A MANUAL OF THORACIC SURGERY (2nd ed.)

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About "A Manual": Every chest surgeon encounters patients with unexpected abnormalities of thoracic anatomy and physiology. These conditions - often life threatening – may evolve rapidly and demand immediate appropriate responses if the patient is to survive. Unfortunately, surgical training programs cannot possibly prepare residents to deal with all such eventualities. Fortunately, they don't need to.

Indeed, thoracic surgical judgement is best developed through careful observations, examinations and discussions of critically ill patients - especially those who are connected to simple chest tube drainage devices. For as such systems function or malfunction, the fundamental logic of thoracic surgery – the essential physiology of respiratory and circulatory systems - reveals itself yet again to physician and nurse. Eventually, if properly handled and interpreted, simple chest tube drainage systems can often reveal more about a patient's recent past, current status and near future than the entire hospital chart.

An easy narrative style and logical presentation of material make this text exceptionally coherent and effective. The innumerable insights and experience-based ideas offered herein will improve patient care and expedite recovery on any thoracic surgical service.

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INTRODUCTION TO THE SECOND EDITION

The total of all information accumulated through recorded history now allegedly doubles every several years. Small wonder that exciting advances are the norm in our recently identified specialty. While recognizing the obvious difference between information and wisdom, there still seems ample justification for a completely rewritten and significantly expanded Manual of Thoracic Surgery. Those familiar with my earlier efforts (Chest Tubes and Chest Bottles, pub. 1970, and A Manual of Thoracic Surgery, pub. 1978) will find that recent advances and refinements fit comfortably onto previously developed concepts and techniques.

Once again, this is a thoracic surgeon's report of what he has found safe, true, practical and rewarding. Therapeutic possibilities that were not explored (for lack of time, interest, skill or opportunity) are clearly differentiated from concepts confirmed to my own satisfaction.

Your hematologist never told you, the things a young surgeon should know;... Fast moving blood is less likely to clot than stagnant blood, unless flow is turbulent;... Some

CHAPTER 1: THE DIAPHRAGM

Anatomy;... Function;... Denervation;... Denervated hemidiaphragm moves paradoxically; ... Eventration;... Diaphragm fatigue and phrenic nerve stimulation; ... Traumatic rupture of diaphragm;... Symptoms of major diaphragm tear;... Radiographic signs of major diaphragm tear;... Direct penetrating injury;... Diaphragm repair;... Morgagni hernia;... Transthoracic hiatal hernia repair using a counterincision;... Transabdominal correction of hiatus hernia;... Transabdominal repair of achalasia;... Diagnosis of congenital Bochdalek defect of diaphragm;... Repair of Bochdalek hernia;... Closure of a large defect in diaphragm;... Slow versus rapid reexpansion of hypoplastic lung associated with Bochdalek hernia;... On stretching the abdominal wall after closure of a Bochdalek defect;... Pulmonary problems after Bochdalek hernia repair; ... Keep them dry!; ... How tight an abdominal closure is too tight?... Case report: Overly-tight ventral hernia repair;... Tight abdominal closure can also cause renal failure;... Acute renal failure following relief of chronic constrictive pericarditis;... Other causes of elevated diaphragm;... Excess intestinal gas;... Prone position for surgery;... Preferential lung ventilation in the lateral decubitus position;... Positive pressure ventilatory support can be fatal;... Esophageal atresia with distal tracheoesophageal fistula;... Other considerations with delayed repair of tracheoesophageal fistula;... Esophageal atresia without distal fistula;... Bowel sounds may be absent during gastrostomy feedings;... Tension pneumothorax is one cause of depressed diaphragm;... Pneumothorax recognized at laparotomy;... A low diaphragm is inefficient;... Free air under diaphragm;... Subphrenic abscess;... Surgical drainage of a subphrenic abscess;... Take care during dissection along diaphragm.

Anatomy

Lined by pleura above and peritoneum below, thin but powerful diaphragm muscles separate thoracic and abdominal contents. Each hemidiaphragm is innervated by its separate phrenic nerve, traveling down the lateral mediastinal surface over pericardium, passing anterior to lung hilum from the neck. The pain of diaphragm inflammation or injury may appear to originate from the same side of the upper abdomen and around back, or from shoulder or neck.

Function

Contraction of a domed-up diaphragm brings down the floor of the chest and stretches lung: Such expansion of the closed thorax causes intrathoracic pressure to fall below atmospheric, which draws air into lungs and aids peripheral venous blood return to the heart. On relaxation, the diaphragm muscle is both pulled upward by lung and pushed upward by abdominal muscles pressing against abdominal viscera. Intrapulmonary air pressure then rises above atmospheric. Normal breathing is consequent to regular variation of intrapulmonary air pressure between more-than-atmospheric and subatmospheric.

Denervation

Damage to innervation may partially or totally paralyze a hemidiaphragm, with the affected portion passively assuming a higher intrathoracic position: A congenital lack of muscle (eventration), large diaphragm hernia or traumatic tear of diaphragm will similarly alter its function. The newborn has poorly developed chest wall muscles and supple ribs, so must rely primarily upon diaphragm movement for effective air exchange. Diaphragm denervation due to phrenic nerve division is generally permanent, although functional recovery often follows many weeks after a hemostat "crush" of the intact nerve.

Phrenic nerve crush or division was occasionally utilized for pulmonary tuberculosis (to decrease

hemithorax size and "rest" the lung), or as an alternative to hiatal hernia repair: (Those who could not reliably keep the stomach "down" could at least bring diaphragm up; diminished subatmospheric pressure in the smaller hemithorax that resulted, was usually associated with less esophageal reflux.) Phrenic nerve was sometimes crushed during esophagogastrectomy (to prevent repeated diaphragm "pull" from disrupting the healing anastomosis) or pneumonectomy (to decrease residual space). Phrenic nerve injury nowadays is rarely purposeful, except during removal of closely adherent tumor, as these other "indications" have been discredited (due mostly to persistent loss of ipsilateral pulmonary function).

Partial denervation of diaphragm often follows central diaphragm incision or repairs. Injury to one or more phrenic nerve branches can cause a localized bulge, simulating tumor of diaphragm or pleura (though a primary diaphragm tumor must be exceedingly uncommon, if it even exists). When major transdiaphragmatic access is required, peripheral detachment of diaphragm from the chest wall preserves its innervation and is therefore preferable to a more central incision. It is usually convenient to leave a short, sturdy rim of diaphragm on the chest wall for easy suture reattachment of diaphragm, unless one intends to relocate that segment of diaphragm in a cephalad direction to diminish hemithorax size; maximum diaphragm length is then best preserved by detachment right at the chest wall.

Denervated Hemidiaphragm Moves Paradoxically

Downward contraction of the opposite normally innervated hemidiaphragm causes passive upward movement of viscera on the denervated side (in response to increased intra-abdominal pressure). Such paradoxical upward movement of a totally denervated hemidiaphragm during inspiration will markedly decrease inspiratory expansion of its overlying lung, which permits the mediastinum to be drawn toward the normal, more subatmospheric hemithorax, diminishing air intake of that lung as well: In effect, therefore, unilateral diaphragm contraction pushes an abnormal, circular pulse of pressure through abdomen and chest.

This circle is reversed on expiration, when the normal hemidiaphragm relaxes upward in response to always-above-atmospheric intraabdominal pressures. Such movement reduces intra-abdominal pressure, allowing upward stretch of paralyzed hemidiaphragm to decrease: Such (paradoxical) downward movement of denervated diaphragm during expiration will inhibit air exhaust from its overlying lung. Mediastinal movement away from the higher pressure (normal) side during expiration will decrease air outflow from the "good" side as well.

Paradoxical movement of diaphragm and upper abdominal viscera may be suggested by diminished breath sounds on auscultation of that side, or abnormal diaphragm excursion detected during percussion; it is confirmed by fluoroscopy of hemidiaphragm movements or comparison of inspiratory and expiratory chest roentgenograms. As one might expect, plication of the paralyzed diaphragm (imbricating the diaphragm in layers by suture through a thoracotomy incision) has been reported effective in improving pulmonary function in good risk patients with a paralyzed diaphragm or eventration.

Eventration

Paradoxical movement may be minimal or absent in eventration, as little elasticity persists in chronically stretched pleuroperitonealmembranes that lack muscular support (a thin, non-muscular

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diaphragm will tend to be supported in a stable, elevated position by intraabdominal visceral pressure).

Diaphragm Fatigue and Phrenic Nerve Stimulation

Reportedly, individuals with congenital or acquired hypoventilation or apnea can benefit from long-term phrenic nerve pacing, by an implanted pacer with an electrode adjacent to one or both phrenic nerves. Although the optimum type of impulse wave and duration of stimulus that can be tolerated is still under study, a number of individuals have been subjected to diaphragm pacing for over 12 hours each day, even for as long as ten years. In many cases this technique has allowed independence from a ventilator for much of each day.

Inappropriate wave forms or too many hours of pacing can apparently lead to nerve changes and diaphragm fatigue. In general, however, the diaphragm is a sturdy, well-vascularized muscle that rarely becomes fatigued unless it has atrophied through disuse during prolonged respirator care, or is at a mechanical disadvantage - as in emphysema (see later this chapter).

Traumatic Rupture of Diaphragm

A hemidiaphragm may be torn by an acute increase of intra-abdominal pressure associated with blunt (usually anterior) abdominal trauma. Major tears commonly involve the central portion of the hemidiaphragm, which receives most of the impact of suddenly accelerated viscera. (Viscera striking the diaphragm more laterally during forceful upward motion will tend to be deflected centrally without significant loss of momentum. Water dams are convex upstream to avoid just such central convergence of forces.)

The right hemidiaphragm is less likely to be disrupted in this fashion, for the entire liver moves upward en masse. Perhaps liver is often split or exploded while dissipating right-sided abdominal compressive forces that on the left side might have torn diaphragm. Splenic rupture, however, usually results only from direct splenic injury: Thus, a major rupture of the left hemidiaphragm will often be associated with an intact spleen up in the chest.

A tear through the dome of the diaphragm sometimes results in "bowstringing" of intact phrenic nerve across the diaphragm defect. Since phrenic nerve approaches the diaphragm from above, it is less at risk from upward injury (which relaxes it) than from a subsequent vigorous surgical repair. The phrenic nerve can often be felt through a large diaphragm defect (from the abdominal side); if watched for, this nerve will not be mistaken for a fascial strand and divided, torn or sutured as viscera are returned to the belly.

Symptoms of Major Diaphragm Tear

A major diaphragm tear should be suspected after significant blunt abdominal trauma, especially if the patient complains of persistent shoulder pain without obvious cause, or post-traumatic epigastric distress, or an inability to swallow, or recurrent vomiting. Other suggestive findings include dyspnea, tracheal deviation away from the side of injury, and decreased breath sounds on the affected side, as well as bowel sounds abnormally present within the chest.

Torn diaphragm generally appears on early chest roentgenograms as elevation of the hemidiaphragm, sometimes with a large air/fluid level (herniated stomach) or "haziness in the left base"

(blood, intestines or spleen) and loss of clear diaphragm outline. Under such circumstances, chest tube insertion for drainage can be both risky and puzzling (see Case #7, Chapter 7).

Radiographic Signs of Major Diaphragm Tear

Any suspicion of diaphragm rupture should be treated immediately by indwelling nasogastric tube and then confirmed by tube location on roentgenogram or a contrast study of the stomach (for evaluation of the left hemidiaphragm), or by a liver scan for liver position, assuming the patient is stable enough for such studies. Otherwise an emergency exploration via chest or abdomen is carried out as indicated.

When stomach appears unremarkable but left hemidiaphragm is high, fluoroscopic evaluation of regular breathing efforts, as well as a study of more rapid diaphragm movements associated with a sniff or cough, may allow demonstration of any diminished, delayed or paradoxical movement of diaphragm and mediastinum. The surgeon should attend such fluoroscopy, if at all possible (or ask that it be recorded for later viewing), for radiologists sometimes miss the paradoxical or delayed diaphragm motion upon which a decision to operate may be based.

Many additional studies have been recommended to detect post-traumatic pleuroperitoneal communications, including introduction of air or fluid into the peritoneal cavity (hoping to visualize or recover this later within the chest); reportedly such tests all have a low yield. Perhaps a peritoneal irrigant containing an aqueous iodine contrast solution (or even a dilute radioisotope) would be more helpful, but I have not tried this.

At present, early surgical exploration seems the most useful test for probable rupture or laceration of the diaphragm (just as a presumption of intestinal volvulus or other internal hernia is considered sufficient indication for laparotomy). Not only is visceral strangulation and perforation an ongoing hazard with any diaphragm defect, but very large diaphragm hernias can also compromise overlying lung function. (In recent years there have been more frequent reports of right diaphragm rupture, often associated with torn liver and hepatic veins. My own experience is almost entirely with left-sided traumatic diaphragm defects.)

Direct Penetrating Injury

The diaphragm is frequently lacerated by a knife, bullet or broken rib. Normal diaphragm excursion can be significant (especially during violent physical activity), so diaphragm injury is always suspected after a penetrating chest wound, and signs of peritonitis or gastrointestinal bleeding watched for (even with a mid- or upper-chest wound from a bullet or long knife). Bullets may follow any straight or irregular path (which is sometimes outlined by bone and metal fragments). They can often be palpated opposite to the site of entry, where elastic tenting of unsupported skin finally stops their advance. Even minor diaphragm lacerations may enlarge with time and entrap omentum or other viscera (often stomach), although liver probably seals many small, right-sided defects, thereby reducing the risk of visceral herniation on the right.

Diaphragm Repair

Unsuspected diaphragm rupture or laceration is often encountered during operative exploration for thoracic or abdominal injuries. While identification and repair of any diaphragm defect can proceed via

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chest or abdomen, liver can interfere with transabdominal exposure of the right hemidiaphragm and retrohepatic vena cava. For diaphragm repair by posterolateral thoracotomy, the seventh interspace is generally most satisfactory; an eighth interspace approach frequently places one behind a bulging lateral diaphragm that must then be retracted in order to gain more medial access. Either a vertical midline or transverse upper-abdominal incision can provide excellent exposure of the diaphragm at laparotomy: A strong table-mounted retractor is of great help during transabdominal diaphragm repair (I use interrupted, 2-0, non-absorbable suture).

When operating through the chest for direct diaphragm injury, careful inspection of hard-to-see costophrenic angles is required, to avoid missing peripheral diaphragm injuries. An anterolateral chest tube is usually placed for postoperative chest drainage, regardless of whether diaphragm repair was undertaken via chest or abdomen (unless the diaphragm was proven intact on abdominal exploration). I have never seen an operation undertaken for ruptured diaphragm in the absence of same, but delay in diagnosis and repair of a major diaphragm tear is unfortunately quite common (sometimes due to distraction of attending physicians by associated injuries).

Morgagni Hernia

In the adult, Morgagni hernia has often been mistaken for pericardial cyst. When such hernia is small, asymptomatic and does not compromise bowel, there seems little justification for repair of this upwardly directed, para-pericardial bulge, often known to have been present for many years. Sometimes, however, the diagnosis is unclear (although sonography or computed tomography may help when other roentgenographic evidence is confusing).

Transthoracic repair of Morgagni hernia is avoided, if possible, for working through a small space in front of the heart (trying to sew diaphragm down toward the upper abdominal wall, away from the surgeon) can be difficult and tedious. During transabdominal repair of a Morgagni defect, one must avoid injury to the nearby phrenic nerve.

Transthoracic Hiatal Hernia Repair Using a Counter-incision

During transthoracic hiatal hernia repair, the surgeon often utilizes a short counter-incision in the diaphragm to help pull stomach down (both ends of a Penrose drain that is looped about the gastroesophageal junction are temporarily brought back into the chest through this counter-incision). Late disruption of a counter-incision repair may lead to strangulation of stomach wall. To avoid such a major complication, the counter-incision (usually in thinner, more tendinous, central diaphragm tissue) is closed very carefully. A counter-incision often causes localized, peripheral diaphragm weakness (because of injury to a branch of the phrenic nerve) which could be mistaken for pleural tumor.

Transabdominal Repair of Hiatus Hernia

If symptomatic hiatus hernia requires surgery, transabdominal Hill repair, perhaps with Nissen fundoplication, now seems preferable to transthoracic correction (if equally skilled surgeons are available for both options), for hiatus hernia patients seem especially prone to persistent, disabling, post-thoracotomy discomfort. Perhaps low thoracic incisions lead to more such sequellae: Certainly, many patients with hiatus hernia symptoms already have significant psychosomatic overlay.

Recurrence of hiatus hernia should be infrequent, whether repaired via chest or abdomen. In an

adult, transabdominal diaphragm hernia repair also allows remedy of unrelated conditions, such as gallstones or ovarian pathology.

Transabdominal Correction of Achalasia:

A good table retractor simplifies transabdominal exposure of the esophageal hiatus and lower esophagus, allowing extensive esophageal myotomy in the usually slender, achalasia patient. To prevent later reflux esophagitis (while still avoiding undesirable narrowing of the already non-propulsive distal esophagus), a transabdominal Heller myotomy is easily combined with the Hill hiatus hernia repair, in order to resecure the freshly mobilized gastroesophageal junction within the abdomen.

Diagnosis of Congenital Bochdalek Defect of Diaphragm

On occasion, a newborn with abnormally scaphoid abdomen and large chest will be found to have dullness and decreased breath sounds or even bowel sounds, usually in the left hemithorax, along with increasing respiratory distress. On roentgenogram, the left "lung" will appear multicystic or show multiple air-fluid levels. A nasogastric tube, and any radio-opaque dyes ingested or inserted via this tube, will pass normally down through diaphragm, then curve back up into the left hemithorax (confirming the diagnosis of abdominal viscera in left chest, if such confirmation is required). Urgent surgical repair is usually essential to allow survival without respiratory support, although I have repaired this defect electively in a two-year-old boy (previously evaluated for "failure to thrive") and an apparently healthy twenty-year-old man (soon after blunt abdominal trauma). In both cases, the Bochdalek defect was encountered unexpectedly during evaluation for intrapleural mass or possible post-traumatic diaphragm rupture.

Repair of Bochdalek Hernia

I prefer transabdominal repair in the newborn, with transverse incision across both rectus muscles (above umbilicus and below ribs). The exact location of such a transverse incision is not crucial, as the loose abdominal wall of infancy makes retraction of any incision easy and atraumatic. Selection of laparotomy over thoracotomy is not simply a question of whether one prefers to "pull" or "push" viscera through the hernial defect, or even which method allows better exposure for diaphragm repair, for these infants also have potential intestinal obstruction from malrotation or peritoneal band across the duodenum. (Although reduction of abdominal viscera through a small diaphragm defect from the chest theoretically leaves one unsure of what abdominal arrangements have ensued, I have not encountered postoperative intestinal obstruction after repair by either approach.)

None the less, at laparotomy, it is relatively simple and atraumatic to stuff intestines back in proper order, while eliminating an occasional Meckel's diverticulum or other likely cause for future bowel obstruction. Gentle, persistent teasing, using opened, dry, 4 x 4 sponges and small ring-forceps such as the Williams clamp (to maintain atraumatic traction on viscera) usually allows reduction of Bochdalek hernia contents with only an occasional minor omental tear or hematoma of the intestinal wall. At times, passage of a finger or catheter through the diaphragm defect (to permit air entry into the chest) will allow easier return of viscera.

The small Bochdalek hernial ring may (rarely) require enlargement to permit passage of displaced abdominal viscera: Good fortune and gentle persistence have so far achieved reduction of Bochdalek hernia contents without such lateral incision in my patients. Once the abdominal viscera have been

removed from the chest, a routine anterolateral chest tube (#8 to #12 French) is inserted through the lateral chest wall. At this time, the easily visible portion of the thoracic cavity is rapidly inspected for lung size and abnormal anatomy (such as pericardial cyst), before beginning closure of the diaphragm defect.

Closure of a Large Defect in Diaphragm

One is often concerned about insufficient diaphragm tissue during closure of larger congenital defects involving the central and posterior diaphragm. More than half the normal diaphragm may appear to be missing when a repair is started. Usually, however, a shelf or ridge of muscle becomes evident behind the left kidney and adrenal gland if these are gently pressed down out of the way. Although they may seem unnecessary, several fine traction sutures along the rim of the defect often help to determine which direction of approximation will result in the least tension (needles are left on these traction sutures so some may be utilized for the closure).

Rather than insert prosthetic material into a large defect, one can also flex the supple infant chest wall inward (by finger pressure from outside the chest) to allow fine-suture approximation of intercostal muscle or rib to the free diaphragm margin. Even if such chest wall-to-diaphragm sutures must be used, no external deformity should result, for newborn tissues heal, grow and readjust rapidly. (However, one patient closed in this fashion, with fine silk sutures around rib and diaphragm edge, developed an "inflammatory chest wall psuedotumor" two years later; it may be that absorbable suture is preferable here, but I cannot comment on that.)

Slow Versus Rapid Reexpansion of Hypoplastic Lung Associated with Bochdalek Hernia

Even with a "hypoplastic" lung, infant tissues are so pliable that an appropriately drained hemithorax can usually be fully occupied by its tiny lung within hours of Bochdalek hernia repair. During this time, gradually increasing (but still "low") chest tube suction will encourage approximation of the small lung to its chest wall, mediastinum and diaphragm. Such suction is most readily applied by an occasional syringe-aspiration of air from the chest tubing above a water seal bottle (to gradually lift a water column into straw and tubing). In the absence of air leak, this simple, low-volume, suction system allows accurate application of chest tube suction (see Chapter 8).

On the other hand, a small (accidental, or purposely retained) pneumothorax will also absorb uneventfully within a few days, barring ongoing pulmonary air leak. I have minimal experience with this passive, less controlled, "no chest tube" method for expanding hypoplastic lung (although equally satisfactory results are alleged). The minor risk of infection with even a temporary pleural space, plus a possibility that new air leak could more easily develop into unsuspected tension pneumothorax in the presence of known simple pneumothorax, has made me reluctant to abandon the use of a chest tube until satisfactory elimination of the pleural air pocket has been achieved: Much of medical practice is similarly based upon personal preference rather than scientific fact.

On Stretching the Abdominal Wall After Closure of a Bochdalek Defect

One sometimes has difficulty closing the tiny abdominal cavity without undue compression of its newfound viscera. This is usually "no problem," as an infant's anterolateral abdominal wall can be stretched significantly. Once diaphragm repair has been achieved (with viscera still mostly displaced onto the abdominal wall), before stuffing intestines back into the abdominal cavity, merely place two

fingers of each hand within opposite sides of the lower abdomen and push the abdominal wall firmly upward and outward, avoiding enlargement of the incision itself by tearing. On occasion, such forceful abdominal dilation (required because viscera did not fit) has caused an umbilical cord to "pop off" internally, but that was simply clamped and ligated without problem (although this could be a greater nuisance if umbilical vessels are cannulated).

One could probably also delay abdominal closure by interposing silastic sheeting between the muscle-and-fascia edges of the incision, then bringing these edges together in stages (as with the "silastic funnel" used for omphalocele repair), though I have not needed this; or even mobilize skin and subcutaneous tissue bluntly from underlying muscle, widely enough to at least achieve visceral coverage by skin. There is more stretch to skin than muscle, and such mobilization was formerly the standard method for achieving omphalocele repair: At times, dissection was even carried around the back of the infant to the midline without apparent ill-effect. The large ventral hernia that resulted was then repaired electively as the child grew. This has not been recommended as far as I know, nor has it been indicated in my own experience with Bochdalek hernia, but another "fall-back position" never hurts.

Abdominal wall closure can be two layers of running absorbable suture, using steristrips to approximate skin edges. Total fluid requirement before and during such procedure, is normally about 50 cc of 5%D/W. Postoperative feeding, starting with dilute sugar solution, can resume whenever the patient is hungry, often within hours.

Pulmonary Problems after Bochdalek Hernia Repair

Newborns with Bochdalek hernia are particularly sensitive to fluid overload. Surprisingly, the mortality of surgical repair within the first 24 hours of life seems to have increased markedly with the advent of neonatology and modern techniques of fluid resuscitation. It no longer appears well known that one should entirely avoid transfusion (or at most, simply replace measured blood loss) of these initially over-hydrated infants. One must remember that all newborns start "wet," and that patients with Bochdalek hernia do not die of renal shutdown, they die of wet lungs.

Keep Them Dry!

If you can prevent any preoperative "fluid resuscitation," and restrict hourly fluid intake after diaphragm repair to 10 cc of 5% D/W or less (for normal size or smaller newborns) until the infant resumes oral intake, you should return a long way toward the functional ("dry") lungs we used to see before we lost control of postoperative newborns to these highly trained and competent "Nursery Commandants." Just as teenagers are sure that they have invented sex, neonatologists cannot believe that successful newborn care antedated their subspecialty.

Interestingly, the current high mortality rate of neonatal diaphragm repair is being touted as a reason to detect and proceed with such repair while the infant is still in utero (a further technical advance that endangers both mother and child). Perhaps it is inevitable that unique training, terminology and equipment, used in highly technical, often life-saving procedures, will increasingly isolate the ever-narrowing interests of the subspecialist from past human experience, which remains the source of that feeling for what is reasonable and likely, known as "common sense."

Summary: Newly expanded lungs are always "wet." If one lung is also markedly hypoplastic, these

tiny patients become the physiological equivalent of an infant who has undergone pneumonectomy. We have all learned "the hard way" that postoperative adult pneumonectomy patients must be kept "dry" (for they readily develop dangerous lung congestion). These newborns, having no "pulmonary reserve" to start with, are even more easily tipped into an ever-descending spiral of pulmonary vascular spasm and shunting that, all too often, ends fatally.

Although a newborn with Bochdalek hernia is generally treated as a surgical emergency, neonatal intensive care on a respirator has made it less so. Is it possible that a desperately ill newborn patient with Bochdalek hernia could be maintained on a respirator without being given any significant fluid intake until he is several days old? Would he then be a better risk for semi-elective diaphragm repair?

How Tight an Abdominal Closure is Too Tight?

That is hard to judge, especially when anesthesia is light and relaxation incomplete, but hypotension (due to interference with return of venous blood), and hypoxia, hypercarbia and acidosis (due to inability of the diaphragm to contract against overly-compacted viscera), are always a concern. Cardiac arrest can be early evidence of such a problem, so especially close monitoring is indicated during (and soon after) surgery.

Case Report: Overly Tight Ventral Hernia Repair

During an externship in Newfoundland (nearly three decades ago), a massively obese, middle-aged female requested repair of an enormous umbilical hernia, the contents of which, in retrospect, had "lost their right of domain" within her abdomen. Serene in our ignorance, we had achieved a very tight repair (under open-drop ether anesthetic) when the surgery was terminated unexpectedly by cardiac arrest. Resuscitation failed, as was usual for that time and place.

On departing, her surgeon ordered us to transfer the corpse from the second floor surgery down to the basement morgue (via a steep and narrow staircase, there being no elevator to the basement). As assistant surgeon, it fell upon me to help effect this transfer. Being taller bearer, I went first, choosing the somewhat lighter foot-end. Exerting every muscle to keep the stretcher up and level, we proceeded down the stairs. George (the orderly) held his end low by stooping, while supporting a good part of her more than 300 pounds. The first several steps passed uneventfully, although each produced a more noticeable tilt of the litter.

At five steps down, her sheet slipped off, to hang about my neck like a scarf. Two more steps and the tight turn at midflight landing were maneuvered with gradually folding ears, as cold, stiff ankles moved relentlessly forward along my head. Emphatic suggestions to George to lower his end were answered by desperate shouts to lift higher. The next three steps passed more rapidly, as icy knees reached my shoulders. She left the litter and landed full astride my neck, three or four steps above the first floor. Together we burst into the thirty-bed ward through swinging double doors, then I stumbled and she rode me to the floor.

Conclusion: The outcome of overtight abdominal closure can be crushing to a young surgeon.

Tight Abdominal Closure Can Also Cause Renal Failure

Complications of extremely tight abdominal closure in the post-trauma adult can allegedly be

avoided by delayed or staged reapproximation of incisional edges, again using temporarily interposed, silastic sheeting. As visceral fluid and gas content, vascular congestion and edema gradually diminish, the separated edges of rectus muscle can then be brought together more easily, to permit completion of the abdominal closure in one or more stages.

Intra-abdominal pressures that remain persistently above 25 cm water in the early postoperative period can reportedly cause acute renal insufficiency, even with apparently adequate cardiac output and blood pressure. Urgent reexploration (for abdominal decompression) has been recommended for patients who develop oliguria in association with such elevated intraabdominal pressures. These pressures are measured from the pubic level by instilling 100 cc of sterile saline into the bladder through the Foley catheter, the end of which is then clamped while intra-catheter pressure is determined, using a hollow needle as side-arm. Presumably one does not have to consider bladder muscle tone during such pressure determinations soon after surgery. Although I have not used this measuring technique, I have encountered posttraumatic and postoperative renal failure, so reexploration for this indication seems an interesting concept to pursue.

Discussion: This reportedly hazardous level of intra-abdominal pressure (above 25-30 cm H2O) is in the range of normal capillary pressures; thus probably also the maximum back-pressure against which one might expect urine delivery into the bladder. When this amount of pressure is sustained on or within the ureter or renal pelvis, perhaps it can stop forward urine flow and thus lead to progressive accumulation of cellular debris and urinary sediment within renal tubules. Presumably similar "back-pressures" cause the renal damage associated with advanced urinary tract obstruction. It is interesting that comparable levels of (mean) pulmonary capillary pressure can lead to pulmonary edema; this 25 cm H2O figure also approximates the inflation pressure within an endotracheal balloon that is known to cause ischemic death of tracheal mucosa; it is also near the maximum venous pressure that can be survived in chronic constrictive pericarditis. Perhaps we can now explain why acute renal failure is occasionally encountered following pericardiectomy for severe constrictive pericarditis.

Acute Renal Failure Following Relief of Chronic Constrictive Pericarditis

Consider first that with stable cardiac constriction, tissue pressure equals and opposes the gradually increased intra-capillary pressure minus the osmotic gradient. The rapid decrease in venous pressure that is expected during successful pericardiectomy would suddenly leave that chronically elevated tissue pressure unopposed: An easily visualized consequence might be renal capillary compression by that unopposed residual renal parenchymal edema within a tight kidney capsule. Should we therefore bring inferior vena caval pressure down more gradually after pericardiectomy (see discussion in Chapter 15)? In any event, it seems appropriate to maintain vigorous mannitol diuresis whenever elevated renal tissue pressure could become a threat.

Other Causes of Elevated Diaphragm

Many conditions, such as advanced ascites, extreme obesity, pregnancy or other intra-abdominal tumor, will elevate the diaphragm and compress lung, thereby interfering with air exchange to some degree. Therapeutic pneumoperitoneum - occasionally utilized in the past to raise both hemidiaphragms and so "rest" the lungs of tuberculous patients - could also encourage sufficient abdominal wall growth to allow delayed visceral replacement after omphalocele repair. Reportedly, induced pneumoperitoneum is still useful during preparation for surgical correction of a massive ventral hernia, with increasing volumes of air instilled by indwelling catheter over one to three weeks as tolerated

(until fascial edges of the hernia defect can be palpated).

Excess Intestinal Gas

Large amounts of gas within the small bowel can also cause major abdominal distension. A post-laryngectomy patient who utilizes air swallowing for an "esophageal voice" should therefore be asked to "shut up" for the two days preceding elective laparotomy, to expedite surgery and reduce the likelihood of postoperative incisional and pulmonary morbidity (consequent to his normal state of tightly compressed, gas-filled intestines). Hiatus hernia repair also, is often followed by ongoing unsuccessful attempts to belch, especially with a more nervous patient. One must often repeatedly explain to such patient that each voluntary attempt to "burp" results in swallowing of more air and leads to increasing abdominal distension rather than relief. Nasogastric intubation may occasionally be required to limit this problem.

Prone Position for Surgery

A beached whale soon suffocates. Likewise, it may be difficult or impossible to ventilate a "pot-bellied" patient safely in the prone position, for the operating table supporting his fat belly will compress his bulky viscera, thereby shoving diaphragms up against lungs: Enlarged emphysematous lungs simply make this situation worse. The ensuing "sudden" death (consequent to inadequate venous return and progressive hypoxia, acidosis and hypercarbia) can be most resistant to resuscitation.

While I have no evidence that a beached whale could breathe (or be ventilated) more readily in the lateral (or supine) position, that is certainly true for fat folk with "beer bellies." Therefore, I plan to turn the next beached whale that I encounter onto his side (even though he could get sand into his "down" eye), in order to see if that position improves his air exchange. Positive-pressure ventilation and elevating the tail to improve venous return might also be helpful, pending return of the tide.

Preferential Lung Ventilation in the Lateral Decubitus Position

Interestingly, it is alleged that an infant lying upon his side will preferentially ventilate the uppermost lung, so that very young children with unilateral lung disease (e.g. air trapping) and marginal oxygenation can often improve their gas exchange when positioned with the diseased lung dependent. Apparently the reverse is true in adults, who derive ventilatory benefit if they lie upon their side with the good lung "down" (but that "adult" pattern again reverses during muscle relaxation and positive-pressure ventilation; then air exchanges most readily into the uppermost lung). Presumably one can explain such findings by the far greater pliability of infant or "surgically relaxed" tissues, and the greater stretch exerted upon the uppermost diaphragm in the adult as the "hanging" mediastinum - and heavy viscera bulging the abdominal wall - both tend to "straighten" and render "less effective" the uppermost hemidiaphragm in the lateral decubitus position.

Positive-pressure Ventilatory Support Can Be Fatal

Additional intestinal gas is particularly harmful to any newborn patient already enduring lung compression by viscera. The classic example is Bochdalek hernia, where positive-pressure ventilation by mask is carefully avoided (and the endotracheal tube placed early) to prevent progressive cardiorespiratory compression by further inflation of bowel trapped within the thorax. Here, an indwelling nasogastric tube will also assist in preoperative removal of swallowed air.

Ventilatory assistance (by face mask or endotracheal tube) can rapidly cause fatal tension pneumothorax if there is any air leak from lung into a closed pleural space (a bronchopleural fistula) unless the pleural space is simultaneously decompressed to the atmosphere (see Chapter 5). Similarly, positive-pressure breathing assistance is avoided when there is wide-open air leak from respiratory tree to bowel (as via a distal tracheoesophageal fistula), or any anatomic abnormality of lung that permits progressive pulmonary air trapping (as seen with congenital lobar emphysema). At thoracotomy for lobar emphysema, positive-pressure breathing can safely be initiated through a previously-placed endotracheal tube as soon as the chest is expeditiously opened and the affected lobe becomes free to balloon forth (see Chapter 13).

Esophageal Atresia with Distal Tracheoesophageal Fistula

A happy circumstance for the preoperative patient with esophageal atresia is roentgenographic visualization of intestinal gas (confirming that a distal tracheoesophageal fistula is indeed present), for this practically guarantees sufficient distal esophageal length to permit primary repair. The skilled anesthesiologist allows such an infant to inhale sufficient anesthetic for surgery to proceed, with the patient kept "light" and breathing spontaneously until the chest has been opened.

The patient can then be ventilated lightly during ligation and division of the azygous vein, which otherwise blocks access to the "fistula" (misconnected distal esophagus). The fistula is then identified and gently suspended on a loop of heavy suture. Thereafter, surgery proceeds with fully controlled ventilation, as the fistula is meticulously ligated "flush" with the membranous tracheal wall, then cut off (leaving only a small button of esophageal tissue at the ligature). Extremities are restrained under circumstances when anesthesia will necessarily be "light." Incidentally, inhaled nitrous oxide aggravates and 100 percent oxygen diminishes any gaseous distention of the intestine (see discussion in Chapter 3).

Other Considerations With Repair of Tracheoesophageal Fistula

It may well be that any "successful" esophageal repair would benefit from leaving a persistent intraluminal stent through that site until oral intake can begin. My only case of complete postoperative esophageal occlusion (requiring revision by thoracotomy) occurred in a three-pound neonate who remained on respirator care for many weeks after a technically satisfactory correction of esophageal atresia: When this child was finally weaned from the respirator (many weeks following our probably inadvisable early repair and a consultant's impulsive, premature removal of his nasogastric tube), no residual anastomotic lumen remained to be dilated; indeed, persistent probing at another institution apparently led to an abscess around the anastomosis that further delayed functional repair.

Anastomotic narrowing of mild to severe degree may occasionally require frequent postoperative dilation with a mercury-filled tube to retain or regain an adequate anastomotic lumen. An infant on liquid diet can develop severe stricture with only minimal symptoms, so it is well to check for esophageal narrowing during early postoperative weeks by repeat esophagram or gentle passage of an appropriate-size mercury-weighted esophageal bougie: To reduce the risk of oil or food aspiration, a water-soluble lubricant is used on the bougie, and the esophagus is only dilated when several hours have passed since the last feeding.

I have not found it necessary to perform circular myotomy of the proximal pouch to gain length, although reportedly this has little risk if needed to reduce anastomotic tension: Such myotomy

supposedly does not alter later esophageal dynamics. Following successful esophageal anastomosis, some of these patients may develop symptomatic gastric reflux that can require additional medical maneuvers (upright or prone position after feeding, frequent small meals) for a period of time.

Early surgical correction of "hiatus hernia with reflux" in these patients may be unwise but I have no relevant experience to relate. Reportedly, chronic bronchitis and asthma can result from persistent gastroesophageal regurgitation or inadequate esophageal clearing. The esophageal discoordination that commonly persists between proximal and distal esophageal segments may permanently slow swallowing: However, at least some of these patients have been able to drink directly from a clean Alaskan stream in the prone position.

Esophageal Atresia Without Distal Fistula

X-Ray examination soon after birth will usually show gas in the stomach or bowel of any newborn, unless he has esophageal atresia without distal tracheoesophageal fistula. Esophageal atresia is also suggested by salivary drooling and overflow aspiration into lungs (especially in a supine patient), and confirmed by inability to pass an appropriate-size orogastric tube for aspiration of stomach content.

While I am not aware of having missed a "double fistula," this error apparently does occur, with or without a preoperative contrast study of the proximal pouch (such fistula can arise above the distal end of the proximal pouch and then angle sharply upward to the cervical trachea): Continued aspiration of food and saliva through such a "recurrent" (actually persistent) fistula can cause ongoing pulmonary morbidity or even mortality. It has been recommended that a proximal "pouchogram" be performed in a lateral position to improve visualization of the anterior esophageal wall, and the study recorded on videotape.

I have generally avoided preoperative contrast study of the proximal pouch because of the high likelihood of barium overflow into the lungs during the examination; even without such a study, aspiration pneumonia is usually the major risk faced by a patient with tracheoesophageal fistula. While it seems likely that a barium study could identify an occasional fistula to the proximal pouch that might otherwise be missed (especially with a longer pouch that requires little mobilization), the potential for increased perioperative pulmonary morbidity due to barium aspiration must also be considered.

It has been alleged that there is no need for barium anyhow, as a "high KV" tracheogram is supposed to improve pouch visualization. If a contrast study is considered essential, a dilute barium sulphate suspension reportedly causes the least pulmonary inflammation if aspirated: No more than 1 cc of diluted barium sulphate is injected through a small end-hole catheter into the proximal pouch.

With a blind proximal esophageal pouch and no visceral air below the diaphragm (thus no continuity of respiratory tree with bowel), past experience suggests that the distal esophageal remnant is usually too short to allow primary anastomosis. Modern intensive care techniques (including continuous aspiration of the proximal pouch and intravenous alimentation) have reduced the rush to operate upon these babies, but eventually one must either perform cervical esophagostomy and gastrostomy (after which the patient is committed to later gastric tube or intestinal bypass of the atretic esophagus), or refer the patient to a surgeon especially adept at making short esophageal ends meet.

If esophagostomy and gastrostomy are selected, they can be performed sequentially during one anesthesia: One should probably perform the gastrostomy first in order to allow roentgenographic

evaluation of the amount of distal esophagus available (by transgastric passage of a radioopaque catheter up the distal esophageal lumen: A lateral roentgenogram with a centimeter ruler behind the spine and another tube pressing lightly on the proximal pouch can apparently allow accurate appraisal of the gap remaining between blind esophageal ends).

Cervical esophagostomy shortens and anchors the cervical esophagus, so it should be avoided while primary anastomosis remains a consideration (provided pulmonary spill of saliva can be prevented in the meanwhile). Any congenital fistula from the proximal esophageal pouch to the trachea can be ligated and divided via the neck; thus thoracotomy is generally unnecessary for patients with esophageal atresia in whom primary esophageal repair is not practical.

Bowel Sounds May Be Absent During Gastrostomy Feedings

Bowel sounds are often absent for months following gastrostomy for esophageal atresia, as no air enters the stomach during gastrostomy tube feedings. This means that the skinny, crying, postgastrostomy child with a hard, silent abdomen and legs drawn up is likely hungry, not suffering from peritonitis; so if the gastrostomy tube is patent and in proper position, one can usually go on with tube feedings (but do not expect to elicit a postprandial "burp"). It is recommended that the child also be allowed oral intake (which simply drains out of the cervical esophagostomy) during his gastrostomy feedings, so that he develops some concept of normal eating prior to his esophageal repair at about one year of age.

Tension Pneumothorax is One Cause of Depressed Diaphragm

Normal diaphragm domes up into the chest on expiration and flattens out during deep inspiration. Only free air or fluid, abnormal tissue or tumor mass, severe air trapping in lung, or excessive ventilation pressure by the anesthesiologist (a stuck valve, perhaps) can actually push diaphragm down into the abdomen. Tension pneumothorax should be suspected whenever a hemidiaphragm bulges into the abdomen during laparotomy.

Pneumothorax Recognized at Laparotomy

Normal lung is easily palpated through relaxed diaphragm. With a little practice, free air and fluid around lung are also readily identified by "feel" and gentle ballottement. When pneumothorax is recognized from within the abdomen, a temporary slit or hemostat puncture through the diaphragm (away from mediastinal structures) could be used to decompress the chest (such puncture might even be preceded by diagnostic needle aspiration). However, immediate closed thoracostomy is the rapid and definitive (meaning "best") treatment for pneumothorax discovered at laparotomy.

A chest tube is readily inserted through the lower lateral chest wall while an intraabdominal hand protects viscera and guides the tube (by palpation and manipulation through the relaxed diaphragm) into an appropriate anterolateral intrathoracic position. The low, lateral chest tube insertion site is usually within the skin preparation zone for upper abdominal surgery, so there need be no contamination of the surgical field by this minor additional procedure.

A Low Diaphragm is Inefficient

A maximally contracted diaphragm is almost straight (horizontal). The upward distance that it then

moves during expiration is determined not only by its relaxed length and the intraabdominal pressure but also by the "relaxed" size of the lung above (plus any other space-occupying collection of air, fluid, tissue, or other foreign material). Upward diaphragm movement is markedly limited in any "full-thorax" condition such as emphysema: That abnormally short range of diaphragm motion is inefficient, as is the reduced interval for respiratory muscle relaxation; yet any further compression of an "already full" thorax by "normal" upward travel of the diaphragm is not tolerated, as it would compromise venous return and cardiac function.

The distressed, full-thorax patient is often markedly relieved by positive-pressure ventilatory support (which increases the downward range of diaphragm movement, thereby, in effect, enlarging the previously limited chest cavity). Not uncommonly, a patient who "does fine" on a respirator that simply supplies room air "under pressure" may, when "on his own," rapidly become short of breath or perhaps even unable to move minimal volumes of air. It seems that some patients with terminal emphysema die as much from inefficient or inadequate diaphragm excursion as from any inability of their lungs to oxygenate enough blood (see Chapters 2 and 13).

Free Air Under Diaphragm

Free air under either hemidiaphragm is best detected in an upright roentgenogram (with the patient remaining upright for several minutes before the film is exposed, if possible). Such free air may result from intraperitoneal perforation of an air-containing viscus, or it could merely represent residual air remaining after laparotomy (especially with cachexia or following removal of a large intra-abdominal mass). Any open drain from the peritoneal cavity normally permits air to escape as muscles tighten: With a scaphoid abdominal wall, however, air may enter the abdominal cavity instead.

On rare occasions, such intraabdominal air may reappear from a chest tube located above a repaired diaphragm, thereby simulating lung air leak. Tiny congenital perforations in the diaphragm may also be associated with passage of air or fluid from the peritoneal cavity to the (usually) right pleural cavity (as is discussed in Chapters 3 and 5). "Benign" intra-abdominal air accumulations (those not associated with infection or perforated viscus) will reabsorb uneventfully in days or weeks.

Subphrenic Abscess

Fluid or infection can localize under or over either hemidiaphragm. A sterile effusion may collect above the diaphragm in response to infection under it. In a septic patient, a diagnosis of subphrenic abscess (located just beneath the diaphragm) has sometimes been difficult or impossible to confirm without surgical exploration. Prior to the advent of clinical sonography and computed tomography, helpful tests for subphrenic abscess included fluoroscopy for diaphragm motion, contrast studies of subdiaphragmatic viscera such as stomach and colon (to identify downward displacement), or even direct angiography of nearby structures, as well as combined lung-liver radioisotope scans. The latter tests are probably no longer relevant to the diagnosis of this problem.

Purulent intrapleural fluid (empyema) is usually widespread throughout the chest cavity initially (although often loculated later) unless of gradual onset or involving already-scarred pleura. On the other hand, subdiaphragmatic purulent collections may rapidly "wall-off" in pockets, due to the early response to inflammation by omentum and adjacent viscera, and the normal patterns of visceral attachment to other viscera and the diaphragm.

Surgical Drainage of a Subphrenic Abscess

An extraperitoneal, lateral or posterior approach to subdiaphragmatic pus collections (via the bed of a nearby but lower, resected rib) was once considered the safest way to avoid pleural or peritoneal contamination during drainage of a subphrenic abscess. Transabdominal drainage now seems equally effective, if intervening visceral adhesions do not preclude safe access, for laparotomy often allows more widespread evaluation and "cleanout."

Modern antibiotics have decreased the significance of any rapidly cleared spill of pus. In fact, sequellae of most spills of infected material within the chest or abdomen should usually be minimal, provided copious irrigation with warm physiologic salt solution and gentle cleanup of all particulate material is backed up by effective antibiotic coverage and appropriate drainage. Of course, when accurate tube placement in one of those "hard to reach" places might conceivably be an appropriate substitute for open surgical drainage of an abscess, it is usually wise to confer with an "interventional radiologist" in whom you have confidence.

The huge or recurrent subphrenic collection of pus is occasionally drained most safely and easily through a lower chest wall hole created by resection of an overlying rib, with peripheral disconnection and higher reattachment of diaphragm. Here, careful placement of the incision (over and paralleling the rib to be resected) is essential: If the pocket has been approached laterally, and the incision appears undesirably high, improved dependent drainage is often possible by extending the incision and rib resection forward and down, along the oblique course of the lateral ribs.

Such a drainage incision should never divide the chondral arch, of course, as a distressing thoraco-abdominal hernia can result. In addition, cartilage infection is allegedly more likely than the remote possibility of bone infection consequent to locating drainage tubes next to the ribs (although I cannot recall any rib or cartilage infection that resulted from tube drainage of a nearby pus collection). If the final incision seems too long, its posterior portion may be loosely reapproximated with one or two fine monofilament sutures through skin.

Careful cleanout of subphrenic debris by this approach, locating the chest tubes well away from drains entering the subphrenic space, has allowed uncomplicated recovery of the critically ill patient with a huge abscess. The large, stiff-walled, subphrenic space is easily marsupialized (until it granulates shut) by reattachment of diaphragm to chest wall dermis (on the upper side of the incision), using a few interrupted monofilament sutures. This higher reattachment of a peripherally detached section of diaphragm effectively seals the chest and separates it from both the intra-abdominal abscess cavity and the outside world. Cleanout of such a directly exposed subphrenic cavity at surgery is simple and convenient, and drains or catheters for irrigant solutions are easily positioned and readily replaced (see also empyema drainage, Chapter 4).

Take Care During Dissection Along Diaphragm

Surgical dissection along the upper or lower surfaces of a scarred or inflamed diaphragm commonly results in diaphragm penetration, as the softer muscle layer (between scarred pleura and fibrotic peritoneum) tends to be the easiest plane for blunt or sharp dissection. Extra care taken to avoid such penetration can prevent undesirable bleeding or even transdiaphragmatic spread of infection.

CHAPTER 2: THE CHEST WALL

Inspiration;... Expiration;... Anatomy;... Intercostal arteries bypass coarctation of the thoracic aorta;... Intercostal nerve injuries; ... Intercostal nerve block;... Case report: Intercostal nerve block;... Major intercostal denervation;... Identification of ribs; ... Rib location through intact skin; ... Thoracotomy; ... Posterolateral exposure of the rib cage at thoracotomy;... Selection of an appropriate posterolateral interspace for chest entry;... Ribs are identified for accurate rib cage entry;... Rib identification is confirmed from within;... Resection of an anterior rib can lead to flail;... Rib resection;... Intercostal approach;... Closure of chest wall muscles;... Thoracoabdominal incisions;... Submammary transsternal entry;... Emergency incision for open cardiac massage;... Anterior thoracotomy;... Congenital defects of the chest wall;... Cardiac displacement with pectus excavatum;... Repair of pectus excavatum;... Details of rib resection technique;... Surgery for chest wall tumor;... Case report: Maturation of malignancy in newborn;... Comments on chest wall malignancies;... Postoperative care after chest wall resection; ... Thoracoplasty;... Apicolysis;... Plombage;... Osteoplastic thoracoplasty;... Standard thoracoplasty;... Obliteration of an infected space by muscle transfer;... Case report: Extrusion of oleothorax;... In how many ways can a restricting thorax be enlarged?... Chest wall infections;... Chest wall injury and "sucking" chest wounds;... Emergency decompression of tension pneumothorax;... The open chest wound and the airtight dressing;... Effects of a small opening through the chest wall;... The wide-open chest;... Flail chest;... Avoid loose sternotomy closure;... Sternal fracture;... Case report: Fractured sternum.

Inspiration

As a consequence of progressive thoracic enlargement during normal inspiration, intrapulmonary air pressures remain subatmospheric until chest expansion is complete. At that point, the spine has straightened enough to lift the rib cage from the abdominal viscera, the normally oblique ribs have moved up anteriorly and out laterally, and diaphragm flattening has pushed abdominal contents down from the chest to bulge the abdominal wall.

Expiration

During expiration, intrapulmonary air is compressed to pressures continuously above atmospheric, as relaxing thoracic muscles allow the rib cage to gradually slump down and in, while abdominal muscles press viscera back up against the relaxing diaphragm. Forced expiration brings additional muscles into play that (1) flex the spine and force the rib cage down against abdominal viscera; (2) directly squeeze the rib cage; and (3) tighten trunk and abdominal wall muscles: Increasingly compacted abdominal viscera then drive the relaxed diaphragm ever higher within the chest.

Comment: The goal of inspiration is to maximize the volume within a given surface (i.e., to cause the chest to approach spherical shape). An ideal expiration would then flatten and curl the thorax to squeeze out any residual air. However, the function and protection of our "air pump" (which concentrically encloses and interacts with the "blood pump") requires a relatively rigid shape that makes it impractical for us to inflate and deflate completely. If we lived only to breathe (rather than vice versa), we could probably come up with a number of more interesting designs.

Anatomy

The bone and muscle chest cage surrounds lungs and heart and provides forelimb attachment. Narrow at its apex, the thoracic cavity gradually widens to the diaphragm level (the upper two ribs and

interspaces are especially short). External body dimensions, however, remain relatively constant, because shoulder muscles fill out the upper torso.

A "large-chested" individual may have ample chest wall muscles or breasts overlying a rib cage of any size. Similarly, the rib cage of a very obese individual need not be larger than that of a very thin person, despite obvious differences in girth.

Bony ribs attach posteriorly onto vertebrae: From here, they curve laterally, downward and forward, then finally medially to their costochondral juction. All but the lowest two ribs have a flexible anterior cartilaginous connection to the flat, midline, usually tripartite, sternum. Ribs 8 - 10 usually connect by cartilage to the rib above, rather than directly to the sternum: The interconnected anterior extent of these ribs is known as the chondral arch. Ribs 11 and 12 are progressively shorter (to provide posterior protection without limiting flexion and extension of the trunk). Two oblique layers of intercostal muscle connect adjacent ribs. One layer lifts ribs forward, up and out, when it contracts; the other layer pulls them down and together. Neck, chest, back, abdomen and forelimb muscles also contribute to rib movements during respiration. With advanced age, the chest cage becomes less mobile, so the diaphragm component of respiration once again becomes increasingly dominant.

A smooth layer of parietal pleura coats the innermost aspect of the rib cage. Intercostal vessels and nerves run just beneath ribs (thus protected by their bony overhang) except near the midline posteriorly. An intercostal needle or tube puncture of the chest wall is unlikely to damage vessels or nerve if it traverses the lower part of an interspace (grazing the upper edge of the more caudal rib). Conversely, intercostal space penetration along the inferior border of any rib increases the risk of intercostal artery bleeding and nerve injury.

Due to excellent collateral, any number of intercostal arteries can be transected for removal of a chest wall tumor, or disconnected during graft replacement of the descending thoracic aorta, without causing ischemia of the chest wall (although spinal cord circulation may possibly be endangered with the latter surgical procedure, due to extensive loss of these vessels or to the often-associated intra-operative, distal hypotension and hypoxia).

Intercostal Arteries Bypass Coarctation of the Thoracic Aorta

In the special case of coarctation of the aorta, however, maximum collateral blood flow through chest wall arteries is required to achieve functional bypass of the severely narrowed segment of upper thoracic aorta. Intercostal flow here is reversed, going from hypertensive subclavian artery branches back through intercostal arteries into the lower-pressure descending thoracic aorta: Adequate distal aortic circulation (to viscera, spinal cord, lower trunk and legs) is thereby maintained.

Since perfusion of chest wall tissues is not altered by all of these very enlarged arteries (which function as an out-of-place aorta), chest wall veins remain normal in size and number. The hypertrophic internal mammary artery encountered under these circumstances may be of sufficient caliber to allow use of an open segment of this vessel as a "live" patch across the coarctation site. Similarly, after subclavian artery patch aortoplasty, the distal internal mammary artery has been implanted into the aortic arch to restore subclavian arterial flow. Such use of this enlarged vessel seems sensible, and early results are reportedly encouraging (but I have no experience with this).

Abnormally dilated arterial collaterals within the spinal canal can apparently (on rare occasion)

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cause direct pressure injury to the spinal cord of a patient with coarctation. Usually, however, the tortuous, perhaps palpably enlarged, intercostal arteries become apparent only through "rib notching" ("wavy" bone resorption along inferior rib borders that often becomes detectable on chest roentgenogram after the first decade of life). At times, the external aortic narrowing and post-stenotic dilation may resemble a "3" on PA roentgenogram. Notching of lower ribs (8-10) may indicate an unusually distal coarctation in the thoracic aorta. The significant hypertension commonly associated with marked coarctation can lead to major stroke, even in the first decade, as well as diminished stature (due to decreased growth of lower extremities).

It is the tortuosity of dilated intercostal arteries ("beating" against the inferior rib margin) that accounts for the rib erosions: Apparently these small arteries can only enlarge in diameter during simultaneous growth in length (which is certainly appropriate for the natural growth of vessels in young tissues). Perhaps only diseased (aneurysmal) arteries can enlarge solely in diameter (through vessel wall aging, over-stretch and breakdown). Hopefully, our ischemic heads, hearts and legs will soon be able to demand "More blood!" through such tortuous collaterals in the chemical fashion of tumors and young tissues, rather than rely upon costly and less effective surgical efforts.

Intercostal Nerve Injuries

Intercostal nerves are commonly damaged at thoracotomy by retractor, suture or electrocoagulation. During recovery from such temporary nerve injury, patients often complain of annoying numbness, crawling, itching, stabbing, or other dysesthesias. These symptoms usually resolve within weeks or months. Ongoing painful "neuroma" problems may subside after intercostal nerve blocks (using a long-acting local anesthetic).

Electrical counter-stimulation of skin (as by the portable Medtronic unit) and acupuncture have both been advocated for relief of postoperative chest wall (or other) discomfort. One of my patients had outstanding relief of "excruciating" postoperative saphenous neuralgia (which started about ten days after coronary bypass surgery). This subsided nicely in the course of several days very strong electrical counter-stimulation to his lower leg (at an "unpleasantly high" setting, he obtained "blessed relief." I remain interested but unsure of the applicability of either modality).

Intercostal Nerve Block

Intercostal nerve block occasionally eventuates in pneumothorax due to penetration of a lung bleb. This may require treatment by chest tube. A paravertebral intercostal nerve block can allegedly also deposit drug within an extended dural sleeve, allowing drug reflux toward the spinal cord and unintentional spinal anesthesia (even transverse myelitis could be caused by ethanol injected here for long-term pain relief). Persistent postoperative complaints of intercostal neuralgia may indicate psychological problems.

Case Report: Intercostal Nerve Block

I have performed only one intercostal nerve block in my office (using xylocaine). This lovely young lady almost instantly developed a severe "local anesthetic reaction" with fierce tremors, gasping and collapse. She was apparently closer to death than to the nearest oxygen tank or epinephrine (hundred of yards away). I even considered "mouth to mouth" before she eventually "came around." My secretary drove her home, after a suitable recovery period, when all three of us showed only fine

residual tremor.

As her pain still persisted, I asked my anesthesiologist colleague to try another block (different medicine, more skill). Shortly thereafter he called with shaking voice to suggest that I take my business elsewhere. There was some question of whether drug had actually been injected before her second severe "drug reaction," so on her next office visit I recommended psychiatric evaluation: Immediately she developed her third, most severe "drug reaction" to date, complete with frothing. This subsided after I walked out, leaving my sensible secretary to keep her company. The psychiatrist never saw her, nor did she ever return to my office.

Conclusion: (1) Intercostal nerve block may sometimes relieve or clarify intercostal pain situations. (2) Psychiatric referral may help differentiate between "functional" and "real" neuropathy, even when the psychiatrist cannot.

Major Intercostal Denervation

Permanent interruption of several adjacent intercostal nerves leaves a belt of altered or absent sensation on the chest wall. Such major denervation is often troublesome and would only seem indicated when chest wall resection makes it unavoidable. Lower thoracic intercostal nerve loss may lead to chronic complaints of upper abdominal "swelling," sometimes with a visible unilateral bulge of upper abdominal muscles when standing or straining. Such swelling need not be obvious to trouble the patient, however, much as a normal-appearing face can feel distressingly swollen after dental anesthesia.

Destruction of a single intercostal nerve is of transient significance, however, as overlapping adjacent innervations usually provide "normal" sensation within six to eight weeks. Persistent hypesthesia of overlying skin commonly follows internal mammary artery mobilization from behind the sternum during coronary artery bypass.

Identification of Ribs

All soft tissues, especially those in obese persons, move about in relation to the rib cage. An elderly female's nipple is commonly most unreliable as an external landmark of underlying structures for, depending upon habitus and position, it may be found near clavicle, axilla or umbilicus. This common observation has not prevented frequent misguided attempts at precise thoracentesis (in a sitting patient, for example, after an appropriate skin location has carefully been marked by the radiologist with the patient supine, on her side or prone).

For even reasonably accurate thoracentesis, one should needle and aspirate with the patient again positioned as when marked. Better yet, if radiologic assistance seems essential, combine fluoroscopy or computed tomography with thoracentesis. Commonly, however, counting ribs from known anatomic landmarks will allow safe and effective chest wall puncture, especially if based upon a recent chest roentgenogram: Preliminary percussion can confirm the diaphragm level when this seems appropriate.

Rib Location Through Intact Skin

Ribs are most readily identified externally at three points: (1) Second rib cartilages meet the sternum at a palpable bulge where upper and mid-sternum join, (2) The lower tip of scapula normally

overlies the seventh rib, and (3) The rib-free soft-tissue triangle between twelfth rib and vertebral column is readily palpated in a comfortably prone patient, after back muscles relax.

Thoracotomy

Routine rib cage incisions either split the sternum or go parallel to the ribs, since adequate exposure and secure closure are difficult to achieve when numerous divided rib ends must be retracted or reapproximated. Thoracotomy can be performed with the patient supine, prone, partway between supine and lateral, in either lateral decubitus position, or even sitting (which adds difficulty in exposure).

In the supine position, the incision can be a full, vertical midline sternotomy or a partial upper sternotomy with one or possibly two lateral extensions (one above the clavicle and the other below it in the second, third or fourth interspace, for a limited "trap door" type of exposure): Common anterior thoracotomy incisions include the bilateral submammary transsternal, the thoracoabdominal, or an anterior or anterolateral submammary approach on either side, with an optional transsternal extension. One can also "develop" a bilateral sub-mammary skin incision sufficiently with dissection under the subcutaneous tissues to allow an underlying vertical sternotomy, but those listed above are the principal openings, with sternotomy and anterior thoracotomy most popular.

Posterolateral Exposure of the Rib Cage at Thoracotomy

Posterolateral thoracotomy is the standard against which other thoracotomy incisions should be compared. Familiarity with the lateral position for posterolateral incision will allow expeditious thoracotomy with adequate or excellent access to lung, esophagus, patent ductus arteriosus, descending aorta, lateral pericardium and heart. Anterior tissues such as thymus that cross the midline may often be removed by thoracotomy from either side or even via a cervical incision, but a sternal split provides the best access for meticulous work in the anterior mediastinum. A posterolateral incision that offers ex-cellent exposure in one patient may turn out to be inconveniently restrictive in another, due to variations in patient height or width, thickness and rigidity of the chest wall, condition of lung and adjacent normal or abnormal tissues, and the conditions under which anesthesia is delivered.

Position: The standard position for posterolateral thoracotomy is with the patient lying upon his side (after anesthesia has been induced and the trachea intubated). Obviously, this is an unstable position in which nerves and vessels to the dependent arm are readily compressed. The chest is therefore lifted from the operating table and braced fore and aft by an ordinary pillow-case under tension between two round sandbags or rolled towels: For an average-sized adult chest, these supporting cylinders should be 6 to 8 cm. in diameter and 25 to 30 cm. in length.

Initially, one sandbag is placed within the closed end of the pillow-case. The open (empty) end is then passed under the chest from behind the patient, pulling the posterior sandbag firmly against the back while spreading the pillow-case widely to eliminate wrinkles. Number two sandbag is now placed barely within, and parallel to, the open end of the pillow-case.

The second sandbag is then "turned under" (rotating against the table top as it rolls along the patient's chest). By this "winding up" of the pillow-case, the sandbags are drawn ever more tightly against, and even partially under, the rib cage, so that the patient's chest is easily elevated on a smooth, ever-tightening, pillow-case sling between the two sandbags (until a hand can slip freely under the

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axilla, which confirms absence of pressure on the dependent arm). I like to place the patient's back close to the edge of the table, as this reduces stress on the surgeon's back, knees and achilles tendons. With the flat surface of the patient's back more or less vertical, the dependent arm is directed forward at a right angle to the chest, while the dependent forearm is flexed up the table toward the anesthesiologist (usually inside the "ether screen").

Resting the lower arm on the table in this fashion (with wrist carefully protected from the screen) eliminates the "arm board," which so often interferes with the assistant and the drapes. The patient is finally adjusted into a true lateral position, with the lower breast pulled forward to avoid undue pressure on it (as might occur if it became folded and jammed between the anterior sandbag and chest).

When there is plenty of help, one person will stabilize the patient's hips until sandbags are in place. Usually, however, it seems best to secure the patient's pelvis to the table with two long strips of 3" or 4" cloth adhesive tape (from metal part of table under mattress on one side, over buttock, to table on other side) as soon as he has been turned to the lateral position. After this pillow-case and sandbag stabilization of the chest, with the pelvis securely taped to the table, the legs are separated to avoid pressure points by placing one pillow (lengthwise across table) between thighs, and another pillow similarly between the lower legs: Check that testicles are not compressed by pillow or thigh. Such transversely placed pillows also stabilize and widen the foot end of the draped patient, which helps the nurse retain loose instruments.

Were it not for the scapula, breast and spine, a standard curved posterolateral thoracotomy incision could simply parallel the interspace to be entered. Obviously, however, the broad, flat scapula will often be "in the way": It is well to skirt the lower scapula tip of an adult with a two cm. margin so that scapula movement does not become restricted by scar tissue.

Similarly, the anterior end of a posterolateral thoracotomy incision should aim at the inframammary fold (although usually not carried forward of the midaxillary line, where momentary sharp mobilization up and down the posterior edge of the serratus anterior often eliminates the need to transect that muscle in order to obtain good exposure).

Finally, the posterior portion of a posterolateral incision in an adult should usually terminate vertically, midway between the vertebral spines and the medial scapula margin, to allow cosmetic extension of the incision if higher access proves necessary. A few dents in the skin (by knife handle or hemostat tip) help to lay out the skin incision.

The smoothest curve results if a left thoracotomy skin incision is made left-handed, starting posteriorly, while the scalpel is best held right-handed for a right posterolateral thoracotomy. (In both cases, the thumb and fingers of the other hand will be on vertebral spines and scapula edge to identify these important points as the incision begins.)

Selection of an Appropriate Posterolateral Interspace for Chest Entry

Interspaces one and two are never opened posterolaterally (too short and too high to provide reasonable access to anything, and would require excessive division of the overlying trapezius and rhomboid muscles). Interspace one can be traversed by an aorto-axillary bypass graft (placed via a left fourth interspace, posterolateral thoracotomy, or else through a sternal split to gain access to the ascending aorta). Interspace three is occasionally used in infancy, while interspace four is commonly

used in infancy and adult for correction of patent ductus arteriosus or tracheoesophageal fistula, and to gain access to high lesions in the mediastinum. Entry through the bed of the resected fourth rib is preferable to interspace incision at the time of coarctation repair (to decrease the likelihood of excessive bleeding from intercostal arteries).

Interspace five, or bed of resected fifth rib, is used for upper and middle lobe (anterior lung) surgery, pneumonectomy and lateral access to the mediastinum (as for thymus) or a midesophageal lesion (usually best approached from the right, or "non-aorta," side).

Interspace six, or bed of resected sixth rib, is used for lower lobe (posterior lung) surgery, pneumonectomy and esophagectomy (by left or right thoracotomy).

Interspace seven can be used for thoracoabdominal access, hiatus hernia repair or traumatic rupture of diaphragm, and distal esophagectomy.

Interspace eight is usually a bit low for hiatus hernia repair (because of frequent necessity for retraction of bulging diaphragm) but eight is adequate for thoracoabdominal surgery, for then diaphragm is detached peripherally anyhow.

Interspaces nine, ten, and eleven are used only during direct access to underlying tissues or fluid collections.

The space below rib twelve is traversed by needle in a high trans-lumbar aortogram (where the intent is to also show renal vessels).

Assume for a moment that upper lobectomy is proposed, but that your incision has been made in the sixth interspace by error. As you open the chest retractor ever more widely, access remains quite limited superiorly, becoming even worse if the seventh rib breaks during retraction. Now what?

Knowing that poor exposure increases surgical risk, and that posterolateral flail is rarely if ever a problem, it is usually best to transect rib six posteriorly, under the edge of the sacrospinalis muscle (our rather dull rib cutters commonly crush intercostal muscle without causing intercostal vessel bleeding, so one need not dissect the rib out before cutting it): Thus divided, the sixth rib will retract far more readily. Of course, one should check the crushed intercostal muscle for bleeding at the time of chest closure. With restricted exposure or a bulky lesion, one may prefer to improve access by cutting two ribs posteriorly, above or below the intercostal or rib bed incision. Routine closure of intercostal muscle (with an occasional, heavy, chromic "figure-of-eight" suture placed next to or around the rib discontinuity) has given excellent postoperative results. Rarely, two separate intercostal incisions (high and low, through a single standard chest wall incision) can improve access to intrathoracic contents.

Ribs are Identified for Accurate Rib Cage Entry.

During posterolateral thoracotomy, ribs are more easily identified once direct palpation of the rib cage is possible. A hand is then passed readily up through filmy adhesions in the subscapular space, to feel the prominent muscle attachments that partially obliterate the superior border of the second rib. (The first rib usually is not felt without indenting or penetrating a resistant musculofascial layer above the second rib: Such confirmatory palpation of the first rib is often unnecessary, as the second rib is readily identifiable in most cases.) Also, in the lateral position, if the shoulder is pressed firmly caudad,

its scapula tip usually overlies the seventh rib.

Rib Identification is Confirmed from Within:

Ribs are even more confidently identified once the chest cage has been entered, by feeling for the soft hollow above the first rib (where the subclavian artery pulse is readily located) and counting down from there.

Resection of an Anterior Rib Can Lead to Flail

The uppermost anterior ribs and costal cartilages are wide and flat in the adult. Although posterior or lateral intercostal muscle closure after rib resection is common, simple and secure, one often cannot achieve firm approximation of intercostal muscles after anterior rib resection, for this leaves too large a gap: These larger anterior gaps are best closed by including good-sized "bites" of the overlying pectoral muscle in the intercostal suture. Bone and cartilage regeneration will maintain chest wall rigidity as postoperative tissue edema subsides, provided most of the perichondrium and periosteum have not been resected along with the rib. On the other hand, persistent noticeable but usually asymptomatic flail often follows resection of even a single anterior rib segment along with its periosteum.

Rib Resection

Subperiosteal removal of a rib segment can markedly improve surgical exposure through a short or "mini thoracotomy" incision. In addition, such removal of rib allows more secure suture-approximation of intercostal muscles than does an intercostal muscle incision, for even if correctly centered, the latter leaves little muscle on opposing rib surfaces for closure.

Chest entry through the bed of a resected rib is preferable when weakness of an intercostal muscle closure is anticipated (e.g. consequent to parietal pleurectomy or extrapleural dissection, for parietal pleura provides much of the strength of an intercostal muscle closure). Rib resection is also helpful if a pneumonectomy is contemplated, or a major pleural effusion may follow the thoracotomy, for a large volume of fluid in a closed space readily transmits sudden pressure changes (as with cough): Under these circumstances, any intercostal weakness is soon demonstrated by increasing paradoxical movement of the chest wall, or even external fluid drainage.

Although a rib that regenerates from residual periosteum will never appear normal on chest roentgenogram, a perfectly satisfactory bony plate is reconstituted that does not alter external appearance or function: Such regrown rib may slow any "repeat" chest entry at the same level, however, due to the absence of normal periosteal tissue planes, as well as by irregular bony extensions into nearby intercostal muscle.

Intercostal Approach

Intercostal incision of the rib cage is usually practical if the anesthesiologist can provide sufficient relaxation so that ribs are not "all bunched together" by contracted intercostals (unless the interspace is narrowed for another reason, such as fibrosis of the underlying parietal pleura). An intercostal incision is always possible, of course, but the idea is to divide intercostal muscles cleanly, midway between the ribs, so that one can achieve snug muscle-to-muscle reapproximation later by suture. Obviously, if simple pericostal suture closure is intended, any intercostal opening will do. The advantage of

intercostal closure is that it is usually "airtight and waterproof" (which can be important in the adult patient).

Once the intercostal muscle has been divided by scalpel at some convenient location, curved Mayo scissors extend this incision in both directions. If the curve of the scissor tip is angled against the external intercostal muscle "grain," such an incision will tend to remain properly centered in the interspace. Incidentally, at least one recent lawsuit has alleged that overly vigorous electrocoagulation of an intercostal vessel in the costovertebral angle caused paraplegia (perhaps by direct "electrothermal" damage to the spinal cord, or by ongoing bleeding with cord compression).

Closure of Chest Wall Muscles

During thoracotomy closure, chest wall muscles must be closed in layers, with care taken to observe the muscle fiber direction within each layer. In a posterolateral thoracotomy incision, muscle will often retract under its overlying fascia or fat, encouraging an erroneous approximation of superficial latissimus on one side to deep serratus muscle on the other. While such a mismatch is readily achieved, it usually disrupts with renewed patient movements as the anesthetic wears off.

It is therefore best to identify layers clearly before beginning the closure, using firm hand pressure on overlying tissues a little back from the edge of the incision, in order to bulge deeper muscle forward into view. It often helps to start suturing at both edges of each muscle belly. One should include the full muscle thickness (fascia and muscle belly) in each interrupted 2-0 Vicryl suture; otherwise a significant chest wall hematoma, with major edema and prolonged, unsightly overhang of the upper skin flap, is likely to follow. Especially in an older or obese patient, this overhang can be severe and simulate an undesirable posterior axillary breast.

Secure approximation of each muscle belly should be with "interrupted" sutures ("continuous" can be satisfactory for thin muscle, but with bulky muscles, any "give" could lead to general separation of the muscle belly, causing a suboptimal cosmetic and functional result). Troublesome blood loss from chest wall muscle during closure is often best controlled by firm suture-approximation of the bleeding tissues. Minor intercostal arterial bleeders are more likely to recur or persist in arteriosclerotic patients.

The chest wall subcutaneous fascia is well developed, and thus easily approximated, using running 3-0 Vicryl suture. Subcutaneous sutures must often be placed obliquely across the incision to restore skin to its preoperative position. Skin is manually pushed about during closure to permit smooth, accurate reapproximation. One should not hesitate to remove and replace unsatisfactory sutures at any level.

Thoracoabdominal Incisions

Other chest wall incisions beside median sternotomy and posterolateral thoracotomy can be of special or emergency utility. A thoracoabdominal incision, for example, may be required for ruptured liver, or when an abdominal aortic aneurysm extends above the renal arteries or even into the chest. In such case, an initial vertical midline abdominal incision can be carried into the left seventh interspace across the mid-costal margin, preferably by curving laterally from the top of the vertical abdominal incision into the chest.

One can also come off the side of a long, high, midline incision at a right angle, before angling

upward into the chest (parallel to the ribs): One rectus muscle is divided en route to the costal margin. (A transverse upper abdominal incision is continued directly into the chest in similar fashion.) As discussed previously, the diaphragm is then detached peripherally until it allows easy access to both chest and abdominal contents.

The costal margin can be divided with a knife, heavy scissors, or bone shears, depending upon its calcification. Reapproximation of interspace muscles and the costal margin is greatly simplified by excision of 1-2 cm of the costal arch (so that cartilages of ribs seven and eight can meet anteriorly). The costal arch can be sutured with heavy pericostal Vicryl, and wire suture brought through the cartilage; interspace muscle may also be closed with 2-0 suture, as in a posterolateral thoracotomy.

For optimal exposure through the interspace during a thoracoabdominal incision, the ipsilateral shoulder can be supported on a sand bag and its forearm tied to the padded cross-bar.of the ether screen: This allows the chest retractor to open the patient most efficiently. (With the patient in a flat supine position, the upper end of the intercostal incision soon dives deep into the axilla, thereby limiting surgical exposure.)

Submammary Transsternal Entry

Unilateral or bilateral transsternal submammary incisions are usually less conspicuous than a sternal split (and have been successfully invisible on high-ranking candidates for "Miss America"): Even with minimally pendulous breasts, only a short transverse scar is visible centrally. Regardless of the interspace to be entered, the curved skin incision follows each submammary fold: All breast tissue (or potential breast tissue) is then reflected up the chest wall to the appropriate interspace. Interspace incisions need not be at the same level on both sides, as the sternum can easily be transected obliquely.

While this approach provides satisfactory access for most heart operations (better exposure of the mitral valve with some decrease in access to the ascending aorta), it has fallen into disuse because of the greater facility of, and familiarity with, median sternotomy. I have also used a bilateral submammary approach for sternal resection: Exposure was satisfactory and the cosmetic result pleasing (an invisible scar, except for a 2 cm, barely discernible line between the breasts). Excessive retraction of the transversely divided sternum can lead to marked postoperative back discomfort, however, presumably due to severe intraoperative angulation of the vertebral column.

Internal mammary vessels bleed significantly during sternal transection if not divided under control. The interspace to be opened is identified by counting down from the sternal prominence where the second rib comes in (at the angle between upper and mid-sternum). That interspace is first entered a few cm lateral to the sternal border in routine fashion (splitting overlying pectoral muscle as much as possible rather than dividing it). When an adequate intercostal opening has been created, a finger is inserted and the soft, pulsatile, spaghetti-size, internal mammary vessels are identified next to the sternal border.

The remaining intercostal muscle is then divided toward the sternum with this finger still in place. A small, closed hemostat can then be drilled downward (avoiding injury to finger or heart) along the sternal border, and also on the lateral side of the palpable internal mammary vessels. Now two hemostats are clamped, cephalad and caudad, on the small, intervening wad of tissue that includes these vessels (which are then divided, and a hemaclip or tie placed on their open ends with some difficulty). Alternatively, the vessels can be divided and hemostats left in place until the sternum has been

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transected by a Gigli or other bone saw.

Regardless of the method used for securing divided internal mammary vessels, ties, clips and hemostats are all knocked off easily during manipulation of the cut bone ends. If vigorous bleeding intervenes prior to sternal transection, the offending vessel is best compressed against the sternum first by finger, and then carefully grasped with a curved hemostat (its tip directed away from the planned line of sternal division).

All four vessel ends are routinely resecured with 2-0 silk suture ligatures, one or two cm back from the raw bone edge, after chest entry has been achieved. The suture needle usually will not pierce the mammary vessels if it is carried along the sternal periosteal surface medially. A chest retractor is then placed between the sternal halves and cranked open gently (crosspiece on the assistant's side as is usual at thoracotomy, for otherwise this gets in the surgeon's way).

Closure: The transverse sternotomy is firmly reapproximated with two or three vertical, transsternal, heavy wire sutures (after chest tubes have been placed, and the internal mammary vessels and surgical areas rechecked for bleeding). Intercostal or pericostal sutures are placed as in posterolateral thoracotomy (but it is hard to get an airtight closure of these wide anterior interspaces, due to prior meeting of the divided sternal ends). Periosteal or muscle sutures are placed as indicated to support the sternotomy and cover bent-down wire ends.

Closure of the split pectoral muscle is minimal, using running 2-0 or 3-0 chromic. One may wish to include a bit of pectoral muscle in parts of the intercostal muscle closure to make this more secure. I have never heard of a transverse sternotomy "falling apart" postoperatively, which unfortunately is not true for vertical sternotomy, but that may relate to the lower frequency of the former incision, as well as the small amount of muscle traction that can be exerted across a transversely closed sternum.

Emergency Incision for Open Cardiac Massage

The common incision for open cardiac massage is left submammary (fourth intercostal space). This incision in the inframmary fold is carried down between underlying ribs and cartilages at the same or a slightly higher level. Avoid carrying the initial intercostal muscle incision too deeply (onto the heart). Once pleural entry has been established, the incision is bluntly ripped open with the fingers. (Don't worry about leaving muscle for closure.) Before putting a hand inside, it is well to widen the incision as much as possible, by a strong, quick, "opening jerk" on each end (especially if a chest retractor is not readily available).

Internal cardiac massage can then begin, but ribs will tend to spring together (giving the surgeon an ischemic hand) until a chest retractor becomes available. When the chest retractor is placed and rapidly cranked apart, several ribs may fracture; a little extra care during closure will prevent flail, however. (This is the only chest incision where the width of the opening may safely equal its length.)

Sometimes open cardiac massage is most effective with the heart held in the hand; at other times it seems safer to press the heart forward intermittently against the sternum with a flat hand (this is also a useful maneuver during temporary massage from within the abdomen through an intact diaphragm). Exposure of the heart is usually poor under emergency circumstances, and one must often open the pericardium widely (vertically, anterior to the phrenic nerve) in order to get effective cardiac access and compression.

With an acute, bloody, cardiac tamponade at the time of emergency chest entry, one may accidentally cut the distended pericardium and also the left phrenic nerve. And after pericardium has been opened for more effective cardiac massage, there is greater risk of penetrating the heart with a thumb or finger. At such times, one must always be sure that ventilation and other aspects of resuscitation are not neglected.

Anterior Thoracotomy

An anterior third interspace incision can provide access to proximal great vessels and aortic arch on the left. Usually (second to fifth) anterior interspace incisions on one side will allow excellent exposure of directly underlying tissues: I have not utilized anterior thoracotomy for routine heart and lung work as I am more comfortable with the exposure provided by posterolateral thoracotomy or sternotomy.

Congenital Defects of the Chest Wall

The partial or total hemi-atrophy of pectoral muscles or overlying breast tissue that is seen on rare occasion in otherwise healthy individuals has only cosmetic significance. An abnormal rib shape or location (e.g. a cervical rib) may lead to a symptomatic thoracic outlet syndrome requiring surgery. Two of the principal congenital defects of chest wall, pectus carinatum and pectus excavatum, appear related to costal cartilage overgrowth. Pectus excavatum, the more common, can occasionally have major physiologic significance.

Cardiac Displacement with Pectus Excavatum

Significant congenital sternal depression causes an abnormal cardiac shadow on PA chest roentgenogram: The diagnosis becomes apparent when a lateral view demonstrates the deep sternal concavity. Major cardiac displacement and lung compression caused by severe pectus excavatum can reportedly result in decreased exercise tolerance (or very rarely, even emphysematous destruction of an entire lung).

Lesser degrees of pectus excavatum, and all cases of pectus carinatum, are merely cosmetic problems that rarely justify radical surgery. Although patients with severe pectus excavatum can benefit physiologically from skilled repair, results of inadequate surgery can be disfiguring, disabling and occasionally even fatal (as by flail chest). Even after successful repair, the entire pectus excavatum thorax will remain abnormally wide and flat (resembling a wooden plank). Fortunately, the cosmetic defect of either pectus deformity diminishes as breasts and pectoral muscles develop during adolescence.

Repair of Pectus Excavatum

Satisfactory repair can be achieved by a safe and relatively simple procedure (developed in Iowa City by Doctors Ehrenhaft and M. Lawrence) based upon subperichondrial shortening of abnormal cartilage (with split, shortened cartilages individually resecured side-to side by fine-wire sutures). Sternal revision by osteotomies is also required. Some wide sternal osteotomies are sutured shut with wire, others are wedged firmly apart with excess cartilage (that is carefully positioned by wire suture). The initial goal of surgery is a slight over-correction of all sternal misalignments, and the lower sternum must be freed from diaphragm, rectus muscles and abdominal fascia.

Postoperative care is as usual, except for avoidance of contact sports and emphasis on a "shoulders-back" posture (keeping hands in back trouser pockets as much as possible). Minor residual or recurrent sternal depression is not uncommon with this technique; another good reason for ignoring clinically insignificant deformities, and for taking preoperative measurements with photographs. A recent article from Iowa City suggests that the currently preferred procedure for correction of pectus excavatum is sternal turnover, based upon an internal mammary artery pedicle (I cannot comment on that more drastic procedure).

Details of Rib Resection Technique

Periosteum can be "stripped" ("reflected" or mobilized readily away) from rib without causing blood loss if the flat periosteal elevator is not very sharp-edged (which would allow it to cut repeatedly into rib cortex or out through periosteum into the intercostal muscles). Top and bottom rib edges are stripped of their periosteum in opposite directions, so that oblique intercostal muscle attachments to rib periosteum direct the ripping force back against rib, rather than guiding the elevator out into the muscle. (One need not try to remember which way to strip which edge, for external intercostal muscle fiber orientation is as obvious at thoracotomy as it is at a barbecue.)

Unsupported (rib-free) pleura and intercostal muscles will normally fall inward from the stripped rib or costal cartilage as soon as the periosteum or perichondrium has been dissected free (much as loss of support by a radial umbrella strut leads to sagging inward of the umbrella cloth). That reflected periosteum and perichondrium soon recreate sturdy bone and cartilage at their new location. To avoid an undesirable recurrence of bone in the thoracic outlet, the first rib is usually removed with its periosteum intact (especially during correction of thoracic outlet syndrome).

Except for the first rib (and the lowest ribs, which are also embedded in muscle), it is not possible to resect a rib along with its periosteum and still avoid pleural entry, unless the parietal pleura has become markedly thickened. An unintended hole into the pleural cavity during periosteal reflection is usually remedied most readily by a small catheter inserted through the hole and connected to water seal or low suction; this chest tube is then removed when lung has expanded and it is clear that there is no air leak.

With no lung air leak, one may prefer to simply close the outer chest wall without a chest tube, allowing residual pleural space air to escape through the accidental pleural defect into overlying chest wall tissues, from whence it will soon be absorbed. (Obviously, the latter treatment is not adequate for unintended pleural entry in the presence of pleural space infection; see below and Chapter 4.)

After any pleural entry, an early postoperative chest roentgenogram is advisable to check for significant pneumothorax. With a patient in the lateral position for posterolateral thoracotomy, good muscle relaxation during closure may markedly increase the volume of entrapped intrapleural air, for when viscera bulges the relaxed abdominal wall outward, it also sucks the upper hemidiaphragm down: This situation can create a surprisingly large pleural air space about the lung (see Chapters 5 and 9).

Surgery for Chest Wall Tumor

If a solitary, possibly malignant lesion does not seem indurated, inflamed or fluctuant, and if the patient has no evidence of disseminated malignancy (such as leukemia, plasma cell or lymphoid cell sarcoma, or lung, liver, bone or other metastases), and if the lesion is fixed to rib, I perform wide en-

bloc excision, staying well clear of the tumor edge by palpation from both within and outside of the rib cage. Palpation from within is accomplished by entering the chest at some distance from the lesion (through a short gap resected in a rib intersecting the lesion, or via a finger-sized, interspace incision next to such rib).

It is important to bring healthy skin and subcutaneous tissue over the Dacron mesh graft that replaces missing ribs. The mesh periphery must be sutured firmly to surrounding periosteum and intercostal muscles ("drum tight"), using multiple, interrupted, 2-0 Dacron sutures. If lung, pericardium, diaphragm, stomach, liver or spleen are adherent to the tumor, part of any such adherent structure can be included in the mass to be excised (provided that there is no evidence of incurability or inoperability). In cases with possible tumor extension along the rib marrow cavity, resection of the entire involved rib or ribs may be appropriate. Judgment and luck become increasingly essential as the extent of potential excision increases.

Incisional biopsy may be useful with larger soft tissue tumors or those impinging upon important structures; too often, however, biopsy merely delays surgery. There is allegedly also risk of tumor implantation, or at least an increased likelihood of local tumor persistence, following incisional biopsy or incomplete resection (of chondrosarcoma, for example), and weeks of consultation may give rise to a "final" - but sometimes not very diagnostic - pathology report.

Case Report: Maturation of Malignancy in Newborn

Unable to resect (without forequarter amputation and removal of one hemithorax), I merely biopsied the huge, undifferentiated sarcoma of the chest wall and shoulder in a newborn: Within weeks of biopsy and patient referral to a cancer center, this lesion spontaneously "matured" and shriveled into an easily shelled out, benign fibroma.

Comments on Chest Wall Malignancies:

Common sense and surgical experience support early complete removal of any rapidly growing, localized, apparently resectable malignancy, regardless of cell type. Similarly, any "obviously benign" lesion may be treated by limited resection, if surgery is even indicated. "Apparently malignant" chest wall tumors usually are malignant, so I perform wide excision whenever practical. This approach has achieved a number of apparent cures with muscle sarcoma and chondrosarcoma of the chest wall. It resulted in little additional distress for two patients with painful, apparently localized, reticulum cell sarcoma (little morbidity, early recurrence elsewhere).

Such a surgical attack was probably too radical, however, for a large, very rapidly growing, chest wall and diaphragm lesion in a young child ("Benign inflammatory," "No organism," "Unknown etiology"). The latter patient (also referred to in Chapter 1) had previously undergone Bochdalek hernia repair; his new lesion may well have been some sort of atypical delayed suture reaction, although that too was considered unlikely after tissue examination. Interestingly, also about 15 years ago, I removed several large lesions of rib (two consecutive anterior first ribs on one woman) that were initially considered "possibly malignant" and later called "Benign, probably inflammatory": Also one enormous, retroperitoneal, invasive process was encountered at that time which I only biopsied before it vanished (almost taking my credibility with it).

In one case, major chest wall resection did provide "permanent" local control and good

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symptomatic relief from a dinner-plate-sized chest wall metastasis, in a patient with metastatic but not preterminal bronchogenic carcinoma. On the other hand, wide resection of a large, extremely painful, solitary sternal metastasis, four years after nephrectomy for renal cell carcinoma, was shortly followed by massive dissemination of malignancy and an early miserable death: It has since been reported that nitrous oxide anesthesia (used in this case) may cause a short-term immunologic depression and thereby increase the likelihood that tumor emboli at the time of surgery will result in metastatic implants.

The advent of computed tomography has greatly improved delineation of tumor margins: Echo studies can be helpful in evaluating soft tissue masses, as can digitally enhanced or other angiography in some locations. Despite technical advances, surgical outcomes remain unpredictable (as do responses to radiotherapy or chemotherapy). As mentioned, the histologic diagnosis may occasionally remain in doubt after extensive consultation ("It could be benign, malignant, inflammatory, proliferative, desmoid or reactive fibrosis" or "Let's wait and see how she does"). Greater diagnostic accuracy has been claimed for electron microscopy of malignant tissues: Whether or how this will influence surgical therapy of chest wall tumors is not yet clear.

Postoperative Care After Chest Wall Resection

Routine water-seal pleural space drainage is used in such cases, with the chest tube located away from the mesh prosthesis. Early chest bottle suction encourages firm approximation of skin flap to mesh: If there is no air leak, one simply aspirates air from the upper chest tubing to raise a hanging fluid column in water seal straw and tubing, thereby applying any desired level of suction (see Chapter 8).

In the presence of pulmonary air leak, application of mechanical (turbine-type) chest bottle suction is desirable, as any slight build-up of air or fluid within the pleural space will cause the skin flap to bulge during each expiration (making it appear that the mesh graft has torn loose), for mesh offers no resistance to movement of air or fluid between the pleural space and the subcutaneous pocket. In such event, improved chest tube drainage draws the bulging skin flap firmly back down onto its mesh backing.

Thoracoplasty

Thoracoplasty means tailoring of the rib cage to diminish hemithorax size. It has been used (prophylactically) to adapt a large hemithorax to a surgically reduced lung, or (therapeutically) to obliterate an underlying, usually infected, pleural space, or (formerly) to compress a subjacent apical tuberculous cavity within the lung: Pulmonary resectional advances (based upon progress in chemotherapy, anesthesiology and postoperative care) had essentially replaced thoracoplastic compression as the primary surgical treatment for cavitary tuberculosis when further advances in chemotherapy also outmoded lung resections for tuberculosis. (At present, only a rare, drug-resistant, localized, atypical mycobacterial lung infection is even considered for resection, with thoracoplasty conceivably still relevant here as a fall-back procedure for the poor-risk patient, or to treat complications of resection).

Apicolysis

The simplest way to reduce the size of a pleural space is to mobilize the parietal pleura down from

the apex so that it can drape over remaining lung like a blanket (the intent being to seal lung air leaks and eliminate the intrapleural air space about the lung). If the extrapleural upper-chest space thereby created (between parietal pleura and rib cage) remains uninfected, it will gradually "scar down." Such a loose pleural blanket seems as likely to plug chest tube drainage holes as lung air leaks, however, and the residual extrapleural space within the chest could also be subject to complications, so apicolysis never "caught on."

Plombage

Plombage requires the local mobilization of rib cage periosteum, intercostal muscles and pleura in one piece. The resulting boneless sheet of soft tissue is then pressed inward from those freshly "skeletonized" ribs. This newly created extrapleural pocket is packed with stiff biologically inert material to compress and reshape underlying diseased lung. Within weeks, displaced periosteum forms new bone. The outcome is a repositioned bony chest wall and overlying dead ribs with foreign material in between.

Plombage remained in common use until the early 1960's, primarily to squeeze an underlying apical tuberculous cavity when the patient was a "poor risk" for lung resection (because of drug resistant tuberculosis, poor lung function or general ill health). Unfortunately, all packing materials then used had a low but progressive rate of erosion into lungs (causing hemoptysis and possibly expectoration of paraffin), or migration out of the desired position, or extrusion out of the skin incision, or infection. Foreign materials that were commonly packed between ribs (sometimes into a polyethylene bag) included warmed paraffin globs and hollow Lucite balls (resembling Ping-Pong balls, but more expensive).

Plombage patients (with wax or balls compressing the soft tissue portion of their rib cage into its new configuration) avoided troublesome postoperative flail and the altered external chest configuration that followed standard thoracoplasty. As in all thoracoplasties, it was considered very important to avoid pleural entry while stripping periosteum from ribs. The major concern during plombage was to mobilize and maintain an adequate pocket of desired shape, deep to the newly bare ribs, without excessive local pressure that could erode underlying tissues.

Recent modifications for treatment of chronic empyema include a strip of live periosteum left on the outer rib surfaces to maintain rib viability, and the use of intrapleural suction to "bring in" the newly "ribless cage," rather than relying upon foreign materials packed under the stripped ribs. Allegedly, with empyema and bronchopleural fistula, this hastens closure of the empyema space, with ongoing fibrosis eventually eliminating the space between ribs and their subjacent intercostal tissues. (This modified procedure seems rather major in comparison to the other methods for treating empyema discussed in Chapter 4, and it creates a temporary extrapleural space that could still cause problems: I have no experience with it.)

Osteoplastic Thoracoplasty

Osteoplastic thoracoplasty (Bjork) represented another effort to eliminate residual pleural space problems while also avoiding post-operative flail movement by tailoring and relocation of upper ribs at the time of lung resection. As a one-stage, upper thoracoplasty (reinforced by upper ribs detached posteriorly and resecured to the sixth rib) it placed a firm new apex just over the lung hilum, providing an immediately-stable reduction of hemithorax size. This procedure allegedly avoided interference with

movement of the scapula and eliminated the necessity for resection of the seventh rib or any transverse vertebral process. (I have not tried it.)

Standard Thoracoplasty

This two- (occasionally three-) stage, commonly seven-rib procedure was also performed through a posterolateral thoracotomy incision down to ribs. The first stage (total removal of first through third or fourth ribs plus associated transverse vertebral processes) was best performed from below upward, to improve access to the shorter upper ribs. Adequate release of the pleural apex usually required division of the first four intercostal bundles posteriorly (with care taken during dissection to avoid injury to the nearby phrenic nerve).

The second stage, performed two or at most three weeks later (in order to avoid undesirable stiffening and new bone formation in tissues mobilized during the first stage), was removal of decreasing (posterolateral) portions of the fifth through seventh ribs, with sufficient seventh rib resected to prevent its remnant from hooking the scapula tip (this tip could also be trimmed, if necessary). During thoracoplasty, damage to the Long Thoracic Nerve of Bell (innervation of serratus anterior) was carefully avoided, to prevent distressing "winging" of the scapula.

The considerable surgical trauma and blood loss of standard thoracoplasty was a major stress for these patients, with their severely infected and compromised lungs. Large compressive dressings sustained the rib cage in its new position, while Penrose drains under the scapula prevented fluid accumulation. Flail chest interfered severely with cough and sputum production, so nasotracheal suction was routinely essential for survival, with bronchoscopy frequently required to overcome lower lobe atelectasis. Often enough, it was a "Do-or-die" proposition, and the latter outcome was all too common.

A first stage thoracoplasty did little to the external appearance, beyond deepening the natural hollow below the clavicle, but the second stage resulted in a far more "squashed" look for that hemithorax. Subsequent to standard thoracoplasty, patients commonly developed thoracic scoliosis, due to loss of lateral support to the spine.

The standard or extended thoracoplasty could eliminate an infected apical. intrapleural space or achieve compression of lung. It could not eradicate an entire pneumonectomy space, especially on the right. The radical Schede thoracoplasty (total removal of overlying rib cage and stiff, thickened parietal pleura to completely unroof an infected pocket - leaving only chest wall muscles and overlying skin), followed by secondary skin grafting, was the only hope for "cure" of a chronically infected pneumonectomy cavity. Many patients with such a cavity therefore required permanent empyema tube drainage; a malodorous alternative that was associated with occasional bouts of superinfection and long-term risk of amyloidosis (see Chapter 4 for a discussion of empyema drainage and the current, antibiotic-era approach to these difficulties).

Obliteration of an Infected Space by Muscle Transfer

According to the literature, bulky chest wall, flank or abdominal muscles can readily be rotated, advanced or transferred on a vascular pedicle into almost any infected pleural or mediastinal pocket to simultaneously eliminate the space problem and the infection. It is suggested that a plastic surgeon familiar with such transplants be on the surgical team. I have no experience with these procedures but

they sound exciting and worthwhile (see also Chapters 4 and 10).

Case Report: Extrusion of Oleothorax

A middle-aged salesman entered with a fluctuant, pointing "abscess" in the mid-portion of his healed, posterolateral thoracotomy incision. The incision was reopened, with evacuation of much cloudy fluid and many wads of wax. Assuming infection, the overlying, long-ago-skeletonized ribs were all resected to allow outer chest wall muscles to fall against the inner, reossified rib cage. Penrose drains were left in the subscapular space until cultures proved negative. The patient was discharged soon thereafter in good health, little changed in his external appearance: Perhaps redundantly, he was also placed on prophylactic antituberculous chemotherapy.

Conclusion: It is technically simple (and easy on the patient) to reopen a plombage incision and complete the thoracoplasty during removal of possibly infected foreign material. The mode and duration of subscapular or chest tube drainage will be determined by any associated sepsis or pleural defect.

In How Many Ways Can a Restricting Thorax be Enlarged?

When a chest cavity is too small for its content, one can either try to remove non-vital content or increase the thoracic volume or both. Much of chest surgery is devoted to removal of space-occupying air, fluid or tissue, in or around the pleural cavities or mediastinum. We also attempt to "correct" certain irregular thoracic shapes, such as pectus excavatum, in a fashion that will enhance thoracic volume within its fixed surface (toward the spherical, in other words).

An abnormally restrictive chest wall decreases the capacity of the air pump and its enclosed blood pump. Since lungs occupy more space, and are usually more pliable, air movement will normally be impacted by such thoracic restriction before blood flow. When enlarging lungs (asthma, emphysema) are blocked from further expansion by a rigid chest wall and flattened diaphragm, one can still gain additional liters of pleural cavity volume by intermittently pushing both hemidiaphragms downward from above through positive-pressure ventilation of the lungs, or by pulling them downward from below, with an "iron lung or abdominal ventilator; the latter devices rhythmically reduce air pressure within a rigid, air-tight shell that either encloses the entire torso or merely the abdomen.

How about an ambulatory abdominal respirator? That should not be impossible to design and construct. It might be too noisy if he sat right behind you at the movies, but it could become as stylish as hoop skirts: For power failure one could add a hand-operated piston. The main question would seem to be whether one could apply sufficient inspiratory pressure transabdominally to a stiff emphysematous lung, considering the added rigidity of the abdominal wall in an upright patient (might the creation of a large ventral hernia then improve abdominal wall compliance?).

Fortunately, preliminary reports suggest that home use of an external (chest and abdomen) subatmospheric pressure respirator can markedly assist breathing of the severely emphysematious patient, by at least allowing him to sleep or rest while this large external vacuum device does the work of breathing: Mobility while off this device is greatly improved, due to the intermittent relief of severe diaphragm fatigue.

Other possibilities (and impossibilities): There is currently no practical way of inverting (bulging)

the diaphragm downward without such "pressure-assist" from above or below. Obviously, any patient with lungs so large that his diaphragms remain flat "at rest," cannot move any air by diaphragm contraction (unless lower ribs are thereby pulled together slightly; a maximum "inspiratory" diaphragm contraction might then force a small amount of air out of the chest).

Might the diaphragm again become an effective breathing organ for a patient with severe emphysema if its bulge could be reversed (downward into the abdominal cavity)? Actually, there is no major anatomical reason why a diaphragm could not bulge downwards and contract back upward. Let's say we maintain an emphysematous patient on the respirator until his lung has bulged "way down": Diaphragm contraction would then become expiration: However, elastic recoil of a compressed, "much enlarged" lung would then be required to push the diaphragm back down into the abdomen on expiration (thereby allowing passive return of air into the lung).

To achieve significant "expansile" recoil of lung might require some biocompatible peripulmonary prosthesis that could "rebound" following its compression by the now-inverted diaphragm. Although increased intrathoracic pressures during breathing would adversely affect cardiac performance, that problem might be overcome by increasing blood volume (which could lead to other problems - see Chapters 14 and 15): All in all, an unlikely prospect.

Well then, how about relocating the diaphragm downward? After all, we have, on occasion, peripherally detached and moved part of the diaphragm upward, in order to diminish thoracic volume or improve our access to a subphrenic abscess. Why not simply free the entire diaphragm margin and reinsert it at the midabdominal level?

Well, for several reasons; (1) little would be gained by such major surgery even if it did work, for the pathologically enlarged lung soon becomes even larger and (2) inspiratory contraction of such a relocated diaphragm would pull the unsupported (by ribs) abdominal wall inward (at a time when it should be bulging out to make room for viscera being temporarily displaced from the "chest" during inspiration) and (3) a relocated diaphragm inevitably loses length so it would be increasingly flattened, and thus at a greater mechanical disadvantage if the diameter of its attachment was unchanged: Of course, one could lengthen the diaphragm with plastic cloth peripherally, perhaps even sufficiently to allow it to be attached to the pelvic brim... Or possibly a rigid upper-abdominal polypropylene hoop of controlled diameter for diaphragm reattachment could improve these dynamics?

How about enlarging the rib cage toward spherical? Unfortunately, there are problems here too (beside those applicable from the above discussion of other options, such as the progression of emphysematous lung enlargement). In the first place, a lateral roentgenographic view of the chest of an emphysematous person already shows a surprisingly circular lung outline, with ribs bulging both posteriorly and anteriorly.

Maybe it is not practical to bulge the ribs laterally a great deal more (apparently this does not occur naturally) as it would restrict arm movement. Could we instead just split the sternum and increase its width, or cut and lengthen the ribs posteriorly? Well, it has been alleged that superior vena caval syndrome can be partially relieved by a few short (3-4 cm) rib segments which prop the sternum apart, yet allow healing when the sternum is securely rewired in this position. This certainly sounds like an interesting laboratory project if such work has not yet been done (perhaps combined with some diaphragm-lengthening procedure).

What about simply cutting off the most bleb-filled part of each lung, to eliminate the excessive lung volume? Unfortunately, generalized bullous disease is usually progressive, and so far there has been insufficient clinical benefit to justify proposing such high-risk surgery to a patient with terrible lungs.

Conclusion: It would be nice if one could enlarge the chest or safely shrink emphysematous lung (see also the discussion in Chapter 13): For the present, generalized pulmonary emphysema by itself is not an indication for surgery, but that abdominal ventilator still sounds interesting.

Chest Wall Infections

Postoperative infections of chest wall incisions are uncommon. Many thoracotomies have been performed with dirty instruments such as pocket knives; and in one emergency I inserted a chest retractor picked off the x-ray department floor. Yet even then, or when operating in the face of established empyema, wound infection is very unusual.

While intact ribs or undivided sternum certainly can develop osteomyelitis, that too is exceptionally rare; such infections would, I assume, best be treated by appropriate antibiotics plus drainage of any abscess (with debridement or subperiosteal resection of any obviously dead or persistently infected bone). Post-sternotomy wound infection can be a major problem, however (see Chapter 10).

Costal cartilage rarely becomes infected, but if such infection develops and becomes firmly established, complete removal of all communicating (and therefore involved) cartilage is generally indicated. This is not always the major task it would appear since, given sufficient time, living perichondrium spontaneously separates from chronically infected (dead) cartilage. Fistulas to the skin surface that develop and heal over many months may in the meanwhile demarcate the area affected.

Once this infected cartilage is fully exposed by incision, the dead cartilage (even a total chondral arch, if that is extensively involved) can sometimes simply be lifted out and easily "snapped off" from its costochondral junction(s) and sternum, and handed to the scrub nurse. This sounds crude, but it is simple and rapid if it works, allowing one to avoid a tedious, bloody dissection through inflamed, fibrotic tissue planes. Thereafter, the loosely approximated, granulating, perichondrial tissues should heal rapidly. Any deep pockets are drained by Penrose drain, or irrigating or sump catheter, and antibiotics are continued as indicated by wound appearance and bacterial cultures.

By the time such a wide resection is embarked upon, after many months of medical treatment and local attempts at surgical drainage, surrounding tissues should be sufficiently indurated and scarred that flail chest need not be a concern: One assumes that early cartilage regrowth from residual perichondrium soon reinforces the chest wall in any case.

Chest Wall Injury and "Sucking" Chest Wounds

Significant chest wall injury may be penetrating or blunt. Penetrating injuries occasionally permit atmospheric access to the pleural cavity. It is theoretically possible that an isolated chest wall defect could cause tension pneumothorax by allowing air into the pleural cavity on inspiration, then sealing shut on expiration. As a practical matter, however, such isolated "sucking chest wounds" do not occur. Penetrating injuries almost invariably damage underlying lung as well, and they very very! rarely (if

ever) function as an efficient, inwardly directed, one-way valve.

Air leak from underlying damaged lung is the only likely cause of tension pneumothorax after penetrating thoracic trauma, regardless of how a chest wound sucks, slurps, gurgles, blows, flutters or splatters. Even if an isolated, sucking chest wound should (or could?) occur, the best treatment would still be that already indicated for the usually associated lung injury (see below).

Emergency Decompression of Tension Pneumothorax

Any accumulation of air within the parietal pleura but outside of lung that remains at pressures consistently above atmospheric ("tension pneumothorax") will interfere with filling of both heart and lungs. Fortunately, tension pneumothorax can always be relieved instantly by venting the chest cavity to the atmosphere. A finger, drinking straw, catheter, spoon, hollow shell of pen, and so on, can be used to prop open any conveniently located aperture into the chest cavity consequent to injury.

One can also create a new chest wall hole for decompression of severe tension pneumothorax with a quick skin incision and blunt finger dissection - or more easily, by insertion of a #14 or #16 hollow needle when available - to allow survival until chest tube insertion can be achieved (see Chapter 5). Sterility is unimportant in such an emergency, especially when compared to the risk of delayed relief of tension pneumothorax in a critically compromised patient. I have never encountered empyema secondary to unsterile venting of the chest, perhaps because chest tube drainage is routinely instituted for the original air leak anyhow - and tube drainage also happens to be proper treatment for any bacterial contamination of the pleura.

The Open Chest Wound and the "Airtight" Dressing

In years past, first aid courses always emphasized the need for an immediately applied, airtight dressing over any "open" chest wound (one that exposed lung to the atmosphere). Fortunately, it is almost impossible to apply a truly airtight dressing to a hairy, sweaty, bloody, open chest wound. Thus, the occlusive dressing that actually resulted under these circumstances, permitted relatively free air egress from the chest opening, and possibly even encouraged partial reexpansion of the underlying lung (by fluttering up from the wound on expiration, then falling back to partially block inspiratory air return).

Once again, a "right result" (the occlusive dressing here serves as a primitive one-way valve) occurs despite a wrong theory ("This dressing must be airtight!"). A truly airtight cover for an open chest wound would almost surely be harmful; causing tension pneumothorax and subcutaneous emphysema by preventing free egress to the atmosphere of air leaking from injured lung.

Effects of a Small Opening Through the Chest Wall

1) A small pleurocutaneous fistula reduces the function of its underlying lung. The same inspiratory pressure drop that draws air into the lungs through mouth and trachea, also brings air into the pleural cavity through any open chest tube or bullet hole. Similarly, as above-atmospheric intrathoracic pressure is generated during expiration, air will flow out of the lung by airway, and out of the pleural space by any open chest tube or bullet hole, until equilibrium (atmospheric pressure both within and around lung) is once again achieved (marking the end of expiration).

2) Air flows in response to chest cavity volume and pressure changes will be apportioned between the trachea and any open chest tube roughly in accordance with the relative cross-sectional area of each channel. A certain part of the total air volume exchanged during each respiration will therefore be moving in and out of the pleural space. Although "free" intrapleural air may eventually be absorbed through pleural surfaces, it is essentially unavailable for immediate gas exchange. So any air moved during breathing that does not pass via airways into alveoli will simply represent "wasted" breathing effort.

3) Sudden occlusion of a hole through the chest wall generally results in a simple pneumothorax. During ready atmospheric access to the pleural space, normal lung tends to assume a smaller "relaxed" size. Such a relaxed lung exchanges less air and does it less efficiently (since movement of stagnant tracheobronchial "dead space" air then accounts for a larger percentage of air that passes through the airways). If we now place a theoretical cork into all open chest tubes or bullet holes (after the patient has been at rest for a few moments, so that chest wall, diaphragm and lung are all in their "relaxed" state), our patient will be left with a fixed-volume intrapleural air space surrounding the lung (there being no air leak from lung in this example).

4) Although simple pneumothorax decreases respiratory efficiency, it still permits comfortable respiration at rest. Despite free air surrounding the lung, any volume change that occurs in the affected hemithorax between end-inspiration and end-expiration, again represents pulmonary air exchange. A "relaxed" lung within an intact chest thus breathes normally, but forceful expiration is naturally avoided as compression of heart and lung would cause increasing distress: The space occupied by free intrapleural air also prevents maximum expansion of the underlying lung during inspiration.

5) An open chest tube also allows comfortable respiration. Clearly, a smaller "relaxed" lung cannot as easily meet increased demands for air exchange, nor is cough as strong (due to diminished air flow); reduced air flow also means that airways are more easily obstructed by mucus. However, even when the airway of a relaxed-size lung must share the total air intake of that hemithorax with an open chest tube, this does not cause a normal person at rest to become noticeably short of breath.

Tension pneumothorax can kill but an open chest tube prevents tension pneumothorax. That conclusion is easily reconfirmed every time a chest tube is inserted for tension pneumothorax: Simply leave the tube open to the atmosphere while chatting with the nurse and patient. Despite competitive room air movement in and out of the chest tube (and a persistent pleural air pocket around the more-or-less relaxed lung), such a patient will usually be quite comfortable (and no longer breathless).

To complete our theoretical discussion of holes through the chest wall, we now uncork all chest tubes and bullet holes while completely obstructing the main bronchus to the "relaxed" lung: Normal volume changes with breathing still lead to air movement in and out of the affected hemithorax, but air exchange now is solely in and out of the pleural space, so no benefit is derived from the work entailed in moving that air.

Of course, at hyperbaric pressures (perhaps eight atmospheres or more), pure oxygen could probably be absorbed through pleural and other surfaces in sufficient quantity to maintain life and circulation, even in the absence of any pulmonary air exchange, for the higher the ambient oxygen pressure, the fewer square meters of exposed capillary circulation are required to achieve adequate gas exchange (the usual area of perfused surface folded within the lungs allegedly approximates that of a tennis court). In fact, many amphibians normally move a significant portion of their required gas

exchange via the skin. Of course, they have a slower metabolic rate and special skin vessels that can dilate (blush) when surroundings are conducive to oxygen uptake.

Conclusion: An open chest tube that is significantly smaller in diameter than the trachea may cause inefficient breathing, but it is usually well tolerated for some time: Compare that benign open-chest-tube "simple" pneumothorax to critical "tension" pneumothorax (where the intrapleural air pressure build-up must be vented by a small hole or tube through the chest wall or the patient may die within minutes) and it becomes obvious that tension pneumothorax is the far greater hazard.

The Wide-open Chest

Consider now a patient with one hemithorax widely opened, as at thoracotomy. Spontaneous breathing is very difficult in this situation for regardless of respiratory effort, intrapleural pressure on the wide-open side remains atmospheric. Thus air cannot enter or leave that exposed lung without intermittent positive-pressure breathing assistance.

The other lung too is compromised by mediastinal movement toward it on inspiration, then away on expiration - which may markedly reduce its spontaneous air exchange. Yet even with an open hemithorax, a patient can still survive on unassisted spontaneous breathing supported by supplementary endotracheal oxygen – at least under special circumstances for moderate periods of time.

During the tuberculosis surgery era, thousands of thoracotomies were performed with spontaneous unassisted respiration and an acceptable mortality rate, using the Overholt prone position (which reduced risk of spilling infectious material into the other lung). However, these expeditious operations were mostly performed on a special Overholt prone operating table that provided no abdominal support and thereby permitted maximum diaphragm excursion. Furthermore, "light" anesthesia was essential to sustain strong inspiratory efforts and ensure abdominal muscle tone for shoving viscera and the active hemidiaphragm back up into the chest on expiration.

Nowadays, a highly skilled anesthesiologist wishing to provide a motionless lung in the open chest, usually intubates the airway of the other lung and ventilates that lung separately (taking care not to block the upper lobe bronchial opening of that working lung with the endotracheal tube balloon).

When a laparotomy is performed without assisted ventilation, adequate relaxation of abdominal muscles (e.g., by spinal anesthetic) may diminish respiratory efficiency too. For in a supine patient with a "wide-open" relaxed belly, spontaneous breathing depends upon residual pressure exerted by bulky abdominal viscera (plus unpredictable upward surgical retraction) - supplemented by normal subatmospheric intrathoracic pressures as elastic lungs spring back to a smaller, more relaxed size - to bring the relaxing diaphragm upward in preparation for the next inspiration.

Flail Chest

With "flail chest," a loose portion of chest wall moves counter to the remaining intact thorax: This impedes breathing dynamics and efficiency in the same fashion as an open chest tube or the open chest. Flail chest can result when several adjacent ribs are doubly fractured (resulting in a "floating" section of chest wall), or as a consequence of extensive resection of ribs (as in thoracoplasty), or following resection of chest wall tumor (if a mesh prosthesis is not used or not sutured "drum tight"), or after poorly designed operations for pectus excavatum that provide no postoperative support between rib

ends and sternum following subperichondrial resection of all deformed cartilages.

If unilateral flail chest develops as a consequence of anterior rib fractures, one will see normal anterior chest wall rising during inspiration, while the disconnected ribs are simultaneously drawn inward by the subatmospheric intrathoracic pressure being generated during chest expansion. On expiration, as normal ribs settle back, the injured side bulges, confirming that there is now above-atmospheric expiratory pressure within the chest. Air exchange may be markedly reduced by such paradoxical "flail" movement of the injured chest wall.

A bilateral anterior flail chest can result from multiple rib and sternal fractures. In this case, the abdomen protrudes normally on inspiration as the diaphragm contracts, while the fragmented anterior chest wall simply "caves in." On expiration, the abdomen flattens as the diaphragm relaxes upward, causing the loose part of the chest wall to bulge. Bilateral paradoxical chest wall motion drastically curtails ventilation (the elastic chest wall of a young child often shows similar paradoxical movement during actual or relative obstruction of the upper airway - while sobbing, for example). Flail movements due to fractured ribs generally stabilize within about two weeks, while a mesh prosthesis usually loosens somewhat as time passes. (Chapter 12 reviews therapy for chest wall injuries.)

Avoid Loose Sternotomy Closure

The improperly closed or infected median sternotomy tends to loosen. Any movement between sternal halves encourages peristernal wires to saw deeper into sternum or to break. Snug closure of a median sternotomy, using heavy peristernal, figure-of-eight (pulley) wire sutures, essentially guarantees sternal stability. One should give a hefty lift on both ends of each wire before twisting, to take up slack and lock the distal loop tightly about the sternum.

This "lift" establishes firm sternal approximation and permits reduced traction during twisting of the wire ends (the time when wire fracture is most likely). Tight approximation of pectoral muscles and fascia over turned-down wire ends along the full length of the sternotomy, using interrupted 2-0 Vicryl sutures, additionally ensures sternal stability and early healing (see also Chapter 10).

Sternal Fracture

The increasing incidence of sternal fracture has been attributed primarily to automobile accidents. Open reduction and fixation by wire sutures has been recommended for patients with significant sternal override (fracture displacement) or sternal instability, in order to avoid the need for respirator care and decrease morbidity. Pulmonary contusion and injury to underlying cardiovascular structures are common accompaniments of sternal fracture (see Chapter 12).

Case Report: Fractured Sternum

An inebriated snowmachine driver entered the emergency room at midnight with a palpably fractured sternum. He stated that he had often watched snowmachines soar over obstacles on slow-motion television advertisements. Thus, when an open stream blocked his path, he simply drew back a hundred yards, then "gave it the gun," expecting to soar over. His friends fished him out when he failed to clear the opposite bank .

Chest roentgenogram revealed a sternal fracture (with subjacent pleural bulge on lateral view) as

well as compression fracture of a mid-thoracic vertebra (a common accompaniment to sternal fracture). He initially refused admission, but returned at 2 a.m. and was admitted with normal "vital signs".

At 6 a.m., the floor nurse called to tell me he was suddenly dying. I ordered another chest roentgenogram and rushed to the hospital. His left chest was now full of fresh blood from delayed rupture of a torn internal mammary artery. He did well after thoracotomy and multiple transfusions.

Conclusions: 1) Chest wall injury can lead to delayed bleeding. 2) A bulging extrapleural hematoma, deep to a sternal fracture site, may indicate internal mammary artery injury. 3) If snow machines were meant to fly, they would have wings.

CHAPTER 3: THE PLEURA

Normal anatomy; ... Function; ... Pleural effusion; ... Evaluation of pleural effusion; ... Is there any hazard in complete removal of a pleural effusion?... It appears that rapid removal of massive ascites can be dangerous;... Case report: The tub overflowed;... Case report: If a little feels good, a lot can kill you;... Peritoneovenous shunting prevents hypovolemia;... Pleural effusion with pancreatitis;... Venous cannulation may cause pneumothorax or air embolism;... Pleural effusion may represent misdirected intravenous fluids;... Entrapped, shrunken or atelectatic lung resists reexpansion;... Pulmonary edema has been reported following reexpansion of lung;... Serous effusion;... Bloody effusion;... Feel for scalene nodes;... Pleural malignancy;... Malignant pleural effusion;... Tetracycline treatment may prevent recurrence of a malignant pleural effusion;... Pleural plaques;... A pleasant surprise: Hemothorax not due to malignancy will usually absorb;... When is it imperative to evacuate a hemothorax?... Breathing exercises really do remove pleural effusion;... Following sternotomy, wide drainage of pericardium into both pleural cavities will also simplify pericardial closure;... Therapeutic pneumothorax;... Principles of absorption of free air;... Warm saline for resuscitation of a hypothermic heart;... Reviving the dead;... Surgical hypothermia;... Hypothermia with inflow occlusion;... Surface cooling;... Case report: Cooling by immersion;... Postoperative shivering;... Extrapleural thoracotomy;... Patent ductus arteriosus closure in the newborn;... Details of procedure for neonatal ductus ligations

Normal Anatomy

The pleural cavity within each hemithorax is filled by lung. Fragile transparent visceral pleura overlies each lung, sliding effortlessly across the thin, slippery parietal pleural surfaces of mediastinum, diaphragm and inner chest wall. A pleural space develops when air, fluid or other material separates visceral from parietal pleura. Clinical consequences of any pleural space are determined by its size, content and the status of underlying lung.

Function

Normal respiration results from regular changes in thoracic volume: These volume changes take place predominantly in the lower chest. The slippery pleura permits more uniform expansion of lung during asymmetric enlargement of its thoracic container.

Pleural Effusion

If production of pleural lubricating fluid significantly exceeds the amount absorbed, a pleural effusion develops; this may be in response to a nearby disturbance or the result of more generalized health problems. An effusion may be barely detectable on roentgenogram (several hundred cc. or less) or more impressive (over 4 liters may fit within an adult hemithorax). Depending upon cause, pleural fluid accumulations may be serous (hydrothorax), purulent (empyema), bloody, or entirely blood (hemothorax). A pleural effusion may also represent an intrathoracic collection of fluid originating at a distance: For example, a lymph fistula can create a chylothorax; abnormally present intra- or retroperitoneal exudates, transudates or secretions may move up through or past the diaphragm; and solutions delivered by infusion catheters may leak directly or indirectly into the mediastinum or pleural cavity.

Evaluation of Pleural Effusion

Diagnostic thoracentesis is usually indicated when a radiographically demonstrable effusion is

discovered (unless the diagnosis is already clear or not likely to be significant). "Acid-fast" (tuberculosis) and fungal cultures are generally requested on the initially retrieved pleural fluid, although the acid-fast culture has rarely (and fungal never) helped me to "diagnose" an effusion. Effusion protein, sugar and enzyme concentrations usually contribute even less to the diagnosis than a cell count or smear (which are only occasionally helpful).

Cytology and "cell block" studies of pleural fluid sediment, or needle biopsy of the parietal pleura, often help to identify an underlying malignancy. Pleural needle biopsy is also an effective method for confirming the presence of tuberculous granulomas or obtaining more-likely-to-be-diagnostic material for culture of tubercle bacilli.

Removal of a large pleural effusion often improves roentgenographic visualization of pleura and lung parenchyma. Early complete drainage of an infected effusion (empyema) helps reduce systemic toxicity and reactive pleural fibrosis, while also improving antibiotic access to any intrapleural bacteria.

Large pleural effusions and major "tight" abdominal ascites both restrict respiration so their complete removal usually results in symptomatic relief. The question then commonly asked? "Might any harm follow rapid, complete evacuation of a large volume pleural effusion?"

Well, experience suggests that rapid and complete removal of massive ascites may actually be dangerous. Indeed, the rapid external drainage of massive ascites has caused syncope or even death.

Case Report: The Tub Overflowed

She finally awoke in response to pounding on the door, then became hypotensive and collapsed. The downstairs tenant had attributed gallons of fluid leaking through his ceiling to an overflowing bath tub but in actuality the patient had spontaneously perforated her umbilicus while asleep, wherefrom drained much of her massive ascites.

Case Report: If a Little Feels Good, a Lot Can Kill You

Another middle-aged cirrhotic got so much relief from the initial, partial removal of his tight ascites at the clinic, that he went home, removed the stitch from the trochar site, completed drainage of his ascites into the toilet, collapsed and died.

Discussion: Whatever its cause (visceral vasodilation, hypovolemia, neurogenic or chemical), hypotension definitely can result from complete or rapid external drainage of tight ascites. On the other hand, hypotension is not seen following removal of a massive pleural effusion (even in those unusual patients whose effusion supposedly represents ascites that has been drawn into the pleural cavity via minute diaphragm defects).

Peritoneovenous Shunting Prevents Hypovolemia

Hypotension is not likely to follow peritoneovenous shunting of ascites either; here the more common problem is an early intravascular fluid overload. Interestingly, the low gradient required to open a peritoneovenous shunt then allows "fine tuning" of such a patient's intravascular fluid volume by tightening or loosening the abdominal binder. Reportedly, ascites may continue to move into the pleural space and produce ongoing effusion, even after successful peritoneovenous shunt, due to the

normal pressure gradient from abdomen to chest.

This gradient (which can be accentuated by inspiration against resistance) also increases flow through the shunt, however, so the effusion usually is no longer significant in those few patients having apparent transdiaphragmatic passage of ascites. However, we are told that this persistent gradient prevents removal of already evident intrapleural ascites by a newly placed peritoneovenous shunt, so thoracentesis may be required initially after shunt placement. (I have no relevant experience with either peritoneovenous shunts or pleural effusions of ascitic origin.)

Pleural Effusion with Pancreatitis

The minor to moderate (usually left-sided) effusion that is sometimes associated with acute pancreatitis will normally subside along with that inflammatory process. On the other hand, massive pleural effusions, with high amylase and albumin content, are reported consequent to posteriorly directed pancreatic duct disruption. As might be expected, retroperitoneal collections of "pancreatic juice" tend to flow into the lower pressure mediastinum, and from there onward to one or both pleural cavities; this process apparently continues until the underlying pancreatic problem has been dealt with.

We are told that pancreatic duct disruption may occur with a first episode of alcoholic or traumatic pancreatitis, but not with pancreatitis due to biliary tract disease. Also that anterior duct disruption is more likely to result in a pancreatic pseudocyst or pancreatic ascites than pleural effusion. Recommended treatment for massive pleural effusions of pancreatic origin is "pancreatic rest": Endoscopic or operative pancreatography, and resection or gastrointestinal diversion of the pancreas are apparently only considered for the patient with persistent leak.

Pancreatic rest therapy allegedly includes avoidance of oral intake, continuous nasogastric suction, intravenous hyperalimentation for two or three weeks and even low dose irradiation of the pancreas (the latter to reduce secretions in poor risk patients). I have not been involved in the care of patients with massive, chronic pleural effusion secondary to pancreatic duct disruption.

Venous Cannulation May Cause Pneumothorax or Air Embolism

The tip of a central venous pressure catheter or hyperalimentation line may initially pass out through the wall of the large vein within which it was placed, or only later erode into the mediastinum or either pleural cavity. Significant pneumothorax can result from lung puncture or intrapleural air delivery during intravenous cannula placement. Of course, atmospheric access to an open intravenous cannula may lead to venous air embolism at any time.

For example, venous air embolism can reportedly become a significant complication of peritoneovenous shunting unless the shunt is kept occluded during any subsequent laparotomy. Of course, all intra-abdominal free air must also be removed prior to abdominal closure in the presence of such a shunt. Fluorocarbons might prove useful for arterial air embolism if they can aid oxygen delivery through arteries temporarily "more-or-less" blocked by bubbles.

The harmful impacts of air accumulations on the venous (right-heart) side, relate primarily to the inability of the heart to propel "froth" effectively. Allegedly a 50 cc dose of intravenous air is enough to block the circulation and cause death. But at least the difficulty in pumping froth (its compressibility and low density) usually prevents the right ventricle from pushing air through to the left side – and it

also allows early rescue of the patient by removal of such froth through suction on a central venous or pulmonary artery catheter (see note on scalene node biopsy, Chapter 14). Incidentally, the high compressibility and low density of froth is the reason that one cannot swim back up to the water's surface from beneath a waterfall or in "high surf" conditions.

A Pleural Effusion May Represent Misdirected "Intravenous" Fluids

Initial misplacement or later migration of a central venous catheter tip should always be suspected in the presence of a rapidly increasing, unilateral or bilateral pleural effusion, for this effusion could represent intrapleural delivery of intravenous fluids. Obviously, such a partial or total intrapleural delivery will result in less fluid being administered intravenously than anticipated: This can easily lead to dehydration, hypovolemia and delayed or inadequate absorption of "intravenous" medications.

In addition, if the misdirected fluid is hypertonic (e.g. hyperalimentation fluid - which can also cause "pleurisy" in this situation), significant additional volumes of extracellular fluids will be drawn into the pleural cavity by osmosis (further accentuating the circulatory hypovolemia). Such excessive (and usually rapidly accumulating) intrapleural collections of "intravenous fluid" can fatally compress lungs and heart in the same fashion as a tension pneumothorax.

Death may follow before the etiology of the increasing dyspnea or extent of the growing effusion have become apparent. To add to the confusion, the malfunctioning intravenous line may previously have been in satisfactory position and working effectively (even for many days) and the patient was most likely seriously ill to start with, for otherwise a central venous catheter might not have been placed.

Under such circumstances, the clinical deterioration that suddenly becomes evident is often attributed erroneously to the primary disease process. On the other hand, the increasing dyspnea can as easily be blamed upon pneumonia or pulmonary edema – the treatment of which might normally include restriction of fluids or vigorous diuresis: Cardiac compression by the massive pleural effusion may also raise the venous pressure, another indication of hypervolemia. If, despite all of these confusing signs and symptoms, circulatory hypovolemia is correctly suspected, the normally appropriate response - to increase the rate of intravenous fluid delivery - would merely expedite an already impending death from massive pleural effusion.

Fortunately, there is a simple, safe and sensible escape from this diagnostic merry-go-round, for any dyspneic patient with significant pleural effusion should have that effusion drained promptly, preferably by chest tube. When such an iatrogenic pleural effusion is drained and examined, not surprisingly it looks a lot like whatever is being delivered intravenously (a serous effusion is likely, with very high glucose levels).

Drainage of this effusion also helps to clarify the hypovolemic state, which should be remedied promptly by some different "assuredly intravenous" route (see Chapter 14). Interestingly, neither the initial misplacement nor a subsequent withdrawal of the intravenous catheter is likely to cause significant intrapleural or mediastinal bleeding in any patient with normal coagulation parameters.

Entrapped, Shrunken or Atelectatic Lung Resists Reexpansion

Major intrapleural air or fluid accumulations displace lung: Their later evacuation usually causes

transient pleuritic pain, cough and expectoration as lung reexpands to fill the additional space thus made available in the pleural cavity. Not uncommonly, however, lung that has been "down" for weeks or months will resist reexpansion: Atelectasis, consolidation and fibrosis of lung, or inflammatory or malignant thickening of its surrounding visceral pleura, may stop lung from "rising" readily (or perhaps ever).

In this event, markedly subatmospheric pressure can develop between a small, rigid lung and its larger pleural cavity as drainage continues: Excessive traction on lung and mediastinum causes severe pain and an increasing "pull" is required on the thoracentesis syringe plunger. One must either discontinue thoracentesis at this point, or allow some air to enter the pleural cavity before additional fluid is withdrawn (see Chapter 6).

Pulmonary Edema has been Reported Following Reexpansion of Lung

"Reexpansion pulmonary edema" is rare, dangerous, and usually unilateral – a result of efforts to "force" an atelectatic lung to reexpand by rapid removal of adjacent intrapleural air or fluid. The problem is that airless lung naturally resists reexpansion as long as endobronchial mucus blocks air intake. The solution is to stop an ongoing removal of intrapleural air or fluid (by syringe, siphon, or mechanical chest bottle suction) from around such a "corked-off" lung whenever this starts to cause severe pleural pain. For otherwise, excessively subatmospheric intrapleural and intrapulmonary pressures will drag fluid out of lung capillaries into nearby collapsed and airless alveoli.

If this explanation is valid (as I believe it to be), one can prevent the occurrence of reexpansion pulmonary edema most simply by just terminating pleural fluid (or air) aspiration whenever this leads to excessive traction on lung (as signaled by increasing pleural pain). It is therefore very important for the patient to cough frequently and vigorously during lung reexpansion, in order to "raise" and thereby eliminate the obstructing mucus.

A logical remedy for reexpansion pulmonary edema (consequent to too rapid removal of an effusion) might be immediate recollapse of the affected lung (letting free air enter the chest through an open needle or open chest tube to establish a stable simple pneumothorax). That new simple pneumothorax could then be removed in small steps by needle or via an intermittently unclamped tube (after pulmonary edema has resolved) using ordinary water seal drainage; then the lung only reexpands as vigorous coughing clears additional airways (certainly a more gentle, comfortable way to bring air back into lung than by application of excessive external traction through intrapleural suction; see also Chapter 5).

Serous Effusion

A minor to moderate, serous pleural effusion cannot always be explained. Careful chest roentgenograms and analysis of pleural fluid are nonetheless indicated. Transient prior or concurrent febrile illness, in the absence of other findings, suggests a viral infection (which is no longer as difficult to document, if further investigation seems essential). If one can reasonably rule out bacterial infection, cardiac failure, significant asbestos exposure, ovarian tumor, apparent pulmonary or pleural abnormality, chronic systemic (e.g. "collagen" or autoimmune) disease, chylothorax, cirrhosis, renal and pancreatic pathology as a cause of the effusion, one should suspect tuberculosis, even with a negative skin tuberculin test (and especially if the "sterile" effusion in a young adult contains a few red and mononuclear cells with "lymphocytic predominance").

Under these circumstances, careful examination and culture of a "good" sputum sample is recommended, as well as a repeat, properly performed, skin tuberculin test (using known-effective antigen). Pleural involvement by malignancy sometimes remains a diagnostic consideration with this sort of effusion, so it is reassuring if one can demonstrate a pleural granuloma by closed needle pleural biopsy, or culture tubercle bacilli. The usual cause of a tuberculous effusion is presumed to be widespread intrapleural dissemination of bacteria and bacterial antigens after rupture of a small, subpleural, primary tuberculous infection.

Despite such intrapleural rupture, tuberculous effusion will rarely, if ever, be grossly bloody in appearance. The incidence of tuberculosis is decreasing rapidly, so this diagnosis becomes less likely in the absence of known exposure: A course of antituberculous chemotherapy may still be a reasonable fall-back position, however, especially if such therapy is guided by an infectious disease specialist familiar with recent advances in tuberculosis chemotherapy.

Although a tuberculous pleural effusion is usually "self-limited", systemic infection with tuberculosis often follows within five years (in the absence of anti-tuberculosis chemotherapy). The declining incidence of tuberculosis has increased the likelihood of primary infection in "immunologically naive" elderly patients.

Bloody Effusion: (1) What is wrong? (2) How bad is it? (3) What can be done?

There are many occasions in medical practice when an expensive finding seeks a question to which it is relevant. Logic suggests, and experience proves, that patients are usually best served (least pain, cost and delay) if evaluation of their complaint proceeds in an orderly fashion. For example, a large-volume bloody pleural effusion, not associated with trauma, pulmonary embolus, clotting abnormality, endometriosis (see Chapter 5) or pneumothorax, is most likely due to pleural invasion by malignancy. In a patient not already known to have malignancy, tissue diagnosis is the first concern, because prognosis and therapy depend upon it.

Unfortunately for the patient undergoing biopsy by a surgeon, evaluation of extent of disease by an internist or oncologist, and therapy by surgeon, oncologist and radiation therapist, there is ample opportunity for delay, poor communication, confusion and loss of rapport. It is therefore crucial that the patient and all involved physicians agree on "who is in charge" at any point in this progression, and that all understand and cooperate with the current "plan" (and then, of course, there are the countless relatives who will want to know "everything". . .).

With bloody effusion and possible pleural malignancy, the surgeon's role will usually be limited to biopsy and drainage (not necessarily in that order). I often find it convenient and helpful to perform an indicated biopsy during the first evaluation, and usually request a "frozen section" microscopic examination (to reduce "patient turn-around time") when there is sufficient biopsy material.

It is kind to the patient with possible malignancy to see him as soon as possible, and to then move him on decisively to the next physician with diagnosis in hand. It also improves patient acceptance of diagnosis and prognosis, as well as compliance with proposed treatments, if endless, costly and inconclusive preliminary evaluations are avoided.

Patients may reach the surgeon at various stages in their "workup." Many internists seem reluctant to consider surgical biopsy prior to extensive, expensive evaluation of all peripheral complaints. Every

specialty has a set of compulsive rituals designed to avoid missing something important, or even (hopefully only rarely) to augment income. And I also am reluctant to send a patient onward without first seeing a recent roentgenogram of the chest ("You saw a chest surgeon and he missed this?!").

Although many patients are only referred after interminable delay, it is well to remember that gradual change under observation is often invisible. It is especially unfair to apply 20/20 hindsight when the patient has finally been diagnosed and referred - for you, too, might have overlooked the problem at that earlier stage. (On the other hand, "How could anyone miss such an obvious... ?")

Question #1... What is wrong?

With a major, bloody effusion, the patient's symptoms and the radiographic evaluation of his pleura are usually both best served if the effusion can be removed by needle aspiration or tube drainage (and occasionally a little air admitted between thickened pleura and lung, to improve roentgenographic contrast at the pleura). If cell block and cytology studies on pleural fluid samples are then indeterminate (as is frequently the case), pleural or lung biopsy (by needle, bronchoscopy or mini-thoracotomy) should be considered.

Feel for Scalene Nodes First, Last and Always

First, however, a thorough, gentle palpation of the supraclavicular area and lower internal jugular vein often identifies a firm lymph node that is appropriate for biopsy. Such nodes are usually far deeper than they feel, and are among the most commonly overlooked abnormalities on routine physical examination.

There is no need for formal scalene fat pad excision here, nor should one worry overmuch about tumor spill, as any scalene node involvement will be a minor aspect of such disseminated malignancy: Just obtain a good tissue sample. (The radiotherapist can include the scalene area in a nearby treatment field anyhow.) Fine needle aspiration for cytology is allegedly usually sufficient for diagnosis of a malignant neck node: I have no experience with this technique, but it could be preferable, especially if neck dissection may follow.

Summary: Whether by cytology and cell block, node aspiration or open neck-node biopsy, endoscopy, needle biopsy of liver, lung or pleura, mediastinoscopy or chest incision, the least traumatic, most expeditious, most-likely-to-be-diagnostic biopsy is usually also the best method for evaluation of a probably malignant pleural effusion.

Question #2... How bad is it?

Computed tomography, sonography, liver, brain and bone isotope scans, and lung tomograms (after removal of effusion) - all are sometimes helpful in evaluating the location and extent of disease in lung parenchyma, pleura and elsewhere. Liver chemistries and gastrointestinal contrast studies often aid in evaluation of symptoms or help detect progression of the malignancy.

Question #3... What can be done for a malignant bloody effusion?

Here the surgeon can offer drainage and common sense, sometimes merely serving as a well-informed friend to the patient (and "interpreter," if oncologist or radiation therapist are too erudite).

Ideally, of course, we biopsy or drain as indicated, and then leave ongoing treatment in non-surgical modalities to those especially qualified.

Conclusion: Possible malignancy in and about the chest is best evaluated by surgery in and about the chest, with the least traumatic, most likely diagnostic procedures considered first. Although, advances in tumor chemotherapy tend to make early tissue diagnosis of increasing importance, the patient usually need not be pushed into more aggressive diagnostic procedures after thoracentesis, if he strongly wishes to delay. Bloody effusion due to malignancy implies that the malignancy is surgically incurable.

Pleural Malignancy

As of 1986, more can be said than done about malignancy originating in pleura. Chemotherapy for mesothelioma in the patient with chest pain, weight loss and bloody pleural effusion appears to reduce survival. Malignant mesothelioma (supposedly often associated with prior asbestos fiber exposure) has fortunately been very rare in my experience. I have not encountered a benign mesothelioma since my residency days.

Logically, any pleural tumor that can be cleanly removed, should be. But mesotheliomas are usually widely seeded over visceral and parietal surfaces before surgical attention is directed to them. It is reasonable to expect that a few malignant cells will multiply and spread rapidly over all communicating visceral and parietal surfaces.

On occasion, a lung lesion penetrating the visceral pleura also disseminates widely in this fashion, possibly because it did not stimulate sufficient inflammatory reaction to cause fusion of the overlying visceral and parietal pleurae before malignant cells penetrated the pleural surface. Anyhow, once such intrapleural seedlings "take," it is (at least surgically) "pretty much all over."

Pleural seeding that is not detectable by chest roentgenogram is often encountered at thoracotomy undertaken for a solitary "coin lesion," some months or years after removal of an overlying carcinoma of the breast; yet one lady in my practice is apparently free of disease, fifteen years following removal of just such a "coin" metastasis to lung.

Computed tomography of lungs and liver can be very helpful in the evaluation of a solitary coin lesion, but one should not consider any "possibly positive" study to be a confirmation of malignancy disseminated into the pleura, lung parenchyma or lymph nodes. Repeat "CAT scan" in two weeks will sometimes clarify that issue, but if surgical cure remains a possibility in a "good-risk" patient, "When in doubt, take it out".

Malignant Pleural Effusion

Significant compression of lungs and heart by an effusion due to intrapleural malignancy, can often be relieved by an indwelling chest tube or repeated needle drainage. Unfortunately, tumor "toxicity" and visceral pleural restriction often limit the symptomatic relief obtained from such fluid removal.

Nevertheless, several days of chest tube drainage may sometimes prevent a major recurrence of the malignant pleural effusion even though the heavily invaded pleura in such cases is often too thick and stiff to allow full lung reexpansion (or permit removal of all fluid, unless air enters simultaneously). It

is possible that intrapleural instillation of one or two modest doses of tetracycline may reduce pleural fluid production or encourage some adherence of these thickened, opposing pleural surfaces.

Tetracycline Treatment May Prevent Recurrence of a Malignant Pleural Effusion

Procedure: Drain all available pleural fluid, adding air to the chest tubing when necessary to relieve excessive suction (see Chapter 6).

Instill 0.5 g or more of "intravenous" tetracycline, dissolved in 50 cc of saline, into the pleural space via a thoracentesis needle or chest tube (I have used up to 2 grams at repeat treatments, but please see tetracycline drug brochure for a discussion of possible tetracycline toxicity).

Turn and tilt patient to distribute the medication. (Tetracycline solution on normal pleura can be excruciatingly painful - see later this chapter.) After an hour or so, again empty the pleural space of fluid.

Thereafter, try to maintain effective pleural drainage (preferably by chest tube) for one or more days, to encourage pleural reapproximation and adherence. With an ongoing large-volume effusion, consider leaving the chest tube in place until systemic tumor chemotherapy reduces the daily fluid output (or the patient dies or insists on chest tube removal).

Pleural Plaques

Not uncommonly, a routine chest roentgenogram of a middle-aged or older patient will demonstrate huge, vague, lung lesions, often multiple or partially calcified, that on tangential chest roentgenogram prove to be smooth-surfaced pleural plaques. Even when extensive, such acellular hyaline deposits are not the site of adhesions and have no surgical significance, except to suggest asbestos exposure some decades before.

These plaques are most commonly seen over the lower parietal pleura and diaphragm; when widespread, they can restrict chest wall movement to some degree. However, most such smooth pleural deposits are not calcified; being invisible on chest roentgenogram, therefore, they are simply incidental findings at thoracotomy: No treatment is indicated.

When calcified, they occasionally have a gritty feel (similar to multiple tiny metastatic pleural tumors, but harder). Prior chest films are especially reassuring in the occasional doubtful case, as any roentgenographic findings have usually been discernible and stable for many years.

A Pleasant Surprise: Hemothorax Not Due to Malignancy Will Usually Absorb

Patient response to free blood within the pleural space is variable. Only rarely will a major, clotted hemothorax finally entrap lung under a thick fibrous peel that requires surgical removal. Delay in surgical intervention for uninfected hemothorax is usually rewarded by satisfactory resolution, therefore, especially if aided by judicious use of chest tubes and deep breathing exercises.

An initially clotted hemothorax will normally liquify within a few days; such clot lysis is demonstrated by the increasing volume and mobility of pleural "fluid" around the lung, as seen on roentgenograms taken in various positions. When a stable and no longer bleeding, patient is not

otherwise a candidate for early reexploration, it is often reasonable to delay efforts at needle or tube drainage of a clotted hemothorax for several days. Incidentally, a clotted chest tube embedded in a clotted hemothorax often remains blocked even after the pleural space clot has liquified.

A clotted hemothorax commonly enlarges as it liquifies and blood proteins fragment into more numerous osmotically active particles. If symptoms increase at that time, the effusion can usually be drained without much difficulty by needle or chest tube. It is well to remember, however (especially when dealing with mild to moderate symptoms in a terrified post-trauma patient), that intrathoracic clot or liquified hemothorax can usually be eliminated by breathing exercises alone.

When is it Imperative to Evacuate a Hemothorax?

If pleural drainage will be required sooner or later, sooner is generally better. Blood is readily absorbed by normal pleura, but when a large-volume hemothorax is superimposed upon an established pleural infection (or combined with gross pleural bacterial contamination), it should be evacuated early, in order to reduce the severity and duration of intrapleural infection and limit reactive scarring about the lung.

With or without bacterial infection, when major postoperative hemothorax is combined with established pleural inflammation and fibrosis (as might be seen following surgical decortication of lung and chest wall) early evacuation of blood and clot should be achieved through more effective tube drainage or else by repeat thoracotomy (see later this chapter). In the absence of infection or established and progressive fibrosis, however, it is usually good judgment to avoid early thoracotomy for major undrainable hemothorax (if the patient is not likely to be lost to follow-up), as thoracotomy can almost always be avoided, and the main risk of delay is the minor possibility that if thoracotomy eventually is required, it could be slightly more tedious.

Breathing Exercises Really Do Remove Pleural Effusion

An especially deep inspiration every five minutes while awake, emphasizing maximum inspiratory movement of the shoulder and accessory muscles of the affected hemithorax (best practiced at first in front of a mirror), can virtually eliminate the need for postoperative thoracentesis. How or why this works is unclear, but it is regularly effective in speeding the absorption of intrapleural blood or serous effusion (but not malignant, congestive, inflammatory or infected effusions, unless and until the cause is controlled).

Following Sternotomy, Wide Drainage of Pericardium Into Both Pleural Cavities Will Also Simplify Pericardial Closure

Following sternotomy and cardiopulmonary bypass, I routinely open both pleural cavities widely (using electrocoagulation to divide each pleural envelope at its sternal reflection). Pleural extensions of the vertically divided pericardium are thus created which allow easy closure of a larger, "more relaxed" pericardium over the heart. This ensures eventual migration of any symptomatic postoperative hemomediastinum into one or both pleural cavities (where such fluid simply becomes another minor-to-moderate, bloody postoperative effusion - see Chapter 10).

All pleural effusions after sternotomy have responded to the breathing exercise regimen described above, plus diuretics for any element of heart failure. Thoracentesis has not been required subsequent

to postoperative chest tube removal, nor has postoperative empyema developed in my 700 consecutive patients who underwent sternotomy for cardiovascular procedures.

Therapeutic Pneumothorax

It is interesting to recall the days when induced pneumothorax - and occasionally, pneumoperitoneum - were used to treat cavitary tuberculosis by partially collapsing and thus "resting" the involved lung. Many lungs kept collapsed in this fashion (with frequent refills of the pneumothorax to replace air absorbed), eventually became "frozen in their squatting position" by fibrosis of the visceral pleura.

Some have since required decortication to achieve pulmonary reexpansion (usually in order to obliterate the residual pleural space after this became infected). In other cases, the bony thorax has been tailored down by thoracoplasty to fit the remaining lung volume. It should be emphasized, however, that the ongoing simple pneumothorax was easily tolerated by these patients (many of whom had very poor lung function), unless addition of too much air created a tension pneumothorax.

Principles of Absorption of Free Air

Air within a closed body cavity that is lined by living vascularized tissue will be absorbed over a few days or weeks, unless new air is added or the gas pressure becomes subatmospheric within a non-collapsing, thick-walled cavity. Any gas enters solution until adjacent fluids are fully saturated (in equilibrium) at that partial pressure. The circulatory exchange of these surrounding fluids will normally prevent such full saturation, however, so a gas-pocket-to-fluid gradient usually persists until all gas has been dissolved.

The rate of gas removal is a function of the solubility of that gas, as well as the saturation and circulation of surrounding fluids. Carbon dioxide is picked up most rapidly, oxygen more slowly and nitrogen even more slowly (carbon dioxide and oxygen undergo reversible chemical reactions in blood, while nitrogen merely dissolves). The gas contents of a non-collapsing, thick-walled cavity eventually come into slow equilibrium with the atmosphere and circulation.

Elimination of gaseous nitrogen (more than 80% of the content of any stable, closed space) can be accelerated by breathing pure oxygen: This decreases dissolved nitrogen in the blood, and thereby increases the gas-pocket-to-body-fluids gradient for nitrogen. Conversely, any "new" (unfamiliar) gas introduced to body fluids (e.g., nitrous oxide at high partial pressures) will increase the gas pressure within any air-containing, closed body cavity or blocked viscus by equilibrating into all such isolated air pockets that are surrounded by living tissue (since it is newly present in tissue fluids but absent from the gas pocket).

Travel by "unpressurized" airplane also can distend an enclosed collection of air by reducing the surrounding "atmospheric" air pressure. In this fashion, a stable simple pneumothorax might become tension pneumothorax during much of the flight, unless vented by an open needle or tube.

Warm Saline for Resuscitation of A Hypothermic Heart

Among undesirable side effects of cardiac (or total body) hypothermia are dysrhythmia, decreased cardiac output and cardiac arrest. "Unpleasantly warm" saline solution (barely tolerable to the

indwelling gloved hand) has often been poured into the pleural cavity during thoracotomy to help rewarm the heart and circulating blood: This is most effective in a small child but also helpful with an adult patient.

Considerable volumes of warmed saline may be required to increase the overall body temperature of larger patients. For example, a 100 kilogram man at 30°C, equilibrated with 100 liters of warm saline at 40°C, would only warm to 35°C (and this example disregards factors such as room temperature, direct heat transfer to surrounding materials, evaporative cooling, respiratory heat loss and metabolic heat production).

Despite its obvious inefficiency, the use of such intrathoracic "core rewarming," (at the time of internal cardiac massage, for example) may be more practical and effective than dipping a moribund, hypothermic patient (perhaps undergoing simultaneous cardiopulmonary resuscitation) into a warm 40°C bath. The warm bath approach also initially increases peripheral metabolism without simultaneously warming the heart, liver and kidneys to the point where they can cope with such additional metabolic needs.

Simple arithmetic suggests that metabolic heat production is essential to the success of any inner, or outer body-surface, rewarming procedure. An essentially dead, hypothermic, large child or adult in metabolic and cardiac arrest, probably cannot be rewarmed successfully except on cardiopulmonary bypass.

The above-described "hot" intrapleural saline was commonly utilized in past years, especially at the close of cardiac procedures on children, in order to reverse intentionally induced hypothermia, or to overcome incidental hypothermia that was preventing successful resuscitation. I have never recognized an intrathoracic complication (such as phrenic or recurrent laryngeal nerve injury, for example) consequent to such application of intrapleural heat, although this would seem possible and has been looked for.

However, I did once cause second-degree skin burns over the neck and thorax of a drug addict who suffered hypothermic arrest at surgery for abdominal gunshot wound (his "friends" had stuffed him into a snowbank). This man was successfully resuscitated, but died a month later of unrelated cause. Evidently his poorly perfused skin was more susceptible to heat injury than my gloved hand. One should probably avoid pouring so much hot saline into the pleural cavity that it massively overflows the thorax (if the anesthesiologist can successfully oxygenate without vigorous lung inflation), but this is of secondary importance compared to survival.

Reviving the Dead

Open chest cardiac massage, supported by heated, humidified, positive-pressure ventilation and warm intrapleural saline, might be worth trying on any salvageable individual recently (within one hour?) dead of hypothermia. Cardiopulmonary bypass obviously provides a more optimal circulation and temperature control, despite the half-hour delay often involved in getting organized (the hypothermic patient in cardiac standstill is meanwhile provided cardiopulmonary resuscitation as indicated). Above 32°C core temperature, cardiopulmonary bypass seems unnecessary to correct simple hypothermia (without some complicating cardiac or pulmonary problem), as gentle external rewarming plus metabolic heat production will usually suffice.

We have heparinized and successfully rewarmed several individuals who were suffering from exposure-induced hypothermia, with core temperatures below 28°C, using femoral vein to femoral artery, partial cardiopulmonary bypass. Femoral vein cannulation for partial bypass can result in poor venous flow unless the catheter reaches high into the inferior vena cava or right atrium. We have found that an appropriate-size chest tube, inserted almost full length (with its tip beveled to expedite introduction) into the barely larger common femoral vein, is a satisfactory venous cannula under these circumstances.

In our very small experience, cardiopulmonary bypass has easily reoxygenated and rewarmed hypothermic individuals with uncontrollable pulmonary edema due to drowning, drug abuse or excessive fluid that was required during cardiopulmonary resuscitation: Bypass has provided time for positive end-expiratory ventilation and appropriate diuresis, inotropic or antiarrythmic therapy, a trial of calcium antagonist (to overcome any coronary, cerebral or visceral artery vasospasm), barbiturate coma and steroid administration.

Surgical Hypothermia

Hypothermia reduces metabolic requirements of living tissues (by slowing chemical reactions), thereby prolonging tissue survival under hypoxic, ischemic or circulatory arrest conditions. As a surgical adjunct, mild hypothermia (to 30-32°C) may be used alone or with cardiopulmonary bypass: If necessary, moderate (20-24°C) or profound (12-18°C) hypothermia, in association with cardiopulmonary bypass, will also allow prolonged periods of total circulatory arrest (reportedly up to 30 minutes, or even more at the profound levels).

This has proved useful in those neurosurgical procedures and major resections of malignancies where excessive bleeding was anticipated. Allegedly, the risk of intravascular air problems is reduced if both arterial and venous cannulas remain clamped during the circulatory arrest, so that the patient stays "full of blood" rather than being "drained out" into the pump.

It is becoming apparent that metabolic recovery from an ischemic insult proceeds more efficiently during normothermic than hypothermic perfusion, so a period of normothermic circulatory support is usually provided before discontinuing cardiopulmonary bypass. We do not correct mild acidosis during hypothermia, for acidosis favorably affects peripheral oxygen release in the face of the temperature-related shift of hemoglobin-oxygen dissociation. A low hematocrit reduces blood viscosity, and thereby apparently improves perfusion during deep hypothermia: Allegedly, sufficient hemodilution (to less than 10 grams of hemoglobin per 100 cc) during surgery also reduces the risk of coagulopathy in polycythemic patients undergoing correction of cyanotic heart disease.

Hypothermia With Inflow Occlusion:

Although rarely used, mild external cooling, followed by inflow occlusion and circulatory arrest, is still an acceptable, reliable adjunct to allow certain brief cardiovascular procedures that can easily be performed without cardiopulmonary bypass. This technique is particularly applicable to congenital valvular pulmonic stenosis, a brief "right-heart" surgical procedure that carries no significant risk of air embolism. (A short interval of inflow occlusion has also been used to lower the tension within the ventricle or aortic wall during repair of a stab wound - I prefer an intravenous bolus of nitroglycerine to momentarily reduce these pressures.)

Inflow Occlusion Procedure: After tightening occlusive tapes about both cavae, wait for two or three heart beats to empty the right heart; then remove the previously placed side-clamp from the proximal main pulmonary artery, allowing the previously made vertical pulmonary arteriotomy to separate (each lip still held by a fine traction suture, already placed). The valve is now visualized in a relatively dry field, and commissures are opened as indicated; caval tapes are then released and the side-clamp reapplied (to exclude the pulmonary arteriotomy) as soon as blood has displaced right heart air: The pulmonary arteriotomy is subsequently repaired with appropriate vascular suture.

Comment: Obviously, any blood passing through a "left-to-right shunt" (such as an atrial septal defect, ventricular septal defect or patent ductus arteriosus) would interfere with necessary visualization of anatomy during this procedure, and also increase blood loss and risk of air entry into the aorta: Of course, with an open ductus, one could simply cross-clamp the distal main pulmonary artery. In any case, I have not utilized hypothermia with inflow occlusion for many years, primarily because we no longer see larger children and adults with pure pulmonary stenosis (and infants with this problem are referred elsewhere). There may be an increased risk associated with the use of temporary circulatory arrest in already very cyanotic patients. A five minute pre-oxygenation by hyperventilation has been recommended (with cessation of ventilation while the circulation remains occluded).

Surface Cooling:

Preliminary external cooling may also be helpful in situations where the possibility of massive intraoperative hemorrhage exists, as hypothermia seemingly allows more time for "recovery" from a "fumble" (as might occur during closure of a calcified ductus or repair of a difficult coarctation in an adult, for example). "It has been said" that a body temperature of 32°C supposedly prevents detectable neurological damage despite six minutes of circulatory arrest (two to three minutes has been given as the tolerable limit with normothermia). Appropriate caution, and familiarity with some reliable technique for moderate external cooling to 30-32°C, will prevent further accidental cooling to 28°C (which carries great risk of ventricular fibrillation).

Surface cooling procedures vary in technique and goal. Under appropriate anesthesia (supplemented by one or two mg. of thorazine intravenously as indicated, to prevent shivering), a normal-sized adult can be cooled with fore-and-aft cooling blankets plus multiple icebags to a core temperature of 30-32°C within 1-11/2 hours. The same temperature drop can be achieved through approximately ten minutes of immersion in ice water.

Skin freezing injury can occur with a frozen icebag (which may be at any temperature below 0°C), but an ice-and-water mixture for patient immersion always remains at 0°C and thus cannot injure skin during this brief, intentional exposure. Although surface cooling (to first reduce peripheral metabolism and thus total metabolic load), followed by core rewarming (to restore core organ metabolic efficiency before increasing peripheral metabolic requirements) has traditionally been considered optimal, the modern heat exchangers incorporated in disposable oxygenators have become so efficient that both cooling and rewarming are most safely and easily achieved during total circulatory support on cardiopulmonary bypass.

Case Report: Cooling by Immersion

Hypothermia, with cooling by immersion, was selected to increase the "safety margin" of an adult coarctation repair: A large clean laundry tub was to be filled with ice and water, but our hospital ice

supply proved insufficient, so the cart was pushed out of the surgery entrance and filled with clean white Alaskan snow. Sufficient water was then added to permit the anesthetized patient to be submerged (with bath temperature known to be exactly 0°C, as some ice and snow remained unmelted).

To avoid apprehension, the patient was anesthetized before the ice tub was brought into the operating room; his blood pressure cuff, esophageal and rectal temperature probes, and intravenous lines were carefully placed and sprayed with sealant or taped with waterproof tape. With the endotracheal tube in position, the patient was transferred to the tub on a canvas sling and completely submerged, except for his face: His exposed forehead was also covered with a wet cloth that was kept cool with ice and ice water.

The exact moment of immersion was recorded; as usual, the intent was to keep the patient submerged for exactly ten minutes, or until a core temperature drop of 1.5°C was accomplished - whichever came first. Both temperature probes were watched carefully for change. (Any sudden drop in the rectal probe temperature indicates an ice-water enema, while lack of temperature change in the rectal probe suggests that its tip is buried in a large, warm stool which might be expected to cool more slowly than nearby living tissues directly exposed to the cooling blood circulation.)

Temperature probes showed 1° and 1.5°C temperature drop after nine minutes immersion, so the patient was lifted back onto the operating table, dried, and placed in lateral position for thoracotomy (while his core temperature drifted on down to 32°C). After uneventful coarctation repair, the warming blanket (plus warm intrapleural saline) brought the patient back up to 34°C by the time he went to the intensive care unit where external warming by the circulating warm alcohol blanket was continued to 36°C, then discontinued. During his early postoperative hours, the patient developed a fever above 38°C (101°F) rectal, which was easily reduced by the circulating (now cooling) blanket; he remained normothermic subsequently.

Early postoperative fever (with cool dry skin) is common after mild to moderate hypothermia, so it is usually best to leave the rectal temperature probe and cooling blanket in position overnight. Prophylactic tetanus toxoid injection was given to this patient after surgery, when the orderly discovered moose droppings at the bottom of the cooling tub. Thereafter, we avoided use of snow by placing our ice orders early whenever possible. The patient did well.

Other Complications of Hypothermic Technique:

One other complication occurred with this external cooling technique; a lifting sheet split in two just as we were raising our heavy patient out of the new, specially constructed fiberglass tub, and the patient sank directly to the bottom. The quick-thinking anesthesiologist immediately covered the endotracheal tube tip with his thumb, while the rest of the operating team "dove to the rescue." Which reminds me, there is often minor leak or spill of ice and water about the operating room floor with this technique, so it is well to have extra bath blankets available, to dry both patient and floor. This patient also did well.

In those years we worked with an Alaskan Indian "surgical technician" who was not distressed by prolonged immersion of his hands in ice-water. (Local researchers have watched northern Alaska natives rewarm their bare hands occasionally by immersion in icewater when fishing through ice at minus 40°C.) I have not had occasion to use external cooling without cardiopulmonary bypass for a

number of years. Nonetheless, it still could be a useful technique for a problem case.

The main risk of this mild to moderate, purposeful "immersion hypothermia" (excluding preventable problems such as drowning, overcooling to levels of ventricular irritability below 28°C, or uncorrected postoperative "rebound" hyperthermia) is that residual hypothermia may not be recognized as a cause of persistent general anesthesia. A patient at 33° or 34°C core temperature may exchange air well, and even move when stimulated, but the endotracheal tube should almost always remain in place until he rewarms to at least 35° or 36°C. All too often, I have seen an anesthesiologist, unfamiliar with this technique or eager to get home ("Once my tube is out, he's yours"), remove the endotracheal tube while the cool patient still appeared somewhat somnolent, only to replace it as a semi-emergency, five or twenty-five minutes later.

Moral: A cool patient wakes slowly, and must be considered still under anesthesia until normothermic.

Postoperative Shivering

Vigorous involuntary shivering is often seen in the recovery room, regardless of body temperature or prior hypothermia. Very small doses of thorazine delivered intravenously (1 mg at a time and wait 5 minutes) will usually control postoperative shivering without significant sedative effect. Arterial blood pressure may sometimes drop with such minimal thorazine administration (especially after cardiopulmonary bypass), but such mild to moderate hypotension usually responds promptly to reexpansion of the patient's blood volume.

Extrapleural Thoracotomy

Elective extrapleural surgery is still recommended occasionally for closure of a ductus arteriosus, repair of coarctation or correction of esophageal atresia, in order to "reduce risk" or "improve sealing in case of a leak." This approach could well have been valuable in the days of leaky esophageal repairs, misunderstood chest drainage, in-adequate antibiotics and primitive anesthesia. I have no experience with extrapleural correction of the above congenital problems; if it is easier or faster, that is reason enough to continue this approach. Dissection in the extrapleural plane can often help the surgeon avoid opening into a nearby malignancy or infection: It may also become necessary for surgical access to lung or mediastinum when post-operative or postinflammatory changes have obliterated the underlying pleural space (see Chapter 4).

Patent Ductus Arteriosus Closure in the Newborn

Premature newborns frequently require surgical closure of a patent ductus before they can be weaned from their respirator: However, transfer of a heavily monitored, critically ill, one-to-five pound infant from the neonatal ICU to surgery and return is a major nuisance at best, and often hazardous to the patient (who may be difficult or impossible to find, deep amongst tubes and wires, if an alarm goes off en route).

For the past few years, we have performed all neonatal ductus ligations in the newborn ICU bed already occupied by the patient, who is simply turned to a lateral position for left 4th interspace thoracotomy (assuming the usual left-sided aorta). Superb echocardiographic studies by pediatric cardiologists have greatly simplified diagnosis of this and other cardiac conditions in the newborn.

With our smallest, sickest infants, we may simply use muscle relaxant, with "anesthesiologist standby" primarily for legal purposes. We have also utilized tiny doses of morphine and ketamine. Because existing renal dysfunction may be aggravated by premedication with tiny doses of morphine delivered into an umbilical artery catheter (and thus more or less directly into the kidneys), we administer such medications only by peripheral venous lines.

Unfortunately, renal, pulmonary and cardiac dysfunction are the norm when these tiny patients are referred for ductus ligation (after failure of indomethacin to bring about ductus closure through prostoglandin inhibition). In my series of about 100 patients there were no surgical complications (although a number of cardiorespiratory deaths and serious renal problems were encountered in these very ill newborns).

Two other premature patients were seen in consultation when such ductus closure led to persistent chylothorax; one eventually required re-exploration (after 5 weeks of high-volume lymph drainage by chest tube) in order to achieve hemaclip closure of still leaking sub-pleural lymphatic tissue over the origin of the left subclavian artery. After the lymph leak was secured, we applied a few particles of tetracycline powder to the pleural edge in this area with a sterile cotton-tip swab (to encourage fibrosis), but I have no evidence that this was either useful or necessary. The infant did well.

It has been reported that chylothorax is a self-limiting condition that rarely lasts over three months. Some surgeons prefer to temporize by insertion of a pleuroperitoneal shunt rather than perform thoracotomy for thoracic duct ligation or direct control of the leaking area. Apparently a Denver double-valve peritoneovenous shunt is inserted (Denver Biomaterials, Inc. Evergreen, Colorado) with the subcutaneous pumping chamber left over a rib for ease in pumping until the fistula seals.

I cannot comment on that technique, but these chylothorax patients had drainage thick enough to block a chest tube repeatedly; hence, if used, such a shunt would probably have to be pumped very frequently. And I might need more time to place such a shunt than to correct the lymph leak, but surgical correction of chylothorax is infrequent so no one can be certain of the best approach.

Details of Procedure for Neonatal Ductus Ligations:

For the duration of the 20-40 minute procedure, neighboring infants are pushed back, relatives excluded, and other nearby traffic limited to those in cap, gown and mask. A surgical headlight, or bed-mounted fiberoptic spotlight, is essential. With increasing age, 1 find 2.5 power loupes helpful in magnifying the ductus area during sharp dissection, and especially to aid in identification of the recurrent laryngeal nerve. Using this magnification in one or two pound patients, it is sometimes possible to see blood flowing within the intact ductus.

Tiny hemostats can control muscle bleeding; they are removed on closure (but careful electrocoagulation is also effective and probably decreases blood loss). The chest retractor can be a small, single or double-action, self-retaining "biopsy" retractor. Small hemaclips are applied to the hemiazygous vein if this must be divided during mobilization of pleura from the aorta (it is often difficult to get a finger or even fine instruments into these squirrel-sized patients in order to tie off their tiny vessels).

A length of fine suture through the medial flap of pleura that was elevated from the aorta, often serves as an appropriately small retractor when weighted with a small clamp. Gently grasping nearby

aorta with a DeBakey forcep and exerting slight traction on this, will often straighten the ductus and bring it to a more accessible position for clip application. Traction on lung may cause additional hypoxia and bradycardia, so the intrathoracic portion of the operation is closely monitored and interrupted as seems necessary.

The ductus need not be circumferentially freed, though its sides must be cleared (a ductus is usually deeper than it is wide, thus readily damaged by blind passage of a clamp about its posterior limit), and the recurrent laryngeal nerve is identified and protected. The ductus is closed with one or two medium hemaclips, using a hemaclip applier that has just been tested on a thin drape edge (defective appliers sometimes "scissor").

CHAPTER 4: PLEURAL INFECTION AND PLEURAL FIBROSIS

Tuberculous effusion and tuberculous empyema;... Other reactive effusions;... Purulent effusion (empyema);... Evaluation of empyema;... Persistent underlying pneumonia;... Intrapleural antibiotics;... On the postpneumonectomy bronchus and its pleural cavity;... Postpneumonectomy empyema;... Clagett procedure;... A chest tube never causes empyema;... Drainage of empyema should be accomplished early;... Rib resection;... Care of empyema tubes;... Antibiotics and superinfection of the chronic empyema space;... It was important to make a trial disconnection of the water seal before converting to open drainage;... Do not discontinue suction too soon;... A reminder: Pleural space obliteration is the goal;... Tube amputation versus the outpatient chest bottle;... Empyema tube removal;... Bronchopleural fistula:... A bad shape may bring chronic empyema to decortication;.. Decortication, with or without lung resection;... Pleurectomy;.. Decortication;... Open the fissures during decortication;... Lung surface is often torn during decortication;... Mobilizing the lung at thoracotomy;... Case report: Torn pulmonary artery;... Mobilized lung reduces postoperative complications;... Adhesions in perspective;... Congenital fusion or absence of pleura;... Apical pleurectomy for spontaneous pneumothorax;... Pleural irritation to promote symphysis;... Extrapleural dissection for surgical access or en masse resection;... Case report: Extrapleural pneumonectomy for primary osteogenic sarcoma;... Role of pleural thickening in the prevention of recurrent spontaneous pneumothorax;... Instillations to achieve pleural space obliteration following spontaneous pneumothorax;... Tetracycline instillation during persistent minor to moderate air leak.

Tuberculous Effusion and Tuberculous Empyema

Among the many causes of minimally symptomatic serous pleural effusion is tuberculosis. Most such "benign" (self-limited) tuberculous effusions are probably secondary to intrapleural rupture of a primary infection, although effusion is also seen with established disease or disseminated (miliary) tuberculosis. A minority of tuberculous effusions reveal mycobacteria on culture or acid-fast stain of the fluid; serous effusions seen in a patient with a history of exposure to tuberculosis, or that antedate the diagnosis of tuberculosis, or resolve during a trial of antituberculous chemotherapy, are often labeled only in retrospect.

We have been taught that the more diligently we seek tubercle bacilli (in material derived from a tuberculous effusion), the more likely we are to find them. Sometimes, however, even when the diagnosis appears self-evident, "They just ain't there, Doc." This means that (despite its declining incidence) one must still keep tuberculosis faintly in mind whenever evaluating an ongoing serous effusion, even after negative studies on the fluid and negative skin tests (although closed-needle pleural biopsy allegedly increases diagnostic accuracy).

The indolent pleural response to tubercle bacilli or their breakdown products is quite unlike the inflammatory and pus cell response of pleura to other bacterial invaders. As a result, the usual serous tuberculous effusion need not be evacuated by chest tube or needle while awaiting the clinical benefits of chemotherapy. Indeed, undrained tuberculous "empyema" usually resolves spontaneously without local sequellae, regardless of chemotherapy (although a rare, sterile, chronic empyema cavity that contains milky, birefringent cholesterol crystals is allegedly the consequence of a prior tuberculous effusion).

When a tuberculous empyema is complicated by secondary infection, however (meaning superinfection by pus-forming, inflammation-causing bacteria), it should be treated as an ordinary bacterial empyema: Such fluid is then evacuated as soon as possible, to prevent progressive pleural

inflammation and fibrous restriction of underlying lung. Logic suggests care in the handling and disposal of all "possibly tuberculous" drainage or dressings, even though there is supposedly no evidence for transmission of tuberculosis by such fomites. (As pointed out in Chapter 3, an untreated tuberculous empyema will usually "clear," even though the patient often develops some other manifestation of tuberculosis within the following five years. Of course, reactivation of tuberculosis is always a possibility in immunologically deficient individuals.)

Other Reactive Effusions

A sterile effusion is often seen in association with mild or moderate pneumonia: This usually disappears following control of the inflammatory process, leaving insignificant filmy to fibrous interpleural adhesions in its wake. More severe and persistent lung infection is often associated with increasing loss of pleural capillary integrity. Blood plasma then accumulates within the pleural space, leading to shaggy gray fibrinous deposits on pleural surfaces and large gray-white clumps of stringy debris drifting in the proteinaceous pleural fluid. In time, this situation, too, often resolves (perhaps after causing significant pleural scarring), unless active pleural space infection intervenes.

Purulent Effusion (Empyema)

Any major bacterial invasion of a pleural space will be associated with an inflammatory pleural response and effusion; the toxic purulent intrapleural accumulations of varying severity and chronicity that result are all labeled empyema.

Healthy pleural surfaces commonly resist bacterial invasion and "clean up" minor infective spills at surgery (especially if the underlying lung parenchyma is not also diseased). Many minor accumulations of infected debris in the pleural space also wall-off and eventually absorb without long-term ill effect, except for local pleural scarring. These benign outcomes are aided by systemically administered antibiotics that seep into the pleural fluid.

As the volume of intrapleural fluid and debris builds up, however, tissue resistance and antibiotic access from the circulation will no longer suffice. The surgeon must then help pleura to fight this expanding infection by encouraging reapproximation of infected visceral and parietal pleurae through needle or chest tube drainage of the pleural space or (increasingly rarely) by open evacuation of semi-solid pleural content through the bed of a resected rib. A laparoscope is reportedly also useful for cleanout of such debris from the closed pleural space under general anesthesia, while each lung is ventilated separately.

Summary: Complete lung expansion (which means elimination of all significant accumulations of air, fluid and debris from the pleural space) is the best way to prevent or treat intrapleural infections.

Evaluation of Empyema

In treatment of purulent effusions, the surgeon must provide adequate samples of pleural fluid (for bacterial smear and culture) at the outset. To derive information that is helpful in the care of these severely ill patients, such samples must be submitted properly and processed promptly. Even when bacterial infection is apparent on examination of material obtained at thoracentesis ("positive gram stain, many polymorphonuclear white cells"), a specific bacterial agent may never be identified, especially if antibiotic treatment is already under way.

Persistent Underlying Pneumonia

Empyema is sometimes associated with chronic underlying lung infection. Anatomical causes of persistent or recurrent pneumonia include obstruction of a bronchus, as by benign or malignant tumor, or foreign body, or enlarged lymph nodes (as in "middle lobe syndrome"), or calcified lymph nodes eroding into a bronchus (as a "broncholith"). Lung can also become predisposed to infection by more peripheral pathological changes, as seen in bronchiectasis.

Systemic abnormalities that can lead to ongoing or recurrent lung infections include cystic fibrosis and immunologic depression (possibly associated with remote malignancy or caused by tumor chemotherapy, steroid treatment, AIDS, or other unusual inherited or acquired disease states). Cigarette and alcohol abuse and poor dental hygiene also predispose patients to pneumonia, lung abscess and empyema. Rare amoebic or fungal infections (more often burrowing, localized abscesses) should be kept in mind, as well as infection of a congenital lung anomaly or cyst, or superinfection of an echinococcal cyst.

It is often the chest surgeon who must guide the evaluation of a persistent pneumonia; the timing and goals of such a search are frequently confusing to the medical specialist. Helpful diagnostic maneuvers include chest roentgenograms in various positions (to demonstrate fluidity of pleural content), fluoroscopy, computed tomography, bronchoscopy, bronchograms and (occasionally) selective bronchial brushing (for cytology) or possibly even bronchial cannulation (reportedly useful for drainage of a lung abscess, utilizing one of the many special "heart" catheters now available).

Good surgical judgment will hold many or all these diagnostic modes in reserve, however, if the patient is recovering. A two-week delay in discovering obstructing carcinoma or chronic bronchiectasis will likely have little long-term significance. Possible unnecessary tests that stress already toxic patients are to be avoided, especially since most such patients will improve under treatment and thereby become "better risks" in the interim. A far smaller number of tests can then be performed on less acutely ill patients (surprisingly many patients recover completely without the benefit of a definite diagnosis).

It is important to determine how the patient is faring by repeated physical examinations and observation of his clinical course, as well as by evaluation of sequential X-ray changes: All too often, decisions for further diagnostic or therapeutic interventions are based upon roentgenographic findings only; yet roentgenographic evidence of lung consolidation may persist for weeks or even worsen after the onset of definite clinical improvement. One can often estimate how much time will be required for consolidated lung to clear on roentgenogram, from the prior duration of such consolidation: In other words, the longer it took to get "that way," the more time it takes to improve.

As an example, acute pulmonary edema often "clears" on x-ray examination within hours after it has resolved clinically. Similarly, acute pneumococcal pneumonia may not even become apparent on chest roentgenogram until the day after effective therapy has started, when the patient already feels much improved: These delayed x-ray "findings" then vanish within days. If one thinks about it "microscopically," there is at least a week's work to do, in addition to killing bacteria, before alveoli of a heavily consolidated lobar pneumonia can even be partially cleared of debris - in order to allow some reentry of air - and reentry of air must occur before the roentgenogram begins to look normal.

Intrapleural Antibiotics

Intrapleural instillation of antibiotics sometimes permits delivery of therapeutic drug concentrations into regions not otherwise accessible to circulating drugs. This is particularly true of the postpneumonectomy cavity that was contaminated by a spill of pus during removal of an infected lung. In this situation, frank empyema can often be avoided by vigorous intraoperative irrigation of the pleural cavity with warm saline, followed by one or more intra- and postoperative instillations of intrapleural antibiotics.

Additional postoperative doses of the more irritating antibiotics such as aqueous penicillin, can be very painful when delivered onto "normal" pleura (that is, pleura not submerged in fluid or coated by fibrin or invaded by granulation tissue or tumor). It is the prophylactic and therapeutic use of systemic antibiotics, however, (before surgery, during cleanup, and after such spills) that has almost eliminated chronic postpneumonectomy empyema as a significant clinical problem. (Of course, postoperative empyema should be very rare when underlying lung remains available to fill the pleural space.)

On the Postpneumonectomy Bronchus and Its Pleural Cavity

Amputation of an entire lung leaves an "empty," air-filled pleural cavity that gradually fills with bloody fluid. In the first day or two after pneumonectomy, it is commonly necessary to vent the overlying air pocket several times, to correct an undesirable increase of the intrapleural pressure. The air remaining thereafter usually absorbs uneventfully over subsequent weeks (see Chapter 9).

In the meanwhile, the increasingly "stale" collection of pleural fluid (its contents neither circulating nor regularly exchanged) gradually becomes concentrated: Walled-in by thickening, progressively less vascularized fibrous tissue, the enclosed proteins gradually denature and precipitate, losing their osmotic effect but still stimulating surrounding tissue surfaces to more scar production.

As thick pleural surface scar gradually contracts and intrapleural air is absorbed, the pleural cavity turns into a stable shrunken fibrothorax. Typically this will have a small central core of turbid fluid encased in a thick layer of "old fibrin" (not unlike the "crud" found within many chronic abdominal aneurysms), all enclosed by heavy layers of avascular fibrous tissue.

Before that stable state has been reached, however, the tenuous bronchial suture line must heal and remain airtight: Bronchial healing depends upon preservation of the (bronchial artery) blood supply to the healing edge. Living pleura sutured over the closed bronchial stump probably provides additional security.

Monofilament suture material, such as wire, polypropylene or staples, apparently reduces the risk of suture line infection (absorbable suture material has also been reported safe and effective in sleeve resection of a bronchus, but I have no experience with this use). It could be that the buried monofilament suture line is what prevents the ordinary postpneumonectomy empyema (without air leak) from affecting the healing bronchus.

Despite all efforts, however, a rare, postpneumonectomy bronchial stump will leak or disrupt following pneumonectomy, especially with prior mediastinal irradiation or residual tumor at the bronchial margin. In addition to allowing pleural fluid to overflow into the airway, a bronchial-stump air leak allows any airway bacteria to enter the stagnant pleural cavity.

Even a "minor" bronchial-stump air leak soon after pneumonectomy may go on to disruption of the

entire bronchial closure. Therefore, a chest tube is always inserted as soon as air leak is suspected, so that all fluid can be drained from the postpneumonectomy pleural cavity (for a major spill of pleural fluid into the remaining lung could drown the patient). At such times, one especially tries to avoid introduction of bacteria into the "possibly still uninfected" pleural cavity, for surgeons occasionally err, and hopefully that will be when they "fear the worst."

Definite enlargement of the postpneumonectomy apical air pocket (on an upright chest roentgenogram that is comparable to previous films) is considered presumptive evidence of new air leaking in through the closed bronchial stump, if there has been no chest tube present or thoracentesis during this time. Such an X-ray finding may be associated with fever and increasing amounts of bloody expectoration, perhaps five to twenty days after pneumonectomy.

Any patient with suspected bronchial stump air leak must immediately lie upon his operated side, and then remain in that position during transport and evaluation, until tube drainage of his pleural cavity fluid has been completed. Obviously, this "worst-case" type of approach could be over-treatment for a postpneumonectomy patient with minimal hemoptysis, but major fluid spill through a defective bronchial closure into the remaining lung is more easily avoided than tolerated.

In any case, if the initial attempt at chest tube drainage soon leads to markedly subatmospheric intrapleural pressure, with a painful shift of the mediastinum toward the pneumonectomy side, this is good evidence that there was no air leak after all. Provided that tube thoracostomy was performed as a totally sterile procedure, no harm should follow partial or total removal of a postpneumonectomy fluid accumulation (if neither air leak nor infection prove to be present, and intrapleural pressures are adjusted to slightly subatmospheric). Even with no air leak, however, if there is any likelihood of empyema, it is reasonable to leave the chest tube in position until the results of gram stain (and possibly even culture) of the pleural fluid become available; meanwhile one can provide vigorous parenteral and intrapleural antibiotic therapy (see below).

If postpneumonectomy bronchopleural fistula is confirmed (unfortunately the norm in this unusual circumstance), the patient will thereafter require continuous drainage of his pleural cavity to prevent any fluid buildup, as long as air leak and infection persist. Major corrective procedures are generally reserved for the reasonable-risk patient with a good prognosis. Others are treated by a lesser procedure (permanent chest tube drainage, or rib resection and open empyema tube drainage), to allow individuals with limited life expectancy more time outside of the hospital.

If major surgical intervention is selected for a postpneumonectomy empyema with bronchopleural fistula, reamputation and closure of the bronchial stump, or various techniques of living-tissue flap repair (using pleura, pericardium, omentum or intercostal muscle) have been reported as sometimes effective in achieving closure of the bronchopleural fistula. Such techniques can be combined with efforts to eliminate the infected space by thoracoplasty, or to fill it by transfer into the thorax of a bulky chest wall or abdominal muscle on its vascular pedicle. Muscle transfer procedures are allegedly less disfiguring and more effective than thoracoplasty, and they may even eliminate the need for direct attack on the leaking bronchus.

It is evident that any thoracoplasty less extensive than a "radical" Schede thoracoplasty cannot obliterate an empty, infected pleural cavity (especially after right pneumonectomy, where the heart does not occupy any of the residual pleural cavity). Transfer of a large chest wall muscle or two certainly seems more economical than discarding the entire rib-containing portion of one hemithorax during the

Schede procedure.

Postpneumonectomy Empyema

An especially careful and sterile thoracentesis is performed to sample the pleural cavity fluid of any "recent pneumonectomy" patient with unexplained fever: If gram stain reveals bacteria, or culture proves positive, one should then insert a low-lateral chest tube for completion of pleural space evacuation (and plan to initiate irrigations with antibiotic solution). Following pneumonectomy, any thoracentesis or chest tube insertion should be performed with particular care, for the diaphragm is usually elevated (although obscured on chest roentgenogram by overlying pleural fluid), while persistently subatmospheric pleural cavity pressures may also have pulled vital mediastinal structures well over into the "empty" hemithorax that is being penetrated (See Chapter 9).

Clagett Procedure

An infected postpneumonectomy pleural cavity with intact bronchial stump can usually be "cleaned up" by continuous or intermittent irrigation (using one or two chest tubes and appropriately diluted antibiotic or antiseptic solutions) over a period of several weeks. If instilled slowly, such solutions need not be warmed above room temperature (see Chapter 3).

Intermittent drainage with continuous irrigation allows episodic pooling of the antibiotic solution, which may improve cleansing of dependent pockets. Rib resection sometimes provides useful access for removal of thick debris and permits more dependent placement of drainage tubes (just above diaphragm) than could safely be achieved by closed thoracostomy: Allegedly, thoracoscopy can similarly expedite removal of debris and appropriate tube placement.

When the time comes to discontinue the irrigation catheter and drainage tube, the finally sterile, "clean" and well-drained pleural cavity can be filled with a warm antibiotic solution (appropriate for what-ever bacteria were present before the cavity became culture negative). If tube sizes have been progressively decreased as chest drainage diminishes, the small remaining tube site holes will close promptly without significant loss of antibiotic solution: A simple skin suture will also serve.

Care is taken not to overfill the empty pleural cavity, air being removed intermittently or continuously as fluid is added. After the chest has been filled, any tracheal deviation at the sternal notch should still be toward the pneumonectomy side, to avoid compromise of the heart or remaining lung.

Esophagopleural fistula can reportedly be encountered decades after pneumonectomy. Allegedly, the thick pleural peel in such a patient will safely allow daily hydrogen peroxide and povidone-iodine solution irrigations without leading to clinical toxicity by significant iodine absorption. Although I have never encountered this problem, such a fistula reportedly often closes during a prolonged course of these irrigations combined with nasogastric tube feeding: The closed space is then irrigated and left full of antibiotic solution, as described above (see also Chapter 10).

Some modification of Clagett's technique can usually eliminate any postpneumonectomy empyema not associated with bronchopleural fistula. If one should fail to achieve a permanent cure of the pleural space infection on the first attempt, it can always be repeated. I have successfully used an abbreviated version of Clagett's method on an occasional, chronic "pleural space problem" (with underlying lung airtight and maximally expanded by suction, and this residual pleural space already or still quite clean),

but I have not had occasion to try it on an infected postpneumonectomy space.

Although an underlying pulmonary or bronchial stump air leak prevents one from filling the residual pleural space with fluid, regular or continuous irrigation will reportedly sometimes allow a small bronchopleural fistula to heal (while also cleaning up an infected postpneumonectomy cavity). It has been recommended that the patient with such minor air leak remain sitting, or else lie upon the "bad" side, during these antibiotic irrigations, and that continuous pleural drainage be provided.

If spill into the airway through a defective bronchial closure is such a concern, why is this not an equal worry during intrapleural tetracycline instillations that "aim for" inflammatory obliteration of the pleural space? Apparently the irritating tetracycline solution is not inhaled into underlying lung (through the ruptured bleb in the presence of spontaneous pulmonary air leak) because, (1) the volume instilled is not sufficient to submerge the lung apex, where such a spontaneous pulmonary air leak usually originates, and (2) peripheral lung tissue usually leaks air in only one direction anyhow, as leaking lung parenchyma collapses upon reversal of the inside-to-outside pressure gradient. Any "hole" allowing air to escape from a rigid airway, however, will probably permit pleural fluid to flood back into the respiratory tree as well.

A Chest Tube Never Causes Empyema

Despite "theoretically justifiable" concern over minor bacterial contamination of the pleura at the site of tube entry, a chest tube is never withdrawn in fear that it might serve as a source of intrapleural infection, even if the skin entry site becomes inflamed or purulent (unless another location for tube placement would better serve patient comfort or tube function). Handling tubes or thoracenteses so that visceral and parietal pleurae are brought together is the best way to prevent or eliminate pleural space infection: Chest tubes are normally kept in place, therefore, until further accumulation of intrapleural fluid or air is unlikely.

Persistence in substernal tube drainage after resuture of an infected sternotomy is similarly reported to reduce the likelihood of ongoing or recurrent sternal infection. But protracted chest tube drainage is undesirable after pneumonectomy, where a long-term pleural effusion is unavoidable and the residual pleural space should remain sterile. Prophylactic systemic antibiotics presumably increase the bacteriocidal properties of pleural fluid accumulations after pneumonectomy.

Drainage of Empyema Should Be Accomplished Early

Pleural space infection may occur with underlying lung absent, diminished by surgery or disease, or present and basically healthy (as with the common postpneumococcal empyema of the preantibiotic era). However, regardless of underlying lung, any significant collection of purulent material must drain freely if the patient is to regain good health.

When intrapleural pus is too thick for effective needle aspiration or is accumulating rapidly, a chest tube should be placed. Several tubes may be needed simultaneously or sequentially if empyema fluid becomes loculated. In chronic empyema, there is often fibrosis with distortion of normal anatomy. This must be kept in mind to avoid chest tube placement in liver, spleen, aorta, heart or other undesirable location.

Rib Resection

Rib resection was classically utilized for a chronic empyema cavity containing large amounts of necrotic debris, as it allowed optimum cleanout and the most dependent drainage. Long-term open tube drainage was usually easier to maintain and more comfortable if an appropriately located subperiosteal segment of rib had been resected: Chronic rib discomfort was thus avoided and larger chest tubes could be used, or skin sutured permanently to pleura (thereby reducing or even eliminating the need for an indwelling drainage tube). Rib resection was regularly required in the preantibiotic days: It is rarely needed any more.

Care of Empyema Tubes

When a chest tube is placed to drain an empyema, it is never removed merely because drainage slows, or the sometimes associated air-leak ceases. One must leave such empyema tube in place until the pleural space being drained is completely obliterated (until it heals "from the bottom"), for otherwise the predictably swift and spontaneous closure of the chest wall tube site hole by contraction of scar would soon cause the no-longer-drained intrapleural abscess to recur.

There may be minimal up-and-down movement of fluid in the chest tubing or water seal straw during effective drainage of a stiff, immobile empyema cavity, but wide respiratory "tidal" is also seen, especially with a larger or less-rigid empyema space. Any tidal under these circumstances merely confirms that a volume change has occurred in the pleural space during respiration - the pressure change (extent of tidal) will be greater if the ability of scarred diaphragm and chest wall to enlarge the space greatly exceeds the remaining capacity of restricted lung to balloon forth.

Inflammation and fibrosis progressively thicken and stiffen the pleural walls around chronically infected spaces, thereby decreasing the possibilities for rapid pleural approximation and space closure. In the course of draining such a stiff-walled pleural pocket, it used to be common practice to eventually transect the chest tube just outside of the skin, thereby opening the chest tube lumen and pleural space to the atmosphere: Thus shortened, the tube was drained into a frequently changed dressing or bag (that might be more convenient for an ambulatory or working patient than a bulky chest bottle).

A sterile safety pin thrust crosswise through such a shortened chest tube (near its outer tip) helped prevent tube displacement (into the pleural cavity or out of the chest wall). Although an empyema tube could be sewn directly to skin with a loose wire suture, it was more comfortably held in place by suspending the above-mentioned safety pin on a large monofilament loop of suture, which in turn was taped to the chest wall above the drain site (where tape and suture remained cleaner and gave more support to the downward-inclining tube).

The actual length of tube remaining after empyema tube transection was unimportant, as long as one end was definitely within the pleural space to be drained and the other end reached outside of the chest wall. Drainage escaped both through and around such an occasionally changed tube, which provided more-or-less continuous (while dependent) evacuation of the infected pleural pocket.

Antibiotics and Superinfection of the Chronic Empyerna Space

During chronic empyema drainage, antibiotics were given only for the occasional bacterial superinfection that caused systemic toxicity (often Streptococcus). Even effective systemic antibiotics and local irrigations only eliminated specific toxic flora, for it was impossible to sterilize a chronically draining empyema cavity while open drainage persisted.

It Was Important to Make a Trial Disconnection of the Water Seal Before Converting to Open Drainage

One could usually assume adequate pleural stiffening to permit open empyema-tube drainage within ten to fourteen days from the onset of chest tube drainage for major pleural infection. In other words, after about two weeks of trying to get a pleural space closed (or as small as possible), the remaining space usually would not enlarge significantly if the chest tube was opened to the atmosphere.

This presumed stabilization of pleura (by inflammation and fibrosis) was confirmed by a trial disconnection of the water seal. During such trial, the extrathoracic end of the chest tube was loosely wrapped in a sterile sponge, permitting ready atmospheric access to the open chest tube and underlying pleural pocket.

Significant "tidal" while connected to water seal, or even a persistent large bronchopleural air leak, did not deter a trial of tube amputation and open drainage, provided the patient experienced no untoward symptoms due to excessive pleural mobility and competitive air flow in and out of the chest tube, or signs such as additional separation of visceral and parietal surfaces on chest roentgenogram: (That is, repeat roentgenograms should not show increase in size or extent of any intrapleural air pocket as a consequence of ready atmospheric entry into the chest tube.) If the patient did develop symptoms, or lung fell away from the chest wall elsewhere than at the residual pocket, the chest tube was simply returned to water seal or chest bottle suction for a few more days.

Do Not Discontinue Suction Too Soon

If "high" suction is applied continuously over a period of weeks, it can generally approximate the visceral and parietal walls of even the most stiff-walled pleural pocket, or at least bring its size down to a minimum. This high suction will be equally effective whether it is applied by a 60 mm Hg setting of a suction gauge or (when there is no pulmonary air leak) by withdrawing air from the upper chest tubing to lift a hanging water seal fluid column to an equivalent height. A total of two or three weeks at this "high" suction setting may be necessary to stabilize any residual pleural pocket into its new, maximally shrunken, condition.

Even when high suction is only instituted late, after weeks of water seal or "low" suction chest tube drainage, one can often markedly reduce or even obliterate a troublesome pleural space within another two or three weeks. Once a chronically infected pocket has been "firmed up" (by high suction) to its new, minimal size, one can discharge the patient with his chest tube still connected to a modern, lightweight water seal bottle (or else amputate the chest tube for open drainage) while awaiting completion of pleural space obliteration by fibrosis and granulation tissue (as discussed below).

A Reminder: Pleural Space Obliteration is the Goal

A temporary pleural space between lung and chest wall is not uncommon after spontaneous pneumothorax, accidental chest injury or chest surgery. Such pleural space may primarily contain air, fluid or other material; it could be symptomatic or asymptomatic, as well as minor, moderate or major in size, and sterile, contaminated or even grossly infected. In addition, the space may be associated with or due to normal, abnormal or necrotic tissues, which may or may not be leaking air or fluids. Clearly, a single, routine response to all pleural space problems could be inadequate, excessive, or simply inappropriate.

An asymptomatic, minor, sterile, pleural air or fluid pocket not associated with ongoing air or fluid accumulation is unlikely to need surgical attention (it is not a space "problem"). In contrast, to prevent significant infection, a moderate-sized postoperative pleural space that has been contaminated by bacteria spilled at surgery, is best evacuated early and efficiently (although this space too can sometimes resolve without intervention of any sort). It is by early, vigorous and appropriate treatment of the grossly infected pleural space (with or without underlying diseased or necrotic tissue needing simultaneous therapy), that the chest surgeon makes an essential contribution to the welfare of his patient.

Comment: Skillful pleural space obliteration, perhaps the essence of thoracic surgery, is commonly required to restore good health.

Tube Amputation Versus the Outpatient Chest Bottle

If an unavoidable space persists between visceral and parietal pleurae, it is best kept as small as possible. Stabilization of the gradually stiffening pleural walls of such a space, using "high" chest tube suction, is therefore a prominent part of initial therapy. Once the space has been held to its minimum size until it becomes "stuck" in that position, further treatment will depend upon the size and shape of this space, presence of infection, condition of underlying tissues and any ongoing fluid or air accumulations.

Any ongoing air or fluid accumulation within a significant pleural space must be vented out of the chest by appropriate drainage (to prevent progressive distension of this space by high pressure, possibly toxic, accumulations) until the space has finally been obliterated by scarring. When a clean-walled, uninfected space is not associated with ongoing air or fluid accumulation, it can eventually be filled with antibiotic solution and external drainage then discontinued (as described by Clagett): Hopefully, the shrinkage of such a space will thereafter progress by fluid resorption, without further need for tube or needle drainage.

Outpatient follow-up during continued simple "water seal" chest tube drainage (in preference to an amputated empyema tube), permits persistent application of chest bottle suction by the hanging column of water (if there is no air leak - see Chapter 8), while such "closed" drainage also decreases the risk of pleural space superinfection. The modern-day patient whose drainage problem is likely to resolve within a few weeks is probably better served by continued chest tube drainage into a chest bottle (in his home or at work) than by cut-off empyema tubes protruding from his chest wall. Either method will require on-going tube-site dressing changes until the tube is removed, but empyema tube dressings must often be changed several times a day, while chest tube dressings only occasionally require renewal.

Empyema Tube Removal

Granulation tissue and fibrosis will gradually obliterate the average persistent air leak or residual pleural space; this healing process will often force a still-remaining empyema (or chest) tube slowly out through the chest wall. To avoid leaving a potential abscess space behind, however, healing must progress from the depths of the pleural space, so an empyema tube must sometimes remain in place for months or even years.

When there is no pulmonary air leak, a contrast study (injecting water-soluble, radiopaque dye via

the chest tube) can sometimes aid roentgenographic appraisal of the residual pleural pocket (computed tomography without dye is most likely even more helpful, but I have had no experience with that). If it is clear that the empyema cavity is shrinking well around the empyema tube tip, the tube may properly be pulled out 1 to 2 cm, trimmed and resecured. Thus assisted, continued healing will press the empyema tube out of the chest over ensuing months, as the problem resolves.

Bronchopleural Fistula:

Lung resection or other injury will occasionally be followed by persistent pulmonary air leak ("bronchopleural fistula"). It is well to be patient when dealing with persistent air leak; even minor leaks may seal slowly on occasion. As a rule, a chest tube cannot be removed until air leak ceases: However, if the patient is otherwise well, home care of the chest bottle, or amputated tube (after pleural stability has been demonstrated), are both effective methods for handling the air leak.

A bronchopleurocutaneous fistula is, of course, an unprotected opening into the respiratory tract: Fistula submersion while bathing or swimming could therefore cause drowning. Similarly, explosive cough, gasping and wheezing often results from even small instillations of diluted, water-soluble, radiographic contrast material into a residual pleural pocket in the presence of air leak. Therefore this study is only requested if planning a several cm. withdrawal of the tube (to encourage obliteration by fibrosis and granulation tissue of a hopefully "long and skinny" bronchopleurocutaneous fistula).

Any contrast evaluation of a pleural pocket is performed solely to prevent accidental withdrawal of the tube tip from a large residual cavity during elective tube shortening, or to determine if the tube is being extruded prematurely. It is likely that computed tomography has eliminated the need for such pleural space dye instillation, especially during air leak from underlying lung (but empyema is now so rare that I cannot ascertain this from my own experience).

On occasion, despite all precautions, a residual pleural pocket will lose continuity with any drainage tube: Despite this, effective drainage may continue, but now in a retrograde fashion, back through some bronchopleural fistula into the lung (from whence it emerges as noticeably distasteful expectoration). Such a fortunate outcome is often followed by successful obliteration of the pleural space. This is more likely to occur when adjacent lung parenchyma has been resected or otherwise damaged; one assumes that nearby infection and local lung necrosis make available some small open bronchus as a "low-volume" (large enough to empty the pocket but small enough to prevent sudden overflow and drowning) passageway for chest drainage.

Relatively intact peripheral lung parenchyma apparently cannot drain small empyema pockets in this fashion, due to obligatory one-way outward flow at any site of air leak from a ruptured bleb. To what extent infection can drain via lung is not clear, but lung will sometimes drain a surprisingly large pleural collection, especially from the more inaccessible upper reaches of the chest (here any open airway would also provide dependent drainage). If a bronchopleural fistula allows unobstructed, bidirectional, air or fluid passage, it can prevent enlargement of any stable pleural space by entrapped fluid or air under increased pressure: When thus stabilized, an appropriately-shaped space will usually be obliterated by progressive fibrosis (see also decortication discussion below).

Final cessation of a chronic air leak is most readily detected if a patient has remained on water seal drainage: Tube advancement or complete tube removal is then appropriate, provided that the only re-maining purpose for the drainage tube was to vent the no-longer-open bronchopleural fistula to the

atmosphere.

As has been mentioned, any grossly infected intrapleural cavity or space that persists after cessation of air leak, must be handled as an empyema space; here "grossly infected" implies systemic signs and symptoms of infection plus purulent drainage. If such residual space has remained or becomes "clean," however, (with no systemic symptoms and minimal nonpurulent drainage), the cavity can be irrigated to an optimal state and then left filled with dilute antibiotic solution, prior to final tube withdrawal.

A Bad Shape May Lead Chronic Empyema to Decortication

When not adequately drained, any empyema will tend to enlarge and produce systemic toxicity, while also stimulating pleural inflammation and fibrosis. If the patient survives, such an empyema may eventually become evident as a subcutaneous bulge, or even drain spontaneously (an "empyema necessitans").

Pleural surfaces around an appropriately drained empyema cavity will continue to contract and thicken until they choke off their own blood supply or grow up against other living tissue. Even after ribs have become maximally drawn together and immobilized by underlying scar tissue, rib cage (parietal) pleura may still continue to thicken (sometimes to several centimeters in depth) in response to the irritating contents of a subjacent pleural space. Final obliteration of such a cavity seems to stop further formation of restrictive scar: In fact, the thickness of pleural scar displayed on chest roentgenogram may thereafter gradually diminish.

We usually credit the "Wisdom of Nature" for the progressive contraction and obliteration of abscess cavities after drainage. Is it her "Stupidity" then, when an infected space surrounded by contracting scar tissue has such a complex shape that scar shrinkage paradoxically enlarges and perpetuates the empyema cavity?

Unfortunately, an ordinary empyema involving the entire pleural cavity has just such a "worst possible shape": What might, under other circumstances, have become "helpful scar contraction" for obliteration of an abscess cavity, rapidly becomes "harmful progressive constriction" when a fibrotic process surrounds lungs (or heart). Time and scar contraction then help the enemy to persist, for the longer that fibrosis proceeds unchecked, the greater the disparity in size between progressively constricted lung and its surrounding, non-collapsible hemithorax.

Early drainage of empyema is always important for elimination of toxic intrapleural material. With total pleural cavity empyema, however, the scarring process and chest tube suction become directly opposed; application of "high" chest bottle suction (if there is air leak, or else a 60 cm. tall fluid column in the chest tubing) is now essential, in order to draw the progressively restricted or entrapped lung up against the increasingly-immobile and rigid rib cage "before it is too late."

Such forceful suction is usually sufficient to complete the evacuation of noxious cavity contents and achieve full lung expansion: Here, the hoped for "best possible result" is a lung with somewhat diminished freedom to expand but no pleural space and thus no pleural infection. However, when days and weeks of "high" chest tube suction not only fail to bring lung and diaphragm together, but also cannot reexpand lung into the apex of the chest, it becomes increasingly apparent that "something more" will be required if obliteration of this infected pleural space is to be achieved.

When scarring is the enemy, and the enemy appears to be winning, we either change our tactics or our goals: A bloody surgical decortication can be carried out, if warranted by the patient's condition and prognosis; we can perform a Clagett procedure and "hope for the best" (see elsewhere in this chapter); or we can simply claim a "moral victory" and declare an end to our fight against fibrosis with suction: We must then admit that these new pleural boundaries are "permanent," and perform a "humbling" rib resection and long-term open drainage, recognizing that there may well be additional losses of lung function to the overwhelming forces of fibrosis.

Interestingly, an intermediate "no win - no lose" position can occasionally be sustained with a major empyema of this sort, provided the battle between scarring and suction have "come to a draw," with apex and diaphragm stuck to lung: At that point, any additional fibrosis and scar contraction along the lateral lung surface may have little further effect on chest wall, lung size, diaphragm movement or empyema cavity itself.

If this stable intermediate position is associated with ongoing air leak, tube drainage will still be required: However, without air leak (or persistent underlying lung infection or necrosis), the large residual space can be treated as an infected postpneumonectomy space (cleaned by irrigation along with local and systemic antibiotics, and then filled with an appropriate antibiotic solution in "modified Clagett" fashion).

Case Report: A Spontaneous "No Win - No Lose" Fibrothorax

I can recall one patient whose scarring process became stuck in this fashion between "forward" around an empyema cavity and "backward" around the lung. This resulted in a flat (fortunately sterile) cavity that stretched from top to bottom of his hemithorax: The tight, thick scar over his chest wall paralleled the tight thick scar over his lung. The cavity contained pearly, birefringent, high-cholesterol fluid, sterile on culture (probably an old tuberculous effusion, according to what I read).

I could not "close" this cavity with high suction, and the patient was "too busy" to undergo decortication: With my tube removed, his situation remained stable (and cavity sterile) for almost a decade thereafter. Eventually, his underlying lung disease reinfected the old empyema space; he then underwent decortication elsewhere, and probably should have had pulmonary resection along with his decortication, for he subsequently developed many of the complications discussed below.

Decortication, With or Without Lung Resection

To improve lung function and shorten the duration of illness, an occasional large chronic empyema cavity can be considered for surgical decortication (which means stripping away diseased, restrictive pleural surfaces, along with evacuation of remaining debris from the empyema cavity). When such a chronically infected pleural space is consequent to persistent underlying pulmonary disease, the problem becomes more complex.

To reduce the risk of catastrophic failure of a decortication procedure, any suspected lung disease must be evaluated by preoperative bronchogram. If lung abnormality, such as saccular bronchiectasis, is detected, it is often best to combine decortication with definitive resection of all diseased portions of lung.

Decortication plus lung resection can bring about a dramatic cure of the severely ill empyema

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patient, but this is feasible only if one has effective antibiotic coverage (to prevent spread of infection over newly-created raw surfaces). During "decortication resection" the empyema is evacuated, lung and chest wall are decorticated, and diseased pulmonary tissue is resected, as indicated: Especially vigorous and devoted postoperative management of the two or more large-bore chest tubes is then essential, to promptly eliminate any residual pleural space between remaining lung and chest wall.

Needless to say, decortication in the face of established empyema, with or without pulmonary resection, is major surgery, causing significant air leak and bleeding: Systemic antibiotics are used freely. This situation is a substantial test of one's abilities in thoracic surgery and chest tube management. When successful, as such surgery must usually be, the course of this serious (now increasingly rare) disease is dramatically improved, and pulmonary function also benefits.

However, there is a strong tendency for these patients to develop postoperative clotted hemothorax. Clotted hemothorax, superimposed upon "already activated" pleural fibrosis (combined with infection and maximal surgical stress), can easily create a clinical problem worse than that for which surgery was originally performed; so place plenty of large tubes and make sure that they work perfectly, especially through the initial postoperative day and night (when some blood loss is unavoidable, and clotting within both chest and tubes appears to develop "instantaneously."

Pleurectomy

Thin, normal pleura can usually be stripped from the inner chest wall without excessive blood loss, but mediastinal and diaphragmatic parietal pleura should be "left alone" or merely "irritated" if pleural space obliteration is required. For flimsy normal visceral pleura is inseparable from peripheral lung tissue: Attempted removal usually produces only small pleural fragments with underlying lung surfaces bleeding and frothing from inadvertent parenchymal tears.

Decortication

Abnormally thickened pleura over lung (or chest wall) can usually be stripped away rapidly and effectively, once a thick pleural edge has been elevated: Finger(s) are inserted under this edge to keep enlarging the subpleural pocket by a back-and-forth sweeping movement, with the finger rolling over lung (and thus rotating against pleura) to minimize laceration into lung parenchyma. Such burrowing, blunt dissection tends to proceed in the proper (least vascular) plane: On the chest wall, this plane is extrapleural; on lung it is apparently just outside of the original, thin, visceral pleural layer. To prevent needless parenchymal injury, unusually adherent patches of thickened visceral pleura are usually left in place on the lung surface.

Open the Fissures During Decortication

Virgin visceral pleura can often be exposed by dividing a few adhesions and sweeping the finger through any well-developed (and usually not fibrotic) interlobar fissure. When thus released, normal in-terlobar pleura usually balloons out sufficiently to compensate for scattered lung surfaces still restricted by scar after decortication.

Lung Surface is Often Torn During Decortication

The less experienced or more aggressive surgeon, especially if dealing with emphysematous lung,

will occasionally complete his removal of thick restrictive visceral pleura, only to be faced by badly torn lung surfaces frothing and bleeding "all over the place." At this juncture, any attempt to suture major air leaks just tears lung (as intact visceral pleura provides the main "grip" for a suture line in peripheral lung). And while the anesthesiologist complains about huge air flows needed to inflate the newly leaky lung, the surgical team shares whatever inhalation anesthetic is being used.

Fortunately, significant blood loss from ragged lung surfaces usually stops promptly if a dry sponge or lap pad is placed firmly against torn lung for five or six minutes. Still bleeding vessels are then secured and major sources of air leak are ligated since ligatures tend to "hold" better than sutures when visceral pleura is absent (their circumferential stress being more evenly distributed over weak tissues). Staples may be helpful on occasion. In any case, torn bloody lung heals surprisingly well as long as healing is supported by competent chest drainage.

Chest tubes require especially careful placement when overall blood loss is great and there is significant air leak. They must also receive compulsive postoperative attention under these circumstances if they are to remain patent and allow continuous easy egress for the high-volume air leak from lung (see Chapter 5). Clotted hemothorax, tension pneumothorax, massive subcutaneous emphysema and empyema all lurk nearby in this situation, and those chest tubes are your only defense.

Mobilizing the Lung at Thoracotomy

When visceral and parietal pleurae have fused consequent to previous pleuritic inflammation and fibrosis, lung will probably be torn inadvertently as its lower chest wall and diaphragmatic surfaces are freed, for access to these areas from a fifth or sixth interspace incision can be awkward. In such circumstances, one might as well rip lower lobe surfaces free of chest wall and diaphragm as quickly and carefully as possible, then cover raw surfaces with a dry lap pad to encourage sealing of air leaks and reduce blood loss, while the intended operation moves ahead. On those occasions when fingers preferentially "poke into" lung parenchyma rather than dissect along appropriate pleural planes, the side of a tightly rolled sponge can be swept firmly forward into the adhesions while rolling along the lung surface (thus rotating against adherent parietal pleura), to achieve more rapid, less-traumatic mobilization of lung from chest wall.

"Thoracotomy proceeds best after the entire lung surface has been freed." While this statement is generally true, common sense should prevail. For example, minor pleural biopsy or wedge resection of lung will rarely require or justify major dissection. Nevertheless, there are five closely related reasons for freeing the entire lung surface of adhesions during major intrapleural surgery. These are safety, surgical access, postoperative bleeding, postoperative lung expansion and effective chest drainage.

For safety, it is important to free the entire lung early in the surgical procedure. If unexpected bleeding, cardiac arrest, or other emergency should then arise, lung can simply be pushed aside without wasting valuable time on dissection to achieve adequate access or exposure. It can be very difficult to regain control of a torn pulmonary artery with lung still densely adherent to chest wall, mediastinum and diaphragm. Indeed, a pulmonary artery tear will likely extend during manipulations required for its control and repair unless lung is carefully relaxed upward to take all tension off the damaged vessel. Suture repair of a pulmonary artery under tension is essentially impossible, for it remains fragile until the suture line is complete, and merely flimsy thereafter.

Case Report: Torn Pulmonary Artery

The rough and rather inexperienced surgeon initially pulled down sharply on the left lung, thereby "uncorking" the left pulmonary artery (by ripping an adherent, calcified, peribronchial node out of the pulmonary artery wall). Impressed by the vigorous hemorrhage, he repeatedly applied pressure to the bleeding site with large lap pads, while he pulled himself together and the artery further apart (for the lower lobe was still densely adherent to the diaphragm). Unable to visualize and correct the damage, he issued another call for help.

On arrival, the consultant immediately controlled bleeding by gently compressing the torn vessel between his fingers. He then prevented further traction on the artery by supporting nearby lung, as another surgeon mobilized the lower lobe from chest wall and diaphragm (these adhesions had been neglected in the rush to "beat the clock"). With lung no longer stretched, it became easy to free up the proximal pulmonary artery. Good exposure, plus proximal and distal control, allowed successful repair of this not unusual (but exceedingly dangerous) pulmonary artery injury.

Mobilized Lung Reduces Postoperative Complications

Another advantage of completing lung mobilization at the start is the greater opportunity thus provided for intraoperative cessation of minor bleeding. The few small vessels still bleeding just prior to chest closure can then be given extra attention. A surgically diminished lung will also expand far more readily into the apex if it is totally freed of all attachments to the lower chest wall, diaphragm and mediastinum (including division of the inferior pulmonary ligament).

In addition, after right upper lobectomy, if middle and lower lobes are not separated, they tend to move upward en masse; this creates a hard-to-fill apical pleural space above the now horizontal, upper lung surface. Such a persistent apical pleural space can prolong the duration of postoperative pulmonary air leak and chest tube drainage, thereby allowing more time for fibrous restriction to develop about the remaining lung.

When lung cannot be drawn up into the apex, there is also increased risk of postoperative empyema. Once the flat upper surface of the middle lobe ascends past the chest tube tip, it will separate the chest tube from the remaining apical pneumothorax: A perfectly placed chest tube and well-mobilized middle lobe reduce the likelihood of this problem.

When diaphragm and lung are not "stuck" to each other, they move upwards more readily: Residual lobes can then swing toward the apex on their bronchovascular connections to the mediastinum, while the central diaphragm moves straight up like a piston. As long as the chest tube remains within the apical pleural space, however, continuous high suction can help overcome a great deal of lung immobility or non-conformity to the shape of the thoracic apex (see Chapter 8).

If the pleural cavity is freed of restrictive adhesions at thoracotomy, there is less opportunity for isolated pockets of air and fluid to develop, and chest tubes can be placed in the most advantageous location. Intrapleural clot accumulation is diminished when freshly shed fluid blood has ready access to properly positioned chest tubes.

Caution: An occasional shrunken, bronchiectatic, lower lobe is far better left undisturbed (except just prior to its planned resection), as any mobilization of such a lobe can cause excessive bleeding from large, high pressure, bronchial artery collaterals, or lead to pleural space contamination from easily torn, chronically infected air pas-sages. In this circumstance, the simple desire to improve lung

mobility could create major ongoing difficulties for the surgeon and his patient (such as empyema, decortication-resection, permanent open drainage, muscle transfer, thoracoplasty or all of the above).

In other words, not every adherent lower lobe requires mobilization, just most of them. Although severe bronchiectasis is an increasingly rare disease, "forewarned is forearmed": A preoperative bronchogram during evaluation of "possibly inflammatory" lung disease can occasionally prevent unnecessary surgery, or reward you with a grateful postoperative patient instead of an albatross hung from your neck.

There are times when mobilization of a very adherent lower lobe will leave small lung fragments adherent to diaphragm or inner chest wall. If easy to remove, one may wish to "tidy up" and pull these "lung bits" off the chest wall, but there appears to be no ill effect from leaving such tissue scattered about. However, during resection for bronchiectasis, it may be better to remove possibly infected residual bits of lung tissue. Undoubtedly, bronchiectasis is a vanishing disease, but consider when you last saw excision biopsy of a rather vague coin lesion that turned out to be bronchiectasis or some other inflammatory lesion. Not only would a preoperative bronchogram most likely have shown the lesion to be inflammatory (with surgery therefore not indicated), but the surgeon now has the worry that his biopsy closure may break down and eventuate in empyema. At the very least, this may lead to additional unnecessary hospitalization while a prolonged course of antibiotics is given "just to be safe" (see Chapter 13).

Adhesions in Perspective

The normal pleural cavity is free of adhesions between visceral and parietal pleurae. Not uncommonly, however, the problem leading to this thoracotomy (or some prior, possibly unrelated, pleuritic inflammation) has resulted in adherence between opposing pleural surfaces: Sometimes the visceral and parietal pleurae can readily be separated by gentle traction or dissection with the exploring hand, at other times this plane will be found to be more or less obliterated. Rarely the two pleural surfaces will have become so severely fused by fibrosis that their separation requires sharp or even extrapleural dissection which can be most taxing (see also later this chapter).

Congenital Fusion or Absence of Pleura

Congenital absence or fusion of pleurae is seemingly not encountered by surgeons. This suggests that free pleural layers are essential for lung bud development or successful pulmonary expansion at birth.

Apical Pleurectomy for Spontaneous Pneumothorax

Some surgeons routinely combine apical parietal pleurectomy with wedge resection for leaking upper-lung blebs. However, this pleurectomy increases blood loss, prolongs surgery, and extends the duration of postoperative chest drainage, thereby increasing the length, risk and cost of hospitalization. I prefer brisk, dry sponge abrasion of the pleura, hoping thereby to promote pleural thickening and symphysis.

Pleural Irritation to Promote Symphysis

Pleural thickening is readily caused by routine surgical manipulation. At several reoperations for

bleeding, I have been amazed to find the mediastinal anatomy obscured by pleural edema and swelling, just hours after the initial operation (pleural flaps over bronchial stumps were particularly swollen, which could explain their apparent utility in buttressing the closure of a large bronchus).

In our ongoing efforts to assure firm fusion of visceral to parietal pleura, we have utilized many methods of pleural irritation, ranging from talc poudrage to chemical burn. Although we know it can be done (because we run into such fusion regularly in our work), it is not always easy to achieve reliable pleural fusion "on demand." With an occasional patient, in whom even gentle abrasion by dry sponges leads to pleural "rips" and undesirable bleeding, I have simply applied uncomfortably warm saline solution to inflame the fragile pleura (instead of further sponge abrasion).

Having seen only one recurrent spontaneous pneumothorax after any form of direct surgical attack (and that was loculated, in a patient with very severe bullous emphysema), it is clearly impossible to demonstrate superior results for any particular approach. If results are comparable, however, one must assume that the lesser procedure or "violation" of anatomic planes is usually better, in which case abrasion clearly "beats" apical pleurectomy.

Perhaps even pleural abrasion is meddlesome and excessive; simple wedge resection of a leaking bleb has long been alleged to be equally effective. At least abrasion is simple and rapid, with the extra-pleural plane still intact if another thoracotomy should ever be required.

Extrapleural Dissection for Surgical Access or En Masse Resection

When visceral pleura has been "welded" to parietal pleura by vigorous fibrosis, one sometimes cannot reopen the pleural space: "Sharp" dissection under these circumstances may become so tedious and bloody that it is both impractical and unsafe. If routine intercostal entry to the thorax is blocked by such pleural fusion, one must mobilize underlying lung in another plane.

Unlike repeat sternotomy, where vital structures can be closely adherent to a relatively small area on the back of the breastbone (from which they must be separated with great care), peripheral lung parenchyma can usually "take a lot of surgical abuse" during mobilization without altering the patient's prognosis. Which is fortunate, because when faced with a firmly stuck lung surface, one must often rip lung forcibly from chest wall in order to achieve chest entry within a reasonable period of time.

The extrapleural plane usually turns out to be quite adequate for this purpose, with thick pleura bluntly separable from rigid ribs (which can require considerable force) along a relatively avascular, relatively easily maintained plane. However, the apex, diaphragm and mediastinum are three "soft" areas where continued forceful blunt dissection can easily carry one into or around crucial structures that one had intended to pass over.

In forcing a passage along the extrapleural plane, between bone and heavy scar, I have several times unknowingly mobilized aorta, esophagus or neurovascular structures in the apex (once even the heart, while removing a bulky malignancy), fortunately without harm: There is no doubt that important structures can be "out of place" - or that the surgeon may not always be working exactly where he thought he was.

Intact pleura (or peritoneum) sometimes serves as a barrier to some malignancies, which may continue to grow and seed locally and eventually kill the patient without ever spreading outside of their

pleural container. Such a "corral" effect is allegedly common with intrathoracic mesotheliomas; we have encountered it with bronchial adenoma and one massive neurofibrosarcoma. In contrast, most intrapleural malignancies (typically lung carcinoma), if undeterred, will spread out of the chest directly and/or by metastasis. It might be important to learn why mesothelioma seems restricted in this fashion.

Direct extension of an underlying lung cancer into the chest wall has long been considered a sign of surgical incurability except (reportedly) in the apex, following preoperative irradiation for Pancoast tumor by the method of Paulsen and Urschel. This brings up an important point: The initial operation often (usually? always?) represents a patient's very last chance for "surgical cure" of a malignancy, as incomplete extirpation of any malignant lesion that is not curable by chemotherapy and/or radiation, generally enables that cancer to spread. Thus routine surgical exploration "to determine operability of a tumor" generally satisfies diagnostic curiosity: However, if appropriate complete removal is not then properly achieved, it also eliminates a patient's "last best chance".

For example, forceful blunt separation of a pulmonary malignancy involving parietal pleura from directly overlying ribs is often quite easy, but a local or distant recurrence soon thereafter suggests that this was not a useful operation (perhaps due to "too little margin" and "too much manipulation"). Not unexpectedly, prognosis worsens when intrapleural cytologic washings reveal malignant cells at the time of surgery: Which is consistent with the known dismal prognosis of malignant pleural effusion, and a hint that incisional biopsies may not be as "recurrence-risk-free" as proponents currently allege.

If preoperative irradiation has not been given, and if the presumed malignancy is closely approximated to pleura on chest roentgenogram or computed tomography, it would seem possible to improve the prognosis by gentle palpation to locate the lesion from within the chest, followed by resection of whatever sections of rib overlie its (hopefully undisturbed) pleural attachments. To limit further manipulation, such a peripheral tumor can then be easily "wedged" out of the lung (see Chapter 13).

While I cannot prove that this suggestion will improve outcomes, one surely won't worsen the prognosis of a malignancy by not breaking into tumor (especially during dissection in the extrapleural plane where that event too often occurs). Furthermore, chest wall resection from inside the rib cage is surprisingly easy. And if the defect resulting from such a rib resection underlies very thick chest wall muscle or scapula, a mesh prosthesis may be unnecessary (see Chapter 2).

Is it possible that the unusually good results reported for formal resection of Pancoast tumors following irradiation may be a consequence of the apparently dismal outlook of patients with chest wall and brachial plexus invasion? Consider the following "psychological" aspects of surgery: 1) A surgeon expecting to do a routine lobectomy is more liable to do a routine lobectomy, even when this results in "poor margins of resection": 2) The surgeon who goes into a chest following irradiation of a presumed or proven malignancy, is better prepared for a major resection - the operation has been appropriately scheduled, he is already committed to the diagnosis of "cancer," aware of the need for wide resection, and his patient understands that the preoperative disability may be worsened by surgery: 3) Also the "occasional" chest surgeon is more likely to "refer" this case to someone more qualified, because of that immediate postoperative morbidity - which is still preferable to dying of metastases "a year down the road" (but such a late death is harder to blame on malignant tumor being inappropriately torn away from unresected chest wall): And 4) Preoperative irradiation may further decrease the risk of tumor seeding during resection.

Extrapleural pneumonectomy is sometimes advisable for removal of nearby bulky tumor, or of advanced suppurative disease and destroyed lung (to decrease the chance of tumor or bacterial spill). Extrapleural dissection often is carried around rib surfaces of a lobe to be resected for similar reasons; the pleural envelope is then entered over an uninvolved part of the lung where it is not adherent. Portions of nearby non-vital structures (such as diaphragm and pericardium) that are involved by expanding malignancy can be removed as indicated. Reportedly, one can also leave thickened parietal pleura in place and perform pneumonectomy right through an empyema, with a reasonable likelihood that local and systemic antibiotics and other irrigations will allow delayed but primary healing.

Case Report: Extrapleural Pneumonectomy for Primary Osteogenic Sarcoma

This healthy 23 year-old white female entered the hospital ten years ago for evaluation of a recently discovered, roentgenographic opacification of the left lung field. She reported cough and weight loss of six months duration, with recent minor hemoptysis.

On exploration, a bulky malignancy was encountered that required extrapleural mobilization, with intrapericardial ligation of pulmonary veins, sharp dissection of a calcified ligamentum arteriosus from the aortic wall, and transection and closure of the left main bronchus at the carina. A bony-hard mass filled the left pulmonary artery and extended into the main pulmonary artery.

Left pulmonary artery transection required a heavy, double-action, bone cutter: The open-ended main pulmonary artery did not bleed; in fact, it did not have a lumen, and resembled normal tibia in all respects, including a central bone marrow (both grossly and microscopically). Soft tumor masses connected to this "lung bone" (which appeared to originate at the calcified ductus) extended into and almost blocked the main and right pulmonary arteries as well: This explained why her heart had dilated and arrested (requiring vigorous cardiac massage several times) during mobilization of the hemithorax-sized malignancy.

The remaining main-pulmonary-artery-bone and soft intravascular tumor was removed through an extension of the incision across the left pulmonary artery origin onto the main pulmonary artery: The entire mass was mobilized and extracted, apparently intact with its many clumps of soft adherent malignancy. The vascular clamp was then hastily applied tangentially to exclude the incision and control blood loss from the main pulmonary artery (while control of cut artery edges was maintained with multiple fine traction sutures).

At this point, the patient developed right heart outlet obstruction and cardiac arrest that responded only to very forceful cardiac compression: Presumably this represented either an obstructing intimal flap, a dislodged blood clot (that had formed locally), or a tumor embolus. In any case, the obstruction was broken up and forced distally or displaced by that last successful intraoperative resuscitation and she did well thereafter.

Ten days postoperatively she was discharged to complete a course of Adriamycin chemotherapy. Following a year of good health, she developed generalized metastases and died less than two years following pneumonectomy.

Conclusions: 1) Extrapleural dissection can aid in the removal of bulky tumors.   2) This young female had tolerated subtotal blockage of her main and right pulmonary arteries, in addition to total obstruction of her left main pulmonary artery. Had we recognized that her right heart outlet was

severely compromised, we might have resorted to cardiopulmonary bypass during resection (but that could have increased postoperative blood loss due to our extrapleural dissection, and also disseminated malignant cells).

Comment: In 1963 I was shown roentgenograms of a solitary osteogenic sarcoma metastasis to lung that spontaneously matured into radiographically normal bone and had remained so for at least two decades. But even though the proximal intravascular portion of my own patient's primary pulmonary osteogenic sarcoma had matured into normal appearing bone, the major bulk of its massive intrapulmonary extension was "poorly-to-well-differentiated osteogenic sarcoma."

Role of Pleural Thickening in the Prevention of Recurrent Spontaneous Pneumothorax

Predisposition to spontaneous pneumothorax may be congenital or acquired. The twenty-year-old with normal appearing or slightly macroalveolar lungs and one or more thin-walled blebs at the apex (or with multiple protruding blebs along the sharp upper or anterior lobe edges), probably has a different condition from the fifty-year-old chronic smoker, leaking air from a leathery, bullous apex (although both conditions apparently can be worsened by smoking).

Why simple resection of the leaking area (or merely oversewing the most obvious blebs) is so effective in preventing recurrence of pneumothorax is a mystery. Perhaps a second mystery... "Why are there so few spontaneous pneumothoraces with so many emphysematous lungs out there?"... can help explain the first. One must presume that almost any direct surgical attack on the leak site will sufficiently alter a rare (air-leak prone) bronchus-bleb configuration, so that the likelihood of recurrent air leak becomes minimal.

Instillations to Achieve Pleural Space Obliteration Following Spontaneous Pneumothorax

Pleural thickening or fibrosis (due to surgical abrasion of visceral pleura) may help prevent recurrent pneumothorax on the operated side, but oversewing or wedge removal of a specifically-abnormal leaking site is probably of more basic importance. The unaltered persistence of an abnormal, leak-prone area of lung may account for past failures to chemically obliterate a pleural space without thoracotomy (by introduction of various irritating substances into the pleural space through needle or tube). In addition, continued air leak can make it difficult or impossible to keep the lung properly positioned while the interpleural obliterative process is under way.

Applications of talc powder are known to cause chronic pleural granulomas and progressive fibrous restriction of the lung. Thoracotomy after talc poudrage is apparently a major undertaking as-sociated with much bleeding and air leak (due to fibrous destruction of intrapleural and extrapleural tissue planes). In addition, some talc allegedly includes enough asbestos to be a risk factor for later development of malignancy (an increased likelihood of ovarian carcinoma has also been reported in those exposed to repeated talc dusting of the perineum).

Despite the above logic and past experience, however, I am increasingly encouraged by the results of intrapleural tetracycline instillation for air leak after spontaneous pneumothorax (although I remain unsure about when to recommend tetracycline in preference to thoracotomy). Especially for the younger patient with major recurrent or persistent spontaneous pneumothorax, surgery may still be the more reasonable option (at least until more information becomes available on the recurrence rate of spontaneous pneumothorax after tetracycline instillation). It is not likely that any instillations can

substitute for direct closure when major air leaks prevent lung expansion (see Chapter 5).

Tetracycline Instillation During Persistent, Minor to Moderate Air Leak

On a first attempt, I usually instill one-half gram of "intravenous" tetracycline dissolved in 50cc of sterile saline. To avoid tension pneumothorax, I do not clamp the chest tube (especially if air leak still persists): While on water seal chest drainage for air leak, lung usually falls away from the chest wall if the patient breathes very quietly. There should be minimal early loss of instilled tetracycline solution out the open chest tube if this tube is pinched or kinked momentarily during any cough or forceful expiration (or while the patient is rolled and tilted to ensure widespread intrapleural drug distribution).

Although I am unsure how best to apply tetracycline, or even how long to leave it in the chest, air leak seems more likely to cease if full expansion of lung and complete pleural apposition can be achieved soon after the 15-60 minute treatment (when instilled fluid and any residual free air are again encouraged to escape by vigorous cough or chest bottle suction). In addition, tetracycline seems to have more impact when post-instillation fluid drainage is grey and turbid, rather than when the returning fluid is an unchanged clear yellow (but that, too, is a very preliminary observation).

I have instilled up to two (2) grams tetracycline dissolved in 100cc saline at one time (for a very sick old man with persistent air leak who refused surgery - the tetracycline finally worked). Increasing the tetracycline dose seemed to increase its effectiveness, but it probably also increases potential for toxicity (please do read the tetracycline brochure).

While I have not recognized any side effects except nausea and vomiting, one can achieve extremely rapid absorption of any dissolved material that is dispersed over pleural surfaces. Normally, the safe dose for intrapleural instillation of any chemical substance would be that amount which can also be tolerated if given intravenously over a few minutes. By that "rule," the above tetracycline treatment and the Clagett procedure would both be considered excessive, but every treatment has both known and unknown risks on the cost side of the "cost-benefit" calculation.

Tetracycline can cause extreme pain on contact with unprotected "normal" pleura (pleura that is not covered with fluid, or by protein precipitates, or replaced by scar tissue or malignancy). I have given a number of tetracycline treatments in cooperation with an anesthesiologist friend who provided short-duration pentothal drip anesthesia to the patient in bed (the same technique that we use for cardioversion). The chemical pleurisy was still painful when they awoke, however, and narcotics were often required for the first hour or so.

We have been favorably impressed with the pain relief afforded by addition of a moderate dose of Marcaine (Bupivacaine) to the tetracycline solution, without apparent loss of the "tetracyline effect." I do not know if a Marcaine-tetracycline mix could replace the less than-ideal pentothal ceremony but it certainly decreases the call for narcotics. However, no more Marcaine should be administered intrapleurally than one would give intravenously, for pleural absorption can be rapid.

It is important to note that Marcaine is a potentially toxic drug with long established as well as more recently discovered, adverse side effects; these include a number of maternal and fetal cardiac arrests (after convulsions) that were most resistant to resuscitation: Reportedly these deaths were associated with epidural or paracervical obstetric blocks, respectively. Presumably this long-acting local anesthetic causes a prolonged version of the cardiac arrest occasionally encountered when an

internist gives a one-gram intravenous injection of xylocaine instead of the intended 100 mg dose. The latter arrest usually obligates one to an hour or so of vigorous cardiopulmonary resuscitation (CPR) before recovery.

Since Marcaine is a long-acting local anesthetic that is likely to have ongoing effect for at least eight to twelve hours, prolonged cardiopulmonary bypass should allow a more appropriate resuscitation effort (than a brief hour of CPR). At this writing, Marcaine is no longer recommended for intravenous regional anesthesia, although it is still considered useful for early post-thoracotomy pain relief, when injected into the intercostal nerve area from within the chest, or from outside of the freshly closed rib cage.

Summary: Tetracycline in increasing doses seems increasingly likely to help "seal off" a continuing, minor to moderate air leak. At present, tetracycline instillation is not as reliable as thoracotomy (with wedge resection and brief dry-sponge pleural abrasion), nor has its long-term effect on the recurrence rate of pneumothorax yet become clear.

Tetracycline plus Marcaine is far less painful on normal pleura than tetracycline alone (which can be agonizing), so this mixture may be preferable, but rapid transpleural absorption of Marcaine could be risky, and I have minimal experience with this drug. Be sure to read all package inserts and use the smallest effective dose (see also Chapter 13).

CHAPTER 5: INTRAPLEURAL AIR ACCUMULATIONS AND THEIR TREATMENT

Definitions;... Mediastinal displacement by tension pneumothorax;... Limited volume pneumothorax;... Pneumothorax after pneumonectomy;... How lung blebs develop, and why spontaneous pneumothorax occurs;... Therapy of spontaneous pneumothorax;... Treatment of pulmonary air leak;... Case 1: Minor pneumothorax;... Case 2: Early moderate pneumothorax;... Case 3: Tension pneumothorax;... On reexpansion of lung by closed thoracostomy;... Inadequate lung expansion with major air leak may necessitate thoracotomy;... Case 4: Staphylococcal pneumonia with pneumatoceles and pneumothorax;... Case 5: Unnecessary thoracostomy;... Percussion and ausculation of pneumothorax;... Approach to pneumothorax in review; ... What role do antibiotics play in the treatment of spontaneous pneumothorax?... Reexpansion pulmonary edema;... Mechanism of reexpansion cough and expectoration;... Case 6: Bronchocutaneous fistula without pneumothorax or subcutaneous emphysema;... Subcutaneous emphysema;... Pulmonary air leaks and subcutaneous emphysema;... Decompression of a high-pressure pleural air pocket will end subcutaneous air accumulation;... Case 7: Subcutaneous emphysema without pneumothorax;... Mediastinal air, pneumothorax and pneumopericardium in the newborn;... Chest tubes in the newborn;.,.. Mediastinal air tamponade in the adult; ... Roentgenograms are confusing; ... Locating the source of air leak;... Major traumatic tracheobronchial air leak;... Massive subcutaneous emphysema and resuscitation;... "Benign" subcutaneous air;... Case 8: Recurrent interstitial pulmonary, mediastinal and chest wall air;... Death from massive subcutaneous emphysema;... Subcutaneous emphysema overview.

Definitions:

Pneumothorax refers to air outside of lung, yet within the pleural cavity. The free air of a pneumothorax causes visceral and parietal pleurae to separate. Such air, and any associated fluid or other abnormally present material, occupies and thereby creates, a pleural space. A pleural space may develop almost anywhere within the pleural cavity: It can have any size less than that of the pleural cavity. After pneumonectomy, the pleural space (surrounded only by parietal pleura) fills the pleural cavity.

When healthy lung is not stretched (by subatmospheric intrapleural pressure, or by adhesions between visceral and parietal pleurae) or compressed, it assumes an intermediate relaxed size (at which point, resting pleural space and airway pressures are both atmospheric). A "relaxed" normal lung surrounded by free air still remains expanded (air-filled) rather than atelectatic (collapsed) or con-solidated (fluid-filled).

When diaphragm and chest wall muscles relax on expiration, the pleural cavities decrease in size. Any pneumothorax smaller in volume than the readily available pleural space between relaxed lung and relaxed hemithorax is called a "simple" pneumothorax. Air accumulations that overfill this readily available space and therefore compress the lung and heart are labeled "tension" pneumothorax (meaning pleural space pressures above atmospheric at rest). Even a small pneumothorax remains at above-atmospheric pressures in the usual patient receiving positive-pressure ventilatory assistance, so any minor pneumothorax can then be considered a tension pneumothorax.

Serous, purulent or bloody effusions that are greater in volume than the readily available space between relaxed lung and relaxed chest wall and diaphragm, can similarly be labeled tension hydro-, pyo- or hemothorax. Although intrapleural blood or purulent material can indeed compress the lungs and heart, such a patient untreated is more likely to die of hypovolemia due to blood loss, or toxicity

from rampant infection, than from pulmonary or cardiac compression by massive bloody or purulent fluid collections, unless he has also been transfused, or treated with an inadequate course of antibiotics, or is suffering from additional space-occupying intrathoracic problems (such as tumor, pneumothorax, emphysema or severe bronchospasm with marked pulmonary air trapping).

Death can result when the size of relaxed lung markedly exceeds the size of the relaxed pleural cavity available to contain it. This may be encountered with a crushed or compressed chest or abdomen, or in patients suffering from terminal emphysema or fatal bronchospasm (where lung may entrap so much air that it even balloons out from the open hemithorax at autopsy; see Chapter 13). As a practical matter, "free air" is usually the principal cause of any rapid buildup of intrapleural pressure: Tension hemopneumothorax or tension pyopneumothorax are examples in which the intrapleural blood or pus may be crucial factors in the patient's illness, but the associated air collection accounts for the increasing intrapleural pressures.

Regardless of cause, an overfilled pleural space requires the patient to keep his chest at least partially expanded at all times, for relaxation beyond that point during expiration causes symptomatic compression of lungs and heart. Under these circumstances, a patient can initially compensate for the reduced volume of each breath by breathing more frequently. Efficiency declines, however, as he moves smaller volumes more rapidly, and stale, dead space (airway) air becomes a larger percentage of total air being moved: Intrathoracic airways also narrow significantly during increasingly forceful expirations, which further impedes efficient air exchange.

Although a normal person at rest can temporarily accommodate to mild tension pneumothorax, a simultaneous decrease in blood volume, perhaps as a consequence of trauma, could lead to drastic (even fatal) diminution of venous return and cardiac output at that same intrapleural pressure. The toxicity and inflammatory fluid losses seen with mild tension pyopneumothorax can have a similarly adverse effect. It should thus be apparent that tolerance for any tension pneumothorax that is less severe than "always fatal" will be determined in part by the patient's current state of health.

Mediastinal Displacement by Tension Pneumothorax

As pressure builds up within one hemithorax, mediastinal structures are pushed toward the opposite lung, impairing its function as well. Mediastinal compression also decreases cardiac filling by diminishing blood return from increasingly distended veins to the compressed heart. If intrathoracic pressures continue to build up, the circulation eventually collapses suddenly as the patient suffocates with his chest maximally expanded. Spontaneous tension pneumothorax remains an occasional cause of death in otherwise healthy individuals.

Limited Volume Pneumothorax

Therapeutic pneumothorax is outmoded for the treatment of pulmonary tuberculosis but a minor diagnostic pneumothorax can occasionally improve roentgenographic assessment of the pleura. Limited accidental air entry into the pleural cavity sometimes accompanies thoracentesis or tracheotomy, or the brief positive-pressure resuscitation of a newborn. All these asymptomatic, uninfected, non-replenished, intrapleural accumulations of free air should absorb uneventfully within days or weeks, without leading to significant pleural thickening that might interfere with reexpansion of the underlying normal lung.

Pneumothorax after Pneumonectomy

The empty hemithorax following pneumonectomy is a special type of pneumothorax. Removal of an entire lung leaves a closed, fluid-and-air-filled space that undergoes little volume change but endures considerable pressure variation, for it lacks the responsive air exchange of an underlying lung that normally cushions respiratory and even cough pressure changes.

Significant side-to-side mediastinal movement can therefore be anticipated during the several weeks required for those tissues surfacing the residual pneumonectomy space to thicken and become immobile. Such mediastinal movement appears to potentiate atrial dysrhythmias (particularly over the age of fifty). One can often prevent an overly rapid ventricular rate response to atrial dysrhythmia after pneumonectomy by prophylactic treatment with digoxin.

How Lung Blebs Develop, and Why Spontaneous Pneumothorax Occurs

Inspiratory air flow reaches the alveoli through bronchi that have widened passively in response to subatmospheric intrapulmonary pressures. Conversely, expiratory intrathoracic pressures (being above atmospheric) compress air passages as well as lung parenchyma, thereby prolonging expiration. Positive-pressure ventilatory assistance will hold airways open more widely (especially with "positive end-expiratory pressure" added) but, even then, relative expiratory narrowing may still be detectable.

When, by irritation or disease, there is already narrowing of air passages, the greater ease of air inflow over egress may lead to intra-alveolar "air trapping." Although progressive mechanical overinflation alone can certainly lead to alveolar rupture, the gradual, generalized, alveolar destruction seen with emphysema, which eventuates in poorly vascularized bullae or blebs, is a more complex (chemical and physical) process.

Any ruptured or torn bleb will continue to pump air into the pleural space until its air supply is interrupted - that is, until air pressure around the bleb exceeds air pressure within. Positive-pressure ventilatory assistance raises airway pressures and thus supplies more air to the leaking bleb; this maximizes the likelihood of fatal tension pneumothorax.

Regardless of the pressure gradient or its direction, however, intrapleural air cannot reenter torn lung parenchyma, for leaking blebs always collapse (and thus become blocked) as soon as intrapleural pressure exceeds that within the lung. The example of a punctured balloon serves: One can easily blow air through it with spluttering or other sounds, but try to suck air back and it simply collapses.

"Spontaneous" (non-traumatic) pneumothorax is often encountered during the newborn period (presumably related at least in part to positive-pressure ventilatory assistance), in young adults to their mid-thirties (possibly due to congenital cystic changes of the lung), and in later middle age (as emphysema develops). While that sounds like "most of the time," it really means that pneumothorax is uncommon in children and adolescents, as well as individuals between 35-45 years of age (I cannot vouch for the latter observation, but spontaneous pneumothorax and persistent, peripheral lung air leak are certainly very rare in kids).

Therapy of Spontaneous Pneumothorax

The initial treatment of spontaneous pneumothorax is determined by its clinical presentation.

Depending upon symptoms and circumstances, one might reasonably choose simple observation, needle aspiration, closed thoracostomy or immediate thoracotomy: A simple "First things first" method of evaluation is demonstrated by the following seven questions:

1) Is the situation critical, demanding immediate decompression of the pleural space? If so, do it.

2) Is the diagnosis of spontaneous pneumothorax as definite as time and facilities will allow?

Roentgenographic differentiation of a large lung cyst from pneumothorax may be difficult. Absence of the faint visceral pleural line that represents outer surface of lung displaced away from the chest wall, or presence of anything resembling "lung markings" in an area of supposed pneumothorax, should make one suspect that the radiolucent zone on chest roentgenogram is really a chronic lung cyst and thus unlikely to be the cause of any new symptoms. Localized interpleural adhesions can also distort the roentgenographic appearance of pneumothorax and confuse an appraisal of its extent.

3) Is the pneumothorax major or minor?

The clinical severity of a pneumothorax depends upon a number of factors, including volume and rate of air accumulation, volume and distensibility of the chest cavity, function and compressibility of the underlying and opposite lungs, other space-occupying conditions in chest or abdomen, pericardial disease, blood volume, vasoactive medications, and so on.

Clearly, therefore, the clinical severity of a pneumothorax cannot be determined solely by the estimated percentage of pleural cavity volume not occupied by lung (as projected by x-rays onto the two-dimensional chest roentgenogram). Nonetheless, symptoms often increase as lung is progressively displaced by accumulating intrapleural free air, so it helps to be able to "quantify" the original roentgenographic findings and subsequent changes in some meaningful fashion.

Conveniently vague designations of extent of pneumothorax are: (1) minor (less than 15 or 20%); (2) moderate (less than 50%); and (3) major (with free air occupying over 50% of the pleural cavity, as projected onto the PA or AP chest roentgenogram). That these are radiographic, not clinical, designations of "pneumothorax severity" becomes apparent when one remembers that with advanced emphysema, a "minor" (under 20%) pneumothorax may actually represent critical tension pneumothorax.

Incidentally, a PA chest roentgenogram is considered a "standard chest x-ray" when taken with a 180 cm (six-foot) separation between x-ray source and film: "PA" implies that the x-ray beam has passed from posterior to anterior through the patient's chest (with the film holder therefore pressed against the front of the patient): An "AP film" (usually taken at variable distances and positions in sicker patients who are unable to stand) is not as readily compared with previous or subsequent films, or with a theoretical norm.

Anterior mediastinal structures commonly appear enlarged on AP films, which means that they also obscure more of the lung: This occurs because the x-ray beam continues to diverge after passing through anterior cardiovascular structures, until it strikes the posteriorly-placed film casette (similarly, the shadow of a hand on the wall will enlarge as the hand is brought toward the light bulb).

If one requests a "standard left lateral" chest x-ray, this means that the film will be located at the

patient's left side, again at a 180 cm distance from the x-ray source. For maximum clarity, the side of most interest is usually placed against the film casette (to decrease magnification and blurring by beam divergence). If one does not specify "PA and right lateral" on the x-ray request however, the conventional "PA and left lateral" roentgenograms of the chest will generally be taken.

It is sometimes useful to order a "lordotic" view of the chest. This is an AP film of the chest taken with the patient leaning back. If an "apical" lordotic is ordered, one usually gets the same view, but it may only include the upper chest. The purpose of this view is to see the lung apices more clearly by tilting the chest until the ribs are parallel to the x-ray beam (thus elevating the first rib "shadow" away from the lung field).

In addition, anterior lesions will seem to move upward on the lordotic film (when compared to the standard PA view), while posterior lung abnormalities appear to move downward in association with the posterior rib cage. Thus the depth of a lesion within the lung can be usefully defined on the frontal view, through its relationship to the center of rotation in the thorax. (In other words, a central lesion does not change its relative intrapulmonary position between a PA and a lordotic film.)

Left and right anterior oblique films are similarly useful at times. The LAO and RAO views are best ordered 20 degrees off the flat PA position (as in 20° LAO, which means that the left anterior chest contacts the x-ray film cassette). Once again, posterior lesions will "move" in the same direction as the spine, anterior the reverse. "Coin lesions" that are invisible on a lateral chest x-ray can often be located and defined by such oblique views. (I suspect that computerized tomography of the lung has increasingly replaced such special views, but they remain helpful and ought to be far less expensive.)

4) Is the lung still leaking air?

A pulmonary air leak persists as long as air still enters a leaking bleb. When lung falls away from the chest wall, subatmospheric intrapleural pressure diminishes and the less-stretched bleb often loses its air supply. If a "minor" to "moderate" pneumothorax remains unchanged on subsequent roentgenograms of the chest, this indicates that the recently symptomatic air leak from lung has ended: However, that leak will most likely resume if lung is reexpanded "too early" (before the site of air leak has "healed over") by removal of intrapleural air, or if air is forced into the collapsed bleb by positive-pressure breathing support.

5) Is the pneumothorax recurrent? If so, how severe were previous episodes?

As a guess, the likelihood of later recurrence of spontaneous pneumothorax for the average patient increases by perhaps 20 percent with each episode (although it probably never reaches 100%). Patients who have Marfan's syndrome with bullous lung disease are supposedly faced with a far greater incidence of recurrence of any spontaneous pneumothorax (allegedly up to thirty times normal; I certainly cannot confirm that figure from my own practice, as I have not recognized Marfan's in any patient with spontaneous pneumothorax).

The pattern of pneumothorax usually remains the same. This means that a person who has suffered several minor pneumothoraces, requiring little or no treatment, will most likely develop another minor pneumothorax (rather than tension pneumothorax) during the next episode on that same side. On the other hand, a person in reasonable health who has had one episode of life-threatening tension pneumothorax should stop smoking (to minimize inflammatory bronchial narrowing and alveolar air

trapping) and expect to undergo thoracotomy for a second similar occurrence.

Surgical resection of the leaking bleb can be appropriate treatment for a first episode of tension pneumothorax if the major air leak shows no sign of slowing, or with special occupational hazards. Military pilots or underwater workers who are frequently exposed to a rapid decline of airway pressure are supposedly at increased risk for overdistension and rupture of any pulmonary bleb: Often such employment does not allow early return to medical attention either.

6) Does the patient have access to competent medical care?

The patient who lives far from medical attention may be another candidate for definitive treatment of tension pneumothorax by early thoracotomy.

7) Is there significant bacterial contamination of the pleural space along with this pneumothorax? For example, is this pneumothorax a consequence of ruptured pneumatocele in a patient with staphylococcal pneumonia?

Bacterial contamination makes early lung reexpansion (for elimination of any pleural space) an urgent goal, in order to reduce the risk and sequellae of empyema.

Treatment of Pulmonary Air Leak:

The following case reports illustrate therapeutic decisions based upon these considerations:

Case 1: Minor Pneumothorax

This female sought medical attention two days after an episode of right-sided chest pain: Chest roentgenogram revealed a minor pneumothorax. She lived far from medical care and was to return home by unpressurized airplane.

Comment: When a spontaneous pneumothorax remains "minor" for many hours after the onset of symptoms, it suggests that the causative air leak is neither major nor persistent: Such a pneumothorax is unlikely to require treatment, though temporary enlargement of the pleural air pocket can be expected during any significant in-flight reduction of airplane cabin air pressure with increasing altitude.

If needle aspiration of intrapleural air seems likely to cause less anxiety and discomfort than temporary in-flight enhancement of a minor pneumothorax, one might reasonably attempt air removal (when a few days have elapsed since the onset of pneumothorax). An open, 14 or 16 gauge, plastic needle, inserted through the second anterior intercostal space with the patient leaning back comfortably, can release much of the free intrapleural air during several gentle Valsalva maneuvers (see Chapter 6).

When the site of pulmonary air leak has not yet healed, needle aspiration may just remove air without emptying the pleural space. In this case, follow-up roentgenogram usually reveals little or no change in the pneumothorax (which confirms resumption of air delivery to a still-leaking bleb during the attempted lung reexpansion).

With such a persistent and presumably sterile, minor spontaneous pneumothorax, one might as

well await lung healing and reexpansion without further therapy. An occasional chest roentgenogram confirms progressive resorption of air until lung expansion is complete (but I have seen one chronic smoker sustain a minor, barely symptomatic, pneumothorax for many months).

Any person with pneumothorax should avoid exposure to smoke or other lung irritants. Patients with persistent air leak may also benefit from medications to decrease peripheral air trapping, such as saturated solution of potassium iodide (to liquify thick sputum), antibiotics (to help relieve chronic bronchitis) and aminophylline or other bronchodilators (to treat the bronchospasm often associated with airway irritation or congestion).

Case 2: Early Moderate Pneumothorax

This patient presented at the hospital fifteen minutes after the onset of sudden, severe left chest pain and "pleurisy." Immediate chest roentgenogram revealed a moderate pneumothorax.

Comment: There is little likelihood that this air leak has yet sealed. Needle aspiration of the pleural space could be carried out, but logic suggests (and experience confirms) that this intrapleural air accumulation will continue (for little time has elapsed since bleb rupture, and a significant amount of air has already leaked out into the pleural space): Thus it would be good judgment to perform an early closed thoracostomy. A well-placed well-secured chest tube (connected to simple water seal) is all that is required to move such a patient from the "impending emergency" category to "routine home care."

Case 3: Tension Pneumothorax

The patient wheeled into the emergency room was obviously short of breath, cyanotic and agitated, with neck vein distension while sitting up on the litter (he curtly refused a nurse's suggestion to "lie down and relax"). His trachea was deviated to the left at the sternal notch, and he had a fixed, over-inflated right hemithorax with rapid, labored, predominantly left-sided respiratory excursions. (Such unilateral hyperinflation and opposite-side respiratory predominance are most evident when inspecting the chest from the head or foot of a semisitting patient, rather than on direct frontal view).

Initial treatment: A roentgenogram was not taken, nor was paradoxical pulse evoked (although probably present). One hundred percent oxygen seemed helpful and so was administered by face mask (using the patient's own breathing efforts for air exchange - not assisted ventilation!). Immediate right-sided closed thoracostomy was carried out (after it was decided that he could survive the several minutes required for routine preparation and placement of a chest tube).

In the meanwhile, a 14 gauge needle was kept in readiness for emergency chest wall puncture (via the second anterior intercostal interspace, with or without local anesthesia, depending upon urgency), in case the patient should suddenly start "fading away," or if unavoidable delay was encountered during preparation for thoracostomy (e.g., "We'll have to send for a chest bottle. The night shift must have forgotten to restock... ").

As the Kelly hemostat clamp penetrated the pleural cavity and its blades were opened, a gush of blood-tinged air escaped; the patient expressed immediate relief ("Looks like you're on the right track, Doc!"). The hemostat was left penetrating the chest wall (to permit ongoing pleural space decompression) until leisurely chest tube insertion could be completed.

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The patient was now comfortable and insisted on removing his oxygen mask, for his still-open-to-the-atmosphere chest tube had relieved the critical tension pneumothorax. As soon as water-seal chest bottle drainage was established, forceful bubbling could be seen throughout the respiratory cycle (with decreased bubbling during inspiration).

On Reexpansion of Lung by Closed Thoracostomy:

A chest tube inserted for ongoing major lung air leak is not likely to achieve immediate, complete lung expansion unless relaxed lung is about the same size as the relaxed hemithorax. "Satisfactory" reexpansion is usually signalled by pleuritic pain, coughing spasms and copious expectoration (all lasting several minutes), while lung is stretched and reinflated, retained secretions are mobilized by the renewed and vigorous air exchange, and visceral pleura once again slides against parietal pleura (suddenly "awakening" all of those pleural nerve endings).

After closed thoracostomy, a single cough will usually initiate this short-term cycle of pain, involuntary cough and expectoration. Administration of narcotics and sedatives, in order to diminish these few minutes of "pleurisy," is usually not justified, especially with an ambulatory patient who may shortly return home alone.

Inadequate Lung Expansion With Major Air Leak May Necessitate Thoracotomy

Continued bubbling at the water seal straw tip (persisting even through inspiration) documents a major pulmonary air leak and also shows that subatmospheric intrapleural pressure has not been achieved during inspiration: The absence of subatmospheric pressure within the pleural space during normal respiration suggests that the underlying lung is not exchanging air, so obviously something more must then be done to expedite the removal of this free air (such as additional chest tubes, "high" suction, or both).

If an additional chest tube improves lung expansion and establishes inspiratory tidal (an upward movement of the fluid column in the water seal straw during inspiration, which documents subatmospheric intrapleural pressure), surgery becomes a less urgent concern, for this inspiratory pressure drop at least allows the underlying lung to resume some function. (Incidentally, regardless of air leak, positive-pressure respiratory assistance or chest bottle suction will also eliminate any inspiratory "tidal," for both of these interventions should cause intrathoracic air pressure to remain continuously above the pressure within the water seal bottle.)

In any case, when a very large pulmonary air leak persists, and moderate to major pneumothorax also remains despite large chest tubes and 60 mm Hg chest bottle suction, surgery will almost surely be necessary, so it might as well be performed forthwith. Nonetheless, there are times when several days on chest drainage can clarify the need for surgery to a reluctant patient. Meanwhile, such major air leak may safely be watched by fully trained, conscientious nurses (or by a reliable, well-informed, minimally medicated patient at home; see Chapter 6).

However, if a large air leak prevents any air exchange within the underlying lung, atelectasis and fever soon follow, especially if the patient already has a chronic productive cough from smoking. Within three or four days, such persistently collapsed lung usually develops a significant pneumonia that can be quite resistant to therapy until lung compression is relieved.

Summary: Once it becomes clear that surgery will be required to control major pulmonary air leak, unnecessary delay should be avoided, especially if the affected lung remains atelectatic.

Case 4: Staphylococcal Pneumonia with Pneumatoceles and Pneumothorax

A three-month-old child with acute staphylococcal pneumonia entered the hospital with bilateral pneumatoceles and a minor right-sided pneumothorax.

Treatment: Right closed thoracostomy was performed.

Comment: Pneumatoceles can rupture into either pleural space, although they will disappear as the child improves on antibiotics. By definition, pneumothorax due to ruptured pneumatocele means "early empyema," as the air source was an infected, acute lung cyst. Therefore, the contaminated pleural space should be obliterated, preferably by continuous chest tube drainage since antibiotic access improves with removal of infected material - which helps prevent early lung entrapment under an infected peel.

Intrapulmonary (subpleural) staphylococcal pneumatoceles may balloon to impressive dimensions and become indistinguishable from large intrapleural air-fluid levels. A closed thoracostomy tube placed into such a cyst can help a toxic child enormously. The theoretical risk of such thoracostomy leading to empyema need not deter the surgeon, for this represents a lesser risk than spontaneous evacuation of a huge, germ-laden, fluid-filled cyst into pleural space or bronchus.

When punctured by a chest tube, air leak from even the largest pneumatocele will only be minimal to moderate anyhow (for a wide-open bronchial connection would have prevented pneumatocele formation in the first place). In addition, the very same chest tube that initially penetrated the cyst wall will then provide appropriate drainage for any pleural contamination and air leak.

Case 5: Unnecessary Thoracostorny

A well-endowed young woman entered the hospital with complaints of right-sided chest pain of recent onset. She was restless, slightly cyanotic and dyspneic, and showed flaring of her nostrils on inspiration: Diagnostic percussion of her chest suggested left lung hyperresonance.

Treatment: The distracted intern placed a closed thoracostomy tube in her left chest, with some air return and slow bubbling but no clinical improvement. Follow-up chest roentgenogram showed the left lung expanded and chest tube in proper position, as well as pneumonia of the right lung.

Discussion: Diagnostic errors are commonly based upon distraction (a "lovely" lady, "aggressive" relatives, a "disgusting" drunk), unnecessary haste (too busy, a need to appear competent or in control, unrealistic confidence in oneself, or in some symptom, sign or test), preconceptions that persist despite clinical evidence (the pain was on the side of less resonance, but there had just been a lecture on tension pneumothorax) and poor judgement or insufficient skill (there was time to wait for a roentgenogram before dealing with this non-critical problem, careful auscultation would have revealed rales on the right). An "initial clinical impression" is just that; expect it to be wrong much of the time.

Therapeutic injuries: The wrong thing done at the wrong time is more likely to be done poorly by the wrong person. An actual pneumothorax usually protects underlying lung from penetration by the local anesthesia needle passing through the chest wall; in addition, one should always perform

confirmatory aspiration of free air through this thin needle before inserting a chest tube.

The results of "acute incompetence" (due to physician fatigue, illness, medication or inattention) may closely mimic those of chronic incompetence or an inability to work under pressure (such a physician is miscast for a role which includes insertion of chest tubes). Lastly there is the occasional (intellectually dishonest) physician who "needs the procedure worse than the patient does." (He, too, usually has a high complication rate, for he will be as inadequate at evaluating the evidence, or learning from his own mistakes, as he is at understanding himself.)

Accepting responsibility for error can be lifesaving: We do not always do "just right." If we strive to minimize untoward results of our errors of omission or commission, the patient and physician both benefit. It is tempting to quickly remove a chest tube that was placed in error, or to avoid mentioning a "probably unnecessary" needle insertion that may have penetrated lung, especially when another physician will be taking over the care of the patient. (He might criticise... but then again, he might not notice...)

One bad deed leads to another: In this case, immediate removal of the chest tube by the offending intern in the emergency room (as soon as he realized that the problem was pneumonia, not pneumothorax) would likely have been followed by a real pneumothorax (which this patient would have tolerated especially poorly, due to pneumonia on the opposite side). It is quite plausible that her chest tube skin incision site in the submammary fold might then never have been noticed, that the initial x-ray film could have been "lost" before it was "reported out" by the radiologist, and that an increasingly dyspneic lady with "bad pneumonia reported on x-ray examination by the admitting physician" would be intubated for positive-pressure ventilatory assistance when she "got into trouble" (which might promptly result in fatal tension pneumothorax).

On the other hand, if this patient is taken to the ward with the chest tube in place, the peripheral parenchymal lung injury should soon heal without harmful sequellae, allowing chest tube removal as she recovers from her severe pneumonia. Clearly, the patient is best served by the honest physician who is able to face his own embarrassing errors when the need arises (the practice of medicine is routinely stressful, frequently embarrassing, and often rewarding). Surprisingly, patients appreciate a competent physician who "cares when he goofs" far more than "Dr. So-And-So who thinks he is perfect." Both make mistakes, and in the long run it is easier to recover from mistakes than to cover for them.

Percussion and Auscultation of Pneumothorax

Both percussion and auscultation can be misleading, even with major pneumothorax. On occasion, one may even elicit dullness to percussion or hear normal breath sounds on the side with pneumothorax. This again demonstrates the desirability of a chest roentgenogram before therapy, when time and circumstances permit.

Incidentally, a finger tapped directly upon the mid-clavicle will minimize the normal variation in percussion tones between opposite hemithoraces (that are caused by minor asymmetries in soft tissue development or location). Such "direct percussion" of the clavicle is especially helpful in diagnosing pneumothorax.

Approach to Pneumothorax in Review

Diagnosis of pneumothorax is based upon suspicion, observation, auscultation and percussion, with history and roentgenograms taken as time permits. Appropriate therapy depends upon the cause of each air accumulation, as well as its extent and the likelihood of its persistence and recurrence.

It is reasonable to merely "observe" a minor pneumothorax that is not associated with significant symptoms or infection: A larger air collection that is unlikely to recur can simply be aspirated by needle. Closed thoracostomy (with one or more chest tubes) may be required for acute tension pneumothorax with major air leak. Thoracotomy for suture of leaking lung is generally reserved for the severe and persistent or recurrent case.

In view of endoscopic progress to date, it seems likely that thoracoscopic application of a sealant, patch or plug to the leaking bleb will eventually prove practical for closure of persistent spontaneous lung air leaks. Perhaps a small, intrableb injection of fibrin glue might be a good project for evaluation in the laboratory.

What Role do Antibiotics Play in the Treatment of Spontaneous Pneumothorax?

When a patient presents with spontaneous pneumothorax, one often obtains a history of chronic bronchitis or recent upper respiratory infection. This suggests that inflammatory narrowing of air passages may have contributed to the current air leak by promotion of peripheral air trapping. Antibiotic treatment seems reasonable for any bronchitis associated with spontaneous pneumothorax.

In theory, any bronchopleural air leak could carry airborne bacteria from the atmosphere directly into the pleural space: Yet empyema is rarely if ever seen as a consequence of "ordinary" spontaneous pneumothorax (meaning pneumothorax not associated with clinically evident, significant infection of the underlying lung). This again suggests that the presence of chest tubes will not cause empyema (although they certainly help cure it).

One gathers that uninfected lung may continue to "filter out" most airborne bacteria, even with minor to moderate bronchopleural air flow, or perhaps that more bacteria are required to initiate an empyema than can gain access to pleura by this route. Maybe long-term pleural space sterility cannot be maintained in the presence of a persistent major bronchopleural fistula, but that is only a theoretical consideration, as atelectatic lung underlying such a major air leak soon becomes infected anyhow (with strong likelihood of empyema developing soon thereafter, unless the air leak is surgically corrected before such complications can arise).

Experience suggests that prophylactic antibiotics are not helpful to the patient with a chest tube, regardless of air leak (despite persistence of an "infection-prone" pleural space until all air leak has ceased). It also appears that spontaneous air leak rarely develops from an already infected lung, except in certain unusual situations (e.g., intrapleural rupture of a lung abscess or staphylococcal pneumatocele): Most likely the decreased air content associated with atelectasis and consolidation, and the overlying inflamed and thickened pleura, both diminish any tendency toward "spontaneous" air leak under these circumstances. Therefore, in the absence of clinical symptoms or roentgenographic findings of pneumonia, any patient presenting with spontaneous pneumothorax may be assumed to have uninfected lung (and no indication for prophylactic antibiotics).

Prophylactic antibiotics cannot prevent bacterial pneumonia from developing in atelectatic lung anyhow: They merely ensure that the eventual flora will be more antibiotic-resistant. On the other

hand, measures to correct atelectasis and restore normal ventilation (such as chest tube drainage of a large pleural space and frequent vigorous cough, or even positive-pressure assisted ventilation and endotracheal suction) will usually prevent such pneumonia. Lung infection by virulent organisms may occur with or without atelectasis, of course, but such episodes usually cannot be prevented by prophylactic antibiotics either (except possibly during some epidemics, or when treatment is only directed at certain bacteria, or used to counter a known, increased susceptibility to infection).

When pleural infection does develop during or after chest tube drainage of air or fluid, this empyema almost surely was due to nearby pulmonary parenchymal infection, rather than pleural contamination by the air leak or chest tube. It appears that harmful quantities of bacteria can only gain access to a pleural space across infected lung. (The special case of postpneumonectomy empyema is discussed in Chapter 4.)

Onset of empyema while a chest is still being drained will be heralded by fever, malaise and increasing amounts of turbid chest tube drainage (containing bacteria and white cells on microscopic examination). Obliteration of the infected pleural space then becomes urgent, and any persistent major air leak that prevents full lung expansion should be eliminated. With or without surgical closure of such air leak, effective and perhaps prolonged chest tube drainage will be essential. Appropriate antibiotic therapy can greatly reduce the morbidity and mortality of pneumonia associated with empyema and air leak.

Summary: Antibiotics are important in the treatment of bronchitis, pneumonia and pleural space infection; they have nothing to contribute at closed thoracostomy for spontaneous pneumothorax. Antibiotics are also unlikely to prevent pneumonia consequent to atelectasis. Atelectasis due to a mechanical problem, such as a compressed lung or blocked bronchus, is best treated by mechanical means (pleural space evacuation, deep breathing, coughing, endotracheal suction, incentive spirometry, positive-pressure ventilation, and so on).

Reexpansion Pulmonary Edema

Rapid reexpansion of compressed, presumably atelectic, lung has reportedly been associated with severe or fatal pulmonary edema: While theories abound, the cause of that complication usually remains unclear to the participating physicians.

It is self-evident, however, that compressed or atelectatic lung can either be reinflated from within, or reexpanded by "suction stretching" from without, or that both methods can be combined. Furthermore, we have all learned that successful lung reexpansion by any method will regularly result in copious expectoration. In fact, those who do not go through a short paroxysm of cough and pleurisy upon institution of pleural space drainage have either endured only a simple pneumothorax (that has allowed uninterrupted lung function), or else lung reexpansion has not yet been achieved (as follow-up roentgenograms will soon demonstrate).

Mechanism of Reexpansion Cough and Expectoration:

When lung compression is relieved, air again passes in and out of the pulmonary parenchyma. Previously stationary mucus now gets blown about, irritating the airways and causing paroxysms of coughing. Pleural reapproximation in the course of lung expansion results in similarly heightened pleural awareness or "pleurisy" for a few minutes, which leads to gasping respirations, further mucus

dislodgement, increasing cough, and so on.

This uncomfortable process concludes after several minutes, when airways have been cleared and pleural surfaces are once again sliding smoothly. All in all, this seems an admirable arrangement for reestablishing lung function, with each cough further inflating distal alveoli from within (at pressures above atmospheric).

In contrast, it is "unnatural," painful and probably hazardous, to try to "stretch" a lung with external suction while its airways are still blocked by mucus; the marked decrease of intrapulmonary air pressure associated with significantly subatmospheric intrapleural pressure, could easily draw "edema fluid" into the alveoli. Perhaps additional neurogenic or humoral mechanisms come into play when the very rarely reported bilateral pulmonary edema follows forceful unilateral lung reexpansion (but that, too, could simply be consequent to vigorous inspiratory efforts needed to draw air in through upper airways filled with frothy secretions).

Theory aside, I have never encountered reexpansion pulmonary edema. This suggests that our usual approach is quite safe. It does seem appropriate, however, to encourage frequent cough (or to apply some other form of intermittently elevated endobronchial air pressure) during reexpansion of previously compressed lung: In addition, "high" chest bottle suction or the rapid tube drainage of a large pleural effusion should not be permitted when it might force reexpansion of an atelectatic lung that is still full of secretions.

It has always seemed reasonable to avoid application of chest bottle suction unless vigorous coughing (while the chest tube is connected only to a simple water seal apparatus), fails to achieve good lung inflation. When such vigorous coughing proves insufficient, especially in the presence of major air leak, a gradually increased application of chest bottle suction (while coughing continues) should be safe, provided that one reduces the suction level slightly whenever lung stretch becomes painful.

When a patient is too weak to cough and raise secretions successfully, one should consider reexpanding the atelectatic lung by endotracheal inflation, combined with intermittent endobronchial catheter suction as needed for removal of secretions. Chest surgeons routinely utilize this approach during thoracotomy for major air leak; at times we even place an endotracheal tube electively (using topical anesthesia), to perform a vigorous, short term "pulmonary toilet" in the face of severe, unremitting atelectasis.

Conclusion: Pulmonary edema following reexpansion of compressed or atelectatic lung is either a "rare and random" event not yet subject to prediction or remedy, or it is rare because we usually do everything "about right." Present knowledge suggests that a collapsed lung should be reinflated by vigorous coughing (or positive-pressure ventilation) during removal of a major effusion or pneumothorax - rather than relying solely upon externally applied chest bottle suction or a fluid siphon to achieve such expansion.

Frequent cough is especially encouraged during chest tube drainage of a major chronic hydrothorax, as the siphon effect can rapidly exceed "high" suction. A published suggestion that reexpansion pulmonary edema be remedied by immediately opening the chest tube to the atmosphere (to allow recollapse of the involved lung, in order to curb secretions), also tends to incriminate externally applied suction as an important factor in reexpansion pulmonary edema. It seems likely that

endotracheal intubation and vigorous "pulmonary toilet" along with positive-end-expiratory pressure would also be very helpful in correcting life-threatening pulmonary edema from this (or other) cause.

If lung reexpansion is restricted (by thickened visceral pleura, pulmonary fibrosis or inflammation, malignant infiltration of lung or endobronchial mucus and atelectasis), any non-obtunded patient will loudly declare when removal of fluid or air must be halted, or at least decreased in rate. Complaints of discomfort during lung reexpansion (or at any other time) should be respected and evaluated, rather than overwhelmed by narcotic administration. One could argue that a markedly thickened visceral pleura or pulmonary fibrosis might reduce the risk of reexpansion pulmonary edema, but even that does not justify a forced rapid lung expansion, for under those circumstances, the mediastinum is subjected to the excessive suction.

Excessive or unnecessary sedation increases the likelihood of retained secretions and recurrence of atelectasis. On the other hand, judicious postoperative or post-trauma narcotic administration as needed may help control severe or persistent pain that prevents adequate cough or deep breathing. (In other words, an oversedated, undisturbed patient is not likely to cough as well as a normal patient, or one pharmacologically relieved of discomfort who is then repeatedly encouraged to "clear his chest" by cough.)

Case 6: Bronchocutaneous Fistula Without Pneumothorax or Subcutaneous Emphysema

A twenty-seven year old male came to the hospital with a parasternal stab wound (by scissors) in the right second interspace. His wife was right-handed and the blades apparently penetrated the right lung, for the stab wound bubbled whenever the patient coughed or strained. Initial and repeat chest roentgenograms were unremarkable, so he was discharged with instructions to apply antibiotic ointment to the wide-open wound and avoid irritating his wife.

Comment: Suture-closure of this stab wound into lung would have caused subcutaneous emphysema. Presumably, the patient had obliterative interpleural adhesions at the stabbed area due to prior pneumonia, for he failed to develop pneumothorax. The small, peripheral lung air leak was perfectly vented to the atmosphere through its conveniently located, overlying stab wound. No therapy was required and recovery was uneventful. There was no pleural pocket to drain, so no indication for closed thoracostomy. A chest tube placed in this patient would simply have created a second, possibly more serious, iatrogenic stab wound of lung.

Subcutaneous Emphysema

Air bubbles within soft tissues give a unique crackling or crepitant sensation on deep palpation of overlying skin. Although such air may be distributed widely (through muscles, fat and other soft tissues), this condition is referred to as "subcutaneous emphysema". Soft tissue gas bubbles most commonly originate from lungs or esophagus: Palpable gas in tissues may, on rare occasion, also result from infection by gas-forming bacteria, hydrogen peroxide irrigation of open wounds, blast or air hose injury, sucking soft tissue wounds (as with an unstable compound fracture), and rapid decompression ("the bends").

There is a natural tendency to swallow air in response to esophageal distress, and the extraperitoneal, extrapleural location of the esophagus probably contributes to air buildup in surrounding tissues after esophageal injury: Air that escapes from more distal gastrointestinal tract

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perforations is commonly restricted to the peritoneal cavity by the surrounding intact peritoneum.

Pulmonary Air Leaks and Subcutaneous Emphysema

Lung is the usual source of subcutaneous emphysema, and the only significant source of massive subcutaneous emphysema. Air that escapes from lung can only enter the soft tissues via a localized pocket of air that reaches pressures well above atmospheric (usually in combination with a parietal pleural defect that reduces tissue resistance to air penetration). When a fractured rib has punctured lung, or an inter-pleural adhesion tears as the pneumothorax increases, the resulting pleural defect can channel minor or even massive volumes of air into the soft tissues.

Generalized balloon-like inflation of a patient cannot result from a spontaneous pneumothorax surrounding an entire lung, however, for fatal compression of lung and heart would occur long before the intrapleural air pressure became high enough to force large quantities of gas out into the tissues. Therefore, any severely inflated patient who is still alive must either have been "blown up" gradually, or intermittently, or from a high-pressure air pocket that was not large enough to compress an entire lung.

An unfortunate exception is the injured patient with pulmonary air leak who is given positive-pressure assisted ventilation during resuscitation without simultaneously being provided adequate pleural space decompression. Such a patient is rapidly "ballooned" by the ventilatory assistance (which commonly continues long after his immediate death from tension pneumothorax, if he was not dead already).

The only patient with a closed chest and torn lung who can possibly survive vigorous positive-pressure ventilatory assistance is one who has had fibrous obliteration of the involved pleural space: Air leaking from damaged peripheral lung will then be driven directly into soft tissues, without simultaneously creating a fatal tension pneumothorax.

Decompression of a High-Pressure Pleural Air Pocket Will End Subcutaneous Air Accumulation

If an air leak from lung can be vented adequately to the atmosphere (to prevent intrapleural or air pocket pressures from rising significantly above atmospheric), there will be little further accumulation of soft tissue air. Even with proper chest tube drainage, however, it is not uncommon to palpate crepitus near a closed thoracostomy wound, or over the lower neck after a thoracotomy that has involved injury to parietal pleura near the apex: This air was pushed into those nearby soft tissues during expiration or by momentarily heightened cough pressures.

On the other hand, when a patient with moderate postoperative air leak develops marked subcutaneous emphysema during the night, one can safely assume that chest tubes were not properly tended. It usually turns out that chest tubing was allowed to hang from the bed in fluid-filled dependent loops, or that the patient rolled onto or otherwise blocked his tubing while heavily sedated or asleep: Either situation prevents air escape, thus causing temporary tension pneumothorax.

Attentive nursing can usually prevent such intermittently excessive intrapleural pressures (despite the presence of properly positioned and functioning chest tubes) until the patient becomes alert and strong enough to detect and deal with these hazards himself. In addition, as pleural healing progresses, the chest wall becomes less susceptible to air penetration.

Case 7: Subcutaneous Emphysema Without Pneumothorax

On admission, this patient had air in the soft tissues overlying her fractured rib, as well as palpable crepitus in the neck. A chest roentgenogram revealed chest wall and mediastinal air, but no pneumothorax.

Comment: When lung laceration by broken rib causes subcutaneous emphysema without pneumothorax, this simply confirms post-inflammatory fibrous union of the overlying visceral and parietal pleurae. In this case, the localized collection of air from torn lung was decompressed through fused pleurae into the chest wall (along a path slashed by the broken rib at the time of injury), as well as back into her mediastinum and neck along loose perivascular tissue planes within the lung.

Note: Although mediastinal air accumulations from an esophageal leak regularly cause significant dysphagia as a consequence of leakage of irritating gastric fluids, any less-than-extreme mediastinal air buildup from a pulmonary source will not interfere significantly with swallowing. Minor mediastinal air collections that are not associated with infection soon absorb uneventfully.

Mediastinal Air, Pneumothorax and Pneumopericardium in the Newborn

Not uncommonly, a newborn requiring resuscitation will develop roentgenographically visible mediastinal air; presumably this air accumulation is consequent to rupture of an alveolus inside the lung, with air dissecting back from the small, high-pressure intrapulmonary air pocket into the mediastinum along the pulmonary artery, or into the pericardium along the pulmonary veins. (Pulmonary arteries continue to be invested in a loose perivascular sheath in the adult: Pulmonary veins less so. Pneumopericardium secondary to interstitial alveolar rupture is rarely, if ever, encountered in later life.)

Anyhow, we are told that a progressive pericardial or mediastinal air accumulation may significantly compress the heart unless that air "ruptures out" to create a unilateral or bilateral pneumothorax (which is then remedied by chest tube as indicated). Recently there have been reports of symptomatic pneumomediastinum or pneumopericardium in newborns that required prompt surgical decompression to assure survival.

Closed or "open" (by incision, with tube placement under direct vision) substernal chest tube placement was therefore recommended for symptomatic episodes, which sounds appropriate The apparent increase in number of newborns who allegedly suffer from pericardial or mediastinal air collections requiring drainage, is probably explained by the large number of very premature infants now being kept alive for prolonged periods on positive-pressure ventilatory support.

Chest Tubes in the Newborn

Chest tubes often seem less effective in tiny children; certainly tube position is far more critical here than in the adult. In addition, air leaks are usually small and expiratory pressures low, so it can be exceedingly difficult to maintain tube patency and function. In fact, a supine neonate will often develop tension pneumothorax despite a patent chest tube properly inserted through the anterior chest wall. The apparent explanation follows:

If the tube tip and side holes are located a mere centimeter or two deep to the anterior chest wall,

then a one or two centimeter thick anterior air pocket may develop. Particularly when a baby is already in desperate straits, with consolidated and relatively incompressible lungs that require high inspiratory pressures to achieve minimally adequate ventilation, the air pressure within such an anterior pocket can rapidly rise to intolerable heights as lung air leak continues.

At this point, one or more simple openings "poked" through the anterior chest wall can provide lifesaving release of the tension pneumothorax, and so allow enough time to get the chest tube or tubes exactly positioned and working properly.

Procedure: To immediately release such an anterior tension pneumothorax in a supine infant on a respirator, simply insert the relatively sharp tip of a small hemostat through intact skin or a tiny skin stab wound into the appropriate anterior interspace (preferably somewhere between ribs 2-4, away from nipples and mediastinum) and separate the hemostat blades. Under these circumstances, lung will not be displaced by any atmospheric entry through the new chest wall hole, since persistently above-atmospheric respirator pressures still keep the lungs maximally expanded (except for space occupied by the tension pneumothorax; see also following chapters).

No tube can improve on the chest decompression provided by such a newly made anterior transthoracic hole into the pleural space - though a chest tube often interferes with necessary air egress in these tiny patients. Without haste, therefore, carefully position an anterior chest tube through this hole, so that it lies entirely in front of lung (and make sure that it is working effectively). A simple water seal bottle of appropriate size may release trapped intrapleural air more readily than a chest bottle suction device, for suction can easily draw soft newborn tissues onto all of the chest tube drainage holes (thus blocking the tube and allowing tension pneumothorax to reaccumulate, despite a perfectly-positioned chest tube that is also patent when "milked").

Mediastinal Air Tamponade in the Adult

On rare occasions, pulmonary air leak in an adult may lead to mediastinal air accumulations under considerable pressure. Dangerously high mediastinal air pressures will cause major distress to any non-obtunded patient – and paradoxical pulse is usually present. Although paradoxical pulse normally is best detected and followed by frequent blood pressure measurements, taking blood pressure by cuff and stethoscope can be a very noisy business in the patient with advanced subcutaneous emphysema (for the crackling is greatly amplified by auscultation): Radial artery cannulation, or simple palpation of systolic blood pressure variation at the wrist while the blood pressure cuff is slowly decompressed on the upper arm, will then usually suffice (see Chapter 15).

Symptomatic mediastinal air tamponade in the adult is readily relieved via a short pretracheal (or lateral cervical) incision under local anesthesia. Blunt (finger) or sharp dissection is readily carried down through the distorted, air-inflated tissues until penetration of the pretracheal fascia (or its more lateral equivalent) releases a rush of air.

This may emerge so forcefully that one momentarily fears accidental tracheal transection. A soft rubber drain or catheter can then be left in the incision for several days to assure continued air release, and the incision covered with a loose, frequently changed, povidone-iodine-soaked gauze dressing.

Roentgenograms Are Confusing

Chest roentgenograms of a patient with marked subcutaneous emphysema are often impossible to interpret, as the medley of highlights and shadows caused by air dissecting around and through chest wall structures, completely distorts and obscures the familiar roentgenographic appearance of tissue boundaries. Pneumothorax is difficult or impossible to detect under these circumstances.

Locating the Source of Air Leak

Fibrous union of visceral to parietal pleura permits torn lung to pump air into the mediastinum and chest wall without creating a pneumothorax. The difficulties encountered in locating the very small, high-pressure, air pocket presumed responsible for such dissemination of air, usually makes it impractical to decompress the air source by tube (although secondary mediastinal air tamponade is watched for and treated, if need be).

Under these circumstances, when progressive gaseous inflation of soft tissues leads to severe symptoms, one sometimes considers performing a thoracotomy for open decompression and suture of the leaking lung bleb: If the hemithorax of origin remains unclear, a sternotomy would even allow bilateral access. The best or only "lateralizing" clue may come early in the episode, when subcutaneous air accumulation is most marked near the site of origin. Thus, with rib fractures in an unconscious patient, air may first appear in soft tissues near one of the involved ribs (as well as in the neck).

A few coughs or respirator breaths later, however, air dissection will often be so generalized, and x-ray studies so misleading, that only the patient's symptoms can thereafter guide the surgeon back to the source of air. Usually, therefore, when the patient has marked subcutaneous emphysema and no pneumothorax, one will not approach the unidentified "air pocket of origin" surgically, unless major airway injury is suspected and identified at bronchoscopy. (I have not performed thoracotomy or sternotomy solely for unrelenting subcutaneous emphysema, so this must be an uncommon indication for surgery.)

Major Traumatic Tracheobronchial Air Leak

The initial treatment of a critically injured patient with major air leak is often non-operative, using one or more closed thoracostomies to decompress any intrapleural air pockets. Often enough, a tracheal or bronchial injury will not be recognized until bronchoscopy is carried out later to assess persistent massive air leak or explain atelectasis or partial airway obstruction. Urgent repair of major bronchial or tracheal tears seems appropriate whenever this is possible.

A tracheostomy can reduce expiratory resistance (and thus expiratory pressure and lung air leak) in patients with major, poorly controlled air leak who are not on a respirator. Positive-pressure ventilatory support may become difficult or impossible in the face of massive pulmonary air leak; thus "inability to ventilate" could conceivably become the major indication for an early surgical attack on the source of air leak (although I have not encountered that necessity).

"Inability to ventilate" here means that air blown into the endotracheal tube flushes directly out of a large hole in the lung, thereby depriving both lungs of the inspiratory pressure build-up that is necessary for assisted ventilation. A large flow of air "whistling" through lung from the respirator to the chest bottle could even tend to suffocate the patient by its Venturi effect.

(I occasionally suffocate yellow-jackets that way - sucking individuals or even an entire nest

cohort into my vacuum cleaner hose - then I leave the vacuum cleaner running for a few minutes. However, if the nest is underground or well away from the house, it is simpler to hit a few yellow-jackets near the nest entrance with a shot of chili pepper "bear spray" – which leads to lack of chemical recognition and total nest-mate fraticide. At these times it is best to wear a protective screened hat and thick clothes. But I digress).

The above-stated reasons explain why a patient with large air leak may achieve more effective pulmonary air exchange through his own respiratory efforts than during assisted ventilation (provided he is awake and strong enough to breathe). For by generating his own subatmospheric inspiratory pressure, he still draws air into one or both lungs (especially if, despite major air leak, effective chest bottle suction keeps the pneumothorax relatively small while the patient breathes spontaneously).

Under such circumstances, even a massive lung parenchyma air leak will be mostly or entirely one-way, from lung to pleural space and not the reverse. This means that whatever subatmospheric inspiratory pressure drop the patient is able to achieve will be rewarded by effective air movement through trachea and bronchi.

Such a situation, which may follow decortication of a very emphysematous lung, for example, can be most taxing for all involved. While I have not encountered massive pulmonary air leak for a number of years, it seems probable that high-frequency ventilation could improve ventilatory control under these circumstances (but that is merely a guess).

Massive Subcutaneous Emphysema and Resuscitation

On occasion, one is called to treat a severely injured or spontaneous pneumothorax patient who is grossly distended with subcutaneous emphysema and undergoing cardiopulmonary resuscitation. Although this patient most likely died of severe tension pneumothorax at (or before) the time that assisted ventilation was initiated, it is always possible that immediate decompression may still help (perhaps he was fortunate enough to have previously fused pleural surfaces).

In such desperate circumstances, rather than first requesting or viewing an undecipherable chest roentgenogram, or preparing sterile supplies for chest tube placement, one swiftly makes two short bilateral anterior chest wall skin incisions and forces a finger or hemostat through distended chest wall tissues into each pleural space (it only takes a moment to penetrate both sides, and one must always anticipate bilateral air collections in any overinflated, terminal patient).

Even without a chest tube, these anterior air holes can satisfactorily decompress (and at times, visibly deflate) such a patient, especially if he is suffering from tension pneumothorax (intrathoracic finger exploration is sometimes appropriate for identifying nearby injuries as well).

Introduction of a chest tube through such a decompressive finger hole can further improve pleural space drainage, and allow preservation of free intrapleural blood for possible autotransfusion. Here again, the water seal (one-way valve) function is not required during resuscitation, as air cannot reenter the chest via the finger hole or open chest tube, as long as inspiratory and expiratory pressures are both kept above atmospheric by assisted ventilation.

Anterior chest wall needle or "finger holes" (using an unwashed ungloved finger if need be) can stabilize the injured patient requiring assisted ventilation or the patient dying of tension pneumothorax

far from a hospital, for many hours pending formal insertion of a chest tube. If it turns out that the pleural space was previously obliterated by pleural fusion, a hole from the skin down through fused pleura can still decrease subcutaneous emphysema of the chest wall to some degree (and there should be no harm from such emergency finger exploration, even if lung parenchyma was penetrated, provided the cut is thereafter kept clean and left open until it heals).

Obviously, with ordinary spontaneous pneumothorax, it is only necessary to vent one hemithorax (assuming the usual diagnostic criteria or a patient who is able to identify the side of painful onset). Paramedics or ambulance personnel have learned that 100% oxygen can be lifesaving when delivered by non-pressurized face mask; they must always be reminded that positive-pressure breathing assistance can immediately and fatally aggravate tension pneumothorax, unless the pleural space is simultaneously decompressed by needle, tube or finger hole.

A large bore needle can successfully decompress tension pneumothorax temporarily, while the patient is breathing on his own, but the change to assisted ventilation usually demands that a much larger chest tube or finger hole be placed through the chest wall, in order to prevent fatal tension pneumothorax as a result of the markedly increased pulmonary air leak.

"Benign" Subcutaneous Air

An unusual but benign form of subcutaneous emphysema is seen in individuals up to middle life, perhaps more during pregnancy, who experience sudden, sharp (occasionally minimal) chest pain, often on coughing or straining. Examination reveals minor subcutaneous crepitus in the lower lateral neck. Chest roentgenogram shows mediastinal emphysema but no pneumothorax. Barium swallow would show normal esophagus, as one might also guess from the lack of dysphagia. There is no obvious air source in these patients, and their clinical course is benign, as these small air accumulations disappear within a few days.

Mechanism: Most likely a bleb ruptured somewhere within the lung substance. Air accumulating there under pressure then dissected back along perivascular tissue planes into the mediastinum and neck: Antibiotics, bronchodilators or potassium iodide can be used for any associated bronchitis, wheezing or thick mucus.

Case 8: Recurrent Interstitial Pulmonary, Mediastinal and Chest Wall Air

A thirty-two year old female presented in the early 1970's with a chief complaint of painful monthly swelling and crackling of her right breast. Eventually, after many non-contributory studies, a right thoracotomy was carried out (through fused pleurae) for wedge resection of large, crepitant, apical blebs that showed interstitial emphysema on microscopical examination. The operation relieved her of such episodes for many months.

The "pneumomammae attacks" subsequently resumed, however, associated with (and attributed by me to) a total inability to discontinue heavy smoking, and finally right upper lobectomy was recommended. The patient rather wisely declined further surgery and her attacks gradually decreased in frequency and severity.

Since that time, several reports have alleged that endothoracic endometriosis can cause recurrent pneumothorax at the time of the menses. Reportedly, this "catamenial pneumothorax" has been treated

successfully by tubal ligation as well as by suture-closure of small diaphragm perforations.

This suggests that air enters through the abdomen via the tubes as the cervical canal opens during menstruation. Not surprisingly, the apparent reversal of flow through the fallopian tubes is often associated with a widespread distribution of endometrial implants through the peritoneum and even thorax (where they reportedly also cause bloody pleural effusions).

The increased risk of ovarian cancer, many years after perineal dusting by talcum powder that presumably contained asbestos, is additional evidence that "wrong-way tubes" exist (especially in nulliparous women under 40 years old). I suppose the subcutaneous air in my patient either entered the chest via diaphragm perforations or leaked via an endometrial implant on the lung surface through pleura fused by chronic endometriosis: However it came about, this is another of medicine's unlikely stories. Incidentally, a hemothorax due to endometriosis allegedly builds up more gradually, so that the monthly incidence is not as apparent. Fortunately, it responds to total hysterectomy or Danazol therapy (according to the literature).

Death from Massive Subcutaneous Emphysema

Massive subcutaneous emphysema is generally due to ongoing air leak from a ruptured bleb or ruptured bronchus. It may spread over the entire body in a few coughs, with especially early and massive distension of "loose" tissues such as scrotum and eyelids. Whatever its origin, we were once taught that this grotesque inflation of the patient is not hazardous in itself. That is certainly untrue in the unusual circumstance of mediastinal air tamponade discussed above. Even without such a rare mechanical problem, some of these markedly inflated patients do die, often after a period of delirium. (One can sometimes decrease their confusion by intermittently pressing air from the swollen eyelids to permit some vision.)

In the past, we attributed this "toxic psychosis" to liver disease or delirium tremens. With gross gaseous invasion of all soft tissues, however, it seems equally plausible that various lymphatic-venous connections could provide ready access of air to the right heart. With a continuous flow of small air emboli reaching the right heart and lungs, it is certainly conceivable that some bubbles can pass through to the left heart and on to the cerebral arteries. Unexplained delirium and death in the patient with massive subcutaneous emphysema could, therefore, be due to intermittent cerebral air embolism.

Even if not large enough to block a tiny cerebral vessel, these small bubbles might stimulate cerebral vasospasm in similar fashion to the known effect of small (less than twenty micron) particulate emboli on cerebral or coronary vessels. The latter are associated with deleterious functional effects that can be eliminated by ultrafiltration of "intravenous" or radiographic contrast solutions with a less-than-one-micron filter. It is likely that in years to come, all fluids - at least those to be administered on the arterial side (cardioplegia solution, angiography dyes, fluids via left atrial pressure lines, and so on) will undergo routine ultrafiltration prior to administration, for the few particles that intravenous fluids and angiography dyes do contain are clearly unacceptable on the arterial side.

Subcutaneous Emphysema Overview:

Effective decompression at the source of air leak by a chest tube or "finger hole" can prevent or stabilize significant subcutaneous emphysema. This is not always possible. Mediastinal decompression may relieve cardiac tamponade symptoms due to a high pressure mediastinal air pocket. And

tracheotomy can reduce the expiratory pressure and thus pulmonary air leak of a patient who is breathing spontaneously. These secondary modes of treatment may be considered when symptoms and signs warrant.

However, if a massively inflated patient seems to be "doing poorly" for no apparent reason other than subcutaneous emphysema, one can embark on decompressive interventions of increasing magnitude (under appropriate local anesthesia). I would probably start with a chest wall skin incision over any broken rib (or perhaps with bilateral anterior chest wall incisions if there was no hint as to the location of the high pressure air pocket), and carry blunt finger dissection down through inflated chest wall tissues and between the ribs, hoping to decompress the air source by finger (and thereafter by chest tube if a larger pleural pocket was encountered, although this might simply confirm local obliteration of the pleural space).

Although a delirious ballooned-up patient, poked full of intermittently bubbling holes, represents a very uncommon manifestation of a very uncommon problem, this is still not a very satisfying picture; especially when "all those cuts" through grossly distended tissues "might get horribly infected." Fortunately, with reasonable cleanliness and modern antibacterials like povidone-iodine or antibiotic ointments, such open holes should rarely, if ever, become infected (small Penrose drains help to delay hole closure, and thus allow persistent air drainage through such cuts as long as this is needed).

As a final resort, I might consider (but have never performed for this indication) a sternotomy, or bilateral anterior submammary incisions (with or without transternal extension), in order to dissect both lungs free for leak closure, or at least create a free pleural space to allow tube decompression of any leaking bleb. One would anticipate massive postoperative lung air leaks following any such procedure on a severely emphysematous patient with obliterated pleura (who is the person most likely to suffer ongoing pulmonary air leak after injury): Extrapleural dissection hopefully could reduce such lung damage to a minimum, but it may be that satisfactory closure or free drainage of every pulmonary air leak will remain an elusive goal.

CHAPTER 6: PRACTICAL ASPECTS OF THORACENTESIS AND CLOSED THORACOSTOMY

Definition of thoracentesis;... Location of thoracentesis;... Avoid penetration of diaphragm during thoracentesis;... Significance of a bloody tap;... Case report: Thoracentesis with friends in the room;... Aspiration of hemothorax;... Accidental penetration into lung;... A Valsalva maneuver allows open-needle removal of intrapleural air; ... Aspiration of intrapleural fluid; ... Syringe technique; ... Comments on plastic needles with hollow metal stylets;... Thoracentesis of an effusion without using a syringe;... Follow thoracentesis with a roentgenogram;... Indications for closed thoracostomy;... A simple chest tube flutter valve;... Water seal;... Keep the chest bottle well below the patient;... Transporting an injured patient with chest bottle attached;... Larger air leaks demand better decompression, a review;... Dynamics of assisted ventilation with pulmonary air leak;... Controlled ventilation should eliminate inspiratory tidal;... A simple water seal permits normal respiratory pressure changes to empty the pleural space;... Self-prime of an empty water seal bottle;... Avoid back-pressure to chest tube drainage;... Dependent loops can kill;... Closed thoracostomy;... Chest tube size;... Misplacement and displacement of chest tubes;... Technique for closed thoracostomy;... Closed thoracostomy can be dangerous;... Insertion of a chest tube in the scar of a previous thoracostomy;... Closed thoracostomy in the newborn;... Case report: Neonatal death following thoracostomy;... Infant restraint and risk of aspiration;... Oversized water seal apparatus causes air lock in infants;... Outpatient treatment of spontaneous pneumothorax;... Outpatient thoracostomy instructions;... Discussion of outpatient thoracostomy;... Other advantages of outpatient thoracostomy.

Definition of Thoracentesis

Thoracentesis usually means penetration of the chest wall and parietal pleura by a hollow needle on a syringe, in order to aspirate fluid or air lodged in the pleural space. Rarely, thoracentesis also includes aspiration of a subpleural lung abscess, when inflammatory fusion of overlying pleural layers is anticipated. The thoracentesis needle is always directed just over a rib, to avoid injury to the intercostal vessels and nerve which lie along the upper edge of each interspace except near the midline posteriorly.

When present in large amount, pleural fluid may initially flow out from the open thoracentesis needle at some pressure above atmospheric: That pressure will be related to the height of intrapleural fluid above the needle penetration site, as well as any "push" or "pull" exerted through this fluid by the surrounding, perhaps displaced, tissues. As a rule, however, subatmospheric pressure must be applied to the outer end of the thoracentesis needle in order to maintain one-way outward flow of fluid or air through all phases of respiration.

The amount of suction required for such aspiration is related to needle size, effusion viscosity, friction between syringe barrel and plunger, tendency of lung to relax away from chest wall, inspiratory pressure, and so on. In addition, nearby tissues or precipitated proteins may obstruct the aspirating needle at any time.

Location of Thoracentesis

The exact site for aspiration is determined by chest roentgenograms in various projections, percussion and possibly computed tomography or sonography. One must remember that skin markers can move relative to the bony thorax during position change, especially in elderly or obese women. If fluid is free within the pleural cavity, the diaphragm level can often be estimated by tilting the patient away from the side to be aspirated, then percussing for diaphragm (actually subdiaphragmatic visceral) dullness. It is usually simplest to penetrate any hemithorax laterally (unless fluid is loculated

elsewhere), as less muscle is thereby traversed, and mediastinal structures remain well away from the exploring needle.

Avoid Penetration of Diaphragm During Thoracentesis

Aspiration low in the chest risks penetration of the diaphragm, which is often elevated when fluid and atelectasis are combined. Free intrapleural fluid usually obscures the diaphragm-lung "interface" that would normally be displayed on an upright chest roentgenogram (unless diaphragm and lung remain "stuck together" as a result of previous pleurisy).

Occasionally, the effusion will preferentially locate below lung, even though there are no adhesions between lung and chest wall. Perhaps such a lung is more resistant to compression from the side than from below (maybe it is "a little bit stiff" or emphysematous, or perhaps so pliable that it simply floats upward), but that is not always apparent on exploration. Some idea of diaphragm level can often be obtained from a lateral decubitus (AP or PA) roentgenogram of the chest (taken across the x-ray table with the uninvolved hemithorax down, to displace free intrapleural fluid onto mediastinum).

After successful pleural entry on a "low tap," one should angle the needle upward if it is to be advanced, to avoid subsequent penetration of a domed-up diaphragm. In general, a needle striking diaphragm will cause "referred" shoulder or neck pain (transmitted from peritoneal and pleural nerve endings). This indicates that the aspiration attempt was too low and should be relocated.

If such shoulder pain is associated with a return of blood which clots in the syringe, especially on a low left chest aspiration, the patient should be observed carefully for intraperitoneal bleeding from the spleen. Fortunately, simple puncture of the spleen is usually innocuous, although a simultaneous deep breath could conceivably cause major splenic laceration. When aspirating intrapleural pus, it is especially important not to tap so low that first chest cavity and then diaphragm are penetrated, for subphrenic abscess could easily result.

Significance of a Bloody Tap

It can sometimes be difficult to evaluate bloody pleural fluid obtained at thoracentesis, as minor amounts of blood may originate from intercostal vessels or muscles lacerated by needle. When aspirated fluid is grossly bloody, however, it is sometimes helpful to measure its hematocrit or wait for clotting, especially if one wishes to determine whether the tap was "traumatic" or if the pleural effusion really is "mostly blood" (blood that has been free in the pleural cavity for a time, usually becomes diluted, defibrinogenated and incoagulable. However, any high protein pleural effusion may tend to "gel" in the chest bottle as it cools to room temperature).

When bleeding is caused by a "traumatic tap" while aspirating a serous pleural effusion, the mixture of blood and effusion has a much lower hematocrit than the patient's own venous blood. Such "secondarily bloody" fluid is also likely to layer or vary in color as it is drawn from the chest. I have never seen clinically significant intrapleural or chest wall bleeding from a needle injury to intercostal vessels, except in the presence of gross clotting abnormality or when the huge collateral arteries bypassing a coarctation of the aorta were punctured.

Case Report: Thoracentesis With Friends in The Room:

During a three-month "refresher" fellowship in cardiac surgery, one patient (of Sicilian extraction) had a friend (the owner of "a little book store," as well as two pink Cadillacs) who never left his side, except to purchase all meals for the patient. At one point during his convalescence, the patient required thoracentesis for a bloody effusion. I briefly explained the proposed procedure as I prepared the syringe.

The indication for thoracentesis seemed to vanish, however, when the friend stood up, came close to me, and gently whispered, "Doc! If you hurt him, I'll get you, no matter where you go!." Our patient did not seem unhappy with my unexpected decision to cancel his procedure. Then, as I neared the door, his friend said "Look Doc, if you really think this is necessary, just go ahead. We all want him to get better, don't we?"

After a long pause, I said, "O.K., but you have to leave the room."

"Why?" the friend asked.

I said, "Because there's a lot of blood in there, and you might get sick."

The book store owner patiently explained, "Look Doc, I've seen more blood than you'll ever see! You just go on. I'll sit over here, out of the way."

So I performed my usual successful thoracentesis (perhaps a little heavy on the Xylocaine) and went to the showers.

Conclusion: (1) There are other hazards associated with thoracentesis besides bleeding and tension pneumothorax; (2) Italian "book store" owners sometimes see lots of blood; and (3) It is best to keep an open mind about allowing relatives or friends to remain in the room during minor procedures (even though they may occasionally faint and injure themselves).

Aspiration of Hemothorax

A stable, clotted hemothorax must liquify in order to flow out through any needle or chest tube. Sometimes it takes several days before roentgenographic signs of clot lysis (such as an increased volume of intrapleural "fluid," a fluid meniscus curving up alongside ribs within the hemithorax, or fluid displacement with position change) suggest that thoracentesis or thoracostomy can probably empty the pleural space efficiently.

As mentioned, however, an occasional effusion or hemothorax will preferentially layer entirely below the lung on the upright chest roentgenogram (thus no fluid meniscus, and this appearance is easily confused with elevation of the diaphragm). Breathing exercises are also effective, and often seem a more appropriate method for the elimination of an effusion or hemothorax (see Chapter 3).

Accidental Penetration Into Lung

Needle penetration into lung during thoracentesis is common and usually harmless. Lung puncture is often signaled by return of small amounts of pinkish froth through the needle during pull on the plunger. Needle withdrawal from normal lung is usually followed by prompt collapse of any injured alveoli and little further air leak. However, an occasional patient (with emphysema, or on assisted

ventilation) will continue to pump air from the needle hole and develop simple or tension pneumothorax.

Iatrogenic air leak (due to needle puncture of lung) is evaluated and treated just as any other pneumothorax. When general anesthesia or other positive-pressure assisted ventilation is soon to follow, any detectable pneumothorax after lung injury is usually treated by chest tube: It is simpler and safer to place a chest tube electively than to risk having the lung air leak resume insidiously during the assisted ventilation of an anesthetized or sedated patient.

However, one must avoid placing a chest tube entirely prophylactically, for lung is easily injured when there is postinflammatory fusion of the overlying pleural layers. Such fusion clearly does not exist when free intrapleural air or fluid have separated the pleurae in that part of the chest. In other words, "no pleural space" means "no chest tube," for otherwise the tube could end up spearing lung that is stuck to the chest wall.

A Valsalva Maneuver Allows Open-needle Removal of Intrapleural Air

This technique can be used after spontaneous pneumothorax, provided the air leak has most likely sealed and the intrapleural volume of free air justifies such removal (see Chapter 5).

Materials:

1) Povidone-iodine solution (plus a razor if the chest is hairy).

2) 5 to 10 cc 1% Xylocaine without epinephrine, plus a small syringe with a No. 18 needle to draw Xylocaine into the syringe, a short No. 25 or No. 27 needle to create a skin wheal, and a 5 cm, No. 23 needle for deeper infiltration.

3) A current PA roentgenogram of the chest (preferably taken within the past hour or two).

4) Sterile gloves and sponges.

5) A No. 14 or No. 16 plastic intravenous needle (with the plastic outer cannula on a hollow metal needle stylet).

6) A pointed (No. 11) scalpel blade.

Method: With the sitting patient leaning back comfortably (so that lung falls away from the proposed puncture site), raise an intradermal wheal over the upper border of the third rib at about the midclavicular line (unless there are nearby interpleural adhesions). Creation of a skin wheal is painless if Xylocaine is injected slowly and continuously as the needle is inserted (with its bevel down). The dimpled raised skin wheal thereafter identifies the "numbed" site, to allow painless needle reinsertion.

Following skin anesthesia, periosteum and pleura (the other pain-sensitive areas of the chest wall) are blocked in a fan-like distribution by multiple, short "needle passes" that first touch the upper border of the third rib, then proceed just over this rib for an additional 2 to 4 mm (all the while injecting 1% Xylocaine). There is no danger to constant anesthetic injection without first aspirating in each new position, provided the needle tip is kept in motion, for accidental intravascular injection by this

technique will be insignificant (even if epinephrine has been added to the local anesthetic). To confirm the diagnosis and proper needle placement, free air is then aspirated from the pleural space before the slim (No. 23) anesthetic needle is finally withdrawn.

Now sterile gloves are put on, the area receives another Betadine wipe, and the skin wheal is punctured with a sharp blade to ease passage of the plastic cannula. An open No. 14 needle is inserted along the previously anesthetized tract (through the lower aspect of the second interspace) into the pleural space. The inner metal needle can then be removed (or its sharp tip is simply retracted until shielded within plastic, if it appears that heavy chest wall muscles might otherwise kink the unsupported cannula).

The patient is told "Please bear down": Air then hisses out through the plastic needle for a few seconds until a sterile, gloved fingertip covers the needle hub to prevent air return while the patient relaxes and breathes normally (for during inspiration, intrapleural pressures remain below atmospheric). Several gentle Valsalva maneuvers, with intervening "rest periods" during which the needle is again blocked, soon remove all accessible air (unless air leak persists or resumes).

As expanding lung blocks the plastic cannula tip, hissing becomes intermittent and finally ceases. At this point the patient may note temporary pleuritic discomfort. The open cannula is slowly withdrawn during a final gentle Valsalva maneuver, and the patient is then sent to the hospital coffee shop. (If one prefers to remove intrapleural air with a glass syringe, one can diminish air leak between plunger and syringe barrel by first moistening the plunger with local anesthetic solution or saline.)

Although complete lung reexpansion by thoracentesis is unusual, repeat chest roentgenogram (one-half hour or more after air removal) should reveal a diminished pneumothorax: The remaining free air normally absorbs uneventfully, as can be documented by subsequent roentgenograms. Follow-up chest roentgenograms should duplicate the original technique and position in order to properly document any change, for any intrapleural air collection will appear larger when displayed against a smaller hemithorax (as on an "expiratory film").

Comment: Open-needle Valsalva venting of intrapleural air is usually successful in appropriately selected patients. With recent onset, or major, or increasing pneumothorax, a chest tube usually seems more appropriate than attempted needle aspiration (provided air removal is warranted). If a minor pneumothorax persists or recurs shortly after aspiration, the air leak probably resumed as lung was reexpanded (in which case, leave it alone and wait for healing). Only when there is significant bacterial contamination of the pleural space from underlying lung infection, is it important to achieve early complete obliteration of a small pleural air space by frequent needle aspirations or, preferably, by continuous chest tube drainage (see Chapter 4).

Aspiration of Intrapleural Fluid

Intrapleural fluid may be aspirated for diagnostic or therapeutic purposes: A recent chest roentgenogram should usually be at hand. If only a small fluid sample is required, this can be obtained in the small syringe being used for local anesthesia infiltration. To withdraw larger volumes of fluid, materials as for air removal are required, plus the following:

1) An aspirating syringe (20 to 50cc),

2) A three-way stopcock, and

3) Drainage tubing, small specimen tubes, and a sterile basin or bottle of adequate size.

Procedure: A midaxillary or posterolateral tap (fifth through eighth interspace, with the patient sitting) will usually provide access to any significant volumes of free intrapleural fluid. The patient's feet hang over the edge of the bed while he rests his elbows on the bedside table (unless very weak, in which case a lateral tap is easily performed with the head of the bed cranked up and the patient brought to the edge of the mattress).

As always, the final step of local anesthetic infiltration includes attempted aspiration. Free air or thin fluid are easily withdrawn to document appropriate needle placement, before substituting a larger (No. 14 to No. 18) steel needle or plastic cannula for definitive aspiration.

Syringe Technique

The "pull" required on the plunger of a glass syringe to maintain slow aspiration is proportional to the subatmospheric pressure between lung and chest wall (provided the needle is adequate in size and the fluid not excessively thick). When the plunger is released, therefore, syringe contents often tend to be drawn back into the chest.

A three-way stopcock (interposed between needle and syringe) permits intermittent dumping of syringe contents without admission of air to the pleural space (as would occur via a temporarily open needle if the syringe had to be disconnected each time for emptying: Such atmospheric entry into the pleural space would then interfere with lung reexpansion).

Before inserting an aspiration needle into the chest, it is well to see that the stopcock works properly and the plunger fits the syringe barrel. A short practice with a three-way stopcock is worthwhile, to ascertain which way does what. Fluid is occasionally pumped back into the chest when a stopcock valve is turned the wrong way: Air may also enter the pleural space through a loose connection.

If either problem occurs while using proper sterile technique, no harm should result: Accidentally returned fluid is simply drawn out again, while only a rare misadventure would introduce enough air to warrant efforts at its removal. Air that does enter the pleural cavity during thoracentesis will usually move up toward the apex of the sitting patient, however: Vigorous attempts to retrieve this free air could certainly result in unintentional needle puncture of the more-likely-to-be-emphysematous upper lung (which must somewhat increase the risk of persistent air leak, although I have not encountered that complication under these circumstances).

The more likely problem in such a case, is that any open-needle Valsalva effort undertaken to remove such "inadvertent air" will simply increase the pleural space air accumulation: This is especially likely if a troublesome apical air collection has occurred during removal of a large pleural effusion, and if the previously relaxed, compressed, restricted, or surgically diminished lung was significantly "stretched" by removal of all that fluid: Unlike normally expansile lung, such an "already stretched" lung may "recoil" during any subsequent open-needle Valsalva maneuver.

Under these special circumstances, only a cough may momentarily raise "peak" intrapleural air

pressures above atmospheric, while any moderate, well-tolerated, Valsalva effort could not even achieve atmospheric pressure within the pleural cavity. Thus an intrapleural chest tube attached to water seal would still allow "cough pressure peaks" to blow-off intrapleural air and thereby complete the lung expansion (perhaps skillful "fingering" of the intrapleural needle during cough could achieve the same goal, but I have simply avoided open-needle removal of air from around restricted lung, and either rely upon the air soon being absorbed from the sterile pleural space or else insert a chest tube).

In any case, near-completion of air or fluid removal during routine thoracentesis is usually signalled by an "increasing pull" required on the syringe plunger, and also by intermittent blockage of the aspirating needle on each inspiration (as the remaining rim of air or fluid about the lung is spread more thinly over a larger surface when the chest cavity expands). Because this intermittent obstruction is usually due to lung contacting the needle tip, even more care must now be taken to avoid lung injury: This is especially the case if using a sharp, steel needle and planning further pleural space evacuation. Incidentally, needle contact with visceral pleura often induces an involuntary cough, for visceral pleura is not "blocked" by any amount of local anesthetic injected into the chest wall.

Note: The fact that intrapleural pressure around a very stretched lung only exceeds atmospheric during a strong cough does not mean that the lung cannot exhale effectively, for regardless of how much tension there is between visceral and parietal pleurae, or how expanded or contracted the chest cage, intrapulmonary air pressure soon becomes atmospheric when lung parenchyma communicates with an open airway: From that stable position, intrapulmonary pressures then move above and below atmospheric in response to changes in the size of the hemithorax and its obediently following lung (until air once again stops entering or leaving the airway because intrapulmonary pressure has returned to atmospheric).

Comments on Plastic Needles With Hollow Metal Stylets

The blunt plastic intravenous cannula, introduced over a hollow steel stylet in the fashion described for air removal, is also preferable when complete pleural fluid evacuation is intended. Significant air entry can be prevented after stylet withdrawal by occluding the needle hub with a gloved fingertip until the sterile aspirating syringe is reattached.

If the hollow metal stylet is left within the plastic outer sheath in order to stiffen it, the diminished needle lumen will retard fluid removal. Another disadvantage of leaving a partially withdrawn metal needle within its plastic outer sheath is the possibility of slow air leak into the pleural space between inner and outer cannulae.

A metal stylet is never advanced into the outer plastic cannula while both are in the chest wall: The needle is only replaced within its cannula after complete removal of both the steel and plastic portions from the patient, to avoid leaving a cut-off plastic cannula tip within the chest wall.

Thoracentesis of an Effusion Without Using a Syringe

Sterile intravenous tubing, attached directly to the thoracentesis needle, can often siphon large pleural effusions down into a container placed on the floor: A gentle Valsalva maneuver by the patient may help to start this flow. As long as the lower (container) end of this tubing is then kept submerged, air cannot reenter the tubing (this setup thus serves as a "simple water seal" device).

However, a chest tube with multiple side-holes is far less likely to become occluded by intrapleural tissues or minor debris than a needle. One advantage of a syringe for aspiration (over the needle-to-intravenous-tubing siphon), is that the syringe can readily blow the needle tip free of debris. A glass vacuum bottle (of the type formerly standard for blood donation) can also be convenient for withdrawing larger quantities of fluid from the chest, as it similarly eliminates tedious pumping of the syringe.

Follow Thoracentesis with a Roentgenogram

Several thoracenteses may be required to complete the evacuation of a loculated pleural effusion. It is usually helpful to take repeat chest roentgenograms between thoracenteses. A film taken soon before aspiration is essential, as the entire configuration can change in a remarkably short time. Complete aspiration of a pleural effusion from the patient in cardiac failure or with tuberculosis or tumor is often unnecessary, as vigorous treatment of the basic disease frequently results in resorption of fluid. Chest roentgenogram after a "chest tap" may also reveal a significant, unsuspected, iatrogenic pneumothorax.

Indications for Closed Thoracostomy

Closed thoracostomy permits continuous thoracentesis by a chest tube instead of by needle. Sites for elective tube placement are similar to those for thoracentesis. Tube thoracostomy is generally indicated for fluid too thick to flow through a needle, or when air or fluid is likely to reaccumulate.

A chest tube is commonly inserted to drain a spontaneous or post-traumatic pneumothorax, or to treat subacute or chronic pleural space infections (where response to antibiotics will not be immediate and fluid accumulation cannot conveniently be controlled by multiple thoracenteses) and at thoracotomy or after other injury (when ongoing air leak or persistent bloody or serous fluid accumulation can be anticipated from damaged surfaces). In questionable instances, it is often reasonable to insert a chest tube prophylactically into the free pleural space. Risk of infection from such intubation is essentially nil, and the tube can soon be removed if it proves unnecessary.

A Simple Chest Tube Flutter Valve

The properly located chest tube will decompress tension pneumothorax: It also allows intrapleural fluid and air to escape during expiration (while pleural space pressures are above atmospheric). If return flow can then be prevented during inspiration, rapid reexpansion of lung should follow (unless significant air leak continuously replenishes the pneumothorax).

A low-resistance one-way valve can be fabricated by tying a slit condom or balloon over the external opening of a chest catheter. This will permit easy air and fluid egress, yet collapse and block the chest tube on inspiration.

Water Seal

Such an emergency flutter valve arrangement is messy, unsterile and somewhat unreliable (as the slit could be held open by clots, thereby allowing to-and-fro air movement). A neater method for establishing reliable one-way flow is to lengthen the chest tube and barely submerge its open end in sterile physiologic salt solution. With this "water seal" arrangement, intrapleural free air and fluid can easily bubble and flow from the submerged chest tube tip on expiration, but "room" air cannot reenter

the tube tip when intrapleural pressure becomes subatmospheric (so inspiration only lifts saline a few centimeters up the tubing. This upward movement of fluid during each inspiration is referred to as "tidal" within the water-sealed tubing).

For neatness and sterility, the water seal can be enclosed in a bottle with a large air vent (Fig. 1). Although a "skinny" bottle would serve for a small child or minor air leak, a larger-volume bottle is preferred following thoracotomy (since significant air and fluid drainage may occur). The clear glass or plastic water seal straw (a rigid continuation of the chest tubing) is suspended from the well-vented water seal container top, so that the straw tip is initially stabilized one or two centimeters below the water seal surface.

If the air vent extends into the chest bottle for an appropriate distance, this allows a temporarily inverted water seal bottle to drain excess fluid from its air vent without loss of the fluid prime (Figure 6). Chest bottle "prime" refers to that minimum fluid volume necessary to guarantee that the water seal straw tip will remain submerged in an upright bottle, even after a maximum inspiratory effort has drawn fluid well up into the chest tubing: With appropriate-size tubing and allowing for a safety margin, at least 200 cc of saline will be required (if the container also has a rounded bottom).

Keep Chest Bottle Well Below The Patient

The water seal bottle should be kept well below the patient (on or near the floor), for otherwise a deep inspiratory effort might draw water seal saline up into the patient's pleural space. Although this should not occur, it is easily remedied; merely place the bottle on the floor and have the patient cough or "bear down" gently for a few seconds. If that maneuver does not immediately bring the fluid "prime" back to its bottle from the pleural space, one can apply strong suction momentarily to the chest bottle air vent, or simply reprime the water seal bottle with additional saline: The original water seal saline should soon return to the reprimed water seal bottle (or else be absorbed by the pleura).

Unless pus is being drained from an infected pleural space, chest bottle contents are best kept sterile. In addition, nothing irritating or potentially harmful should be added to the water seal bottle. One never primes a water seal bottle with distilled water, for example, as distilled water refluxing onto pleura is extremely irritating (and in large amounts it might even cause hemolysis, although I have not heard of that happening under these circumstances).

Transporting An Injured Patient with Chest Bottle Attached

On occasion, a patient with pulmonary air leak must be transported on the floor of a vehicle, with his chest and the water seal bottle therefore at the same level. Depending upon respiratory efforts, air leak and bottle height, the water seal prime might well be pulled into the pleural space for the duration of the trip. This does not mean, of course, that the chest tube should be clamped, for that could cause tension pneumothorax, which is far more dangerous than the absence of a water seal.

When a water seal system loses its prime, it functionally becomes a lengthy open chest tube. The consequent relaxation of lung, and to-and-fro movement of air within this long chest tube, simply decrease breathing efficiency. If such inefficient breathing is poorly tolerated despite supplemental oxygen, then a flutter valve (even an unsterile, slit condom or balloon) or some "safe" chest bottle suction system should be considered.

If these are not readily available, one can delegate an individual with a good sense of rhythm to compress the chest tubing during inspiration and leave it open on expiration; lung is reexpanded just as quickly in this fashion as by a water seal or flutter valve. (This is mostly a "theoretical-review problem" anyhow, as any significant pulmonary air leak will usually prevent a high inspiratory tidal from developing.)

Practical Aspects of Thoracentesis
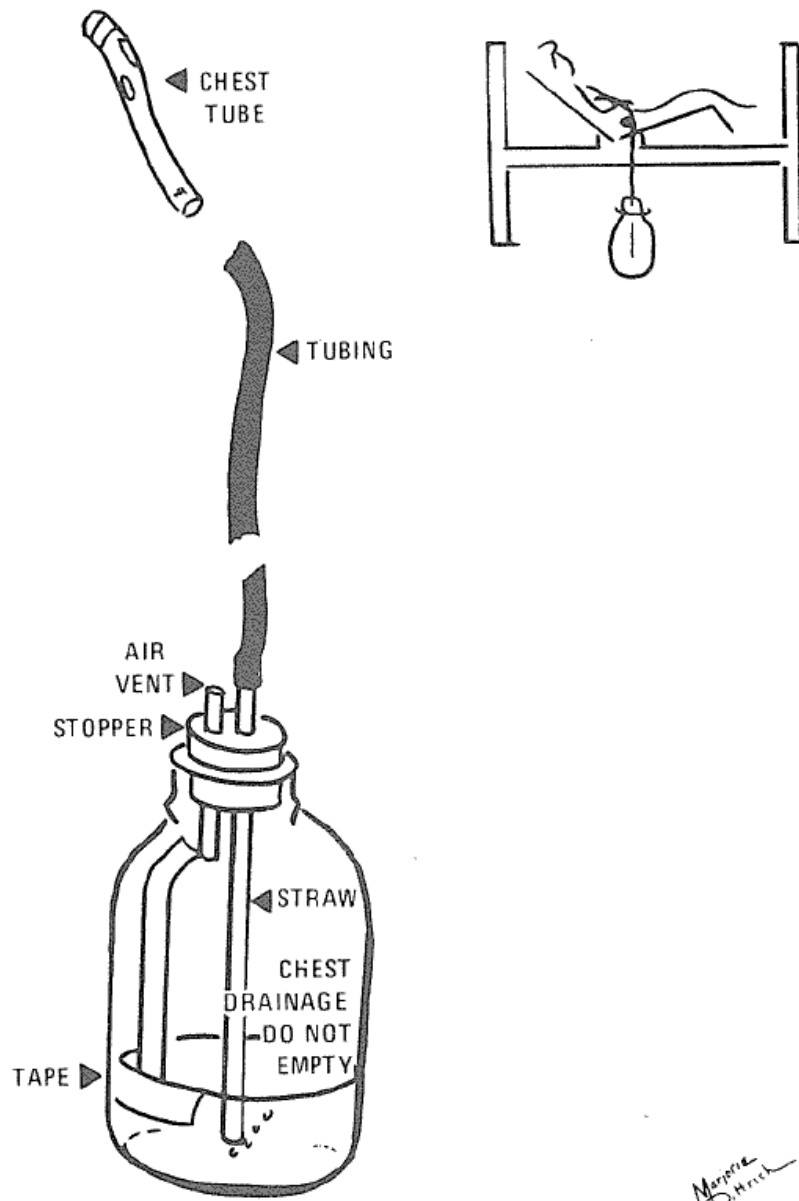
Figure 1.

Larger Air Leaks Demand Better Decompression: A Review

A water seal bottle that bubbles continuously (through inspiration as well as expiration) is both "good news and bad news." The good news is that a one-way valve need not be connected to the chest tube as long as pressure within the pleural space always exceeds atmospheric. The bad news is that the patient cannot utilize the underlying lung during spontaneous breathing.

When lung that is free within the pleural cavity is subjected to intrapleural pressures that remain above atmospheric during unassisted inspiration, it is no longer available for air exchange (a condition also known as "tension pneumothorax"). On the other hand, even higher and continuously elevated pressures within a small intrapleural air pocket may have little effect on cardiac or respiratory function (although this could easily lead to subcutaneous emphysema).

Continuously above-atmospheric (or positive) pressure within any pleural air space simply documents inadequate decompression of an underlying pulmonary air leak to the atmosphere. Normally, the air pressure within such a pocket will not exceed peak airway pressures generated during a Valsalva effort or cough (or the maximum pressure reached in the course of positive-pressure ventilatory assistance).

Appropriate therapy of such "excessive" air leak can include; (1) better drainage (insertion of more or larger chest tubes, better tube placement, application of "high" chest bottle suction); (2) a surgical effort to close off the air leak; or (3) attempts to reduce the mean airway pressure (such as discontinuing positive-pressure ventilation if the patient can breathe on his own, or even performing a tracheotomy for the truly marginal case, in order to reduce expiratory pressures during spontaneous respiration. (An external abdominal respirator should also help to lower airway pressures and thus reduce a massive air leak, but I have not tried that.)

Dynamics of Assisted Ventilation With Pulmonary Air Leak

Positive-pressure ventilation may improve pulmonary air exchange in a critically-ill patient with a chest tube and moderate air leak. On the other hand, tension pneumothorax can result if the air leak is markedly increased by such assisted ventilation (this can occur despite the presence of a functioning and previously "sufficient" chest tube). In fact, "useful" assisted ventilation may be difficult or impossible to deliver in the presence of very large pulmonary air leaks. (In theory, "high frequency" or single lung ventilation might favorably affect these adverse ventilatory dynamics; see also Chapter 5.)

Controlled Ventilation Should Eliminate Inspiratory Tidal

With positive-pressure fully controlled ventilation, the patient's lungs are intermittently pumped full of air by the respirator, then allowed to deflate passively to the atmosphere. Under these circumstances, inspiratory and expiratory intrapleural pressures both remain well above atmospheric, so chest tubes can only drain in one direction (from the continuously above-atmospheric pleural space out to the atmosphere): chest tubing is then left connected to a water seal device mostly to assure convenient fluid collection and sterility. An empty (unprimed) chest bottle would serve these functions equally well, as the one-way valve function is not needed, but one would then lose "bubbling at the water seal straw" as a visible reminder of air leak.

The regular upward movement of fluid in the water seal straw known as "tidal" simply reflects

each subatmospheric inspiratory effort. Thus tidal is not seen while breathing remains controlled on a respirator unless (1) serious respirator malfunction or airway blockage stimulates the increasingly anoxic patient to make major inspiratory efforts or (2) the patient requires additional sedation to allow him to "cooperate" more comfortably with the respirator. Unfortunately, mistaking (1) for (2) is not an uncommon cause of death in critically ill patients.

While it is common for an uncomfortable patient to cough or "gag" on the endotracheal tube (or an associated oral airway) during adequate ventilation, one must always assume that the respirator is "guilty until proven innocent" (so check volumes exchanged, listen to lungs, recheck endotracheal tube position and airway patency, get new blood gases, and in the meanwhile, ventilate the patient by a hand-held ventilation bag if not entirely confident of ventilator adequacy). To sedate a patient into "cooperating" with a defective respirator (or dislodged endotracheal tube, or trachea full of mucus, or whatever) merely adds the patient to that long list of those who are unaware of or unconcerned with his impending death by suffocation.

A Simple Water Seal Permits Normal Respiratory Pressure Changes to Empty the Pleural Space

Any patient strong enough to breathe and cough effectively will readily evacuate air or fluid from the pleural space through a properly located chest tube connected to an adequately designed and correctly utilized water seal bottle. As a bonus, fluid fluctuation in the water seal straw serves as a manometer of intrapleural pressure changes. An occasional glance at this manometer keeps the observer informed about respiratory effort, possible airway obstruction, blocked or improperly located chest tubes, atelectasis, air leak and much more.

Self-Prime of An Empty Water Seal Bottle

Following closed thoracostomy, a large pleural effusion may initially flow unassisted from the chest tube without "hesitating" or reversing direction during inspiration. This indicates that the intra-pleural fluid height above the tap site, plus any displacement pressure, exceeds the subatmospheric inspiratory pressure currently being generated.

As the pleural fluid volume then diminishes, drainage will be maintained by the weight and cohesion of the enclosed, downwardly moving column of liquid (commonly referred to as a "siphon"). This intact fluid column will exert increasing pull on the pleural space as lung gradually resists further expansion and mediastinum, diaphragm and chest wall are drawn together (after having been pushed apart by the effusion).

When the pleural space being drained of a large effusion does not close readily, excessive intrapleural suction can develop that may be harmful as well as painful. Fortunately, the siphon can easily be stopped by finger-compression of the chest tubing (unless the tubing is excessively thick-walled or rigid): Release of tubing compression then allows drainage to resume.

One may also choose to break the siphon by allowing air entry into the upper chest tubing with a hollow needle (one can then "lower" the no-longer-moving fluid column until its height in centimeters provides an appropriate suction setting). However, if much pleural fluid still remains when the siphon is thus "broken", siphon flow may spontaneously resume despite this newly established air lock, for that intervening air is readily carried distally if vigorous fluid drainage is reinstituted by a strong cough or other Valsalva-type exertion.

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An enclosed, downward-flowing column of fluid will exert suction above and pressure below. If it stops flowing suddenly, it exerts an additional momentary suction or pressure pulse that is related to its mass and deceleration. A tall, hanging column of fluid exerts more suction than a downward flowing, liquid column of similar height (for the latter is only partially supported).

In any case, a large effusion and effective siphon make it unnecessary to prime the water seal chest bottle; simply allow direct flow into an empty water seal unit and the straw tip is soon submerged. Some high-protein or very bloody pleural effusions may form a soft gel in the bottle, but such a gel only resists air escape slightly more than saline, and it is equally effective in maintaining unidirectional flow.

Avoid Back-Pressure to Chest Tube Drainage

Big air leaks from lung are uncommon: Unfortunately, many commonly used chest drainage devices become obstructive in the face of large air flows from the chest tube (which means they work "just fine" until really needed). Any part of the chest drainage system may cause increased resistance to air escape from the chest tube: For example, a blocked (or unduly narrow) chest bottle air vent can permit build-up of air pressure within the water seal bottle, and thus also within the pleural space.

If an air leak averaging 5 liters/minute must be evacuated, then a chest tube and drainage system capable of passing 10 liters/minute at physiologic pressures, will briefly but regularly become obstructive to peak expiratory flows of possibly 20 liters/minute. Even such momentary back-pressure diminishes respiratory efficiency.

Dependent Loops Can Kill:

Fluid in a dependent loop of tubing will create back-pressure to air leak. A 50 cm tall fluid column trapped in a hanging loop of tubing causes the same back-pressure to air escape as a water seal straw tip submerged 50 cm below the fluid surface in a water seal drainage bottle (Fig. 2): Obviously, 50 cm back-pressure to air escape from either cause can lead to severe tension pneumothorax.
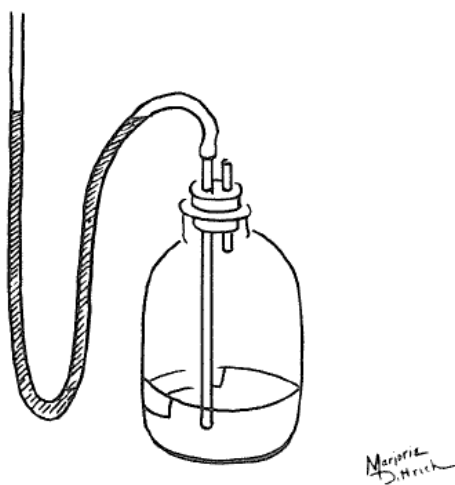
Figure 2.
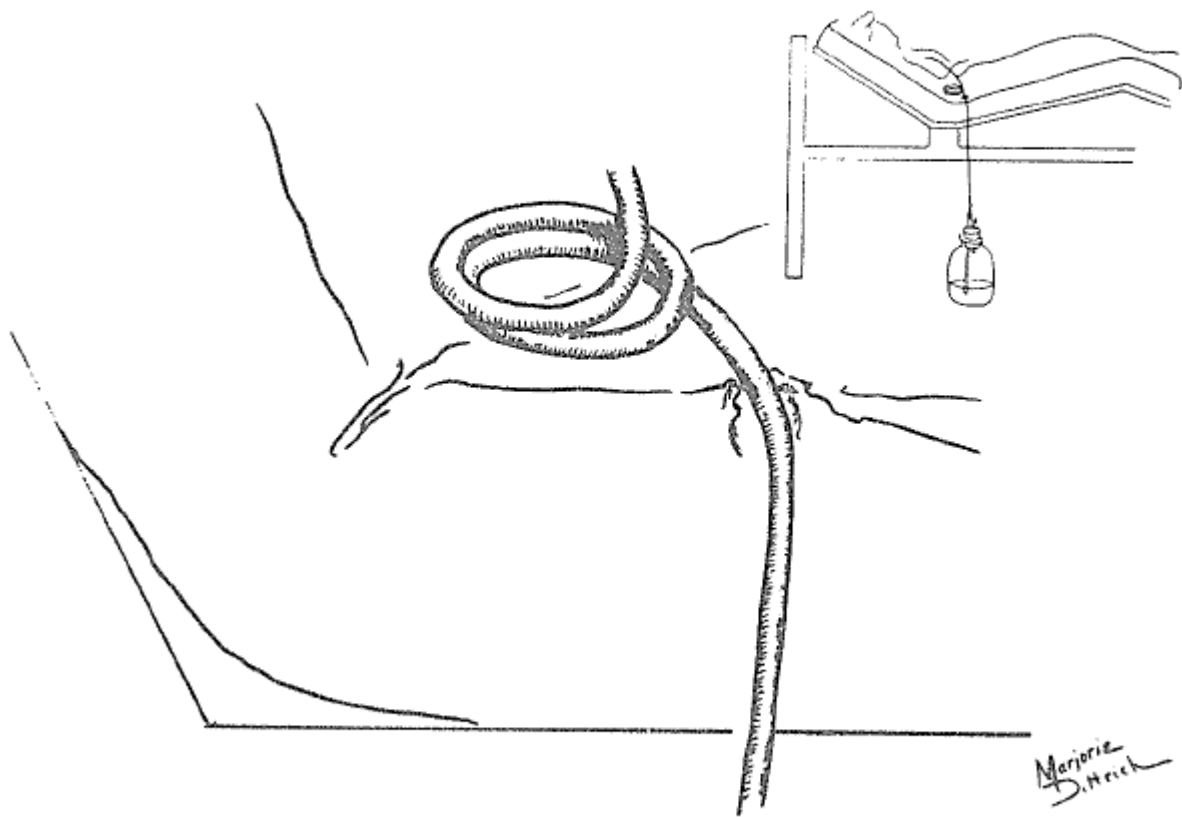
Figure 3.

Under these circumstances, dramatic relief is afforded by merely coiling redundant tubing flat on the bed so that it leads directly down to the bottle (Fig. 3), or by decanting excess drainage from the chest bottle (or replacing the full bottle with a more appropriately primed unit). In fact, simply disconnecting the chest tube from the chest drainage system, so that it is left open to the atmosphere, is far safer for the patient than the tension pneumothorax so easily caused by fluid-filled dependent loops of chest tubing or a "too full" chest bottle when there is pulmonary air leak .

The apparently simple way to "eliminate dependent loops forever" by just shortening the chest tubing is unsatisfactory, as the patient cannot then move about in the bed while still keeping the chest bottle on the floor. In addition, a shortened or tied-down tubing may soon jerk the chest tube from the chest. An unfastened loop or two of excess tubing placed upon the bed is usually most satisfactory, especially with the slightly "tacky," easy to coil, disposable, clear plastic tubing now available.

Closed Thoracostomy

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"Closed thoracostomy" means insertion of a chest tube into a closed hemithorax (in other words, not during thoracotomy). Although one could refer to chest tube insertion during thoracotomy as "open thoracostomy," it is more usually referred to as "intraoperative chest tube placement" or something like that. A chest tube should be inserted through the anterior or lateral chest wall whenever possible, so the patient can lean back without being poked by or blocking the tube.

The tunnel (tube path) created during tube insertion through the chest wall, serves to guide and support the crucial intrapleural portion of the chest tube. One attempts to place the chest tube tip in an anterolateral location within the rib cage, barely below the first rib. This position usually prevents lung from "sealing over" the chest tube end and side holes until the apical pleural air space has been eliminated: Especially for air removal, the chest tube is always positioned so that it remains uppermost within the chest of a supine or semi-sitting postoperative patient.

In theory, any chest tube ought to be pushed into the absolute apex of the chest, to remove that very last bubble of air: Unfortunately, an apical tube position could injure delicate subclavian vessels and brachial nerves. In contrast, any low or posterior chest tube placement allows premature blockage of chest tube holes by gradually expanding lung: Although fluid drainage might still be satisfactory, a low or posteriorly placed tube cannot usefully drain air or even relieve tension pneumothorax.

The least thickly muscled areas of the chest wall are preferred for chest tube insertion sites. One tries to avoid chest tube placement through a major glandular portion of the female breast (the inframammary fold is sometimes a cosmetically acceptable tube insertion site; a small scar here is usually not visible). An anterior, second intercostal space, chest tube insertion usually traverses less muscle and is more comfortable than an "anterior axillary" tube location, for the latter penetrates thicker, more mobile muscle (which also increases the risk of tube displacement). It seems appropriate to avoid chest tube insertion near breast implants.

Overall, the second anterior interspace at roughly the mid-clavicular line is the simplest, safest site for rapid chest tube insertion through an intact chest wall: A tube entering just above the third rib in this location can safely drain both air and fluid.

Chest Tube Size

A closed thoracostomy tube should fit readily between the ribs and still be large enough to transport all anticipated drainage without causing back-pressure. Although it is often easier to maintain the patency of a larger tube, excessive tube capacity can be undesirable in children (see later this chapter). In practice, a No. 28 or No. 32 French chest tube is usually adequate for adults, while 16, 20 or 24 serve well in larger children; proportionately smaller sizes are required for infants and small children.

A No. 28 plastic chest tube is small enough to permit painless insertion into an adult under local anesthesia. I tend to utilize a No. 28 tube in essentially all adult outpatients requiring closed thoracostomy, and usually place one or more No. 32 chest tubes at thoracotomy. The more posterior tube (of two placed at thoracotomy) will primarily drain fluid and may reasonably be one or two sizes larger than the somewhat higher, anterior tube. Currently I prefer silastic chest tubes after chest surgery, as they seem less prone to clot.

Misplacement and Displacement of Chest Tubes

More than once, my perfectly positioned intrapleural plastic No. 20 or 24 French chest tube has later been found doubled up, kinked, and entirely within the chest wall, presumably as a result of chest wall muscle movements; small plastic chest catheters simply may not be stiff enough to hold a reliable course through adult chest wall muscles (and these smaller tubes frequently droop out of proper position once inside of the pleural space, as well).

It is difficult to hold any intrapleural chest tube in proper position when fastened to the loose skin of a newborn or cachectic patient, as any traction on the chest tube will easily "tent" the skin, thereby bringing the tube tip back into the chest wall. The prophylactic insertion of an additional length of chest tube at the time of thoracostomy may then be reasonable, but great care must always be taken in newborns to avoid any posterior drooping of a chest tube tip when placed to release all available air.

The chest tube tip above lung removes air until lung comes up to meet it (which is how a well-drained pleural space is usually eliminated). A chest tube misplaced beside or behind lung (or even between diaphragm and chest wall) quickly loses its capacity to drain air, due to collapse of surrounding tissues onto tube holes whenever intrathoracic pressure rises above atmospheric; yet such a tube still seems "wide open" to air being pushed in from outside of the chest during efforts to "clear the tube."

In the presence of ongoing pulmonary air leak, any posterior or inferior chest tube placement will allow an anterior intrapleural air pocket to accumulate (in a supine patient) at pressures persistently above atmospheric. If such a tension pneumothorax is strictly anterior in location, it may only be detectable on a lateral roentgenogram of the chest - a finding that has been referred to as pancake lung.

As might be expected, individuals with stiff lungs – e.g., newborns "in trouble," or seriously-ill emphysematous patients - who are most likely to develop such a problem, often tolerate it quite poorly: These supine patients are less likely to reveal an anterior tension pneumothorax on routine AP or PA roentgenogram because their stiff lung does not get much smaller than its surrounding pleural cavity even when relaxed (in addition, any air that reaches the side of the lung can still escape from the poorly positioned tube).

In contrast, as intrapleural air accumulates in an individual with normally pliable or "elastic" lung, it is soon announced by the increasing rim of air on AP or PA chest roentgenogram (for a relaxing lung normally becomes smaller in all dimensions). Under this more usual circumstance, significant intrapleural air accumulations commonly cause lung to shrink away from a poorly positioned chest tube as well, which may allow chest drainage to resume before lung can be compressed below its relaxed size (but a chest tube that remains beneath lung will never drain air effectively).

Summary: Poor chest tube placement can result in persistent simple pneumothorax or even tension pneumothorax. In the presence of stiff lung, an anterior air collection (due to undesirably posterior or lateral tube tip location) may cause severe distress or even death, without ever becoming apparent on the portable chest roentgenogram.

On the other hand, normal lung relaxes away from the chest wall during intrapleural air accumulation, so even an insignificant minor pneumothorax soon becomes apparent on AP roentgenogram: Normal lung also is more likely to relax away from a poorly positioned chest tube before tension pneumothorax can develop.

Undetected anterior air accumulations are uncommon except during the newborn period: They are most likely to become clinically significant in a persistently supine patient. (Presumably, posterior air pockets would cause similar difficulties in a persistently prone patient with pulmonary air leak and chest tubes located anteriorly).

Technique for Closed Thoracostomy

Materials include povidone-iodine, a syringe, needles, gloves and Xylocaine (as for thoracentesis), except that thoracostomy may require 10 cc (occasionally up to 15 cc) of 1% Xylocaine: Additional supplies include a No. 28 plastic chest tube, 2-0 monofilament plastic suture swaged onto a skin needle, scissors, a needle holder, tincture of benzoin, 4 x 4 gauze pads, cloth adhesive tape 2 to 3 cm in width, sterile saline to create a water seal, and an appropriate, preferably unbreakable and spillproof, chest drainage bottle with attached chest tubing. (We formerly resterilized one gallon cider jugs, but the hospital lost money processing those bottles for twenty-five dollars at a time when a gallon jug full of fresh cider could still be purchased for two dollars...).

Closed thoracostomy proceeds through the same initial steps as thoracentesis. All pain-sensitive portions of chest wall (skin, periosteum and pleura) are infiltrated with local anesthetic along the proposed tube path. Air (or fluid) is then aspirated from the chest (using a reasonably small needle on a syringe), both to confirm the adequacy of anesthesia and to avoid plunging a chest tube into the "wrong place."

A transverse (for minimal scar) skin incision is made within the skin wheal of local anesthetic until divided dermis separates easily to reveal fat. The "closed" Kelly hemostat is then driven into the pleural space along the course of the previously applied anesthetic (using a "to-and-fro" rotating motion), bumping over the upper border of the third rib before penetrating parietal pleura.

In a young or muscular individual with tough fascia, considerable pressure may be required to accomplish this blunt chest wall penetration by Kelly clamp. It is usually well to place the patient flat or leaning back in bed, therefore, so that he will not be pushed about. Although he may dislike the pressure required, the patient should have minimal pain, provided local anesthetic was properly placed.

The customary site of tube insertion can be altered when necessary (to get away from intrapleural adhesions or fractured ribs, for example). A broken rib cage tends to "give" and be pushed away during tube insertion, making pleural entry more difficult, as well as far more painful. Once the Kelly clamp has penetrated the pleura (keeping in mind the location of nearby structures such as diaphragm, heart, aorta and internal mammary vessels), it is opened slightly to gently dilate the hole (taking care thereafter not to close the clamp on lung tissues, by withdrawing the clamp somewhat as it is closed - this could be an unnecessary bit of advice, as I have never seen anyone "grab" lung in this situation).

When tension pneumothorax is present, a gush of blood-tinged air under pressure is released as the pleural space is entered and the hemostat blades spread; a conscious patient usually expresses immediate relief. The chest tube tip is now beveled and grasped in the hemostat, one blade of which is inserted through the tube tip side-hole (opposite the bevel), so that soft plastic chest tube tip precedes the hemostat points (this prevents snagging of tissues during tube insertion through the chest wall; see Fig. 4).

Hemostat and tube tip are driven in an anterolateral apical direction, and following its release from

the hemostat, the tube is advanced to position with all side-holes well within the pleural space. It is important to avoid chest tube erosion into vital structures such as brachial plexus, subclavian artery, aorta or esophagus. Therefore, one usually pulls the chest tube back "a little" when something stops its advance, prior to securing the tube in position. Anterolateral chest tube placement is best achieved while standing beside the patient. A surgeon standing at the head of the patient tends to direct the tube tip inferiorly and even medially, well below its optimal location.

Figure 4.

FOLD & STICK TOGETHER

1" ADHESIVE TAPE

CHEST TUBE

Figure 5.

Marjorie
Dittrich
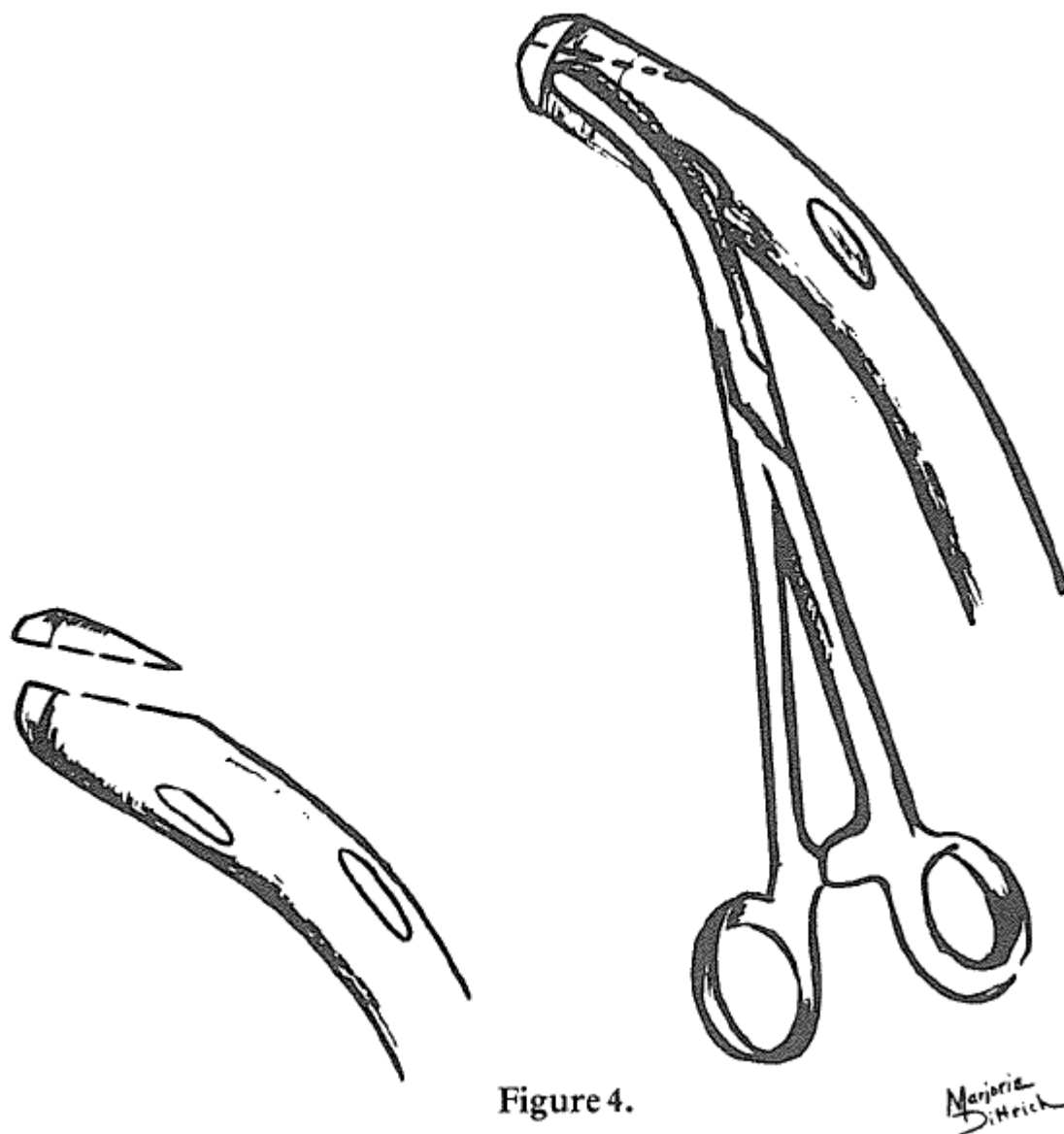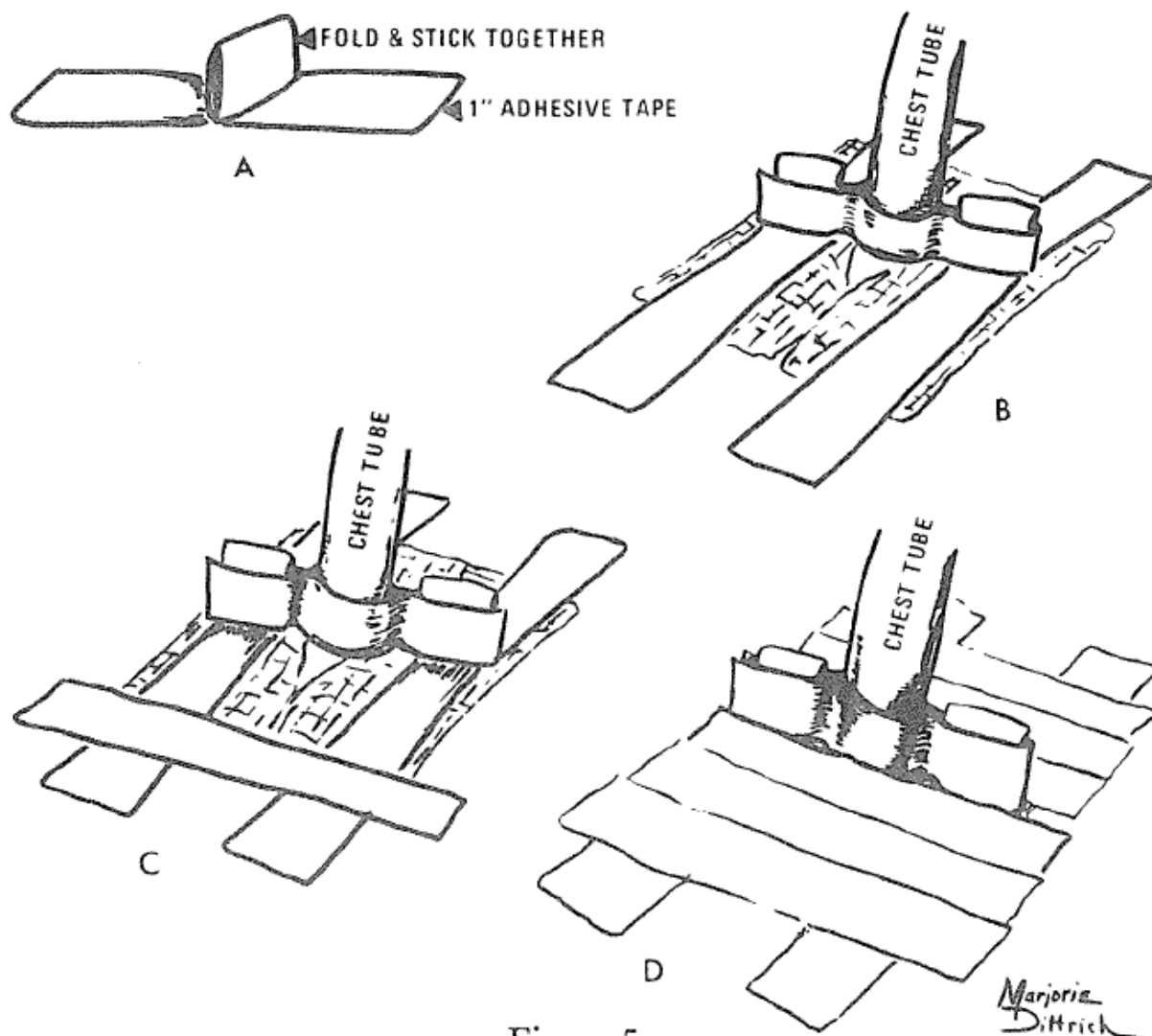
An open chest tube in the pleural cavity draws air in during inspiration and pushes air out with expiration. Except for a whistling sound, this does not visibly inconvenience a patient just relieved of tension pneumothorax. Thus there is no need for hasty hookup of the chest tube to water seal. Indeed, the patient's obvious relief and easy breathing despite that "open chest tube" should be drawn to the attention of anyone who still believes that an open chest tube is a life-threatening abomination.

Otherwise such an individual will one day "religiously" CLAMP! some poor patient's chest tubes for some antiquated reason like "moving the patient" or "frothing out of the chest bottle" and thereby bring on a stealthy ("l can't remember where I left my clamp?") - if not an immediate – life threatening tension pneumothorax.

When a chest tube is not marked along its length (or provided on a trocar), it is helpful to have another sterile tube available (or the original tube container held up nearby), to allow estimation of how much tube length has been pushed into the chest. If still unsure, the chest tube can usually be advanced an additional short distance through the rib cage, unless it meets resistance or causes pain. At this point,

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it is pulled back a centimeter or two (as mentioned, an "exactly correct" tube position is most critical in infants with ongoing air leak).

Rarely, in the presence of several rib fractures, the tip of a properly intrapleural chest tube will persistently deviate out into nearby chest wall through torn intercostal muscles. This position (with the tube tip end-hole outside of the parietal pleura and blocked) may have to be accepted as long as enough side holes remain unobstructed within the pleural cavity to permit good tidal and proper drainage.

Before or after connection to the chest tubing and water seal bottle, a chest tube can be advanced or withdrawn to assure optimal tidal and drainage. It is then secured by a monofilament skin suture (a loose loop of which is first knotted firmly above the skin, then the suture ends are tied back-and-forth tightly about the chest tube at skin level). A small, 5 cm. square, split-sponge dressing is then applied, after which the nearby tube and surrounding skin are moistened with benzoin. A secure method of taping the tube is essential (Fig. 5).

The patient is now instructed to cough, while bubbling is observed in the water seal bottle located on the floor. After an initial attempt to cough, the patient often develops a brief involuntary coughing spasm with pleural pain and copious expectoration (the standard signal of successful lung reexpansion). Satisfactory intrapleural tube position is suggested by a normal respiratory tidal of fluid in the water seal straw, and confirmed by chest roentgenogram.

Closed Thoracostomy Can Be Dangerous

One grey Sunday afternoon, while removing sutures during hospital rounds, I received a phone call from a general physician in a near-by town. He reported having just placed a chest tube in a young adult female who had stopped by the local hospital for evaluation of some bumped ribs: His parasternal chest tube insertion had been rewarded by copious bloody drainage.

During our brief phone consultation, her blood pressure became unobtainable, so I suggested cross matching six units of blood and dashed to the local airport for my airplane. Dodging a couple of rain showers enroute, I landed and was rushed to the hospital. There lay a deathly pale, non-responsive, 21 year-old female - obviously gasping her last: Her chest bottle was full of clot, and fresh blood was still draining through and around the left parasternal chest tube.

With local anesthesia unnecessary, the chest tube was removed and her parasternal incision enlarged to identify cleanly divided, still bleeding, internal mammary vessels. The experienced nurse controlled these vessels with finger pressure while I placed a saphenous vein cutdown and started transfusing the six pints of cross-matched blood.

While this blood was pumped in, her divided internal mammary vessels were secured with hemaclips and then by suture. An appropriately positioned chest tube was placed; the original tube site was closed loosely with a skin suture. By now the patient was awake and requesting a bed pan. Detailed instructions were left for chest tube removal and she did well.

Conclusions: (1) An unnecessary procedure by an untrained person is likely to be done poorly. (2) There are times when an airplane becomes a legitimate tax deduction.

Insertion of a Chest Tube in the Scar of a Previous Thoracostomy

When a patient presents with recurrent pneumothorax, one may prefer to use the previous thoracostomy site for tube reinsertion, either because it is properly located (unusual) or because another scar is undesirable. I often fight back the desire to show off my smaller, better scar and reinsert through the same skin location (if lateral roentgenogram shows lung not adherent to this area of the chest wall). However, local anesthesia, skin incision and drilling in by hemostat are all more difficult through such a scarred site.

Closed Thoracostomy in the Newborn

Anesthetic is not required, as newborns seem to feel little pain, and that only briefly at the moment of incision. (For saphenous vein cutdown at the ankle, local anesthetic is even contraindicated, as it is difficult enough to find and cannulate the little vein without adding distortion and edema by infiltrated anesthetic.) A minimal skin stab wound is made for chest tube placement, carefully avoiding the potential breast area of the newborn girl (a thoracostomy or thoracotomy scar near the nipple can severely distort later breast development). The small hemostat is bored and popped into the chest in the usual fashion, then blunt-tipped tubing with appropriate side-holes is thrust into the proper anterolateral position and secured (see pancake lung discussion above and also Chapter 5).

Case Report: Neonatal Death Following Thoracostomy

Beware of chest tube insertion in agonal infants for minor degrees of pneumothorax. I was once talked into placing a chest tube in a dying premature infant (with severe brain and lung damage), because prior resuscitation had caused minor to moderate pneumothorax. All physicians involved agreed that he was dying, but some insisted that a chest tube might improve him slightly (and perhaps "get them off the hook" for the pneumothorax). Anyhow, I placed an appropriately small tube in this apneic infant's pleural space (in my usual atraumatic fashion) and went home.

The following day, I was told he had died (as expected). Another day later I began to get funny looks from some of the pediatricians. Finally the pathologist gave me a call, and in his "I think there is something you should know" fashion, told me that the child had died with his chest full of blood because of my chest tube insertion: So I got credit for the "kill" (and learned that premature infants, especially if severely ill, may have a bleeding tendency, and that their dilated, anoxic vessels can continue to ooze after trauma).

Moral:

1) Not every pneumothorax requires a tube.

2) If intervention cannot correct the primary defect, let terminally bad cases be.

3) There is always enough blame to share.

Infant Restraint and Risk of Aspiration

Infant arms must usually be restrained while a chest tube is in place; even newborns are capable of pulling out a chest tube. Precautions may include padded elbow splints and pinning the child's sleeves to the sheet. An infant or small child should never be pinned out flat on his back, however, as this can lead to sudden death by aspiration of vomitus. Risk of aspiration must also be kept in mind when

initiating oral feedings following thoracotomy, but oral fluids are often well tolerated by infants within hours after routine chest surgery (start with a little sugar water before offering breast or formula feeding).

Oversized Water Seal Apparatus Causes "Air Lock" in Infants

A chest tubing and water seal straw that is inordinately large for the infant or small child can cause "air lock" and persistent pneumothorax. Any wider-than-necessary glass straw and tubing (one ancient straw that someone resurrected and used had a 1.5 cm internal diameter) has considerable volume capacity and can "damp" (render ineffective) the desired transfer of intrathoracic expiratory pressure through the tubing to the water seal (thus no bubbling despite free intrapleural air).

In this situation, vigorous spontaneous inspiration may only produce 1 to 3 cm upward fluid movement or "tidal" in the water seal straw: Even without air leak, the infant's pleural cavity cannot blow off or absorb enough air from the large tubing and straw (an intervening air space) to develop the upward tidal (subatmospheric intrapleural pressure) required to achieve full lung expansion.

This situation was easily rectified by replacement of the oversize unit with another having a narrower (pencil-sized) tubing and water seal straw; more recently we have simply aspirated air from the upper chest tubing – thus creating suction by lifting the water seal fluid column up in the straw and tubing (of course, a hanging fluid column descends if an air leak is present; external chest bottle suction may then be required).

Obliteration of the pleural space usually follows within hours after infant chest surgery, unless there is significant air leak, or the underlying lung is hypoplastic or surgically diminished. Thus, when the infant's chest tube is connected to simple water seal, it should soon show a high straw fluid level with minimal fluctuation.

Optimal tubing and straw diameter thus depend upon the patient's size, as well as the type and amount of fluid or air to be evacuated. Except with infants and small children, however, when the size of straw or tubing has presented a problem, they were always too small rather than too large. (Chest bottle suction can sometimes help compensate for an inadequately small drainage lumen by increasing the pressure gradient, and thus flow, along the tubing.)

Many chest surgeons do not routinely place chest tubes during infant thoracotomy. Some remove an intrapleural catheter on mild suction while lungs are still being inflated by the anesthesiologist at the completion of thoracotomy closure. Others use an appropriate-size water seal apparatus, with or without suction, or connect the chest tubing directly to suction.

In theory, simple water seal is safest: As a practical matter, however, an infant's lung seldom leaks air after lung surgery, and postoperative fluid drainage is normally insignificant, so the chest tube is placed mostly for the surgeon's peace of mind (or to be ready for the rare anastomotic leak after esophageal surgery). Of course, thoracotomy on an infant with bacterial contamination of the pleura, or infection in or near the pleural space, or a possibility of considerable postoperative lymph drainage, should usually be followed by careful and sometimes prolonged, chest tube drainage.

Outpatient Treatment of Spontaneous Pneumothorax

In a sixteen month period (1973-75), twelve emergency-room closed thoracostomies were performed for progressive, moderate to major, spontaneous pneumothorax. A similar number of patients seen with stable, minor to moderate pneumothorax were successfully managed by observation or needle aspiration alone. Two of the twelve patients undergoing chest tube insertion were admitted to the hospital, one directly to surgery for recurrent tension pneumothorax, and the other for in-hospital treatment of "associated conditions" (he was an attorney).

Nine of the ten remaining patients were sent home within two hours after chest tube insertion. (The first patient of this mini-series was hospitalized overnight before being discharged to care for his water seal chest bottle). Persistent air leaks eventually brought three of these ten outpatients back for wedge resection of the leaking pulmonary bleb and pleural abrasion (seven, eleven and fourteen days after tube insertion.) The average duration of air leak for the entire group of ten outpatients was seven days.

Following chest tube insertion, each patient (plus any relatives or friends) was sent on a trial expedition to the hospital coffee shop after brief instruction on the basics of simple water seal chest drainage. An occasional patient preferred to lie flat on his back for a few minutes first. Once stable and relatively comfortable, the patient was permitted to leave the hospital.

Outpatient Thoracostomy Instructions

1) Do not smoke.

2) Leave the bottle on or near the floor, so that water seal saline is not drawn up into your chest.

3) Observe the bottle occasionally for bubbling.

4) Keep the water seal bottle upright. (No harm is done if it falls over; merely return it to the functional position.)

5) Call the doctor's office daily and give a status report.

6) Return to the hospital emergency room for any significant problems.

7) Expect some pink or red drainage when the lung stops leaking air and becomes fully expanded (several patients called to report this "hemorrhage").

8) Make an appointment for chest tube removal the day after (7) occurs.

9) Resume activities as desired. (One bartender found his chest bottle a conversation piece; a lady patient took hers to a square dance in a stylish long-handled sack purse.)

10) Pour 2 to 3 teaspoons of table salt into the air vent if outside in freezing weather.

11) Take no pain medication if planning to drive.

Patients were given prescriptions for ten or twenty codeine (30-60 mg.) plus aspirin tablets, and rarely some Valium (but they usually received no oral or intramuscular analgesics prior to discharge).

Some insisted on driving themselves home, although a ride was recommended or occasionally provided.

Discussion of Outpatient Thoracostomy

No antibiotics were used. The three "complications" (persistent air leak requiring surgery) could not be attributed to home care of the chest drainage system, for an identical, simple water seal (primed with sterile physiologic salt solution) was used during home or hospital care. Outpatient thoracostomy (as described) has been shown cost-effective, essentially trouble-free and is now utilized by other Anchorage surgeons.

However, one cannot discharge a patient attached to one of those "tippy," complex, flimsy units that require some form of mechanical suction in order to function at all effectively. It is important that the patient "call in" daily so possible problems can be discussed and the chest tube removed when the appropriate time comes. If the patient lives in another town, effective follow-up and tube removal can often be provided by his own general physician.

Other Advantages of Outpatient Thoracostomy

Compared to in-hospital treatment, the chest bottle never got emptied by mistake, the chest tubing was never clamped, and there were no medication errors or other midnight calls to report slight changes in bubble size or shape. All patients were surprised and grateful that chest tube "home care" was possible. Total medical costs ranged from $150 to $250, unless subsequent thoracotomy was required.

If, as seems apparent, the course of healing after chest tube insertion was unchanged by outpatient care, an average of more than seven in-hospital days was avoided for each patient. A saving of over $1,000 for a ward bed or over $2,000 in an ICU bed was estimated. Interestingly, a previous (identical) moderate pneumothorax, treated in the intensive care unit of another institution, cost one of these same patients $2,500 (1975 costs).

Other advantages of ambulatory chest tube care were some relief of a significant hospital bed shortage, uninterrupted parental care for small children at home, reduced surgeon frustration with persistent air leak, a marked decrease in the use of analgesics and sedatives, and avoidance of all the other complications and delays in recovery that may result from "being sentenced to" inactive, dependent status in a hospital bed.

Conclusions: Self-care at home with a simple, stable, unbreakable water-seal chest drainage system (Fig. 6) is practical for most patients requiring closed thoracostomy after spontaneous pneumothorax. Home chest bottle care is also indicated for an occasional patient with a prolonged postoperative air leak from lung. "Out of sight" does not mean "out of mind," however, as dressings, chest tubes and even disposable bottles will all require occasional attention until they are finally removed (and perhaps then converted into a "terrarium" or miniature greenhouse by the grateful patient).

NOZZLE COVER

CHEST TUBE CONNECTOR

RUBBER INJECTION SITE

DRAINAGE TUBING

AIR VENT DUST COVER

AIR VENT FITTING

ACCESS PORT

WATER SEAL STRAW

VOLUME MANOMETER SCALE
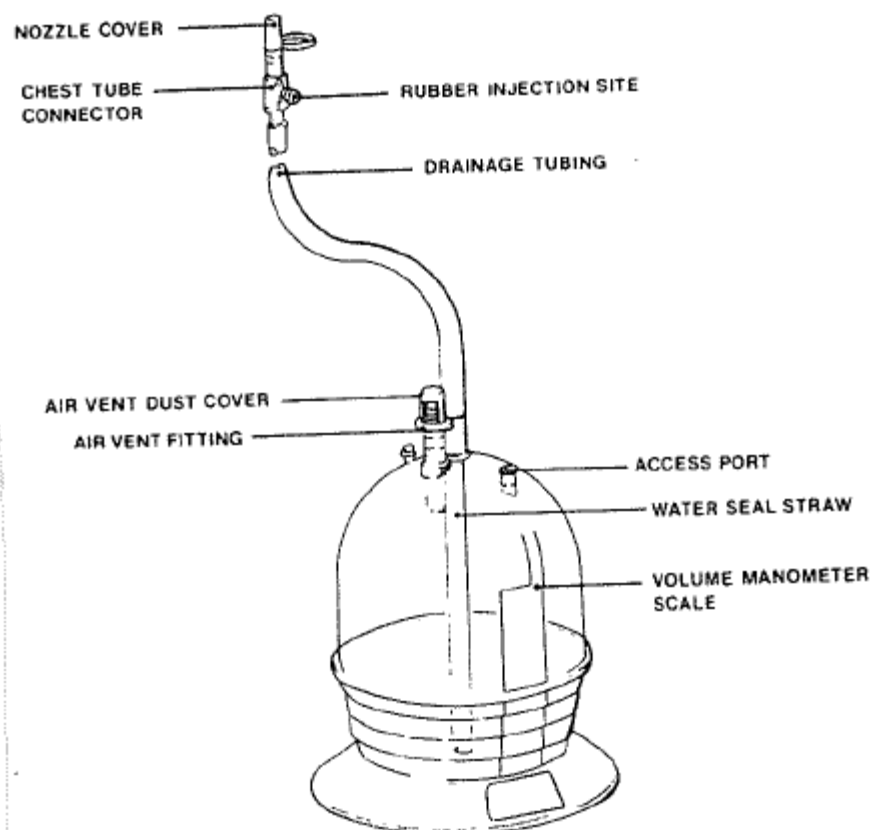
Bentley von Hippel-Tector Pleural Drainage System PDS-100

Figure 6.

CHAPTER 7: THE WATER SEAL AS A MANOMETER OF INTRAPLEURAL EVENTS: A PRACTICAL REVIEW

Normal intrapleural pressure events; ... The water seal straw speaks of many things; ... The water seal manometer can identify respiratory obstruction;... So watch the tidal to evaluate inspiratory effort;... Full lung expansion means loss of the manometer function;... Case 1: Peculiar tidal due to a misplaced chest tube;...Tidal with an occluded or misplaced chest tube;... Case 2: Normal progression of inspiratory tidal in pneumothorax; ... Case 3: High tidal with atelectasis;... Comments on atelectasis; ... Postoperative bronchoscopy for secretions;... Case 4: Massive air leak and no tidal;... Treatment of massive air leak;... Alternatives with major air leak;... Case 5: Intermittent atelectasis;... On keeping a chest tube in its intended location;... Case 6: Unbelievable tidal;... Case 7: Absence of tidal;... Case 8: Persistent minimal air leak is uncommon;... Persistent air leak can be simulated by a defective water seal system;... Or a porous patient;... Detect and correct extraneous air entry;... Or overwhelm the leak with chest bottle suction;... A hanging water column applies suction to the chest tube (and detects air leak);... Case 9: Absence of tidal while receiving positive pressure ventilation;... When is tidal not expected?... Sometimes a one-way valve is unnecessary.

Normal Intrapleural Pressure Events

If unimpeded, air always moves from higher to lower pressure zones. Normal breathing therefore utilizes slow muscle contractions to alternately drop intrapulmonary air pressures below atmospheric and raise intrapulmonary air pressures above atmospheric.

On inspiration, the thin upwardly domed diaphragm muscle shortens actively to push viscera downward while chest wall muscles lift ribs up and out. As a result, air is drawn into the steadily enlarging thorax through open nose and mouth until intrapulmonary air pressure rises to atmospheric.

During ordinary expiration, respiratory muscles relax and ribs move down and in, while abdominal muscle tension pushes viscera and relaxed diaphragm back up into the chest. This steady reduction of thorax size allow stretched lung to shrink back toward its relaxed size. Intrapulmonary air therefore flows out into the atmosphere until intrapulmonary air pressure drops to atmospheric.

Thus small, constantly adjusted intrapulmonary pressure gradients (below and above atmospheric) transfer the exact volume of air required for respiration.

The Water Seal Straw Speaks of Many Things

Respiration-related fluctuation ("tidal") of fluid in the water seal straw and chest tubing, continuously confirms chest tube patency: An occasional quick glance at the simple water-seal chest drainage unit also provides "real-time" insight into intrapleural "happenings." Unlike other measuring catheters which must be inserted and maintained with varying degrees of difficulty and risk, the chest tube is already in place; and it generally remains there as long as anything worth reporting is "going on."

A properly handled, well designed water-seal unit causes minimal back-pressure to chest tube drainage: And the water seal spontaneously applies increasing suction as the fluid level gradually rises

within the straw and tubing. The major advantages of water seal over other methods of assuring one-way, closed-chest drainage are safety, low cost and simplicity: The fact that it is less confusing to nurses, residents and professors, means that "simple water seal" chest tube drainage is more likely to be applied correctly.

The Water Seal Manometer Can Identify Respiratory Obstruction

Bronchi are living tubes that constantly change their diameter in response to physical and chemical influences. Although each inspiration results in passive widening of intrathoracic air passages, cartilaginous stents are necessary to prevent these airways from collapsing during expiration (when air pressure in the surrounding lung parenchyma exceeds that within the airway): Normal bronchial narrowing during expiration increases the severity of any partial distal-airway obstruction (whether due to inhaled peanut, bronchial adenoma or bronchospasm), and delays deflation of the affected portion of lung.

On the other hand, "upper airway" obstruction usually becomes most symptomatic during inspiration (with inspiratory prolongation, "crowing" and wheezing) because; (1) larynx and cervical trachea are not exposed to intrathoracic pressures, so their lumen is not reduced on expiration; (2) loose upper-airway obstructions (e.g. the tongue of a comatose patient) tend to be blown out of the way as the patient exhales, then drawn back into obstructing position on inspiration; and (3) respiratory muscles can apply more force to chest compression than to chest expansion (consequently, musical "wind" instruments are usually "blown" rather than "sucked").

So Watch the Tidal to Evaluate Inspiratory Effort

Increased inspiratory effort is usually accompanied by increased tidal at the water seal straw (as long as the chest tube still communicates with a residual pleural space). However, a major pulmonary air leak often prevents development of any inspiratory tidal - thereby documenting persistently above-atmospheric intrapleural pressures regardless of inspiratory effort; such "pleural-space air-trapping" occurs when more air escapes into the pleural space than is removed by the chest tube during expiration.

The water seal straw usually does not allow estimation of expiratory effort. First of all, once the straw fluid level has been pushed below the water seal surface, bubbling will result (unless the straw tip is submerged to an excessive depth). Secondly, in the absence of air leak, lung tends to occlude chest tube holes during expiration, again limiting downward fluid movement in the straw.

Fortunately, inspiratory tidal turns out to be a fair measure of overall respiratory effort (since dyspnea affects inspiration and expiration simultaneously). Of course, as air leak ceases and the pleural space closes down about the tube tip, tidal will diminish rapidly (regardless of breathing effort). Once tube tip and side holes are sealed off by lung, the water seal bottle no longer serves as a manometer of intrapleural pressures. (And this is often the appropriate time for chest tube removal.)

A chest tube within a residual pleural space signals "Possible inspiratory obstruction!" if its tidal increases markedly (to 20, 30, 40, 50 cm. or even more) with each attempted breath: Such an accentuated tidal will be most noticeable in the absence of a large pulmonary air leak (which limits maximum inspiratory pressures by rapidly refilling a large pneumothorax).

Measures that open the airway (such as posterior pharyngeal or endotracheal suction, or merely lifting the angle of the jaw forward and up - toward the tip of the nose - to bring posterior tongue and epiglottis forward) can markedly relieve inspiratory obstruction in a supine, unconscious patient: This relief is accompanied by a marked decrease in tidal, as each inspiratory effort successfully delivers more air with less pressure change (ordinary "volume work" requires far less effort than poorly rewarded "pressure work").

For similar reasons, high inspiratory tidal may be the earliest sign of major atelectasis, for blockage of the lower airways also prevents lung from accepting a normal inflow of air. In other words, a marked intrapleural pressure drop (high tidal) necessarily results when inspiratory chest enlargement cannot draw air into a blocked lung or closed pleural space.

Tidal at the water seal bottle is therefore observed frequently, especially when a post-thoracotomy patient is first brought to the recovery room. If the patient has been extubated prematurely, a wide tidal may be the first clue that the upper airway is obstructing, and that the patient needs help in order to move air more effectively (his jaw held up, an oral, nasal or endotracheal airway inserted, posterior pharyngeal or endotracheal suction, or perhaps additional antagonist given for previously administered narcotic or relaxant).

Full Lung Expansion Means Loss of the Manometer Function

Intrapleural pressure changes are transmitted through the chest tubing air column to the water seal straw as long as a well-positioned chest tube (that has been "milked" to assure patency) communicates with an air-containing pleural space. When that pleural space is "lost" (by successful pulmonary reexpansion), the manometer function of the water seal straw is also lost.

An elevated, stable, water seal straw fluid level routinely follows cessation of pulmonary air leak and elimination of the residual pleural space. This stable fluid level (within the water seal straw or lower chest tubing) is usually as high or higher than any recent inspiratory tidal, for any small quantities of air that are drawn into the chest cavity from the upper chest tubing (during vigorous inspiration) will soon be absorbed through the living pleural surfaces.

A very high "straw" fluid level, well up within the chest tubing, often results when fluid drainage persists after lung air leak has ceased, because a constant movement of fluid down toward the water seal bottle can carry along (to the bottle), displace (back into the chest), or even "absorb" air from the chest tubing. Not uncommonly, an uninterrupted column of fluid may develop, from pleura to bottle, during drainage of a significant effusion: This tall, hanging fluid column may exert excessive suction (see Chapter 8).

When the pleural space is totally obliterated and pulmonary air leak has ended, aspiration of air from the upper chest tubing will still raise the fluid column within the water seal straw and tubing. Similarly, allowing brief air entry into the upper tubing (as by an open needle inserted through the chest-tube-connector injection site) quickly lowers this fluid level in the straw.

Neither maneuver affects the nearby fully expanded lung, however, for at this late stage, any fluid level in the straw merely indicates what pressure is being applied to the small spot of pleura in direct contact with each chest tube hole. In other words, sucking harder on fully expanded lung does neither harm nor good; adding a little air to the tubing in such a case accomplishes nothing either.

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On the other hand, continued atmospheric access to the pleural cavity (as by an open needle penetrating the chest tubing, or a chest tube left open to the atmosphere for several inspirations) would soon overcome the adherence of moist visceral pleura to moist parietal pleura and thereby permit lung to fall away, allowing a recurrence of pneumothorax. Obviously, a surgically diminished, or a restricted (by thick visceral pleura), or otherwise "small and scarred" lung will have an increased tendency to pull away from the parietal pleura during any atmospheric access to the pleural cavity, unless already secured in its fully expanded position by interpleural fibrosis (which process may occur, but often only days or weeks after chest tube removal).

The following case reports illustrate the usefulness of a simple water seal, chest bottle straw as an intrapleural manometer:

Case 1: Peculiar Tidal Due to a Misplaced Chest Tube

A very obese fifty-year-old female developed a moderate pneumothorax from diagnostic thoracentesis performed prior to laparotomy. Knowing that her injured lung was likely to leak additional air during positive-pressure assisted ventilation at surgery, a chest tube was placed in the second anterior intercostal space. Although tube insertion was rewarded by a gush of air, the water seal straw acted strangely thereafter, showing episodic bubbling not synchronous with expiration. When tidal did occur, it was only one or two cm, and the fluid level in the straw frequently settled at, or even below, the water seal surface within the chest bottle.

Milking or "stripping" her chest tubing had little effect, except to alter the straw fluid level (although the tubing "felt" open). Portable chest roentgenogram after thoracostomy showed persistent pneumothorax, with the chest tube seemingly in satisfactory position. A "technically-poor" roentgenogram, taken the following day (with some rotation of the patient) revealed the chest tube tip to be outside of the bony thorax.

Analysis: Undoubtedly my Kelly clamp initially penetrated the parietal pleura, but the tube was then misdirected into a deep chest wall (retromammary, extracostal) position. The occasional burst of bubbles at the water seal straw tip (not synchronous with expiration) merely signified ongoing decompression of the pleural space through the intercostal Kelly puncture site (with a submammary air pocket venting intermittently whenever surrounding soft tissues ballooned away from tube tip or side holes).

Closed thoracostomy was repeated (for free). With a new chest tube now properly intrapleural, excellent tidal with some expiratory bubbling continued for two days, associated with good lung expansion on chest roentgenogram. Diminishing respiratory oscillation of the increasingly elevated straw fluid level thereafter confirmed that the tube was still patent and that the air leak really had sealed. Chest roentgenogram the next day still revealed satisfactory lung expansion, so the chest tube was removed.

Tidal With An Occluded or Misplaced Chest Tube

Absence of free tidal soon after chest tube insertion suggests a kinked or malpositioned chest tube. A stable straw fluid level below the water seal surface (in a patient not on positive-pressure respiratory assistance or chest bottle suction) must mean that the chest tube is blocked: Otherwise, it would signify a stable, above-atmospheric, pleural-space air pressure throughout the entire respiratory cycle (but then

any air or fluid contained within such a pressurized space should promptly drain out through the open, properly placed chest tube). "The chest tube always tells the truth," but its truth is whatever is experienced by the column of air in continuity with the water seal straw.

An occasional cause of chest tube blockage is postoperative hemothorax, with a patent chest tube embedded in blood clot. In this case, air is easily pushed up the "blocked" tube into the chest, but it cannot return, for surrounding clot immediately reseals the tube holes (another one-way valve): A higher straw fluid level (without resumption of tidal) usually follows milking of a tube buried in clot.

Similar results of "milking" are seen when the tube has been misplaced into chest wall soft tissues, especially when there is no air leak. Subcutaneous or intramuscular misplacement of the chest tube tip is usually more painful than the appropriate intrapleural placement, as every movement causes the tube to poke periosteum or pull against its skin stitch, so recheck the chest tube position when a patient complains excessively.

Case 2: Normal Progression of Inspiratory Tidal in Pneumothorax

A thirty-seven-year-old female entered the hospital with a left hemopneumothorax secondary to self-inflicted gunshot. Her trachea was deviated to the right, neck veins were full, face and body purple, left chest overexpanded and rigid, and her right chest moved with gasping respirations. Blood pressure was reportedly 70/0 mm Hg and the patient was stuporous.

A chest tube on a large hemostat was quickly slipped through the conveniently located (left-third-rib-and-interspace) bullet hole, with release of air and blood under pressure. The immediate improvement in her appearance and vital signs continued after she was placed on water seal drainage and her blood volume restored.

Initially, her chest tube drained blood and much air on expiration; during inspiration, the straw fluid level rose only 1 to 2 cm above the water seal surface. Over the next two days, however, inspiratory straw fluid levels gradually increased (to a 10-12 cm tidal), associated with decreased expiratory bubbling and less drainage. The increased height of inspiratory tidal was noticeable earlier, and easier for the observer to quantitate, than the simultaneously diminishing volume of air bubbles.

This gradual increase of inspiratory tidal usually documents progressive healing of leaking lung (which is confirmed by auscultatory and roentgenologic evidence of increasing lung expansion). When listening with a stethoscope over the site of pulmonary damage, one can often hear air escaping from lung; sounds heard will vary from an expiratory squeak or wheeze, to a rattle, or with very-high-volume air flow, to a bubbling rumble.

In my practice, survivors of self-inflicted gunshot to the heart have all been right-handed, apparently saved by the popular misconception of left-sided heart location (as in "Pledge of Allegiance"). Most commonly, the bullet has passed obliquely through the lingula of the left upper lobe. Where bleeding is not excessive, and the injury appears confined to lung parenchyma and chest wall, a chest tube is usually sufficient treatment (along with appropriate supportive therapy such as intravenous fluids, antibiotics, tetanus toxoid, respiratory therapy, and so on).

Case 3: High Tidal With Atelectasis

A forty-seven year old man underwent right lower lobectomy for an epidermoid carcinoma 4 cm in diameter: Resection was carried through an incomplete fissure between middle and lower lobes. During the postoperative afternoon he demonstrated an unremarkable, 6 to 8 cm inspiratory tidal and moderate expiratory bubbling. All bubbling ceased later that evening, with tidal then oscillating from saline level to 20 or 25 cm above water seal level. Additional findings were slight fever and tachycardia.

Atelectasis was confirmed on chest roentgenogram. Although careful nasotracheal suction was carried out several times that evening, bronchoscopy was required the following day (to evacuate pooled mucus from the rather long, intermediate bronchus). Prompt reexpansion of the collapsed middle lobe was associated with a decrease in his temperature toward normal, diminished tidal to 8-10 cm, and resumption of the moderate air leak on expiration.

Comments on Atelectasis

Wide tidal documents expansion of a relatively "inelastic" pleural air space, as well as considerable respiratory effort (with these vigorous breathing efforts not being rewarded by easy air inflow). Of course, the sudden postoperative cessation of moderate bubbling seen in this case could have resulted from simultaneous sealing of all air leaks - and numerous tiny air leaks sometimes do "seal off" when positive-pressure ventilatory assistance is discontinued - but that was not the sequence of events with this patient, who continued to leak air for a number of hours after "coming off the ventilator."

Moderate postoperative air leaks from lung often take several days to close (even weeks, if issuing from severely emphysematous lungs): Decreasing respiratory effort and gradually diminishing tidal accompany this "routine recovery" from pulmonary surgery. As the residual lung expands to fill the pleural space, it also exchanges air more easily. (Pleural space obliteration commonly becomes complete as the air leak ends, and an obliterated pleural space is always associated with a marked diminution of tidal, with the fluid level remaining higher in the straw.)

Here the abrupt change in chest tube function (cessation of bubbling and markedly increased tidal) and associated slight fever and tachycardia both suggested atelectasis. And this "heavy smoker" could not adequately "clear" his long intermediate bronchus by cough, using air still available in his middle lobe, so that lobe simply "collapsed and drowned" in its own little puddle of mucus. A more appropriate resection would have included that small distal middle lobe, especially when dealing with nearby carcinoma (which, in fact, recurred locally in the bronchial stump within a year).

Summary: Early postoperative collapse of a "leaky" lobe (in this case, the middle lobe), often results in sudden cessation of air leak and wider tidal: Removal of chest tubes soon thereafter could easily lead to a significant "delayed onset" pneumothorax, when air reenters the atelectatic lobe. On the other hand, severe lobar infection and empyema are likely to follow if major (lobar) atelectasis is allowed to persist.

Postoperative Bronchoscopy for Secretions

Endotracheal intubation and suction performed by a skilled anesthesiologist probably causes less hypoxia than competent bronchoscopy: Admittedly, the usual suction catheter does not have the visual and directional control of a flexible fiberoptic bronchoscope: Nevertheless, when gentle irrigation is used together with cleansing suction during temporary orotracheal intubation and positive-pressure controlled ventilation, it consistently achieves excellent reexpansion of severely atelectatic lung (at no

increased risk to a recent bronchial suture line). Postoperative atelectasis will be rare, and indications for such therapeutic intubation rarer still, if patients can avoid smoking cigarettes for two weeks prior to admission for elective surgery.

Case 4: Massive Air Leak and No Tidal

This forty-six year old man came to his family physician with a two-day history of epigastric pain. In the course of his "normal upper gastrointestinal series," fluoroscopy revealed a major right-sided pneumothorax. On arrival at the hospital, examination disclosed leftward tracheal deviation in the suprasternal notch, with a fixed, overinflated right hemithorax: Respiratory motion was only apparent on the left side.

Insertion of a chest tube into the right chest under local anesthesia resulted in immediate relief of the patient's discomfort. Air leak was massive during expiration, with persistent bubbling at the water seal during all phases of respiration. Despite vigorous, voluntary coughing, there was little pleural pain and no increased sputum production (as would be anticipated with successful pulmonary reexpansion).

Treatment of Massive Air Leak

In an attempt to increase air evacuation, chest bottle suction was applied at 40 to 60cm of water. The air leak ceased during such high suction, but symptoms of dyspnea and chest pressure simultaneously returned, despite some increase in breath sounds anteriorly. Each disconnection of suction resulted in recurrence of the massive air leak, as well as relief of the "chest pressure."

Chest roentgenogram revealed that the rather small and limp, No. 24 French, plastic chest tube had drooped sharply downward after entering the pleural cavity. As a result, whenever the upper lobe began to expand anteriorly and upward, it blocked the tube openings. This situation was aggravated by my undesirably low, third anterior interspace, tube insertion site, and could not be improved by significant withdrawal of the chest tube, for bringing a chest tube side-hole out-side of the parietal pleura in this high-volume air leak situation would undoubtedly have resulted in major subcutaneous emphysema.

With lung already collapsed for two days prior to diagnosis, and now another day wasted on my inadequate treatment with a poorly placed, undesirably small chest tube, the situation was that of a dis-gruntled patient with major air leak (water seal bottle "frothing over"), chest bottle suction ineffective in achieving better expansion, and underlying atelectatic lung still full of secretions.

Alternatives With Major Air Leak

When major air leak prevents even partial lung reexpansion (despite well-placed adequate-size chest tubes, vigorous respiratory therapy and "high" chest bottle suction), the air leak must be closed at thoracotomy, to avoid pneumonia, empyema and trapped lung. In addition, bubbling at the water seal straw that persists through inspiration simply documents continuing tension pneumothorax in the pleural space.

Fortunately, any pleural space even partially decompressed by a large-bore needle or tube will not build up enough air pressure to cause a rapidly fatal compression of lung and heart, unless the patient is also on positive-pressure ventilatory assistance (in which case several large chest tubes may be required very soon to release all the air being pumped through leaky lung into the "relatively closed" pleural

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space). With or without a chest tube present and functioning, however, even mild tension pneumothorax will prevent air exchange in its underlying lung (thus an alert patient will voluntarily hold the affected hemithorax in a partially or fully expanded position to gain symptomatic relief).

After considering all options, surgical correction was recommended to this patient with large air leak, inadequate chest tube placement and persistent lung collapse: At thoracotomy, a 4 x 5 mm hole was found in the apex of his emphysematous lung. As is often seen in a major spontaneous pneumothorax that has been unresponsive to closed thoracostomy, a flimsy, avascular, translucent bleb lining had herniated out through the tear in the bleb.

Almost certainly, before such a major bronchopleural fistula could have healed across its avascular orifice, pneumonia and empyema would have supervened: Most likely, a later surgical decortication would then have become necessary to obliterate the chronically infected pleural space and partially regain function of the entrapped atelectatic lung.

In any case, as soon as the ruptured bleb was clamped, excised, and oversewn with 2-0 chromic sutures, the lung reexpanded easily. Several other blebs were also oversewn. During the next few minutes, the anesthesiologist was kept busy suctioning away copious endotracheal secretions released by newly inflated lung.

Visceral and parietal pleurae were then abraded with dry sponges to promote pleural symphysis. Two chest tubes were positioned from low-lateral skin incisions, through appropriately directed, short subcutaneous tunnels; the anterior tube was brought up anterolaterally within the pleural space to the first rib level, while the posterior tube was positioned slightly lower.

Postoperatively, both chest bottles bubbled vigorously on expiration and tidaled 5 to 6 cm up into the straw on inspiration. The patient was quite concerned about this continued moderate air leakage from suture holes in his emphysematous lung, but on the basis of improved tidal, it was possible to reassure him of the temporary nature of these new postoperative air leaks (in fact, they all sealed within four days). Chest tubes were removed on the fifth day after thoracotomy, at which time no further tidal was seen and the chest roentgenogram demonstrated good lung expansion.

Comment on Tube Placement: In a heavily muscled chest, the second rib is best identified where it meets the sternal-manubrial angle. In this slender, very long-chested individual, higher insertion of the original tube (perhaps even just over the second rib, through the first interspace) and use of a larger (at least No. 28 French) chest tube initially, would have been preferable.

Second interspace (midclavicular line) chest tube insertion remains the standard, however, particularly in an emergency. Thoracostomy through this site is generally definitive treatment for free air or fluid not pocketed elsewhere. Subclavian vessel injury during chest wall penetration via the second interspace is virtually impossible, but this risk should be kept in mind when contemplating higher placement of the chest tube.

Case 5: Intermittent Atelectasis

An emphysematous, seventy-two year old male underwent right upper lobectomy for carcinoma. Pulmonary function was marginal, but he was accepted for surgery when he climbed one and one-half flights of stairs without undue distress. Two chest tubes were placed laterally at thoracotomy, the upper,

anterior tube being secured to the periosteum of the first rib with a fine catgut suture. An initial post-operative chest roentgenogram showed good tube position with partial lung expansion: Vigorous air leak and some inspiratory tidal were also noted at this time.

A few hours later, his chest roentgenogram was repeated to evaluate increasing dyspnea, associated with diminished breath sounds and intermittent chest tube bubbling but no regular tidal. This film revealed the remaining lobes now less well expanded, due to the "anterior" chest tube having fallen transversely down behind lung. Here it became blocked whenever the lung expanded slightly, thus preventing vigorous cough or chest bottle suction from evacuating his pneumothorax (although some venting of air occurred through the displaced tube whenever he leaned forward).

An anterior closed thoracostomy, placed between his first and second ribs under local anesthesia, relieved his pneumothorax: There was prompt resumption of the significant expiratory air leak and 6-8 cm inspiratory tidal. Over the next days, his lower and middle lobes gradually ballooned upward and the two lower tubes stopped bubbling: When their tidal and drainage also ceased, they were removed. Meanwhile, the upper tube kept leaking lung stretched using a 60 cm "high" chest bottle suction setting.

Air leak persisted via this upper chest tube for two and one-half weeks. During that time, the patient suffered recurrent bouts of atelectasis, each associated with labored respiration, cyanosis, cessation of air leak and marked increase in tidal (with the straw fluid often fluctuating from the water seal surface over halfway up to the bed level).

Initially, his intermittent atelectasis could only be overcome by nasotracheal suction. Later, as the patient regained strength (after an incidental myocardial infarction), he became able to accomplish his own bronchial cleanout by vigorous coughing. Whether by suction or cough, as soon as obstructing mucopurulent sputum was evacuated, the air leak always resumed through his chest tube as his dyspnea decreased.

Eventually, after gradual cessation of air leak and then decreasing tidal, chest roentgenograms showed complete expansion of his residual lung. The upper chest tube was removed two days later.

On Keeping a Chest Tube in its Intended Location

Larger (therefore stiffer) plastic chest tubes, directed more appropriately upward from the lateral insertion site through better alignment of the "tunnel," would have remained in proper position without a flimsy suture to the periosteum. If the posterior tube had also been brought up to first rib level, this would have been very helpful in improving air evacuation when the anterior tube "malfunctioned" (especially as major postoperative air leak was easily predictable at thoracotomy). Now in his late eighties, the patient remains free of carcinoma, still smoking and drinking excessively.

Case 6: Unbelievable Tidal

Following expeditious wedge biopsy of very stiff, nodular lung, a single, anterior-apical chest tube was placed and the chest closed. As long as the anesthesiologist still provided ventilatory support, each squeeze on the breathing bag was associated with a significant upward movement of fluid in the water seal straw. This paradoxical tidal reverted to normal when the patient awoke and began spontaneous respirations.

Comment on Paradoxical Tidal:

It required moderately vigorous compression of the breathing bag to push air into this small, stiff, wedge-shaped lung. As it expanded, the lung moved downward en bloc toward the wider thorax at the diaphragm level. Intrapleural pressure at the apex (where the chest tube had been placed) therefore decreased as lung moved down and away during each forceful inflation.

Moral: The water seal manometer never lies: Its most puzzling or insignificant pronouncements are real and thus worth interpreting, if only for the insight they provide into this "sign language" that we should all be trying to learn.

Case 7: Absence of Tidal

The small afebrile two-year-old with a flat abdomen had his first-ever chest roentgenogram during evaluation for persistent non-productive cough and increasing dyspnea. Consultation was requested when multiple air-fluid levels were reported in his left chest, along with tracheal deviation to the right. While sitting in an oxygen tent, he appeared minimally dyspneic. But after numerous antibiotic shots for presumed pneumonia, lung abscesses and empyema, he was also uncooperative.

Careful thoracentesis (which could only be performed following sedation) delivered only a small amount of serous fluid. Mostly for lack of a better idea, a small chest tube was then inserted from the left sixth intercostal space laterally. With the chest tube in place, there was no tidal at the water seal straw.

Milking the chest tubing documented tube patency, but resulted only in displacing the stable fluid level in the water seal straw; at times this level settled below the water seal surface. Even with the chest tube again opened to the atmosphere, there was no air movement through it, despite several attempts to achieve better tube position. When air was irrigated in, it did not return.

At this juncture, repeat chest roentgenogram (for tube position) revealed a multicystic, space-occupying mass in the left chest, nicely outlined by the minor, iatrogenic pneumothorax. Barium swallow confirmed Bochdalek hernia, which was repaired uneventfully. Two days postoperatively, the cheerful, potbellied, flat-chested patient was discharged, eating frequent huge meals (without further antibiotic shots).

Comment: Although a chest tube amongst intestinal viscera drains nothing, it still seems patent on irrigation (just as when buried in blood clot). This case represented a medical "fumble with recovery": His scaphoid abdomen should have been a giveaway, as would bowel sounds within the chest, had auscultation been practical. However, he was uncooperative, noisy and insisted on sitting, so the apparent x-ray diagnosis of "lung abscess with empyema" was accepted without independent examination.

Fortunately, chest tube insertion in this properly restrained and sedated child, was atraumatic as well as unnecessary. In any case, peculiar tube function suggested a mass lesion, and chest roentgenogram confirmed this. Once outlined by air, it "had to be" either cystic lung or multiple loops of bowel.

Moral: (1) Once again, the chest tube tells all, although its initial message may be unclear. (2) If possible, reexamine a child after proper sedation - before tube insertion, even if already "completely

evaluated" by a pediatrician (as this child had been, over several previous months, for "failure to thrive").

Case 8: Persistent Minimal Air Leak Is Uncommon

This twenty-five year old bride of twenty-four hours came to the emergency room with a two-day history of chest pain. Major spontaneous pneumothorax was diagnosed and a chest tube placed through the inframammary fold, directed upwards. Expansion of lung was satisfactory, with minimal air leak: The anxious groom was therefore assured that the leak would soon cease. However, despite "high" chest bottle suction, vigorous coughing and frequent deep breaths, a minimal air leak persisted for five days, so consultation was requested.

On examination, no faint apical wheeze or bubbling sound could be elicited (often heard with a cough or Valsalva maneuver when lung still has a slow air leak). When the chest bottle and its tubing of repeatedly autoclaved latex were replaced, the air leak immediately ceased. Follow-up roentgenogram showed full lung expansion, so the chest tube was removed, a dressing applied, and the patient discharged with advice to stop smoking and avoid vigorous activities for a few days.

Persistent Air Leak Can Be Simulated by a Defective Water Seal System

A persistent small (or unexpectedly large) air leak should stimulate a careful search for some site of atmospheric entry into the supposedly "water-sealed" system. A cracked or loose chest tube connector, defective or damaged tubing that is not "airtight," a water seal straw that is cracked or not quite long enough to remain under saline, a bottle with water seal forgotten (or carefully poured off for "intake and output" charting) or a chest tube located in an overlarge bullet hole (where it does not fit snugly), all can permit outside air entry into the water seal system during normal inspiration.

Or a "Porous Patient"

Following thoracoabdominal incision for liver resection, with a chest tube above repaired diaphragm and drains below, one can anticipate copious chest tube fluid drainage for two or more weeks: In addition, with a scaphoid abdomen, subphrenic drains may permit outside air to pass via the abdomen into the chest: The expiratory bubbling that then results will incorrectly suggest "persistent lung air leak." Such an extraneous air leak usually stops within days, but a chest tube is left in place as long as the significant (ascitic?) drainage persists.

Detect and Correct Extraneous Air Entry

A minor persistent "technical" inflow of air to the pleural space may not prevent virtually complete expansion of normal underlying lung if the pleural space is appropriately drained. However, a larger air leak or more restricted lung can easily contribute to a residual "pleural space problem" if ongoing infection, or pleural inflammation and fibrosis, trap a small or incompletely expanded lung within thickened visceral pleura.

The search for any "technical" site of air entry into the chest tube, tubing or straw is expedited if a short segment of chest drainage fluid is carefully followed down through the tubing. Air entry can be seen (through clear plastic tubing) as fine bubbles entering the fluid-filled lumen at the offending site, provided subatmospheric pressure is maintained within the tubing for the duration of this inspection (by

some form of chest bottle suction or a hanging fluid column).

An occlusive dressing with airtight tape (or "spray-on" plastic film) can be used to stop troublesome "incidental" air access at the skin level: Tincture of benzoin applied to skin will improve the adherence of such a dressing. An occasional short-term non-pulmonary source of the air bubbling from a water seal straw may be the hole that remains after another chest tube has been removed (especially from a very thin patient).

Such an unusual leak rarely persists more than two days: In the meanwhile, the offending tube hole can be dealt with by a more or less airtight dressing (faced with a large mound of antibiotic ointment on some Vaseline gauze) or by a suture or metal skin clip (as long as that skin is not excessively indurated).

Or Overwhelm the Leak With Chest Bottle Suction

Some minor "technical" air leaks are not easily identified or resolved. However, they can be rendered insignificant by the use of chest bottle suction until the residual intrapleural space has been obliterated. The chest tube is then removed while still on suction (once confident that no pulmonary or other air source will cause recurrent pneumothorax).

A Hanging Water Column Applies Suction to the Chest Tube (and Detects Air Leak)

Air entry into the chest tubing, or withdrawal of air therefrom, causes the water seal straw fluid level to descend or move up. In fact, this hanging fluid column is by far the simplest, safest method for applying and regulating chest bottle suction, provided there is no air leak. Creating an elevated fluid level in straw or tubing is also the most sensitive way to detect a persistent air leak, for even slight or intermittent air entry into the pleural space or water seal tubing will cause this fluid level to descend.

When Is Tidal Not Expected?

Closed thoracostomy was performed for post-traumatic tension pneumothorax in a patient receiving mechanical ventilatory assistance. Although each of several tube reinsertions released air and seemed adequately positioned on chest roentgenogram, the surgeon was unable to obtain "proper" tidal. Consultation was therefore requested.

"Positive-pressure ventilatory assistance" means that air is forced into the lungs during inspiration, then passively exhausted to the atmosphere on expiration. This means that instead of (the usual) subatmospheric inspiratory intrapleural pressures alternating with above-atmospheric expirations, we see a markedly-above-atmospheric inspiration alternating with an ordinary still-above-atmospheric expiration.

As long as a patient remains on the ventilator, therefore, one will see no tidal, for this regular upward movement of fluid in straw and tubing reflects the subatmospheric inspiratory phase of normal breathing. Disconnecting the respirator momentarily will allow some tidal to become evident if the chest tube is still functional and the patient makes useful inspiratory efforts.

Summary: While breathing is effectively controlled on a positive-pressure ventilator, a patient will not raise the fluid column within the water seal straw during inspiration (for his intrathoracic pressures

remains above atmospheric throughout the respiratory cycle). Under these circumstances, air leak from lung will only be demonstrated by bubbling at the straw tip.

Any persistent increase in intrathoracic pressure, such as that experienced during pneumothorax or ventilatory assistance, may diminish venous return and markedly reduce the cardiac output and blood pressure of a hypovolemic patient; this effect is all too commonly confirmed by cardiovascular collapse during vigorous induction of emergency anesthesia in the badly injured patient.

Sometimes a One-Way Valve Is Unnecessary

With the exception of a rigid, chronic empyema cavity, which can simply be allowed open dependent drainage, effective pleural space drainage requires a one-way valve unless one-way flow is supported by a persistent pressure gradient from pleural space to atmosphere. Such persistent pressure gradient can either be maintained by constant vigorous suction on the distal end of the chest tube, or through controlled positive-pressure (inspiratory) breathing assistance.

When the inside-toward-out pressure gradient along the chest tube never reverses, one-way air flow will be maintained without need for a one-way valve. Thus, an open chest tube (or bullet hole, or thoracotomy incision) will not prevent full lung expansion while the patient remains on positive-pressure controlled ventilation. Similarly, effective chest bottle suction eliminates the need for a water seal or other valve during drainage of air or fluid from the closed pleural space.

But when a patient on a respirator does show some tidal, he must be making a significant inspiratory effort (i.e. his breathing is not "controlled" by the respirator). When a respirator is purposely adjusted to "assist" breathing, the height of tidal becomes a measure of the patient-generated inspiratory suction (subatmospheric pressure) required to trigger the respirator. However, if the respirator is supposed to be doing all his respiratory work, yet the chest bottle straw still shows inspiratory tidal, a serious problem exists that must be remedied at once.

For such tidal in the simple water seal chest bottle tells us that a supposedly fully ventilated patient is gasping for more air than he is being provided; perhaps the endotracheal tube is obstructed or displaced, or the respirator is malfunctioning or misconnected or disconnected or, if air exchange and blood gases prove satisfactory, the patient may need additional sedation to relieve him of the work of breathing (why else would he be on the respirator?) Of course, the patient could simply be gagging on an unnecessary oral airway that feels like "two very long fingers rammed down the throat," or possibly the endotracheal tube itself is gagging a patient who is now awake and strong enough to be extubated.

Whatever the problem, if inspiratory tidal is detected when not expected, one should always consider immediate disconnection of the patient from the mechanical ventilator as a first step in "trouble-shooting". Then simply use a hand-held hand-squeezed Ambu (or other self-inflating) breathing bag - with or without supplementary oxygen - until a respirator-related problem has been ruled out. For inadequately ventilated patients are liable to suffocate unnoticed while respirator mal-functions are studied and corrected.

CHAPTER 8: CHEST DRAINAGE DEVICES AND CHEST BOTTLE SUCTION

Basics;... One-way valve;... Collection of drainage and related problems;... A collection chamber interposed before the water seal simply impedes chest tube drainage;... Then what was the idea?... What is a vacuum breaker?... There is no need for a vacuum breaker except to protect a flimsy drainage unit;... The end result is costly, complex and ineffective;... Other options to assure one-way flow;... Basic considerations: Suction and positive-pressure;... Definitions;... Volume and pressure capacity;... Suction lifts a water column;... A water column can push or pull;... A fluid column too high;... Mechanical suction for intrapleural tubes;... Mechanical chest bottle suction is never needed unless there is air leak;... High suction may occasionally be hazardous;... Mechanical chest bottle suction may improve lung expansion if there is air leak;... A chest bottle suction machine increases risk;... How to connect mechanical chest bottle suction;... Suction is never lifesaving; when in doubt, disconnect;... Suction with major air leak;... How much suction?... Foaming and reflux from the chest bottle;... Avoid "technical" chest bottle air leaks if planning chest bottle suction;... Three bottle suction is outmoded and dangerous;... The vacuum breaker straw tells us if suction is adequate;... The vacuum breaker fluid level drops through evaporation, although the water seal surface does not;... Conclusion and review.

Introduction

Successful chest drainage requires only the simplest tools, yet these are often misapplied. The surgeon usually proceeds by rote rather than reason, while pulmonary physiologists (seeing nothing to study) remain unsure of practical applications. The wide variety of chest drainage devices currently marketed either document such confusion, or they confirm that no completely satisfactory chest drainage device exists, or they imply that there is more than one solution for every drainage problem (or all of the above).

Basics

A properly placed chest tube permits free movement of air and fluid through the chest wall. An effective chest drainage device avoids interference with outward flow while blocking return.

One-Way Valve

To prevent retrograde flow, almost all chest drainage systems either rely on a water seal or on tight approximation of thin rubber or plastic flaps. Water seal is simpler to create but demands definite gravitational orientation. A properly designed, springy flap valve will work in any position (although stiff leaflets may be held apart by blood clot). Flap-valved chest tubes are therefore included in space flight medical kits.

A flap valve system should certainly be lighter than a water seal unit, for at least 200 cc of sterile saline is usually required to keep a water seal straw tip submerged during a maximum inspiratory effort (which draws part of the water seal up into the chest tubing): Although a smaller diameter chest tubing and straw reduces the volume of water seal "prime" required, it also causes back-pressure to major air leak. As a general rule, chest tubing should never be narrower than the chest tube to which it is attached.

The main disadvantage of flap valves is that they provide no visible information on air leak or intrapleural pressure changes. When they lack (or cannot interpret) such information, many surgeons revert to inefficient, potentially dangerous methods of detecting persistent pulmonary air leak, such as

clamping the chest tube for an extra twenty-four hours before its removal.

It must be obvious that such clamping can cause tension pneumothorax if an air leak continues (unless, by good fortune, the air leak stops when lung is only partially compressed). On the other hand, if an air leak has ended (possibly even some time ago), the tube might better be removed, rather than clamped and left in place during an additional, possibly unnecessary day of expensive hospitalization.

Summary: Water seal has important clinical advantages over the flap valve. A sturdy, stable, properly designed, clear-plastic water seal unit with adequate prime can be lightweight and trouble-free when used on Earth.

Collection of Drainage and Related Problems

Collection chamber design must emphasize easy air passage, sterility, trouble-free fluid storage, ready fluid retrieval (for autotransfusion or analysis), and sufficient volume capacity so that frequent emptying will be unnecessary. A major problem with conventional water seal is progressive submersion of the straw tip as drainage accumulates within the chest bottle. Fluid evacuation through the chest tube will not be hampered by straw tip depth, but air escape through an increasingly submerged straw tip is increasingly impeded, leading to air entrapment at above-atmospheric pressures within the pleural space.

Traditionally, this back pressure to air drainage has been kept "within tolerable limits" by simply replacing the entire unit when "full enough," or by intermittently decanting excess fluid, or by gradually withdrawing the water seal straw through the chest bottle stopper (as fluid level rises), or by application of sufficient suction to the chest bottle air vent to overcome all back pressure, or by locating the collection chamber proximal to the water seal or flap valve.

A Collection Chamber Preceding the Water Seal Just Impedes Chest Tube Drainage

Costly complex chest tube drainage devices that interpose a collection chamber between the chest tubing and the water seal straw appear to be "selling like hot cakes". Perhaps one day a marketing historian will reveal how an entire generation of chest surgeons was duped into believing that "complex design deficiencies, counteracted by routine chest bottle suction" (two negatives, as far as patient care is concerned), could possibly provide safe effective chest tube drainage.

If the super-huckster responsible for this misunderstanding is ever identified, he should be strung up. But his full statue still deserves a central place in the "Marketing Hall of Fame", close by a bust of the guy who sold his entire inventory of less desirable pink salmon by inventing the slogan "Guaranteed not to turn red in the can!"

But regardless of sales pitch, a large intervening air pocket (collection chamber) always impedes transmission of small pressure changes from chest tubing to water seal straw because the added compressibility or "elasticity" of intervening air creates an "air lock". That collection-chamber air-lock - interposed between your perfectly placed chest tube and its one-way water seal - naturally and inevitably adds cost, confusion and new complications by necessitating chest bottle suction. For without suction to overcome the built-in "air lock", air and fluid retention in the chest would surely cause healing delays, increased risk of morbidity (including empyema), and higher mortality.

Then What Was the Idea?

It has always been very difficult to market a slightly modified cider jug, or even to justify its increased cost: After all, the hospital staff could always consume more cider and recycle those additional cider jugs. But the big problem in applying simple effective cider-bottle water-seal chest drainage to complex clinical situations – which were quite often situations that no one really understood – turned out to be the absence of clearly written instructions for simple water seal.

This left many surgeons and nurses insecure about chest drainage - about what was really going on within the chest - about the unpredictability of chest complications and postoperative disasters. Clearly a ritual was needed that could be followed "religiously" to ward off such evil results. And lo! the manufacturers were there, ready to guide us ever deeper into confusion at ever greater cost.

So they promoted one impressively complex apparatus after another – all difficult for an observer to understand (none reusable). And crucially, each came with clear instructions that did not depend upon (indeed, were irrelevant to) any understanding of chest drainage. And those "ten instructions" to be followed regardless of outcome – implied that "someone far wiser" had designed this important, impressive, and costly device; and that the wise one would always be at the side of the faithful who utilized his apparatus in their darkest hour. Blind obedience to his designated rituals would be a sign of that covenant. And best of all, those surgeons and nurses who accepted and followed his rituals completely, appeared to regain partial control over the patient's fate.

In comparison, carefully individualized solutions offered by experienced practitioners for problems encountered with simple water seal seemed to pale when compared to the benefits of this "comprehensive but costly" approach. In retrospect, it is clear that someone ought to have developed and distributed simple, concise instructions on how to use an ordinary water seal bottle. Then many of these misunderstandings about chest drainage, and innumerable millions of dollars wasted on worthless chest drainage devices each year, and a great deal of patient morbidity could have been prevented. But there was no money in it.

The main problems with a glass cider jug (it might break, some water seal straws and air vents were too narrow and so interfered with free chest drainage, and excess fluid in the water seal bottle caused back pressure to air leak) were easily corrected (plastic jugs, larger straws and air vents, decanting or replacing full water seal bottles, or applying chest bottle suction) - but acknowledging multiple options seemed unattractive to important professors who preferred "discipline" and "everything done the same way every time" (on patients who all differed) "in order to reduce the opportunity for error."

It was this psychology, rather than knowledge of chest physiology or advances in technology, that "saddled us" with these more expensive, interposed collection chamber systems that always create back-pressure to chest drainage and therefore demand constant chest bottle suction. In turn, continuous chest bottle suction with these interposed collection chamber units, markedly increased the cost, complexity (multichambered drainage units full of mysterious valves, secret passages, and bubbling fluid pockets, noisy machines; more tubes and wires) and thus risks to the patient (by machinery malfunction, errors of management and decreased patient mobility).

In our war against disease (as in any other war), doing it the same way every time cannot substitute for a better understanding among "the troops": In fact, it almost guarantees defeat at the very next

challenge (for "a challenge" implies that opponents are fairly evenly matched, at which point it is particularly important that everyone understand what they are doing and why).

It is clear that great confusion about chest drainage has been perpetuated in the minds of surgeons and nurses by this unfortunately popular type of chest drainage system. For example, many apparently reasonable surgeons have concluded that externally applied suction is always necessary to achieve effective chest tube drainage, or that a water seal unit should never be marketed without a vacuum breaker.

A Vacuum Breaker?

As the name implies, a vacuum breaker prevents applied suction from exceeding preselected limits by allowing atmospheric entry into the suction system whenever that limit is reached. The simple vacuum breaker most commonly used for medical purposes is a tall air-tight cylinder somewhere between the suction source and the point of suction application. An open glass tube projects out through the cap of this cylinder into the atmosphere, from its origin near the bottom of the cylinder.

When excessive subatmospheric air pressure is generated within the vacuum breaker cylinder by the suction source, atmospheric air is drawn in through the glass straw. For example, with enough water in the cylinder to submerge the straw tip 20 cm, there will be no atmospheric entry via the straw until suction reaches 20 cm water subatmospheric pressure: At that point, the underwater end of the always open straw begins to bubble, as gently or vigorously as necessary to "break" (overcome) any suction in excess of 20 cm.

Unfortunately, incompletely humidified room air bubbling continuously through such a vacuum breaker will soon evaporate its fluid "prime" unless this is regularly replenished. And if the vacuum breaker "prime" does evaporate unheeded, gradually diminishing suction will be applied until finally, uncontrolled air entry (via a no-longer-submerged straw) eliminates all suction. However, this equipment is reliable, simple and easy to clean, which probably accounts for the traditional use of "cm water" to measure suction, just as the mercury sphygmomanometer established "mm of mercury" as proper units for expressing arterial blood pressure.

An Aside: How can the height of a fluid column be equivalent to pressure? Well, for conversion purposes, 1.3 cm of water is approximately equal to 1.0 mm mercury (and one atmosphere = 14.7 pounds per square inch = 760 mm of mercury). The reason cm water or mm mercury need not be expressed in units of weight per unit of area (such as "pounds per square inch") is that liquid water and liquid mercury are both essentially incompressible and thus of known density (meaning known weight per unit volume). Therefore the height of a mercury column (h) actually implies (h) times density, which is (h) times wt/ vol. Volume is also expressible as height x area. Thus height of mercury column (h), which really means height times density, is (h) x wt/ volume, or (h) x wt/(h) x area which equals wt/area, as in pounds per square inch or whatever (the h's cancel). Please excuse the digression (at times the answer is more confusing than the question, but every legitimate text requires at least one feeble attempt at a formula).

There is No Need for a Vacuum Breaker Except to Protect a Flimsy Drainage Unit

Some popular lightweight chest drainage systems cannot withstand clinically useful "high" suction (e.g., up to 60 cm of water), so a short vacuum breaker is built into the unit solely to safeguard the unit.

The manufacturer's understandable desire to protect his flimsy product and reputation therefore prevents the application of occasionally essential "high" suction (this represents a rather significant conflict between the manufacturer's and the patient's best interests).

To further protect these fragile "interposed collection chamber" chest drainage systems (but not necessarily the patients attached thereto), manufacturers often include an additional vacuum breaker (a "high negativity" safety valve), in order to prevent a resounding drainage-unit implosion that would startle both patient and physician. And implosion can rapidly follow occlusion of the vacuum breaker atmospheric inlet by a physician trying to apply necessary "high" chest bottle suction (30-60 mm Hg) to the patient's chest tube.

An additional "pop-off" ("high positivity!") valve is often added to such chest drainage systems as well: This reduces the likelihood of patient death from severe tension pneumothorax, which might otherwise result when lung air leak exceeded the "volume capacity" of the suction system to which these units must always be connected, or if the suction system malfunctioned or was turned "off" by mistake.

The End Result Is Costly, Complex and Ineffective

It still seems hard to believe that such needlessly complicated, hazardous and expensive chest drainage systems - which require low suction to function, yet cannot withstand useful levels of high suction (and which must routinely be entirely replaced after accidental inversion, partly because they are so difficult to understand and reprime) - were brought to market merely to avoid the need for occasional removal of excess fluid from the water seal bottle (especially when excess fluid in a properly designed water seal bottle can swiftly and safely be emptied through the air vent by inverting the unit over the nearest wastebasket or sink).

Early postoperative ambulation, a major advance in modern patient care, is unusually difficult and delayed when the patient is anchored to such a complex, easily upset, chest-drainage-plus-suction system (adding immobility and delayed convalescence to unavoidable problems already associated with chest drainage). One sometimes wonders how much of the high cost of medical technology is similarly based upon needless complexity, due to misunderstandings of purpose and function, or perhaps to "creative marketing." The old aphorism "A camel is a horse designed by a committee," especially applies to these more complex chest drainage devices.

Other Options to Assure One-Way Flow

How about placing a superb flap valve at the intake of the collecting chamber? This arrangement should improve drainage (due to obligatory "undamped" one-way flow from the chest tubing into the collection chamber), but it still suffers from invisibility of any air leak. Of course, addition of a water seal beyond the collecting chamber would then provide two valves and restore visible bubbling, but then routine suction might again be required to overcome the intervening "collection-chamber air lock."

As mentioned, chest bottle suction eliminates the water seal straw "manometer function," which again leaves one unable to detect chest tube blockage or minor persistent air leak (both are self-evident when using simple water seal). Without the simple water seal manometer, the physician must revert to more frequent physical examinations and repeated, expensive, chest roentgenograms if he wishes to remain even partially informed on the clinical situation.

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The ultimate irony of the entire promotion of "complex interposed collection chamber systems that always require suction" is that appropriately regulated, constant suction by itself eliminates the need for anything more than an ordinary interposed glass jug. In addition, a badly regulated, ineffective or non-functional suction system can instantly turn the most complex chest drainage unit into a hazardous chest drainage blocker.

Basic Considerations: Suction and Positive Pressure

Among other factors, the ease of air passage through any tube depends upon the diameter and length of that conduit, as well as the pressure gradient along it: That gradient can be increased by reduction of pressure at one end, or by build-up in pressure at the other. In the case of a chest tube, marked pressure changes within the patient are tolerated poorly, but significant beneficial alteration of the outside or "atmospheric" pressure is possible through application of suction to the chest drainage system.

Definitions

Pressure above atmospheric ("positive" pressure) is produced by compression. Subatmospheric pressure within a container (also created by work) is the only source of suction. The term "negative pressure," often considered synonymous with "suction" or "subatmospheric pressure," should be avoided, for it has probably confused more discussions of chest physiology than any other expression.

Rather than tolerate "Ahoy, there! Let's raise the negative pressure about 20 cm?!" - Lewis Carroll might insist that we "Bring that positive suction up." It has never been clear to me what is meant by an increase or decrease in "absent" or negative pressure: Far less confusion results when you simply suggest that the suction be increased, for the suction is then being dealt with as a positive thing rather than as a missing pressure.

Volume and Pressure Capacity

Suction or positive pressure can readily be applied to a drinking straw by sucking or blowing with the mouth: Volume capacity and pressure obtainable will be limited under these circumstances. Volume capacity and pressure need not be related, however: A kitchen faucet and fire hydrant both deliver similar pressures, but they exhibit markedly different volume capacities.

The pressure created by any standing column of water increases in proportion to its height and is essentially unrelated to its diameter: The large lake at 10 feet above reference level will release much water without detectable pressure change, while a thin tubing of similar height loses most or all its pressure on removing one cup of water. In the same fashion, different mechanical suction devices may eventually produce identical subatmospheric pressures, but they can differ markedly in the volume capacity available to sustain that pressure.

Suction Lifts a Water Column

Identical subatmospheric pressures can be created by suspending a column of liquid in thin tubing (lower end submerged in fluid, upper end connected to a closed space) or by a multihorsepower turbine pump. In fact, suction is often measured or defined by the height to which a water or mercury column is lifted. Obviously, the turbine can move a lot of air without "losing suction," while the hanging

column of fluid rapidly drops to reference level on even minor air entry into the tubing or its communicating closed space.

A hanging fluid column is most simply created by aspiration of air from the upper chest tubing (or its communicating closed space) until water seal fluid is lifted to the desired height in the tubing. Any syringe used to withdraw air in this fashion, serves as a very low volume capacity, moderately high pressure, suction machine.

A Water Column Can Push or Pull

The kitchen faucet is an example of a standing tube full of fluid delivering above-atmospheric pressure, while an ordinary siphon is a hanging column of fluid that delivers suction and thus can lift fluid. A siphon can easily be "broken" or blocked by air entry, for a relatively compressible or expansile air pocket within the fluid column may "damp" minor pressure changes that would otherwise have initiated ongoing siphon flow (see Chapter 10).

A Fluid Column Too High

Too tall a fluid column in the chest tubing may pull surrounding lung or mediastinal tissues so firmly against chest tube drainage holes that fluid drainage cannot continue. Momentarily venting the proximal tubing with an open needle then permits room air entry into the tubing, releasing some or all of this fluid column down into the bottle: This usually relieves any "tissue blockade" of chest tube holes that are next to (but not under) lung, thus expediting evacuation of previously sequestered intrapleural fluid. (Latex tubing generally remains air-tight after a #20 needle puncture, but the standard needle injection port in a chest-tube-to-chest-tubing connector expedites air admission and withdrawal or fluid sampling.)

One can also allow atmospheric entry to the chest tubing by momentarily tilting the water seal bottle until the straw tip is no longer submerged. This, too, results in displacement of excess fluid from the chest tubing to the chest bottle, for air entering the exposed straw tip soon rises to the top of the fluid column, bringing its level down. However, while fluid-filled tubing remains unprotected by the temporarily-out-of-water straw, tubing content is readily sucked back into the chest (during a deep inspiratory effort); thus many surgeons find the "open needle in tubing" method more esthetic.

We prefer to discontinue air entry into the tubing when the straw fluid level is still somewhat above the water seal surface. A fluid level just above the bottle is easily seen within clear plastic tubing, and while there, it applies low suction and also confirms the continued absence of any pulmonary air leak.

Occasionally, to speed a surgeon on his rounds, an overly helpful nurse may speed around before him, releasing all chest tube fluid columns to the water seal level (thereby assuring optimum fluid drainage and saving the surgeon from this chore). But in this fashion, a desirable nurse may closely simulate an undesirable air leak, thereby delaying timely chest tube removal. Such confusion is avoided if elevated chest tubing fluid levels are not brought down below the top of the water seal bottle.

Mechanical Suction for Intrapleural Tubes

The role of chest bottle suction during closed chest drainage should be clear. We know that major

pneumothoraces can routinely and reliably be evacuated through any satisfactory water seal arrangement. Such drainage is propelled by the same minor intrathoracic pressure changes that are required for normal air exchange (plus an occasional cough). There is no reason to believe that the routine connection of a mechanical suction device to the water seal bottle air vent can improve on that result.

Mechanical Chest Bottle Suction is Never Needed Unless There is Air Leak

Excluding design deficiencies (such as the interposed collection chamber type of unit discussed above), chest bottle suction is never helpful except with air leak: For merely adjusting the fluid column height in a simple water seal system allows one to apply any desired level of suction to the chest tube. In fact, in the absence of air leaks, fluid gradually builds up within the chest tubing anyhow, often to a considerable height.

Interestingly enough, many surgeons who firmly believe that chest bottle suction settings over 20 cm of water are hazardous, do not become alarmed by a 60 or even 80 cm tall fluid column in the vertical portion of the chest tubing (presumably because they have often seen this occur without harm), yet these two positions clearly are incompatible.

High Suction May Occasionally Be Hazardous

It may be important to avoid any form of suction when a chest tube lies near a delicate structure such as an aortocoronary saphenous vein bypass graft: There have been occasional reports of disastrous bleeding from vein grafts apparently "inhaled" by a nearby chest tube. Tube placement near a vein graft is usually avoidable (see Chapter 10).

The delicate and pliable thoracic contents of a newborn are readily drawn together by chest tube suction settings of less than 20 cm of water. More suction than this is unnecessary and could even be detrimental (see Chapter 1).

Mechanical Chest Bottle Suction May Improve Lung Expansion If There Is Air Leak

Chest bottle suction is indicated primarily to force earlier or more complete lung expansion in the face of any size air leak. Mechanical chest bottle suction is most useful when a small or inelastic lung and larger pleural cavity must be brought together, or to overcome a persistent air leak that prevents complete pulmonary reexpansion. In other words, chest bottle suction can help to minimize the size of any temporary pleural space that has an ongoing air supply.

When used for this indication, a continuous suction setting of 40 to 60 cm is often required for days or even weeks. While many flimsy chest drainage devices currently marketed cannot withstand such "high" chest bottle suction, this degree of subatmospheric pressure greatly expedites full lung expansion - which minimizes pleural fibrosis and lung restriction and virtually eliminates the risk of residual pleural space problems or postoperative empyema. (In fact, I have not encountered empyema as a postoperative complication in more than twenty years, except following pneumonectomy.)

Even at 60 cm, continuous suction may have to be applied for several weeks to obliterate a resistant pleural space. We have never recognized a complication due to such "high" suction: However, an occasional Emerson chest bottle suction machine has overheated after many days at the high setting,

"burning out" the inexpensive, easily replaced "vacuum cleaner type" motor (so I advise patients to un-plug the suction machine if they smell smoke or notice it overheating).

Comment and summary: The height of a fluid column in the straw and tubing determines how much suction is being applied by the simple water seal apparatus: However, if a long, fluid-filled loop of tubing is allowed to droop toward the floor it will obstruct flow rather than apply suction, for it is the vertical distance over which a hanging fluid column "wishes to drop" that determines its suction effect.

With no air leak, pleural surfaces soon absorb small amounts of intrapleural air that were not drained by chest tube, thereby completing lung expansion. The increasingly subatmospheric intrapleural pressure generated while the chest tube still remains is reflected in the gradually rising fluid level within straw and tubing.

When pleural space obliteration finally is achieved, chest tube holes become occluded. Before this time, while the chest tube still communicates with a pleural space, syringe and needle withdrawal of air from a chest tube connector injection port (or from the upper end of a self-sealing latex chest tubing) can establish a fluid column of any height: As mentioned, this is an effective way of applying suction to the chest tube when there is no air leak.

A Chest Bottle Suction Machine Increases Risk

Previously unobstructed air flow through the water seal bottle air vent can be partially blocked by a suction system with inadequate volume capacity. Under these circumstances, tension pneumothorax can easily develop while a chest bottle suction machine is "on" and working well, and the water seal bottle straw is bubbling merrily. In addition, many suction machines, when "off" or unplugged, resist air escape from the water seal bottle air vent or even obstruct the vent entirely; increasingly severe tension pneumothorax will then follow (for as long as life or pulmonary air leak may last).

For this reason, many commonly used chest bottle suction systems, of which hospital "wall suction" is a good example, are particularly unsafe when major pulmonary air leak exists. So take care to use only a turbine or other suction device that will always permit air to pass freely, regardless of whether it is "working" or "turned off."

How to Connect Mechanical Chest Bottle Suction

It is better to connect the sterile suction tubing to the air vent of the water seal bottle, rather than directly to the chest tube or chest tubing: In this way, suction is readily applied or swiftly disconnected, without losing the water seal function. Although significant pulmonary air leak will still be visible as bubbling at the water seal straw tip, tidal should not occur while using effective chest bottle suction (because, with or without air leak, the patient's inspiratory subatmospheric pressure never drops below the subatmospheric pressure being generated at the water seal by the suction device).

Suction is Never Lifesaving; When in Doubt, Disconnect

Addition of chest bottle suction to simple water seal chest tube drainage is never lifesaving. Therefore, suction should not be used if it would endanger the patient in the event of malfunction. Far too commonly, the suction device that is connected to the chest bottle cannot maintain continuously subatmospheric pressure within the chest bottle during rapid expiration or cough. It should be clear that

if the device cannot pump air away as fast as that air leaks from lung, the suction system is obstructive to escaping air (thus not only useless, but hazardous).

Unfortunately this describes far too many of the commonly used chest bottle suction pumps. For example, many years of repeated overflow of fluid and froth into the hospital wall-suction outlet can gradually reduce its effective aperture (and thus volume capacity) to dangerously inadequate levels. Though such wall suction can still achieve respectable levels of subatmospheric pressure in the absence of significant air flow, it can easily cause tension pneumothorax when attached to the chest bottle of a patient with major air leak. Yet, such inadequate or frankly obstructive suction devices are most likely to be utilized for uncommonly large pulmonary air leaks, when poorly functioning chest tubes are particularly hazardous - as in "Do something, even if it is wrong!"

Suction With Major Air Leak

Major pulmonary air leak may occur consequent to spontaneous pneumothorax, lung surgery, or other lung injury (particularly in the presence of emphysema). Lung always tears easily, and it is especially likely to leak huge volumes of air if emphysematous blebs or cysts have been opened in a misguided attempt to "suture-ligate the bronchial air supply" (see Chapter 13). Even when lung cysts are ligated whole, or twisted, bunched and over-sewn, or "wedged out" rather than opened, the flimsy visceral pleura can tear readily at needle or staple holes, causing considerable air leak for several days. A surgical decortication of lung also is likely to cause massive pulmonary air leak.

Adequate care of the patient with major pulmonary air leak begins with properly placed chest tubes, sufficient in number and large enough to permit easy air escape. When a significant air leak or relatively small chest tube results in persistent pneumothorax and lung compression, effective chest bottle suction can usually increase air flow through the chest tube: If air leak from lung is not equally accelerated by the suction, lung expansion should improve.

How Much Suction?

When dealing with many small air leaks, vigorous chest bottle suction may sometimes promote healing by pressing leaking lung surfaces against each other or the parietal pleura. One could equally argue, however, that small air leaks are more likely to close after surgery (by surface tension, fibrin plugs or whatever) if chest tubes are only connected to simple water seal, for small air leaks might tend to be held open when chest bottle suction overstretches surgically diminished lung.

Disregarding theory then, it is fair to say that patients all differ, so each should serve as his own "control": A trial of varying levels of suction often reveals a setting at which there is reasonable lung expansion and minimal bubbling; several days at this suction level will usually outlast these small air leaks. Incidentally, I have never applied more than 60 cm of water ("high") mechanical chest bottle suction to a residual pleural space (perhaps someone will eventually claim that higher-than-"high" suction can rapidly eliminate all residual pleural space problems, but that remains to be shown).

Rapid increases in chest bottle suction can be painful (conceivably even hazardous), so increase the chest bottle suction setting slowly while carefully observing the patient, to give "things inside" a chance to move around and stretch gradually (as mentioned, low suction will suffice to readjust the more mobile tissues of childhood). It is probably safer if the patient coughs vigorously while chest bottle suction is being increased; such voluntary efforts, or using positive-pressure ventilation plus

endotracheal suction to remove mucus plugs, will certainly aid lung reexpansion (see also reexpansion pulmonary edema, Chapter 5).

Foaming and Reflux From the Chest Bottle

Foaming of chest bottle contents can be a problem in high volume air leak situations: Not only is the current extent of bubbling obscured by froth, but that froth tends to travel from bottle to bottle until it is inhaled by the suction source. A bit of silicone antifoam spray, simethicone or ethanol, added occasionally to the chest bottle, can help control this.

But never put anything into a chest bottle that could endanger the patient if it accidentally ran back into the chest (which admittedly is a rather remote possibility during major air leak). This general prohibition includes priming the water seal unit with large volumes of distilled water, which could cause significant pleural reaction and possibly even hemolysis.

Accidental reflux of the water seal into the chest is best prevented by keeping water seal bottles well below the patient's chest, preferably on the floor. Excessively high fluid columns should be released by allowing air entry into the upper chest tubing, whether or not additional fluid appears to be sequestered within the chest.

Release of an unnecessarily high fluid column certainly decreases the likelihood of tubing fluid being pulled back into the patient's chest when the water seal bottle is accidently tipped. A continuous fluid column from the pleural space to the chest bottle would also be an ideal "bug-ladder" in the event of bacterial contamination of the water seal bottle contents (but if so, I have never recognized any problem from such a cause).

Avoid "Technical" Chest Bottle Air Leaks If Planning Chest Bottle Suction

The cap or stopper on a water seal bottle normally does not need to be air-tight for it serves only to stabilize the straw tip under the water seal surface, and to support the air vent. When chest bottle suction is applied, however, the entire chest drainage system should be sealed from the atmosphere to prevent overload of the suction apparatus by incidental atmospheric entry.

It is best if the chest tubing connector has a smooth lumen with the same internal diameter as the chest tubing, so that clots pass through with ease. If the originally supplied tubing connector tip will not enter the flared end of a smaller chest tube, however, a less desirable plastic "five-in-one" tapered connector can often be trimmed adequately from both ends with heavy scissors to fit.

To preserve the original connector (which may be worthwhile if it has an injection port for air entry or aspiration or chest fluid sampling), the original connector can be cut off along with a few inches of tubing (using sterile scissors on sterilized tubing) and reversed; the original connector end is then inserted into the remaining chest tubing: This provides a "connectorless" open chest tubing end that is then attached to the chest tube via an appropriately tailored, five-in-one connector. The final sequence (chest tube to five-in-one connector to short piece of chest tubing to original connector to rest of chest tubing) is more readily created than described.

As an occasionally useful alternative, the flared tip of a smaller plastic chest tube is carefully trimmed, moistened with an alcohol wipe and jammed snugly into (rather than over) the original chest

tube connector. This "insertion connection" can be secure and must be airtight (to prevent atmospheric entry from simulating ongoing pulmonary air leak). The usual lengthwise strip of tape (along chest tube, connector and tubing) gives additional security; one can also spray these connections heavily with a plastic film.

Chest Drainage Devices & Chest Bottle Suction
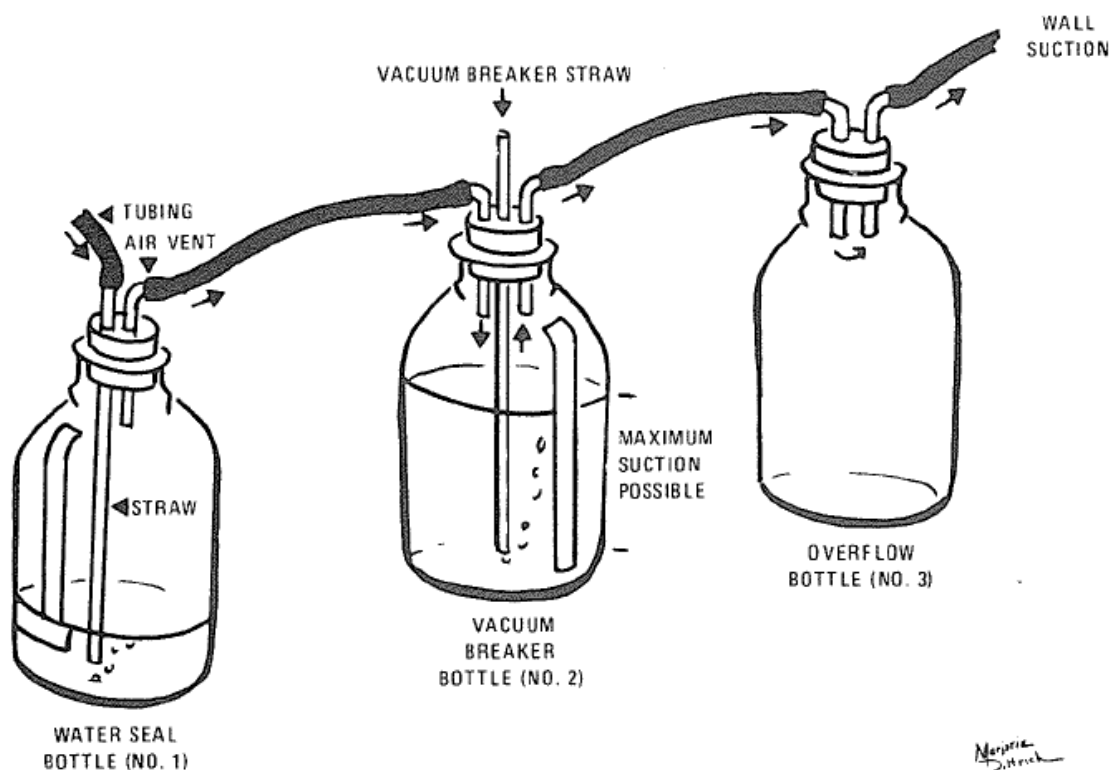
Figure 7.

Three-Bottle Suction Is Outmoded and Dangerous

Consider now the antiquated, three bottle suction system (Fig. 7). Bottle No. 1 is usually the water seal jug, No. 2 is a primitive but effective vacuum breaker (even an ungainly, one-gallon vacuum breaker will gradually evaporate, however, thus diminishing the amount of suction actually being applied) and No. 3 is a final trap bottle before the wall suction.

Undoubtedly this cumbersome arrangement often permits effective chest drainage, but it can also be unsafe, especially with major air leak. Over and over again, an emergency, dead-of-night call from a ward nurse reported, "Mr. X has something wrong with his tubes! They were blowing water all over, so I clamped them." My telephone instructions never varied "Take those clamps off immediately!" and "Disconnect the first (water seal) bottle from the others!."

Almost predictably, however, on my hasty arrival, the occluding clamps would still remain on chest tubes or tubing, with nothing disconnected. "I tried to take the clamps off but he started blowing water again." "I'm never sure which bottle does what." "My instructor told me never disconnect chest bottles...." Typically, at this point, the patient was blue but still gasping, and the floor wet (but

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"housekeeping was on the way"). The night nursing team had gathered at the nursing station and was "taking report."

When the chest tubes were (immediately) unclamped, a geyser spouted from the vacuum breaker straw of the No. 2 Bottle with each desperate expiration. This geyser simply indicated that the suction device was not evacuating air from the vacuum breaker bottle as fast as air was being delivered from leaky lung via the water seal (Bottle No.1).

Now, when any so-called "chest bottle suction" blocks the free passage of air in this fashion, instead of expediting air escape from the chest by sucking, the only way a patient on this three-bottle arrangement can decompress his pleural air pocket to the atmosphere is via the vacuum breaker straw of Bottle No. 2. The height of the geyser above bottle No. 2 bears some relation to the severity of tension pneumothorax that has been caused by the inadequate chest bottle suction system.

Unfortunately, as long as Bottles 1 and 2 remain connected, the patient's obvious tension pneumothorax will persist until enough fluid has been "geysered" out of the vacuum breaker bottle to expose the previously submerged end of the vacuum breaker straw (and that means blowing a lot of fluid). If the patient has the strength to survive this long, he will finally have returned himself to simple water seal chest drainage: However, the "long air vent" (newly established by his extraordinary respiratory efforts) through the vacuum breaker straw in Bottle No. 2, will continue to offer significant additional resistance to escaping air.

Obviously, the only sensible solution is rapid disconnection just beyond the water seal bottle, which immediately returns the patient to simple water seal. If chest bottle suction does seem essential (to increase air exhaust from the pleural cavity, and improve lung expansion by a further decrease in pleural cavity pressure), then a safe, turbine-type, chest bottle suction device may be reconnected to the air vent of the water seal bottle (but all concerned must understand that inadequate suction is far worse than none).

The Vacuum Breaker Straw Also Reports When Suction is Adequate

Electric pumps and wall suction devices sometimes use the underwater-straw type of vacuum breaker as both vacuum gauge and vacuum control. This straw clearly limits suction by the depth to which its tip is submerged. With the vacuum breaker straw tip submerged to 15cm, for example, air will be sucked down within the straw by any effective suction, and it will bubble forth freely from the straw tip (thus "breaking" excess vacuum) whenever the suction source applies more than 15 cm. suction to the vacuum breaker container (which is normally interposed between the suction source and the patient, as in the three bottle arrangement described above).

Not always as obvious, however, is the fact that unless the vacuum breaker straw bubbles continuously, the anticipated 15 cm of suction is not actually being applied. In fact, close observation often reveals "no bubbling" and (1) the vacuum breaker straw fluid level located somewhere between the vacuum breaker straw tip and the overlying water surface: This means that "not enough suction is being applied" to cause bubbling at the submerged vacuum breaker straw tip, or (2) the vacuum breaker straw fluid level may rise above its water surface level during expiration: The latter oscillation documents above-atmospheric pressure within the vacuum breaker bottle, rather than suction.

Above-atmospheric pressure within the vacuum breaker unit again means that more air is passing

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through the water-seal chamber than the suction can handle. In other words, the suction device is actually impeding expiratory air escape from the water seal bottle air vent, or (3) as in the extreme, unfortunately too common, situation described above, the vacuum breaker straw may even be spouting into the air, or fluid may be running down the outside of the vacuum breaker unit: This again confirms tension pneumothorax, due entirely to relative obstruction of the water seal bottle air vent by ineffective suction in the presence of significant air leak (or perhaps totally inadequate suction is blocking any persistent air leak).

Summary: Actual interference with air escape through the chest bottle air vent – due to a partially or totally obstructive suction system beyond the vacuum breaker - is an all too common cause of persistent postoperative tension pneumothorax, clotted hemothorax, empyema and even death.

The Vacuum Breaker Fluid Level Drops Through Evaporation, But the Water Seal Fluid Level Does Not

When incompletely humidified room air is constantly bubbled through a fluid-filled vacuum breaker system, there is a continuing, gradual decrease in the vacuum breaker fluid level, and a corresponding decline of the suction being applied. Obviously, the higher the suction applied and the lower the vacuum breaker setting, the more bubbling is seen at the vacuum breaker straw, and the more rapid the loss of vacuum breaker fluid, and the lower its suction setting, and so on.

Conversely, the water seal bottle fluid level is never diminished by air that bubbles through it, for that air has already been fully humidified during its passage through the respiratory tree (so a large-volume pulmonary air leak should greatly increase "insensible" water losses, although I have not recognized that as the cause of clinically significant dehydration or excessive evaporative heat loss to the point of hypothermia).

As mentioned, turbine-type pumps (such as ordinary vacuum cleaner motors) can safely be used for chest bottle suction; even if unplugged or set for unduly low flows, they will not significantly increase resistance to egress of air from the water seal bottle air vent. Except under unusual circumstances, a chest bottle suction pump should always include such a "blow by" capability, as chest bottle suction is not essential, or even as useful as simple water seal, for most chest drainage problems.

Conclusion and Review

Chest bottle suction is occasionally very helpful in drawing lung and chest wall together when air leak is present. However, regular use of low-level chest bottle suction needlessly increases cost, noise, complexity and risk to the patient. A properly designed (stable, light-weight, unbreakable, spill-proof) inexpensive, simple water seal system should cause minimal back pressure to egress of air from the chest, and its straw fluid level will provide a constant monitor of intrapleural events. Such a simple water seal system makes even outpatient chest tube care both safe and practical.

Accumulating drainage can readily be decanted from the chest bottle air vent without loss of water seal if the air vent protrudes sufficiently far into the chest bottle to guarantee that the inverted unit will retain whatever minimal fluid volume is necessary for a water seal. During the short time that a water seal unit is thus inverted, fluid actively draining from the air vent maintains a gentle suction on the chest tube if there is no air leak (a major air leak may be insignificantly retarded during such brief fluid drainage, as demonstrated when the draining fluid is blown out under pressure). The risk of bacterial

contamination during such occasional decanting via the air vent is probably similar to that faced while voiding (namely minimal).

Unlike more complex pleural drainage systems, an ordinary water seal bottle resumes function as soon as it is righted: Undesirable buildup of drainage within a simple water seal chest drainage bottle is immediately evident and easy to remedy. Conversely, it is less evident and thus more hazardous when a non-turbine source of suction malfunctions (unfortunately, almost a regular occurrence). Only hoary tradition and slick marketing can account for the continued clinical application of either the ponderous, confusing three bottle system or its costly, flimsy, more hazardous, and ever more confusing descendants.

CHAPTER 9: CHEST DRAINAGE WITH THORACOTOMY

Air and fluid tend to accumulate postoperatively;... So place two or more chest tubes at major thoracotomy;... Adequate drainage requires appropriate tube position;... Chest tube placement at thoracotomy;... Errors in setting up a water seal: Partial misconnection;... With total misconnection, the water seal bottles erupt;... No water seal; ... Bed position after thoracotomy; ... Try to avoid reoperation for bleeding;... Intrapleural saline helps detect bleeding during chest closure;... Intrapleural irrigant temperature;... Postoperative hemothorax;... Case report: Reexploration for bleeding;... Reexploration for bleeding;... Bleeding and external blood loss;.. Rules to bleed by;... Treatment of stable clotted hemothorax;... Streptokinase for clotted hemothorax;... Never clamp a chest tube, except after pneumonectomy;... Routine chest tube placement after adult pneumonectomy;... Technique of postpneumonectomy pleural cavity pressure adjustment by chest tube and simple water seal bottle;... Check the tracheal position frequently;... Excessive fluid production after pneumonectomy;... Subatmospheric intrapleural pressure is especially helpful if the phrenic nerve was divided during pneumonectomy;... Antibiotics;... Role of prophylactic digitalization;... Treating Low Serum Potassium or Magnesium Can Reduce Dysrhythmias;… Restrict fluids after pneumonectomy;... Postpneumonectomy air leak from the bronchial stump;... After pneumonectomy: Things for the nurse to consider;... Clean and dirty thoracotomy wounds;.. Poor risk cases sometimes cannot be turned for posterolateral thoracotomy;... Postoperative dressing for posterolateral thoracotomy;.. Tape allergy;... Postoperative roentgenogram of the chest.

Air and Fluid Tend to Accumulate Postoperatively

Pleural fluid production is usually greatest soon after surgery, before raw and inflamed surfaces begin to heal, but such fluid output can persist for days or even weeks. Measurable fluid drainage is generally not encountered after infant thoracotomy, presumably due to more rapid healing.

Excessive trauma and inflammation appear to increase the volume and protein concentration of pleural drainage, and thus the likelihood of intrapleural loculation and coagulation of a serous or bloody effusion. When operating in the face of chronic inflammation, one should realize that the "healing process" (including vigorous fibrosis) is already "under way" at the time of surgery: One must then achieve early reexpansion lest lung become trapped in some undesirable position.

The usual bonus for prompt elimination of any postoperative pleural space is diminished fluid production from pleural or surgically denuded subpleural surfaces. In addition, expanded lung rubbing against the parietal pleura will soon disperse and defibrinate moderate amounts of bloody drainage, thereby expediting chest tube drainage.

Following pneumonectomy, the air-filled pleural cavity gradually shrinks even as it fills with fluid: Intermittent early decompression of the overlying air pocket therefore helps to prevent tension pneumothorax. Within a day or so after surgery, however, fluid produced will more or less replace air absorbed from the remaining gas pocket, so further pressure adjustments of this clotted hemithorax usually become unnecessary as healing progresses.

One might anticipate that a half-empty pleural cavity (a large residual pleural space containing a surgically diminished or atelectatic lung) would undergo a similarly benign series of events. Unfortunately, the small, poorly aerated lung "adrift in a fibrinous sea" will almost surely become infected: Secondary infection of its surrounding, stagnant pleural fluid then becomes almost a foregone conclusion.

It seems clear that inadequate chest drainage and insufficient clearing of pulmonary air passages can greatly increase the likelihood of chronic lung infection and empyema after thoracotomy: If lung then becomes firmly entrapped within an infected pleural peel, surgical decortication will probably be indicated (hopefully, with more attention paid to chest drainage and pulmonary toilet the second time around). In theory, this complication and its correction could alternate indefinitely: In fact, postoperative empyema patients tend to become very sick and die.

So Place Two or More Chest Tubes at Major Thoracotomy

It is "infinitely(!)" easier and quicker to place two or more large chest tubes during surgery for infected lung (when copious or persistent postoperative bloody drainage and perhaps air leaks can reasonably be anticipated) than it is to deal with an inadequately drained, moderate-sized, infected pleural pocket in an unhappy feverish post-operative patient. Postoperative closed thoracostomy is frequently awkward and difficult; if the collection requiring drainage is located next to esophagus, heart, aorta, or high in the apex, such tube insertion may even become "impossible" (a determination made by balancing possible risks and benefits of chest tube placement against the likely morbidity with antibiotic therapy alone).

I have usually opted to "watch" inaccessible pleural fluid collections following lung surgery, until they either grew large enough for safe chest tube insertion or resolved during vigorous antibiotic therapy (see Chapter 4). Certainly, in some cases (as with a possibly infected fluid collection next to a recent aortic graft or suture line), early reexploration and more adequate drainage may be the more "conservative" approach. Radiologists are becoming increasingly skilled in percutaneous drainage of purulent collections, so consultation is often appropriate before embarking on major therapy.

Adequate Drainage Requires Appropriate Tube Position

Any major thoracotomy involving lung (or even just the lysis of dense pulmonary adhesions) justifies the use of two chest tubes: The anterior tube tip is always located close to the apex, while the posterior tube may be positioned nearer the base. As the anterior apical tube is essential to assure prompt lung expansion into the upper hemithorax, it is far more important: Once the lung has expanded to fill the pleural cavity, the anterior tube will evacuate fluid just as effectively as a low, posterior tube.

The posterior tube primarily prevents dependent dorsal blood and serum accumulations early after thoracotomy (which is when such collections pool and clot most readily if not promptly evacuated). If expecting significant postoperative air leak, it is wise to locate the posterior tube tip above midthorax (even up to the first rib), so that its tip also expedites early postoperative air removal (while its lower holes are drawing off fluid).

An occasional case with massive air leak, or significant bacterial contamination, or where copious pleural fluid production can be anticipated, is best treated with three large chest tubes. Extra tubes cost little in money, time or discomfort, and they can markedly shorten hospitalization, or even prove lifesaving.

Intrathoracic procedures not associated with pulmonary air leak (e.g., a simple lung biopsy, some cardiovascular procedures, hiatus hernia repair) may require only a single anterolateral chest tube when hemostasis seems assured. Even if some postoperative blood loss should occur, the anterior tube will serve as an excellent drain for fluid - as long as early, full expansion of a normal lung is confidently

anticipated.

Intrathoracic esophageal suture lines usually warrant a nearby posterolateral chest tube directed toward the apex: This can be left in place until a contrast study demonstrates leak-free healing. Here, the anterolateral chest tube is often removed first, after the lung has fully expanded.

As discussed in other chapters, a solitary, low, posterior chest tube cannot correct pneumothorax, so if an air leak remains possible, a high anterolateral chest tube should always be placed, to prevent pneumothorax and also clotted hemothorax. Pneumothorax increases the likelihood of clotted hemothorax because only a fully expanded lung will redistribute any postoperative blood loss over all pleural surfaces: Unless "disturbed" and defibrinated early in this fashion, fresh blood tends to stagnate, pool and clot, rather than move toward any nearby chest drainage tube.

One patient with unexpected postoperative lung air leak and only a posterolateral chest tube in place, developed tension pneumothorax every time he lay back (because his lung then fell onto the chest tube), until an additional anterior closed thoracostomy was carried out. In this patient, it was the anterior tube that prevented his postoperative blood loss from clotting under an undrained air pocket. And the early lung expansion achieved after placement of the high anterior tube, improved intrapleural hemostasis and also expedited drainage via the posterior chest tube.

Comment: The less experienced surgeon is urged to "err" on the side of too many chest tubes, at least until he is quite sure that his tubes are appropriately placed and will remain so. I have never seen a patient harmed in any way by placement of an additional intraoperative chest tube that later proved unnecessary.

Chest Tube Placement at Thoracotomy

To avoid discomfort and tube blockage when the patient lies back, routine postoperative pleural cavity drainage tubes are placed so that they do not not penetrate chest wall skin much behind the anterior superior iliac spine (which is easily palpated through surgical drapes).

Method: Through a short transverse skin incision, usually well below the thoracotomy wound and close to the level of diaphragm attachment to chest wall, a large Kelly clamp is driven obliquely upwards into the chest cavity (just over a rib, in order to avoid the intercostal nerve and artery). The length and direction of the resulting "tunnel" through subcutaneous tissue and muscle should assure an appropriate "lie" for the chest tube. If two chest tubes are used, they are usually inserted 3 to 5 cm. apart, to permit convenient individual, or rapid simultaneous, tube removal.

One hand remains within the chest during such Kelly clamp insertion, to protect diaphragm, liver, spleen and lung from the carefully advanced (but nonetheless, occasionally "plunging") clamp tip. The chest tube is then grasped and pulled out of the chest by its blunt (usually beveled and flared) "connector" end, and the chest tube tip is appropriately positioned.

A chest tube is always located so that no part of any tube side-hole remains outside of the pleural cavity. Subcutaneous emphysema can result from leaving a chest tube side-hole within the chest wall during significant lung air leak. Furthermore, a side-hole partly exposed to the atmosphere at skin level could mimic major pulmonary air leak, as outside air that enters this side-hole during inspiration will then bubble from the water seal straw tip during expiration.

Of course, with one or more side-holes "emerged" above skin level, one would expect these "outside" chest tube openings to inactivate the distal chest tubing entirely: And the water seal straw would be exposed only to atmospheric pressure, rather than reflecting intrapleural pressure changes. Such an open-to-the-atmosphere chest tube also allows underlying lung to relax, and most probably is about to fall out of the chest wall entirely. (These remarks might not entirely apply to a more chronic chest tube site, for irritated, edematous granulation tissue has an increased resistance to penetration by air: In addition, underlying lung could have become fixed in position, next to a stable pleural space.)

Chest tubes should point cephalad and lie between lung and chest wall. To maintain effective drainage as the diaphragm moves up and lung expands in the early hours after surgery, all tube side-holes are placed higher within the chest than the dome of the diaphragm, since only the most distal (outermost) side-hole may be cleared during "milking" (see Chapters 10 and 11). A tube low in the lateral sulcus (or under lung, or within any other pleural recess) is not uncommonly sandwiched between diaphragm, lung and chest wall, and thereby "prematurely" excluded from the remaining apical pleural space.

The upper anterior tube can also be brought out through skin via the first or second anterior interspace. This leaves only one tube entering laterally but has no effect on tube function. When a significant length of chest tube is inserted from in front, it seems to lie best if directed laterally and superiorly, rather than straight in and up, or medially toward mediastinal structures.

To protect the brachial plexus and subclavian vessels, the chest tube tip is always kept below its adjacent first rib, even if the tube must be pulled back a bit after the initial postoperative chest roentgenogram. Sometimes an "awkward chest tube lie" during thoracotomy is best corrected by abandoning the first skin incision and tube tract entirely, and starting over at a new site.

Chest tube size is generally more important than type. The tube lumen should be adequate to handle all drainage expected, and the tube walls should be stiff enough to maintain patency and stay in the intended position. Various surgeons prefer Foley catheters, bent-tip catheters, or rubber or plastic catheters.

A Foley catheter lumen is smaller for any given external tube diameter, so this speaks against its routine use. Rubber catheters also seem to clot much more readily than plastic chest tubes. Silastic chest tubes are most likely to remain patent during fresh blood loss (if milked frequently) but they too can become solidly blocked by clot, especially after drainage slows.

Errors in Setting Up a Water Seal: Partial Misconnection

Occasionally, during completion of a thoracotomy closure, vigorous bubbling is noted in both water seal bottles, even though the surgeon feels certain that significant lung injury did not occur. In such cases, chest bottles should be inspected for a possible error in arrangement. Not uncommonly, one finds that an inexperienced nurse has misconnected the chest tubing to the air vent emerging through one bottle cap, rather than to the adjacent underwater straw (disposable water seal units generally arrive with chest tubing already secured to the straw, which eliminates such misconnection errors).

If one of two chest bottles is misconnected in this fashion, there is no ill effect while positive-pressure controlled ventilation continues. With the onset of unassisted breathing, however, a vigorous air leak begins (this sequence is opposite to that more commonly seen, when multiple small air leaks

stop soon after positive-pressure ventilation is discontinued).

Close inspection of the correctly connected water seal straw then reveals each inspiration lifting saline as it should; meanwhile, the unconnected and therefore "open" water seal straw in the other bottle bubbles vigorously (serving as a "vacuum breaker" for below-atmospheric inspiratory intrapleural pressures). On expiration, the properly connected water seal straw bubbles while the same above-atmospheric intrathoracic expiratory pressures transmitted into the misconnected bottle just push the fluid level up within the unconnected straw (simulating tidal). This "half-wrong" setup causes persistent partial pneumothorax but can be tolerated for a considerable time.

With Total Misconnection, The Water Seal Bottles Erupt

With both chest tubings misconnected to water seal bottle air vents rather than their underwater straws, the resulting tension pneumothorax is far more serious. To begin with, a large pleural air space commonly persists during closure of a thoracotomy incision (regardless of pulmonary air leak), simply as a consequence of the standard posterolateral thoracotomy position.

For in that lateral position, the mediastinum sags down onto the dependent "good" lung, while abdominal wall relaxation simultaneously allows visceral weight to pull the diaphragm more inferiorly than usual. Thus enlarged, the pleural cavity easily dwarfs its enclosed lung during chest closure, especially if lung size was diminished by surgery.

The enlarged pleural air space between lung and hemithorax usually persists until the patient is turned to the supine position and relaxants wear off. Firm abdominal muscles then push viscera and diaphragm back up into the thorax, and the mediastinum no longer sags away from the midline. Although the increasingly compressed pleural air pocket has ready access to the chest bottle (via chest tubings misconnected to air vents), the only outlet from these "reversed," water-sealed bottles will be via the unconnected water seal straws (still open to the atmosphere above and submerged in saline below).

With its one-way valve function thus intact but "set up backwards," the water seal straw turns into an ordinary vacuum breaker. But a closed vacuum breaker bottle attached to the chest tubing will not relieve tension pneumothorax during positive-pressure respiration, or while coughing or straining on awakening from anesthesia. The only way a patient in this predicament can relieve his non-air-leak-generated, end-of-thoracotomy tension pneumothorax is to evacuate water seal fluid up the underwater straw and out onto the floor (Fig. 8).
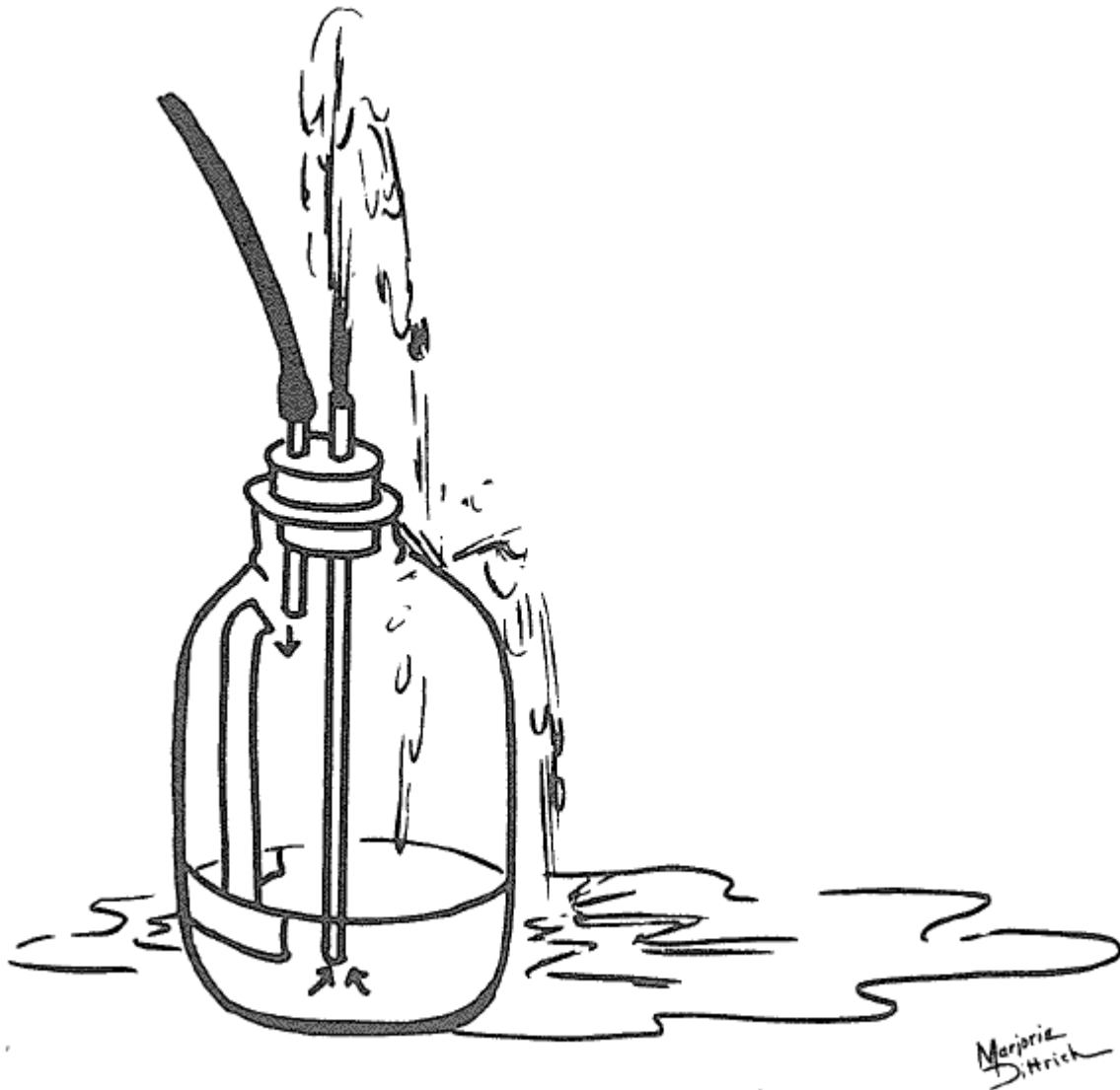
Figure 8.

If the patient can then endure until one of the straw tips emerges from its newly diminished water seal surface, his tension pneumothorax (roughly comparable to the height of the geyser above the "water seal" surface) becomes a much less serious "open chest tube" simple pneumothorax.

No Water Seal

An insufficient or absent water seal "prime" causes persistent pneumothorax without visible bubbling (another "open chest tube" problem). An arrangement where only one of two chest bottles has enough prime to achieve water seal function will also mimic persistent lung air leak; here only one bottle will bubble, although both chest tubes can expel air during expiration.

Bed Position After Thoracotomy

Barring hypotension or other contraindication, it is generally advisable to keep the head of the bed somewhat elevated during the first postoperative days, for this can improve both air exchange and chest

tube drainage. When sedated or uncomfortable patients (who move poorly and avoid deep breaths) remain supine soon after thoracotomy, they tend to develop posterior intrapleural accumulations of fluid or clot, as well as dependent rales, atelectasis and even pulmonary consolidation.

A fully expanded and functioning lung tends to distribute bloody pleural drainage over its entire surface (by respiratory movements and capillary attraction), while a collapsed or partially inflated lung simply floats within or upon any intrapleural fluids. Until lung expansion is complete, therefore, the semi-sitting position at least can bring bloody drainage down onto the diaphragm; there it is far more likely to be agitated, defibrinated and drained than if allowed to lie in still layers along the posterior chest wall of a supine patient.

Try to Avoid Reoperation for Bleeding

Continuing postoperative blood loss can necessitate reexploration for vessel control and evacuation of clot; this is always hard on a surgeon's ego and reputation. Significant postoperative hemorrhage can be kept to a minimum by performing a "final" reinspection for ongoing blood loss after chest tubes have been placed and all is ready for closure. Sites worth rechecking are the divided inferior pulmonary ligament (especially on the mediastinal side), all divided bronchial vessels, any suture lines and ligatures, and all areas of dissection.

"Hard to see" sites of divided chest wall adhesions or stripped pleura are checked for bleeding by lightly pressing a dry sponge against the suspect area for a few moments. This maneuver is also helpful at the intrapleural "site of entry" of a chest tube, especially in the coarctation patient with huge, tortuous, intercostal arteries, or for investigating other inaccessible areas of injury or rib fracture (down between diaphragm and ribs, for example). Divided intercostal muscle is also inspected for any "pumping" arterial branches that might still require control, especially toward the posterior end of the incision.

Thoracotomy for routine coarctation of the aorta is performed through the bed of the resected left fourth rib to reduce bleeding from extensive intercostal collaterals. During this procedure, all of the relatively enormous intramuscular chest wall arteries are ligated individually with fine suture material, rather than electrocoagulated. Even with meticulous hemostasis, reexploration for major postoperative blood loss into the chest wall is not uncommon after coarctation repair (chest wall veins are not enlarged in such patients, of course, as there is no corresponding need for large collateral veins to bypass the normal vena cava).

Intrapleural Saline Helps Detect Bleeding During Chest Closure

It is frequently helpful to leave 200 to 300cc of warmed saline in the pleural cavity, and then watch for significant reddening of this during closure. Pneumonectomy patients, particularly, should have instillation of a few hundred cc of warm saline into the pleural cavity, to see if this fluid remains clear or pink, or if it rapidly turns bloody.

Warmed physiologic salt solution (perhaps 500 cc or so) can also dilute and thereby reduce, early clot formation during thoracotomy closure. Once the patient is placed in the supine position, the anterior tube will be on top of lung, allowing more effective chest drainage, and thus better expansion of lung, which improves drainage, and so on.

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"Soon after the operation ends" is the ideal time to "play with the tubes," draining them repeatedly into the chest bottles until the chest tubing no longer fills and the patient can tolerate a semi-sitting position. Five or ten minutes of persistent tube manipulation right after thoracotomy may be the best way to detect early postoperative hemorrhage and prevent clotted hemothorax.

In addition, the presence of the surgeon is often helpful for the first few postoperative minutes in the recovery room or intensive care unit, when others involved in the patient's care may be too busy sorting out and calibrating fluid lines to notice that the patient has stopped breathing or whatever.

Removal of excess fluid from the water seal bottle will minimize back pressure to air evacuation. Even without pulmonary air leak, it is well to empty chest bottles down to their prime level if there has been significant fluid buildup: The bottles will then be considerably lighter and also less worrisome in appearance (non-surgeons often view any red chest drainage as blood, so this gives them less "blood" to worry about).

Intrapleural Irrigant Temperature

Never irrigate the chest with a cold solution, as this may slow or stop the heart. On the other hand, a significantly hypothermic patient can often be rewarmed effectively by copious intrathoracic irrigation with uncomfortably warm saline (see Chapter 3).

Postoperative Hemothorax

Despite all efforts at hemostasis, an occasional patient will bleed significantly after surgery and require reexploration.

Case Report: Reexploration for Bleeding

A forty-eight-year-old woman underwent thoracotomy for resection of a necrotic neurofibroma with central sarcomatous elements. Although closely adherent to her aorta, the mass was removed intact and without significant blood loss. At the end of surgery, the chest tube tidaled well (2 to 10cm above saline level). There was no bubbling at the straw or significant bloody drainage.

As he was leaving the recovery room, the surgeon became aware that the patient was suddenly "doing poorly." Her chest tubing, now full of blood, felt warm: She was extremely pale, gasping, and obviously agonal. The thoracotomy incision was therefore torn open and her descending aorta compressed barehanded, deep in a pool of liquid and clotted blood. At immediate reoperation, a degenerated, still ligated intercostal artery was found "blown off" the aorta. The origin of this intercostal from the aortic media was carefully sutured, other ties were rechecked, and her chest again closed.

This sequence recurred twenty minutes after her second return to the recovery room, again with warm tubing and a rapidly rising, bloody fluid level in the water seal bottle. Fortunately she was not agonal this time, so thoracotomy could be delayed until the operating room was reached (and her husband out of view). Another intercostal blowout had occurred.

After suturing this second aortic leak, the thoracotomy was left open for two hours, while the team alternated coffee breaks. When no further bleeding developed under direct observation, the incision

was closed for the third time, and she did well, remaining afebrile and healing rapidly, with no air leak and minimal blood loss. Chest tubes were left in place for five days, as some serous drainage persisted until then (with minimal tidal at 15 or 20cm above water seal level in both bottles).

The patient was discharged to her remote rural home on the tenth postoperative day. One week later, her general physician called in to report that he had rehospitalized her because of a sudden onset of left chest pain and dyspnea. Repeat chest roentgenogram showed complete opacification of her left chest, and the patient was markedly anemic (despite a "normal" hematocrit determination the previous week).

No specific treatment seemed appropriate or was required for this third episode of bleeding. Three months later, chest films showed an 8 to 10cm "peel" about her lung. Over the next year, this gradually resolved. Chest roentgenograms were essentially normal two years following thoracotomy, with no evidence of recurrent tumor.

Conclusions: (1) When current blood loss flows out at such a rate that the chest tubing feels warm and the chest bottle fluid level is rising rapidly, the diagnosis clearly is major intrathoracic hemorrhage. (2) Normal pleura can absorb a major clotted hemothorax (this one probably would have cleared far more rapidly with proper breathing exercises - see Chapter 3).

Reexploration for Bleeding

Continued intrathoracic blood loss following thoracotomy or other trauma may be an indication for surgical exploration. Massive blood loss that immediately threatens the survival of an otherwise salvageable patient, clearly requires immediate control. Ongoing moderate blood loss that exceeds blood replacement capabilities must also be stopped. The main decisions to be made in such cases have to do with the timing of an essential intervention and with "prioritizing" crucial matters such as control of blood loss, replacement of blood, ventilation, sterility and available facilities (operating room, supplies and equipment) and skills (anesthesiologist, surgical nurses and assistants).

Common experience and surgical judgement both suggest that maintaining the circulation of blood is more important than uninterrupted ventilation. Under some circumstances, a well-oxygenated healthy individual can even stop breathing voluntarily for more than ten minutes, without coming to grief.

In my own practice, poor communication at the operating table has twice resulted in prolonged absence of ventilation ("off cardiopulmonary bypass" for seven to fifteen minutes before ventilation was resumed), without any apparent ill effect. The more prolonged of these two apneic intervals was quickly identified and corrected when marked bradycardia and hypotension drew our attention to the absence of ventilation.

We now take care to speak loudly and clearly with "pump off" clearly enunciated by both surgeon and perfusionist, followed immediately by a "respirator on" similarly requested and then announced by surgeon and anesthesiologist (even if ventilation is already in progress).

Circulatory arrest, too, can be tolerated for short periods of time under special circumstances (hypothermic drowning or surgery). Cardiac arrest due to loss of circulating blood volume is generally fatal, however, unless immediate volume replacement with oxygenated blood can be achieved, during a

period of externally supported circulation and rewarming. With adequate circulation, ventilation can readily resume following a period of apnea, but no amount of ventilation will help the patient with absent blood volume.

It is evident that control of massive life-threatening blood loss must usually take priority over ventilation and other considerations. That does not mean, of course, that one can ignore ventilation (or the need for a great deal of help and sterile equipment) until final control of bleeding is achieved. The point made here is simply that, in an emergency, the most urgent of various life-threatening problems (such as massive bleeding) is preferentially dealt with first; other life-threatening problems (for example, absence of air exchange) will, in turn, become increasingly urgent, until they finally kill the patient if not corrected.

Bleeding and External Blood Loss

Surgery would be far simpler if all blood that escaped its normal, intravascular location was instantly delivered externally into a sterile, graduated container, from whence it could be reinfused without delay or detrimental change. Despite some improvements in the materials and technique of chest drainage, the above goal is rarely, if ever, attained.

In the first place, we are handicapped by the extent to which we must still rely upon indirect evidence to inform us of the source, amount and timing of blood loss into the closed chest. There are too many variables (such as clotted intrathoracic blood versus warm chest tube drainage, roentgenographic changes that "might" represent blood loss, cardiovascular pressure changes measured directly or indirectly, blood gas, hemoglobin, electrolyte and other solute determinations that might reveal cardiac or metabolic dysfunction which could be from other cause than blood loss), too many machines that can malfunction (from respirators to monitors to blood sugar or blood gas analyzers), and too many people involved (all subject to human error) for estimates of blood loss ever to be consistently accurate.

All too often, when we reexplore a chest for "continued active bleeding" we find only clot; or perhaps we decide that the blood loss has ended and "head home," only to be recalled shortly to correct continuing life-threatening hemorrhage. In some centers, where many individuals participate in numerous, relatively standardized cardiovascular procedures, a relatively specific "cookbook" approach to postoperative bleeding has been instituted: "So many cc blood loss by chest tube over such-and-such time means that you will reexplore the patient" ("Sincerely, The Boss").

Chest tubes can clot, however, and parameters, paradigms and even professors may change (given enough time), so it is wise to think through the decision process, even if currently constrained from using your own best judgement by the edicts of the Absolute Monarch. Ignoring "house rules," therefore, what is the best way to evaluate the patient who is clearly neither bleeding to death nor "unsalvageable" (i.e. could not survive another exploration, regardless of how much he is bleeding).

Rules to Bleed By

Ongoing moderate blood loss requires persistent blood volume replacement (see Chapter 14). Continuous blood volume replacement has risks (hypothermia, coagulation disorders, lung problems, metabolic derangements, allergic reactions, viral infections) that must be balanced against the risk of immediate surgical exploration.

Blood lost from blood vessels but not effectively drained out of the chest, will collect within the chest. In a small "closed" space such as pericardium, relatively minor blood accumulations can be detrimental or fatal. Ditto for larger clot accumulations within the pleural cavity (but the volume of intrapleural blood will usually not become the primary problem unless the bleeding patient with normally elastic lung has received extensive fluid resuscitation, for one hemithorax can accomodate almost an entire normal blood volume).

It is very unlikely that any patient suffering from cardiac tamponade, or severe lung and heart compression by accumulating clot, is ever "too sick" to undergo surgery (unless an additional "definitely fatal" condition currently coexists). Rather, a severely symptomatic patient with ongoing clot accumulation in chest or pericardium is usually too sick not to undergo surgical decompression and hemostasis.

Unexpectedly large blood losses may cause the surgeon to select early reoperation, though an even greater blood loss might properly be treated "conservatively" if anticipated. Thus major decortication or segmental resection is often associated with more postoperative blood loss than routine lobectomy or lung biopsy, because more damage to small blood vessels usually leads to more bloody drainage. However, persistent blood loss from the chest tube following simple lung biopsy suggests bleeding from a significant vessel that could require surgical control (in other words, many small vessels usually stop bleeding soon after surgery, while larger ones frequently do not).

When post-thoracotomy chest tubes become embedded in clot (one can usually detect this by "feel" while "milking" the chest tubing; air is readily pushed into the chest, but nothing comes back out), and x-rays show increasing haziness or fluid density in a patient with other evidence of ongoing blood loss, or if a patient requires continuing transfusion to remain stable when significant blood loss was not anticipated, reoperation is often useful. Sometimes, at reoperation, one or more bleeding vessels can be identified; at other times, only clot is evacuated (but at least the chest drainage tubes work well thereafter; also more and larger chest tubes can easily be placed if that now seems appropriate).

Even after blood loss has ceased, the ongoing breakdown of clot or other blood proteins (into smaller, more numerous particles, with increased osmotic activity) can attract more fluid into a limited space, causing progressive distress. There are times when an appropriate surgical response might include breathing exercises, needle drainage, small tube cannulation, closed thoracostomy, finger mediastinotomy, pericardial window, sternotomy or thoracotomy. These specific topics are discussed elsewhere.

Summary: The surgeon is vitally concerned with intrathoracic blood loss and intrathoracic blood accumulations. When postoperative blood loss does occur, he tries to externalize all blood accumulations by means of effective chest tube drainage: As this drainage is never 100% effective, fluid and clot accumulating within the chest can affect vital functions.

Indirect evidence for ongoing blood loss is often difficult to evaluate. However, when reexploration for bleeding must be carried out within twelve hours of surgery, one will usually require excellent operative exposure to allow direct attack on the source of blood loss; procedures carried out after that time should require only limited access, as by then, decompression of an overfilled space will be the probable technical problem, rather than control of hemorrhage.

Treatment of Stable Clotted Hemothorax

Clotted hemothoraces occasionally require surgery to control ongoing blood loss, decompress a massive symptomatic clot, or evacuate infected clot. When stable, however, they usually respond to appropriate chest tube placement or with simple persistent breathing exercises (see Chapter 3).

The major part of any hemothorax will usually liquify within one to five days after clot formation. Roentgenographic evidence for liquefaction of intrapleural clot includes any significant increase in volume of intrapleural "fluid" that is not due to bleeding, a fluid meniscus or new layer separating lung from chest wall, or a shift of the "clot" location associated with a change in the patient's position.

Not uncommonly, "increased density" (greying) of the normally "clear" (dark) lung fields on a supine AP chest roentgenogram will be the only evidence for a posterior clot or free fluid collection. An x-ray beam carelessly centered over the opposite lung field (rather than over the mediastinum, on a PA or AP film) may mimic the "unilateral in-creased density" appearance caused by intrapleural fluid.

In theory, any early or late increase in pleural effusion might also indicate new, or slow but continuing, intrapleural blood loss: Such late bleeding is very unlikely, however, in the absence of infected vascular suture lines, anticoagulant therapy or other major clotting disorder. In addition, delayed bleeding lowers the hematocrit, while osmotic capture of extracellular fluid by a dissolving intrapleural clot would more likely cause hemoconcentration (either problem can cause hypovolemia as well- see Chapter 14).

Except in the face of pleural infection or an already established fibrotic process (as after decortication), there is no great urgency in completing the evacuation of a clotted or liquifying hemothorax (in terms of a few days), except to relieve severe symptoms and signs of cardiac or pulmonary compression. There is also no harm in leaving a non-functioning plastic chest tube planted firmly in clot for a day or two, pending clot liquefaction.

Under such circumstances, however, any tube may (and a rubber chest tube will) clot too solidly to reopen. It is usually reasonable, therefore, to remove a solidly clotted chest tube from a clotted hemothorax. This often improves patient comfort and mobility during the wait for clot liquefaction (meanwhile, the patient works on his frequent deep-breathing exercises. If still necessary, thoracentesis or closed thoracostomy can be performed later, perhaps through a better site).

Streptokinase for Clotted Hemothorax

I have not ventured into streptokinase treatment of clotted chest tubes or a clotted hemothorax for more than two decades. Perhaps the purified products now available (streptokinase, urokinase or tissue plasminogen activator) will prove more acceptable for intrapleural instillation than the initially marketed "streptokinase-streptodornase" product, which commonly caused fever, allergic manifestations, and sometimes even recurrence of bleeding (one such patient allegedly developed atrophy of overlying chest wall muscles). The significant risk of renewed bleeding still makes me reluctant to instill clot lysis factors into the pleural space (a liquifying clot associated with active intra-pleural bleeding in an unstable patient is hardly an improvement over a stable clotted hemothorax).

Never Clamp a Chest Tube, Except After Pneumonectomy

A chest tube is usually kept clamped in a postpneumonectomy patient because (1) no air leak is expected, (2) the empty hemithorax will gradually fill with serosanguinous pleural fluid, and (3) an open chest tube submerged in pleural fluid could suddenly siphon a great deal of fluid out via the chest tubing: The resulting severe pressure drop in the emptying hemithorax would rapidly shift heart and mediastinum toward that side; such a sudden "siphon shift" is both exceedingly painful and likely to induce dysrhythmia or possibly even death (however, I have never ventured beyond the "painful" stage with my own patients).

A routine postpneumonectomy chest tube only remains unclamped immediately after chest closure, while the patient is slowly and gently turned to the supine position. At that time, air usually gushes from the submerged water seal straw as the mediastinum shifts back toward the empty pleural cavity, and visceral weight combines with increasing abdominal muscle tone to force the diaphragm upward.

If a patient just after pneumonectomy is turned from the lateral position at surgery without such an open chest tube, he can suddenly develop markedly above-atmospheric pressures in the empty hemithorax (a classical tension pneumothorax, with mediastinum and trachea pressed toward the remaining lung). Immediate decompression by thoracentesis (through the second or third anterior interspace) is then required, often to resuscitate the suddenly hypotensive patient.

Pressure within the empty hemithorax is occasionally adjusted thereafter by further air removal until the empty hemithorax remains slightly subatmospheric during both inspiration and quiet expiration. (The expected postoperative effusion, along with a gradual reduction in size of the empty pleural cavity, usually causes pressure within the progressively-more-apical air pocket to increase during the first postoperative day or so; an occasional, repeat pressure adjustment is desirable during this time.)

Air pressure within the empty pleural cavity is readily adjusted, without a water manometer or other special device, by using a normally primed water-seal chest drainage bottle and tubing (connected in some sterile, airtight fashion directly to a thoracentesis needle): Once again, the water seal serves as a very satisfactory manometer of inspiratory intrapleural pressures. Simpler yet, a large-bore, open needle can be inserted (as at thoracentesis for aspiration of air), covered during inspiration and then removed near the end of a quiet expiration (less accurate, apparently just as safe).

My preference, however, is to place a small chest tube at the time of pneumonectomy, and thereby avoid the occasional hypotensive episode requiring urgent chest wall puncture. This tube is usually removed the day after surgery, when pleural air pressure has remained subatmospheric for a number of hours without further adjustment.

Routine Chest Tube Placement After Adult Pneumonectomy

Using a long Kelly clamp inserted via a small, lateral, inframammary skin-fold incision, the beveled connector-end of a #20 French chest tube is grasped and pulled out of the chest cavity through its long extracostal tunnel from an upper interspace, leaving the tip in the usual anterior apical position. This tube is secured to the skin and then connected to a simple water seal chest bottle.

When the patient has been turned to the supine position and his intrapleural pressure initially adjusted, the tube is clamped with a large Kelly hemostat. It may be wise to encircle both Kelly clamp handles with tape, so that the clamp will not pop off accidentally. A "Do Not Remove" note on this tape

can also be helpful.

Technique of Postpneumonectomy Pleural Cavity Pressure Adjustment by Chest Tube and Simple Water Seal Bottle

Pressure adjustments are readily accomplished as follows: First, to avoid any siphon effect, empty the chest tubing distal to the chest tube clamp (by allowing room air into the upper tubing through a hollow needle, or by tilting the water seal bottle to allow air access to the straw tip). Then, with the chest tubing cleared of fluid, place the chest bottle on the bed or bedside stand for easy observation (and also to reduce the siphon gradient if the tubing suddenly refills with fluid).

Finally, when the patient has relaxed and is breathing easily, unclamp the chest tube. At this point, tidal or bubbling from the water seal straw gives an immediate, accurate indication of intrapleural pressures. Ideally, one tries to maintain an intrapleural pressure slightly below atmospheric: This is evinced by a low inspiratory tidal and no bubbling on normal (quiet) expiration.

During pressure regulation with the chest tube unclamped, a cough (or any other sudden effort that tightens abdominal muscles in Valsalva fashion) can easily "blow off" too much intrapleural air, or even initiate a fluid siphon. Such siphon should be stopped immediately by pinching and then reclamping the chest tube (and once again emptying the chest tubing by open needle or tilted bottle, as before).

Excessive (often painful) subatmospheric pressure is readily developed by such a cough or brief siphon; a high tidal is then seen, well above the water seal straw, as soon as the clamp is again removed. This accidentally created, excessive intrapleural suction is relieved by allowing momentary atmospheric access to the unclamped chest tube through a hollow needle into the tubing lumen while the patient again breathes as easily as possible.

Some bubbling is expected at the water seal straw when venting the empty hemithorax during the first twenty-four hours after surgery. However, if the chest tube tip droops down too far within the pleural cavity, or the pleural fluid level rises to an unexpected degree, the tube may drain fluid rather than venting the overlying air pocket.

There is no doubt that fluid removal can reduce the pressure within an empty pleural cavity, but one cannot regulate the intrapleural air pressure easily when the tubing is full of fluid, and siphon shift of the mediastinum always remains a risk. Thus, under these circumstances, one may as well remove the chest tube and perform any further intrapleural pressure adjustments by thoracentesis (unless some well-tolerated change in the patient's position brings the air pocket over to the chest tube holes, or minor withdrawal of the chest tube allows it to be resecured in a more advantageous position, or the surgeon elects to drain the accumulated fluid - see discussion below).

Check Tracheal Position Frequently

Frequent gentle palpation of the tracheal position at the sternal notch is helpful in evaluating the postpneumonectomy patient, for a normally mobile mediastinum always shifts noticeably toward the pleural cavity with lowest pressure. Satisfactory adjustment of intrapleural air pressure by thoracentesis or chest tube will keep the trachea displaced toward the "pneumonectomy" side of the midline. Maintaining such a subatmospheric intrapleural pressure provides space for additional fluid to accumulate

within the empty pleural cavity without unduly compressing the mediastinum (especially during cough).

Each consecutive intrapleural pressure adjustment following pneumonectomy usually releases a smaller quantity of air. If, after several adjustments, pressure within the empty pleural cavity still increases rapidly, that suggests significant intrapleural bleeding, excessive pleural fluid production, or possibly even air leaking through the bronchial closure. In association with other signs of ongoing blood loss (such as early postoperative hypovolemia, hypotension, falling hematocrit, and roentgenographic evidence of a large pleural effusion within the empty pleural cavity), this rapid buildup of intrapleural pressure suggests that reexploration for bleeding could soon become necessary.

When the rising intrapleural pressure is associated with an increase in size of the apical air pocket (and perhaps significant bloody expectoration), this suggests that the patient has a postpneumonectomy bronchial stump air leak: Such a leak places him at risk for drowning if more pleural fluid is suddenly "dumped" into the leaking bronchus; therefore he must be positioned upon his operated side until all pleural fluid has been drained by chest tube (see Chapter 4).

Excessive Fluid Production After Pneumonectomy

Extrapleural pneumonectomy (for bulky carcinoma or lung destroyed by infection) may occasionally result in "excessive" post-operative losses of serosanguinous fluid that will require continuous drainage by chest tube, as well as close attention to the patient's fluid and electrolyte requirements.

To avoid the pain and risk of repeated siphon shift of the mediastinum during such tube drainage (any pressure change within an empty pleural cavity is exerted directly upon the mediastinum, without attenuation by outside air entering or leaving an "elastic" lung), a hollow No. 19 needle can be inserted into the chest tube connector lumen: Continued air access through this needle prevents a hanging fluid column within the chest tubing that could exert undesirable suction or start a siphon).

Once this arrangement is functioning, and the initial, large, post-operative collection of pleural fluid carefully drained, the chest tube Kelly clamp is no longer required. An empty, glass, intravenous-solution bottle can be hung nearby, with its tubing inserted into the open No. 19 needle, to permit continued air access into the chest tubing connector.

The prolonged "air path" through the empty I.V. bottle may reduce the likelihood of bacterial contamination at the needle site (or of pleural fluid leaking from the open needle hub onto the bed). Repeated bacterial cultures of pleural drainage, and vigorous antibiotic prophylaxis or treatment, are also indicated. When using this "apparatus," pleural cavity pressures will remain nearly atmospheric (above during expiration and below during inspiration) and thus need not be "adjusted."

Subatmospheric Intrapleural Pressure is Especially Helpful if the Phrenic Nerve Was Divided During Pneumonectomy

When a phrenic nerve is sacrificed during removal of adherent tumor, intentional accentuation of subatmospheric pressure within the empty hemithorax can minimize paradoxical diaphragm movements and improve (the often marginal) respiratory function until peripleural fibrosis stiffens the postpneumonectomy pleural cavity (see Chapter 4).

One method for maintaining such a persistently subatmospheric pressure is to leave the small postpneumonectomy chest tube open to straight water seal drainage (without use of the above-described indwelling needle air vent) after drainage of the initial postoperative fluid build-up (that fluid removal is achieved by combining fluid removal with air replacement, to avoid excessive intrapleural suction). Once such a patient (with empty pleural cavity and minor pleural fluid production) has been placed on simple water seal, the hanging fluid column in the chest tubing can be adjusted to the appropriate height: That column height is then sustained by any fluid drainage, as long as there is no bronchial air leak (which would require tube drainage anyhow) or sufficient fluid production to restart a siphon.

Antibiotics

Any patient undergoing pneumonectomy should probably remain on prophylactic antibiotics until "all tubes are out": The empty pleural cavity also makes sterile technique especially important. If prolonged drainage of the pneumonectomy space should become necessary, prophylactic antibiotic irrigations might well be appropriate (see Clagett Technique - Chapter 4).

Role of Prophylactic Digitalization

As previously mentioned, older patients commonly experience atrial tachydysrhythmias after pneumonectomy, possibly due to wide pressure oscillations in the airtight hemithorax caused by the normal volume changes of respiration and cough. Dysrhythmia also seems more common with major atelectasis, or after pulmonary lobectomy in older, more emphysematous patients: Here again, an inelastic lung and large pleural space combine to allow significant intrapleural pressure changes (as demonstrated by the associated wide tidal).

Major air leaks, and the large chest tubes that they demand, tend to reduce such pressure oscillations. In any case, the wide tidal will gradually decrease as remaining lung expands to obliterate the pleural space; the improved air exchange of the gradually stretching lung also helps the patient to breathe with less effort. Prophylactic digitalization can limit the ventricular rate in response to atrial tachydysrhythmia (and perhaps even reduce the likelihood of such rhythm disturbance, especially if the prophylactic dose of digoxin is kept "on the low side" and serum potassium levels are maintained above 4.5 mg/L).

Treating Low Serum Potassium or Magnesium Can Reduce Dysrhythmias

American diets often lead to suboptimal serum magnesium levels so a magnesium sulphate solution given intravenously (in lower doses than used for eclampsia) or one or two magnesium chloride tablets by mouth (such as Slow-Mag or Mag-64) daily often reduces muscle cramps as well as tachydysrhythmias. Although a significant excess of serum potassium is dangerous and may cause cardiac arrest, a bit too much magnesium merely makes the patient persistently sleepy (potassium and magnesium are the dominant intracellular cations).

Restrict Fluids After Pneumonectomy

Following pneumonectomy, frequent auscultation of the lung is essential. Any basal rales suggest fluid overload, with a need for prompt diuresis plus fluid restriction (but keep in mind that some patients always have "dry" rales, even when dehydrated or hypovolemic). With or without rales, the

early postpneumonectomy patient must be kept "on the dry side" of complete fluid replacement: Intra-operative and postoperative fluid orders should ignore routine pleural cavity fluid accumulations and under-replace any measured blood loss, for the limited gas exchange capacity of a single lung deteriorates readily with the slightest congestion.

Moderate reduction of urine output is expected and desired during the first twelve to twenty-four hours after pneumonectomy, to avoid any possibility of early postoperative fluid overload (when the risk of decompensating the remaining "good" lung seems especially high). Why pulmonary congestion later becomes less of a problem is unclear, although the answer probably includes short-term pulmonary-irritant and vasoreactive changes due to anesthesia and other medications, as well as ongoing pulmonary vascular and lymphatic adjustments.

In any event, it is wise to oversee and limit any preoperative, intraoperative and post-pneumonectomy fluid therapy, and not to leave this critically-important matter to a new house officer, unfamiliar anesthesiologist, concerned internist or even chest medicine specialist, as these folk are often less attuned to this critical problem, and more worried about maintaining a normal urine output and rosy cheeks.

Pulmonary artery pressures should never guide postpneumonectomy fluid replacement, as fatal pulmonary edema (due to excessive hydration) has been increasingly encountered in the presence of normal or even low wedge pressure: Wedge pressure measurement has little purpose here anyhow, for the left heart is protected from fluid overload through the increased resistance offered to right heart output by the single remaining lung. It is easy to see that fluid accumulation within that remaining lung - due to overhydration - could require its lymphatics to cope with far more fluid than usual.

Summary: Dangerously wet lungs are all too common after pneumonectomy: However, I cannot recall a single patient who was pushed into renal failure by fluid restriction at the time of this proce-dure. Operative blood replacement should only suffice to maintain adequate blood pressure during normal cardiac rhythm, and a hematocrit of less than 40; in particular, blood replacement should never exceed measured losses. Fluids for hydration of a normal postpneumonectomy adult male in the first 24 hours after surgery are usually started at less than 75 cc of dilute electrolyte solution per hour (and decreased or increased according to clinical judgement thereafter).

Postpneumonectomy Air Leak from the Bronchial Stump

Early intrapleural fluid build-up following pneumonectomy often exceeds the volume of chest tube drainage commonly encountered after other lung surgery: Perhaps this is due to loss of lung lymphatics as well as lack of postoperative interpleural approximation.

Some surgeons insist that postpneumonectomy patients lie only upon their operated side (to prevent drowning in case of catastrophic bronchial stump disruption). I have not heard of acute bronchial disruption during hospitalization, however (except consequent to vigorous endotracheal suction. One resident proudly reported over a liter of sputum removed by suction catheter. His patient did well after reexploration and suture). It seems preferable, therefore, to encourage early mobilization of the postpneumonectomy patient.

As soon as a pneumonectomy patient has regained his positive attitude toward recovery, I advise him (usually just prior to discharge) that in the remote possibility of the onset of copious expectoration,

he should immediately lie upon the operated side and have himself thus transported to the hospital emergency room. Incidentally, comparable sequential chest roentgenograms after pneumonectomy never show significant enlargement of the residual pleural cavity air pocket unless there is air leak. (I have seen a minor apparent increase in the apical air collection within an empty hemithorax without subsequent development of bronchial disruption or empyema, but this is always cause for worry, and usually chest drainage as well; see Chapter 4.)

Eventual rib resection with permanent open drainage was the outcome in my three patients who developed postpneumonectomy bronchial stump air leak. Early closed drainage of pleural cavity fluid (when the leak was first suspected, before it enlarged to a major disruption) successfully converted each of these potentially fatal catastrophies into a prolonged aggravation for the patient.

These bronchial disruptions were attributed to; (1) tumor at the tracheal margin of a post-irradiation palliative extrapleural resection of a chronically obstructed, infected lung; and (2) chronic cachexia with postoperative "strep throat" (a 75 pound, elderly female in the days before hyperalimentation, with a beta Hemolytic streptococcus infection of her throat and bronchial silk closure; first hint of leak was two weeks postoperatively); and (3) presumably excessive bronchial artery mobilization that devascularized the cut bronchial edge.

In recent years, bronchial stump closure with 4-0 cardiovascular (monofilament) polypropylene sutures (either simple or figure-of-eight) has proven very satisfactory, and I try much harder to obtain a live pleura tissue flap for coverage of the post-pneumonectomy bronchial stump (none was available or used in the above three cases): Perhaps some bronchial air leaks are inevitable (regardless of surgical technique or prophylactic antibiotics), but competent surgery should make bronchial disruption a very uncommon complication.

After Pneumonectomy: Things for the Nurse to Consider

1) Listen for rales at the posterior lung base. Minor fluid overload is very dangerous after pneumonectomy.

2) Palpate the trachea at the sternal notch to be sure it remains midline or toward the pneumonectomy side.

3) Avoid poking at (or through) the bronchial suture line with an endotracheal suction catheter.

4) In the event of copious expectoration, have the patient turn immediately onto the operated side; bronchial suture line disruption could otherwise allow massive spill into the remaining lung, with likelihood of drowning.

5) Report any arrhythmia to allow early therapy, meanwhile provide oxygen (there have been allegations that supplemental oxygen given after myocardial infarction does not improve oxygenation, but this "evidence" may not be relevant to other, dissimilar clinical situations).

6) Leave the chest tube clamped (but only after pneumonectomy) unless otherwise directed.

Clean and Dirty Thoracotomy Wounds

When a patient with a chest tube comes to thoracotomy, one must decide whether the tube should be taken out on admission to the operating room, cut off or removed before skin preparation, or left in place until the chest is open. Esthetically, it is preferable to remove all contaminated bottles and tubes from the surgical area when there is no air leak (or active blood loss being collected for possible autotransfusion). Usually, however, there is pulmonary air leak, which will probably become more significant when positive-pressure ventilation is instituted.

Only after the patient with pulmonary air leak has been positioned for skin preparation and thoracotomy is the skin suture cut and the chest tube removed to allow optimum skin cleansing about the tube site. If a chest tube has been present for several days, the tube tract probably has become so stiffened by inflammation that it will remain patent without internal support for a short while: However, if concerned about continuously venting a possible tension pneumothorax until the chest can be entered, it is easy enough to insert a new sterile tube, or even slip a Kelly clamp temporarily into the povidone-iodine soaked tube tract in order to maintain its patency.

Especially in a septic case, where additional minor contamination is less significant, the tube can also be cut off close to skin level and skin prep done directly around this. This discussion may be unnecessary, as wound infection is extremely rare following thoracotomy: Perhaps wound splinting by ribs and the copious intercostal circulation are important "protective" factors.

Poor-risk Cases Sometimes Cannot be Turned for Posterolateral Thoracotomy

I have not used the prone thoracotomy position for many years (and no longer have access to an Overholt prone operating table anyhow). Intensive medical therapy, and preliminary tube drainage of purulent collections in or about the lungs, can markedly decrease the risk of spill into the opposite (down) lung during posterolateral thoracotomy.

In experienced hands, double-lumen endotracheal tubes, inflatable balloon catheters to block the bronchus, special length or diameter items such as main bronchus tubes for selective one-lung ventilation, and other applicable anesthesia monitoring and supportive techniques for the severely infected or compromised lung can make the familiar posterolateral incision relatively safe for essentially all pulmonary work.

Even the occasional patient who is "too sick to turn" can still undergo limited exploration and excellent drainage via a lateral or even slightly posterior incision, despite remaining in the supine position (especially when secured with his chest protruding slightly off the edge of the operating table, and carefully draped).

For a truly borderline patient, the sitting position also is practical. Such a patient is secured to the table at the hips with his "away" arm fastened to an armboard as an extra precaution against falling. While anterior submammary thoracotomy is always an option for exploration, it is far less helpful when rib resection for dependent drainage of empyema is required (and that is the usual problem being dealt with when a patient is this marginal, yet still considered an acceptable candidate for thoracotomy).

Postoperative Dressing for Posterolateral Thoracotomy

The skin is cleansed with a 3% (or less concentrated) hydrogen peroxide moistened sponge (which readily removes blood) and dried carefully. A minimum dressing (4 x 4 inch gauze pads opened

lengthwise) is applied to the posterolateral chest incision, using momentary pressure if oozing persists. Postoperatively, a minor but persistent skin bleeder may increase blood loss, and it is far less obvious under a bulky dressing.

One should avoid placing a bulky posterolateral dressing over a thoracotomy incision anyhow, for this is very uncomfortable with the patient leaning back, since it puts weight directly on the incision. (It might be more helpful to place such a bulky dressing on the healthy side as a back support, but I have not tried this as I doubt the patient or his relatives or attorney would appreciate my great idea.)

The bandage is secured by wide plastic or paper or "unstretched" elastic tape (also in minimum fashion). A split-sponge dressing is placed around the chest tube or tubes, which are then taped securely to benzoin-prepared skin with cloth adhesive tape (see Figure 5) unless the patient has "tape allergy."

Tape Allergy

Tape allergy is uncommon and should not be confused with blistering from "tape shear." The latter can usually be avoided by loosening the anterior end of a posterolateral thoracotomy dressing in the recovery room, for this part of the dressing regularly comes under increasing tension after the patient is turned to supine and his shoulder falls back. Several hypoallergenic tapes now available have further diminished "allergy" problems. Keep the possibility in mind, however, to avoid an occasional nasty "burn," with possibility of permanent scarring and pigmentation changes.

Tape allergy may, in part, be mechanical hypersensitivity (such as leads to dermatographia, for example). Generally, the patient with early blistering will complain that the dressing feels " all bunched up" or that it is "burning." Irritated or blistered skin is best treated "open and dry" or with antibiotic or antiseptic ointment if slightly purulent.

Postoperative Roentgenogram of Chest

I usually order and view an initial portable chest roentgenogram within the first half hour or so after any thoracotomy, to confirm satisfactory location of chest tubes, endotracheal tube and venous catheters, as well as to identify unexpected intrapleural accumulations of air or fluid, or detect evidence of fluid overload or atelectasis. Thereafter, the stable patient has repeat films on a daily or less frequent basis, de-pending upon the clinical situation.

While an endotracheal tube or pulmonary artery catheter remain in place, regular roentgenographic confirmation of their position can be reassuring: But except for the most critically ill patients, I hardly ever use pulmonary artery catheters: perhaps they are more valuable than they seem, but often they "restate the obvious" or leave the matter in doubt (and like many other technical advances, they tend to increase both cost and risk).

It can be false economy to require permission from the physician-in-charge prior to ordering postoperative chest roentgenograms, especially in the ICU, for skilled and interested ICU nurses and respiratory therapists reliably discover and correct endotracheal tube displacement, and also detect other problems (pneumothorax, hemothorax, atelectasis, effusion) that could become disastrous if allowed to persist.

When more people feel directly responsible for the patient's welfare, his passage through the

hospital is likely to be "increasingly safe and sure". Shared responsibility need not mean "No one is in charge here"; rather it implies delegated and overlapping concerns - a true "team" approach.

CHAPTER 10: THE MEDIASTINUM

Introduction

This chapter will initially focus on that retrosternal inter-pleural zone located anterior to heart and pericardium, superior to diaphragm and inferior to anterior neck tissues. To expedite our discussion, let us agree here (and confirm elsewhere) that blood accumulating postoperatively within this potential space usually escapes from damaged arteries; that such retrosternal collections need only accumulate until they rise above venous pressure levels to stop venous return and cause fatal cardiac tamponade; that severe postoperative bleeding can overwhelm and block any size or number of drainage tubes; that remedy of cardiac tamponade within twelve hours after sternotomy closure requires a fully equipped and staffed operating room (often in a hurry); that the unstable bleeding patient is best kept completely paralysed on ventilatory support (to prevent a cough or minor exertion from suddenly increasing mediastinal pressures to catastrophic levels); and that the occasional, truly marginal patient with severe postoperative bleeding will more likely survive a rushed return to surgery if his sternotomy is first reopened in the ICU bed, as additional bleeding or unanticipated delays might otherwise prove fatal. (One surgeon relates how his postoperative patient was "turning sour" en route to the operating room when a "sweet little old lady" at an intermediate floor became so fascinated by this "true to life" hospital drama that she kept pushing the elevator button every time the door closed. The patient finally died there: I forgot to ask what happened to the little old lady.)

More commonly (I am glad to report), troublesome postoperative mediastinal bleeding subsides "spontaneously" or in response to the correction of clotting defects, institution of mild "controlled hypotension" or application of vigorous PEEP (positive end expiratory pressure). Many possible sequellae of minor to moderate poststernotomy mediastinal hemorrhage are considered in the first part of this chapter.

Substernal Drainage: Methods and Problems

Although well placed, properly tended drainage tubes of an appropriate size are supposed to prevent mediastinal clot accumulation, some intrathoracic clot almost always builds up while bleeding persists. Subsequent clot lysis and blood protein fragmentation then draws additional fluid into the closed anterior mediastinal space by osmosis. This process may lead to increasingly severe cardiac tamponade.

Despite the fact that continued active bleeding is hardly ever a problem more than twelve hours after sternotomy closure, a formal reexploration can become necessary to relieve persistent severe tamponade by mediastinal clot in the first day or two poststernotomy: Thereafter, progressive clot lysis usually allows satisfactory cardiac decompression by simple "finger mediastinotomy." Obviously, the timing of such mediastinal drainage will depend upon physician awareness and the patient's condition; an initially retarded convalescence may only turn "critical" 5-7 days after sternotomy.

"Finger mediastinotomy" refers to a finger-sized midline infra-sternal Betadine-soaked reopening through a very recent upper abdominal fascial closure (usually without anesthetic), in order to permit Retrosternal! passage of a sterile gloved finger. Prompt decompression of a substernal fluid pocket is thereby achieved with little likelihood of causing infection or developing a hernia at the fascial opening. There is minimal space for mediastinal air accumulation under these circumstances so a temporary Penrose drain may even be laid into this finger tract and covered by a frequently changed Betadine-soaked occlusive dressing, pending completion of clot lysis (see Chapter 15).

While competent tube management cannot eliminate such delayed retrosternal fluid accumulations, one can minimize their likelihood and volume by early application of suction to the mediastinal tube, as soon as the sternal approximation seems airtight, in order to lift fluid drainage out over the anterior chest wall of the supine poststernotomy patient: Fresh blood pools and clots readily under a substernal air pocket in a supine patient, so any delay in initiation of mediastinal drainage may allow significant build-up of intrathoracic clot.

Suction is unnecessary when a pulmonary air leak drains through the substernal tube, however, for positive-pressure ventilation then provides a steady outward flow of air to carry fresh blood along as froth. In other words, moving air assists fluid drainage (which is why "sump suction" can be so useful), while blood tends to pool and clot under a stagnant air pocket. (One could argue that a "stagnant" air pocket merely identifies inadequate substernal drainage, rather than causing it, but see discussion later this chapter.)

When there is no air leak, early substernal-tube suction is applied most readily by aspirating about 100 cc of air from the chest tube connector, in order to lift a hanging fluid column above the simple water seal chest drainage bottle: This column removes retrosternal air and usually initiates mediastinal tube drainage. Barring later air entry, that hanging fluid column is sustained or reconstituted by any blood lifted out over the sternum.

Any time that a proximal segment of tubing fills with fluid, this allows an equal volume of air to move distally and thereby decrease the height of the hanging fluid column. Encouraging prompt passage of proximally located fluid and clot into the distal tubing (by appropriate tilting and squeezing) will not only help keep the proximal chest tubing functional and free of obstructing clot, but such prompt fluid passage will also sustain the distal hanging fluid column that is providing suction.

With retrosternal air evacuated, fresh blood spreads widely between contiguous tissues, often becoming partially defibrinated during its passage over moving heart and lung surfaces toward the drainage tube. Persistent chest tube care remains essential while fresh blood loss continues: One need not worry about creating new retrosternal air pockets while "milking" the chest tube, however, for such air is rapidly retrieved by the hanging fluid column, as long as the tube continues to function. In fact, increasingly edematous and cohesive mediastinal tissue surfaces soon resist air return anyhow (see "finger mediastinotomy" below).

Hanging fluid columns and slight drainage delays become unimportant when active blood loss ends. While the tube tip within an increasingly mobile patient has often clotted by this time, effective tube drainage continues as long as the most distal side-hole remains open and within the pocket to be drained. Functional substernal drainage tubes are best left in place until fluid production becomes minimal in volume and barely bloody in color, to avoid further osmotic fluid ingress.

A Model of Mediastinal Drainage

Aspiration of air from an inverted "U" of tubing (with each end of the "U" being submerged in its own pool of fluid) will lift fluid an equal distance above the surface of origin within each leg of the tubing, regardless of the relative levels of the two pools. If the height to which fluid is thus lifted exceeds the length of the shorter arm of the inverted "U" (so that a fluid column reaches from one of the pools to the top of the "U"), drainage of the upper pool will commence. Flow then proceeds until the upper pool has emptied, or both pool surfaces reach equal level (equilibrium), or air enters the tubing and breaks the siphon (partially or totally releasing the hanging fluid column), or the open tube tip within the upper pool becomes blocked by debris or the container wall.

Tube Placement at Sternotomy Closure in the Adult

A single No. 32 French silastic chest tube should reliably drain any substernal fluid accumulation except continued active bleeding (for which reoperation may soon be indicated anyhow), provided the tube is properly located and carefully tended. Tube placement is simplified if the sternal retractor remains open while the retrosternal tube is positioned from a short, transverse, upper abdominal skin incision.

The beveled "connector end" of the chest tube is grasped within the chest and pulled out through an appropriately directed subfascial tunnel (created by the Kelly hemostat from outside-in). This tunnel should support the tube in an appropriate lie. The most distal side-hole must remain above diaphragm level, as this may be the only hole cleared of clot by vigorous milking. Tough fascia in young adults is more readily penetrated if a smaller hemostat is pushed through first, before inserting the larger Kelly clamp to grasp the tube.

One should secure the chest tube to the skin entry site at this time, using a 2-0 or 3-0 monofilament skin suture (tied first in a loose loop above the skin, then tightly "back and forth" about the tube); for it can be most irritating if a tube slips out during sternotomy closure. Unlike the usual thoracotomy, where closed thoracostomy can generally be performed postoperatively with less "hassle" than reopening the chest, it is best to reopen any sternotomy for proper tube replacement if the only drainage tube is inadvertently yanked out while the patient is still in the operating room.

Such reopening is not necessary, however, if a wide pleural entry has been created that assures easy mediastinal drainage into either pleural space; in that case, a closed thoracostomy can generally substitute for the inadvertently removed substernal tube. Accidental tube removal after the patient has left the operating room is evaluated and treated according to "drainage past" and "drainage anticipated" (see later this chapter and Chapter 15).

Air Leak Through a Small Defect in Mediastinal Pleura May Bubble Intermittently

Sharp dissection, electrocoagulation of "bleeders," or the wire sternotomy closure can easily injure

mediastinal pleura and its underlying lung. A pulmonary air leak sometimes results that may be delayed in onset, with air either passing directly into the mediastinum or first causing pneumothorax. In such a case, air entering the mediastinum through a small pleural defect usually forms a gradually enlarging mediastinal air pocket: This "balloon" finally deflates (in a brief flurry of bubbles) when it extends onto an open chest tube hole.

The rate of air accumulation, distance between pleural defect and substernal tube, and patient position will all affect the frequency and "vigor" of such intermittent bubbling (which is often relatively regular but not synchronous with respirations). Infrequent bursts of bubbles may even slip out unobserved; but on simple water seal, such air leak is always betrayed by loss of any hanging fluid column.

In contrast, when using mechanical chest bottle suction, infrequent or intermittent bubbling from the water seal straw becomes difficult to detect, especially given the constant mechanical vibration of a flimsy plastic 3-bottle unit by its bubbling vacuum breaker, and the usual low fluid level in its water seal straw during suction). This can lead to premature chest tube removal in the presence of ongoing but intermittent air leak.

The embarrassed surgeon must then perform an urgent closed thoracostomy to correct the "delayed-onset postoperative pneumothorax". Presumably, an occasional experience of this sort is what has caused many otherwise reasonable chest surgeons who "always use low suction" to "always clamp the chest tube for 24 hours prior to its removal": Fortunately, this expensive and unjustified "suction and clamp routine" is easily avoided by utilization of a simple water seal chest drainage bottle within which a hanging fluid column is easily created to detect air leak or to apply suction when there is no air leak and suction might be helpful. Also "high" chest bottle suction can be applied (without collapsing sturdy water seal bottles) if that might help to close a persistent pleural air pocket associated with an ongoing air leak.

When a minor air leak through the mediastinal tube is not associated with pneumothorax, such air leak will usually seal within one to three days, delaying substernal tube removal only slightly. I do not use right-angle or other intrapericardial tubes following cardiac repairs, but it is worth mentioning that such mediastinal tubes are less useful for air drainage because their tip is positioned more posteriorly: In fact, any deeply intrapericardial drain will not evacuate mediastinal air under "normal" postoperative circumstances (a supine patient and some mediastinal tube fluid drainage) unless it is a sump tube on suction.

If a mediastinal air leak is associated with moderate to major pneumothorax, that pneumothorax is usually best treated by an additional closed thoracostomy. Once appropriate pleural space drainage has been achieved by chest tube, the substernal tube is removed by the usual criteria (based upon past, present and anticipated mediastinal drainage). In general, less significant intrapleural or mediastinal air collections and air leaks can be treated by either the existing substernal tube or a new closed thoracostomy.

Good judgment may suggest that both a substernal and an intrapleural tube be positioned prior to sternotomy closure when there has been inadvertent pleural entry. As mentioned, wide communication between the anterior mediastinum and one or both well-drained pleural cavities may make the substernal tube unnecessary (e.g. following subtotal pericardiectomy).

Cardiac Pulsation May be the Only Tidal Seen in a Mediastinal Tube

Admittedly this is not a very important observation, but why does the only visible tidal in a substernal tube sometimes coincide with the heartbeat and not respiration?

Well, we would expect respiratory tidal to be absent during positive-pressure controlled ventilation, which never allows the pressure within the chest to fall below atmospheric. Furthermore, emphysematous lungs containing trapped air might continue to "crowd" the mediastinal tube during spontaneous respirations, and therefore transmit inspiratory pressures poorly, if at all.

As for prominent cardiac pulsations at the water seal, these simply signal that a mediastinal air space is still in contact with the mediastinal tube holes. This mediastinal air space undergoes a fall in pressure during systole (as the heart retracts and its size decreases), causing upward tidal at the mediastinal straw: The heart need not be in direct contact with the air space as such pressure changes are readily transmitted through intervening incompressible emphysematous pulmonary blebs.

In any case, suction on the chest tube by a hanging water column or mechanical device, readily draws nearby air through the substernal tube, thereby eliminating that "cardiac tidal."

Case Report: Unsatisfactory Substernal Tube Drainage

A plastic, No. 28 chest tube was placed in retrosternal position following an aorta-to-right-coronary-artery (saphenous vein) bypass. Control of chest tube position was attempted by inclination of the subcutaneous and subfascial tunnel, but it remained dangerously far to the right, near the vein graft: Therefore it was pulled back and also cut shorter. The tube drained only for a moment and never thereafter, despite obvious postoperative mediastinal widening and my frequent worried milking of the tube.

Tube removal at thirty-six hours confirmed a firmly clotted tube tip, with only the most distal side-hole (the one farthest from the tube tip) still patent. Although air forced out of this side-hole while milking had easily lifted adjacent soft tissues away, only the firmly clotted tube tip was properly located within the anterior mediastinal space needing drainage – namely, above the diaphragm level.

Approximately 100cc of "old" (dark red, non-clotting) blood drained via the tube tract after tube removal, during a couple of mild Valsalva maneuvers. The patient stated that this drainage somewhat relieved his substernal oppression.

Nonetheless, "finger mediastinotomy" was required on the fifth postoperative day, to release an additional 600cc of old blood. On the ninth postoperative day he was discharged, still complaining of back and neck distress, with persistent mediastinal widening and minimal paradoxical pulse.

He returned a week later with recurrent tamponade and 25 to 30 mm paradoxical pulse. This time he was taken directly to the operating room and his sternotomy completely reopened: at reexploration, another liter of old dilute defibrinated blood gushed from a large stiff-walled pericardial pocket (located next to his right atrium and vein graft).

Drainage Options to Consider after Reopening the Sternotomy

A chest tube on "high" suction would soon obliterate this pericardial pocket: Opening the pocket widely into an appropriately drained right pleural space should also succeed. However, the phrenic nerve and vein graft were both invisible, embedded somewhere within the greyish-yellow edematous distorted epicardial and pericardial tissues, deep to adherent greyish-yellow fibrin deposits.

As a "chicken" compromise, therefore, the thick walled pericardial space was carefully irrigated out and left full of warm, physiologic salt solution. A single, carefully tunneled tube (with all its side holes carefully located within the substernal space) was directed leftward over the heart - away from the invisible vein graft and its "undrainable" pocket. The hope was that this salt solution would soon drain out or be absorbed, and that in the meanwhile it would decrease the reaccumulation of (and also dilute) any osmotically active, protein rich inflammatory exudate which might otherwise promote persistent atrial tamponade.

This new, "perfectly" located (but once again non-functioning) substernal tube was removed after forty-eight hours, as the intrapericardial pocket had again sealed off from the anteriorly located tube. Fortunately, his asymptomatic mediastinal widening gradually resolved over several weeks of home convalescence as the saline absorbed, and he finally "did well."

Discussion: Clot within a chest tube lumen can often be blown back into the chest by "milking" the tubing, or by disconnecting the chest tube to irrigate it directly: I avoid the latter for fear of delivering bacteria to a fluid pocket that already is resisting drainage and may continue to drain poorly after such irrigation.

It seems clear that any pressure surge sent up through the chest tube by milking must primarily affect the distal side-holes. In fact, once these holes are cleared, further milking or low volume irrigation can have little effect on clot within the tube tip (due to rapid escape of the pressure-pulse from milking at the first open side-hole).

A single well-placed chest tube side-hole can certainly drain any fluid accumulation (after all, each side-hole opening is as large as the chest tube lumen). When that open side-hole is buried in clot or located outside of the cavity to be drained, however, milking can still send a pressure wave through air or fluid up the tube and out of the open side-hole, but return flow will then be obstructed as clot or tissue fall back onto the "open" side hole (as soon as pressure around the tube exceeds that within it). Under these circumstances the chest tube itself behaves as a "wrong-way" one-way valve.

When retained blood proteins degrade into smaller molecules, the increase in osmotic activity extracts additional water from nearby body fluids. In contrast, a pocket of isotonic physiologic salt solution is simply absorbed: During this process, any but the most rigid walled space will shrink down and become obliterated. Perhaps the large majority of uninfected intrapleural or intrapericardial blood collections would eventually absorb in similar fashion, but confined spaces (such as skull, pericardium and even pleural cavity) may tolerate the intervening osmotic expansion of a blood-filled pocket very poorly.

In addition, a significant fibrous reaction by surrounding tissues may encapsulate and indefinitely delay such fluid removal - which proceeds most efficiently through normal pericardium or pleura (rather than via chronically inflamed, scarred and thickened tissue surfaces). Fortunately, bilateral wide pleural entry and loose pericardial closure have eliminated delayed postoperative cardiac tamponade from my practice.

Pericardial Closure

As a practical matter, a volume of intrapericardial fluid sufficient to cause acute cardiac tamponade will have little adverse effect if released into the less constricted anterior mediastinum. Similarly, a liquid blood and clot accumulation that causes cardiac tamponade while trapped within the anterior mediastinum can readily be tolerated after drainage into an opened pleural space.

To reduce the likelihood of postoperative tamponade, many surgeons leave the pericardium entirely open after cardiac repairs. In addition, surgeons who routinely open the pleura to mobilize an internal mammary artery for coronary artery bypass will automatically benefit from anterior mediastinal decompression into the pleural space.

It seems, then, that delayed cardiac tamponade (by trapped fluid at persistently above-atmospheric pressures within the pericardium or anterior mediastinum) could be avoided entirely if we simply open both pleural cavities widely after cardiac surgery, and leave the pericardium open as well.

Yet I still prefer to close pericardium when this can safely and easily be achieved in order to (1) separate the sternotomy closure from the heart, pacer wires and vein grafts; (2) stabilize vein grafts in good position; and (3) detect intrapericardial bleeding prior to closing a still-oozing sternum: A loosely closed pericardium may also aid intrapericardial hemostasis. Snug or tight pericardial closure, however, can impair cardiac filling, trap bloody fluid and compress vein grafts (making them more likely to kink where they bend).

So an ideal pericardial closure should (1) envelop but not constrict the heart within healthy, vascularized tissues; and (2) permit easy fluid egress, even after removal of mediastinal tubes. Such bloody drainage should then (3) move "away" spontaneously so that normal osmotic increases in its volume cannot compress the heart. This implies (4) that an ideal pericardial closure must also eliminate any potential retrosternal space.

Even when not compressing the heart, retrosternal fluid provides a liquid culture medium and thus potential bacterial pathway or "bug ladder" between subcutaneous tissues and pericardium via post-sternotomy bone gaps. A seemingly minor retrosternal fluid pocket may thus help initiate and then enable a superficial poststernotomy wound infection to persist, when it might otherwise have responded to subcutaneous drainage and antibiotics.

And that retrosternal fluid pocket may even encourage that local wound infection to expand into a messy life-threatening mediastinal or pericardial abscess that will require major surgical intervention. The fact that anterior mediastinal abscess becomes a major worry whenever sternotomy wound infection is diagnosed may be just another indication of the general inadequacy of our anterior mediastinal drainage techniques.

Effective pleural drainage clearly expedites recovery from thoracotomy. Experience has also shown that a sternotomy infection is likely to resolve only when it has good subcutaneous drainage above and healthy tissues below. It is time that we devote more attention to elimination of stagnant fluid collections from the retrosternal space.

Fortunately, a readily drained, loose pericardial closure that also eliminates the potential retrosternal space is easily constructed through lengthening of the divided pericardial edges with

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pleura. Since 1980, approximately 300 consecutive patients undergoing a primary cardiac procedure have had such an "ideal" closure. I am intimately aware of a much larger series of similar pericardial closures by Tector, who routinely utilizes the internal mammary artery for coronary artery bypasses.

Procedure For Extending the Pericardium With Pleural Flaps

A routine vertical midline pericardial entry is completed with the usual horizontal anterior pericardial incisions at the diaphragm level (forming an inverted "T"). Following the cardiac surgical procedure, pleural extensions of those vertical pericardial edges are created by making an electrocoagulation incision into both pleural envelopes right beneath and parallel to the sternum (with fingers protecting lungs and heart). This bilateral retrosternal pleural detachment is safely and easily achieved: The resulting flimsy 2 cm long pleural extensions of pericardium are then approximated loosely over the heart by fine absorbable sutures. In comparison, similarly wide pleural entries made near the pericardium (rather than just under the sternum) leaves floppy useless curtains of pleura hanging from the sternum: If not excised, such loose tissue can occlude all nearby drainage tube holes.

Free escape of fluid from the newly closed pericardium into either pleural space is ensured by not reattaching the horizontally incised lower pericardial edge back down to the diaphragm: Pericardial openings are left over aorta and elsewhere for fluid drainage as well. If pleural cavities are opened earlier in the cardiac operation, considerable amounts of blood worth salvaging (in sterile cases) can collect unnoticed in deep pleural recesses. And always think again about sucking fluids out of both pleural cavities before final sternal closure.

Drainage tubes: With bilateral wide pleural entry and loose pericardial closure, two anteriorly placed No. 32 silastic chest tubes have routinely provided satisfactory drainage of the entire thorax. I usually bring an intercostal tube in from the right side, and incline the midline substernal tube toward the opened left pleura. The substernal tube initially serves as the "main drain," then often clots as fresh blood loss slows and stops (a good time to remove this tube). The "more distant" right chest tube receives partially defibrinated blood from the start, so it usually remains open to vent serosanguinous fluid as any residual intrathoracic clot liquifies.

Complications

During electrocoagulation at the upper end of the right pleural entry, I "sparked" the right phrenic nerve on two occasions, causing diaphragm to "jump" (fortunately without postoperative evidence of phrenic nerve injury). I have also "sparked" lung sufficiently to cause insignificant short-term air leak. One postoperative reexploration was required to control a bleeding retrosternal vessel in the divided pleural reflection.

Other Observations

These minor complications were outweighed by the safety of sternotomy closure with vein grafts protected, by the minimized risk of cardiac injury during any repeat sternotomy (the heart was not adherent to sternum following such pericardial closure, although lung has been injured superficially by the sternal saw), and by elimination of the potential retrosternal space between intact pleural envelopes (thereby eliminating the occasional "finger mediastinotomy" to relieve subacute fluid tamponade, as well as any stagnant substernal culture medium). There has been no apparent change in pulmonary function or other delay in recovery. Combined operative and hospital mortality has remained about 1%

for elective cardiac procedures and 2% overall.

Following such wide pleural entry, it is not uncommon for a roentgenographically obvious pleural effusion to suddenly develop at the same time as the postoperatively enlarged pericardial shadow becomes smaller. This new pleural fluid, which represents liquified clot moving out of the pericardium, has routinely disappeared with the help of deep breathing exercises (one extra-deep breath on each side every five minutes while awake), thereby also eliminating the need for postoperative thoracentesis. However, depending upon patient position, the effusion may alternate sides between studies and considerably perturb the radiologist.

In the absence of severe pulmonary restriction (which might delay pulmonary expansion), such a major anterior interpleural communication allows any upper-anterior chest tube in a supine or semi-sitting patient to drain non-loculated air or fluid from anywhere within the chest. This eliminates the risk of unilateral tension pneumothorax (which formerly developed occasionally within centimeters of a functioning substernal tube) during the period of postoperative ventilatory support.

Postoperative empyema has not followed such "wide pleural opening," nor has bilateral spontaneous pneumothorax been seen. If it is deemed important to keep left and right pleural spaces separated (perhaps a patient has known pulmonary disease on one side), a loose pericardial closure can usually be achieved after taking down only one pleural reflection (leaving the other pleural envelope intact): When faced with a strong likelihood of recurrent spontaneous pneumothorax, one might consider oversewing or stapling both lung apices at the time of wide pleural opening (during pericardial closure).

Interestingly, the single sternotomy wound infection among these 300 patients was cured with antibiotics following subcutaneous drainage of creamy pus, despite a pledgeted closure of an underlying ventricular aneurysm. (Except when faced by an exceedingly soft ventricle or aortic wall, I prefer pericardial "bits" or strips for pledgets: Non-constricting pericardial closure is not compromised by removal of pericardial strips from the free pericardial edge, since directly overlying pleura remains intact.) The "slightly loose" sternum of this patient soon stabilized as his wound infection resolved.

Poststernotomy Mediastinal Infection

Many people, objects, techniques and bacteria have been blamed for causing sternotomy wound infections. The present low and declining incidence of this major complication, and the multitude of possibly related variables, have created an environment more conducive to ritual than reason.

During our long surgical training and early years in practice, we often accommodated to the "beliefs" of more experienced surgeons. At various times I have been forced to forgo pleasant conversation in the operating room, laugh only during inspiration (it sounds funny, but you get used to it), use only this or that special scrub and antibiotic (there have been many dozens of these miracle combinations over the last 30 years), wear double gloves or double masks, change gloves every ten minutes, use skin towels, count to three, or simply mumble incantations while irrigating with antibacterial solutions, gain entry to the operating room by bowing to press a foot switch that dispensed antibacterial solutions, or watch others stuff Neosporin ointment up their nose: Subsequently, I have violated all of these "minimum precepts for uninfected surgical wounds" without adverse consequences.

Of course, one problem with any prophylactic routine is the difficulty encountered in explaining the rare infection that still occurs. Although some would simply redouble their efforts under such circumstances (i.e., twice as much Neosporin, twice as far up the nose), it may be more useful to view an unusual event like wound infection to be independent of anything that is truly "routine" (although if one forgot or had no time to perform such routine, this could still be related to the unusual event, being itself uncommon; for infrequent events that are truly unrelated do not usually coincide). So how does this help us prevent sternotomy infection?

Well, those early poststernotomy wound infections that I have seen were all heralded by serosanguinous (or less commonly, purulent) drainage and progressive sternal instability: Other signs such as fever, leucocytosis and chest pain were less specific. If one can assume that every sternotomy wound receives some bacterial contamination, what local factors are likely to increase susceptibility to wound infection?

Early in my series, a diabetic patient with resolving lateral chest wall carbuncle required urgent coronary bypass; not surprisingly, about ten days after surgery, the patient developed fever, sternal instability and a serosanguinous wound discharge: His obvious wound infection required reexploration and closure, which delayed complete recovery by several weeks. Common enough: But why did he heal secondarily, yet not primarily; after all he still had diabetes and a resolving carbuncle? Could we perhaps prevent poststernotomy wound infections if we routinely reexplored all sternal closures a few days after operation? How does a secondary closure differ from the first?

Perhaps there are other factors involved here besides nearby infection and "low host resistance." Take serosanguinous drainage, for example: A simple subcutaneous wound infection is generally purulent; if serosanguinous, it probably represents infection of a wound hematoma, rather than secondary bleeding into an already infected wound. Although not "normal," serosanguinous poststernotomy wound drainage is seen far more frequently than sternotomy infection, and such drainage generally appears to come from deep to the sternum, over areas where the sternum is broken or fits back together poorly, especially with Valsalva-type efforts by the patient that increase intrathoracic pressures.

The apparent preponderance of serosanguinous over "just plain purulent" drainage from infected sternotomies, suggests that incompletely drained, postoperative, anterior mediastinal fluid collections increase the likelihood of sternotomy wound infection. Could the appearance of creamy pus even be taken as an indication that the wound infection is probably limited to subcutaneous tissues and the underlying bone?

I mentioned that the diabetic patient with chest wall carbuncle was operated upon "early in my series." One might conclude that I was inexperienced then and should know better by now, but I haven't seen another such case and might do it again anyhow, so perhaps my judgement has not improved. Although most surgeons would probably agree that their wound complication rate decreased as they learned from past mistakes, why should experienced surgeons have fewer sternal infections?

Casual observations do not suggest that operating room silence or clean shoes, clean nose or clean fingernails coincide with increasing experience: Sometimes there is a little improvement in draping technique, but often the reverse appears true. Experienced surgeons do get better sternotomy closures, however, and they tend to encounter fewer bleeding and drainage problems. The relationship between bleeding and drainage is obvious: A more secure sternotomy approximation also decreases bleeding and expedites healing.

While the secondary closure of an infected sternum may well be weaker than a "routine" (uninfected) sternotomy closure, such secondary closure can still heal successfully if secure bone-to-bone approximation can be achieved and maintained. So only under these circumstances, I leave the numerous twisted ends of my figure-of-eight peristernal wires "long" and pointed anteriorly (which does endanger anyone performing CPR or "chest thump").

This allows me to tighten the patient's wires in their bed whenever loosening is detected. For if movement between infected sternal halves is not prevented, infection persists and disruption soon follows (appropriate substernal tube drainage obviously remains important after reclosure of an infected sternum).

Conclusion: It seems likely that inexperience becomes associated with an infected sternotomy via excessive bleeding, inadequate drainage and insecure sternal approximation. All of these factors also contribute to a persistent, serosanguinous, anterior mediastinal fluid pocket and delay healing, thereby prolonging the time during which the sternotomy wound remains susceptible to infection.

It seems likely, therefore, that sternotomy wound infection can usually be prevented or cured by a better, more stable, sternal approximation with more effective substernal drainage. Which may be why increasing experience improves results and why it is not necessary – or even useful - to reopen all sternotomies a few days after surgery in order to prevent or preempt wound infection.

Prompt healing of the rare sternotomy performed for wide pericardiectomy in the presence of underlying pericardial infection and empyema, is further evidence for the importance of optimal substernal drainage (see Chapter 15).

And while one might anticipate that a hemi-sternum should show increased susceptibility to infection after relocation of its underlying internal mammary artery, it is reported that the incidence of sternotomy wound infection has not been altered by such vessel removal. This suggests a countervailing benefit from the wide pleural entry commonly created while mobilizing the internal mammary artery.

Under these circumstances, however, it is alleged that any sternal infection that does appear, tends to become "more severe" and difficult to eradicate. Whether such "severity" refers to the degree of sternal necrosis, or to the extent of anterior mediastinal and pericardial involvement in such infection, has not always been clear.

The preceding discussion leads me to guess, however, that even "severe" sternal infections rarely extend through a well-drained anterior mediastinum to involve the properly decompressed postoperative pericardium. Perhaps those with larger surgical practices or more postoperative infections can help to clarify these issues.

Summary: An "ideal" pericardial closure can reduce poststernotomy morbidity by eliminating substernal fluid collections.

Approaching the Infected Sternum

Poststernotomy wound infections vary widely in virulence and destructive involvement of surrounding tissues: Any routine response to the wide variety of possible clinical problems associated

with sternotomy wound infection might thus be inappropriate, inadequate or excessive.

Matters for individualization include how widely to drain any subcutaneous infection, whether to reopen the sternotomy, whether to irrigate all exposed internal surfaces with sterile saline, or with antibiotic or antiseptic solutions, whether and how much to explore or debride, whether and how to reclose the sternotomy, or even whether and when to resect sternum widely or perform a muscle transplant.

It is clear that obviously dead tissues and infected fluid collections demand debridement and ongoing drainage. Specifically appropriate, full course antibiotic therapy also seems crucial with any but the most limited subcutaneous infections.

All of the early poststernotomy wound infections that I have encountered had only minimal sternal necrosis and minor mediastinal fluid collections (and thus did not require debridement, beyond wiping sternal edges free of exudate). Maintaining firm approximation of sternal halves, as well as ensuring free substernal and subcutaneous drainage, were key to uneventful healing of all these wounds.

More specifically, any sutures placed during reexploration for infection should probably be of "minimally reactive" monofilament (e.g. wire or polypropylene). At least one No. 28 or No. 32 French catheter is usually left in a retrosternal position and brought out inferiorly, or else the mediastinal pleura is opened widely into one (or preferably both) pleural cavities. Each contaminated, widely opened hemithorax is carefully irrigated before being drained separately by an anterolateral chest tube - and generally another more posterior tube is placed as well. The sternum is then rewired securely.

How Loose is Too Loose? A Review

A stable bone closure is essential for sternotomy wound healing, especially in the presence of infection. Independent movement of reapproximated sternal halves is always cause for worry: When encountered soon after sternal closure or in the presence of infection, such movement generally progresses to sternotomy dehiscence within a few days.

Dehiscence of an increasingly loose sternal closure either follows wire breakage (due to repeated flexion of wires by the independent motion of each sternal half during respiration and other movements), or it results from still intact wire loops finally "sawing" at least one of the sternal halves into multiple independent segments.

Significant and progressive sternal loosening within a few days of competent closure may be an early sign of wound infection and impending dehiscence. Major, independent movements of the sternal halves can eventually erode underlying tissues, such as saphenous vein grafts or right ventricle.

Later loosening (say 5 to 15 days postoperatively) may not progress if the wound remains uninfected, although a rare pseudarthrosis could possibly develop if such motion persists. Minor sternal clicks, or other occasionally noticeable movements at the site of a sternal or nearby rib fracture, may cause discomfort for some weeks but they are not hazardous. When possible, it does seem best to avoid positions or exertions that cause these sternal or rib movements - until further healing can eliminate the problem.

Technique for Sternal Closure

A stable well-drained sternal approximation will heal uneventfully unless badly infected. "Figure 8" or pulley sutures placed just around sternal periosteum can maximize the early strength of this bony approximation. Wire on a heavy needle may also be driven quite readily through bone where the sternum is very wide, as it usually is up near the clavicles.

Frequent palpation of the sternal notch, with an "upward" (toward the patient's head) or downward (toward the patient's feet) pull on appropriate wire ends before they are twisted together, will prevent misalignment of sternum (with one-half of the sternum more cephalad than the other). Significant vertical displacement of this sort can allow prolonged oozing from poorly compressed portions of opposing sternal marrow; it may also be quite noticeable to the patient, and an ongoing focus of unnecessary concern.

A total of five heavy monofilament wires will routinely achieve strong sternal approximation: Bony closure begins by placing a simple wire suture across the top (manubrium) and then continues with figure 8 wire sutures below, with each figure 8 spanning one costal cartilage. Thus, the first figure 8 enters the left first anterior inter-space, comes out through the right second, in left second, out through the right first. Second figure 8 goes in left second, out right third, in left third, out right second and so on (with the most distal wire sometimes only a simple suture). One can usually avoid injury to internal mammary vessels by passing the needle closely along the sternal periosteum at the most medial aspect of the anterior interspace.

Heavy wire sutures placed near the xyphoid are more likely to break and become symptomatic after some months or years, due to constant respiratory movement of the costal cartilages, so one usually ends the stainless steel wire approximation of sternum just above the xyphoid. Nevertheless, an occasional patient will return after some months with discomfort that seems possibly or definitely due to broken wires.

Painful wires are (sometimes, preferably?) removed during a brief general anesthetic, for their removal can sometimes require a very strong pull (and posterior sternal periosteum cannot safely be "frozen" with local anesthetic to allow the wire loop to slip out painlessly). If intact, a figure 8 wire for removal is best divided at the anterior loop that does not contain twisted ends, then pulled out by the "knot."

An already broken wire is best removed by tugging on the knot after the distal loop is mobilized - and perhaps after the longer wire end has been trimmed. Once they have become deeply embedded in a young person's growing sternum, some wire sutures simply cannot be pulled out prior to repeat sternotomy (just as barbed wire may become irreversibly incorporated in a growing tree).

At initial sternotomy closure, one should check for substernal bleeding during insertion of these sternal wires, before they are tightened. Any significant substernal vessel bleeding, including internal mammary, should be suture-ligated (by a horizontal suture around the injured internal mammary vessel above and below site of injury, or a vertical suture placed around any horizontal vessel branch that is bleeding closer to the sternal edge). Exposure can be awkward with wires in place, so occasionally these must be removed, then replaced after troublesome substernal bleeding has been controlled.

The true utility of sternotomy closure with a figure 8 suture then becomes apparent: As a first step, one can lift the patient part-way off the table with the two suture-ends held in heavy clamps, thus securely tightening the lower half of each wire loop, without significant likelihood of wire breakage. It

then requires only minimal traction while twisting the wire ends, to ensure a tightly wired sternum. There is far less risk of wire breakage when gently twisting (around each other) the two ends of such an already tightened figure-of-8 wire loop, than during attempts to improve poor sternal approximation by further twist-tightening "the knot" - for metal fatigue rapidly weakens a wire as vigorous twisting continues.

After twisting, trim the tightly twisted wire ends and then bend the knot inferiorly into the sternotomy (to permit downward sponging of the wound without snagging wire tips). Firm presternal approximation of pectoral muscles and fascia is then carried out, using interrupted 2-0 Vicryl. This sturdy muscle-fascia layer markedly strengthens the sternotomy closure and decreases stress on wire sutures, especially after an off-center sternotomy, or with an osteoporotic or devascularized sternum. In fact, that anterior muscle-fascia closure essentially guarantees sternal healing unless severe infection supervenes. The prominence that this unnatural muscle approximation causes over the center of the sternum will diminish to normal as months pass.

A calcified anteriorly curved xyphoid can be removed to expedite careful approximation of the anterior rectus fascia; such a xyphoid could otherwise become the weak point in an upper abdominal closure and give rise to a symptomatic, perhaps not easily detectable, epigastric hernia. One attorney patient almost filed suit against his cardiologist after xyphoid removal and incisional hernia repair readily corrected his "abnormal xyphoid fixation": Almost two years had been spent in unsuccessful psychotherapy for this "interesting neurosis."

Wires Usually Require Intermittent Tightening Following Secondary Closure of an Infected Sternum

As mentioned earlier, on reclosure of an infected or probably infected (not just contaminated) sternum, I leave the twisted wire ends long and pointing directly anteriorly, with intervening monofilament sutures approximating whatever pectoral muscle-fascia layer is available to strengthen the sternal closure: Most of the skin and subcutaneous tissues are left open. These long wire ends make it possible to correct later sternal loosening as it is detected (by another vigorous "lift" on the twisted ends to "snug up" the distal loop of the Figure 8 suture, before again carefully twisting it tight).

Loosening of the sternal halves is expected and looked for daily in all reclosed, infected cases. An initial sign may be visible or palpable independent motion or "clicking" of the sternal halves; such movement is always associated with a small amount of "froth" appearing between the sternal halves or even through nearby wire suture holes. This froth is generated as sternal movements churn and drain the underlying protein-rich fluids.

"Frothing at the sternum" has had no obvious ill effect upon the patient's ability to move air efficiently during unassisted respiration. If a patient with widely opened pleura were to develop a severe sternal infection, it is certainly possible that several large chest tubes on vigorous suction (and even prolonged positive-pressure assisted ventilation) might become necessary to maintain lung expansion while the sternum remained porous and frothing: Early muscle transfer might be another method of sealing off the mediastinum if free pleural air access did develop (but I have had no experience with this postulated problem).

Early and repeated wire tightening - performed in the ICU or ward bed after appropriate narcotic analgesia - has prevented recurrent sternal dehiscence until healing was achieved in perhaps a dozen

consecutive reclosures of infected sternotomies. Three of these infections came from my own practice, the rest appeared after sternotomy performed elsewhere.

The momentarily painful but essential "tightening procedure" described above has provided sufficient stability to allow healing of the infected sternum in all but one patient (this man was referred with an infected sternal separation as well as hepatorenal failure, and closed for "cosmetic" reasons; he died two days later with a still-stable sternum). Tightening may be required about every 2 days for over two weeks while sternal fibroblasts race to achieve healing before the wires cut completely through the sternum.

Once the formerly-loose-and-infected sternum has stabilized, (usually about two weeks after reclosure), I clip the twisted wires off at the skin surface and schedule an overnight readmission about a month later for complete wire removal. (I prefer to remove the wires solely because of a postulated greater risk of ongoing sternal osteomyelitis - it certainly might be sufficient to merely trim the wires back well below the skin.)

Here again, it is persistence that "pays off" (rather than genius or great skill), as well as good rapport with the patient who is repeatedly hoisted by his own sternal wires as they are snugged up. Incidentally, if you even think that these sternal wires are loosening on Friday afternoon, tighten them on Friday afternoon; otherwise the sternum will probably have been cut into many segments before Monday (and your "on-call" friend will likely procrastinate if not already familiar with this rough and ready "technique").

Caution: Cardiopulmonary Resuscitation (CPR) must be performed with great care on such patients, to avoid severe injury to hospital personnel from those sharp, bacteria laden, anteriorly directed wires. Always leave instructions to place a thick towel or bath blanket over the sternotomy bandage prior to CPR, should this prove necessary (so far it has not). Especially advise any colleagues with a flair for the dramatic, not to "fist thump" such a patient during CPR.

A substernal drainage tube is generally left in place under the reclosed sternum for as long as it continues to function: This tube can often be removed at about the time that sternal stability seems assured. I have not used irrigations to any great extent. Antibiotic therapy (for presumed early sternal osteomyelitis) is continued for a number of weeks after apparent complete healing.

Other Approaches to the Infected Sternotomy

Muscle transfer procedures are reportedly very effective in achieving healing when severe sternal necrosis precludes sternal rewiring (is this more common after utilization of one or both internal mammary arteries for coronary bypass?). According to the literature, the pectoralis major muscle can readily be advanced onto the heart (via the open debrided sternotomy) with its thoracoacromial arterial supply intact: This usually requires division of some or all of its humeral attachment. The contralateral muscle is then advanced to meet the first, without humeral detachment.

Problems with the lower sternum reportedly are better dealt with by swinging up omentum (or rectus muscle if the internal mammary artery is still intact), with split thickness skin grafts recommended 48 hours later to cover the omentum. I have no experience with such transfers, but they sound appropriate for circumstances where secondary sternal closure is not feasible. (It has also been recommended that an experienced plastic surgeon take charge of such muscle transfers.)

Case Report: Erosion of an Unstable Sternum into Right Ventricle

Many years ago, a patient with acute poststernotomy infection (after coronary artery bypass surgery) had his sternum minimally debrided and packed open, with tubes placed for irrigation and drainage. The gauze pack was gradually teased out over several days, as irrigation continued. Thereafter, the unwired sternal edges moved back and forth several cm during each spontaneous respiration. About one week after his sternal reopening, the patient was rushed back to the operating room when a large gush of blood issued from the open wound as it was being dressed.

As the free sternal edges were elevated at surgery, a large erosion into the right ventricle became copiously evident. This was tamponaded by fingers at first, then closed with heavy monofilament sutures (that included nearby edematous, contaminated pleura and pericardium in each stitch, to help bolster the damaged right ventricle wall): Bleeding stopped promptly.

The sternum was then securely rewired and the skin closed loosely. Regular antibiotic irrigations into the substernal chest tube were continued, as were systemic antibiotics. The patient recovered slowly but uneventfully.

Summary: This full-thickness defect of the right ventricular wall was closed with nearby living tissues: It healed despite the presence of gross infection. "Possibly helpful" factors included the relatively low chamber pressures of the right ventricle, and excellent blood flow through that chamber, which allowed easy antibiotic access to the infected tissues.

Posterior Drainage of Mediastinal Infection

The vertical paraspinal incision formerly undertaken for posterior mediastinal drainage ("costotransversectomy") included resection of short posterior segments of ribs with their adjacent transverse processes. This extrapleural approach was helpful for drainage of posterior mediastinal (paravertebral) abscesses, especially tuberculous collections and particularly on the right side (where aorta was not "at risk"). This "direct" drainage path helped prevent intrapleural (or intraperitoneal) spill of infected material in the "good old" pre-antibiotic days.

Transpleural Drainage by Posterolateral Thoracotomy

Transpleural access to the mediastinum simply requires thoracotomy and division of the overlying mediastinal parietal pleura. There is little reason to reapproximate any divided mediastinal pleura (except perhaps locally to help bury a pneumonectomy stump, or possibly to buttress an esophageal suture line or cover a vascular graft). In fact, widely opened mediastinal pleura permits free drainage into the pleural cavity, converting a potentially complex and hazardous mediastinal drainage problem into an ordinary pleural drainage situation. With bacterial contamination or infection of the mediastinum, therefore, an especially long incision of the mediastinal pleura (it only takes a moment) can help prevent residual pockets of mediastinal abscess from building up under a rapidly thickening layer of parietal pleura.

Tube placement in "septic mediastinum" cases should follow the standard rules for tube location for major empyema involving the entire pleural cavity (at least two tubes, positioned primarily to achieve early, full expansion of the lung). In addition, it is desirable to have the tube drainage holes as close as convenient to the site of sepsis, while still avoiding tube impingement on (and possibly erosion

into) mediastinal structures.

With mediastinal sepsis (mediastinitis), the primary drainage tube is placed closer to the mediastinum than is usual after routine lung surgery, for the shortest drainage path is usually best under these circumstances. When in doubt regarding insertion of additional tubes for drainage, consider whether it is easier to remove a redundant chest tube early after operation than to perform an accurate closed thoracostomy into a contaminated fibrotic distorted inflamed postoperative pleural cavity with rapidly developing adhesions.

Anterior Mediastinal Approach for Mediastinal Drainage

Sternotomy, currently the most common approach to the anterior mediastinum, is regularly performed without opening either pleural envelope. After unintended pleural entry, it is sometimes best to enlarge the pleural defect in order to position an additional anterolateral intrapleural chest drainage tube.

How about accidental pleural entry from an infected substernal space? That could create a greater nuisance: Here it is usually best to open the pleura widely, then place two intrapleural tubes for optimum drainage of the potential empyema. Purposeful wide entry into both pleural spaces during repeat sternotomy can also expedite safe separation of sternum from heart or simplify exploration of a friable, infected mediastinum: Vigorous bilateral pleural drainage then becomes part of the remedy.

When a potentially infected pleural space is partially obliterated by fibrous adhesions, however, it becomes a matter of judgment how widely to open the pleura and divide adhesions. On the one hand, if the contaminated pleural cavity has scattered areas of free pleura between adhesions, there is an increased likelihood of loculated purulent collections after operation. On the other hand, extensive decortication of very fibrous adhesions could be unreasonable if it significantly increases the likelihood of major air leak and blood clot accumulations, for these also increase opportunities for intrapleural "pus pockets" to form.

Anterior Mediastinal Drainage in Perspective

Malignancy: Even when malignancy involving the anterior mediastinum is "incurable," associated fluid accumulations may kill the patient "prematurely": Some of these fluid collections respond to chemotherapy or radiation treatment, others must be drained. Surgical decompression can benefit a few of these patients significantly, but most continue their relentless downhill course.

Pericardial effusions due to malignancy are best drained externally by the shortest practical route: To avoid further dissemination of malignancy, the drainage path should not penetrate or traverse un-involved (pleural or peritoneal) areas. Malignant effusions are usually drained only to control severe symptoms or prevent their recurrence.

Infection: A mediastinal or pericardial abscess should be drained entirely and continuously over sufficient time to achieve obliteration of its cavity of origin (in order to prevent reaccumulation). A mediastinal abscess is often unroofed and converted into an empyema (which is more readily drained and cured through appropriate chest tube placement and antibiotic therapy). Purulent pericarditis is sometimes best treated by pericardiectomy and bilateral pleural drainage (see Chapter 15).

Other: Non-malignant uninfected pericardial fluid collections that have significant local effects and do not respond to or "cannot wait for" ordinary medical therapy, should be "permanently released" from their confines to a more volume-tolerant or absorptive part of the body, or else temporarily drained externally (in part, depending upon the anticipated duration of such excessive fluid production).

Pericardial fluid accumulations may be released (via a small pericardial window or subtotal pericardiectomy) into either or both pleural cavities, into the peritoneal cavity (not recommended, due to reported risk of visceral herniation into pericardium: Would wide-mesh polypropylene screen solve that problem?), or even to a subfascial, extraperitoneal location. A quantity of sterile fluid sufficient to cause pericardial tamponade is readily absorbed by peritoneum or pleura (just as excess cerebrospinal fluid has no apparent effect when transferred to these "more tolerant" locations).

Postoperative pleural drainage techniques are unchanged when pericardial fluid has been vented into the pleural cavity. Routine cessation of any air leak and diminishing fluid drainage allow chest tube removal by the usual indications (chest tube drainage of less than about 100 cc/day sterile fluid, appears readily absorbed by healthy adult pleura, so chest tube removal need not be delayed on this account). Pericardial fluid production must usually cease, however, before removal of an intra-pericardial or anterior mediastinal drainage tube is advisable (unless the pericardium was also decompressed into the pleural cavity or some other absorptive space).

Perforation of Thoracic Esophagus

The diagnosis of perforated esophagus is usually made on a patient with history of recent esophageal instrumentation, protracted vomiting or preexisting esophageal disease. After difficult passage of an esophagoscope, for example, a patient suffering perforation will note progressive anterior or mid-dorsal chest pain, aggravated by swallowing.

Within hours of such injury, examination will usually reveal rapid pulse, elevated temperature, and palpable crepitus in the lower, anterolateral neck. On auscultation, a crunching sound is sometimes heard with every heartbeat (due to regular displacement of an air pocket next to the heart). If the diagnosis remains in doubt, extravasation of swallowed, water-soluble contrast media provides radiographic confirmation: Also any ingested food coloring or methylene blue will reappear shortly from a chest tube placed within the often associated "contaminated hydropneumothorax" (empyema).

Mediastinal air from ruptured esophagus may break into either pleural cavity to cause pneumothorax. I have not encountered tension pneumothorax from this cause, perhaps because the volume of swallowed air is limited by dysphagia; also, it is likely that any "excessive" intrapleural air can easily reenter the open communication between the pleural space and the gastrointestinal tract.

A major tear of the lower esophagus permits free reflux of gastric contents into the pleural cavity. Even with adequate tube drainage, this is poorly tolerated, especially over a prolonged period. Unlike the standard empyema, gastric juice cannot be "walled-off" effectively within the pleural space (however, continuous saline irrigation by mouth, with appropriate chest tube suction drainage, reportedly allows healing - but watch the fluid and electrolyte balance).

Drainage

A chest tube should be inserted as soon esophageal perforation with pneumothorax or hydropneumothorax is diagnosed. If clear, yellowish fluid with curd-like fibrin floaters is obtained, this is diagnostic of perforated esophagus with gastric reflux from an empty stomach. Standard treatment of esophageal perforation includes cessation of oral intake, intravenous fluids, antibiotics and continuous nasogastric tube suction (unless trans-esophageal irrigation is proposed).

Repair

In almost all cases, thoracotomy should be performed promptly for significant full-thickness thoracic esophageal tear or rupture, as this approach usually carries less risk than "non-surgical handling. A "little" tear (possibly not full thickness, or not associated with significant extravasation of air and fluid) can sometimes be handled by nasogastric suction and antibiotics without thoracotomy, but such a program can be taxing. (I have no experience with the irrigation method of Santos. It could be an important advance.)

Reportedly, "spontaneous" late perforation of the mid-thoracic esophagus can also occur even decades after pneumonectomy for inflammatory lung disease: Such a fistula will allegedly often heal during a few weeks of nasogastric tube feeding (allowing eventual closure of the postpneumonectomy cavity by some version of the Clagett procedure; see Chapter 4). Muscle transfer procedures are reportedly helpful when the spontaneous esophagopleural fistula does not heal following several weeks of tube feeding.

One naturally hopes to minimize the seriousness and possible sequelae of any iatrogenic injury, and this must be kept in mind to avoid under-treatment. And in a delayed or neglected case, diversion or even esophagectomy may be safer than simple repair of a torn esophagus. In any such high-risk case, effective chest tube drainage is mandatory. One might consider performing cervical esophagostomy, gastrostomy and heavy chromic suture ligation of the abdominal esophagus in the truly desperate case (but I have no experience with that either).

A full-thickness tear in cervical esophagus is usually most safely handled by early repair and drainage or, occasionally (with minor tear), by antibiotics and nasogastric tube feedings for a few days. The therapeutic approach depends upon a number of factors, such as apparent size of tear, collections of fluid and air requiring drainage, and response to antibiotic treatment alone (if that course is selected initially).

Absence of gastric reflux to the cervical level is a therapeutic plus with the usual tear at this level, so one might reasonably keep the patient in a semi-sitting position as an adjunct to healing, regardless of therapy selected (and especially while the nasogastric tube remains in place, for an indwelling NG tube will itself promote reflux). As a "less than regular" esophageal surgeon, I find the cervical esophagus easier to identify with an indwelling NG tube in place: At surgery, a finger exploring down onto the prevertebral fascia initially can mobilize the esophagus gently on three sides, before careful separation of esophagus from trachea is undertaken.

Case Report: Perforation of Esophagus by Chest Tube

Consultation was requested when a mid-esophageal leak was demonstrated soon after transthoracic hiatus hernia repair (plus long esophagomyotomy for mid-esophageal diverticulum).

Findings: In an effort to improve posterior mediastinal drainage, a transversely directed chest tube had been so located that its tip rested against the esophagus: After two days this tube eroded into the esophageal lumen.

Thoracotomy was required for successful repair of this esophageal leak. Following suture repair (buttressed with a pleural flap) the chest was irrigated out carefully. Two large chest tubes were placed laterally, this time directed toward the apex (as they should have been initially), with the more posterior tube passing close by the perforation. The more posterior chest tube remained in place for five days, until barium swallow examination showed absence of esophageal leak: By that time, chest drainage was serous and less than 50 cc per day.

Comment: A chest tube (and especially its tip) must never press upon significant intrathoracic structures such as subclavian vessels, aorta, heart, brachial plexus or esophagus: Even the side of an overly stiff rubber chest tube may erode into lung (this used to be an occasional cause of recurrent postoperative air leak), so chest tube material, size and positioning are crucial to benign recovery.

Important neurovascular structures in the apex are adequately protected when the tip of the properly secured, anterior chest tube reaches no higher than the anterior first rib on chest roentgenogram. (I currently prefer No. 32 French silastic chest tubes for postoperative pleural drainage in the adult.)

Case Report: Spontaneous Esophageal Leak

Over two decades ago, I had just entered private practice when an old gold miner came in with a major left pleural effusion and pneumothorax. Closed thoracostomy drained serous fluid with curdlike floaters: Water-soluble contrast study of the esophagus confirmed esophageal leak.

Esophagoscopy showed gray, friable, tumorous tissue, heaped up into the esophageal lumen and bleeding easily on biopsy. The pathologist reported fragments of esophageal muscle and granulation tissue. Repeat esophagoscopy and biopsy had same diagnostic result. The nasogastric tube had to be passed into the stomach during esophagoscopy, for it kept deviating out of the huge esophageal rent into the pleural space.

This old man with perforated lower esophagus and copious chest drainage had no history of recent emesis: In addition, the lesion grossly resembled esophageal carcinoma. I recommended exploratory thoracotomy. The patient refused, unless I was sure I could "fix it." I said I doubted that I could. He said, "Doc. If I don't get back to my gold mine, you don't get paid."

I didn't get paid. He died after more than a week of intensive chest drainage and intravenous fluid therapy (presumably of infection disseminated by intrapleural gastric juice). Autopsy showed a several-inch-long esophageal rent and no evidence of malignancy.

Conclusions: (1) Esophageal rupture can present without a history of pain or vomiting. (2) "Benign" rupture can closely resemble malignancy on visual inspection through an esophagoscope, especially when many hours have passed since perforation. (3) With cervical esophagostomy, transhiatal esophagectomy and gastrostomy - or cervical esophagostomy, ligation of the distal esophagus, gastrostomy, and later colon transplant - we might have saved this tough old Alaskan.

Histoplasmosis

"Sterile" fibrosing mediastinitis is a "benign" unusually nasty condition that can lead to superior vena caval obstruction with severe, symptomatic swelling and congestion of upper arms and head.

Treatment of Fibrosing Mediastinitis

I am aware of no reliable, long-term cure for this condition, which has allegedly followed a variety of medical treatments and illnesses. Successful mobilization of a still-patent but entrapped superior vena cava has been reported; an enlarged saphenous (spiral venous) graft or externally-stented Gore-Tex tube have both been utilized successfully to bridge the gap between the innominate or internal jugular vein and the right atrial appendage, usually with a relatively short-term "follow-up": The superficial femoral vein has also been recommended as a bypass conduit. Successful bypass has also been reported using a tube constructed of pericardium. Even sternotomy has been suggested for palliation, with the firmly wired sternal "closure" held apart during healing by several 3-4 cm long rib struts (to help decompress the "full" mediastinum).

My only operative experience was with a 10 year-old girl "bypassed" with a dacron graft more than a decade ago at the time of exploration for a presumed "mediastinal mass" obstructing the superior vena cava: This graft clotted after several months of medium-dose heparin therapy at home. The 1986 choice of venous bypass material would include spirally sutured (and thereby widened) saphenous vein, superficial femoral vein, pericardium and externally stented Gore-Tex; certainly, knitted and woven Dacron and Teflon bypass grafts no longer seem useful for this purpose.

Prevention of Fibrosing Mediastinitis

On occasion, one encounters a middle-aged patient from the Midwest, with a solitary, paratracheal, bilobed convexity bulging into the upper right pleural space (a "reverse three" appearance on roentgenogram). The skin histoplasmin test is commonly (though not always) positive, and the diagnosis is almost surely caseating mediastinal histoplasmosis involving the paratracheal lymph nodes. As mediastinal histoplasmosis is reportedly one important precursor of "benign" mediastinal fibrosis, any surgical intervention that markedly reduces or eliminates that likelihood would seem a prudent investment for the "good risk" patient.

Operative Procedure: The operation consists of unroofing these ballooned-up nodes, then scraping and suctioning out the profuse creamy mess within. These infected nodes need not be (indeed cannot be) excised, for in their deeper aspect, these multiseptate nodes commonly burrow between trachea, esophagus and great vessels. Concurrent antifungal therapy is not indicated, and routine postoperative chest drainage appears sufficient to prevent pleural complications.

To the best of my knowledge, such patients have all done well after surgery. Postoperatively, it is easier for the formerly dubious assistant surgeon to agree that further extension or rupture (of such an ongoing sclerosing burrowing infectious mediastinal process) might well lead to severe mediastinal fibrosis with superior vena caval obstruction.

CHAPTER 11: CARE AND REMOVAL OF CHEST TUBES (partly a review)

Never obstruct a chest tube;.. Ambulating the patient with a chest bottle;... The water seal `prime";... Loss of water seal;... A high fluid column may interfere with chest drainage;... Needling latex tubing;... Needling plastic tubing;... Milking tubing;... Stripping tubing;... Full lung expansion brings free pleural fluid out through the chest tube;... Pleural fluid draining around a chest tube may also signify full lung expansion;... Timing of chest tube removal;... Remove chest tube only after air leak ceases;... Remove chest tube only after fluid output decreases;... Technique for chest tube removal;... Continued fluid drainage from the tube site after tube removal;... Skin edge bleeding;... Suture closure of chest tube holes is not advisable;... Special case: Tube removal after pneumonectomy;... A skin clip can close the chest tube hole after pneumonectomy and in young children;... Removal of a tunneled tube;... Mediastinal tube removal;... Accidental chest tube removal;... Chest tube placed into liver or spleen;... Case report: Removal of chest tube from the liver;... Case report: Removal of chest tube from lung parenchyma;... Residual pneumothorax following chest tube removal;... Residual air/fluid level following chest tube removal;... Sample nursing instructions for chest tube care;... Sample instructions for chest tube removal.

Never Obstruct A Chest Tube

Unobstructed drainage is essential with any pleural drainage system. Therefore, especially if there is ongoing pulmonary air leak, never allow chest tubing to be clamped or otherwise obstructed, for that can rapidly lead to tension pneumothorax and subcutaneous emphysema. Removal of all clamps from the vicinity of any patient with chest tubes can help reduce the likelihood of such error.

Even with minimal air leak, a chest tube should never be clamped. Too often, the patient who gradually "took a turn for the worse" over several hours, is found to have clamps on the chest tubes under his gown (or even under a reinforced dressing) and again, tension pneumothorax. Those clamps usually were applied "just for a minute" while moving the patient, then forgotten. The ever-present risk of chest tube disconnection or accidental withdrawal should encourage careful handling, secure connections and firm fixation of the chest tube to the skin, rather than such clamping.

An absolute "never clamp" must also be emphasized for the patient with or without pulmonary air leak who is being transported by "less-pressurized" aircraft, for a 25 percent in-flight reduction of "atmospheric" pressure will expand by one-third any intrapleural air pocket unless ongoing open-tube decompression is provided.

In the presence of air leak, when "something seems wrong" with the chest drainage system and the patient is "in trouble," immediate disconnection of the chest tube from any possibly blocked tubing or drainage apparatus will relieve tension pneumothorax: Surely such decompression makes more sense than to clamp a chest tube that was inserted specifically to prevent or relieve exactly that serious life-threatening condition.

Clamping, kinking, tube displacement from the pleural space, a tube tip buried in clot, a clotted tube or obstructing clot "hung up" in the tube or tubing, dependent fluid-filled loops, a patient lying upon his tubes, fluid accumulation far above the water seal straw tip, a large "collecting-chamber" air space inserted between chest tube and water seal, or a malfunctioning or inadequate suction device, all prevent easy release of above-atmospheric-pressure air collections from the pleural space. Any obstruction to free air drainage promotes intrapleural air and fluid retention and increases the risk of lung and heart compression, empyema and lung entrapment. Under such circumstances, a chest tube temporarily opened to the atmosphere can bring great relief.

Ambulating The Patient with a Chest Bottle

In the past, ingenious bottle holders on wheels facilitated patient ambulation with heavy glass chest bottles. Some modern well-designed lightweight low prime volume, plastic water seal containers can simply be carried about by their tubing. It is bad practice to tape any chest bottle to the floor as ambulation should be frequent; also, a glass bottle fixed to the floor is more readily broken (by accidental kick or dropped bedrail) than a bottle mounted on wheels.

A well-designed carriage made the one-gallon glass cider jug less likely to tip, but accidental upset of a simple water seal chest drainage bottle is never a disaster anyhow. If broken, the water seal bottle should be replaced as soon as possible; meanwhile the tubing is left open, especially if there is air leak. On the other hand, if still intact, a tipped water seal unit is merely righted to restore function. In contrast, the complex flimsy plastic "three-bottle-equivalent" systems may require replacement of the entire costly drainage unit each time it is accidentally knocked over; another unnecessary restraint on adequate patient mobilization.

The Water Seal "Prime"

Usually 1000ml of normal saline was poured into the sterile glass one-gallon "cider jug" in order to cover the glass straw tip and establish a water seal. Sterile chest tubing was then connected between the water seal straw and the chest tube. A short horizontal strip of tape on the outside of the water seal bottle marked the original fluid level.

At present, commercially available water seal units vary considerably in volume capacity, weight, complexity and stability; often they require far less prime than the old cider jug. With any water seal system, daily or hourly fluid drainage increments may be marked as indicated (date and time) on a tape running up the side of the water seal bottle.

Such marks are not helpful when accumulating drainage is frequently decanted from the water seal bottle to prevent back-pressure to air leak: Under these circumstances, the volume removed should be recorded on the intake-output sheet if it has relevance to therapy (which it often does not).

Loss of Water Seal

If the chest bottle has a removable cap, a large label on the bottle should state, "Chest Drainage, Do Not Empty." Too frequently, in the rush to complete "intake and output" charts, a chest bottle with removable cap is completely emptied by an aide; the resultant loss of the one-way water seal function usually leads to persistent simple pneumothorax.

A High Fluid Column May Interfere with Chest Drainage

When significant fluid drainage persists for several days after cessation of air leak (or if there never was an air leak), the entire chest tubing may fill with fluid: This fluid column can exert considerable suction and pull lung tightly against the chest tube, blocking further drainage. To improve drainage, high fluid columns are usually released by allowing air access (see Chapter 8).

Needling Latex Tubing

Latex chest tubing is most easily emptied of excess fluid by piercing the tubing wall near the chest tube connector with a sterile hollow No. 19 or No. 20 needle (after antiseptic wipe of tubing): This allows atmospheric air entry into tubing lumen and chest cavity, hopefully without introducing bacteria or causing a permanent air leak in the tubing wall.

An excessively tall fluid column released by such air access will drain rapidly into the water seal bottle. Residual intrapleural fluid may then have access to previously occluded chest tube holes and escape from the chest.

A hollow needle with syringe attached can also be used to aspirate air from the upper part of the chest tubing, thereby raising a fluid column within straw and tubing (this hanging fluid column delivers sustained suction when there is no air leak; see Chapter 8).

Needling Plastic Tubing

Needle penetration of clear plastic tubing is undesirable as it usually leaves a permanent hole for atmospheric air entry. Although tilting the water seal bottle will allow air access to the open straw tip and chest tubing (as will disconnection of the chest tube), an needle port on the chest tube connector provides more practical access to the chest tube lumen (for sampling fluid, or entry or withdrawal of air).

Some formulations of plastic tubing are too thick walled or rigid to allow effective milking or stripping; softer, slightly tacky plastic tubing is easy to milk or strip, and it coils more readily than latex tubing, without springing off the bed or kinking. Another major advantage of properly formulated clear plastic tubing over latex tubing is the ready visibility of fluid drainage, as well as a decreased tendency of such drainage to clot within and block the tubing.

Milking Tubing

In milking, a pressure wave is repeatedly forced back into the chest through the air or fluid column within the tubing: This is achieved by pinching or sharply angulating the upper chest tubing with forefinger and thumb, while repeatedly compressing tubing closer to the patient between fingers and palm of the other hand, in order to force chest tube contents proximally. When the distal chest tubing is adequately pinched shut, enough pressure may be created during milking to blow clot or debris back out of the chest tube into the chest, thereby reopening the chest tube lumen. (Any small clot pushed back in this fashion will soon liquify and drain out or be absorbed.)

With experience, the difference between milking of an open, or a clotted, or a "kinked off" chest tube soon becomes palpably evident. In the early postoperative period, it is helpful to milk the chest tube every time you pass the patient, as this improves tube function, allows an ongoing evaluation of drainage and is minimally disturbing to the patient.

When a chest tube tip is embedded in clot, one can easily flatten a hands-width of tubing that was just full of air or fluid, so this content must have moved proximally into the pleural space (provided finger-occlusion completely blocked the distal tubing); however, the chest tube still will not drain, nor does any air that was "squeezed in" come back out, and there is no tidal. A one to four day wait for clot lysis may then be necessary before drainage can be resumed (unless early reoperation must be undertaken to control ongoing blood loss or decompress lung and heart).

Similar ease in milking (also without resumption of chest drainage) is seen if the side-hole most distant from the chest tube tip is not within the space to be drained: Although "tube" air or fluid is readily pushed back out of this distal side-hole, surrounding soft tissues immediately reocclude it. A clotted tube tip and more proximal side-holes often remain blocked despite "successful" milking, due to the fact that the most distal side-hole allows easy escape of each milking "pulse."

Reemphasis: Milking often cannot blow clot from the chest tube tip, so take care to position all tube side-holes within the space requiring drainage. Although any tube side- or end-hole can serve during chest drainage, the hole closest to the outside atmosphere is likely to serve longest, especially when removing fresh bloody drainage; proper placement of this distal side-hole then becomes the most critical factor in tube position (this discussion refers only to fluid drainage. Upper anterior tubes for air removal are unlikely to become embedded in clot in any case: Obviously, however, in the presence of air leak, the upper anterior tube must always remain open).

Stripping Tubing

"Stripping" the chest tubing applies a pulse of suction to the chest tube: This is not usually necessary or helpful except to push clot or thick fluid along manually within easily clotted latex tubing. To "strip" tubing, it is necessary to block the proximal tubing with one thumb and forefinger, then slide the other thumb and forefinger distally from this point while completely compressing the tubing lumen. Some folks grease the chest tubing (outer surface) prior to stripping; others even use specially designed rollers.

The idea behind stripping is that a sudden release of the proximal compression will allow just-flattened distal tubing to spring open, sucking fluid and clot down from the chest tube. Depending upon tubing type, size and elasticity, there may be more or less reason for clot to hang up. Stripping can momentarily apply strong suction at the intrathoracic chest tube drainage holes. This could be hazardous if the tube was poorly located (next to a vein graft, perhaps).

Modern non-wetting plastic tubing on a connector with the same internal diameter, offers less reason for clot to hang up. A clotted segment of plastic tubing can usually be cleared by repeated squeezing to directly "fragment" its contained clot, while keeping the tubing tilted toward the chest bottle so that loosened broken clot can more easily move distally (downhill).

When freshly shed blood drains through plastic chest tubing, it will deposit a strongly adherent, irregular fibrin and clot layer inside the tubing. This indicates that the "foreign" tube surface has partially defibrinated the fresh bloody drainage in a similar fashion to that seen when fresh blood is stirred with a glass rod (the rod soon becomes coated with fibrin, while the blood loses coagulability).

Such a relatively opaque, fresh fibrin-and-platelet deposit usually "disappears" (liquifies and drains onward to the chest bottle) within hours after bleeding subsides: Thereafter, one observes only "old" and usually darker fully-liquid defibrinated blood (with occasional shrunken, irregular, dark clots) passing easily through clean-walled chest tubing.

The rapid disappearance of this fibrin layer from within the clear plastic chest tubing soon after bleeding stops is due to the minimal thickness and large surface area of this layer, which makes it very susceptible to passing fibrinolysins. On the other hand, if the chest tube remains entirely clotted for any length of time, it may never reopen, even with vigorous milking and stripping of the more distal chest

tubing.

There are times when premature chest tube blockage can be a significant setback for the patient: Perhaps irrigation of an important but clotted chest tube with diluted streptokinase (or some other plasminogen activator) will become an acceptable way of correcting this problem or returning an important but clotted, intravenous needle or catheter to service (but I have not tried this).

Full Lung Expansion Brings Free Pleural Fluid Out Through the Chest Tube

When a chest tube has been placed for spontaneous pneumothorax, cessation of air leak is commonly associated with appearance of small amounts of serosanguinous drainage in the previously clear tubing and bottle. This pink drainage indicates that reexpansion of the underlying lung is complete: It occurs because those last few cc's of pleural fluid can only become widely dispersed by lung movement and "capillary attraction" after lung has completely filled the remaining pleural space.

Until that time, small amounts of fluid settle between nearby pleural surfaces where they form a "meniscus" below the air space. A chest tube can usually be removed within 24 hours after an air leak gradually ceases and such "final" serosanguinous drainage is noted. (As used here, the term "full expansion" does not necessarily mean "elimination of all atelectasis," but simply that the lung now fills the entire pleural cavity.)

Pleural Fluid Draining Around a Chest Tube May Also Signify Full Lung Expansion

Sometimes serous or serosanguinous pleural fluid leaks from the pleural cavity along the outside of a chest tube, usually after the tube has been present for several days. This simply means that the tube tip and side holes (which are located high within the pleural cavity) no longer provide the easiest escape route for a low-lying fluid pocket. Such fluid, which may not be evident on roentgenogram, is then forced out alongside the chest tube by expiratory pressures and gravity, as expanding lung obliterates the remaining pleural space.

When such drainage around a tube is associated with cessation of chest tube drainage and tidal, this usually indicates that the chest tube is "no longer useful" and can be removed. In other words, the tube has either clotted-off, or the tube holes have become sealed between adherent lung and chest wall (and thereby excluded from draining any residual pleural space).

At this point, chest tube removal may release additional volumes of previously blocked intrapleural fluid (perhaps a diaphragm sulcus seroma) into the occlusive tube site dressing. In some cases, a sudden, impressive evacuation of very bloody fluid may occur from the residual skin hole, hours or even days after chest tube removal: Often this is consequent to a vigorous cough, or strenuous Valsalva effort when constipated. If unexpected, such bloody drainage can be most unsettling to the patient, nurse and internist (see later discussion of tube-site drainage, this chapter).

Timing of Chest Tube Removal

Chest tubes are removed when they become non-functional or unnecessary (i.e., firmly clotted or no longer open to a pleural space). Satisfactory pleural space obliteration is most likely to be seen when tube drainage and tidal both diminish gradually, as the fluid level rises well up into the water seal straw.

A rubber chest tube will usually clot, or at most show a 1-2 cm tidal, when it is ready for removal. A plastic chest tube can generally be removed once the straw fluid level remains well above the water seal surface for one or two days (documenting "no more air leak") and chest roentgenogram shows satisfactory lung expansion.

Plastic tubes provoke less pleural reaction than rubber, and are less likely to become embedded in or occluded by fibrin. An open plastic chest tube may sometimes continue to show minor to moderate "cough tidal" for many days after cessation of air leak (despite "full expansion" of lung on chest roentgenogram, so it is impractical to leave a plastic tube in place until there is no more tidal. If concerned about removing a chest tube in the presence of such residual tidal, simply increase the height of the "hanging fluid column" and most of that tidal should vanish).

Remove Chest Tube Only After Air Leak Ceases

A chest tube can usually be removed two to six days after thoracotomy, but never while air leak persists. In fact, it is often well to delay tube removal for another twenty-four hours after cessation of bubbling. During this time, tidal excursion with cough or deep breath will generally diminish to less than 2cm, and the straw fluid level usually remains more than 10 cm above the water seal surface (the straw or tubing fluid level can also be "lifted" much higher, of course).

When there is no intrapleural air pocket in contact with an open chest tube and tubing, the water seal does not reflect pressure changes within the hemithorax: When milked, vented or drawn higher, the elevated, stable straw fluid level that remains, simply suggests that subatmospheric intrapleural pressure is present as usual without specifying how much. Tube removal under these circumstances depends upon past, present and anticipated fluid output.

As long as pleural surfaces adhere about, and thus seal, the chest tube holes, intrapleural pressure changes cannot be transmitted to the water seal straw. Likewise, copious fluid drainage and a tall fluid column in straw and tubing can overwhelm the manometer function of the water seal straw.

One should avoid chest tube removal soon after sudden cessation of air leak, or in the presence of significant atelectasis, especially if the atelectasis involves a previously leaky pulmonary lobe: An endobronchial mucus plug can cause atelectasis and stop air leak for one or more days; such a leak may then suddenly resume when the collapsed lung is again aerated. Low grade fever, dyspnea and wide tidal usually accompany early postoperative atelectasis, if the chest tube remains in proper position (see also Case 3, Chapter 7.)

Remove the Chest Tube Only After Fluid Output Decreases

Chest tube removal from an adult is usually delayed until daily fluid output by chest tube is 200cc or less (some serous drainage may persist as long as the chest tube remains in place). Particularly heavy and prolonged chest tube drainage is expected after thoracoabdominal incision for major abdominal trauma (e.g. with right hepatic lobectomy).

In this circumstance, removal of the chest tube before two weeks have passed is often unwise, even though any air leak may long since have ceased. "Grossly bloody" drainage may sometimes be another reason to delay chest tube removal, for ongoing clot lysis can later result in significant fluid accumulations.

Technique for Chest Tube Removal

Chest tubes (along with their closely adherent tube-site dressings) are best removed smoothly and rapidly, preferably while the patient performs a brief mild Valsalva maneuver (after the tape has been "unstuck" from the chest wall and the skin suture cut). A previously prepared Vaseline gauze dressing (faced by a glob of antibiotic or Betadine ointment and backed by 4 x 4 gauze) is immediately pressed onto the post-tube-removal "skin hole" and held firmly in place until secured by overlapping strips of wide tape.

The fatter the patient, the more rapidly a just emptied transthoracic tube tract becomes "air-tight" – the occasional very thin individuals may need a post-removal tube site dressing in place for a third day to guard against air entry. Usually the tube site dressing is routinely removed about forty-eight hours after the chest tube was withdrawn. Thereafter the small wound remains uncovered and is washed by the patient during his daily shower.

Occasional applications of antibiotic or antiseptic ointment can help "clean up" local inflammation. However, if a moist punctate "diaper" rash develops close by the tube site, this may indicate allergy to Neosporin ointment rather than a new tube site skin infection (so Neosporin is discontinued and the tube site heals rapidly anyhow). Polysporin ointment rarely causes skin allergy.

Exposed and even gaping tube-site skin wounds heal quickly without infection, leaving a minimal scar. On the other hand, immediate closure (by stitch or skin clip) of the small contaminated skin wound after chest tube removal - or any bandage that thereafter keeps that site moist and macerated for over two or three days - markedly increases the likelihood of a local infection that could even delay discharge from the hospital.

Regarding the tube tunnel or path: When closed thoracostomy is performed under local anesthesia, the chest tube inserted through a small skin incision then passes across the upper border of an underlying rib to directly enter the pleural space. On the other hand, chest tube placement during thoracotomy offers more options and positional control as the tube can easily be guided via a "longer" path (spanning one interspace and rib) from pleura to skin. Such a "tunnel" not only helps control tube position – it also reduces risk of air entry into the tube-site wound after tube removal.

A tunneled tube path tends to allow late evacuation of residual pleural fluid without permitting air to enter because the chest wall tunnel can fill with pleural drainage during expiration, yet it collapses down against ribs on inspiration, blocking air return. While removal of any chest tube that has been in place for many days may leave a persistently open channel for at least a few hours (until edematous, indurated tissues gradually slump back together), the more direct tube pathway through the chest wall is more likely to allow air entry and thus cause pneumothorax.

When a chest tube on water seal has many side-holes, it is especially important to remove that tube with one swift motion since open side-holes already "outside" immediately admit atmospheric air to the remaining, intrapleural portion of an unclotted chest tube: Thus the act of chest tube removal remains a common cause of minor pneumothorax. But a momentary gentle Valsalva maneuver by the patient during chest tube removal can help prevent such air entry (and it may express residual pleural fluid as well).

Of course, even a vigorous Valsalva effort (which could readily cause a somewhat hypovolemic

postoperative patient to faint) may not prevent atmospheric entry through a briefly open chest tube that recently was on high suction in order to expand a small underlying lung. Here it is especially important to remove the just-clamped tube swiftly and block its subcutaneous tube tunnel immediately thereafter (by gentle finger pressure just above the skin hole) until the skin opening can be buried in ointment under an "airtight" tube site dressing. Although a simple pneumothorax that results from chest tube removal usually resolves without problems, it is better prevented, as any persistent pleural space increases the risk of empyema.

Continued Fluid Drainage from the Tube Site After Tube Removal

Not uncommonly, several hundred cc of straw-colored to dark bloody fluid will pour from a chest tube skin-site shortly after tube removal, especially with a cough or during straining at stool: This may occur even when no pleural fluid was evident on the most recent chest roentgenogram. Fortunately, such evacuation of pleural fluid remains "a good thing" even after chest tube removal, for full lung expansion requires elimination of all pleural fluid pockets.

Experience assures us that neither pneumothorax nor empyema will result from this occasional "voiding" of pleural fluid through the tunnelled tube site wound. The nurse should be instructed not to lift away the saturated dressing early after tube removal, however, for this sloppy bandage allows the outward flow of fluid while preventing air reentry. It is better practice to reinforce the soggy dressing, and simply change pajamas and bedding as necessary, until the wet dressing can safely be removed (forty-eight to seventy-two hours after tube removal).

It is extremely uncommon for air to enter any "tunneled" chest tube wound site, however, and poststernotomy tubes are not left in place for prolonged periods anyhow (so they do not leave an indurated tube path that tends to stay open). Thus post cardiotomy tube-site dressings are often changed routinely by the intensive care nurse within hours of tube removal.

Summary: Only with a small or surgically-diminished lung that is "under a lot of stretch" to achieve full expansion, or a "chronic" chest tube tract (temporarily held open by edema and inflammation), or a really thin patient, or following closed thoracostomy, is air reflux a real possibility (and these are not "run-of-the-mill" patients in a modern postoperative intensive care unit).

On the other hand, while soggy tube-site dressings may sometimes be distasteful, they certainly present no hazard if left in place for two or three days: Particularly in a "combined cardiac-and-pulmo-nary-surgery" intensive care unit, therefore, one might err on the side of caution and avoid the routine early removal of postoperative chest tube-site dressings (in order to prevent an occasional pneumothorax after lung surgery).

Reminder: A residual bloody postoperative (or post-traumatic) pleural effusion is usually darker and more dilute in appearance than fresh blood; nevertheless, its first gush from the chest tube site can be disconcerting. Even forewarned patients may interpret such sudden tube-site drainage as catastrophic blood loss or disintegration of their operative incision, so repeated patient teaching is helpful here.

Conclusion: Bloody drainage that persists or occurs more than 48 hours after tube removal seems to present no significant risk of pneumothorax (by air reentry through the still-patent tube site, with or without an overlying bandage) or empyema. After 48-72 hours, a continuously-wet dressing is

undesirable, so we (who "don't do beds") accept episodic drainage from an open tube site into readily changed sheets and bed clothes: Antiseptic ointments can be applied to the open tube site wound whether it is currently draining pleural fluid or not.

A tunneled, low lateral, chest tube insertion wound that enters the chest at the diaphragm sulcus will have far less tendency to suck air (especially in a sitting or ambulatory patient) than a high, anterior, intercostal tube site, for the same reason that a high anterior chest tube is more likely to remove air, and a lower tube to remove fluid. Thus, an increasing pleural effusion, combined with cough, can readily be forced out of a low tube site wound, but diaphragm and lung will tend to occlude a tube hole just above the diaphragm when there is no fluid present to separate the pleurae.

Skin Edge Bleeding

It is, of course, also possible to have fresh blood loss from the tube site wound, before or soon after removal of a chest tube (especially if the patient has been taking aspirin). Unlike the more common, en-tirely fluid tube-site "dump" of a residual pleural effusion, this fresh bloody drainage is always identified by a build-up of jelly-like clot under the dressing, and readily corrected by a skin suture.

I have never encountered active bleeding from the tube tract deeper within the chest wall except following coarctation repair, so one can place a "full-skin-thickness" skin suture with minimal concern about hiding active "deeper" bleeding (if persistent, such hypothetical "internal bleeding" would undoubtedly be signaled by an increasing pleural effusion or chest wall hematoma, along with the usual signs and symptoms of blood loss).

Suture-Closure of Chest Tube Holes is Not Advisable

It is common practice in some training programs to lay, but not tie, a tube entry site skin suture at surgery: That suture is then immediately tied after chest tube removal, to close the chest tube hole. In view of the often-important drainage function of the tube site wound after tube removal, and the proven safe and rapid healing of such skin wounds when left open (in many thousands of patients in Anchorage, Iowa City, Milwaukee, Boston and undoubtedly elsewhere), immediate tube-hole closure seems undesirable at least, and possibly even dangerous.

For in addition to the increased risk of local abscess or inflammation caused by suture closure of a contaminated, possibly infected, tube site wound, residual pleural fluid will then remain entrapped in the pleural space: At the very least, this must increase the need for postoperative thoracentesis - and nutrient rich pleural fluid extending along the open tube tunnel to the bacteria-infested skin opening ought also increases the possibility of postoperative empyema.

Normal pleura certainly can absorb a great deal of sterile fluid. And since "no pleural space" means "no pleural fluid" and "no pleural fluid means no empyema" what's not to like about letting pleural fluid escape freely and safely from an open tube tunnel. It has been decades since I performed postoperative thoracentesis for a persistent pleural effusion. And postoperative empyemas are rarely seen except following pneumonectomy, and breathing exercises really do speed up pleural fluid absorption...).

Well anyhow, an unsutured chest tube skin wound certainly closes rapidly and leaves minimal scar (as long as no soggy, germ-laden dressing covers the tube site for more than 48 to 72 hours after tube

removal, for that can easily delay healing). Incidentally, the "cross-hatch" scars caused by a snug suture tied across of the chest tube skin hole can sometimes be more noticeable than the actual chest tube skin incision that was made along skin cleavage lines.

Summary: Suture closure of the chest tube "skin hole" after chest tube removal is an unnecessary intervention with potential for harm.

Special Case: Tube Removal After Pneumonectomy

Following pneumonectomy, the small chest tube is usually removed from its extra long chest wall tunnel about twenty-four hours after surgery: One especially wishes to avoid any unsterile fistulous tract between pleura and skin (and, as discussed, the longer a chest tube remains in place, the more likely that a temporarily open tract will remain through inflamed and edematous "soft" tissues).

No such problem exists if the postpneumonectomy chest tube is removed within a day or two, provided that tube and hole were not excessively large and that the pleural entry site of the tube lies within the apical air pocket rather than being submerged under accumulating fluids. If the intrapleural end of a chest tube tunnel becomes submerged too early, however, the fluid weight - supplemented by any cough or Valsalva effort - may balloon a not-yet-healed tube tract and encourage continuing pleural fluid drainage.

With no underlying lung available to fill the remaining pleural space, such overflow drainage via the tube tract simply establishes a fluid path ("bug ladder") through the chest wall that increases the likelihood of empyema. Any significant escape of fluid from the postpneumonectomy space could also lead to excessive "siphon shift" of the mediastinum.

A Skin Clip Can Close the Chest Tube Hole After Pneumonectomy and in Young Children

Tube removal from a pneumonectomy patient, or a child younger than ten or twelve years (whose dressings never seem to stay on), can be supplemented by a metal skin clip for approximation of the skin edges as soon as the tube has been removed. In this case, the chest tube is pulled out with one hand (after loosening the tape and cutting the skin stitch) while skin and subcutaneous tissue are bunched about the tube hole with the clean thumb and forefinger of the other hand, until the first hand can apply a previously-prepared, sterile skin clip to close the hole.

The skin clip is then submerged under a small mound of antibiotic or antiseptic ointment, and covered by a light dressing for two more days. The hole is usually neatly healed when the clip is then removed. After infant lung surgery, a chest tube can often be removed within twelve to twenty-four hours, and the skin clip twenty-four hours later (due to minimal air and fluid drainage and greater rapidity of healing). These skin wounds heal rapidly because they are almost sterile; however, the prevalence of "almost sterile" tube sites becomes "vanishingly small" after several days with a chest tube in place.

Removal of A Tunneled Tube

Expedience and local anesthesia combine to keep a closed thoracostomy tube pathway as short as possible (except for minor obliquity to improve the intrapleural "lie" of the tube). At thoracotomy, however, one can safely and comfortably lengthen the "subcutaneous" (extrathoracic) portion of the

tube tract by several centimeters, before "poking in" through the intercostal space. This longer, "S"-shaped path, commonly referred to as a "tunneled" insertion, can theoretically increase the risk of kinking or displacement of the chest tube or cause unnecessary discomfort.

Kinking only occurs with sharp angulation, however, or on bending a stiff but thin-walled tube such as the usual red rubber "rectal" catheter: Similarly, tube displacement and increased discomfort are not a problem if modern, clear plastic or silastic chest catheters are utilized.

Undoubtedly, very thick-walled tubing would cause more pressure and discomfort at each bend, while an undesirably small tube (with two bends already in it) might more easily become doubled-up and displaced by muscle shear within the chest wall. These problems simply do not arise if the commonly-marketed #28 or #32 French chest tubes are used for postoperative chest drainage in an adult.

The principal advantage of tunneled chest tube insertion (besides positional control and the reduced risk of air entry) is that it allows ongoing chest drainage after tube removal when that is appropriate, yet makes it easier to seal the tube tract promptly if that becomes important. After pneumonectomy, it also increases the physical separation of skin bacteria from the pleural entry site.

An extended subcutaneous tunnel is readily blocked during tube removal by gentle finger pressure away from the actual wound. This closure of the tract itself can then be maintained by an appropriately lumpy dressing that covers both tube tract and skin wound. As previously mentioned, I routinely tunnel all operatively placed chest tubes, penetrating the pleura at least one intercostal space cephalad to the skin incision.

Mediastinal Tube Removal

Removal of mediastinal (substernal) tubes is generally delayed until drainage is quite serous and very low-volume, to avoid further osmotic fluid accumulation within the mediastinum during breakdown of residual blood clot (especially if pleurae are intact). Commonly there is little inspiratory pressure drop transmitted into the edematous, postoperative, anterior mediastinum, so unless the patient is very thin, the tube-site dressing can be changed at any time after removal of the substernal tube from its necessarily long tunnel, without fear of significant air entry: Rectus fascial planes traversed by the substernal tube usually prevent significant drainage via the empty tube tract anyhow (see Chapter 10 and "finger mediastinotomy," Chapter 15).

Accidental Chest Tube Removal

If an intrapleural chest tube is accidentally "jerked out" or prematurely removed some days after its insertion, it is usually possible to slip an identical, or next-size-smaller, sterile tube back into the previous tube tract (when tube drainage is still required and one wishes to avoid thoracentesis or a "formal" closed thoracostomy at another site). If the replacement tube does not go in readily, bevel its tip, then try again while pressing the tip gently inward through overlying skin at the point where the tube tract dives between ribs.

One should probably soak the skin opening and tube tip with Betadine during such manipulations, and avoid many hours delay, as tissue edema holds these tracts open for only a short time. Such gentle reinsertion, usually without infiltration of local anesthetic, is generally associated with minimal patient

discomfort; it is also very unlikely to cause infection. In fact, chest tube reinsertion should help to prevent intrapleural infection if residual fluid or air still requires drainage.

One would, of course, avoid such "semi-sterile" chest tube reinsertion after pneumonectomy, where the "permanent" pleural fluid collection maximizes the opportunity for empyema. In this situation, one or more careful thoracenteses will adequately substitute for a chest tube (during the first twenty-four to forty-eight hour, postoperative period when air removal for adjustment of intrapleural pressure may still be indicated).

I would not reinsert an accidentally removed substernal tube blindly in this fashion, as one could easily jam such a tube into an undesirable or hazardous location. If tube replacement soon after sternotomy is deemed essential to prevent tamponade or infection, it is usually safest to at least reopen the upper abdominal fascia, if not the entire sternotomy incision, in order to visually ascertain satisfactory replacement of an essential drainage tube in the retrosternal space between intact pleurae (an adequate opening between a pleural cavity and the retrosternal space would make this unnecessary - see Chapter 10).

Chest Tube Placed into Liver or Spleen

A "low" closed thoracostomy may endanger liver or spleen. As an intern, I once observed placement of a chest tube directly through the spleen; emergency splenectomy was required to stop the steady drainage of fresh blood.

I have had several patients referred with a low right-sided chest tube planted firmly in the liver. In fact, I recently placed one there myself, when the patient blew out a lot of air (forced expiration leading to increased doming of the diaphragm during the necessarily low chest tube insertion, even though needle aspiration had indicated that this was the proper level). Fortunately, this very ill old man (with loculated basal tension pneumothorax) did well after blood transfusion and an "extremely" careful, repeat closed thoracostomy. (Surprisingly, patients generally continue to appreciate a physician who inadvertently does harm, as long as he seems truthful, otherwise competent and caring.)

Case Report: Removal of Chest Tube from the Liver

A middle-aged male was referred from a distant rural area with a low-lying right-sided chest tube in place. The initial problem had been chest injury with minor hemopneumothorax: Referral was for follow-up care of persistent hemothorax. The chest tube was reported to have drained blood initially, but the hemothorax persisted after this bloody chest tube drainage ceased.

The patient was taken to the x-ray department and an undesirably low tube location (in liver below diaphragm) was confirmed at fluoroscopy. Based upon previous experience, it was reasonable to expect one-pint-or-less blood loss (and no other residual problem) from removal of this off-target tube.

With patient cross-matched, precautionary large-bore I.V. in place, and operating room personnel aware of this problem, I pulled the tube out, holding a kidney basin against his side while the patient lay very still for a few minutes until bleeding ceased, to avoid muscle layers prematurely closing the tube hole. The patient and his hematocrit remained stable. He was soon allowed "up and about," returning home after his persistent hemothorax was evacuated by needle (and inguinal herniorrhaphy by another surgeon).

Case Report: Removal of Chest Tube From Lung Parenchyma

An elderly, emphysematous alcoholic was brought to a rural clinic with a bullet injury to his lower left chest. After a chest roentgenogram was taken, an emergency closed thoracostomy was performed. The patient was referred for follow-up care two days later, with roentgenogram in hand.

At the time of admission, the patient was in no distress. Initial examination was not remarkable, except for moderate subcutaneous emphysema near the chest tube. The patient's main concern was his persistent minor hemoptysis "ever since the shooting." The original chest film showed an abnormal lower lung density (hematoma) associated with the bullet, but no pneumothorax or subcutaneous emphysema.

When PA and lateral chest roentgenograms were now repeated, they revealed the chest tube skewering his upper lobe, with the tube tip lying in the superior segment of his left lower lobe. On further questioning, the patient recalled an episode of left-sided pneumonia with pleurisy, some fifteen years earlier.

Discussion: This patient survived an unfriendly bullet through his left lower lobe as well as a "friendly" chest tube spearing the upper: Either or both contributed to his hemoptysis. Not having been present at the shooting, I cannot say whether that was justified, but since the x-ray revealed no evidence of free intrapleural air or fluid, the chest tube insertion clearly was unwarranted.

Presumably, pleural obliteration by prior inflammation prevented this bullet injury to emphysematous lung from causing pneumothorax or hemopneumothorax. Perhaps the original lung perforation in this patient was rapidly tamponaded by blood from a closely adherent, higher pressure, intercostal artery (but in any case, "no air build-up" at the site of injury clearly signified "no air leak").

Undoubtedly, however, if positive-pressure assisted ventilation had become necessary, it would have resulted in severe generalized subcutaneous emphysema. Fibrous pleural obliteration also held the lung firmly in place as the chest tube passed through, instead of the tube simply pushing lung aside or, at worst, gouging it superficially if tube inserted was on a sharp stylet, for example).

It seems unlikely that a bullet would pass through chest wall and lung without causing any air leak or blood loss while penetrating a free pleural cavity; certainly hemopneumothorax is the more usual result. In any case, the absence of free air or blood within a pleural space is never an indication for closed thoracostomy. A really prophylactic chest tube is rarely indicated anyhow, except possibly prior to initiating positive-pressure ventilatory assistance after lung injury: Even then, one should first identify free intrapleural air or fluid on chest roentgenogram (as discussed in Chapter 5).

That is all very well, but how and when is an obviously intrapulmonary chest tube to be removed? The patient currently has a minor air leak through this chest tube, as well as hemoptysis and a presumably obliterated pleural space: Certainly reinsertion of a more appropriately-positioned (intrapleural) tube is not an option.

Although removal of a chest tube while it is still draining pulmonary air leak goes against our teaching, there is little reason for air to stop entering a chest tube that is skewering lung. Possibly the tube could be removed gradually, but this patient has no infected pocket that must scar down about the tube tract prior to tube removal (and meanwhile, there is always a worry that the tube could erode a

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pulmonary blood vessel).

Thus the tube was carefully withdrawn, and the tube site dressed with antibiotic ointment on Vaseline gauze for forty-eight hours. His lung remained fully expanded, so the patient was discharged two days later (when the likelihood of delirium tremens had also diminished).

Residual Pneumothorax Following Chest Tube Removal

Not infrequently, a chest roentgenogram taken after chest tube removal reveals a minor apical pneumothorax. What should one do?

In general, nothing, except to encourage one extra-deep breath every five minutes while awake (emphasizing maximum chest wall movement on the affected side) to hasten resorption of residual air and fluid.

A common cause of residual apical air is a chest tube that was placed too far below the apex and then became prematurely occluded as expanding lung rose past the tube tip. Air can also be introduced accidentally at the time of tube removal, especially if the patient draws a deep breath when he should be performing a gentle Valsalva, or if the chest tube is pulled out too slowly.

In either of the latter circumstances, an intermediate event (side holes outside, tube tip inside) creates a momentary open hole into the chest, allowing air entry as lung shrinks toward its "relaxed" size. On rare occasion, intermittent positive-pressure breathing therapy may also reopen an air leak, causing delayed pneumothorax after chest tube removal.

In most cases of residual minor pneumothorax, there is little likelihood of continued air leak or fear of infection, so resorption and full expansion can confidently be predicted. Nevertheless, I keep such patients under surveillance with an occasional chest roentgenogram (and have them report any fever) until complete lung expansion is finally achieved (see Chapter 5).

Residual Air/Fluid Level Following Chest Tube Removal

I do the same, but worry more, when there is increased potential for infection in association with a residual air pocket. This situation should not arise during therapy of frank empyema, for an empyema tube is then left in place until any pleural space closes down about it (or permanent open drainage is performed). The "problem patients" are those with inaccessible air-fluid levels high in the apex, and a low-grade or spiking fever.

Closed thoracostomy into the apex is technically difficult, posing obvious risk to surrounding structures. I usually temporize, therefore, adding a vigorous ten-day or two week course of hopefully appropriate antibiotics to the previously described, deep-breathing exercise regimen. An intelligent and cooperative patient (who will not be lost to follow-up) need not remain in hospital during this entire time.

Within a week or two, the clinical condition of such a patient either improves or deteriorates: If he becomes febrile, it may be due to the roentgenographically visible air-fluid pocket (or possibly to some other problem). If such a "probably infected" apical air-fluid pocket is the cause of increasing toxicity, it generally enlarges to a size and location that will permit safe tube thoracostomy (or at least needle

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and catheter placement under x-ray guidance). Often, however, post-operative apical air-fluid collections drain per bronchus spontaneously, resolving this therapeutic dilemma before closed thoracostomy can safely be attempted.

Sample Nursing Instructions for Chest Tube Care

1.With air leak bubbling from the water seal straw, any resistance to air escape from the pleural space will increase pneumothorax, so A) coil or place excess tubing flat on the bed, B) lead the remaining tubing directly down to the water seal bottle without allowing intervening, fluid-filled, dependent loops of tubing, and C) decant excess fluid from the water seal bottle when necessary to prevent or correct deep submersion of the water seal straw tip (if sure that you know how to do this).

2.When there is no air leak, air can be withdrawn or added at the chest tube connector needle port in sterile fashion to maintain a (hanging) fluid column of desired height in the straw and tubing; this column applies suction to assist fluid drainage. If a water seal fluid column subsides soon after it has been lifted in this fashion, it indicates minor air leak (or perhaps that tubing air has been displaced distally by a proximal tubing segment full of fluid or clot, or that the bottle has been tipped or sloshed, allowing air to enter the water seal straw tip).

3.Check the chest tubing every time you see the patient. Always encourage fluid within the chest tubing to move toward the water seal bottle by lifting the tubing nearer to the patient. Squeeze the tubing frequently to loosen thick or clotted drainage, so fluid will move on down to the bottle.

Sample Instructions for Chest Tube Removal

1. Chest tube removal leaves a hole through which pleural fluid may escape (which is desirable).

2. Reinforce the tube site dressing p.r.n., but avoid removal of this dressing until forty-eight hours after the tube was removed, to minimize the risk of air entry.

3. After forty-eight hours, a dressing is no longer placed upon the tube site; the skin wound can then be washed and antibacterial ointment applied, if indicated.

4. A sterile skin clip and clip applicator should be available during chest tube removal from children and pneumonectomy patients.

CHAPTER 12: POSTOPERATIVE OR POSTINJURY CARE

Introduction;... On the unpredictable effects of aspirin and other therapy;... On the nurse, or off the medication;... Also observe the patient;... Healthy patients may have sick electronics;... Anything attached to the patient may malfunction;... Roentgenograms and auscultations supplement each other;... Lighten the patient's load;... Postoperative pain;... Weakness and shivering;... Ambulation, muscle spasm and protective rigidity;... Venous stasis and phlebitis;... Provide real food and drink;... Hallucinations and nightmares;... Gas and nausea;... Encourage deep breathing and arm movements;... Early removal of dressings and sutures;... Tub soak of a recent surgical incision may be unwise;... Delayed cellulitis of the post-venectomy leg;... Treatment of chest wall injuries;... Temporary measures;... Operative fixation;... Flail chest often leads to retention of secretions;... Nasotracheal suction: Indications and technique;... Tracheobronchial toilet;... Tracheostomy and endotracheal tubes;... Overview of respiratory care;... When respirator function is in doubt, ventilate by hand;... Pneumothorax following tracheotomy;... Case report: Tracheostomy can correct pulmonary edema due to severe laryngeal obstruction; ... Whenever possible, place an endotracheal tube prior to tracheotomy; ... Comments on tracheotomy technique;... Tracheostomy care;... Chest tubes are often helpful in "chest injury" care;... Positive-pressure ventilation and tension pneumothorax;... Avoid premature tube removal;... Cigarettes;... Bronchodilators and expectorants;... Wheezing as a sign of impending pulmonary edema;... Retrosternal noises in the night;... Occasional problems with nasogastric tube drainage;... On Foley catheter removal with the balloon inflated;... Resuscitative efforts;... Case report: Artificial respiration;... Mouth-to-mouth resuscitation;... Case report: External cardiac massage;... Miscellaneous aspects of cardiopulmonary resuscitation;... Case report: Massive pulmonary embolus;... Irreversible Sudden Dilation of Heart;… Open cardiac massage still has a place in resuscitation;... Case report: No electrical activity.

Introduction

Recovery from major surgery is complicated by pain, weakness and disorders of respiration, circulation and other visceral functions. Evaluation of postoperative progress is based in part upon laboratory study of blood samples: Lab results reported may be correct, useful, incorrect, irrelevant, significant, non-representative, out-of-date or any possible combination of the above.

Most patients will resolve minor biochemical abnormalities spontaneously, given a reasonable diet and supportive intravenous electrolyte solutions (without necessity for corrective hypertonic infusions, for example). Unexpected or inconsistent clinical findings or laboratory results are always kept in mind, but drastic remedy is avoided until clearly required. Thus one must often resist the natural urge to correct any detectable biochemical "abnormality," remembering that misguided attempts to fully remedy one postoperative "problem" may seriously aggravate another.

On The Unpredictable Effects of Aspirin and Other Therapy

A treatment becomes increasingly accepted as results become more predictable; even aspirin, however, is still an unknown. Whether you prescribe it as aspirin, acetylsalicylic acid or the acetic acid ester of orthohydroxybenzoic acid, you really have no way of knowing what desirable or unexpected biochemical, physiologic, idiosyncratic or antagonistic effect may ensue, assuming that (1) you correctly ordered, (2) the nurse correctly delivered and (3) the patient successfully ingested and retained this small white tablet in a timely fashion (none of the above being "sure things" by any means).

On The Nurse, or Off The Medication

Although patients differ and treatments inevitably vary as well, a patient with favorable prognosis will usually recover health and happiness after competently performed surgery, provided the surgeon remains alert, available, interested and considerate (which may not be so easy). For example, it is often impossible to find out what is troubling the patient except from his nurse; many otherwise intelligent patients will be very open with their nurse, yet reluctant to complain to the surgeon who "just saved their life." On the other hand, some nurses misunderstand or even manufacture evidence (perhaps to demonstrate dedication or overcome boredom at 4 a.m.), so "know your nurse.

At their best, these wonderful, hardworking, caring, underpaid, intelligent and perceptive people can make you look like the world's greatest surgeon (then be appreciative and generous). The reverse also holds true (for example, when Rollins, "the G.I. bleeder" receives the heparin drawn up for Collins, "the phlebitis"), so make it a habit to read labels and recheck medication sheets occasionally. One is sometimes amazed at antibiotics that are still being given (or not given when ordered), or embarassed by the difficulty encountered in anticoagulating a postoperative heart-valve patient who inadvertently received prophylactic Vitamin K (in a routine "inherited" postoperative multivitamin preparation).

Also Observe The Patient

Most important, however, is a frequent look at the patient, especially soon after surgery. First of all, does he look "sick"? Dehydrated? Pale? Stressed? Circumoral pallor? Deeper or larger circles under his eyes? Breathing easily? Nostrils flaring? Rapid shallow breaths? Position and movement of eyes and extremities? How about the electronic monitor readout and waveforms?

Healthy Patients May Have Sick Electronics

Always check the arterial pulse wave on the monitor screen rather than rely solely upon a digital readout, especially when the ECG "looks funny" or numbers are "out of line." Blood-pressure cuff readings are less likely to be wildly inaccurate than a digital arterial-line readout. No stethoscope is required if confirmatory cuff pressures are taken on the arm bearing the radial artery cannula. Simply inflate the cuff until the arterial wave form disappears, then deflate slowly.

Systolic pressure is that cuff pressure reading at which any pulse wave reappears on the monitor screen; diastolic is the number on the aneroid dial when the entire arterial wave form (peaks and valleys) has just become visible. The sounds that are heard when taking a blood pressure, between systolic and diastolic values, are caused by intermittent blood flow: The "thump" is not heard above the systolic value (no flow) or below the diastolic pressure (continuous flow), except that it may sometimes "carry" through stiff vessels to some extent at any pressure. In any case, a well-calibrated aneroid or a mercury sphygmomanometer will often reveal satisfactory arterial pressure even though the malfunctioning digital readout has just declared "Disaster!"

Of course, one completely ignores the numbers on the digital readout for arterial pressure as long as a blood pressure cuff remains inflated proximally, for numbers displayed during such cuff inflation will only (inaccurately) reflect central arterial pressure minus cuff pressure: In other words, inflation of a cuff reduces the intraarterial pressure distal to it. (This sort of thing may seem obvious as you read it, but it can cause confusion when there is "a lot going on.")

It is alleged that cuff pressures may become spuriously elevated above the "real" intraarterial value in a patient with "stiff" vessels, for it then takes a greater cuff pressure to occlude flow. A patient likely

to have such "psuedohypertension" (or the comparable hypotensive patient who still reads "normal" by cuff) is also likely to have a palpable artery distal to a blood pressure cuff that has been inflated well above systolic pressure (a "really stiff" vessel wall does not require intravascular pressure to become palpable).

Anything Attached To The Patient May Malfunction

Further visual inspection of the patient should include drip rate of all intravenous solutions as well as their appearance and labels, function of the nasogastric tube - including a quick disconnect to be sure that it gurgles with air entry, thus confirming that useful suction is actually being applied (whenever fluid in the NG tube is tidaling "to and fro," this indicates absence of effective suction), and respirator tubes (perhaps full of water or disconnected?).

You should also lift the clean white covers occasionally, especially if "things don't seem quite right," to see whether venous or arterial lines have accidentally become disconnected and are now bleeding freely (the mattress may also have received the most recently ordered intravenous solutions and medications), or if chest drains are clotted or full of fluid that should be encouraged onward to the chest bottle (try to keep chest tubes over rather than under the covers).

Roentgenograms and Auscultations Supplement Each Other

If practical, sit the patient up and listen to each lung base posteriorly for rales, rhonchi, wheezes and bronchial or absent breath sounds (each lung base is best evaluated during one or more coughs and the associated vigorous breaths). One should correlate auscultation findings with every chest roentgenogram, as the combined "audio" and "visual" findings eventually meld into a useful mental replication of "what is going on in there."

I rarely listen to the heart (or anterior or lateral lungs) following cardiac surgery, except to reassure a nurse or relative concerned about the many noises heard here (including rubs, squeaks, rattles and crunches) that are of little or unknown clinical significance. Details of the sort described above become almost a subconscious part of any brief inspection of the patient during "rounds" or random visits.

Lighten the Patient's Load

The same technology that makes it possible to monitor the ECG of a migrating humming bird in flight (or an underwater whale) should make it possible to monitor a "coronary" patient more reliably and comfortably. There is no reason why the patient could not have a tiny individualized microchip and transmitter (in his ear, perhaps, or comfortably adherent elsewhere) that would warn the nurse's station of his temperature, pulse, cardiogram and perhaps, disposition.

The currently utilized heavy, monitoring "brick" that is hung around the patients neck (to repeatedly thump his sternotomy incision) or installed in his bathrobe pocket (ditto for the groin) also has an unacceptably high failure rate. It seems likely that the Amazons who often thunder into a sleeping patient's room to administer CPR (for what turns out to be another electronic malfunction) could actually have an adverse effect on his chances for survival. Certainly every aspect of patient monitoring is long overdue for "an upgrade" by Silicon Valley.

Postoperative Pain

A surgical or other injury usually "hurts" or "throbs" more-or-less continuously for the first twelve hours, unless one intervenes with analgesia. After this initial period, severe postoperative pain is usually secondary to movement or muscle spasm (or possibly a reflection of anxiety or even drug addiction). Increasingly severe late postoperative pain may represent infection: There are many times when postoperative pain is difficult to evaluate and treat.

In common parlance, someone who is "feeling no pain" may be stuporous or comatose, agitated and wild, unable to control or protect himself, possibly nauseated, incontinent, hypoxic, hypotensive, and so on. A patient who wishes to feel no pain after surgery, must understand that complete analgesia is neither practical nor safe, once he is awake and has been extubated. Although I was trained in the "get them awake and coughing" era, recent improvements in monitoring, anesthetics, nursing and respiratory care have made it increasingly practical to keep many patients safely intubated and pain-free during or through the most acutely uncomfortable, first postoperative night.

A long posterolateral thoracotomy incision or complete sternal resection appear to be among the most painful operations that we thoracic surgeons perform, with a long vertical midline abdominal incision "somewhere close behind." While long incisions "heal from side to side," patients allege that they "hurt from end to end," so I no longer rush these patients off the respirator after surgery.

On the other hand, sternotomy and pneumonectomy usually cause less pain than one might expect. Presumably, the reason that sternotomy hurts less than thoracotomy (unless the patient is rolled far onto his side, temporarily dislocating all recently "sprung" ribs) is that it is followed by a rigid, bone-to-bone closure. Similarly, with pneumonectomy (after the first twelve hours of acute incisional distress) there will be minimal respiratory movement and no pleuritic pain on the operated side (for no visceral pleura remains to irritate the fluid-soothed parietal pleura).

Unusually severe postoperative pain, or severe distress in less stable patients early after surgery, is best treated by incremental doses of intravenously administered narcotic, until it becomes clear how much narcotic is needed and can be tolerated. One should not hesitate to administer a narcotic antagonist to revive the obtunded patient, giving incremental intravenous doses - one fourth cc (1/4 cc) Narcan I.V., every two or three minutes as needed. At times, an immediate larger dose (one cc or more) of Narcan may be justified, but this often delivers a remarkable "jolt" to the previously obtunded patient who is thereby suddenly aroused, confused and in great distress from multiple causes (in other words, the patient may "come up fighting").

Weakness and Shivering

Modern anesthesia can leave the strongest man weak. Whether due to depolarizing muscle relaxant drugs, or muscle petechiae secondary to severe shivering, or temporary muscle atrophy following short term denervation, or disuse atrophy from immobilization, or whatever; the fact remains - after waking from an operation on your leg, your arms may be too weak to pull you into a sitting position.

Patients should know that they may be very weak for several days after surgery, that this is not a "bad sign," and that it may take months to fully regain the strength they had when "going to sleep" for surgery. (My anesthesiologist friends allege that succinyl choline is more likely to have such adverse effect than the newer curare analogues.)

There are many other causes for marked muscle weakness (e.g. in the chronically-ill or alcoholic

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patient, hypophosphatemia has recently received attention as a correctible cause of muscle weakness, especially after long-term maintenance on hyperalimentation fluids deficient in phosphate).

When a patient waking from anesthesia develops severe shivering, a tiny (one or two milligram) dose of thorazine will usually correct this (with close observation of arterial blood pressure all the while, in case additional intravascular fluid volume becomes necessary). Early control of severe post-anesthetic shivering will hopefully prevent the occasional occurrence of generalized postoperative myalgia (total body "shin splints"), or even sutures "torn out" by such involuntary and painful muscle contractions.

Ambulation, Muscle Spasm and Protective Rigidity

Many physical and chemical stimuli cause skeletal muscles to contract. Injured muscle, or muscles of an injured person, often will not relax fully (this is called "spasm" or "protective rigidity"). Muscles tend to shorten with inactivity as well as with pain. Even dead muscle develops contractile rigor prior to autolysis. Only mechanical stretching will lengthen a muscle beyond its "relaxed length." People and animals therefore "stretch" frequently, especially on awakening.

Early ambulation is a most effective way to overcome the protective rigidity and progressive spasm caused by postoperative pain and immobility. Even if such "ambulation" is initially limited to sitting up and dangling feet over the bedside, or later to an expedition from bed to chair, the patient will be much more comfortable on his return to bed (having stretched his muscles).

A patient following sternotomy or an abdominal incision can be helped into the sitting position by a supporting hand behind the upper thoracic vertebrae. Avoid pulling or pushing a thoracotomy patient by his sore shoulder or arm. After posterolateral thoracotomy, small children or infants are best lifted by a hand under their seat, rather than under the arms.

Venous Stasis and Phlebitis

Blood flow through any vein with competent valves is always toward the heart. The numerous veins draining a lower leg, for example, are compressed intermittently (and thus emptied) by contraction of nearby muscles during ordinary movements. If such a lower leg becomes immobile (because the patient is "under anesthesia," sedated on a respirator, paralyzed, in a cast, or "too sore to move"), venous blood "return" will be propelled primarily by distal arterial inflow (which is usually diminished within an immobile extremity).

Since all veins are not created equal (in size, position, patency, valves, or in relation to enveloping fat, muscle or fascia), forward flow through different veins will vary markedly. Under conditions of enforced inactivity, therefore, some veins will contain blood that is relatively stagnant (and thus more likely to clot, especially during the hypercoagulable post-traumatic period).

The question often arises whether steady external compression by elastic hose is likely to improve flow through previously stagnant veins. It seems probable that such compression has similar effect to an increase of tissue pressure from any cause (including simple dependent edema). One would therefore anticipate some decrease in the subcutaneous venous blood reservoir locally, but certain veins will still carry most or all of the diminished flow (regardless of external elastic support), while other veins continue to retain more-or-less stagnant blood.

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In theory then, unless external compression is sufficient to obliterate the lumen of most or all of the stagnant veins, venous stagnation will persist (but in veins of reduced volume). It is not likely that really vigorous external compression can be evenly applied by a tight garment without delivering excessive pressure in some areas (unless a fluid or air-filled garment is used to deliver such pressure uniformly, as in the military "anti-shock" garments).

Elevation or intermittent compression of an immobile lower leg will reduce venous stagnation. The simplest effective intermittent compression is by a firm dorsiflexion of each foot, repeated every 5 minutes while awake. This simple movement has become an important aspect of our early postoperative "exercise program" and its purpose is explained to the patient on admission and once again after surgery.

During the time that the patient remains on a respirator, or still asleep after anesthesia (thus not moving), regular dorsiflexion of each foot is carried out by the nurse (as well as by the physician whenever visiting the bedside). Clinically detectable phlebitis has been very rare since this regimen was instituted (under 1% of 700 cardiopulmonary bypass patients, including the rare acute phlebitis encountered at operation) and detectable pulmonary emboli have been essentially non-existent.

Active or passive dorsiflexion also helps to prevent "heel cord shortening" (calf muscle contracture) in comatose or chronically bed-ridden patients; once developed, this can be very resistant to corrective physiotherapy. A firm vertical "foot board" at the end of the mattress may be useful here, as well as for those patients who need blankets lifted away from their feet.

The too common "metal cradle" placed over the feet to support the bedding, often endangers the lower limb that it is supposed to protect: Although the cradle hoops easily support a sheet or blanket, the patient often injures an already damaged or ischemic lower leg on its supports (while moving about or trying to dangle his feet over the edge of the bed).

It may be prudent to withdraw non-essential female hormone therapy for the duration of hospitalization, for this allegedly increases the risk of venous thrombosis. An occasional patient at high risk for developing postoperative phlebitis (who previously had phlebitis or pulmonary embolus, or perhaps prior or concurrent splenectomy and high platelet count) should probably be managed on postoperative anticoagulants. Except in dealing with varicose veins or persistent lower leg edema, I have usually found elastic stockings unnecessary: Too often elastic hose will be found "bunched" or rolled down as a mild tourniquet, thus more likely to induce venous stasis than improve venous return.

Provide Real Food and Drink

The hospital broth often offered as initial postoperative sustenance, and prominently featured on the "clear liquid diet," can make a strong man gag: Similarly, the "low cholesterol egg." A typical patient recovering from sternotomy, or a thoracotomy not involving his esophagus, is far better allowed to select the least loathsome items from the "regular diet" hospital menu (beginning on the morning after surgery, once he can tolerate water in small amounts): An early return of normal gastrointestinal function (other than emesis) can then be more reliably anticipated. The occasional compulsive eater must be reassured that poor appetite for solid foods may be normal during the early postoperative days. In fact, this could be an ideal opportunity for him to experiment with the oft-dreaded results of not eating when not hungry.

Hallucinations and Nightmares

An agitated, tremulous patient who normally has "several highballs" or "a few beers" every day, is far less likely to develop delirium tremens or nocturnal hallucinations if allowed to continue a limited alcohol intake (unless hepatic function is already compromised, or reduced resistance to infection becomes a major issue). Continuous television pictures can help overwhelm unpleasant nocturnal hallucinations related to discontinuation of narcotics that only become troublesome at low light intensities (the sound can be left "off" if it keeps others awake).

"Sleeping pills" taken while still suffering significant pain, often promote wild hallucinations and nightmares; at such times (especially early after surgery), appropriate analgesia is preferable for achieving comfort and thus some restful sleep. It is also far safer to give narcotic early postoperatively than Valium; narcotic can always be reversed by a narcotic antagonist if it has an undesirable or excessive effect.

Many patients consider it essential that they sleep entirely through every night, especially after major surgery. Admittedly, such nights spent awake can often seem endless, lonely, and associated with a frightening flight of ideas: Nonetheless, it is usually preferable to avoid the heavy sedation or excessive analgesia that such a prolonged stupor would require, in favor of better oxygenation, reduced hallucinations and the physical benefits of an occasional nocturnal "ambulation" (in search of a drink or pleasant conversation).

The postoperative patient who does sleep through the night, often pays in stiffness and pain the next morning. If a sedative seems indicated, Benadryl is preferable to a drug like Dalmane (which has a prolonged action, and often surprisingly adverse cumulative effects on a patient's cognitive functions and personality if used repeatedly).

Gas and Nausea

One should temporarily avoid intake of beer or other effervescent liquid following any antireflux procedure, to prevent painful gaseous distension that might require nasogastric tube reinsertion. Symptomatic acid reflux into the distal esophagus is common after any surgical stress, especially when the patient must lie flat in bed for a longer period, so appropriate antacid is freely provided, and the head of the bed elevated as well (as long as this seems otherwise appropriate): Although patented antacids have become a very common cause of troublesome post-operative diarrhea, Tums still seem useful. Oral simethicone will sometimes reduce patient complaints of gaseous distension.

But to pass flatus successfully, one must not only be able to forgo several breaths, but also to create and sustain a certain amount of abdominal pressure. Dyspneic patients commonly cannot hold their breath long enough and, early after surgery, they are often too uncomfortable to "bear down" appropriately anyhow. At such times, a return-flow enema or simple rectal tube can markedly relieve troublesome intestinal gaseous distension.

Laxatives are freely provided on request, and Benadryl (25-50 mg) or sometimes Valium (5-10 mg) are given as needed for relaxation or sleep. Compazine (5-10 mg IM. every six hours P.R.N.) often relieves postoperative nausea; so may Tagamet, antacid, dry toast and poached egg, or even just explaining that undesirable air swallowing inevitably accompanies every voluntary effort to "burp."

Encourage Deep Breathing and Arm Movements

An especially deep breath on the surgical side every five minutes, using accessory chest and shoulder muscles to overexpand that hemithorax, significantly improves initial chest tube drainage, as well as ongoing fluid and air resorption after chest tube removal (see Chapter 3). Early encouragement of useful arm and shoulder motion will also prevent residual shoulder stiffness, formerly a problem for some elderly patients after thoracotomy or thoracoplasty.

Early Removal of Dressings and Sutures

In the past, it was commonplace to leave dressings in place over "clean" thoracic and abdominal incisions until sutures were removed, possibly a week after surgery. Interestingly, the smelly bandages that often resulted never led to wound infection. More recently, I have come to remove 4-0 nylon skin sutures (or metal clips) early, often even on the first or second postoperative day, replacing each suture loop or clip sequentially as it is removed (by a sterile, pre-packaged, butterfly tape) to prevent wound-edge separation.

Tincture of benzoin is usually applied to the operative site and allowed to dry for a few moments before such "early" suture or clip removal begins. The benzoin helps keep these wound cross-tapes in place for 2 weeks after surgery; then they are readily peeled off. Early dressing and suture removal increases patient comfort and allows regular observation of the wound, as well as earlier showers.

Emphasis: Whenever removing skin sutures or staples "early" in this fashion (meaning in less than a week on chest or abdomen, perhaps slightly longer on the leg), it is well to "tape as you go." An overlapped (poorly approximated) skin closure is especially likely to separate if not supported by a nearby already-in-place Steristrip. Alternatively, subcuticular wound closure (with fine 4-0 or 5-0 Vicryl or other absorbable suture) maintains excellent skin approximation and makes such cross-tapes optional (but expect occasional minor complaints about fine suture material appearing at the wound surface during healing of a subcuticular closure).

To prevent painful blistering by skin shear, Steristrips are not secured with benzoin when there is likelihood of early postoperative swelling of the incision (e.g., at a fresh vein donor site on the lower leg): Steristrips are then placed directly on clean dry skin, and simply replaced if they loosen prematurely. Liquid or spray benzoin may be helpful later, when leg swelling has stabilized or subsided, but avoid getting any benzoin spray on the scrotum, as rapid loss of patient rapport can ensue.

Tub Soak of a Recent Surgical Incision May Be Unwise

One full-leg-length (E. coli) wound infection developed more than two weeks postoperatively, soon after a diabetic patient tub-soaked his vein donor site to help get the Steristrips off. The soaking was unnecessary, for dry Steristrips peel off easily, and it certainly seems undesirable to contaminate a macerated waterlogged recent skin incision with bathtub fecal organisms anyhow. Perhaps one should simply avoid prolonged bathtub soaks of clean, recent surgical incisions under ordinary circumstances.

If you must soak incisions, injuries, burns or whatever, it might also be wise to incorporate a mild antiseptic solution; however, a shower can usually serve for routine postoperative cleanliness. Of course, it is common for a warm shower to diminish venous return in an early postoperative hypovolemic patient, who may then become syncopal. For those early postoperative patients who are

unlikely to tolerate a brief standing or sitting shower with lukewarm water, a bed-bath remains a very reasonable option.

Delayed Cellulitis of the Post-Venectomy Leg

Rarely, a patient who has undergone saphenous vein bypass of his coronary arteries will return many weeks or months after surgery with an erysipelas-type cellulitis involving the vein donor site. This infection is usually sudden in onset and associated with fever, malaise and local discomfort: It generally responds rapidly to anti-streptococcal antibiotics. A common cause of this cellulitis appears to be chronic fungal infection between the toes, and it is recommended that the fungal problem be treated vigorously to reduce the likelihood of future recurrences: The increased susceptibility of the post-venectomy leg to such cellulitis has been attributed to its altered lymphatic drainage.

Treatment of Chest Wall Injuries

Proper treatment for a chest wall injury may range from aspirin and advice to tracheal intubation and prolonged respirator care or even open stabilization. It is the physiologic severity of the injury and the patient's overall condition that determine therapy, for the "anatomically severe" injury may well be unimportant physiologically. For example, extensive posterior rib fractures are well splinted by the scapula, as well as by heavy back muscles further stiffened with post-traumatic hematoma; significant posterolateral flail movement of the chest wall is thus rare.

Temporary Measures

An unstable anterior or lateral chest wall can sometimes be "improved" for a time by having the patient lie upon his injured side, or by placing a sandbag on the large, loose portion of the chest, or by towel clip or wire traction on any loose rib: Additional hours of benefit may be obtained by breathing high concentrations of oxygen; but none of these measures is very satisfactory. After breathing for a few minutes with a sandbag on one's own chest, it is easy to imagine that flail chest breathing must really be difficult, if breathing against a sandbag represents relief. Nonetheless, any slight improvement in the efficiency of each breath can have rapidly cumulative, beneficial effects on the maximally stressed patient.

Operative Fixation

Operative fixation of fractured ribs can be performed by various techniques, from simple suture stabilization of jagged rib ends to intramedullary fixation or customized metal rib splints. I have been satisfied with the result of heavy chromic suture stabilization of rib fractures (round and round the rib, back and forth in muscle, or what-ever) when exploring an injured chest for some other purpose (usually bleeding), but have not operated electively solely to correct chest wall flail (though that could be appropriate on occasion).

In any case, since a flail chest stabilizes spontaneously within about two weeks, it has usually seemed preferable to avoid added surgical stress after trauma, and also not to "open" badly damaged soft tissues to possible infection. However, recent reports indicate that a simple heavy-wire suture repair of an overriding or unstable sternal fracture can markedly expedite recovery and reduce pulmonary complications: This sounds like a reasonable approach for such a problem.

Flail Chest Often Leads to Retention of Secretions

When the chest wall moves paradoxically, effective coughing can be painful and difficult, or even impossible. Heavy smokers who have had difficulty raising thick secretions before injury, become particularly poor risks after major chest trauma. Ineffective cough, chronic bronchitis and inadequate ciliary action in such severely injured patients, can lead to rapid accumulation of bronchial secretions and progressive atelectasis. Flail movement of the chest usually becomes increasingly pronounced some hours following injury, reflecting the increased respiratory effort required to stay alive.

Flail chest is a continued strain upon the patient, and requires persistent treatment if survival is to be achieved. As each breath becomes smaller and more difficult, a greater percentage of "total respiratory effort" is devoted to moving "used" air back and forth through respiratory passages: Declining respiratory efficiency means that the respiratory rate must increase. Even at bed rest, a sick patient may perform as much work "just breathing" as a healthy athlete who is exercising.

A subtle retention of secretions after chest injury commonly leads to progressive hypoxia, agitation and hypertension. Too often these signs of inadequate ventilation and pain are "treated" only with narcotics and sedatives; further depression of ventilation by these agents may then cause death. Therefore, an injured patient who is unable to raise his secretions and cough (which at first may seem like "no big deal") must receive intensive care ("a big deal").

Endotracheal suction can be the key to maintaining sufficient air exchange for survival, whether or not an endotracheal tube and respirator are used. It is important to institute endotracheal suction as soon as the patient demonstrates an inability to cough effectively, not just after he shows cyanosis, for that is often very late.

Ongoing epidural anesthesia or repeated intercostal "rib blocks" are reportedly very helpful in managing chest trauma. These methods of pain relief often improve patient cough and ventilation to the point where intervention with mechanical breathing assistance becomes unnecessary.

It is always desirable to avoid prolonged mechanical ventilation because of the likelihood that pulmonary barotrauma and infection, or even disuse atrophy of respiratory muscles, could result: This is particularly true for the emphysematous patient, who also is least able to tolerate significant chest wall flail.

Nevertheless, the early institution and prolonged maintenance of mechanical ventilation is often life-saving for the patient with flail chest who cannot generate significantly subatmospheric inspiratory pressures (because the size of the "flail" pleural cavity during inspiration remains about the same as the relaxed size of its underlying lung): Clearly "no subatmospheric inspiratory pressure" means "no air exchange in that lung" - see also Chapter 2).

Nasotracheal Suction: Indications and Technique

Adequate tracheobronchial cleanout is crucial to the management of injured or postoperative patients. One can usually predict that certain patients will require repeated nasotracheal suction and institute this early, before symptoms develop. (These patients used to include anyone undergoing resection for tuberculosis or bronchiectasis: Even after resection of all grossly diseased lung tissue, such patients initially had a minimal cough reflex along with copious secretions.)

Many currently and chronically addicted cigarette smokers require nasotracheal suction after any major surgery or other injury, due to continuing excessive mucus production within their irritated, narrowed bronchi, and inadequate clearing of such secretions by damaged cilia, as well as weak cough due to emphysematous changes.

Tracheal placement of a nasotracheal suction catheter can usually be achieved by gently probing via a nasal passage, using sterile catheters of varying sizes and stiffness (lubricated with Xylocaine jelly), while the tongue is held forward with a gauze pad by a nurse or the seated patient himself, in order to lift the epiglottis from the larynx. Catheter introduction to the trachea is often easiest at the time of a vigorous inspiration.

Secretions are then evacuated during five- to seven-second periods of nasotracheal suction: Each short period of suction is routinely followed by an appropriate interlude of oxygen administration (at 10 to 15 liters per minute by mask or cannula, unless the patient can obviously manage without).

If the suction catheter remains within the trachea during these pauses for "catch-up" breathing (or oxygen therapy), such catheter MUST be sharply kinked between fingers to occlude its lumen: This "kinking- off" of a tube that is still within the trachea but not "officially sucking" is essential, for a catheter tip continues removing endotracheal air even when a more proximal side-hole on the catheter stem has been left uncovered: Many already hypoxic patients have died of suffocation during therapeutic endotracheal suction, simply because inadequate consideration was given to their ventilation.

You might wonder why a patient cannot utilize some of the "new" air moved through the trachea by the suction catheter: After all, new air brought in during high-frequency ventilation certainly diffuses about effectively. Most likely, endotracheal suction causes "suffocation" by reducing the partial pressures of all endotracheal gases well below their current equilibration pressures in the pulmonary capillary blood. At the very least, this should lead to a sudden drop in blood oxygen, nitrogen and carbon dioxide: At worst it could also result in pulmonary edema and even microbubble formation within the pulmonary capillaries (see also reexpansion pulmonary edema; Chapters 3 and 5). It could well be that any pulmonary parameter that is improved by positive end-expiratory pressure or PEEP will be worsened by suction (at least momentarily).

In any case, at the point where further twisting and gentle probing while on suction produces minimal additional secretions from the trachea, the catheter is withdrawn on "full" suction (which may be much more than you think; gauge pressures are often expressed in a variety of different units), in order to lift out any thick mucus adherent to its tip: Oxygen is again provided. After a few of these suction sessions, most patients learn to cough well whenever a suction catheter is even mentioned.

Tracheobronchial Toilet

A sometimes satisfactory alternative to nasotracheal suction, once an endotracheal suction catheter has been positioned, is instillation of up to 60 cc of sterile Ringer's Lactate Solution (in 15 to 20 cc amounts) through this catheter. This stimulates the postoperative patient to cough vigorously, while also diluting his thick mucus.

Such a "tracheobronchial toilet" can result in productive, area-wide expectoration, so it is well to place large towels around the patient. Oxygen should be available at the time of this irrigation, just as

with endotracheal suction, even though arterial and alveolar oxygen levels may not fall quite as precipitously.

It has been reported that hypoxia and dyspnea can occasionally follow lobectomy or pneumonectomy, entirely as a consequence of right-to-left shunting through a patent foramen ovale (despite the absence of elevated right-sided pressures). This condition apparently results from a postresectional shift in heart position that directs the inferior vena caval venous stream onto an open foramen: Diagnosis by contrast echocardiography or cardiac catheterization allows correction through direct suture of the interatrial communication.

Tracheostomy and Endotracheal Tubes

Positive-pressure controlled ventilation is often indicated for the patient with serious chest wall injury, during the seven to fourteen day period required for adequate rib cage healing and stabilization. Endotracheal intubation can also help prevent aspiration of saliva or regurgitated material, or control pulmonary congestion and edema through application of positive end-expiratory pressure (these functions depend upon an effective inflated balloon cuff in the adult patient). On the other hand, a tracheostomy often causes aspiration of oral feedings because it anchors the airway and thus interferes with the normal airway occlusion during swallowing.

Tracheostomy (without ventilatory support) can dramatically decrease a massive air leak through the chest tube, by reduction of expiratory airway pressures (through elimination of grunting, coughing and other expiratory resistance). Tracheostomy can also cure the rare pulmonary edema that results from desperate inspiratory efforts against an almost complete upper airway obstruction, by providing relief from the markedly subatmospheric intrapulmonary pressures generated during such prolonged inspiration (see case report below).

A bonus of tracheotomy, in the presence of severe subcutaneous and mediastinal emphysema, is mediastinal decompression (which is achieved as the pretracheal fascia is incised; see Chapter 5). Of course, either tracheostomy or endotracheal intubation will usually expedite evacuation of airway secretions by suction catheter. Tracheostomy is reportedly useful for removal of endobronchial foreign bodies, sometimes using a Fogarty balloon catheter to aid in the extraction: Rigid tube bronchoscopy was allegedly easier through a tracheal stoma.

Tracheostomy used to be considered an important aspect of the prolonged respiratory assistance provided to an exhausted postoperative patient in cardiopulmonary failure, or an injured patient with contused or "wet" lung, or flail chest: At least the excessive metabolic demands directly related to vigorous respiratory work could be eliminated by controlled ventilation.

A tracheal stoma still seems appropriate at times for the control of persistent, excessive pulmonary secretions in the chronically debilitated patient with a large lung abscess, and apparently it has a limited role in the medical treatment of chronic pulmonary insufficiency. Overall, however, indications for tracheotomy have diminished markedly in recent years, as less irritating, more flexible, plastic oro- or nasotracheal tubes with improved, low-pressure, balloon cuffs have become available.

Although these more recently developed "airways" are readily inserted and replaced, we usually leave that to the anesthesiologist, except in acute emergencies: Some of the burden of urgent respirator management has also shifted to our anesthesiologists. I mention this because we all recognize that

every physician should be able to intubate a patient in an emergency, or regulate a respirator: Yet respiratory therapists and ICU nurses usually understand the controls and capabilities of advanced-model, properly humidified, volume respirators better than we, and most MD's would be well-advised to await an anesthesiologist before attempting endotracheal intubation (in any but the utmost emergency).

The literature documents an increasing incidence of laryngeal and endotracheal ulceration and stricture with longer duration of endotracheal intubation, but I still prefer an endotracheal tube to a temporary tracheostomy (for one or two weeks), as long as this seems well tolerated. A patient soon develops significant laryngitis and tracheitis if allowed to cough and gag on his endotracheal tube, of course, so it seems likely that the quality of nursing and respiratory care can help to determine how long an endotracheal tube will be tolerated.

Certain head and neck problems either mandate for or against a tracheostomy (versus another airway). A pulsatile anterior neck mass speaks strongly against tracheotomy, for example. There are times when nasotracheal intubation can initiate severe epistaxis, or unduly limit the size of endotracheal tube placed: Reportedly, it can also precipitate severe sinusitis, although I have not seen that. Tracheostomy may be required, however, when oral pathology, or lack of patient acceptance, or the need for blind intubation, prevent satisfactory orotracheal intubation.

It is always wise to cooperate in patient care with appropriate sub-specialists. Amazing advances in specialties other than our own may be less visible to us, engulfed as we are in the rapidity of change in our own field. For example, head and neck surgeons are making such interesting progress in laser resection or vaporization of airway or esophageal malignancies that one would be wise to discuss any problem case with an ENT specialist before proceeding with tracheotomy. (In fact, they have routinely taken such good care of patients who do require tracheotomy that whenever possible I prefer to let them perform the procedure and only become involved in follow-up.)

Temporary orotracheal intubation with a standard endotracheal tube has replaced postoperative therapeutic bronchoscopy in my practice, for such intubation allows controlled lung inflation (and thus improved oxygenation) during a leisurely aspiration of secretions; perhaps skillful fiberoptic bronchoscopy still has an occasional therapeutic role, when visual inspection or controlled manipulation of the suction tip seems essential, but I have not encountered such a problem postoperatively for many years.

Overview of Respiratory Care

Positive-pressure breathing assistance (by a reliable volume respirator or anesthesiologist) via a cuffed tracheostomy tube or cuffed endotracheal tube allows (1) internal support of the unstable chest wall, (2) proper endotracheal cleanout by suction as necessary, (3) air exchange without effort by the patient, (4) reliable delivery of well-humidified air at desired oxygen levels, (5) sufficient analgesia and sedation (including muscle relaxants if necessary to keep the patient from breathing against the respirator - a postulated cause of intracerebral bleeding in the premature infant) to permit the patient complete physical and mental rest, and (6) improved oxygenation and "control" of a "wet," "insufficient," chemically injured or "contused" lung (for all of which, positive end-expiratory pressure may be crucial).

But, the physician who selects intubation for respirator care, along with heavy sedation, takes on

an immense obligation, as the patient thereby becomes unable to cough, raise secretions or often even breathe without assistance. (During my general surgery training at Boston City Hospital, for example, even emergency tracheotomy was performed with extreme reluctance, for it carried a high probability of suffocation by retained secretions during the poorly staffed night shift.)

When Respirator Function is in Doubt, Ventilate by Hand

When every breath depends upon competent staff and reliable equipment, a three-minute lapse in nursing care can be fatal. Similarly, if the hospital "power" fails, or a respirator becomes electrically disconnected, or a tracheostomy tube becomes obstructed or displaced, death may ensue within minutes. An effective manual breathing bag system such as the Ambu bag (Air Shields, Inc., Hatboro, Pa.), should always be available for use while an actual or suspected respirator malfunction is confirmed and repaired.

Too often, one finds worried nurses or respiratory technicians drawing blood gases or nervously peering into the respirator's bowels while the patient turns ever more blue, gasping without avail against some mechanical malfunction. It must be obvious that neither blood gas determination nor respirator repair has immediate value in the emergency resuscitation of an acutely unstable patient. (A good flashlight also belongs at the bedside of every respirator patient, for use during those unexpected late-night, hospital power outages).

Pneumothorax Following Tracheotomy

After completion of a difficult tracheotomy on a patient showing significant respiratory distress, one may discover unilateral or even bilateral moderate pneumothorax on routine chest roentgenogram. Such pleural air accumulations are usually asymptomatic, having occurred during relief of severe airway obstruction. The common "medical rounds" explanation has been that pleura in the base of the neck was inadvertently injured at tracheotomy, permitting direct atmospheric entry.

That purported etiology is usually unacceptable to the surgeon, who knows "damn well" that he never came anywhere near the pleura. As is usual under such circumstances, the surgeon is correct, for the pneumothorax was a direct result of desperate and prolonged inspiratory efforts against a partially obstructed upper airway.

Mechanism: Once the pretracheal fascia has been opened during tracheotomy, any major drop in pleural cavity pressure can easily draw air into the mediastinum along the tracheal outer surface: This "mediastinal air" may then burst into one or both pleural spaces, with additional air then being drawn in and "pumped along" by the marked pressure changes of each violent respiratory effort.

Mediastinal air pumping stops, however, as soon as the inspiratory obstruction has been relieved by tracheostomy, and the patient can once again breathe easily. Such non-recurring intrapleural air collections can thus be ignored or removed by needle as seems appropriate (perhaps even by an open needle during Valsalva efforts against a momentarily occluded tracheotomy tube), as they are almost surely not the consequence of an ongoing pulmonary air leak (see Chapter 5).

Case Report: Tracheostomy Can Correct Pulmonary Edema Due to Severe Laryngeal Obstruction

On one occasion, an emergency tracheotomy (performed to bypass severe laryngeal edema in an

adult) rapidly terminated a copious production of frothy pulmonary secretions indistinguishable from pulmonary edema. Presumably, the patient's intense and prolonged inspiratory efforts dropped intrapulmonary air pressures sufficiently to draw fluid into the alveoli from nearby capillaries (in similar fashion to the mechanism postulated for "reexpansion pulmonary edema" - see Chapter 5).

Pulmonary edema related to laryngeal obstruction (e.g., during post-extubation laryngospasm) has been commented upon by others, who have attributed it to varying malign influences including low albumin, hypervolemia, cardiac reflexes, and so on. It seems far more likely that such edema is caused by the unusually low intrapulmonary air pressure during each prolonged and desperate inspiration (which is "important" since predisposing mechanical problems are far more readily prevented or treated than "intangible" conditions, portentous "imponderables" or even hypothetical reflexes).

Whenever Possible, Place an Endotracheal Tube Prior to Tracheotomy

Serious complications are more easily avoided if tracheotomy can be performed electively, after an oro- or nasotracheal tube has been placed. With good airway control and pain relief by an anesthesiologist, the procedure then requires no further haste; indeed, it usually becomes possible to defer the procedure until better light, suction, and other facilities become available. The difficult-to-intubate patient can sometimes have successful orotracheal or nasotracheal tube placement over a small fiberoptic laryngoscope or bronchoscope.

Preliminary endotracheal intubation in infants and young children has the additional advantage that the trachea is more easily identified if it has a relatively stiff tube inside it. The trachea in a young child is soft walled and readily collapsed or pushed aside: This may result in disastrous cannulation of nearby tubular structures of similar appearance (such as esophagus and carotids). The cricothyroid membrane is allegedly the safest site for an emergency tracheotomy, but I have no experience with this.

Comments on Tracheotomy Technique

The usual sharp metal retractors, hooked under a cartilage ring, provide excellent positional control of the trachea during its manipulation for tracheostomy. A loose loop of 2-0 monofilament suture around a cartilage ring on each side of the proposed tracheal incision can similarly increase the safety of this procedure, especially in children.

Traction on these two "suture loops" lifts and stabilizes the trachea, simplifying the tracheal incision as well as tracheostomy tube insertion. (One must be prepared to proceed efficiently, once hooks or sutures are applied, for accidental perforation of the underlying endotracheal tube balloon-cuff frequently eliminates positive-pressure airway control).

Emergency reinsertion of a tracheostomy tube is also simplified by such "thread handles," which are therefore left in place until the tracheostomy tract has "stabilized" (two or three days after tracheostomy). Later removal of these tracheal wall sutures is expedited if the initial knot on the closed loop of suture was made well above skin level.

Caution: During a hurried, anterior tracheal incision, it is not difficult to cut into the posterior tracheal wall as well (or even the underlying esophagus), especially when dealing with the airway of a tiny newborn.

It is evident that positive-pressure assisted ventilation in an adult (other than high-frequency ventilation) requires the use of a snug-fitting endotracheal or tracheostomy tube. Traditionally, the necessary "tight fit" has been achieved below the larynx by placing an inflatable rubber cuff around the airway cannula. Improper tube fit or angle, or prolonged over-inflation of the older hard-rubber cuffs, often eroded the tracheal mucosa and tracheal wall (and occasionally even the nearby innominate artery): Severe tracheal stricture was an all-too-common result of prolonged respiratory support in past years.

Another hazard associated with use of old-fashioned metal, tracheostomy tubes was that the inflatable cuff could slip down over the "out of sight" tracheostomy tube tip within the trachea. This might still allow air to be pumped into the lungs during inspiration, but it could easily block adequate expiration, leading rapidly to an in-tolerable increase of intrathoracic air pressure (within lungs, in this case, so "tension pulmothorax?") as well as cessation of air exchange.

Cuff pressure of any endotracheal tube should routinely be kept well below the capillary perfusion pressure (which is normally around 25 to 30 cm H20) even if some air then leaks back out around the tube during positive-pressure ventilation. Silastic balloons allegedly are less irritating to the tracheal mucosa than other rubber formulations: Reportedly, some endotracheal tube balloon-cuffs may enlarge markedly in the presence of certain anesthetic gases that diffuse rapidly into the balloon: This could easily increase cuff pressures to unacceptable levels (see Chapter 3).

All tracheostomies produce some late narrowing at the site of tube insertion, especially if a portion of the tracheal wall is "needlessly" (my opinion) excised for creation of a stoma (any excised tissue or other small object also has a tendency to disappear into the tracheal stoma, thus complicating an already dangerous emergency procedure). Postoperative tracheal narrowing is usually not crucial, except in infancy or childhood, where removal of the tracheostomy tube may be followed by persistent, partial tracheal obstruction that can be most troublesome.

A tracheal stoma centered more than two rings below the cricoid cartilage will allegedly increase the risk of massive hemorrhage due to innominate artery infection and erosion. Temporary control of sudden and significant innominate artery bleeding (usually occurring many days or weeks after tracheotomy) is sometimes achieved by endotracheal balloon-cuff overinflation, but more reliably by a "pretracheal index finger" inserted into the tracheotomy skin incision just below the tracheostomy tube to compress the pulsatile innominate artery against the deep surface of the sternum (pending emergency sternotomy for innominate artery ligation and division).

The literature strongly suggests that vessel repair or grafting under these circumstances is contraindicated, and that it is important to allow the divided ends of the innominate artery to retract well away beneath nearby healthy tissues. Surprisingly, neurological harm rarely follows emergency innominate artery division.

Troublesome minor ooze from the thyroid (at the time of tracheostomy) can often be controlled by a temporary hemostatic pack of absorbable coagulating gauze. Avoid tight packing of an entire tracheotomy incision, however, or tight suture closure of skin around the tracheostomy tube, for either may cause minor to marked subcutaneous emphysema due to paratracheal trapping of air that leaks back along the tracheostomy tube (often at much-above-atmospheric pressures, if the patient is receiving ventilatory assistance).

Tracheostomy Care

Copious tracheobronchial sputum production that interferes with air exchange is often a continuing problem for tracheostomy patients. The entire tracheostomy tube must be removed and replaced occasionally when it becomes heavily encrusted with dried secretions. An effective cold-fog tent or vaporizer, blowing directly over the tracheostomy (or a properly-humidified respirator) can markedly reduce both secretion formation and sputum tenacity. This equipment should be an integral part of the care of any patient with a recent tracheostomy. Enzymes, detergents and other preparations are of minimal significance in comparison to the importance of moist air.

Tracheostomy suction must be taught as a uniform special hospital procedure; otherwise a safe and sterile suction technique cannot be enforced. Severe hospital-acquired bronchopulmonary infection is probably the most common complication of tracheostomy. As discussed above, it is important to avoid prolonged episodes of endotracheal suction (more than five to ten seconds), and oxygen should be immediately available for administration between suction treatments.

Chest Tubes Are Often Helpful in "Chest Injury" Care

Minor to moderate pneumothorax is not uncommon after chest injury. Such a pneumothorax may be stable and reabsorb uneventfully (peripheral lung injury rarely leads to persistent air leak in children and young adolescents, for example). Nonetheless, if it is unclear whether a pneumothorax is stable (and early after chest injury it is often unclear), closed thoracostomy can easily prevent hazardous, undetected progression of the pneumothorax: Such progression is especially likely when a patient with already injured lung requires positive-pressure ventilatory support (at surgery or on a respirator).

Positive-Pressure Ventilation and Tension Pneumothorax

Tension pneumothorax may develop after "spontaneous" bleb rupture, thoracentesis, rib fracture, or other trauma to the lung: However, it is far more likely to follow positive-pressure ventilatory support (during resuscitation, anesthesia, or longer term respirator care), for such support "abnormally" elevates intrapulmonary pressures, causing air leaks to develop, reopen or increase. Under these circumstances, a tension pneumothorax that is not promptly recognized and treated may rapidly kill the patient (see Chapter 5).

As an isolated occurrence in the ambulatory patient, tension pneumothorax is usually fairly evident (pain, dyspnea and tracheal deviation away from the fixed hyperinflated hyperresonant hemithorax with decreased breath sounds). Death in such a patient is attributed primarily to hypoxia rather than circulatory arrest by cardiac compression: Prompt relief of the intrapleural pressure buildup can allow immediate recovery, even following loss of consciousness with respiratory arrest.

In a draped patient under anesthesia (or comatose or heavily sedated on a respirator) tension pneumothorax may not be nearly as obvious (or even detectable); tension pneumothorax should come to mind, therefore, whenever such a patient inexplicably "gets into trouble": Then one should palpate for tracheal position, auscult and percuss the chest, compare respiratory movements of the two sides, check volumes exchanged and respirator pressures, see if the abdomen is "newly distended," get an urgent chest roentgenogram and "if worst comes to worst," quickly insert a large plastic needle or tube into one or both sides of the chest without a roentgenogram.

Even without prior direct lung injury, prolonged respirator care will occasionally result in a sudden devastating tension pneumothorax, especially when continuous "positive end-expiratory pressure" (PEEP) is being applied. Death from tension pneumothorax in a patient on a respirator is apparently consequent to loss of cardiac output.

With the lungs being ventilated "involuntarily" at high pressures to "the bitter end," any blood that reaches systemic arteries should still be well oxygenated: Therefore, any emergency evaluation of arterial blood gases cannot contribute to the diagnosis of tension pneumothorax in the ventilated patient while his blood continues to circulate (not surprisingly, "unexplained" hypotension due to reduced cardiac output is alleged to be a more useful early sign of tension pneumothorax under these circumstances).

Previously we defined tension pneumothorax as "free intrapleural air at persistently above-atmospheric pressures." Although this definition was developed for the spontaneously breathing individual, it is also appropriate during positive-pressure ventilation: Any pneumothorax then becomes a tension pneumothorax by our definition, and thus an urgent indication for a chest tube.

This approach even makes therapeutic sense, for it has been reported that any significant intrapleural air accumulation increases the intrapleural pressures of a patient being ventilated, and often measurably diminishes his cardiac output (in similar fashion to "vigorous" positive-end-expiratory pressure: Since rapid enhancement of blood volume often improves cardiac output in the face of strong PEEP, it is likely that the hypotensive patient with stable tension pneumothorax might also respond temporarily to expansion of his blood volume): Closed thoracostomy remains the appropriate treatment for any tension pneumothorax - on or off the ventilator.

Avoid Premature Tube Removal

It is well to avoid chest tube removal while the patient remains controlled on a respirator, even if air leak and fluid drainage have long since ceased. Not uncommonly, a new air leak will suddenly develop in a patient still being ventilated (usually at 2 a.m.); it is much better to learn of some recent-onset air leak during morning rounds than to be called in for an emergency life-saving closed thoracostomy. Surprisingly, there is often considerable pressure from medical colleagues to remove any chest tube that is not obviously essential: Despite the slight, added nuisance for nursing care, however, such a tube could still prove lifesaving.

The chest tube will hardly restrict a patient who is being ventilated, anyhow, and it never causes empyema (even in a septic patient). If an underlying lung infection should lead to empyema (whether the patient is on a respirator or not), any appropriately positioned intrapleural tube also becomes therapeutic for the empyema.

Cigarettes

Improvement in lung function will occur within a fortnight off cigarettes: The patient should understand that cessation of smoking prior to elective surgery can greatly decrease postoperative pain and pulmonary problems (less coughing, less endotracheal suction) and thereby speed recovery.

Bronchodilators and Expectorants

Oral intake of 10 drops of a saturated solution of potassium iodide, three times a day (better yet, 0.25 to 1.0 gm. of sodium iodide in 500 cc of the patient's regular intravenous fluid), will help loosen tenacious secretions (as does adequate patient hydration and proper humidification of inspired air). Some individuals are allergic to iodides, of course, and old or weak patients receiving sodium iodide intravenously should be watched closely, with suction at hand, so that they do not drown if unable to raise stimulated secretions. (I have not resorted to such iodide therapy for years, but it is very effective and thus worth considering on occasion.)

Intravenous aminophylline is usually well tolerated and helpful in opening the peripheral airways of asthmatic, emphysematous, bronchiectatic or cigarette-smoking patients. Alternate routes for aminophylline administration are by rectal suppository (every six to eight hours) or by mouth. One will occasionally wish to discontinue aminophylline in the presence of unexplained nausea, tachycardia or other cardiovascular manifestation. Serum aminophylline measurements often allow more effective therapy.

Atropine-like drugs may soon contribute to the treatment of bronchospasm in emphysema and chronic bronchitis, although they are allegedly less effective for asthma than presently utilized adrenergic agents (such as Albuteral). The narrowing of trachea and bronchi that is seen with vagal stimulation is blocked by atropine. Allegedly Albuteral plus atropine has no more effect on widening the emphysematous airway than atropine alone. (I have no experience with the use of atropine for this indication.)

Although I have not recognized bronchospasm secondary to aspirin administration, this is supposedly another cause of asthma attack, by its blockage of Prostoglandin E2. Perhaps a useful prostoglandin bronchodilator is "just around the corner."

I often resort to significant doses of intravenous steroids for the initial control of asthmatic-type wheezing or postoperative pulmonary insufficiency (see Chapter 14).

Frequent intermittent positive-pressure breathing treatments (up to several times per hour, sometimes with Alupent, Albuteral or Bronchosol alternating with saline) have helped many critically ill post-operative patients through their borderline pulmonary insufficiency (undoubtedly due in part simply to the increased attention directed toward their respirations). Any of these bronchodilators can probably potentiate dysrhythmia, and at maximal dose they should only be used short-term, depending upon dosage given and patient need and response. (And review drug package inserts occasionally: They may change and we often forget.)

Incentive spirometry also can help the patient clear his lungs of loose mucus as he becomes stronger. However, no mechanical breathing treatment (other than PEEP on a respirator) is likely to benefit the patient with consolidated lung parenchyma, when this is not secondary to endobronchial blockage by mucus. For example, incentive spirometry does little more than harass the patient with postpericardiotomy syndrome, widespread minor atelectasis and non-productive cough.

Wheezing As a Sign of Impending Pulmonary Edema

Overhydration is a common cause of postoperative wheezing. Such wheezing, and an increase in daily weight of the ambulatory patient, may be the only signs of an impending, florid, pulmonary edema. Usually, therefore, I administer Lasix (20 to 100 mg IV or by mouth) to any postoperative

patient who is not obviously dehydrated, whenever new wheezes are detected. It is surprising how often diuresis eliminates respiratory wheezing, even with no rales detectible at the lung bases.

An accurate, daily A. M. weight of each patient can be very helpful. Why hospital scales are so often inaccurate, when trucks and vegetables must be weighed reliably, has always been a mystery: Perhaps there ought to be a law about accurate patient weights. Now if daily hospital charges could be based upon so many dollars per kilo of patient....

Retrosternal Noises in the Night

On occasion, after closure of a thoracotomy or sternotomy (or with spontaneous pneumothorax, or pneumomediastinum from any cause), a loud crunching, ripping or wheezing, retrosternal sound is heard, usually in association with every heartbeat. Internists or cardiologists may then "fear the worst" (whatever that is): However, a change in position of the usually stable postoperative patient will alter this sound, which is seemingly due to compression of an air pocket between the heart and anterior chest wall. (It often sounds like regular wrinkling of stiff cellophane, or intermittent squeezing of subcutaneous emphysema.)

Rarely, an intact, emphysematous lung, compressed between the anterior chest wall and the heart, will emit a similarly unusual and insignificant sound. Pericardiotomy is often followed by an audible rub, as is reapproximation of roughened pleurae during lung reexpansion, lung inflammation, or with pulmonary embolus. A persistent loud pericardial rub may be an early sign of "postpericardiotomy syndrome" (see Chapter 15).

Torn esophagus can also provide the mediastinal air needed for this sort of sound, in which case the diagnosis is usually evident. Similarly, intrapericardial perforation of a gastrointestinal ulcer can lead to such noises: In both cases, urgent treatment will be required, but there will be many other serious findings in that very sick patient, in addition to a "noisy" heart beat.

Occasional Problems with Nasogastric Tube Drainage

At times, it may be lifesaving to keep a Levine tube properly positioned and functioning, especially when gastrostomy is impractical or undesirable (as with the stomach surgically absent or up in the chest, or during the non-surgical treatment of severe pancreatitis). Unfortunately, patients in these dire straits are often intermittently confused, uncooperative, suffering from delirium tremens or frankly psychotic.

To prevent such a patient from repeatedly pulling the tube out, some surgeons have resorted to suturing the nasogastric tube to the nose. However, it seems preferable to place the nasogastric tube in such fashion that it causes progressive distress only during unauthorized attempts to remove it. A practical method of nasogastric tube placement for such unusual occasions is described below:

Procedure: The Levine tube is inserted by nares, with an appropriate number of assistants controlling the patient. If rapidly inserted, the tube will usually be spit out of the mouth of such an uncooperative individual. If not, it can be grasped in the posterior throat with a blunt sponge forceps or swept forward with a padded throat stick.

The tube tip is then pulled out of the mouth so far that, when reinserted into the same nostril and

gently passed down the esophagus, it will easily reach the desired position in the stomach. Satisfactory tube location is confirmed by blowing 5 to 10 cc of air down the tube (while listening or feeling for a corresponding gurgle over the stomach) or by syringe aspiration of stomach contents from the newlyplaced tube.

The nasogastric tube can then be secured without any tape or suture to the patient: The loose "first loop" of tube (from mouth back to nose) is simply taped "side-to-side" to tube that still remains outside of the nostril (that never was inserted) before leading whatever tube length still remains outside the patient off toward the suction device or drainage bag.

With the first loop and proximal tube securely taped together in this fashion, the nasogastric tube remains in a relatively comfortable position unless it is pulled: The harder the patient pulls, the worse it feels, so a tube inserted in this fashion is essentially impossible for the disoriented patient to remove.

Elective nasogastric tube removal by the physician is simple, however: When the appropriate moment has arrived (leaving the tape undisturbed), the first loop of nasogastric tube is cut somewhere in front of the lips, and the whole thing then smoothly pulled out of the nose. This is not as unpleasant for the patient as it may sound. He merely has two tubes passing into one nostril and one emerging from his mouth.

With the first loop taped so that it remains loose, this is not much more aggravating than a normal nasogastric tube. Taping the tube firmly to itself in the above fashion (perhaps after moistening the tubing with Benzoin) prevents permanent tightening of the first tube loop around the soft and hard palate. It is only when tugging on the tube, therefore, that the palate is momentarily distressed.

As mentioned, it is well to disconnect any nasogastric tube from its suction source every time you visit a postoperative patient. The momentary "gurgle-slurp" provides reassurance that suction actually is being applied. Too often, this is not the case, even with all gauges and red lights apparently in their optimum state. Meticulous attention early after surgery can prevent miserable distention later postoperatively. Some nurses tend to "believe" all lights and gauges, so simple gravity drainage of the nasogastric tube (into a small, vented plastic bag) will often prove more effective than a troublesome suction device.

On Foley Catheter Removal With the Balloon Inflated

On occasion, a forgetful elderly individual will remove his own Foley catheter with the balloon still inflated: Although signs of trauma are usually evident, he may not remember why this happened, nor can hospital staff figure it out. The answer came to me one day when an older patient far down the corridor accidentally stepped upon the loop of tubing between his catheter and the drainage bag. Having thus yanked the Foley balloon well down into his urethra, he became electrified (so to speak) and moved with astonishing rapidity to complete the excruciating extraction. When queried a few moments later, he only recalled having suddenly developed an extreme dislike for the offending tube.

Resuscitative Efforts Case Report: Artificial Respiration

As a medical student, I once trailed the usual entourage of interns and residents into a private room crowded with relatives, just as the eighty-five year old patient expired. The chief orthopedic resident, a former "linebacker" who had recently nailed her hip, leaped onto the bed and proceeded with vigorous

"artificial respiration."

Perhaps I should mention that in those days, artificial respiration consisted of vigorous compression of the rib cage, which was then allowed to spring back, sometimes aided by lifting the patient's arms ("out comes the bad air, in goes the good"). This particular effort to do "everything possible" became more complicated, however, when the patient's rib cage collapsed permanently on the first compression. Our chief resident's resuscitative efforts thereafter were designed mainly to fluff up this ladies' chest, so that she would not appear so awfully flat to the surrounding relatives.

Mouth-to-Mouth Resuscitation

Sometime later, mouth-to-mouth resuscitation came into vogue: Although I always blew in, then moved swiftly aside to avoid expiratory germs and gastric content, I gradually became disenchanted with this method as well; more so after prolonged unsuccessful efforts on a young female with disseminated tuberculosis and a small child with meningococcemia. Undoubtedly, mouth-to-mouth resuscitation still has its place: However, early, effective cardiac massage often supports the circulation sufficiently to allow continuation of effective spontaneous breathing until the patient can be defibrillated, or until someone arrives with a breathing bag and mask.

Case Report: External Cardiac Massage

One afternoon, as I passed a back-corridor four-bed ward, an agitated nurse's aide approached me and asked if Mr. X was my patient. I said he was not, and asked what was the trouble. She said he seemed unwell. When I urged her to notify his doctor, she rushed off to that center of modern nursing care, the chart rack.

After an indecisive moment, I wandered in to look at the patient in question, before visiting my own patient in the next room. He was in the corner bed, curtains drawn and agitated wife at his side; an obviously terminal, middle-aged man, taking occasional gasping breaths. I asked his wife to step out for a moment while I felt briefly for carotid or femoral pulse, then began external cardiac massage when no pulse was evident.

The day was warm, the bed bouncy, and my back aching after several minutes of massage. As long as I worked hard at his circulation, however, the patient breathed satisfactorily. After a while, between pushes, I opened the curtain in an attempt to attract help. Some relatives of another patient sleeping in this room started to come in, then left when they realized that I was "busy."

The bedside buzzer resulted in the usual absence of response. I rushed to the hallway door twice between compressions, but everyone was out of sight. For perhaps ten more minutes, I massaged, waiting for help, expecting at least that the worried aide would soon return (but she was still busy on the phone, conscientiously describing the problem to the urologist who had circumcised this man an hour or two before).

Eventually, someone happened by, and I sent him off to get help. Although delayed by the fact that the aide was still on one phone, and the head nurse on the other as usual, a "Code 99" was finally broadcast and dozens of people rushed in: The patient recovered nicely after one external countershock.

Conclusion: Adequate external massage sometimes supports adequate circulation and, thereby,

spontaneous respiration.

Miscellaneous Aspects of Cardiopulmonary Resuscitation

On rare occasion, effective external cardiac massage may first require one or more very forceful chest compressions, in order to fragment and displace distally a massive pulmonary embolus blocking the right heart outlet or main pulmonary artery.

Case Report: Massive Pulmonary Embolus

This middle-aged patient had no known cardiac or pericardial disease when he collapsed suddenly in the x-ray department, just after lower leg venogram revealed thrombophlebitis. The patient's full veins and the absence of palpable pulses during vigorous external cardiac compression suggested main pulmonary artery obstruction by a large pulmonary embolus.

That presumed right heart outlet obstruction was partially relieved, and cardiac output restored, only after a discerning anesthesiologist compressed the patient's sternum so vigorously that both sternum and clot were fractured. Shortly thereafter, the still comatose patient underwent successful pulmonary embolectomy (using cardiopulmonary bypass), followed by full recovery.

Note: It can be very important NOT to open the pericardium of such a patient until superior and inferior cava have both been cannulated (via the common femoral and internal jugular veins), for otherwise the acutely obstructed and suddenly unsupported right ventricle may balloon massively out of the new pericardial opening "never to beat again".

Irreversible Sudden Dilation of Heart

An equally disastrous overstretching of heart muscle may occur during surgery for aortic insufficiency if ventricular fibrillation occurs soon after cardiopulmonary bypass is instituted - before the ascending aorta is cross-clamped. For after a moment or two of vigorous fibrillation, such a non-beating (hence unprotected) left ventricle will suddenly distend acutely in response to pressurized blood from the aortic cannula being delivered forcefully across the insufficient (open) aortic valve.

Sudden massive overstretching of heart muscle presumably leads to immediate irreversible loss of contractility because it forcibly rips myosin ratchets away from the actin filaments along which they formerly moved.

Open Cardiac Massage Still Has a Place in Resuscitation

External cardiac compression may be ineffective when the patient has a very stiff (due to heavily calcified chondral cartilages) and emphysematous "deep" chest; in that circumstance, one must revert to a swift left submammary incision for open cardiac massage. Open massage can also be essential to gain control of cardiac wounds or relieve posttraumatic pericardial tamponade. The necessarily swift incision needed for open cardiac massage may occasionally wound the heart or transect the left phrenic nerve - especially when this nerve is pushed forward by a ballooned-out pericardium in the presence of cardiac tamponade.

Case Report: No Electrical Activity

"Multiple defective defibrillators" was the initial conclusion, that early morning at change of shift, when the patient undergoing resuscitation would not "jump" despite maximum electrical discharges. The defibrillators proved blameless, however, after it was demonstrated that death had occurred the night before (patient lividity and rigor, with the body already at room temperature).

CHAPTER 13: THE LUNG

Lung as a filter;... Regulating the size of an alveolus;... Air trapping;... Case report: Air trapping by a bronchial adenoma;... Lobar emphysema;... Diagnosis of lobar emphysema;... Treatment of lobar emphysema;... Atelectasis;... Lung compression;... Lung abscess;... Chest surgery has not yet outgrown its need for bronchography;... Indications for bronchogram;... Bronchogram technique;... Treatment of lung abscess;... Tube drainage of lung abscess or lung cyst;... Chest tube suction helps obliterate a lung abscess;... Case report: Tube drainage of infected echinococcus cysts;... Chest exploration for lung injury;... Dealing with posttraumatic bleeding from lung;... Persistent air leaks;... Open fissures;... Indications for segmental resection;... General comments on segmental resection technique;... Indications for wedge resection;... Practical aspects of wedge resection technique;... Wedge resection in spontaneous pneumothorax;... Steroids and reopened air leak;... Lung cysts;... Avoid mobilization of unresectable apical tumors;... Middle lobe torsion.

Lung as a Filter

All blood returning to left heart chambers from the right heart must first filter through lung. Not surprisingly, lung plays a vital role in metabolic and cellular processes other than gas exchange. As a low-pressure in-line nondisposable filter, lung also deals economically with all but the most massive clots and embolic showers.

Eliminating what it can, absorbing much of the rest as necessary punishment, scarring down around harmful particles when all else fails, the lung plays a major role in vital body processes. It protects delicate end arteries of the high-pressure, systemic circuit from damage by impure blood, prevents a carious tooth or infected finger from causing brain abscess, and disposes of small clots or fragments of crushed muscle before they can lead to blindness or renal hypertension.

As an airway also, lung must take constant particulate invasion in stride. Inhaled solids or liquids are usually cleared by high-velocity outgoing air currents (cough) and the continuous slow outward flow of mucus; some particles undergo preliminary digestion by bacteria or white blood cells before they will disimpact from an airway, others may permanently block and thereby "destroy" distal lung being served by that blocked airway: Nearby "breathing units" then enlarge and "take over.

While our "to-and-fro" method of air exchange is not as efficient for oxygen uptake or carbon dioxide elimination as a simple "flow through" ventilation system might be, the constant "back flushing" of air passages by expiratory air movements does help to prevent progressive distal impaction of all inhaled foreign matter. Lung would soon lose all function if it had to serve as an in-line air filter; unlike the material removed from our blood during each circuit, even clean air carries a suspension of indigestible or inorganic materials that would soon pack our lungs until they resembled the collection bag of a vacuum cleaner.

Our "to-and-fro" system of ventilation retains some "used" or dead space air within the lungs at the end of each expiration, but at least "to-and-fro" avoids the need for additional valves or exhaust openings, and it also maintains essential CO_2 levels while limiting water and thermal losses. Appropriate airway size becomes quite crucial when only incomplete air exchange is achieved during each breath, however; not only must air passages be large enough to conduct maximum air flow freely, but they must also be small enough to prevent excessive storage of used air.

Regulating the Size of an Alveolus

The natural tendency of normal lung is to decrease in size when possible. This primarily reflects the surface tension in millions of tiny alveoli, with a lesser contribution by pulmonary elastic tissue. The extremely strong tendency of an "incomplete" or "perforated" fluid bubble (i.e. an alveolus attached to a bronchiole) to collapse, is controlled by secretion of lung detergent (surfactant) which lowers the intraalveolar surface tension to levels compatible with normal lung expansion.

Changes in surfactant concentration apparently help to regulate alveolar size. Thus, rapid alveolar overexpansion dilutes available surfactant, while sudden collapse increases its concentration per unit of surface area. A nice feature of this design is the fact that surface tension never "gets tired": To the degree that lung "elasticity" depends upon surface tension, therefore, it can continue expanding and recoiling indefinitely without this component of its elasticity "wearing out."

When foreign material is inhaled, it may partially or totally block a bronchus: Because the diameter of any bronchus or bronchiole changes in response to intrapulmonary pressures, inspiratory widening and expiratory narrowing of a partially blocked airway may lead to distal "air-trapping" as long as the airway occlusion remains incomplete; atelectasis soon follows when the bronchus becomes totally obstructed.

Either air trapping or atelectasis can lead to distal tissue destruction and fibrosis, depending in part upon the foreign material blocking the airway, and the associated bacterial flora. The more irritating the impacted foreign material (a peanut, for example), the more rapidly a partial blockage will continue on to total obstruction and atelectasis (due to endobronchial inflammation and spasm, as well as locally induced secretions). Of course, a partially obstructing foreign body may also be expelled, or drawn distally until it completes the bronchial closure.

Air trapping by foreign body impaction within segmental or sub-segmental bronchi or bronchioles is difficult to diagnose on chest roentgenogram, although a partially blocked airway may become evident when it causes an audible wheeze or diminished breath sounds over contiguous portions of the chest wall; collateral air flow between neighboring lung segments may limit segmental atelectasis or air-trapping from such cause. Even a small zone of persistent air trapping can become hyperlucent on chest roentgenogram, but complete bronchial obstruction usually supervenes before detectable ballooning is caused by a foreign body in a peripheral airway.

Fluoroscopy often demonstrates air trapping when due to a larger foreign body, bronchial adenoma or lobar emphysema: One may see delayed ventilation of a lobe or lung, with mediastinal movement toward that side on inspiration and away on expiration. Roentgenograms taken just at the end of a rapid, forced expiration may show the affected side still relatively expanded. The surgeon is advised to be present at such fluoroscopy, or to have it recorded on cine film, for a decision to proceed with bronchoscopy or thoracotomy must often be based on evanescent findings.

Case Report: Air Trapping by a Bronchial Adenoma

One young woman with a bronchial adenoma wheezed only during a maximal inspiratory effort (which expanded the bronchus sufficiently to allow air entry past the tumor). Following two or three such efforts, however, the inspiratory wheeze would cease, for air could no longer enter the now overfilled lobe. This intermittent wheeze caused confusion between referring physician and several

consultants, as the patient had been advised to see the consultant only if she was wheezing at that time, so she faithfully wheezed herself "full and quiet" just prior to each examination (just another case in which the history was more important than the physical examination).

Lobar Emphysema

As a surgical emergency, congenital lobar emphysema affects mostly the very young, although an unusually mild case may survive without surgical treatment into, or even past, childhood: A rather distended, "macroalveolar" lobe (with easily visible, enlarged alveoli) is occasionally encountered at surgery for spontaneous pneumothorax in young adults. At that stage, I have been hesitant to remove the entire lobe, if it was not significantly enlarged, for fear of overstretching the remaining "almost normal" lobe(s).

Diagnosis of Lobar Emphysema

The progressively distressed newborn with lobar emphysema is typically "a little blue" at birth, perhaps requiring resuscitation. Roentgenograms of the chest reveal one lung relatively hyperaerated, the other moderately atelectatic, with the heart shadow displaced toward the "atelectatic" side.

Pediatricians commonly focus on the less well-aerated lung and assume that the heart has been pulled over by lung collapse. An anesthesiologist may therefore be the first physician consulted, with a request to suction out and fully inflate the atelectatic lung (a very bad idea, for additional endobronchial pressure can rapidly balloon the opposite, emphysematous lobe to a size not compatible with continued respiration or cardiac output).

Usually several roentgenograms have been taken at various stages of respiration by the time a chest surgeon is consulted - for the rapid breathing efforts of this dyspneic newborn almost guarantee that both inspiratory and expiratory films will be available, regardless of the X-ray technician's intent (which normally is to take only "inspiratory" chest roentgenograms).

Careful comparison of these repeated roentgenograms will reveal a full range of chest wall and diaphragm respiratory movement on the apparently poorly inflated or "atelectatic" (but actually normal) side, while the diaphragm remains low and ribs stay farther apart on the side with lobar emphysema. On the other hand, if the respiratory abnormality really is true major atelectasis, ribs often stay persistently closer together than normal on the poorly inflated side, and that hemi-diaphragm remains relatively elevated, with x-ray revealing normal respiratory movement on the fully inflated side.

If regular or cine fluoroscopic evaluation of lung fields is also carried out (and usually this is not necessary), the surgeon should be present (or personally review the cine) in order to form his own opinion on which side is doing the most respiratory work, which side has delayed air exchange, and which way the mediastinum is pushed or pulled with respiration. (Of course, there are no ventilatory delays when the patient only has atelectasis.)

The rapid respiratory rate of a dyspneic infant makes ordinary fluoroscopy especially hard to interpret, but lobar emphysema affects respiratory dynamics much like a major pneumothorax: In both cases, a large volume of air is more or less trapped within the overfilled pleural cavity, so the heart moves away from the side with lobar emphysema on expiration and toward it on inspiration.

It is reported that congenital cystic adenomatoid malformation and acquired persistent interstitial pulmonary emphysema also trap "useless" air within the thorax, and that they may require direct surgical attack for similar indications. (My experience has been almost entirely with single-lobe disease, which we have simply referred to as "lobar emphysema.") Interstitial pulmonary emphysema supposedly is consequent to respirator therapy of premature infants, and may be associated with varying degrees of bronchopulmonary dysplasia as well as pneumothorax, atelectasis, and pulmonary infection.

Treatment of Lobar Emphysema

Proper treatment for lobar emphysema is immediate lobectomy. In the meanwhile, positive-pressure breathing can markedly aggravate the air trapping problem, so the anesthesiologist must always be reminded that a few squeezes on the ventilating bag could kill. Rather than having his respiration controlled, therefore, the tiny patient is allowed to breathe himself down with inhalation anesthesia to an appropriate stage for surgery to commence.

When such a marginal newborn patient gets into "real trouble" during induction of anesthesia, an immediate, wide, intercostal opening of the rib cage allows the overexpanded lobe to balloon forth from the chest, thereby converting a major emergency with a dying child into a routine lobectomy (which can then proceed safely under gentle positive pressure ventilation). Hemaclips allow rapid and effective control of smaller vessels and bronchi, and lobectomy is further expedited by the fully open interlobar fissures that are regularly encountered during infancy (when not operating for lung infection).

Atelectasis

Total obstruction of a lobar bronchus usually results in rapid absorption of any air remaining distal to the blockage. This absorption occurs through the peri-alveolar pulmonary capillary circulation: The resulting "collapsed" or airless state of lung parenchyma is termed atelectasis: Semi-rigid airways within such an atelectic lung cannot really collapse, of course, but they usually fill with mucus distal to the blockage: Thus an atelectic lung or lobe soon appears "airless" on chest roentgenogram.

Simple displacement of parenchymal air by fluid (transudate or exudate) during pulmonary edema or pneumonia can also create obviously airless (in this case "consolidated") lung on chest roentgenogram. One often sees air-filled lobar and segmental bronchi within airless lung: Such an air bronchogram, which is visible on standard chest roentgenogram, strongly suggests that the patient has pneumonia (rather than atelectasis due to a major bronchial obstruction).

However, multiple mucus plugs in smaller bronchi may cause significant atelectasis in the presence of an air-filled lobar bronchus, especially in a postoperative "current cigarette smoker" who is reluctant to cough. There are other clues that can help differentiate between various conditions that give similar roentgenographic shadows, for example, while lobar pneumococcal pneumonia may cause some decrease in size of the affected lobe, Friedlander's pneumonia (Klebsiella pneumoniae) will often expand the airless lobe beyond its normal size (due to an excessive build-up of intra-alveolar exudate).

As time passes, however, the initial appearance of these various conditions can become obscured by secondary changes. While the low-grade fever and tachycardia associated with atelectasis are initially quite reversible, simply reopening a blocked airway may no longer achieve pulmonary

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reexpansion after the onset of significant infection: The same parenchymal damage and fibrosis may result from lung infection consequent to an initial atelectasis as when the process began with severe pneumonia.

In addition, the primary pneumonia patient may soon fill his lobar bronchus with mucus, or the patient with an endobronchial foreign body causing atelectasis may finally dissolve or clear the impacted material (leaving open bronchi and varying degrees of distal consolidation that further confuse the roentgenologic evidence).

Summary: Chest roentgenography and physical findings more accurately portray "where you are" than "how you got there." Effective treatment often requires an understanding of both.

Lung Compression

One condition that clearly is different from atelectasis (lung collapse) or lung relaxation (toward its preferred resting size) is extrinsic lung compression by air, tissue, foreign material or fluid. Moderately compressed lung commonly remains aerated (but its portion of pulmonary blood flow is usually reduced): Cessation of air exchange occurs when lung is compressed at greater than atmospheric pressure during all phases of inspiration (keeping it persistently smaller than its relaxed size). Compressed lung often collapses secondarily, due to mucus blockade of the airways (this occurs especially early in cigarette smokers).

However, the parenchyma of a relaxed-size lung may remain aerated and still functional for many years after its entrapment by a thickened pleural peel. This is in marked contrast to the progressive lung destruction that commonly accompanies chronic atelectasis complicated by parenchymal infection.

Conclusion: We have learned that free drainage is usually essential for correction of any significant infection that is associated with accumulation of necrotic debris. When such infection occurs within lung parenchyma, there must usually be drainage via an open bronchus before the lung can recover.

Lung Abscess

Lung abscess may result from any necrotizing bacterial pneumonia. Predisposing conditions include diabetes, alcoholism, epilepsy, stroke, head injury, carcinoma, or aspiration of foreign material or gastric content into the airway from any cause (often while unconscious). Poor dental hygiene allegedly increases bacterial contamination of the airway from any aspiration (and thus the opportunity for lung infection and abscess).

Abscess may occur distal to an obstructing foreign body or malignancy, or even within a necrotic carcinoma. Symptoms in the latter patients, however, are usually due to the carcinoma itself, rather than the superinfection of an already necrotic cavity. Therefore, "necrotic carcinoma" patients are less likely to show septic fever and more likely to cough blood (along with the usual purulent debris), than are patients with "benign" lung abscess.

Rarely, one may also encounter a slowly resolving air-fluid level in an area of major lung infarct and necrosis: The initial roentgenographic appearance here may be that of an excavated, necrotic tumor. Only a bronchogram, fortuitous pulmonary angiogram, recent history of pulmonary embolus, or gradual clinical or roentgenographic improvement can help differentiate these two conditions on plain

roentgenogram. When in doubt, however, the diagnosis is almost surely carcinoma rather than excavated lung infarct.

Cavitary tuberculosis, or apical blebs with surrounding consolidation and fibrosis, or bronchiectasis - all may give the thick-walled, shaggy, roentgenographic appearance of excavated tumor. The aspergilloma or fungus ball occasionally found "rolling about" (with a "crescent" of air above it on roentgenogram) in a bronchiectatic, tuberculous, sarcoid or malignant cavity, is often associated with hemoptysis as well.

Reportedly, many patients with aspergilloma will eventually require a surgical resection for major hemorrhage. Direct transthoracic or endobronchial instillation of antifungal agents has often proved successful in eliminating aspergillus; apparently systemic agents cannot "reach" this (occasionally invasive) overgrowth of fungus in a warm, moist, chronic pulmonary cavity. (I have not knowingly encountered this condition in over twenty years.)

Chest Surgery Has Not Yet Outgrown Its Need for Bronchography

A bronchogram is crucial to the differential diagnosis of most chronic inflammatory lung conditions. Bronchoscopy may also be helpful for what is or is not seen, as well as the bacterial and cytologic specimens obtained. Thus bronchoscopy and bronchogram should normally be an integral part of any preoperative evaluation of lung abscess.

Why then is bronchography (simple, safe, very informative and inexpensive) so rarely taught or performed? It only requires a small catheter, a little local anesthetic, some iodine-containing contrast material, and an ordinary fluoroscope (preferably operated by a competent radiologist).

Indications for Bronchogram

A bronchogram is essential for the evaluation of segmental lung disease. It is also essential when planning lung resection or pulmonary decortication on a patient with possible bronchiectasis or persistent lobar collapse. For example, one usually cannot "get away with" upper lobe resection for carcinoma if the remaining lower lobe is so severely bronchiectatic that it will never expand or function adequately. Similarly, one ought not try to decorticate a bronchiectatic lower lobe and leave it in place.

Certainly one cannot resect a lung abscess safely from a patient with chronic lung disease without first evaluating the remaining lung. (How else could you decide how much to resect and what may safely remain?) The often "unremarkable" external appearance of a bronchiectatic lung at surgery can easily mislead the surgeon who does not have a recent bronchogram in hand.

"Modern" chest surgeons can often "get by" without performing a bronchogram because of the marked decrease in chronic inflamatory lung disease (such as bronchiectasis) that has resulted from childhood immunizations against pertussis and measles, and the wide-spread use of effective antibiotics. Improvements in anesthesia and other medications also have markedly reduced the risk of performing an unnecessary thoracotomy (but not the pain, cost and aggravation for the patient).

Admittedly, a bronchogram can be tedious and it only justifies a small fee, but it really could save many patients from a costly thoracotomy, or help the surgeon plan a more appropriate operation that might prevent major postoperative morbidity. The next time that your open lung biopsy (performed for

possible malignancy) is reported back as "inflammatory tissue," stop and consider whether a simple bronchogram might not have provided the same information more easily.

A bronchogram can usually differentiate between pulmonary malignancy and post-inflammatory lung changes, silicosis or pleural disease: Malignant bronchogenic tumors displace and obstruct adjacent bronchi as they enlarge; an inflammatory lung process is still penetrated or traversed by relatively undisplaced, open bronchi (which may, of course, become blocked by mucus, but not in the pattern expected with a single enlarging mass).

The bronchogram of an atelectatic lung will display "bunching" of bronchi: Any pleural effusion or empyema displaces adjacent consolidated lung and thus bronchi; a bronchogram can help locate the source of abnormal cytology, and much more.

Comment: Bronchography may be indicated whenever one wishes to determine bronchial anatomy, pathology, distribution, or displacement. It is usually best not to perform a bronchogram immediately after a bronchoscopy with biopsy, as blood clot near the biopsy site can mimic an endobronchial lesion or simply prevent good filling of adjacent bronchi.

Bronchogram Technique

Appropriate premedication and local anesthesia are necessary to assure patient cooperation. This means an intravenous narcotic, followed by a local anesthetic spray into nose and throat, as well as anesthetic-moistened pledgets in both pyriform sinuses (5% cocaine or its equivalent). A small catheter inserted through the nose (lubricated with xylocaine jelly) will then enter the trachea most easily during deep inspiration, with the tongue pulled forward.

Additional, more dilute local anesthetic is repeatedly injected via this catheter into the trachea (in 2cc doses) with the sitting patient alternately leaning to one side or the other (expect an explosive cough with the first two or three doses of endotracheal local anesthetic, such as dilute Dyclone, Cyclaine, 0.5% Cocaine or 2% Xylocaine without epinephrine). Local anesthesia is deemed adequate when further local anesthetic injections and movement of the bronchography tube no longer elicit cough (but keep in mind the total dose delivered and please read the drug brochure: It is often very helpful).

The small endotracheal catheter is then filled with "oily dionosil dye" (bronchography contrast material) to make it visible at fluoroscopy. Thus opacified, the catheter is guided under fluoroscopy to the desired side and positioned next to the appropriate bronchial orifice: Then someone slowly injects dye while the patient breathes quietly (and stops breathing during film exposure). Hopefully, he can also avoid coughing until the examination has been completed. The patient is rolled into lateral, prone and supine positions as indicated, to fill and visualize segmental bronchi: "Spot films and "overhead views" are taken, and the films are developed and proven satisfactory before the endobronchial catheter is removed. The patient is then encouraged to cough out dye material: Nevertheless, some dye often remains visible within the lung on roentgenogram for several days, especially in poorly ventilated bronchi. The oily carrier may persist in such areas even after elimination of its dissolved iodine, for it is the "heavy" iodine atom that makes the oily substance radioopaque.

To avoid causing hypoxia, one generally studies only one lung by bronchogram at a single procedure, especially in heavily sedated children. I find it easier to get a satisfactory bronchogram on

smaller children if they are kept asleep with an endotracheal tube in place. A small bronchogram catheter is then inserted through the endotracheal tube via a perforated elbow connector that also permits ongoing positive-pressure ventilation by the anesthesiologist.

Bilateral bronchogram can certainly be performed when this seems necessary, if the first side was well tolerated; often little additional dye is required, as merely turning the patient other-side-down lets excess dye flow across from the first side. The anesthesiologist carries out careful endotracheal suction and lung inflation after the procedure is completed (when films have been declared satisfactory), before the patient is extubated.

Treatment of Lung Abscess

Acute lung abscess usually responds to appropriate and persistent antibiotic treatment, combined with indicated respiratory therapy (which may include postural drainage, bronchodilators, expectorants and cessation of smoking). Directed transbronchial drainage (using cardiac catheterization techniques and catheters) has been reported effective for aspiration of liquid abscess content, but I have no experience with this.

The very peripheral or huge lung abscess can often be cured by thoracentesis and then closed thoracostomy through its overlying pleural layers (that have usually fused in response to this nearby inflammation). Surrounding lung tissue commonly heals promptly, once such an abscess has been drained by tube. In general, surgical resection of a benign lung abscess is undertaken only if the abscess does not respond to appropriate medical management within "a few weeks" (more or less, depending upon how the patient is faring).

The unusual abscess that requires resection, therefore, will have demonstrated an unremittingly septic course clinically, as well as stable or worsening roentgenographic findings (such as air-fluid levels or fluid densities within or about the lung). One must remember, however, that the patient with lung abscess can also have some other cause for a persistent septic course, and that chronic roentgenographically demonstrable densities about and within the lung may also take a long time to finally become roentgenographically "clear" - even when no longer symptomatic (see Chapter 4).

Tube Drainage of Lung Abscess or Lung Cyst

A delayed two-stage approach to tube drainage of a lung abscess (or massive pulmonary air cyst) was sometimes utilized in the past; the first extrapleural stage promoted "pleural symphysis" (interpleural adhesion) by placing an irritating iodoform-gauze pack just outside of the intact parietal pleura (as nearly over the abscess as possible). Closed thoracostomy was then performed through this site, several days later.

Contamination of the free pleural space by pus or air was thus avoided (in the often poor-risk patient), at a cost of several days delay in drainage. With intrapleural spill of pus from tube drainage of such a lung abscess always unlikely, and no longer the potential catastrophe that it was before effective antibiotics became available, such a delay for staged transpleural drainage of toxic abscess contents usually seems unnecessary: In fact, I have not utilized this approach for more than 20 years.

Tube drainage of a huge pulmonary air cyst (to attempt its suction collapse) can lead to major pneumothorax (with further deterioration of the often already marginal pulmonary function), so a two-

stage approach designed to avoid causing pneumothorax might sometimes seem warranted. Emphysematous lung cysts are commonly multiloculated, however, which means that they cannot be obliterated by an intraluminal chest tube on suction anyhow (so here we describe a hopefully safer way to perform a normally useless procedure).

Ruminations: Perhaps meticulous bronchoscopic occlusion of one or more specific small bronchi feeding a massive emphysematous bleb will be used to promote its collapse in the future. Although an endobronchial approach should be more successful than a chest tube thrust through the chest wall into the cysts, one might still be defeated by air access via collateral intralobar passages, or by distal infection, or displacement of the "cork" that was used.

Nonetheless, the idea of instilling a little powdered antibiotic distally and/or otherwise occluding the appropriate airway may deserve laboratory investigation (one might try an irritating "tetracycline tampon," or perhaps even wedge a small endobronchial end-hole suction catheter into or through the appropriate segmental orifice for ten days, or merely electrocoagulate, or physically or chemically denude the appropriate endobronchial area. If nothing else worked, one could always insert a peanut; we know that will work if it remains in place for a few days).

Comment: The patient who is usually considered for excision of huge lung cysts has marginal pulmonary function and thus tolerates any complications poorly. At present, only thoracotomy can reliably eliminate such bullae (by resection or direct suture or staple obliteration): Unfortunately, with generalized pulmonary emphysema, the risk of thoracotomy can be high and any benefit fleeting (as relentless pulmonary destruction "marches on").

Chest Tube Suction Helps Obliterate a Lung Abscess

Once a transthoracic drainage tube has been placed within a lung abscess, it is treated as a regular chest tube, except that early "high" suction is often very helpful in promoting closure of any intrapulmonary cavity that has some ongoing source of air. Air entry into the abscess cavity through an open bronchial connection may prevent significantly subatmospheric pressure from developing within the cavity during inspiration (thus no upward tidal at the water seal straw): Under these circumstances, there is little reason for the residual intrapulmonary pocket to "pull itself together."

It seems likely that a lung abscess won't form or persist if there is a large open bronchial communication that would permit easy drainage of its fluid content: Thus we must assume a small bronchial communication, and further - that such connection is either one-way or intermittent. If a small bronchus is also severely inflamed, one-way flow (during inspiratory widening only) can readily lead to air-trapping, with continued enlargement of the abscess cavity (see earlier discussion of incomplete bronchial occlusion in this chapter).

Bronchodilators and antibiotics therefore make an important contribution to closure of such a cavity, and "high" suction on an indwelling chest tube or catheter, is useful for staying ahead of the "one-way" air inflow that would otherwise tend to keep the cavity open. If a lung abscess cavity is not obliterated by seven to ten days of high suction, the tube can be cut off and handled as an empyema tube.

Clinical judgment is indicated here, for the smallest possible cavity around the tube tip heals soonest, so do not discontinue suction prematurely. "Prematurely" would be confirmed if the abscess

cavity enlarged significantly (on roentgenogram) during a trial disconnection of the chest bottle suction. (Although I have not encountered significant bleeding from a chest tube placed within a lung abscess, this may be a legitimate worry until the tube has been withdrawn.)

Case Report: Tube Drainage of Infected Echinococcus Cysts

A 5-year-old Caucasian Alaskan child had three huge bilateral lung abscesses from secondarily infected Echinococcus cysts. A separate closed thoracostomy into each abscess allowed copious free drainage and rapid obliteration of all cavities: With no air leak, simple water seal drainage was sufficient. As soon as full drainage was established, this small chronically ill "failure-to-thrive" patient began to thrive.

Incidentally, sylvatic Echinococcus granulosus lung cysts of the North American type are allegedly devoid of significant anaphylactic risk with rupture, and also unlikely to recur if spilled at surgical removal. Apparently, this is in marked contrast to the Greek version of this disease. I am informed that sylvatic cysts are commonly seen and ignored at the Alaska Native Medical Center (or merely aspirated by needle without ensuing complications).

The only time an Echinococcus cyst has a diagnostic appearance on chest roentgenogram is when there is early separation of the cyst wall; this creates a smooth, crescent-shaped rim of air outside of the membrane. Lacking this finding, only multiple additional cystic areas in liver (plus epidemiologic likelihood of this infection) would make one feel comfortable about not removing such a dense, discrete "coin" lesion from the lung.

Chest Exploration for Lung Injury

Most wounds of lung (by knife, rib or bullet) that reach the hospital in reasonably stable condition, can be handled by simple closed thoracostomy. After first placing an anterior mid-clavicular line, second interspace chest tube (at least #28 French in the normal sized adult), a second large tube may be placed through a mid-axillary fifth interspace site to improve the evacuation of blood from such a patient. Major ongoing blood loss (or rarely, massive air leak) may require direct surgical correction at emergency thoracotomy.

Dealing with Post-traumatic Bleeding from Lung

At thoracotomy for ongoing excessive blood loss after lung injury, the major source of bleeding may be chest wall (intercostal arteries at systemic pressures) rather than lung (low-pressure pulmonary vessels): Occasionally, however, it is lung that bleeds freely. A self-inflicted gunshot may have blown off the lingula, for example, or shattered it badly.

In this case, simply lift out the shot-off portion at thoracotomy, secure bleeding and major air leaks with big chromic mattress sutures, irrigate carefully, check for any obvious bullet or other foreign material (without causing additional injury by digging for these), inspect the mediastinum and diaphragm for injury, place at least two chest tubes and close up.

If an intercostal artery is found to be the source of bleeding at thoracotomy, one need not also "repair" nearby holes through lung, unless these are still bleeding actively or leaking large volumes of air. In addition to clot-sealed holes in lung or a cleanly shot-off lingula, a considerable variety of

shattered lung can be encountered.

I generally remove only very loose or devitalized pulmonary tissue, preferring to control major parenchymal bleeding and air leak with a few interlocking, 0 chromic sutures (followed by minimal debridement and swift closure), but that is a matter for individualization. Certainly, major proximal lung or hilar injuries will require meticulous emergency repair or resection (but these "potential patients" rarely reach the hospital alive).

Two or three times, however, the large mattress sutures placed to control external blood loss from torn and bleeding lung, had to be removed rapidly when it became apparent that the blood loss had merely been redirected into the airway (nearly drowning the patient). One need not worry about "silent bleeding" in this situation, however, for the anesthesiologist always shouts early and loud.

On occasion, emergency control of ongoing severe hemorrhage from torn lung can only be achieved by placing large vascular clamps (or even large Kelly-type clamps, if nothing else is available) across or proximal to the visible lung injury (if repair is not immediately possible), before transporting the patient (with clamps in open chest) from the emergency area to the operating room for more definitive repair or resection.

Although leaving behind numerous large chromic mattress sutures and much possibly devascularized, ripped and ragged lung is always a legitimate cause for concern, it is usually best to treat critically injured patients only for actual, rather than potential, problems. Surprisingly, these patients usually heal uneventfully (or else die of associated head injuries with the chest "repair" still intact).

I have not recognized an air embolism (due to bronchovenous communication) although it has been reported with lung injury. Common sense suggests that low ventilation pressures are preferable with any damaged or recently repaired lung; perhaps low ventilation pressures can also reduce the incidence of this catastrophe (which allegedly should be treated with a steep, head down position during cardiac massage).

It is also possible that air embolism only becomes likely in the presence of combined injuries to large vessels and airways in the lung hilum (which are often fatal before hospital admission anyhow). Smaller lung vessels are soft and likely to collapse if air pressure around them exceeds their intraluminal blood pressure, so they are more likely to let blood escape than imbibe air, except with side-wall injuries of an adjacent airway and vessel (which certainly could allow air into the left side of the heart via the pulmonary veins).

Persistent Air Leaks

Persistent air leaks after lobectomy usually come from transection of emphysematous lung at an incomplete interlobar fissure. I have recently paid more attention at thoracotomy to preventing moderate air leaks from raw lung surfaces, sometimes using 2-0 chromic figure-of-eight or mattress sutures to reduce air leak and speed expansion of the residual lung: The consequent earlier cessation of air leak seems to have decreased pleural fibrosis and inflammation, thereby benefitting postoperative pulmonary function. Whether such early advantage persists is less clear: Even marked pleural thickening on chest roentgenogram usually diminishes as months pass.

Open Fissures

Superior segment and lingula are sometimes separated from the rest of their lobe by an incomplete fissure. More commonly, however, anticipated interlobar fissures are "absent" (or obliterated by scar and inflammation) when they would have been most helpful in expediting resection. Lung tissue planes must then be developed by segmental resection techniques.

The rather common absence of complete (open) fissures in the adult undergoing lung surgery is in marked contrast to the open major fissures uniformly encountered at infant thoracotomy for non-inflammatory disease: It seems likely that the later absence of open major fissures can often be attributed to post-inflammatory changes.

Indications for Segmental Resection

Segmental resection may be indicated for any benign pulmonary disease requiring removal of less than an entire lobe of lung, especially when preservation of lung function is critical. For many years, bronchiectasis and tuberculosis (now uncommon and usually "non-surgical" diseases) were the principal indications for segmental resection.

General Comments on Segmental Resection Technique

Irregular segmental borders commonly are displayed as faint lines crossing the lung parenchyma under the transparent visceral pleura. It is simple to begin a sharp division of visceral pleura and lung parenchyma along these lines. However, efforts to carry this sharp dissection along segmental surfaces down to the hilum frequently become anatomically confusing, and "stir up" bleeding.

Fortunately, peripheral intersegmental bleeding (away from the hilum) is generally from very small pulmonary artery branches or somewhat larger intersegmental veins: When troublesome, this bleeding normally stops if a large sponge remains pressed against the raw lung surface while dissection proceeds elsewhere. The filmy coagulum that forms within five minutes under this sponge will also close numerous tiny air leaks from the raw segmental surfaces.

Dissection can usually proceed peripherally from the origin of a segmental bronchus without a great deal of difficulty. Segmental borders become especially easy to identify if lung is momentarily deflated as the segmental bronchus is occluded, for the blocked segment then remains deflated and visibly unaffected by ongoing ventilation of the remaining lung.

During segmental resection, the segmental pulmonary artery branch (which lies adjacent to its bronchus) is best divided early (and its stump mobilized away from the bronchus by sharp dissection), for this moves the larger proximal artery "out of harm's way" while the segmental bronchus is further mobilized, stapled and transected, or divided and sutured.

The distal end of the transected bronchus is grasped with a Kelly clamp after stapling (or while the short proximal stump is closed with fine polypropylene sutures). Traction on the Kelly toward the hilum can provide useful countertraction for a sweep with the blunt side (not tip) of an exploring finger along the intersegmental plane toward the pleural surface (a fingertip usually penetrates pulmonary parenchyma rather than following intersegmental planes).

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Progressive traction on the divided segmental bronchus away from the hilum will sequentially tense all remaining venous connections, making it clear which vein should be divided, and where. Any strong traction on the segmental bronchus away from the hilum should always be balanced by wide-sponge counterpressure on hilum and lung (as finger counterpressure will easily tear into lung).

Thus delineated, a segment can be torn cleanly away from remaining healthy lung, especially if it has already been freed from surrounding lobe(s) on all but one major surface, through prior sharp dissection and blunt "fingering." Non-threatening bleeding is then controlled by gentle pressure with a large sponge, as described above.

When residual lung will be "small" (compared to the pleural cavity it must fill), one avoids any parenchymal ligation or stapling that would "bunch" raw surfaces and further diminish lung volume. Although open fissures and raw segmental surfaces allow maximum lung expansion, excessive air leak from raw lung surfaces can reduce or eliminate that benefit, by delaying or interfering with lung expansion (so once again, "judgment is required").

Wedge resection or lobectomy usually leave less raw lung surface behind than segmental resection, and less raw surface usually means a diminished postoperative air leak of shorter duration, thus fewer in-hospital days after surgery. An occasional ambulatory postoperative patient can safely be sent home with water seal bottle in hand to await closure of a persistent air leak (see Chapter 6).

Such outpatient care is not a good idea, however, if the residual lung tends to remain much smaller than its surrounding pleural cavity: These lungs are best kept maximally expanded by "high" chest bottle suction (sometimes even for two or three weeks) until the air leak ceases, or the lung remains fully expanded despite opening the chest tube draining this "bronchopleural fistula" to the atmosphere. (Obviously, a simple water seal cannot maintain a hanging fluid column to apply suction in the presence of air leak.)

Thus, it is normally not wise to send a post-thoracotomy patient home on simple water seal if he has a persistent pleural space with air leak, for this could allow the shrunken or surgically diminished lung to become encased by progressive pleural thickening. Lung entrapment in its "squatting position" and late empyema are likely to follow if high suction is discontinued before the remaining lung is "full, up and stuck."

Indications for Wedge Resection

Wedge resection refers to removal of a "more or less" wedge shaped portion of peripheral lung parenchyma, along with its overlying visceral pleura. Small or large pieces of lung can be wedged out: These often originate from more than one lung segment, and occasionally from two separate lobes. Effective closure of the pulmonary defect after wedge resection depends upon staple or suture compression of transected vessels and airways between opposing layers of visceral pleura.

As a simple, rapid, and minimally traumatic procedure, wedge resection is preferable to more extensive surgery (such as segmental resection, lobectomy or pneumonectomy) whenever it is likely to be equally safe and effective. Wedge resection is commonly utilized for operative closure of air leak following spontaneous pneumothorax, for biopsy of peripheral lung and pleura, and for indicated removal of a single or multiple metastatic malignancies (if wider excision is not possible or safe or likely to increase curability).

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Computed tomography of both lungs is very helpful in determining the likely extent of pulmonary metastases preoperatively; one must also inquire specifically about headache, personality change or trouble with balance, and order a brain scan and computed tomography of the head if there seems to be any indication. Previous x-rays should be inspected, and palpation for detectable neck nodes is a "must."

At times, repeating a chest roentgenogram during a moderate Valsalva maneuver will demonstrate significant change in the size of one or more "coin" lesions: This suggests that they may actually be pulmonary vascular anomalies. A pulmonary arteriogram may then be appropriate (if doubt persists).

It is usually also a good idea to request and view a repeat roentgenogram at the time of admission for chest surgery, for an occasional definite "coin" lesion may turn out to be a psuedotumor that simply vanishes in the interim - similarly, a new lesion or effusion can develop in a remarkably short time.

Sternotomy appears to be an effective approach for bilateral pulmonary surgery at "one sitting." I have minimal experience with lung resection via a sternotomy, and therefore do not consider it as convenient for access to lower lobes or hilum as a posterolateral approach. However, I do believe those who allege that they can achieve decent surgical exposure using close monitoring, double lumen endo-tracheal tubes, tilting the patient for exposure, packs behind the lungs, and appropriate retractors.

It is well to recall that a sternotomy incision should not be opened too widely, nor should the sternal retractor be placed so that it spreads mainly the upper half of the divided sternum, as such efforts can result in first rib fracture with possible brachial plexus injury (previous shoulder area trauma and fibrosis increase that risk).

In recent years, I have come to prefer wedge resection for small solitary primary malignancies of lung that are peripheral (subpleural) in location. My impression is that the "five year cure rate" for these patients is unchanged or improved, while morbidity and hospital stay are certainly diminished (although I have never had a hospital death with any elective pulmonary resection). Undoubtedly my "sample size" is not statistically significant (nor am I a significant statistician), but my rationale for wedge resection of a peripheral or sub-pleural carcinoma - in preference to lobectomy or pneumonectomy - is as follows:

Anecdotal: Over the course of a decade, I wedged out 3 separate solitary lung carcinomas from both lungs of one elderly emphysematous lady (still "well," no evidence of recurrence, after more than ten years). I can recall two or three other patients in my practice, also with limited pulmonary reserve, who underwent "curative" wedge resection for peripheral lung malignancy, first on one side and then, some years later, on the other;

Logical: On a number of occasions, I simply could not justify bilobectomy or pneumonectomy for a small peripheral "coin lesion" carcinoma of one lobe that had crossed an obliterated major fissure to also involve the adjacent lobe; under these circumstances, I simply wedged the lesion out from both lobes;

Intuitive: It seems unlikely that a small cancer very near one margin of resection (the pleura) will have a significantly increased likelihood of cure if one extends the margin of resection on the pulmonary side by removing the entire lobe or lung within which that malignancy originated;

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Supported by Reported Results: Published figures suggest that the five year cure rate with wedge resection for carcinoma of lung is equal to or better than the cure rate following lobectomy or pneumonectomy. Of course, a counter-argument would be that "only the little early ones get wedged, so the cure rate for wedge should be better". This is not the entire answer, however, for some surgeons "religiously" perform lobectomy for all small lesions while others strongly prefer to "wedge".

Strengthened by Compatible Hypotheses: There is allegedly some evidence that certain anesthetic drugs (e.g., nitrous oxide) may temporarily depress the immune system, thereby potentiating intraoperative dissemination of metastases. Similarly, intraoperative blood transfusion is alleged to have an adverse immunological effect on the recurrence rate of carcinoma, while postoperative transfusion supposedly does not; this difference in the effect of blood transfusion also suggests that manipulation of the tumor at surgery may be a significant risk factor in tumor recurrence.

It is very likely that there will be less anesthesia, less intraoperative blood loss, no transfusions, and also less manipulation of a tumor, during a brief wedge resection than in the course of a longer-lasting lobectomy. In addition, wedge resection should carry less risk of disseminating tumor than a more limited incisional (or even needle) biopsy performed in anticipation of possible lobectomy; and finally:

Simple Rapid Procedures Usually Expedite Recovery: Patients subjected to expeditious, "less major" surgery, simply "do better." Just because a procedure is easier on the patient and simpler for the surgeon does not mean that it is necessarily bad. Yet many surgeons have apparently concluded that wedge resection is too easy to give comparable results to lobectomy, and further, that performance of wedge resection for any indication as serious as "Cancer!" must be a sign of weak will, an impending golf game, or plain incompetence.

Conclusion: A malignant lung lesion can sometimes be "cured" by a lesser excision than lobectomy or pneumonectomy: Such lesser procedure may still be advantageous even when pulmonary function would permit more extensive resection (especially if a more extensive resection might simply leave the patient with less pulmonary reserve, while offering no evident countervailing benefit).

At present, lobectomy or pneumonectomy often seem indicated for centrally located, potentially curable, malignant lesions (however, there is currently also increasing interest in careful cautery excision of more deeply located "coin lesions," but I cannot comment on that technique). While it remains to be determined which, if any, surgical procedure can best eradicate a particular lung cancer, wedge resection often appears to be the "better procedure."

Practical Aspects of Wedge Resection Technique

Depending upon the location and size of the lesion, wedge resection can often be performed through a small chest wall incision that allows surgical access only to whatever lung can be delivered into the wound. A carefully placed, 7 to 8 cm "minithoracotomy" (through intercostal muscle or via the bed of an 8 to 12 cm resected rib segment), using two Gelpi retractors or an infant- or child-size Finochietto retractor, can serve nicely for simple lung or pleural biopsy procedures. As proximal control of major vessels will be difficult or impossible through such a minithoracotomy, one may prefer to place the incision in a fashion that allows easy and rapid extension if a "technical problem" should arise.

A minithoracotomy skin incision is often shorter than the underlying cut through fat and muscle (or the resected segment of rib), for a skin incision stretches well and is easily moved about for short distances over the more fixed, underlying tissues. Perhaps surprisingly, a short skin incision vigorously overstretched usually leaves a less noticeable scar than a longer cut.

When removing an isolated peripheral lung nodule that may be malignant, one must resect an "adequate" margin of normal lung along with the "coin" lesion (while minimizing its palpation): I prefer a 2-0 chromic suture line for firm compression of transected vessels and air passages between layers of visceral pleura, as stapler heads may interfere with the continuous evaluation of margins that is needed during tumor excision, especially through a minithoracotomy.

Other Pertinent Details With Wedge Resection

Grasp the lesion so that fingertips and thumb "meet" by compressing lung parenchyma deep to the mass. Place one or two curved Potts vascular clamps around the lesion from the lung edge (clamps go deep to fingertips that were placed beyond the palpable edge of the lesion, to secure about a one centimeter margin of normal lung).

If proximity of large vessels or bronchi (or, conversely, a poorly circumscribed lesion or unexpectedly deep tumor extension) make an adequate margin of resection seem unlikely at this point, it is usually better to proceed with lobectomy (or segmental resection), rather than struggle to complete a wedge resection with probably inadequate margins: Excessive manipulation or accidental transection of tumor during close dissection could easily leave the patient with a worse "tumor burden" than he had preoperatively.

On the other hand, during resection of multiple metastases, or while performing "tumor debulking" procedures in association with effective chemotherapy, one tries to minimize the loss of useful lung tissue; then it is not always possible or necessary to maintain margins that might be appropriate when "going for a surgical cure."

After satisfactory Potts clamp placement (or stapling), the excluded lung lesion, with its surrounding parenchymal margin, is sharply excised. One can usually avoid slippage of raw lung edge through the vascular clamp (following lesion removal) by minimizing traction on the clamp and also leaving a small wad or cuff of lung tissue on the biopsy side of the vascular clamp, especially at the heel of the clamp (well away from the lesion), where lung tends to slip through the clamp most easily.

If a portion of raw lung edge should slip through the Potts clamp, a few rapid horizontal-mattress sutures (or a basting suture and whip stitch) will resecure the slipped section, pending routine completion of closure. While awaiting the pathologist's "frozen section" report on the lesion and its margins, a 2-0 chromic, back-and-forth, progressive basting suture is placed on the hilum side of the vascular clamp (to prevent retraction of divided vessels and airways into lung parenchyma until they can be oversewn).

The clamp is then removed, the basting suture "snugged up" (actually the lung is simply pushed centrally from both ends of the tightened-and-now-straight chromic suture), and an over-and-over whip stitch returns to where the basting suture began, so that the two ends of this chromic suture can be tied firmly together. Raw lung edge above the Potts clamp may also be electrocoagulated prior to placement of the returning whip stitch, if at all concerned about tumor at the margin of resection (as during the

resection of metastases).

A surprisingly large piece of lung can be removed expeditiously by wedge resection. On occasion, for rapid resection of a bulky, infected or bleeding, peripheral but incurable carcinoma with hilar involvement by large nodes, I have even wedged out the distal half of an upper or middle lobe: Such a large wedge is usually not indicated or recommended, however, as it may lead to later air-fluid levels in the upper chest.

Inflammatory postoperative pleural obliteration has prevented these delayed apical air-fluid levels from leading to a major infected pneumothorax. Presumably a small, reopened bronchus serves both as the air source and drainage tube for such an apical pocket, which commonly causes fever and malaise until it clears. Undoubtedly, as more lung is squeezed within heavier interlocking-chromic sutures, increased tissue necrosis and healing delays are likely to be encountered.

Even the most carefully planned surgical intervention for more advanced malignancy may be fruitless, of course, but an occasional patient who is "otherwise in good shape" may derive months or even years of benefit from palliative chest surgery, especially when low-grade malignancy or metastases are limited to the chest.

It is often difficult for a young and healthy surgeon to decide whether some extra months or even a year of life can adequately compensate for the trouble, pain and cost of even a well-designed, competently performed "palliative" surgical procedure: Patients usually treasure their "extra" time, however, provided they can remain functional and relatively pain-free. As with most things, time takes on more value when it is in short supply.

Wedge Resection in Spontaneous Pneumothorax

Not uncommonly, in the course of a routine thoracotomy, one or more "ballooning" lung cysts may be encountered that seem to compress adjacent lung or pose risk of future pneumothorax. One can grasp such a cyst with a large Kelly clamp and twist, fold and gather it optimally, then staple or gently oversew the entire wad (with one or several rows of atraumatic 2-0 chromic suture, taking large bites and pulling the swaged-on needle carefully through visceral pleura in order to avoid excessive postoperative air leak).

Although isolated intact blebs or cysts can simply be ligated at their neck, such ligatures have an annoying tendency to be forced outward or roll off entirely during positive-pressure ventilation, which can lead to more air leak if the bleb or cyst was damaged at the level of original ligation. One can also "whip" a chromic stitch along the occasional lobar edge that displays a whole line of translucent, small or medium-sized blebs, "in hopes" that the natural tendency of an air cyst to enlarge will thereafter be overcome by scar.

When exploring the chest for persistent air leak after spontaneous pneumothorax, I have usually resected enough lung tissue from the ruptured apical bleb area to permit a suture closure of "more normal" lung tissue: This has provided material for microscopic examination and various cultures (although such studies have never been of much help clinically).

Steroids and Reopened Air Leak

A patient on high doses of steroid may develop a new air leak requiring another chest tube, ten to twenty days after wedge resection. Presumably, the absorbable sutures lost strength before adequate healing occurred. After two such experiences, I switched to heavier, 2-0 or 0 chromic suture for over-sewing the base of the wedge resection, and that seems to have eliminated this problem (presumably staples or a longer-lasting monofilament absorbable suture would be equally or more satisfactory).

Lung Cysts

Some textbooks of chest surgery formerly recommended opening larger cystic areas of lung to oversew their bronchial air supply from within. Having seen this tried a time or two in vivo, and having examined pathology specimens with such a procedure in mind, I cannot believe that any experienced surgeon would recommend that approach.

Certainly the average multiloculated emphysematous lung cyst has only one semi-strong tissue layer for suture, and that is its intact visceral pleura. Once this layer is opened, one encounters blood and froth coming from every direction, with no tissue in sight that can withstand a suture or even ligation.

The originator of this idea may have tried it successfully once on a young adult with the rare congenital giant pulmonary air cysts (which admittedly resemble severe emphysema at first glance). These large, congenital air cysts may be multiple and so distort a lobe or lung that it appears practically destroyed.

Yet, after a few giant cysts have been shelled out easily (by incising visceral pleura and then blunt finger dissection), the no longer attentuated lobes will appear practically normal. Surprisingly, this "finger enucleation" can be accomplished without significant air leak or blood loss (merely "unroofing" might be just as effective, with or without suture of the small bronchial connection).

These huge simple air cysts have been very unusual in my practice, however. I would guess that I have only encountered four cases: The first was treated by lobectomy, as I did not realize that the otherwise normal lobe was simply attenuated around its large air cysts.

Admittedly, this is a tiny series from which to recommend "shelling out" congenital lung cysts, so I simply mention this therapeutic alternative (but, again, advise against its use in older adults with emphysematous blebs, for these can neither be shelled out nor effectively closed from within after they have been opened widely).

Summary: Emphysematous bullae (multiloculated air cysts) should be wedged-out of the lung or oversewn if surgically significant. The rare large simple "congenital" air cysts of lung are easily "shelled out."

Avoid Mobilization of Unresectable Apical Tumors

When an apical mass lesion is likely to be unresectable, it is well to confirm this fact before all of its adhesions to the chest wall have been divided, for if mobilized, the weight of such a lesion usually prevents upward reexpansion of lung (leading to a subsequent apical "space problem," and high risk of empyema with "permanent" tube drainage).

As an alternative solution, one might even consider glueing or sewing the lesion back to the upper ribs (I do not know if that has ever been attempted, and if it has I doubt any surgeon would admit to it). A "copious" wedge resection of the tumor mass could also be less risky than dissection through a hilum invaded by malignancy, but this problem is best avoided by careful palpation of the mediastinum and hilum before the tumor is mobilized.

Middle Lobe Torsion

When closing the chest, be sure that lung lobes are normally oriented and not twisted. A floppy, fully mobilized, middle lobe might best be secured to its adjacent lobe before closure, to prevent torsion and consequent gangrene. On two or three occasions, I have helped a young surgeon take a desperately ill patient back to surgery to remove a "rotten" middle lobe and evacuate the foul stinking empyema that was caused by such a volvulus after "routine lobectomy."

I therefore "tie" any mobile lobes together across an open fissure (whenever this seems necessary and possible), to prevent such a disaster. "Instant adherence" of adjacent pulmonary lobes can most easily be achieved by gathering small hemostat-held directly-opposite fragments of pleura and lung within a single tight 3-0 or 4-0 ligature at two or three separate sites (having first arranged the two remaining lobes in their best relation to each other for optimal filling of the pleural cavity).

CHAPTER 14: THE CIRCULATION

Blood flow depends upon the pressure gradient;... Nature abhors a vacuum;... She is not too fond of turbines either;... How elastic should our arteries be?... The intraaortic balloon pump;... Elastic and muscular arteries contain a pressurized reservoir of blood;... Venous return;... Left ventricle works harder;... Left ventricle may stretch atria;... Storage of excess blood;... Liver congestion;... Case report: Acute liver congestion;... Lung congestion;... Intrapleural pressure changes can affect cardiac output;... Evaluation of blood loss in general;... Estimating the amount of an acute blood loss;... Recovery or replacement of intravascular fluid losses;... Fluid versus blood replacement for acute blood loss;... Untreated blood loss;... Tissue edema;... Case report: Pulmonary edema from mobilization of dependent edema;... Posttraumatic edema increases fluid requirements;... Mobilization of edema reduces fluid requirements;... On fluid losses into dead and dying tissues;... Anaphylaxis, edema and epinephrine;... Venous oxygen and venous pressure measurements;... Changes in central venous oxygen saturation usually reflect changes in cardiac output;... Rigid blood vessels have a relatively fixed volume;... General aspects of intravenous fluid therapy;... Dextran and albumin;... Oliguria;... Mannitol diuresis;... Diuresis;... Lasix diuresis;... Hypotension and minimum adequate perfusion pressure;... Hypotensive control of blood loss after cardiopulmonary bypass;... Hypertension and blood volume after cardiopulmonary bypass;... Vasoactive medication is always administered into the side-arm of a continuously flowing primary intravenous line; ... To err is human, to forgive (without knowing what happened) is stupid;... So cultivate a little paranoia;... Case report: Incorrect label;... Calcium administration corrects hypotension;... Solumedrol for hypotension;... Cardiopulmonary resuscitation after sternotomy;... Outline of an approach to post-bypass hypotension;... A nurse check-list for viewing the poststernotomy chest roentgenogram.

Blood Flow Depends Upon the Pressure Gradient

Just as water runs downhill, so "blood always flows from higher toward lower pressure zone if unimpeded." Variables such as artery size, flow characteristics of blood, pulse transmission through fluid and vessel walls, cardiovascular resonant frequencies, elastic and muscular responses of individual vessels, angle of branching and difficulties in nondisruptive flow measurements, all add complexity without changing the laws of physics.

Nature Abhors a Vacuum

Blood will only circulate if it is pushed or pulled (or both). If our circulatory system relied solely upon "pulling" blood around we would need a stiff non-collapsible venous system: This would mean the loss of our important, variable volume "venous reservoir".

A non-collapsible venous system can certainly be designed: In fact, our own internal jugular vein is a good example of this, for it is held "wide-open" by its fixation to the surrounding neck fascia. So if you happen to cut into this vessel accidentally at scalene node biopsy in a sitting patient, you are unlikely to ever forget this glistening blood free fascial space – a channel which won't at all look the way you'd expect a big vein to appear from the inside ("lots of blood splattering all over the place").

On the only occasion that I opened this "glistening tissue plane" at scalene node excision, only a little "slurp" told me that all was not well: The patient was immediately dropped to a horizontal position while I kept the hole compressed. Then I asked him to "Bear down. Please!" His Valsalva maneuver pushed a lot of bloody froth back from the heart side to well up from his internal jugular vein into my waiting suction. Following vessel repair, the node biopsy was completed uneventfully, and the

patient did well until the malignancy "caught up with him."

I am aware of several deaths by venous air embolism during scalene node biopsy under local anesthesia in the sitting position: Some of these were only recognized at the end of the procedure, on removal of the drapes - so try to engage the patient in conversation under these circumstances; it helps them relax and keeps you "in touch."

If our internal jugular vein were free to collapse rather than being externally supported, the hanging fluid column, from head down to heart could cause intermittent venous flow, or a "venous chatter" or "water hammer" effect as venous walls were repeatedly approximated by markedly subatmospheric intraluminal pressures. At worst, the head-to-heart siphon might bring these hypothetical soft venous walls together so tightly that they could block venous outflow from the brain.

A giraffe probably has low arterial pressure to the brain while munching tree tops: This should make him particularly susceptible to any simultaneous venous pressure elevation that could further reduce the pressure gradient between brain arteries and veins. Permit me to guess, therefore, that the giraffe has a particularly well stabilized deeply located internal jugular vein that always remains wide-open in the "heads up" position (and that this siphon aids brain perfusion).

If we were redesigned so that our entire venous return depended upon some sort of central venous suction system, however, a firm exoskeleton might also be useful, to assist perivenous tissues in applying appropriate countertraction. Of course, the non-collapsible venous channels necessary for a suction-driven blood circulation could also pass through our long bones (but that would displace marrow and require extensive redesign of the joints).

Many problems come to mind in trying to design a subatmospheric venous return system, including the possibility of intravenous bubble formation ("outgassing") from venous blood subjected to excessively subatmospheric ("siphon") pressure. How does a giraffe get around that one? Furthermore, inadvertent air entry at a site of tissue injury could readily overload or block such a venous suction system with froth.

Air flowing into an accidentally opened vein would also sweep away any blood that might otherwise clot at the opening: If "self-sealing" fluids from within the vein could not block further air entry at the wound, then any "circulatory sealant" would have to originate from tissues around the injured veins.

There might also be difficulties in maintaining tissue hydration, for tissue fluid would tend to be pulled back into the venous (subatmospheric) end of the capillaries, unless a strongly reversed osmotic gradient was created (toward tissues, away from capillaries); that, in turn, would unpredictably increase fluid losses from the circulatory system (as would minor changes in the subatmospheric venous cir-culatory pressure).

It is easy to see why subatmospheric venous return systems never became popular with Mother Nature. Therefore, it appears likely that significant "pulling" on blood cannot be the main impetus to a useful blood circulation (except that one could perhaps design a no-heart or no-pump circulatory system that was based upon slow rotation of the entire organism, or intermittent inversion, to allow gravitational pull to draw blood in a circle around an appropriately-valved series of conduits. Evolutionary theory suggests that dizzyness would be an uncommon trait amongst such "spinners" or

"flip-flops"). Well then, what options are available if we choose to "push" blood through the circulatory system?

She is Not Too Fond of Turbines Either

A turbine or other rotating pump maintains forward fluid momentum by very frequently or continuously pushing on a moving column of fluid. In a closed-circle system, such a fluid column tends to continue "circulating" by its own inertia: Once it is "up to speed," therefore, the pump only has friction to overcome.

Cardiac patients often comment that it takes them a few minutes "to get started," but that they can thereafter perform significant physical activity. Similarly, athletes find it beneficial to "warm up" prior to vigorous physical exertion. One gathers that a steady state of increased cardiac output becomes easier to maintain when adjustments in preload, stroke volume, heart rate and afterload have been completed.

An intermittent "fill and empty" cardiac-type pump must overcome repeated inertial losses as it "ratchets" the entire blood volume forward with each stroke. Such "to-and-fro" pump action depends upon one-way valves to prevent the higher pressure "output side" from rapidly refilling the pumping chamber, for in the absence of valves, input and output pressures would always remain equal, unless forward flow was supported by "cardiovascular peristalsis" or some type of high frequency, more or less continuous pumping that did not allow time for reflux (e.g., a turbine).

A continuous flow "turbine heart" would require relatively rigid arteries, to allow demands for increased output to be met by speeding up the pump (while also bringing additional peripheral channels to "full flow" by opening more "gates"). With appropriate pump design, power source, tubing arrangement, fluid flow characteristics, and an adequate venous reservoir, a widely variable flow rate might thus be achieved without greatly altering the arterial pressure.

Our own to-and-fro hearts, complete with valves, have a stroke volume that can approach one percent of our blood volume. We achieve pulsatile blood flow as a result of intermittent addition of incompressible fluid to full but expansile pipes. Blood vessel elasticity moderates blood pressure changes and decreases inertial work: This permits the flowing blood to transfer more of the total kinetic energy in each heart beat onward to the tissues.

A variable peripheral resistance is crucial to sustaining forward blood flow as these distended arteries rebound from overfilling, for with inappropriate peripheral resistance, there could either be an insufficient or overly rapid decline of arterial pressure after each heartbeat: The peripheral resistance must also be able to compensate for the heart rate, especially with a slow pulse.

If our arteries were entirely rigid, each heart beat would have to accelerate intra-arterial blood to high speed (requiring a great deal of "inertial" work) as the arterial pressure went up to whatever very high level would be necessary to obtain the appropriate forward movement (unnecessary pressure work). Arterial pressure would then drop to about zero while the heart refilled from the venous side for "another squirt." It seems evident, therefore, that the increased stiffness of aging arteries will require the heart of an older person to perform more work to achieve the same "normal" cardiac output.

As we replace an increasing percentage of any individual's arteries with bypass grafts, perhaps we

should evaluate the elasticity of these large vessel substitutes: If they were also durable, more elastic grafts would reduce peak systolic pressures and thus cardiac work, as well as "wear and tear" on other arteries. It might also be interesting to investigate the "mechanical" role of bone-marrow fat in long bones and venous bulges within flat bones, to see how much "elasticity" our intra-osseous circulation contributes.

The elastic qualities of our larger arteries derive from the relatively stiff (especially as we age) intimal layer, the springy media, and the rather expansile adventitia. Significant medial degeneration or necrosis leaves an unsupported overstressed intima that is liable to tear and release blood at arterial pressures into the potential space between stiff intima and expansile adventitia. The almost immediate, very painful, result of a pulsatile, high-pressure blood column dissecting into this space is the creation of a small blood-containing intimal pipe within a larger, blood-containing, hopefully not "leaky," adventitial pipe (as in an acute aortic dissection).

The extreme pain caused by this process in these often hypertensive individuals, tends to keep their blood pressure elevated while these concentric aortic pipes separate (tear apart) rapidly (except at their most adherent portions), until the adventitia reaches its elastic limit. During this separation, many holes are torn between the inner and outer channels, where arterial branches formerly passed through both.

The "end result" is a markedly distended outer vessel, hopefully still "feeding" those attached arteries that were lifted away from the perforated intimal tube by the expanding adventitia. Where inner and outer vessels remain adherent, the blood supply to surrounding tissues continues to derive from the inner channel, through the common wall, as before.

One reason that the adventitial tube surrounding such an "auto-endarterectomy" has far greater likelihood for rupture than the usual surgical endarterectomy is the far greater tension exerted on the vessel wall, for this tension goes up rapidly as vessel diameter increases: This "explains" why a "radical" (intima plus media) endarterectomy is generally not performed on any vessel greater in diameter than a small to normal size mid-abdominal aorta.

Vessel wall tension is also the reason that "spontaneous" dissections commonly originate in the largest artery first, so we refer to it as an "aortic dissection" (and try to identify the most proximal site of aortic involvement, or determine the site of aortic intimal tear when we can) even though its distal evolution often involves significant aortic arterial branches in a crucial fashion that may determine patient outcome after surgical repair.

A far less extensive, intramural dissection results when the site of intimal disruption underlies "still normally elastic" aortic media. Thus, the post-traumatic aortic tear in a younger individual most commonly remains localized at the left subclavian level. The mechanism of such an intimal tear, with no preexisting cystic medial necrosis, appears to be an extremely strong surge of blood back up the aorta, as a result of sudden severe abdominal or leg compression during auto accident, ditch cave-in, and so on.

When intraaortic pressures rise suddenly to the point where "something has to give," that "something" could also be aortic valve or even left ventricle wall; perhaps this depends in part upon the presence of preexisting disease, or even the position of the aortic valve cusps (open or closed) and whether the ventricle is contracting or relaxing at that moment, but those who arrive alive at the hospital usually have their aorta torn at the level of the obliterated ductus "where the aorta becomes

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more fixed."

Findings at surgery in my first three cases of traumatic rupture of the descending thoracic aorta (all in their late teens or early twenties, well before we had cardiopulmonary bypass available) suggested that the mechanism of this disruption might be other than the above mentioned "fixation" of the aorta: Rather the fault seemed to lie in the locally diminished distensibility of aortic wall at the site of the obliterated ductus arteriosus.

Thus, the first patient had an intimal tear only at the ductus (as if it had been "popped out like a cork"), with only local, subadventitial extravasation of blood. The next patient had the same finding, plus a tear halfway around the aorta through intima and media, centered on the "uncorked" ductus site (with far more subadventitial extravasation). My third patient again had the ductus "pulled off," and his intima and media were torn completely around the aortic circumference at this same level, with the two widely separated open ends contained within a tightly distended adventitial balloon that was "sweating blood" (as was I).

All three did well after simple reapproximation of the separated intima-media component. These sturdy layers came together easily end-to-end, in marked contrast to flimsy tissues encountered at acute (not trauma related) aortic dissection - which hold sutures about as readily as wet toilet paper.

It is said that aortic dissections develop sturdier tissues for suture as they become more chronic, but one could also claim that it requires a relatively fibrotic adventitia to "hold" long enough to become chronic. My first three acute ascending aorta dissections certainly would not have lasted to any "chronic" stage, for all presented with intrapericardial blood and acute aortic valve insufficiency. Two survived hospitalization, one is alive and well - the other died at surgery for a later repeat dissection and valve replacement (a number of years ago, before composite grafts became available to us).

In other words, it is likely that some patients with acute dissection have sturdier tissue than others, which may indeed allow them to survive to the chronic stage. This suggests that early repair of all salvageable candidates with this condition will occasionally be rewarded by an easier repair on a patient who otherwise would have become chronic. Also that patients with this condition who survive transfer to a distant "teaching center" may be "better surgical candidates" (easier cases) than those who require an emergency operation locally, soon after their first symptom.

How Elastic Should Our Arteries Be?

Let us assume an "almost perfectly elastic" large vessel system that could absorb an entire cardiac stroke volume with minimal change in pressure. As long as such a vascular tree remained somewhat "stretched," systolic and diastolic pressures would be almost identical. In theory then, very elastic arteries should convert the output of a to-and-fro pump with valves to a relatively pulseless, continuous flow.

Among the disadvantages of such a perfectly elastic arterial system would be its relatively enormous blood storage capacity: This might permit economical blood storage at arterial pressures during times of low-flow requirement, but a reservoir large enough to provide significant supplemental flow during exertion would be an obvious burden and present significant risk of rupture (we might resemble a postprandial mosquito, with distended red or blue reservoirs confirming our state of discharge or repressurization). Apparently there is good reason for the compromise between rigid and

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elastic pipes that has been provided by Nature.

Pulse pressure must remain with us, therefore, while we still use an intermittently ejecting heart rather than a continuous turbine. The turbine might work with partially elastic vessels, but this would imply increasing blood storage in increasingly stretched arteries at increased flow rates, so an adequate venous reservoir would be needed (though not as large as in the mosquito analogy above). So how much pulse pressure do we need? That is, can peripheral tissues prosper with continuous arterial inflow at a steady pressure?

Well, first of all, pressure regulation by any mechanism is easier when a desired mean pressure is continuously "bracketed" by actual pressures just above and below it. It is characteristic of self-correcting systems that small adjustments frequently applied from both sides will tend to maintain the desired output, while the delay involved in waiting and then reacting to an adverse trend usually leads to overcorrection, which results in wider oscillation of the desired output: So if you try to avoid a pulsatile pressure, you may simply create an oscillating pressure. (Thus, to steer a straight course with a ship requires bracketing of the desired heading with frequent small rudder changes, each of which, if maintained, would have caused a significant deviation.)

If it could be delivered, what might one expect from pulseless constant-arterial-pressure perfusion of an immobile tissue? Well we know that tissue "turgor" and local lymphatic pressure are both closely related to mean capillary blood pressure minus the osmotic gradient back into capillaries from the tissues: Also that a certain amount of interstitial fluid will accumulate around soft-walled capillaries and lymphatics, as well as within them.

Under pulseless, steady state conditions, the gradient along capillaries and veins or lymphatics (upon which the outflow of blood or lymph depends) will still be the difference between tissue pressure and the pressure within its principal venous or lymphatic outlets. It also seems intuitively obvious that there must be considerable resistance to flow within any tiny soft-walled lymphatic vessel, which may extend a meter in length and begin just a few microns in width.

It further seems evident that pressure within any lymphatic channel must remain at or below that of surrounding tissues, or it cannot accept fluid passively through its wall (of course, if tissue pressure were to considerably exceed endolymphatic pressure, the soft-walled lymphatic might well collapse). Can one perhaps postulate an additional way to control tissue edema fluid accumulations, besides an ever increasing osmotic gradient back to blood, or propulsive lymphatic peristalsis, or active transmural lymph secretion (at higher than tissue pressure) into the lymphatic lumen?

Studies of lymph flow and tissue weight during cardiopulmonary bypass reportedly indicate that tissue edema becomes an increasing problem during pulseless perfusion. How does our normally pulsatile perfusion avoid this state of gradually worsening tissue perfusion and edema?

It is evident that distensible arteriolar and capillary walls readily transmit arterial pulse pressure changes to surrounding tissues; "tissue pressure waves" must result when these tiny vessels widen simultaneously (and thereby compress surrounding tissue cells and fluids). It seems likely that such continuously passing tissue pressure waves then create local microgradients in properly aligned lymphatics and venules which will tend to "milk out" these small valve-containing structures and thereby maintain their flow, even when detectable gradients over comparatively huge distances could not be expected to achieve the same goal.

Capillary and lymphatic flow must be included in any study of the conservation of blood momentum: Although one might expect the rapid slowing of blood flow peripherally to generate unacceptable amounts of friction and heat, the elasticity and compressibility of tissues (or parts of tissues in the case of bone marrow fat) apparently convert blood kinetic energy into potential energy that has utility for the local tissue economy.

We seem to have concluded that some pulse pressure is desirable, which in turn requires moderate arterial wall stiffness. We have reasoned that excessive pulse pressure consequent to overly rigid vessel walls can lead to unnecessary cardiac work. The general "goal" of any circulation, appropriate blood flow rates, also demands that capillary pressures be kept below edema and above ischemia levels.

The progressive stiffening of aging vessels, and the often associated limitations of cardiac output, makes it increasingly difficult to stabilize the older circulatory system under the stress of surgery or exercise. One must then begin to make trade-offs, using medication and other manipulations to achieve the more essential goals (e.g., using nitroglycerine to decrease peripheral resistance also lowers perfusion pressure, and so on).

The Intraaortic Balloon Pump

A discussion of vessel capacity would be incomplete without some mention of this technique for raising diastolic and lowering systolic pressures. In brief, the stroke-volume-sized intraaortic balloon inflates during diastole (while the aortic valve is closed), delivering a pulse of increased arterial pressure to all (or mostly the proximal) tissues including the heart; the balloon is then actively collapsed during systole, thus lowering intraaortic pressure while the aortic valve remains open for cardiac ejection: This balloon sequence markedly decreases cardiac power requirements during systole (less pressure work and less fluid inertia to overcome).

Balloon pumping can be of great value for temporary support of a failing left ventricle when the balloon is properly located within the upper descending thoracic aorta. Right-sided failure following cardiopulmonary bypass allegedly also responds to balloon pump placement in the pulmonary artery, through (and perhaps still partly within) a blood-filled fabric diverticulum.

Satisfactory control of critical intraoperative right-ventricle failure in the adult apparently has also been achieved by a simple 2 cm. long incision in the interatrial septum that allows the failing right ventricle to unload through right-to-left shunting at the atrial level (a possibly useful temporizing measure).

Elastic and Muscular Arteries Contain a Pressurized Reservoir of Blood

Opening a major artery to the atmosphere causes a gush of blood much greater than the current cardiac output, for the entire arterial tree is thereby rapidly decompressed. The same elasticity and muscular contraction of vessel walls that normally smooths pulse waves and supports intraarterial pressures also enhances excessive blood loss from an open artery (until hypotension, local arterial contraction and retraction, obstructive clot formation, or cardiac arrest bring hemorrhage to a close).

Indeed, an uncontrolled artery spraying blood across the room is so impressive that the usual sedate and orderly advance of blood through intact arteries seems unreal. Since uncontrolled arterial bleeding is the only time one actually sees blood "flow" (fly), misconceptions abound. Angiograms

offer more reliable views of circulatory speed.

Venous Return

Cardiac output depends upon venous return: The forward movement of blood through capillaries, along with intermittent tissue pressure changes, "powers" the returning blood through many parallel, distensible, venous channels toward the sponge-like pulmonary vascular bed where blood is regenerated after each circumnavigation.

Unlike arterial pressure (which is far higher and thus less affected by local factors), "venous blood pressure" can change markedly in accordance with local tissue or cavity pressures to which that portion of vein is exposed: Numerous one-way valves within peripheral veins provide support to the venous blood column, allowing flow only in a central direction (whenever pressure gradients are favorable).

During relaxation, muscle tissue pressure often falls below nearby subcutaneous tissue pressures. However, the pressure within contracted muscle generally exceeds the intraabdominal venous blood pressure. Similarly, on inspiration, intrathoracic pressures fall below atmospheric (thus below all peripheral and intraabdominal venous pressures).

This "design" causes increasing muscle activity and more vigorous respirations to improve the systemic venous return and cardiac output to levels appropriate for exercise. Conversely, marked blood stasis in some peripheral veins accompanies the absence of any muscle movement (during anesthesia, while on assisted ventilation and muscle relaxants, or following heavy sedation).

When there is difficulty in filling the right heart (e.g., following surgical repair of tricuspid atresia), intermittent abdominal compression reportedly may assist venous return and improve cardiac output. Manual compression of the abdomen for half-minute intervals apparently was comparable in effect to intermittent abdominal compression at 45 mm Hg by Mast suit or by a periabdominal blood pressure cuff (placed around the abdomen of small children): Such manipulations or devices are said to decrease arterial-venous oxygen differences and (usually) improve urine flow.

Apparently periabdominal cuff inflation during expiration (achieved by a ventilator attachment) was equally useful; perhaps because expiration is the time of lowest intrathoracic pressure while on ventilatory assistance, and that is when viscera must push the diaphragm back up anyhow. Although continuous excessive elevation of intraabdominal pressure may cause renal failure (see Chapter 1), one would guess that intermittent compression, as described, is free of such risk.

The "blood pump" interacts with its surrounding "air pump" in a similarly beneficial or complementary fashion. Thus, pleural space pressures are normally kept below peripheral airway (intrapulmonary) pressures by the tendency of normal lung to relax away from the chest wall (to a smaller relaxed size): This especially enhances filling of thin-walled, low-pressure, subpleural structures such as systemic great veins, pulmonary veins, atria and esophagus. Not surprisingly, ordinary intrapleural pressure changes (as between vigorous inspiration and expiration) can noticeably alter systemic and pulmonary venous flows.

The same subatmospheric intrathoracic pressure that improves blood return also aggravates esophageal reflux, however. It is good design, therefore, that the superior vena cava bulges into the pleural space while the esophagus burrows deeply within the mediastinum.

One might anticipate that any esophageal replacement passing through the pleural space (or even bulging under intact parietal pleura from a substernal location) would show increased tendency to gastroesophageal reflux: Similarly, the ante-thoracic (subcutaneous) location of such an esophageal conduit should diminish the likelihood of significant reflux (only a guess).

Left Ventricle Works Harder

It requires very little right heart pressure to propel blood through a normal pulmonary circuit. Right and left ventricles have the same rate and stroke volume, but left ventricle must support the systemic pressure (a lot more work).

Left Ventricle May Stretch Atria

Free cardiac movement within the pericardial sac and chest probably improves atrial filling: As each bolus of blood (stroke volume) is forced into the aorta, the left ventricle recoils and rotates in an opposite direction (which may enlarge the right atrium at a time when upward movement of the tricuspid valve would otherwise reduce the right atrial volume).

Storage of Excess Blood

Pulmonary and splanchnic (visceral) vessels and peripheral veins, all have a significant blood storage function. Loss of splanchnic and peripheral venous tone, for example, leads to peripheral venous pooling, decreased venous return, lowered cardiac output and perhaps fainting (hopefully to a flat position in which venous return, cardiac output and thus cerebral perfusion can again improve).

On the other hand, any excess in blood volume will cause peripheral veins to dilate, while ankles (or other dependent areas such as the back and buttocks of a supine patient) become edematous: Liver, spleen and lungs then become congested as well, for excess fluid naturally tends to pool in organs with low capillary pressures and sponge-like parenchyma. Gas exchange at the delicate blood-air interface of lung is easily deranged by such fluid accumulation (undoubtedly congestion also interferes with liver function, although tenderness due to stretching of the liver capsule is often an early complaint).

Liver Congestion

In an awake patient, liver pain from sudden congestion can mimic an "acute abdomen." On the other hand, during laparotomy, visible purpling and swelling of the liver may be the first sign of an acute intraoperative fluid overload, or the sudden onset of right heart failure. Significant capsular indentation on gentle compression can then confirm that liver is indeed severely congested.

While normal, living liver is firm to the touch, and a minor squeeze leaves only a momentary, minor, yellowish-brown dent, an acutely congested liver is purplish, softer and far more compressible, and it rapidly resumes its smooth, featureless, distended appearance after compression is released: In fact, such liver closely resembles a full plastic blood bag (for the obvious reason that it really is a bag full of blood, plus "some other stuff").

Rapid confirmation of acute cardiac failure (by measurement of the peripheral venous pressure), and immediate therapy with digoxin and diuretics plus fluid restriction, has prevented catastrophic pulmonary edema in several similar cases that were first detected at laparotomy by simple observation.

Case Report: Acute Liver Congestion

A middle-aged male was brought to the hospital after he collapsed with a sudden onset of abdominal pain and breathlessness. Diagnostic paracentesis in the right mid-abdomen returned "gross blood." Emergency laparotomy revealed a huge purple distended liver bleeding significantly through its torn capsule at the needle puncture site. That overstretched capsule then tore relentlessly during subsequent attempts to suture the bleeding site.

Autopsy revealed that a massive pulmonary embolus had caused acute right heart overload (right heart failure), severe liver congestion, liver pain and circulatory insufficiency. The urgent diagnostic stab and subsequent therapeutic suture needle wounds in this dying patient had then elicited uncontrollable hemorrhage.

Perhaps locally applied hemostatic powders (and/or some sort of inflatable "liver compression bag" or a flexible soft-surfaced horse-shoe-shaped adjustable hinged compression device to apply a temporary gentle local two-sided liver squeeze) could help reduce bleeding from flimsy liver surfaces that cannot be sutured. And nowadays cardiopulmonary bypass is occasionally life-saving when it is possible and essential to recover and reuse lost blood during temporarily uncontrollable surgical bleeding.

Lung Congestion

Symptomatic lung congestion can often be relieved by increasing the airway pressure. It may be that the "secondary" bronchospasm of early pulmonary edema has a similarly beneficial effect, as it prolongs an increasingly forced expiration.

Any good "volume respirator" (as opposed to a pressure controlled respirator) should be able to maintain bronchiolar and alveolar air pressures at selected values above atmospheric throughout the respiratory cycle (while also eliminating the need for respiratory work by the patient). Above atmospheric airway pressures presumably drive excess fluid back from lungs into splanchnic and peripheral veins (as well as liver); the improvement in pulmonary and tissue oxygenation that follows, then provides time for diuresis and other therapy to proceed.

Intrapleural Pressure Changes Can Affect Cardiac Output

The effect of intrapleural pressure changes on heart function depends partly upon the blood volume. Any hypovolemic patient given ventilatory assistance is likely to suffer diminution in his already marginal blood return (or perhaps even a catastrophic loss of blood pressure and cardiac output).

In contrast, a hypervolemic patient, with venous reservoirs maximally distended and even overflowing into tissue edema, should not have noticeable alteration of cardiac output under these circumstances. Therapeutic interventions must be attuned to such realities, for harm can result whenever any "routine" - such as the induction of anesthesia - becomes a "ritual" that is followed "religiously" (without critical thinking).

Evaluation of Blood Loss in General

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Blood loss or inadequate fluid intake can cause hypovolemia. The blood volume deficit from an acute blood loss can be estimated through careful observation of the patient (movements, mental state, skin color, breathing), while also checking pulse, blood pressure, conjunctival color, skin temperature and peripheral venous filling. Anyone who has ever been seasick or watched someone faint, however, will realize that not every pale, greenish, dizzy, sweaty person is in dire need of blood transfusion.

On the other hand, a supine adult with 80/40 blood pressure, pulse of 150, and clinical likelihood of ongoing major blood loss, probably has a 1.0 to 1.5 liter blood volume deficit. Confirmatory signs of such deficit are flat or invisible peripheral veins (empty reservoir), cool extremities (peripheral vasoconstriction associated with a diminished cardiac output), blanched-out conjunctivae on the inner surface of the lower eyelid (often signifying a low hemotocrit; less vasospasm here, so get used to checking this), and constant restlessness with complaints of thirst ("Water! Water!").

Hypotension due to blood loss should never be confused with the preterminal hypotension of severe head injury. The latter is usually a very late finding, associated with slow pulse, coma, and (too often) apnea. The occasional "vagal reaction" or "indeterminate cause" hypotension that occurs with slow or normal pulse, warm extremities (if they were warm before), and normal atrioventricular conduction (especially during or soon after surgery), usually responds nicely to atropine (0.5 to 1.0 mg, given intravenously), but ignore "eye signs" (pupil size and reactivity to light) for a while thereafter.

Estimating the Amount of an Acute Blood Loss

A quick, imaginary visit to a blood bank can often help evaluate the severity of acute blood loss. Imagine first, that 500 cc of blood has been taken slowly from a healthy adult volunteer. No effect? Well, if he sits up slowly and drinks a little fluid, he can usually walk off with minimal symptoms or change in his pulse and blood pressure.

Now, take a liter of blood from another healthy adult. He may not notice any problem while supine, although he will probably be "a bit thirsty," but any rapid "sit-up" will likely be followed by an equally rapid return to the horizontal, possibly even on the floor. His blood pressure while still flat on the table was probably "down a little" and his pulse undoubtedly somewhat increased: While still horizontal, however, he was most likely not "in shock." If examined during his brief attempt to sit and stand, however, significant hypotension would have become evident (and that might well persist for a time after his collapse).

Lastly, withdraw 1500 cc of blood from a third, unsuspecting, hypothetical donor (thus taking one-third to one-fourth of his entire blood volume). Not surprisingly, his arterial pressure will go down and his pulse will go up: With flat veins and pale conjunctiva, he will be vocal about thirst, distressed, anxious, and almost surely horizontal by preference.

Recovery or Replacement of Intravascular Fluid Losses

Normal compensatory mechanisms promote rapid recovery of the intravascular (blood) volume at the expense of other extracellular and intracellular fluids. This hemodilution is associated with diminished urine output, due to marked fluid resorption as well as decreased renal perfusion. Clinical estimates of acute blood loss (keeping in mind the above-mentioned hypothetical blood donors) take all of this into account, however, so one can replace blood pretty much on the above "eyeball" basis (whether or not it agrees with measured blood loss - see also below).

During rapid blood replacement, always monitor blood pressure, pulse, venous filling (including neck veins), urine output, and breath sounds at both lung bases (posteriorly and laterally on the thorax). Resumption of satisfactory urine output, visible filling of peripheral and neck veins, and return of blood pressure and pulse to near normal - any or all of these signs suggest caution in further fluid replacement, especially if the patient now remains "stable" in a semi-sitting position.

New crepitant rales at the lung base indicate possible fluid overload (especially when traumatic lung damage or any preexisting lung disease is unlikely); temporary cessation of fluid therapy is then in order, perhaps with stimulation of diuresis. On the other hand, one must occasionally push the blood volume gently up beyond the full-vein level, when an elevated venous pressure appears necessary to achieve good right heart filling and ejection (as during pericardial tamponade, or sometimes following tetralogy repair, or with cardiac dysfunction from "some other" cause): When venous blood volume and pressure are carefully increased under these circumstances (to improve the subnormal arterial pressure), it should not overload lungs and cause rales unless the left heart also is compromised (which is not usually the case).

Fluid Versus Blood Replacement for Acute Blood Loss

Unless autotransfusion of hemothorax is an immediate possibility, severe blood loss often requires temporary volume replacement by physiologic salt solution, pending availability of properly cross-matched blood (which could easily mean a 45 to 60 minute delay). Type-specific, incompletely matched, or "low titer" 0-negative blood are considered safe alternatives to fully cross-matched blood in an emergency.

One is sometimes caught between the possibility of renal shutdown from trauma and poor perfusion, and the (minimal) risk of renal shutdown from incompletely-crossmatched blood. I have generally selected the latter course when such a choice presented, but it is not always easy to predict who can wait (or what a jury might decide).

Untreated Blood Loss

Around 1960, the mortality rate for acute severe upper gastrointestinal blood loss, optimally treated by full blood volume replacement and emergency surgery, was reportedly the same as that of comparable patients who received neither volume replacement nor surgery. While not espousing similar non-intervention today, it still seems undesirable to strive for normal pulse, blood pressure and urine output in an actively bleeding, preoperative patient (prior to achieving partial control of bleeding or, at least, to obtaining properly cross-matched blood).

On numerous occasions, I have watched vigorous preoperative volume replacement of still-uncontrolled blood loss (using physiologic salt solution and bank blood) cause a patient to become extremely anemic and dangerously hypothermic, and his remaining intravascular "blood" rendered incoagulable. There are many acute surgical situations when one might better allow moderate hypotension to persist until the patient can be brought to the operating room; hoping thereby to retain more of the bleeding patient's own fresh, warm, perfectly cross-matched blood within his own blood vessels.

Limited acute blood loss (up to one liter, perhaps) is often quite well tolerated by the previously healthy patient, provided that the blood volume lost can be replenished from intracellular and

extracellular sources, or replaced by external supplies of "less volume effective than blood" physiologic salt solution. Normally, over 75 percent of all extracellular fluids are located outside of blood vessels (although all "real blood" should be found inside): Theoretically then, if administered fluids were similarly distributed, and other corrective fluid shifts not already under way, more than four times the estimated acute blood loss would be an appropriate volume of Ringer's Lactate Solution to administer for temporary physiologic correction of blood volume.

In practice, it may require significantly less externally supplied salt solution than anticipated to restore the blood volume (or occasionally far more; see below). Once stabilized by this fluid replacement (one to two liters of Ringer's Lactate Solution for each 500 cc of blood lost acutely), a basically healthy patient can soon correct his mild residual anemia and hypovolemia, just as after blood donation. Obviously, there must be an initial weight gain if a given volume of blood is "replaced" by a far larger volume of colloid-free solution, but that fluid excess usually lasts only a few days (until postoperative edema is eliminated and the normal volume of "healthy blood" is rebuilt).

Of course, with a functioning gastrointestinal tract, a limited blood volume loss is rapidly and easily replenished by oral fluids and salt, ad lib. Even the patient who has been denied oral or intravenous volume replacement for moderate blood loss can soon make up most of the intravascular deficit (by mobilizing large volumes of intracellular and extravascular fluid): As time passes, following an acute major blood loss, therefore, any patient may show far less tolerance for transfusion than one would expect, based upon the known blood loss and lack of supplementary intravenous or oral fluid intake. This is another good argument for "treating the patient" rather than simply replacing the blood loss.

Single unit blood transfusions can often be avoided: There is certainly no need to add the risk of unnecessary transfusion to that of the patient's primary illness or operation. Especially in marginal or elderly patients, however, single unit whole-blood transfusions will often achieve better stabilization than larger volumes of salt solution.

Quite commonly, more Ringer's Lactate Solution is required to achieve adequate replenishment of intravascular volume (and regain acceptable pulse, blood pressure and urine output) than might have been predicted from the known volume of blood lost, for tissue edema follows all trauma (including surgery). It may be hard to believe that a massively edematous patient can simultaneously be desperately "short of fluid," but any major loss of intravascular volume (even if "only" consequent to fluid leakage from capillaries) must be replaced by additional fluid, regardless of whether, or even how much more, the patient may swell.

Tissue Edema

Local conditions and specific organ requirements routinely regulate cerebrospinal, ocular and inner ear fluid secretion and removal. However, intracellular and extracellular fluids of most other tissues also relocate passively in response to circulatory and hydration changes, osmotic differences, tissue pressures, inflammation or injury. Extravascular extracellular fluid accumulations that are not readily exchangeable with intravascular and intracellular fluids are often referred to as "edema fluid" located in the "third space".

This definition implies that edema fluid is "sequestered" extracellular fluid, temporarily unavailable to the patient's circulation. Indeed, on a short-term basis, edema fluid may be as

inaccessible as urine in the bladder or the water in a photograph. Of course, edema fluid is a far greater burden that can cause possible complications ranging from decubitus ulcer to pleural effusion or pulmonary edema.

An important consequence of ongoing immobility on any "dependent" (below the heart) part of the body is the persistent back-pressure exerted against that immobile tissue by an uninterrupted column of venous blood extending from the affected site up to the heart. Edema of dependent tissues may therefore result from inactivity (e.g., an injured leg in a cast - or ankle edema in older adults who stand a lot - or back and buttock edema in a persistently supine, sick patient in bed) as well as from incompetent venous valves which allow persistent back-pressure to be exerted on dependent tissues regardless of activity.

An active muscle contraction squeezes veins in or near that muscle, thereby forwarding nearby venous blood toward the heart. With competent venous valves, muscle contractions thus reduce venous back-pressure on tissues and alleviate dependent edema by lifting venous blood onto competent venous valves closer to the heart. Frequent changes of position also alter the dependent status of affected tissues (as when we put our feet up on the desk, for example).

Tissue edema fluid accumulates between cells, outside of capillaries and lymphatics, in the "third space" (wherever there is "room" for more fluid). Although dependent edema may occur from scalp to toes, it is most common in the lower legs, or over the back and buttocks of hospitalized patients (where, at times, it may become massive, especially in the more "fragile" tissues of older or very sick patients).

Edema fluid can often be partially "recaptured" (mobilized into the intravascular space) within hours, through a change of position (elevation), by external compression (elastic hose, pneumatic counter-pressure garment, or other device), or following a decline in venous pressure (as seen after pericardiectomy for constrictive pericarditis).

Whenever possible and practical, the best way to eliminate dependent tissue edema is to elevate the affected dependent tissue above heart level and keep it there for the number of days required. It is also important to encourage exercise, with a full range of motion (and to avoid, or try to overcome, any venous or lymphatic obstruction). Long-lasting dependent edema can lead to tissue fibrosis, ischemic changes and an increased local susceptibility to infection.

Case Report: Pulmonary Edema from Mobilization of Dependent Edema

A young disabled veteran of the Vietnam conflict underwent left thoracotomy for peripheral coin lesion (adenocarcinoma was found and removed by wedge resection). Postoperatively he developed fulminant pulmonary edema soon after being extubated. Eventually he required endotracheal reintubation for prolonged positive-pressure ventilatory support while he underwent a multi-liter diuresis.

Relevant history was a gunshot through his left groin and buttock, with complete loss of femoral vessels: Saphenous vein bypass of the destroyed femoral artery was followed by postoperative infection, pneumonia, pulmonary emboli and vena cava ligation. His persistent chronic disability was massive post-traumatic swelling of the entire left leg. A venogram before thoracotomy revealed that all venous return from his entire left leg drained via a single tortuous vein through the buttock.

Discussion: With a history of multiple pulmonary emboli and recurrent pneumonia, "discretion" (wedge resection) seemed the better part of "valor" (lobectomy), for this patient was already chronically dyspneic. The severe postoperative pulmonary edema certainly supported that decision, but why did it occur? (My competent anesthesiologist friend had delivered less than two liters of intravenous electrolyte solution during the entire operative period.)

Eventually we realized that the patient had been able to mobilize much of his massive leg edema while in the lateral position for thoracotomy (as the swollen leg then remained above his heart, and body weight no longer impeded venous drainage via the buttock). Now, almost two years postoperatively, the patient has done well (except for an intervening acute appendicitis).

He also markedly improved his ambulation when we were able to overcome much of his limb swelling with a large hydraulic compression device (provided for his home use by the V.A., after some judicious prodding). Prior to this time, with only custom-made elastic hose, he either had to lie down all the time or accept swelling, and this young "survivor" was not about to lie down.

Posttraumatic Edema Increases Fluid Requirements

Posttraumatic edema grossly resembles dependent edema and is similarly worsened by immobile dependency (and any venous valve incompetence or other interference with venous blood return to the heart). The duration and extent of posttraumatic edema will be determined by local and regional vascular and lymphatic damage, as well as any release of tissue breakdown products or complicating infections.

Early tissue edema usually accumulates in rough proportion to the extent of tissue injury: Generally, therefore, the postoperative or injured patient will require intravenous electrolyte-solution supplements to replace his fluid losses into the third-space (which can be considerable, up to many liters).

Unless much of this edema fluid is secondarily lost into the environment (through injured skin, or by external drainage from internal surfaces such as pleura, peritoneum or the gastrointestinal tract), one can expect edema fluid to eventually reenter the circulation, as inflammation subsides and healing progresses.

In the course of uncomplicated postoperative healing, mobilization of third space fluid usually becomes significant within 36 to 48 hours after surgery: The resulting blood volume expansion may cause severe lung congestion, and will normally be associated with vigorous diuresis.

Mobilization of Edema Reduces Fluid Requirements:

The only goal of routine fluid administration after surgery or other trauma is to satisfy current fluid and electrolyte requirements. Volumes administered should usually be sufficient to support normal blood pressure and urine flow, but not enough to cause lung congestion: Within these parameters, we depend upon "physiologic adjustments" to "keep things in order" (under our watchful eye).

We antcipate and welcome a routine spontaneous postoperative diuresis as this provides indirect evidence of satisfactory progress in healing (for significant infection, inflammation or tissue necrosis would all "third space" additional fluid, rather than releasing previous edema fluid to the circulation).

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While such postoperative diuresis persists, however, the patient will have a reduced or even "negative" fluid requirement.

A negative fluid requirement calls for fluid administration to be kept at a bare minimum (only that amount required to deliver essential medication intravenously), while appropriate diuretic therapy simultaneously reduces the fluid overload. An occasional patient must also be severely restricted on oral fluid intake during this diuresis, so that his temporarily inappropriate thirst does not delay recovery.

Following chest surgery, the patient is usually eating and drinking before the onset of this postoperative diuresis: Regardless of oral intake, however, one should NEVER attempt to replace such unusually large-volume urine "losses" (that signify edema fluid is now being mobilized and excreted) in the "daily fluid orders."

We have all seen patients tolerate considerable volumes of intravenous solution, even though these "maintenance fluids" were administered at the height of a normal diuresis, but such burdening of an already hypervolemic postoperative patient is illogical, unnecessary, stressful and too often, even hazardous.

And if you still worry that your apparently recovering patient may suddenly have somehow developed "high volume renal failure" - or think that he has failed to produce sufficient antidiuretic hormone – there is still no need to run costly renal or ADH tests: For within four to six hours, any patient undergoing an appropriate spontaneous postoperative diuresis will still look like he is recovering well, while the patient suffering from inappropriate diuresis will definitely begin to show signs of hypovolemia with increasing thirst and pulse rate and decreasing blood pressure (for further details on renal function, ADH and human physiology in general, see Human Evolutionary Biology (Human Anatomy and Physiology from An Evolutionary Perspective) by Arndt von Hippel – available for free download from textbookrevolution.org).

On Fluid Losses Into Dead or Dying Tissues

Capillary permeability is increased by direct trauma (including thermal injury) as well as "toxic" substances (septic shock, anaphylaxis, various chemicals) and inadequate circulation. By the time simple ischemia causes a major increase in capillary endothelial permeability, significant damage will already have occurred in surrounding soft tissues. Nonetheless, a worthwhile recovery of function is sometimes still possible if adequate tissue perfusion can be maintained by fasciotomy, thrombolysis, and perhaps therapeutic agents that decrease "free radical" injury.

Apparently, oxygen-derived free radicals are abundantly produced in ischemic tissues as a result of enzyme damage (these highly-reactive forms of oxygen or hydroxyl, minus an electron, can allegedly induce an ongoing chain-reaction of destructive oxidation or reduction of tissue molecules). Not surprisingly, free radicals have been implicated in "reperfusion injury" (tissue damage that only becomes apparent on restoration of blood flow after ischemia).

There is some evidence that allopurinol pretreatment prevents production of superoxide (oxygen free radical) by xanthine oxidase (a damaged form of the normal xanthic dehydrogenase) and thereby decreases reperfusion injury. The primarily intracellar enzymes that normally destroy any free radicals are known as superoxide dismutases ($O2-. + O2-. + 2H + = H2O2 + O2$). What all this theory means for

the future treatment of ischemia is still unclear, but it could lead to exciting advances.

In any case, if adequate circulation is restored after a period of ischemia sufficient to damage surrounding tissues, extracellular fluid will be drawn out through intact or damaged capillary walls by the osmotic effect of tissue breakdown products (as intracellular enzymes also make their appearance in the blood stream). If, in addition, capillary endothelium is so damaged that capillaries simply cannot "hold their water" (or even proteins and blood cells), massive swelling is bound to ensue.

In the natural course of tissue death and necrosis, arterial inflow (and thus tissue swelling and edema) will tend to be limited. Continued local sequestration of essential body fluids is prevented because direct fluid loss from damaged capillaries leads to local hemoconcentration, sludging and thrombosis; endothelial cell swelling and increased tissue pressures also tend to occlude the microcirculation. A relatively sudden "natural" death of an entire individual by circulatory failure (total body ischemia) proceeds in similar fashion, also without excessive tissue swelling, as the patient and his circulation cease to function more or less simultaneously.

It is when circulation is "unnaturally" restored (to a dying extremity by revascularization, or to a dying patient by cardiopulmonary bypass), that truly massive, ongoing fluid losses are encountered. Modern interventions can prevent final thrombosis (with heparin), delay hemoconcentration and sludging (with copious infusions of balanced salt solution), and decrease the likelihood that severe limb compartment edema will block arterial inflow (by fasciotomies); but the advanced tissue necrosis usually associated with a significant loss of capillary integrity due to ischemia is usually irreversible: Then even "timely" fasciotomies which successfully restore circulation may simply maintain patent vessels to a "dead" leg.

The patient that we try to resuscitate on cardiopulmonary bypass (after a hypothermic ischemic period of unknown duration), will usually prove to have been ischemic too long for successful resuscitation if he requires massive volumes of blood and salt solution to maintain effective perfusion. While different tissues in the body seem to differ in their ability to tolerate and recover from ischemia, the core organs are "gone" when their capillaries are "blown". Perhaps continued perfusion even at that advanced state could still "save" the bony skeleton or whatever, but severe capillary leakage as a consequence of ischemia commonly signals a significant (local or generalized) termination of function.

Anaphylaxis, Edema and Epinephrine

Anaphylaxis is an occasional indication for a relatively massive short-term infusion of fluids to maintain the circulating blood volume until a "toxic event" can be overcome by steroids (such as solumedrol in repeated, 2 gram "industrial" doses), antihistamines in large amount, respiratory support as needed, and epinephrine as tolerated intravenously. It is truly amazing how much epinephrine may be required during resuscitation of a patient who develops severe anaphylaxis in the operating room.

However, just because it takes a lot of epinephrine "to keep the patient going" does not mean that the case is hopeless; rather it simply confirms the diagnosis of anaphylaxis (for under other circumstances, such an epinephrine dose would likely have "blown the patient's head off" with hypertension, or caused ventricular fibrillation).

However, once such an epinephrine drip has been started and proven effective, Do Not Stop It Too Soon; keep the epinephrine running as an IV drip while necessary, and taper the dose only very slowly

if it is still well tolerated (even after the patient could seemingly do without), for if fulminant anaphylaxis then recurs, you may not achieve a second successful resuscitation.

Venous Oxygen and Venous Pressure Measurements

Various catheters can help determine fluid and blood requirements. A "central venous pressure" catheter (preferably with its location confirmed in the superior vena cava by chest roentgenogram) is often very helpful, perhaps more by the direction and degree of change than the precise values measured (differences in calibration or patient requirement for a certain minimum venous pressure are not always obvious or predictable, and many variables can alter venous pressure readings: For example, hypoxia allegedly causes pulmonary hypertension, which may raise the venous pressure in the same fashion as right heart failure).

A pulmonary wedge pressure or left atrial pressure determination (apparently these are not "always identical") often report the loading pressure of the left ventricle more specifically and accurately; that information is particularly helpful when "fine-tuning" a patient with marginal cardiac function or "following" a patient during massive fluid loss and replacement.

Published reports suggest that the patient with normal pulmonary vascular bed, mitral valve and left ventricle, will have a measured pulmonary artery diastolic pressure which is the same as the pulmonary capillary wedge pressure and also the same as the mean left atrial pressure and the left ventricular end-diastolic pressure; this relationship apparently does not hold with pulmonary hypertension or during tachycardia (which does not allow enough time for equilibration).

We are told that continuous positive end-expiratory ventilator pressure may increase end-diastolic pressure without increasing left ventricular end-diastolic volume. Under these circumstances, the pulmonary capillary wedge pressure or left ventricle end-diastolic pressure does not represent the actual left ventricle preload. Many other variables can also confuse the significance of these measurements. For example, furosemide allegedly lowers the pulmonary artery pressure within minutes, even before diuresis occurs.

Balloon-tipped flow-directed pulmonary artery catheters also have risks (as do central venous lines - see Chapter 3) such as inducing large-vein thrombosis or pulmonary artery rupture. Hemoptysis in the presence of a balloon-tipped pulmonary artery catheter is reportedly a sign of impending "bad trouble," especially with pulmonary hypertension or during anticoagulation. It may then be best to leave the catheter in place undisturbed, as a guide for the most-likely-impending resection of involved lung. In the face of severe bleeding from a balloon-ruptured pulmonary artery, reinflation of the balloon at the same site or slightly proximally has been recommended.

Fortunately, there is always a balloon available to solve any problem caused by another balloon: With a ruptured pulmonary segmental artery, one can also pass an appropriate-sized Fogarty catheter under fiberoptic bronchoscopic guidance to tamponade the bleeding segmental or lobar bronchus opening (thus protecting the uninvolved airway from an overflow of blood until resection can be accomplished). Fuji Systems Co. of Tokyo apparently even markets an endotracheal tube with a built-in moveable "blocker" balloon to control endobronchial bleeding.

On the premise that it is easier to stay out of trouble than get out of it, I rarely use flow-directed balloon catheters, so the above discussion represents "second-hand" advice. The time, skills and risk

involved in cannulating a preinfarction patient for complete monitoring can sometimes be put to better service in effecting a speedy repair (which usually reduces the need for such intensive monitoring).

In addition, an accidental carotid artery puncture, during placement of a pulmonary artery catheter through the internal jugular vein, greatly increases the danger of subsequent anticoagulation for cardiopulmonary bypass, and routine left-atrial lines carry the ongoing risk of "minor" air or clot embolism (even when they only reveal what is clinically evident).

Despite being somewhat of a "monitoring nihilist," my hospital death rate for elective and urgent coronary bypass (1974-84) was about 1% (with an overall 2% mortality rate for the entire 700 patient experience, including all valve cases, reoperations and emergency cardiopulmonary bypass procedures for repair of heart and great vessels).

Anyhow, a finding of elevated venous pressure with normal pulmonary artery wedge pressure suggests a more proximal flow restriction, such as pericardial tamponade, pericardial constriction or major pulmonary embolism: "Equalization" of the central venous pressure with right atrial, right ventricular and pulmonary artery diastolic pressures usually permits the diagnosis of pericardial constriction or tamponade (as does sonography).

An acute massive pulmonary embolus is associated with markedly elevated venous, right atrial and right-ventricular pressures. This becomes only too evident if one opens the pericardium in such a patient before instituting cardiopulmonary bypass (by internal jugular vein and femoral vein cannulation): Under these circumstances, the markedly overloaded right heart may simply balloon out massively and stop beating forever.

It can reportedly be dangerous to rely upon wedge pressures for fluid therapy management after pneumonectomy, as that can lead to fatal fluid overload despite a normal pulmonary artery wedge pressure. The importance of fluid restriction at and after pneumonectomy has long been emphasized (see Chapter 9).

At the termination of cardiopulmonary bypass, one reloads any repaired heart gradually, providing partial flow support until the heart seems able to propel the entire cardiac output. Sometimes a heart requires an additional normothermic interval on total bypass before it can carry this load, and it is unwise (even dangerous) to try to shorten such a recovery period by "flogging" the heart with "inotropic support."

When reloading the heart "to get off the pump," one can transfuse far more rapidly than even the normal cardiac output, and the immediately-post-repair heart is certainly not up to its normal performance. It is especially important to avoid overdistending the easily visible right heart at this time, which can occur even if the rapid transfusion from the cardiopulmonary bypass reservoir is being delivered through the aortic line (perhaps a low peripheral resistance allows more rapid return of blood to the right heart).

Anyhow, it has been reported that an overdistended right ventricle can prevent adequate left ventricle filling and function (by bulging the interventricular septum into the left ventricle lumen and outflow tract); and that this condition can be diagnosed by elevation of the CVP over the simultaneously recorded pulmonary capillary wedge pressure; and that it can be remedied through withdrawal of the venous overload, to allow right heart distension to diminish so the left ventricle can

fill more adequately (and thereafter support the circulation).

I have not encountered this problem (perhaps because I usually take a short coffee break after inducing anything more than a brief period of hypothermic arrest, while the heart gradually returns to normothermia and normal sinus rhythm, still on total circulatory support). However, the above-described difficulties with a deviated ventricular septum certainly emphasises the need to watch the heart and avoid "unnecessary distension" while "coming off the pump."

Perhaps it becomes more important to monitor the wedge pressure if you plan to "rush the patient off the pump." As for "rushing," I prefer to think that the surgeon has "nothing better to do" than bring his patient off the pump "slowly but surely."

Changes in Central Venous Oxygen Saturation Usually Reflect Changes in Cardiac Output

The oxygen content of mixed venous blood is reduced below that of arterial blood, by metabolic oxygen consumption. Therefore, in a relatively stable patient, a decline in venous blood oxygen content usually suggests a decline in cardiac output (same oxygen extraction from a diminished flow of blood), although it could also represent an increase in metabolism (more oxygen taken from a relatively constant flow of blood), or a lower oxygen content of arterial blood (less oxygen to start with), or some combination of the above.

Soon after coronary bypass surgery, the patient is usually at rest, normothermic and well-ventilated (stable metabolism and relatively constant arterial saturation), so changes in his venous-blood oxygen content tend to parallel changes in his cardiac output. Such a patient may be somewhat hypervolemic soon after surgery (due to "hemodilution" during cardiopulmonary bypass), but he commonly undergoes copious diuresis in the early postoperative hours, and sometimes suffers postoperative blood loss as well.

Any consequent significant decrease in his blood volume leads to decreased venous return, which in turn will reduce cardiac output. A major drop in cardiac output may impair vital signs and even endanger survival, so a simple test to detect decreasing cardiac output (before this alters pulse, blood pressure or urine output) can be most helpful.

It turns out that a decline in mixed central-venous oxygen saturation usually precedes other signals that the supine patient is becoming increasingly hypovolemic: "Semiprophylactic" intravenous administration of blood or electrolyte solution can then be started to prevent any impending undesirable drop in arterial pressure. To be of any help in patient care, however, the mixed venous blood sample must be taken from the superior vena cava, right heart or pulmonary artery, for more peripheral venous samples commonly show significant variations in response to local circulatory changes (consequent to arm position, arm movement, arm temperature, or even alterations in the rate of fluid administration through a nearby intravenous cannula).

Summary: A central-venous blood oxygen saturation that remains stable (preferably over 60 percent) usually reflects a satisfactory circulatory state, while any progressive decline in central venous blood-oxygen saturation (to and below this level), especially within the first few hours after coronary bypass when the patient is still properly oxygenated on controlled ventilation, suggests an undesirable decline in blood volume and thus cardiac output (this is usually associated with slowing of the urine output).

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If a patient develops obvious shock, or has multi-organ failure or adult respiratory distress syndrome, the correlation of central venous oxygen saturation with cardiac output supposedly becomes unreliable, but that does not detract from the usefulness of this determination under other circumstances.

Rigid Blood Vessels Have a Relatively Fixed Volume

Some older persons with a truly rigid vascular tree and limited cardiac output, may demonstrate a very narrow transfusion tolerance; 50 cc of blood removed or added can move the patient back and forth between hyper- and hypovolemia, especially during early postoperative hours when their venous blood storage reservoir is also relatively non-responsive as a result of anesthesia and other medications. This is in marked contrast to a healthy younger person who can readily compensate for a 500 to 1000 cc acute blood loss (when not additionally depleted by post-traumatic tissue edema or severe dehydration).

General Aspects of Intravenous Fluid Therapy

Postoperative intravenous fluid therapy should support serum potassium at optimal values. After non-cardiac procedures, a dilute multi-electrolyte solution containing five percent glucose can be supplemented with potassium (appropriately diluted and infused via a pediatric drip chamber) as necessary to maintain serum K+ at 4.5 to 5.0 milliequivalents per liter (which is helpful in reducing the risk of dysrhythmia, especially in older patients). Barring unusual losses or deficits, additional sodium has not seemed necessary in the usual post-operative heart patient who begins oral intake on the morning after surgery.

During, and for the first day or so after adult cardiac surgery, a "polarizing solution" of 10% glucose in water, containing 50 mEq of potassium chloride and 10 U of regular insulin/liter, is usually given as "maintenance" fluid therapy (at 75 to 100 cc/hour). Here also, the intent is to support serum potassium over 4.5 mEq/liter and, hopefully, to build up intracellular potassium and glycogen reserves. Of course, this solution is discontinued if (and for as long as) serum potassium becomes elevated.

This glucose-insulin-potassium infusion supposedly enhances myocardial preservation in the face of ischemia, possibly by supporting myocardial ATP levels and shifting myocardial metabolism to glycogenesis and glycolysis (both before and after heart surgery). Allegedly there is not much glycogen in normal myocardium, and anaerobic glycolysis is generally less important than lipolysis as a source of myocardial energy. (Whatever that may mean to the biochemists among us, the fact is that this "polarizing solution" is well tolerated, and it has not caused postoperative electrolyte difficulties "when used as directed.")

Potassium solutions that are infused into peripheral veins at concentrations over fifty mEq/liter will cause pain, spasm and often early thrombosis of such vessels. However, if these high potassium concentrations must be given through a small vein, 3 or 4 mg. of morphine injected through the same vein at the start of potassium infusion may eliminate local discomfort and spasm. One should always run concentrated potassium solutions slowly, especially if through a central venous line, to allow early dilution in the vena cava (rapid infusion of a "high" potassium solution through the central venous pressure line has caused hypotension, bradycardia and even cardiac arrest).

Standardized multi-electrolyte maintenance solutions will meet routine postoperative fluid and

electrolyte requirements if kidneys are functional. These multi-electrolyte solutions usually include sodium (at levels slightly more than one-fourth that of normal serum), potassium (also over 30 mEq/l), and adequate amounts of other essential cations and anions.

Such maintenance solutions are given to a normal adult patient at 50 to 150 cc/hour and are entirely different in purpose and function from solutions designed to replace extracellular fluid volume, such as Ringer's Lactate Solution (with or without 5% glucose): "Ringer's Lactate" closely mimics serum electrolyte concentrations and can therefore be given in large amounts for replacement of massive fluid losses without causing marked electrolyte disturbance.

Dextran and Albumin

The use of Dextran-40 currently (in 1985) is permitted for plasma volume expansion, priming of extracorporeal circuits and prophylaxis against venous thromboembolism. Dextran 70 and Dextran 75 are approved only for plasma volume expansion in the treatment of shock. In view of the efficacy of blood and plain electrolyte solutions for plasma volume expansion, and the adverse reactions to Dextran that are reported (such as anaphylactic or anaphylactoid responses by those patients who are sensitive to dietary or bacterial polysaccharides), it seems unnecessary to use Dextran except for prophylaxis against thromboembolism (it "appears helpful" while an intraaortic balloon is being used, for example). At this time the FDA is reevaluating the use of Dextran.

Human serum albumin was formerly used extensively for volume resuscitation of the injured or burned patient. I cannot comment on either use except to note that it, too, appears unnecessary in the treatment of shock; according to the literature, the use of albumin for volume resuscitation has been associated with an increased incidence of acute renal failure, left ventricle dysfunction and posttraumatic pulmonary insufficiency. (Those complications may have something in common with the allegation that intravenous hyperalimentation leads to an increased incidence of pulmonary sepsis and prolongation of hospitalization when used perioperatively for patients with esophageal carcinoma.)

Reportedly, the use of colloid rather than crystalloid may help prevent pulmonary toxicity during Bleomycin therapy (inspired oxygen concentrations should also be held low during such treatment). I might also use albumin infusions a few days postoperatively to improve intestinal healing and function in a patient with very low serum albumin after bowel or liver surgery, but I have had no recent experience with that problem, and otherwise have given up the use of this product.

Many successful surgeons utilize serum albumin infusions to maintain the serum osmotic pressure during hemodilution bypass in these days of questioning the safety of blood transfusions, so there is probably no "right" or "wrong" in this matter; just do "whatever works for you" (while remembering that all interventions carry cost and risk).

Oliguria

Hourly urine output is usually a fair measure of hydration, barring renal disease, unusual osmotic load, or active diuretic treatment. Posttraumatic renal shutdown is best prevented by avoiding dehydration and maintaining normal blood volume and copious urine flow. Prolonged hypotension (or other ischemic insult), tissue breakdown products and many other toxic substances, can severely damage kidneys within hours, so always monitor and support urine flow meticulously (above perhaps 50 cc per hour in any critically ill or injured, adult-size patient), except following pneumonectomy,

when the risk of renal shutdown is minimal and it is all-too-easy to overload the remaining lung with fluid (see Chapter 9).

Intravenously administered diuretics such as Lasix (furosemide) and mannitol are of great help during the prevention, evaluation and treatment of oliguric states, but inspection of the patient with poor urine output, as well as his chart, is also essential. If fluid replacement seems adequate, but lungs are still dry and neck veins flat (or central venous pressure is not markedly elevated, with CVP catheter patent and in good position), one can run 500 to 1000 cc Ringer's Lactate rapidly into an adult patient's vein, to see if this oliguria responds to volume expansion. In the presence of congested lungs or high central venous pressure, an early trial of vigorous diuretic therapy will usually be preferable.

Mannitol Diuresis:

With no response to Lasix and apparent "good perfusion" (full veins, warm extremities, normal blood pressure), a mannitol load can help decide the matter. If there is significant renal perfusion, 25 to 50, or even 100 gm. of mannitol (delivered as an intravenous solution) can provide a sufficient renal osmotic load to oblige urine flow through still-functioning nephrons within minutes. Mannitol stimulation of urine flow can be helpful or essential in clearing tubular debris, preventing protein precipitation, washing out intratubular accumulations of toxic solutes, or even reopening renal tubules (through reduction of renal intersitial edema). In other words, "it takes urine to make urine."

Mannitol may be less useful once blood urea (also an effective osmotic diuretic) becomes quite elevated. Although mannitol can expand the blood volume and perhaps shrink red cells, reduce blood viscosity and decrease cerebral swelling (until it is excreted), these often beneficial side-effects should not alter its application in appropriate amounts when sorely needed: A common, more easily corrected, cause of severe oliguria is a plugged or displaced Foley catheter (or even some other blockage of the urine drainage tubing, so look for this initially, or replace the entire catheter system if doubt remains).

Summary: Renal shutdown may be a response to one or several simultaneous insults, including direct trauma, embolism or other compromise of renal circulation, tissue breakdown products, circulating toxins including peripheral vasoconstrictors (such as dopamine in high doses), low blood pressure, elevated intraabdominal pressure, hypoxia, infection, hypovolemia, dehydration, renal contrast or retrograde studies, urinary tract obstruction or other pre-existing renal disease.

The best way to prevent renal shutdown is rapid restoration of blood volume and urine output: One or two hours delay can be critical in the development of renal shutdown, so pursue urine output vigorously. Since renal failure can apparently result from different causes or through several mechanisms, several simultaneous remedies may sometimes work better than one. We frequently utilize Lasix (for both its possible vasodilating and urine-producing effect) simultaneously with mannitol in generous doses: Low dose dopamine infusions may also be appropriate (for their renal vasodilating effect).

"Calcium blocker" agents and renin antagonists are currently being studied for their value under such circumstances (in preventing renal vasospasm). A kidney during diuresis is supposed to be less sensitive to ischemia, so some authors recommend establishing a diuretic state before the aorta or renal vessels are cross-clamped electively (I prefer mannitol for this use).

The usual hemodilution technique for cardiopulmonary bypass, plus vigorous prophylaxis as

described, have prevented the development of post-bypass oliguric acute renal failure in my 700 cases (excluding those already in renal failure before surgery). Postoperative or posttraumatic renal failure still carries a very high mortality, so early consultation with a competent renal specialist is recommended in any potentially troublesome case.

Diuresis

As discussed above, postoperative patients commonly have a moderate diuresis while mobilizing tissue edema, usually about thirty-six to forty-eight hours after surgery. The postoperative cardiac patient routinely excretes many liters of urine per hour, starting immediately after the termination of cardiopulmonary bypass (hemodilution technique). On other occasions, a postoperative or injured patient may develop massive diuresis for no apparent reason.

Again, consultation is urged if the situation becomes hazardous or overly complex, but it is likely that the patient either has diabetes insipidus, diabetes mellitus with a glucose osmotic diuresis, fluid overload, excessive diuretic therapy (possibly meant for the patient in the next bed, or ordered by a consultant without your knowledge) or high-output renal failure. Fluid treatment must be persistent and electrolyte monitoring close in any of these conditions, until the patient is stable.

Diuresis is the normal response to fluid overload, however, so (as mentioned above) avoid being in the ridiculous and harmful position of trying to replace urine volume, plus provide additional generous maintenance fluids, in a patient undergoing major diuresis consequent to hypervolemia (during mobilization of edema, for example).

A surgeon is rarely called upon to replace the actual urine output in any case, for his usual task is simply to support it above minimally adequate levels. The patient in obvious oliguric or anuric renal failure who does require volume replacement of any urine that is produced will usually require the attention of a nephrologist as well.

Fluid administration and losses during and after surgery are quite variable and not always recorded: In addition, "third spacing" of fluid (as tissue edema) is unpredictable, as is the mobilization thereof. It is the patient's circulating blood volume and electrolyte status (especially K+) that is important, even if he is in renal failure. We do not need a direct "measure" of that blood volume, however, in order to deal with it.

In other words, one must watch the patient's appearance, vital signs and urine output closedly (as well as serum electrolytes, hematocrit and renal function when indicated), and not worry if "intake" and "output" don't "match." An accurately measured and correctly recorded, daily A.M. patient weight can be of great help when evaluating the overall fluid balance (although recorded weights seem subject to more frequent and larger errors than almost any other clinical measurement).

How can one tell if a given diuresis is "normal"? Well, first of all, "Relax." Excessive fluid and electrolyte loss by urine is no more dangerous than excessive fluid and electrolyte loss by perspiration or other cause, and we have all survived that. Next, observe the patient: If he is not complaining of thirst, if his tongue is moist and eyes do not appear "sunken" (in other words, if he does not look "dry"), he is most likely just disposing of excess fluid.

Finally, watch his course closely, especially checking pulse, blood pressure, temperature,

hematocrit (for hemoconcentration) and serum (but only very rarely urine) glucose and electrolytes for any progressive abnormality or unusual loss. If "something really is wrong," it will become clinically evident within a few hours: One can then em-bark on further appropriate therapy or bring in a consultant to help you care for just these few "troublesome" patients.

When a patient has become severely dehydrated, his intracellular and extracellular solute concentrations rise well above normal: If then provided with large volumes of dilute oral or intravenous fluids, the abnormally high intracellular solute concentration may cause his cells to imbibe excessive fluid from the suddenly diluted blood. Severe swelling of cells may then lead to the patient's death from cerebral edema, especially in children (and sometimes the elderly).

For this reason, oral or intravenous administration of isotonic salt-containing solutions is recommended as the initial therapy for severe hypovolemia due to dehydration. The intent here is to bring the blood volume back toward normal with isotonic fluid, then allow a more gradual loss of solute on resumption of urine production; ingestion of increasingly dilute fluids is then permissible. (My personal clinical experience dealing with severe dehydration is minimal.)

Lasix Diuresis:

Diuretics such as Lasix can be used to manipulate a "not always understood" state of hydration, in order to find out whether your patient is "sort of wet" or "rather dry." Thus I often administer Lasix to an anemic, hypervolemic or hypertensive patient early after surgery, until the central venous pressure and arterial pressure begin to drift below desirable levels: At that point, it is easy to rehydrate appropriately, or transfuse, while still keeping the patient comfortably over "on the dry side."

In similar fashion, preliminary diuresis allows a very anemic patient to receive one or two units of less expensive, easier to administer "whole" blood (versus packed cells, which also lack useful plasma components). The non-bleeding patient will usually benefit from an additional dose of Lasix between blood transfusions (to prevent pulmonary congestion consequent to the second transfusion).

It often becomes necessary to supplement serum potassium during Lasix diuresis, but our experienced nurses do this quite skillfully from standing orders. We also utilize Lasix, along with potassium restriction, to bring down any undesirably high serum potassium; more urgent measures include intravenous glucose and insulin, or even potassium-absorbing resins by enema (especially for the hyperkalemic oliguric patient, pending dialysis). Incidentally, it has been alleged that hemodialysis for impending renal failure may increase the likelihood of persistent renal failure: Whether or not that is true, it seems sensible to manage the fluid and electrolyte status as carefully as possible, so that the need for dialysis can be kept to a minimum.

Hypotension and Minimum Adequate Perfusion Pressure

In theory, there is some minimum arterial blood pressure at which essential core organs will survive but not function: The lowest arterial pressure which also allows continuing satisfactory function of brain, heart, liver and kidneys should therefore provide some margin of safety. Externally delivered kidney product is the simplest indicator of adequate core organ perfusion in a heavily sedated patient.

Satisfactory urine output at a stable, low, blood pressure (below 100, over perhaps 80 mm Hg.

systolic) is thus a reasonable indication of adequate circulation. In other words, under "normal" circumstances (horizontal position and patent vessels), brain, heart and liver circulation can be assumed adequate in the presence of significant urine flow. If, in addition, the patient is not cold, uneasy or restless, perspiring, or complaining of severe thirst, these are additional "good signs."

Such a stable "functional hypotension" need not be treated (attacked) by pressors or balloon pump. In fact, this "low normal" arterial pressure is presumably associated with adequate blood volume and good arterial runoff through a moderately dilated peripheral vascular tree. Somewhat low arterial blood pressure can be a desirable early postoperative condition (adequate cardiac output with minimal cardiac work required to overcome low circulatory resistance). The body will usually shift to a higher pressure as recovery progresses (usually by the day after surgery): In any case, 80 mm. systolic is the "normal" arterial pressure for some patients.

Hypotensive Control of Blood Loss After Cardiopulmonary Bypass

Relative hypotension not only reduces cardiac work but it can also decrease worrisome postoperative blood loss. We have administered a continuous nitroprusside or nitroglycerine "intravenous drip" following sternotomy on numerous occasions, in order to achieve "controlled hypotension" and thus reduce, or hopefully stop, ongoing mediastinal blood loss.

Hypertension and Blood Volume After Cardiopulmonary Bypass

Not uncommonly, a patient may develop significant hypertension soon after sternotomy closure and admission to the intensive care unit. Postulated causes of such "cardiopulmonary bypass related" hypertension (which lasts but a few hours if untreated) include delayed renal responses to hypotension during bypass, diminished postbypass pulmonary norepinephrine inactivation, untimely beta-blocker withdrawal, a relative increase of arterial PCO_2 to normal (after intraoperative "blow-off") and altered vascular reactivity secondary to non-pulsatile perfusion.

It is evident that beta-blockers have not created this entity, for such hypertension was encountered before beta-blockers became available. The regularly seen "post-dilutional hypervolemia" is probably also not a major factor, although Lasix is sometimes effective in reducing both blood volume and any associated mild hypertension.

In any case, this particular sort of hypertension (which increases cardiac work and also the likelihood of poststernotomy bleeding) often responds within minutes to one or more tiny (one mg.) doses of thorazine given intravenously (usually up to a total of less than 10 mg.). One must often wait a number of minutes after each one mg. dose to determine its eventual effect: Also, regardless of whether post-bypass hypertension is being treated with thorazine or an intravenous nitroprusside or nitroglycerine drip, one must anticipate that such vasodilator therapy will expose any blood volume deficit (which, surprisingly, often coexists with this hypertension).

Be prepared, therefore, when treating postbypass hypertension, to administer significant volumes of blood or Ringers lactate solution in order to fill newly dilated vessels and reestablish adequate venous return. Not uncommonly, if such hypertension is left untreated, it will collapse "spontaneously" within hours, even in the absence of significant blood loss. The transition from mildly worrisome, hypovolemic hypertension to mildly worrisome hypovolemic hypotension can be almost instantaneous (I just turned away for a moment, and look what... !).

Whether achieved gradually or suddenly, the "newly refilled" vascular system (with vasospasm and hypertension spontaneously or medically relieved) is "like money in the bank," available to cover "unanticipated expenses" such as postoperative bleeding: A fully "replenished" patient will also be more responsive to dopamine, should that be required.

If well tolerated, the vigorous application of positive end-expiratory pressure can also help increase the volume of blood held "in reserve" (stored in veins): Reduction of the "effective blood volume" in this fashion helps one to control postoperative hypertension, as well as relieve lung congestion due to temporary early postoperative hypervolemia (see Chapter 16).

Vasoactive Medication is Always Administered into the Side-Arm of a Continuously Flowing Primary Intravenous Line

Incorrect administration of vasoactive drugs (based upon common misconceptions about intravenous injections) is a very frequent cause of postoperative hypotension. When a drug must be given intravenously and very slowly, the concerned nurse will not uncommonly also reduce the flow rate through the I.V. tubing into which she injects. Then, after exactly five minutes of meticulously slow injection (during which time she conscientiously monitors the patient for any conceivable adverse effect), having delivered exactly the right dose, she resets the primary intravenous line for full flow and leaves the room for her next patient, serenely confident of a job well done.

It may only be at this point, however, that the drug finally is flushed from the intravenous line into the patient, who thus receives an unintended, concentrated, bolus injection which often is tolerated poorly. If the surgeon can take time to check, and especially if he avoids "bullying" the other members of the health team, he may discover that many otherwise-inexplicable "idiosyncratic" drug reactions can be attributed to just this one minor misunderstanding. Although minimal preparatory teaching can prevent such a simple mistake, it will rarely if ever come to the attention of a surgeon "who shouts" so his patients remain at higher risk for that reason as well.

Many intravenously delivered drugs can alter circulatory dynamics: If not injected into a vein under direct control by hand-held syringe, such drugs must, at least, be delivered into a continuously-flowing I.V. line, as close to the needle entering the patient's vein as possible. Such proximity to the patient's vein will minimize accumulation of drug within the intravenous tubing, and make it more likely that the patient actually receives the medication at about the rate and time that it appeared to be "given."

Appropriate regulation of any rapidly acting, vasoactive drug demands that drug entry into the patient at least parallel the visible "drip rate" from the I.V. medication bottle. Even then, any consequent vasoactive effect is normally delayed (after drug entry into the patient) for at least one "circulation time."

Many factors can destroy a linear relationship between administration of a medication into the I.V. line and its effect on the patient: Medication delivered into the primary (maintenance fluids) I.V. tubing, from the medication-containing (secondary) I.V. bottle, may have a very delayed and uncertain expression that depends upon the primary I.V. flow rate, relative heights of primary and secondary bottles, extent of streamline flow, size and shape of the primary I.V. tubing, relative density of primary and added solutions, and much else.

This suggests that if excessive or undesirable effects ensue during such an intravenous injection, all interconnected intravenous lines and bottles must be suspected of containing additional drug; hence all flow into this intravenous needle or catheter must be completely interrupted until the drug effect has worn off and more is needed, or the tubing (and perhaps bottles) can be aspirated, flushed out or replaced.

To Err is Human, to Forgive (Without Knowing What Happened) is Stupid

A "nitroprusside drip" is often required to control early post-operative hypertension. Intravenous administration of this vasoactive drug usually is slowed and finally stopped when it is no longer required: Then, often just before "change of shift," the "nitroprusside bottle" is finally replaced by plain Ringer's lactate. Soon thereafter, the increasingly hypovolemic patient's blood pressure drifts on down, so the nurse who has just come "on duty," very appropriately administers a few hundred cc. of Ringer's Lactate solution from the full bottle.

All too commonly, she thereby also delivers the relatively huge, unlabeled bolus of nitroprusside that still remains within the temporarily unused secondary intravenous line: As a result, the patient's slowly declining blood pressure promptly disappears. (A recorded arterial blood pressure of 40 mm. Hg systolic is not uncommon at this juncture.)

Fortunately, this sort of "catastrophic" hypotension is "self-limited," commonly lasting about five minutes, and surprisingly well tolerated by the obtunded patient (the foot of whose bed is immediately elevated while help is summoned from the four corners of the hospital). Presumably some cardiac output can be maintained despite the severe, temporary loss of peripheral resistance, perhaps in part because of the foot-up position and simultaneous, copious fluid administration (which help to maintain adequate venous return until the unexpected drug effect has worn off).

On occasion, a previously utilized tubing, still full of succinyl choline muscle relaxant, has similarly been flushed unknowingly into a patient during transport to or from the recovery room (reportedly once even in the elevator): This amount of drug can suddenly "relax" the patient so well that he even becomes unable to breathe. (The patient is the only person within hearing range who remains relaxed at such a time).

Needless to say, it is important to discontinue the intermittently utilized secondary tubing along with its attached succinyl choline bottle, and to be sure that the principal "maintenance" intravenous line has been flushed clear of drug at that time.

Summary: Any intravenous "drip" delivering vasoactive medication such as nitroglycerin or dopamine will require constant surveillance and regulation. Adjustment of the drug-containing solution flow rate through a side-arm from the secondary I.V. into the primary tubing is a common method for regulating the effect of such medications. A needle, inserted in sterile fashion into the distal rubber tubing of the primary intravenous line, often serves as a simple side-arm (where concentrated drug can enter the ever-flowing primary intravenous line).

The continuous flow of maintenance solution through the primary line serves to dilute the vasoactive drug before it reaches the patient, while also preventing significant retention of drug in the primary tubing. Significant "backup" of vasoactive medication into the primary I.V. tubing, or even up into the "maintenance fluid" bottle, cannot occur if the primary bottle is kept "dripping" continuously

from the moment before the secondary line is inserted. The primary tubing flow rate also can be adjusted, in order to further "fine tune" administration of the vasoactive drug.

All flow (in both primary and secondary "side-arm" lines) is stopped at once, of course, if the patient shows sudden signs of excessive drug effect: More commonly, flow through the side-arm line will gradually be decreased (while the primary I.V. flow rate remains constant) as the patient stabilizes and is "weaned" from medication. This results in the primary line carrying ever-diminishing drug concentrations until the side-arm is shut off and disconnected; soon thereafter, the primary line will have been flushed free of all drug by the continuing primary flow (even if no special precautions were taken to achieve this).

Policy: It is the responsibility of the physician or nurse regulating any vasoactive drip to remove all intravenous tubings that may contain such drug at the time the drip is discontinued. Therefore, if a drip must be discontinued suddenly (leaving significant vasoactive drug in the primary line between side arm and patient), the primary line should be cleared by aspiration, disconnected and flushed, or even replaced, if need be.

Query: Why not simply forgo all this bother and connect or disconnect any vasoactive drip right at the needle?

Well, a vasoactive I.V. commonly is discontinued for significant periods of time, during which the needle could clot. In addition, every manipulation of the needle or catheter causes additional trauma where it enters the vein. Finally, such manipulations are tedious and distracting, and often there are too few satisfactory intravenous access ports to dedicate one solely to each use. Undoubtedly, computer regulated, feedback controlled, one-way intravenous fluid delivery systems will soon outmode much of the complicated network of tubing that must currently be utilized to achieve rather poor control of blood pressure in the seriously-ill postoperative patient.

So Cultivate a Little Paranoia

Any intravenous line with an additional side-arm line "simply cannot be trusted," for one carefully labelled bottle can easily empty into the other (rather than into the patient), especially when both are supposedly running rapidly and drip chambers are too full. Until color-coding of intravenous bottle contents becomes available, one should at least suspect intermingling of contents of any interconnected bottles: Certainly, intravenous fluid container labels must always be kept "up-to-date" for all known contents.

Case Report: Incorrect Label

During my internship at Boston City Hospital, a patient with Foley catheter had "24 hour urine collection" ordered, for some long-forgotten, probably unnecessary test (it was, after all, a "teaching hospital"). The collecting bottle first utilized happened to be a recently emptied glass intravenous-fluid bottle.

Once full of yellow urine, the first collecting bottle was disconnected from the Foley and temporarily placed on the bedside stand by an aide. When notified that his intravenous fluids had run dry, the just-coming-on-duty-and-already-overworked nurse quickly checked the label and hung the "new" bottle that "someone had left, ready to go" on the bedside stand, still labeled "vitamins added"

from its initial use.

Conclusions:

1) Even if pea green, color coding of intravenous fluids probably cannot prevent such errors if the label is not current;

2) I.V. bottles should not be used for other purposes;

3) "Previously opened" I.V. bottles must always be discarded;

4) Foley or nasogastric tubes and their connectors should be manufactured in such sizes or shapes that they cannot possibly be attached to any I.V. tubing or connector (for anything that can be misconnected, will be misconnected);

5) The cost, discomfort and risk associated with any test, must be balanced against its likely benefit: An unnecessary urinalysis proved fatal to this patient. Perhaps if a "second opinion" were required before all urinalyses...

Calcium Administration Corrects Hypotension

Calcium chloride given intravenously usually "improves" dangerously low blood pressure (at least following adult cardiopulmonary bypass procedures utilizing hemodilution, which is where we have had considerable experience with its use). Such improvement may be transient or long lasting, perhaps depending upon whether the ionized serum calcium level was normal or diminished (in other words, whether calcium acted upon a symptom or the cause).

If the first one gram "push" of calcium seemed helpful, and the patient's pressure is again drifting dangerously downward, the calcium infusion can usually be repeated with expectation of similar benefit, regardless of current serum calcium levels (which are generally not available at the time of such an episode anyhow). Under emergency circumstances, I have unknowingly boosted the serum calcium markedly above "normal" without apparent detriment (although I certainly cannot recommend this, based solely upon my own limited experience).

Solumedrol for Hypotension

Solumedrol is routinely administered intravenously in large doses to all of our patients during heart surgery (and sometimes after). It allegedly may stabilize cell membranes, reduce white cell clumping (and otherwise decrease the risk of "pump lung"), improve patient tolerance for anoxia and hypotension, prevent ischemic spinal cord injury following aortic cross-clamping, and reduce the likelihood of bronchospasm or allergic responses to medications and blood.

In other words, I am not sure what it does: But, at least Solumedrol has not increased infection rates or caused adrenal failure or pancreatitis. At surgery, we give two grams, and sometimes one gram every four hours for a day or so thereafter (for critically ill or merely hypotensive adult heart surgery patients).

Solumedrol improves blood pressure within 30-45 minutes in the face of (stress-induced relative,

or absolute) adrenal insufficiency: It often increases blood sugar, but otherwise has no obvious adverse effect (even when not needed), so I order it freely while seeking other causes of postoperative hypotension.

Perhaps one percent of patients will develop a symptomatic duodenal ulcer after cardiac surgery, so we use antacids vigorously, often adding cimetidine when routine perioperative steroids must be continued for adrenal insufficiency (rare) or resumed for postpericardiotomy syndrome (more common).

We have detected no ill effect from sudden discontinuation of steroid administration after one or two days of perioperative (or probably unnecessary) steroid therapy. It is alleged that synthetic ACTH can rapidly identify adrenal insufficiency by the absence of a cortisol response, but I have no experience with such tests (nor do I usually have time to await results of any test when the patient suddenly is "doing poorly").

Comment: Dangerously low blood pressure in an unstable post-bypass patient is usually not caused by steroid deficiency, but low blood pressure surely must stress the adrenals. Some anesthetics (such as etomidate, an intravenous sedative-hypnotic) allegedly suppress adrenal cortical steroidogenesis within hours of their administration: That effect may reportedly last one to four days.

In any case, Solumedrol clearly can be lifesaving when needed (which need may demonstrable at the time only by the apparent benefit associated with its administration). Solumedrol tends to raise blood sugar and also causes a pleasant euphoria during an otherwise unpleasant, first postoperative day.

The mild postoperative depression not uncommonly seen one or two days later could perhaps be related to "Solumedrol withdrawal," but it is of little concern, especially if anticipated by the forewarned patient. An elevated white blood cell count (over 20,000 cells per cubic mm.) is also common after Solumedrol administration; this elevation may last for several days, during which time the white cell determination usually has little clinical relevance.

Cardiopulmonary Resuscitation After Sternotomy

In our experience, an unhealed freshly wired sternum can sustain whatever external compression is required for resuscitation without detectable weakening (even a series of several dozen "restarts" has been followed by uneventful healing). Indications for C.P.R. are thus unchanged following sternotomy. In several cases, such C.P.R. has dislodged clot from within the pericardium and reopened blocked chest tubes, but these additional benefits are not currently considered indications for C.P.R.

Outline of an Approach to Post-Bypass Hypotension

Dangerously low arterial blood pressure soon after cardiac surgery is a relatively common "real-time" event (usually involving many variables and insufficient data). Analysis must often proceed simultaneously with treatment, if valuable minutes (and perhaps the patient) are not to be lost.

First: Confirm hypotension and its severity by a brief survey of the patient and his monitoring equipment. Glance at the arterial pulse wave tracing: When this is regular and of good size, it is likely that the patient is fine and that the problem is in the digital readout; check EKG for rhythm and presence of "P" wave (dysrythmia or loss of the "atrial kick" usually lowers cardiac output and thus

blood pressure); see if the patient has a swollen purplish neck and face (signalling superior vena caval or right atrial compression by intrapericardial clot, or perhaps a more generalized cardiac tamponade); check femoral pulses (usually these are the most readily palpable pulses to confirm high, normal or low arterial pressure); look for hives or other acute allergic rash; take an "old fashioned" but usually reliable cuff blood pressure (possibly even on both arms to rule out normal arterial pressure on only one side, as might be expected with long standing atherosclerotic disease of one subclavian artery); observe chest bottle drainage (quality and volume) and "milk" chest tubes as you check for currently excessive bloody drainage.

Second: Rule out a respirator defect, discontinue all PEEP, confirm adequate air exchange, inquire about recent medications (especially vasodilator therapy) and recheck known allergies against recent medications given or ordered. Most of the above steps can be part of a "first glance." The sequence is unimportant: The big questions are "Is something acutely wrong?" and "If so, what obvious clues or crucial evidence can we detect that might guide us to a likely cause?"

Third: Start treatment of likely causes, or of the low blood pressure symptom itself; give blood or physiologic salt solution rapidly by vein (overtransfusion rarely, if ever, causes a problem in post-bypass patients still on a respirator. A marked hypervolemia can always be corrected by blood removal or diuresis. The hematocrit normally drops during early postoperative days anyhow); give calcium (a rapid one gram calcium chloride intravenous injection for its possible "boost" effect); give solumedrol, as well as mannitol (25-100 grams to preserve renal function) or Lasix (same rationale); elevate legs (to improve venous return); with slow pulse, start the heart pacer (an increasing rate, even up to 100 beats/minute, is occasionally very helpful, but patients seem to differ on their optimum rate); for some reason, atropine is usually ineffective in altering the pulse rate soon after bypass surgery (at least if the surgery was performed under heavy morphine anesthesia); a dilute isuprel drip often speeds the heart rate and improves cardiac output by its inotropic and peripheral vasodilating effect; one can also use a dopamine or epinephrine drip; some effective antihistamine is given if there is any question of a possible allergic reaction; Xylocaine is the initial treatment for any ventricular irritability (which may also result from overly vigorous therapy with isuprel, dopamine or epinephrine). Medications that are given for an urgent effect must usually be administered intravenously, provided an appropriate form of the drug is available for such use.

Fourth: Gather more data (serum Na, K, Hgb, Ca, glucose, blood gases and pH, and another chest roentgenogram); any continuing downward trend of an already low hematocrit, despite whole blood transfusion, is often the best clue to blood loss. The hematocrit can also drop by simple dilution, however, when the blood volume has been expanded with asanguinous intravenous fluids or by the patient's own fluid reserves.

Fifth: More specific remedies should be possible by now, including beginning or discontinuing some of the above. Any patient who develops severe, unexplained, inexorably progressive hypotension is a candidate for reopening of the sternal closure, either immediately in bed or soon in the operating room, for cardiac tamponade by fresh bleeding is the most likely cause for such deterioration (unless obviously due to rhythm disturbance, and even then, tamponade often coexists). Prompt surgical remedy of tamponade is life-saving (and at worst, an "unnecessary" reopening of the sternum should cause minimal delay in the course of recovery).

I have never regretted reopening the sternum when "in doubt" (except for the extra effort and "uproar" involved). Once in the operating room, with his chest open and clot removed, the critically ill

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patient is guaranteed optimal management by a competent anesthesiologist and surgeon, now able to directly manipulate and see the heart's response to therapy (including the use of copious sterile warm saline irrigations to prevent hypothermia).

The above outline is for discussion purposes only (being obviously incomplete, overly complex, or out of proper sequence for most patients). However, any urgent problem with many variables - that will surely be encountered - is best considered in advance.

A Nurse Check-List for Viewing the Post-Sternotomy Chest Roentgenogram

1) Read label on film (date, time, patient's name)

2) Inspect film to confirm correct labeling: Is the image displayed consistent with the patient who is named (in overall size and shape, skeleton, soft tissues such as breasts, and artifacts such as wires, staples and tubes)? Is it also comparable with the image seen on prior films?. Has it been put on the "view box" correctly (patient's left to your right, as if you were facing each other)?

3) Check tube positions:

A. The endotracheal tube tip should be located below the larynx and above the tracheal bifurcation, within the tracheal air shadow;

B. The central venous pressure catheter tip should reach the mid-line in the mediastinum but remain above the right atrial bulge (to allow sampling of "mixed" venous blood without inducing atrial dysrhythmia);

C. The nasogastric tube should extend well below the diaphragm: Avoid instilling antacid if the N.G. tube tip is in higher position, as antacid can easily reflux into the pharynx from within the esophagus (and a higher, intrathoracic, N.G. tube might also be misplaced into the trachea or a bronchus);

D. Confirm the location of chest tubes, pulmonary artery catheter and visible pacemaker wires (one will occasionally detect broken or disconnected pacer wires, fractured sternal closure wires, or a first rib fracture posteriorly).

4) Look for an abnormally clear space in either hemithorax (absence of lung markings could mean pneumothorax).

5) Look for increasing densities encroaching on either lung field, or "mediastinal widening" (such densities often represent blood accumulation or atelectasis of the adjacent lung). However, a reduction of air pressure within the lungs (as when recently taken off PEEP or "off the respirator"), or an "expiratory" roentgenogram (diaphragms will be higher), or a change in patient position or distance from the x-ray source - all can cause the mediastinum to appear wider.

There can be a truly remarkable (but not significant) difference in appearance between an inspiratory film taken during maximal mediastinal compression by PEEP, and a later, post-extubation, more-expiratory film (with diaphragm up and "full" heart now in a more transverse position): Therefore, a "somewhat worse-appearing" film can be expected during normal recovery from heart

surgery.

If a chest roentgenogram is taken with the patient somewhat "rotated" (which results in a slightly-oblique film, rather than a "straight" AP or PA), it can mimic mediastinal widening, so avoid overinterpretation of a roentgenogram in which there is asymmetry of clavicles with respect to normally midline structures (such as the tracheal air shadow or vertebral spinous processes.

CHAPTER 15: THE PERICARDIUM

Role of normal pericardium;... Pericardial fat;... Pericardial pain;... Accumulating pericardial fluid may compress the heart;... And affect heart rate;... Paradoxical pulse;... Procedure for cuff measurement of paradoxical pulse;... Acute cardiac tamponade;... Pericardial effusion;... Bacterial pericarditis;... Pericardiocentesis;... Case report: Fatal pericardial tap;... Pericardial window;... Technique of pericardiotomy;... Pericardiectomy;... Case report: Pericardiotomy for obvious pericardial effusion;... Postoperative cardiac tamponade;... Technique of finger mediastinotomy;... Course of pericardial disease;... Case report: Tamponade not relieved by a needle through the window;... Surgery for viral pericarditis;... On the heart falling out of the window;... Minor pericardial entry;... Not all indications for "total" pericardiectomy are valid; ... Be aware of venous overload and peripheral edema before pericardiectomy;... It may be important to reduce venous pressure slowly;... First left, then right;... A free pleural space is necessary for drainage of pericardial infection;... Postpericardiotomy (postinfarction, Dressler's) syndromes.

Role of Normal Pericardium

The slippery translucent pericardial envelope limits heart size and position without restricting its contractions. Opposing epicardial and pericardial surfaces normally are separated by only a few cc of serous fluid. Although not readily expansile, pericardium will usually balloon greatly in response to chronic cardiac enlargement or progressive fluid accumulation. Diseased pericardium may become remarkably inflamed, thickened, shaggy, calcified and adherent to neighboring structures. Various intrapleural, mediastinal and subdiaphragmatic disease processes may extend into the pericardium.

Pericardial Fat

The outside surface of pericardium is covered by varying amounts of moderately lobulated fat: Ditto for the outer surface of the heart. However, the inner aspect of the pericardial envelope has a smooth, glistening, serous lining. Why the fat deposits? Why lobulated? Why not also on the inner pericardial surface? Why even have a pericardial space?

From the mechanical point of view, fat is an efficient cushion and thermal insulator. Smoothly surfaced epicardial fat lobules skate far more easily over the flat serous inner pericardium than they would across another fatty, lobulated surface, so the fact that the inside of pericardium is very smooth speaks to the importance of free heart movement for achieving optimum cardiac output. For example, systolic rotation of the left ventricle encourages atrial filling and also helps to empty its overlying right ventricle (see Chapter 14).

As for the fat lobules, their cushioning function becomes more apparent when inflammation and fibrosis have obliterated the pericardial space: Thereafter, each heart beat still results in significant cardiac movement within its now-adherent pericardial sheath, but this time the movement mostly takes place between the muscular base and fibrosed top of each epicardial fat lobule, as well as between pericardium and its surrounding tissues. Apparently those little epicardial and pericardial "fat plateaus" retain sufficient stretch to prevent ordinary postinflammatory changes from significantly restricting cardiac movements.

Every surgical resident soon learns that important coronary arteries usually hide deep within epicardial fat. If we find this inconvenient, that may be because our recently developed coronary repair skills have not yet had a measurable impact on the evolution of human coronary arteries and their fatty

environs. And until now, larger coronary arteries presumably "did better" by lying low loosely within soft mobile greasy epicardial fat.

Incidentally, epicardial fat is "brown adipose tissue" which differs in at least one obvious way from the vast majority of white adipose tissue that surrounds us. Of course, white fat similarly cushions, insulates and makes for nice curves (by filling in nooks and crannies between and around muscles and organs). And both sorts of adipose tissue also store triglyceride for export to other body tissues as free fatty acids.

But in addition, brown adipose cells contain many more large mitochondria that can directly produce heat (as by a short-circuit - without also doing work) whenever a person is exposed to cold or eats more food calories than can currently be used. Mitochondrial heat production in brown fat is stimulated and controlled by norepinephrine, epinephrine and insulin. Incidentally, those captive hardworking mitochondria – which all descend from free-living proteobacteria – now live within and support every nucleated plant and animal cell (by supplying most of its immediately usable energy as ATP, in exchange for glucose fragments).

The ability of brown fat cells to do a "slow burn" explains brown fat's patchy distribution (interscapular, axillary, nape of neck, subscapular, perinephric, pericardiac, along great vessels, and between ribs) since that is advantageous for keeping crucial blood and spinal cord inputs from "running cold" - which explains why cold hands are more comfortably warmed in axillae than on the flanks.

Considering the fact that atrial and ventricular muscles usually move in opposite directions (after all, they are not supposed to contract simultaneously), it makes sense that the atrioventricular groove (and its enclosed important vessels) is buried within large accumulations of this soft light-brown stuff (which appears increasingly attractive as you get to know it). Despite all of this theory, what would happen if, in the interests of simplifying surgery, we could dispense with all this fat and simply bury larger coronary arteries within cardiac muscle or let them drape along the heart surface?

Well, in the first place, a coronary vessel buried in muscle will be squeezed during systole (a time when an epicardial vessel within malleable fat is filling for its heart's content). In the second place, a larger coronary artery draped upon a fat-free epicardial surface would have difficulty shortening along with underlying muscle fibers during systole - particularly if there was fibrous obliteration of the pericardial space but no surrounding fat to protect the vessel from all the shear stress with cardiac movement. Similarly, if buried within muscle, a larger artery would be subject to repeated flexion and at risk for regular kinking and atheromatous degeneration (which might cause it to become less functional, even more difficult to flex, and so on).

Conclusion: In the right places, fat is beautiful. It allows our skin to move freely over our well-concealed muscles. It liberates the heart, coronary vessels and phrenic nerves from the likelihood of dysfunction or damage by nearby fibrosis and movement. And furthermore, brown fat truly "warms the cockles of our hearts" (assuming "cockles" refers to heart valves, perhaps just the mitral, as cockles and mussels are bivalves).

Pericardial Pain

An inflamed or distended pericardium can cause precordial or referred pain quite similar to angina, except that it tends to vary with changes in position (from supine to sitting, for example). Pericarditis is

often associated with electrocardiographic changes (low voltage and ST abnormalities).

Pericardial pain may be referred to the neck, arms or abdomen; it can even mislead the unsuspecting general surgeon to unnecessary laparotomy for a patient dying of cardiac tamponade - at which point, making a small hole in the bulging inferior surface of the pericardium would offer blessed relief - but no, the exploration merely reports "dusky bowel," and so the patient expires.

Comment: General surgeons now receive sufficient cardiothoracic surgical experience during residency to view gentle finger-tip palpation of the diaphragm underlying heart and lungs as part of their exploratory laparotomy routine (or at least they might consider feeling this upper border of the peritoneal cavity in any puzzling case).

Accumulating Pericardial Fluid May Compress the Heart

The pericardium usually offers sufficient space for the normal heart filled with blood, plus a bit of room for free fluid. When the volume of pericardial fluid exceeds that available space, a rapid pressure build-up occurs within the pericardial envelope since pericardial fluid and myocardium are both incompressible, and pericardium is acutely unstretchable.

A pericardium could become fully distended by 200 cc fluid acutely, but permit more than one liter of fluid to build up over weeks or months). Beyond that "full" point, every additional cc of pericardial fluid would displace an equal amount of blood from within the coronary sinus (say 2 to 3 cc), or from the intrapericardial vena cava (perhaps 15 to 30 cc), or from the atria (maybe 150 cc).

Rising intrapericardial pressures thus reduce atrial filling (especially of the entirely intrapericardial right atrium), while also limiting diastolic expansion of the ventricles (especially the flat thinner-walled right ventricle). Studies suggest that a pericardial pressure of 15 cm water is well tolerated, but that over 25 cm causes acute tamponade.

It is also reported that venous pressure must exceed intrapericardial pressure by at least 4 cm (water pressure) in order to provide sufficient "cardiac input" to sustain adequate cardiac output. So since a venous pressure greater that 25-30 cm "pretty much stops" the capillary circulation, one might have guessed that a pericardial space pressure that reached 25 cm could be incompatible with continued health and survival. And that under these circumstances, the agonal patient would naturally maximize his inspiratory efforts - in order to produce the lowest possible intrathoracic pressures - as the only way to enhance his critically compromised venous return.

Significant pericardial fluid accumulations can thus reduce the volume of blood ejected by each heartbeat. Although the heart "should" compensate for any reduction in stroke volume by increasing its rate (at least to the point where output again diminishes due to inadequate time for chamber filling, valve closure and ejection), the actual result of acute pericardial distension or cardiac compression often is "marked hypotension with bradycardia" (sometimes to the point of cardiac arrest).

Experimentally, atropine will allegedly block this "reflex bradycardia" of acute tamponade. Clinically we have relied upon isuprel or dopamine, because atropine seems less effective following cardiopulmonary bypass under morphine anesthesia.

Conclusion: It does not require a great deal of pressure within the pericardial space to interfere

with cardiac filling. Acute cardiac tamponade develops whenever cardiac compression by intrapericardial (or extrapericardial) air, tissue, or fluid brings the cardiac output down to "marginal" levels: Thereafter, any slight increase in cardiac compression can cause death. On the other hand, removing even 5 or 10 cc of fluid from the tightly distended pericardium is often dramatically lifesaving, for a corresponding small increase in the volume of each marginal heartbeat will be rapidly cumulative and beneficial.

Paradoxical Pulse

Whenever there is diminished acceptance of venous return by the heart, blood becomes "backed-up" peripherally in the veins. As compromise of cardiac filling becomes more critical, the cardiac stroke volume responds noticeably to any small improvement or deterioration of cardiac filling: Even the "normal" alterations of intrapleural pressure may then have significant "impact" on the systolic blood pressure. External compression of the heart can thus lead to the regular respiration-related variation of systolic arterial pressure commonly referred to as "paradoxical pulse".

This arterial pressure variation (or "paradox" as it is known) is often a useful measure of cardiac embarrassment from such compression. Where an arterial-cannula pressure wave is being displayed on the monitor screen, paradoxical pulse will be demonstrated as a regular (sinusoidal) variation in the height of succeeding arterial pressure waves. Paradoxical pulse is also detected readily by careful blood pressure cuff measurement of these regular changes in arterial systolic pressure during normal respiration.

Not all respiration-related variations in systolic pressure represent "true" (meaning pathological) paradoxical pulse. For example, a paradox of less than 10 mm mercury is usually considered normal or "not significant." On the other hand, paradox of 20-30 mm mercury often signifies serious cardiac tamponade (and a marginal cardiac output that should be improved promptly).

One cannot measure paradox in the absence of normal sinus rhythm, however, for major variations in cardiac filling are always encountered during irregular heart beats, and these variations can obscure any respiration-related alterations in systolic pressure. Perhaps a computer analysis of rate versus filling for every beat could eliminate that rhythm variable, but the market for such software is probably limited.

Cardiac failure or marked hypovolemia can also invalidate efforts to determine paradox, as does any abnormally forceful respiration (whether related to physical exertion, illness such as asthma, or respirator-controlled ventilation). However, the gasping agonal respira-tions ("air hunger") that can be associated with preterminal acute cardiac tamponade, do not invalidate an associated finding of severe paradox (though such respirations undoubtedly contribute to the paradox). Additional evidence for tamponade should be sought in all cases of possible tamponade (history, physical examination, sonography, flow-directed pulmonary artery catheters, roentgenography and so on) as time allows.

Prompt pericardiocentesis or emergency-room thoracotomy can be lifesaving for a patient with acute cardiac tamponade and severe paradox, so treatment must occasionally proceed on clinical impression alone (with few if any "studies"), if fatal outcome is to be averted.

Paradoxical pulse is frequently reported "absent" in chronic constrictive pericarditis: Perhaps the chronic elevation of venous pressure causes enough venous distension so that venous return (and thus

cardiac output) remains unaffected by relatively normal respiratory pressure changes. In addition, if cardiac constriction endures long enough to become "chronic," this implies persistence of a "more than minimally-adequate" cardiac output, which is not likely to cause "air hunger."

Summary: The combination of an "absolutely full" venous reservoir and absence of air hunger may help to account for the lack of paradoxical pulse with chronic constrictive pericarditis. Constrictive pericarditis is rarely associated with orthopnea, but dyspnea is usual when the constriction becomes "critical."

Procedure for Cuff Measurement of Paradoxical Pulse

Careful blood pressure cuff measurement of paradox is preceded by a quick approximate systolic pressure reading: During the repeat, more accurate measurement, the cuff is very slowly deflated, starting from a somewhat higher pressure. By auscultation (or less accurately, by radial pulse palpation at the wrist), it is then possible to determine the maximum cuff pressure at which any systolic beat "gets through."

That "beat" represents the strongest heart stroke of each complete inspiration-and-expiration respiratory cycle; intervening heartbeats of each cycle will not bring the arterial blood pressure up to this cuff pressure, although they will be detectable as regular vibrations of the aneroid needle. (Remember, the only time you hear a beat through the stethoscope is when blood flow is intermittent. Above systolic you hear nothing, below diastolic also very little; but you can feel the radial artery pulse as soon as the proximal cuff pressure falls below systolic: Furthermore, regardless of whether flow is intermittent or continuous, the pulse remains palpable down to a cuff pressure of zero.)

Anyhow, as slow deflation of the blood pressure cuff continues down past the highest audible systolic blood pressure reading, more and more of these regular (intervening, lower pressure) pulse wave peaks will get through under the cuff, until a cuff pressure is reached at which all of the pulse beats are heard or felt. This "lower" (all-beats-are-just-now-available) systolic blood pressure is also recorded.

The cuff pressure at which the "strongest" regular systolic beat of each respiratory cycle is first detected, minus the highest cuff pressure at which all the arterial pressure waves can be heard or felt, represents the systolic pressure variation with respiration, or "paradox" (expressed in mm of mercury). Up to 70 mm paradox can be sustained briefly by some patients dying of cardiac tamponade (during deep agonal respirations), but this much paradox without early remedy is a "short lived" phenomenon.

Comment: Cardiac tamponade can be acute, subacute or chronic - and due to liquid, solid or air within (or even outside of) the pericardial envelope. Except with chronic constrictive pericarditis, the amount of paradox usually parallels the severity of cardiac compression: Forceful respirations, hypovolemia, cardiac failure or irregular pulse may simulate or interfere with measurement of paradoxical pulse.

Acute Cardiac Tamponade

Acute cardiac tamponade caused by significant blood "loss" into the intact pericardial envelope (consequent to myocardial or aortic injury or disease, or a coagulation defect), presents as an apparent hypervolemia (full peripheral veins and frequently, a tender, congested liver) combined with shock

(hypotension, decreased urine flow, and tachycardia or bradycardia). In general, when this combination ("plenty of blood but not much blood pressure") develops acutely, it suggests that the heart has suddenly become weak or injured, or that there is interference with blood return to or through the chest; such interference most commonly results from increased pressure within or on the mediastinum, or blockage of the pulmonary artery by a major embolus.

Hemopericardium or cardiac tamponade soon after severe compression of the legs and abdomen (or blast, or other direct closed injury to the chest), supposedly indicates a high likelihood of cardiac rupture and the need for urgent surgical repair. Penetrating cardiac injury with continued bleeding may either lead to early cardiac tamponade (hypervolemic shock) or hemorrhagic shock (obvious hypovolemia), depending upon whether blood lost from the heart wound can readily escape the lacerated pericardial envelope or not.

Pericardial Effusion

Sterile pericardial effusion may be a response to local inflammation or trauma, or a delayed response to irradiation, or a manifestation of some systemic condition such as uremia or a "collagen disease." At present, echocardiography is the most appropriate technique for detecting and quantifying pericardial effusions: Computed tomography, and many other roentgenographic (catheter, dye or isotope) techniques, can also demonstrate abnormal separation of a cardiac chamber from the pericardium (or its adjacent lung).

Viral pericarditis is often suspected in otherwise healthy persons who develop positional anterior chest pain, often with globular en-largement of the heart shadow on roentgenogram of the chest, diminished cardiac pulsation on fluoroscopy and echo evidence of pericardial effusion. Tissue or fluid samples, or increasing blood titers, will occasionally confirm the viral etiology of such a "sterile" inflammation with effusion, which can recur and be most troublesome (and perhaps even cause constrictive changes after many years, although by that time it may be impossible to document the original viral etiology).

Bacterial Pericarditis

In decades past, tuberculous pericarditis was a not uncommon, relatively indolent infection that could lead to chronic thick-walled fibrous (or even calcific) constrictive pericarditis. Non-tuberculous "bacterial" pericarditis was unusual even before modern antibiotics became available; in recent years it has disappeared from my practice (except for one Russian fishing-boat captain who had a supply of inappropriate or inadequate antibiotics with which he insufficiently treated his own pneumonia and empyema). Reportedly, bacterial pericarditis in immunodeficient individuals still comes to surgical drainage on occasion.

It seems evident that any congenital defect or window through the pericardial envelope could allow an adjacent empyema ready access to the pericardial space. Although such a window is understandably difficult to demonstrate in messy "end-stage" bacterial pericarditis, I have twice noted large, congenital, anterior pericardial windows in the course of partial pericardiectomy for relief of cardiac tamponade (from "inflammatory" exudate or purulent pericarditis).

Since both of these patients also had massive pleural effusions of the same fluid, we can presume that inflammatory sealing of the pericardial window edges to epicardium occurred after intrapericardial

extension of the infectious or inflammatory process: The newly walled-off, irritating pericardial contents could then cause tamponade as they increased in volume.

Obviously, it takes more than two open pericardial windows to establish a trend, but we might note that empyema has always been far more common than bacterial pericarditis, and that these two conditions are usually encountered independently. This suggests that intact pericardium is an effective barrier to infection. Might the fat surrounding pericardium even have a protective function against nearby infection, similar to that of the more mobile omentum (which it grossly resembles)?

Less than one percent of sternotomies will reveal a congenital anterior pericardial window ("side-windows" are far less common, if they even occur). This low incidence could increase the significance of encountering pericardial windows in association with my last two cases of massive, subacute inflammatory or bacterial pericarditis in private practice - for a rare pericardial defect should hardly ever be associated with another rare event such as bacterial pericarditis if both occur independently.

Treatment: The treatment of intrapericardial bacterial abscess, with or without acute tamponade, is the same as the treatment of any other walled-off abscess, namely "wide open drainage" (with the urgency of therapy determined by the severity of symptoms). However, the same vigorous fibrosis and contraction of scar tissue that is relied upon to obliterate a small infected pleural space, becomes very undesirable around the heart. Therefore, any major significantly advanced purulent pericarditis is probably best treated by "prophylactic" subtotal pericardiectomy (which results in wide-open drainage into both pleural cavities).

On the other hand, early slightly purulent pericarditis, diagnosed on pericardiocentesis, has often been treated successfully by a "somewhat prolonged" course of appropriate antibiotics (with or without tube drainage, as determined by the patient's course and symptoms). This "medical" experience suggests that early elimination of noxious intrapericardial content can keep inflammation and scar production to a minimum (with little likelihood for later development of constrictive pericarditis).

Poststernotomy wound infection with anterior mediastinitis may also involve pericardium: This condition is usually diagnosed relatively soon after surgery, and treated by simple drainage of infected pockets. Although significant intrapericardial loculations of pus must all be drained, the principal purulent collection is usually located in front of the open or closed pericardium (within the space bounded by both pleural reflections, the heart and pericardium, and the infected sternotomy; see also Chapter 10).

Pericardiectomy is usually unnecessary and undesirable in the treatment of poststernotomy mediastinitis and pericarditis: Not only is late pericardial constriction a very uncommon sequela to such postoperative infection, but subtotal pericardiectomy could easily endanger any "patent" (still open) vein grafts. With or without postoperative infection, I have never encountered (or at least recognized) chronic constrictive pericarditis following sternotomy, although this has certainly been reported (especially as a consequence of povidone-iodine irrigation of the pericardium at surgery).

Pericardiocentesis

Chronic or recurrent pericardial effusions, or an acute pericardial tamponade following injury, can sometimes be "handled" by one or more therapeutic needle aspirations. Using a No. 18 or larger needle, of the length commonly used for "spinal tap" (8-10 cm), subxyphoid insertion is made in a flat manner

that keeps the cephalad-moving needle point close under the sternum. As the needle is advanced, continuous slow injection of local anesthetic keeps its lumen patent and the procedure painless.

It is alleged that echocardiographic guidance has significantly reduced the risk of pericardiocentesis. If echocardiographic guidance is not available, aspiration is attempted after each tissue plane is crossed, until fluid is obtained or one feels the heart bumping the needle tip, usually with a grating sensation. A sterile alligator clip attached to the needle will allow electrocardiographic confirmation of cardiac contact (if such is desired). This is probably most helpful when one is not sure whether blood being aspirated originates from the pericardial space or right ventricle.

If the exploring needle enters the pericardial sac of a patient suffering from cardiac tamponade, immediate clinical improvement may follow successful withdrawal of any fluid. With relief of tamponade and aspiration of all easily obtained fluid, systolic blood pressure vari-ation with respiration (paradox) rapidly returns toward normal (less than 10 mm. mercury). Frequent follow-up examinations (including measurement of paradox) will prevent "silent" reaccumulation of pericardial fluid in hazardous amounts.

Aspiration of only a few cc of fluid, or merely poking several needle holes in a tense pericardium during the attempt, can occasionally provide marked temporary relief. Rapid recurrence of paradoxical pulse, or failure to alleviate tamponade initially, indicates that surgical control of any intrapericardial bleeding, or at least better pericardial decompression by placement of a drainage tube, is likely to be "next on the agenda": Pericardial exploration will also be required when there is a symptomatic intrapericardial clot accumulation, for one cannot drain a clotted pericardial sac by needle.

If the pericardiocentesis needle point is not kept close behind the sternum, it can lacerate thinner-walled, more posterior cardiac structures such as left atrium or coronary sinus. These parts tend to bleed more than ventricles if injured by needle (unless a major coronary artery on the ventricle is slashed by an unlucky stab). The aspirating needle should not have a long or very sharp bevel: If care is used, serious accidents will be uncommon by this approach.

The surgeon must realize that a long needle can easily follow a curved or even tortuous course, depending upon the direction that pressure is exerted on the needle hub while the needle point is advanced: Although a curved needle path is often used to advantage (e.g., for intraaortic placement of a needle at translumbar aortography), it is not always intuitively obvious that a needle tip under the skin will tend to curve more posteriorly as the needle hub is aimed more anteriorly.

Conversely, the best way to keep a needle close under the sternum (especially with a subxiphoid insertion over a fat belly) is to aim the needle hub more posteriorly as the needle is advanced - once the needle point is firmly under the skin. One can demonstrate this concept during insertion of intravenous needles or while delivering local anesthesia, if visible confirmation is desired.

Case Report: Fatal Pericardial Tap

Many years ago, an elderly male with unresectable epidermoid lung carcinoma (asymptomatic except for recurrent pericardial tamponade) underwent regular pericardiocenteses by me without difficulty. Concurrently, he was maintained on low-grade chemotherapy, which he also seemed to tolerate well. On one occasion, at the request of his oncologist, I injected chemotherapeutic drug directly into the pericardial space in a semi-successful effort to diminish the frequency of therapeutic

pericardiocenteses.

Unfortunately, the next pericardiocentesis following this chemotherapy was slightly traumatic, with myocardium grating across the needle tip. "Minor bleeding" from the scraped myocardium simply would not cease (only later did I learn that chemotherapy had eliminated his platelets). Each time that I removed the intrapericardial needle, he gradually redeveloped cardiac tamponade. Twice I reinserted the needle into his pericardial space: Then he, his wife and I all sat and watched it drip slowly for more than an eternity. Finally, I performed a formal thoracotomy to control the bleeding caused by this "minor procedure" (pericardial needle stick), thereby converting the potentially fatal, minor epicardial ooze into definitely fatal generalized bleeding (from all tissues incised at thoracotomy).

Moral: Try to avoid deep needling or other invasive procedures on persons whose blood won't clot. Even if preoperative "bleeding-clotting studies" are often a waste of time and money, I have no desire to repeat the above experience. In retrospect, an initial pericardial window, performed via the subxiphoid approach, might have prolonged his course and avoided need for further pericardiocenteses (all performed in the "good old days," before platelet transfusions became available to us, and before the subxiphoid approach for a pericardial window had been described).

Pericardial Window

The above-described fatality from pericardiocentesis is not unique. Most physicians who are frequently called upon to "tap the pericardium" have had similar disasters. Except in an emergency "acute-tamponade" situation, there is no need to subject patient and surgeon to such risk; it is usually far safer to create a pericardial window (which allows direct inspection and pericardial drainage) than to perform a diagnostic pericardiocentesis.

You may also discover that the referring physician who insisted a patient be "needled" will later develop a memory lapse or regrettably miss attending the meeting at which "your complication" is discussed. "Nowadays" if needling is insisted upon, I usually say, "Please do it yourself. I will be available if you get into trouble." Such a comment by the surgeon often leads the previously intrepid internist to select a safer diagnostic approach (or a more intrepid surgical consultant).

Technique of Pericardiotomy

A pericardial window for long-term decompression of a pericardial effusion is most simply created via a small vertical incision over the xiphoid. Following xiphoid resection, extraperitoneal dissection can easily be carried cephalad along the under-surface of the sternum to the pericardium: The diaphragmatic or low anterior surface of the pericardium is readily entered and drained by this route. However, a more extensive opening of the thorax will be required to gain wide exposure of pericardium and heart.

Simple pericardial window drainage can also be achieved from within or before the peritoneal cavity, or through either pleural cavity, via any opening that provides adequate exposure: The chosen incision is opened, drained and closed in a fashion appropriate for the disease being treated, the condition of the patient and the type of fluid being produced (as well as its anticipated volume and duration of drainage).

Thus, an uninfected non-malignant pericardial effusion can be vented into either or both pleural cavities, into the peritoneal cavity above the liver (not recommended, due to the reported risk of visceral herniation: One might consider covering the drainage opening between liver and heart with a coarse polypropylene mesh to decrease that risk, but I have not done this), into an extraperitoneal pocket under the rectus sheath, or externally by drainage tube (as after sternotomy).

Loculated collections within the pericardium usually require a more extensive procedure. And recurrent inflammation can easily "close" a pericardial window (by sealing its edges to the epicardium), which then allows recurrent pericardial tamponade.

While benefit derived from drainage of a malignant pericardial effusion usually depends more upon the "course" of the malignancy than the drainage procedure selected, it has become increasingly clear that a substantial minority of patients subjected to pericardial window drainage for benign or malignant pericardial effusions, sooner or later develop recurrent effusion or constrictive pericarditis.

Pericardiectomy

The choice of window drainage versus major pericardiectomy thus depends upon the patient's clinical condition, anticipated surgical risk, general prognosis, and the estimated probability of future complications. Without a reliable crystal ball, and in view of the relative ease and safety of definitive pericardiectomy, and considering the greater hazard and frequently less satisfactory outcome of a major surgical attack on a more chronic, constrictive pericarditis, there has apparently been an increasing tendency to perform pericardiectomy rather than window drainage.

At this point, having expressed a preference for pericardial window over pericardiocentesis, and for pericardiectomy over pericardial window, one might anticipate that I would prefer heart transplant to pericardiectomy. Not so. Even pericardiocentesis has its place, and I would still perform pericardial window in preference to pericardiectomy for many poor risk or limited-life-expectancy patients, or to relieve cardiac tamponade within a minimally inflamed or thickened pericardium (when such tamponade is not due to ongoing blood loss, control of which will require optimal surgical exposure).

There are two anterior incisions recommended for pericardiectomy (a posterolateral thoracotomy provides only limited access to the anterior pericardium, and inadequate exposure to the opposite side). A left anterior submammary incision provides optimal exposure of both ventricles, while the median sternotomy approach also is effective, but it does provide less ideal access to the lateral left ventricle: Thus at sternotomy, one must occasionally discontinue cardiac displacement while the blood pressure "recovers" or an arrythmia subsides.

If the left anterior thoracotomy proves too restrictive, it can be extended across the sternum (or even into a bilateral anterior submam-mary transsternal incision, but a sternotomy is far simpler and more comfortable). My own experience has primarily been with median sternotomy, and I have not encountered problems due to this approach.

While it is easy to convert a vertical midline pericardial window incision into a median sternotomy, one can also switch to a left anterior thoracotomy without difficulty, if the attempted window drainage procedure proves unsatisfactory (due to poor exposure or unexpectedly advanced pericardial disease). I would definitely AVOID median sternotomy for pericardiectomy or cardiac repair work following extensive mediastinal irradiation, however, as the sternotomy simply may not

heal (a bilateral, anterior, submammary, transsternal approach is my preference under these circumstances, if cardiopulmonary bypass is proposed or anterior thoracotomy proves too restrictive).

Case Report: Pericardiotomy for Obvious Pericardial Effusion

A middle-aged man with advanced liver disease and peripheral venous congestion was referred for diagnostic pericardiocentesis because of globular cardiac enlargement with diminished pulsation on fluoroscopy - an "obvious pericardial effusion" (in presonography days). I declined to perform the "tap" but offered to stand by while his referring internist did this. We compromised on a subxiphoid exploration by me, at which time an enormously dilated, extremely thin walled, barely contracting heart was discovered, presumably the result of "myocardiopathy" (whatever that is).

Conclusion: The patient died within a few months of "no cardia." Pericardiocentesis could easily have caused disaster in this patient.

Postoperative Cardiac Tamponade

A clotted hemomediastinum undergoes clot lysis comparable to that within a clotted hemothorax, with the important difference that the intact anterior mediastinum or pericardium has a much smaller volume capacity. If anterior mediastinal or intrapericardial clot already is of sufficient volume to cause symptoms (chest pressure, mid-dorsal backache, neck, arm or abdominal complaints) or signs (paradoxical pulse, swollen purplish neck or face, widened mediastinum, and a generally retarded recovery), one must assume that "it will get worse before it gets better," for blood clot continues to gain in volume as it liquifies.

There are times when adequate postoperative mediastinal tube drainage simply becomes impossible; perhaps the substernal tube has clotted or become buried in clot, or it never was properly placed, or it has already been removed. If a drainage tube still functions usefully, however, it is generally best that this tube remain in place for the three, four or five postoperative days required to allow clot to dissolve and drain out (at which point, symptoms and signs return to their normal convalescent state). A similar resolution of the problem can be anticipated if a wide opening was left into the pleural space, through which such fluid can escape from the mediastinum (see Chapter 10).

The poststernotomy patient without a widely opened pleural cavity, who has symptoms and signs of subacute cardiac tamponade (due to a post-sternotomy anterior mediastinal or intrapericardial clot) presents a problem. Assuming that there is not a large collection of clot or liquid blood within a tightly closed pericardium (which will certainly require reexploration), the optimal approach to drainage of the anterior mediastinum now depends upon the state of clot lysis.

If it is only one day after surgery (or even two), and the patient is barely tolerating his mediastinal compression, and more effective tube drainage seems unlikely, one might as well reopen the entire sternal incision semi-electively: A marginal patient is swiftly converted into a normal postoperative convalescent by simply scooping out the clot (followed by copious, warm saline irrigation).

On the other hand, if it is five or seven days after the original sternotomy, and the patient is just now developing significant signs of anterior mediastinal or pericardial tamponade, one can reasonably assume that much or all of the clot has liquified: An easier method of postoperative anterior mediastinal drainage, known as "finger mediastinotomy," is then likely to be definitive therapy.

Summary: Delayed postoperative cardiac tamponade (caused by clot lysis and osmotic capture of fluid within the pericardium and anterior mediastinal space) can usually be relieved simply and successfully in bed by "finger mediastinotomy" through the reopened upper abdominal fascia. When this simple drainage procedure is only partially corrective, and symptomatic mediastinal widening or paradox persist or recur, then reexploration for definitive removal of remaining clot is better done early (but the finger exploration is generally curative). Of course, if a wide opening was created from the mediastinum into one or both pleural cavities, mediastinal fluid will automatically be released as it forms.

Technique of Finger Mediastinotomy

Following careful Betadine wash of the incisional area (with the patient supine or semi-sitting), skin sutures are removed from the lowest 5 cm of the sternotomy incision. Several of the interrupted, upper midline, abdominal fascia sutures (just caudal to the xiphoid) are divided within this sterile field, using sterile gloves and scissors. A Betadine-moistened gloved finger is slipped through this newly created fascial defect and pushed gently up along the deep surface of the sternotomy, anterior to diaphragm, into the anterior mediastinum. Commonly, an impressive gush of bloody fluid is released by this exploring substernal finger, followed by immediate hemodynamic and symptomatic improvement.

There is usually no reason to "poke or scoop about" with the substernal finger, for this is painful and could cause damage: Any accessi-ble liquid should escape readily via the newly opened finger-tract between stiff, edematous, postoperative tissues. More complete and continued drainage can occasionally be achieved by draping a Betadine soaked Penrose drain temporarily into the pericardial space: Perhaps surprisingly, this can drain the stiffened postoperative pericardium (several days after surgery) as effectively as a chest tube on water seal.

Although an occasional disconcerting gurgle may be heard, there should be no "problem" with air rushing in and out of the pericardium, or entering the pleural space to cause pneumothorax (for if pneumothorax did result from finger mediastinotomy, the unsealed pleura-to-anterior-mediastinum opening would already have decompressed the pericardial tamponade without "fingering").

Good luck, careful sterile technique, and repeated Betadine soak of all exposed subcutaneous tissues during each change of the bulky occlusive "post-fingering" dressing, have prevented infection in these open wounds. Although a preliminary "pain shot" can be helpful, both patient and surgeon are usually far more concerned about the patient's apparently imminent demise than the slight discomfort caused by this procedure when gently performed. Local anesthesia is unnecessary and impractical for finger mediastinotomy within a few days after sternotomy, as significant discomfort results only briefly, while fingering the inaccessible retrosternal area.

Surprisingly, finger mediastinotomy does not lead to ventral hernia, even though careless fascial closure at surgery may easily cause such. For the open wound soon narrows down by scarring, leaving little if any evidence that such finger reentry was required. As far as I know, subsequent constrictive pericarditis has not developed in these patients; presumably any Betadine applied to the external wound is washed or drained away before it can lead to pericardial irritation and constriction.

Course of Pericardial Disease

The goal of surgery becomes more difficult to define when the course of untreated or unoperated disease is as variable and unpredict-able as that of non-bacterial pericarditis. Are you trying to cure recurrent episodes of pain or fluid reaccumulation, or remove affected tissues, or only prevent the eventual, insidious onset of chronic constrictive pericarditis? What is the likelihood that chronic constrictive pericarditis will develop? How disabling are the present symptoms? Do they respond to medical therapy? Some questions can be an-swered, some cannot.

To prevent recurrent uremic pericarditis, the subxiphoid approach for pericardial window would seem the best (least) operation to accomplish the desired result, namely drainage and prevention of future tamponade. The same may hold true for acute, serous, post-traumatic tamponade.

Case Report: Tamponade Not Relieved by a Needle Through the Window

A critically ill patient was first seen some weeks after major blunt chest injury. At the time of consultation, he had severe "post-traumatic" cardiac tamponade plus a huge left pleural effusion. An emergency anterior pericardial tap was non-productive so he was taken directly to surgery.

The operative plan was to create a pericardial window through a left posterolateral thoracotomy, and simultaneously to achieve wide pleural drainage. However, when we turned him to the full lateral position, his blood pressure rapidly disappeared, so we turned him back to a better tolerated oblique position that still permitted posterolateral thoracotomy.

As soon as the chest was entered and the four-liter pleural effusion released, he remained stable when placed in the lateral position: A large, posterolateral pericardial window was then created to relieve his high pressure serous pericardial effusion of more than one liter. He did well thereafter.

Other findings: The patient was found to have a large congenital anterior pericardial window which had sealed to the epicardium (this explained why my anterior needle aspiration never passed through pericardium before contacting heart muscle). Except at the sealed window, his pericardial space was markedly distended and free of adhesions or loculated fluid.

Discussion: The depth of cardiac submergence under his massive left pleural effusion (in the full lateral position) undoubtedly brought the pericardial space pressure above his venous pressure. I do not know why this patient developed massive serous pericardial and pleural effusions some weeks after his auto accident. Regardless of cause, however, a trans-xiphoid effort to create a pericardial drainage window would have proven difficult, due to the preexisting large anterior pericardial window that was already sealed down to epicardium by inflammation.

It was a serious error to persist with posterolateral thoracotomy in a patient who tolerated the lateral position poorly. Even if (back then) "posterolateral" was my customary approach to the pleural cavity, an anterior thoracotomy would have provided adequate exposure for both the proposed pericardial window and pleural drainage. In addition, induction of anesthesia and operative positioning would both have been far simpler following a preoperative closed thoracostomy to drain his massive pleural effusion.

Although I proved somewhat adaptable to the circumstances in which I found myself (turning the patient only as lateral as he could tolerate), my proposed posterolateral approach was difficult: In more recent decades, I have seen several deaths result when surgeons were unable to modify their "plan of

attack" to fit the actual patient. More than one patient with post-traumatic hemothorax has died because the surgeon insisted upon utilizing the lateral position: Fatal bleeding after subclavian artery injuries can sometimes be attributed to similarly inappropriate efforts to gain optimal exposure in the face of obvious hemodynamic and time constraints (see also Chapter 16).

Surgery for Viral Pericarditis

When painful attacks of viral pericarditis with fluid tamponade do not respond well to medical treatment, or if they recur frequently, surgical removal of pericardium may be indicated simply to eliminate the possibility of recurrent pericardial tamponade.

There is little use in trying to remove every tag of parietal pericardium (from the diaphragmatic surface, for example). Leaving a strip of viable pericardium under the left phrenic nerve will help avoid injury here, and it seems meddlesome to remove the small patch of pericardium behind the right phrenic nerve (clearly the great veins are not compressed thereby, regardless of what is said "in the literature").

Unfortunately, recurrent bouts of pericardial-type pain often continue long after subtotal or extended pericardiectomy. Whether inflammation continues in the residual pericardial patches on the diaphragm, behind the heart and on the aorta, or if the pain that occurs after surgery is now due to epicarditis, or viral myocarditis or "serositis," remains very unclear (at least to me).

On the Heart Falling Out of the Window

It is essential that any pericardial window be located so that the heart cannot "fall out." Consider that a heart may swing to right or left, and that ventricle size will vary considerably with changes in position, respiration and phase of cardiac stroke: Therefore, avoid leaving any small, potentially "heart size" holes on either lateral aspect of the pericardium (especially the left). When such a defect unavoidably results from surgery, and partial reapproximation seems difficult or undesirable, simply enlarge the defect so that if the heart should flop out, it can readily return, or at least avoid strangulation.

One can even leave pericardium "wide open" from top to bottom. The heart may thereafter remain displaced into the left hemithorax, especially if surgery was a left pneumonectomy, but there seems to be no ill effect from this. A congenital "total absence of pericardium" causes the roentgenographic appearance of a "boot-shaped heart," sticking out into the left hemithorax (simulating the appearance of a tetralogy of Fallot).

Minor Pericardial Entry

Incidental entry into the pericardial space during lung, mediastinal or ductus surgery is common and seemingly innocuous: The first small gush of pericardial fluid can be disconcerting when not expected by a surgeon who is straining to avoid vessel injury somewhere deep within the chest.

Not All Indications for Total Pericardiectomy are Valid

This procedure is indicated to prevent a likely, or "cure" an actual, constrictive pericarditis, or for

optimum drainage of an extensive pericardial-space infection. It is well to be as sure that this operation is necessary as time permits. Insist upon maximal confirmation of the clinical diagnosis by echocardiography, enhanced computed tomography and cardiac catheterization.

One patient who was referred to me two decades ago as "an obvious chronic constrictive pericarditis" (with "calcified eggshell pericar-dium even visible on plain chest roentgenogram"), did not seem a desirable candidate for any surgery (diagnostic or therapeutic). Fortunately for me, he died suddenly while we physicians were still arguing about whether surgery was indicated. At autopsy, he had thin eggshell calcification, true enough, but this represented the entire thickness of his aneurysmal left ventricular wall (plus adherent pericardium).

Be Aware of Venous Overload and Peripheral Edema Before Pericardiectomy

Some patients with chronic constrictive pericarditis are only referred for emergency surgery when their cardiac output has become critically compromised (having thus received "maximum benefit from medical therapy"). These patients must then undergo urgent surgical decompression of their heart in the presence of massive peripheral edema and perhaps, hepatic and renal dysfunction.

Effective diuretics, volume respirators with PEEP, and much experience with hemodilution during cardiopulmonary bypass have improved our skills in dealing with hypervolemic patients. Nonetheless, all involved medical personnel should realize that surgical relief of chronic venous obstruction (here due to chronic constrictive pericarditis) allows sudden mobilization of previously sequestered blood from within the dilated venous reservoir. Obviously, this large volume of "suddenly extra" blood "must go somewhere". And unless unusual blood loss or phlebotomy leave that blood "on the floor" or "in the bag," that somewhere will likely be the lungs - unless prevented by PEEP.

Which is a roundabout way of suggesting that the lungs can become dangerously flooded during or soon after pericardiectomy, if not protected by PEEP and vigorous diuresis, or even phlebotomy. It is also likely that "too rapid" relief of severe chronic venous hypertension can endanger renal and hepatic function (e.g., high tissue pressure of an edematous engorged kidney could easily occlude its own microvasculature or tubules if suddenly relieved of its customary excessive, previously supportive, venous back-pressure). Hence following pericardiectomy for severe chronic constrictive pericarditis, urine output, as well as renal and liver function, is closely monitored.

The critically compromised preoperative patient with chronic pericardial constriction (or acute cardiac tamponade) - with already marginal cardiac output, must maintain a venous blood pressure greater than his intrapericardial pressure if venous return is to continue. Therefore, preoperative attempts to reduce blood volume and tissue edema in such a patient are contraindicated.

It May be Important to Reduce Venous Pressure Slowly

In view of the above discussion, and having seen acute renal failure occur following skillful pericardiectomy, and having read reports about persistently elevated intraabdominal pressures (over about 25 cm water) causing acute renal failure, it seems appropriate and beneficial to reduce the venous back-pressure on core organs rather slowly (when venous pressure has been chronically sustained at over perhaps 20 cm of water), to more gradual elimination of edema from core organs such as liver and kidney. Fortunately, the same positive end-expiratory pressure that we apply to prevent postpericardiectomy "flooding" of the lungs, simultaneously allows the peripheral venous pressure to

subside in that appropriately slow and desirable fashion.

Summary: Diuresis and continuous PEEP can allow a controlled descent of venous pressure over the initial (8-12?) hours following pericardiectomy for severe chronic constrictive pericarditis: Such gradual decompression appears safe, and it could help prevent renal or hepatic failure.

First Left, Then Right

Many years ago, we were taught to relieve left ventricle constriction (during operative pericardiectomy) prior to freeing the right ventricle, in order to avoid a temporary imbalance of stroke volumes that could rapidly overload the lungs. Even with modern anesthesia equipment and effective diuretics, this still seems logical (although intraoperative PEEP would probably remedy that potential problem as well).

Pericardiectomy for chronic constrictive pericarditis can be a difficult, tedious operation, with an ever-present danger of injury to coronary arteries or right ventricle entry. Surgical dissection is most readily carried through the fatty plane in which the coronary vessels also travel (I have seen the left anterior descending coronary artery mobilized over several cm within a pericardial flap that was to be removed, fortunately without harm).

While I have not attempted pericardiectomy during partial cardiopulmonary bypass (femoral vein to femoral artery) or subtotal cardiopulmonary bypass (cannulating an internal jugular vein as well), this might be helpful for intraoperative support of a critically compromised patient, or to correct inadvertent coronary or right ventricle injury.

One should, of course, encourage the anesthesiologist to discontinue significant fluid replacement once the pericardium has been decompressed, in order to simplify the postoperative care of these massively hypervolemic patients. However, an occasional "critical" patient may require large volumes of intravenous fluid until surgical relief of the pericardial constriction has been achieved. The actual level of venous pressure then becomes irrelevant, for if the patient is "in trouble" and improves with fluid therapy, one must support the venous pressure (to sustain his blood pressure) until surgical decompression can be achieved. And expect your Ringers Lactate to cause tissue edema in the meanwhile, for venous pressure in such patients is right at the "leaky capillary" level.

A Free Pleural Space Is Necessary for Drainage of Pericardial Infection

In uninfected patients, the pleura is often free of significant adhesions. Following bacterial infections within the chest, however, vigorous blunt and occasionally sharp dissection can be required to open the pleural cavities for adequate empyema and postpericardiectomy drainage (see Chapter 4).

I usually remove any loose, fatty pleural reflections from behind the sternum along with pericardium, leaving lungs and heart in a single, common pleural space. This allows ideal pericardial drainage via both pleural spaces. At least two large chest tubes are then left within each hemithorax, to support rapid convalescence of the previously moribund patient. Substernal tubes are unnecessary with the widely opened mediastinum that follows subtotal pericardiectomy and mobilization of the lungs. Appropriate antibiotic therapy and the absence of retrosternal fluid under these circumstances, has allowed prompt healing of the sternotomy (despite ongoing infection around the heart and lungs for which pericardiectomy was performed).

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Postpericardiotomy (Postinfarction, Dressler's) Syndromes

These syndromes probably represent varying manifestations of a group of conditions that could have a common or similar final pathway. Postpericardiotomy syndrome is usually associated with activated complement, consequent to blood damage during cardiopulmonary bypass: Onset may be rapid, at any time following cardiac surgery. Dressler's Syndrome can also resemble an autoimmune process, especially when it occurs after myocardial infarctions. We have also encountered it on the day prior to a proposed cardiopulmonary bypass procedure.

In any event, "postpericardiotomy syndrome" is normally manifested by varying degrees of pleurisy, pneumonitis, pericarditis, fever and malaise. An elevated "erythrocyte sedimentation rate" is the most consistent and helpful (though non-specific) laboratory finding.

The usual postoperative variety of these conditions is most effectively treated by steroids; it almost always responds dramatically within twelve hours to prednisone (20 mg po every six hours). After symptoms have abated completely for a day or two, lower dose steroid therapy can be continued as required (usually for a week or so) to prevent recurrence.

Early steroid treatment for this condition, when seen subsequent to coronary bypass, can certainly accelerate the patient's convalescence: Some highly qualified physicians prefer to utilize aspirin and other antiflammatory agents as initial therapy of postpericardiotomy syndrome, but these drugs are definitely "less effective.

There have been allegations that untreated postpericardiotomy inflammatory changes may increase the likelihood of vein graft occlusion, as well as articles reporting the opposite. My preference for steroid therapy is based upon the fact that it makes the patient feel much better and helps him get home sooner, while causing no apparent harm (elevated white blood cell counts of no apparent significance are commonly seen during steroid therapy).

In the past, with an urgent (disabling) but puzzling case, we have resorted to finger mediastinotomy to rule out tamponade, then started immediate vigorous steroid therapy, without encountering healing difficulties or infection of the open wound. Our present mode of bilateral wide pleural entry has eliminated this tenuous indication for finger mediastinotomy after heart surgery (clinical sonography undoubtedly can help differentiate a postpericardiotomy "pericarditis" from cardiac tamponade by bloody mediastinal fluid, but I have not had a chance to try this).

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CHAPTER 16: SURGICAL ASPECTS OF BLOOD COAGULATION

Your hematologist never told you, the things a young surgeon should know;... Fast moving blood is less likely to clot than stagnant blood, unless flow is turbulent;... Some clots remain, others disappear;... Case report: Thrombosed popliteal aneurysm;... Case report: Ongoing femoral artery embolectomies;... Miscellaneous observations (children);... Compression stops blood loss;... Positive end-expiratory pressure reduces bleeding following cardiopulmonary bypass;... Packs control bleeding;... Kelly clamps also may be left in place;... Operative deaths from blood loss;... Access;... Blood loss must be coordinated with blood replacement;... A time to pack and run blood;... Blood;... Case report: Postoperative disseminated intravascular coagulation;... Intra-arterial transfusions;... Anticoagulants;... Coagulation disorders;... After bypass;... Other causes of coagulopathy;... Case report: Coagulopathy after repair of a ruptured thoracic aneurysm;... Case report: Intentional iatrogenic consumption coagulopathy;... Reoperation for post-bypass bleeding;... On reopening a sternotomy in bed to relieve acute tamponade;... How to evaluate drainage of defibrinated "old blood";... Autotransfusion of major hemothorax;... Materials for autotransfusion of hemothorax;... Technique;... Case reports.

Your Hematologist Never Told You The Things a Young Surgeon Should Know

Successful surgery depends upon clotting and healing; neither process is well understood. With blood clots easier to measure than predict, every operation becomes an experiment in clot production or avoidance. Experience teaches us that blood is no more uniform than those within whom it circulates (hence the limited applicability of any "Case Report").

Our primary reliance upon personal knowledge and experience is supported in law. General reference material, for example, regardless of how erudite and up-to-date, cannot provide legal testimony until declared reliable by an "expert witness" (one recognized as both knowledgeable and experienced, who is thus qualified to evaluate conflicting evidence). So much for "medical science," which remains a distillate of conflicting evidence: What have we "distilled" from our years of study and painful observations?

Fast Moving Blood is Less Likely to Clot Than Stagnant Blood, Unless Flow is Turbulent

Confirmatory (and other) observations: A dacron bypass graft of vena cava will reliably thrombose; the same graft in aorta usually remains patent: If experience makes that observation appear self-evident, why is a valve prosthesis in the tricuspid position more likely to clot than the same prosthesis in a mitral position? Or a valved right-heart conduit more susceptible to neointimal hyperplasia than one on the left side?

Frequent dorsiflexion of the foot reduces venous stagnation and thus the likelihood of lower limb venous thrombosis; atrial fibrillation interferes with uniform atrial emptying and is associated with increased risk of atrial thrombus; an anterior infarction of the left ventricle is far more prone to blood stasis and mural thrombus than an equal-sized inferior infarct; mural thrombus is common in abdominal aneurysm (due to lateral turbulence) although aortic occlusion by clotting of an abdominal aneurysm is rare (in fact, purposeful clotting of such an aneurysm allegedly requires blockage of all flow); damage to venous endothelium more commonly leads to vessel occlusion than similar damage on the arterial side (as can be seen by patency rates after venous thrombectomy versus thrombendarterectomy); furthermore, poor arterial "run-off" diminishes flow and increases the risk of thrombosis.

Some Clots Remain, Others Disappear

An uninfected hematoma in otherwise healthy tissue will resolve in such a predictable fashion that its current appearance can be used to estimate the time elapsed since injury (or prior to death). Larger accumulations of blood within tissues or body cavities undergo a similarly steady progression of events, from the original bleeding and clot-ting episode through clot lysis, absorption of intact cells and blood proteins and final disposal of their remaining breakdown products.

Although myocardial infarction is usually caused by a coronary artery thrombus, this clot often disappears spontaneously within hours, days or weeks, sometimes leaving a very small recanalized vessel that may stay open despite persistently reduced flow to the infarcted area. Similarly, a cannulated radial artery will often clot, yet "frequently" reopen later. Clotted veins are regularly returned to service by dissolution of the contained clot (such "postphlebitic" veins often have "lost" their functioning venous valves, subjecting distal tissues to higher venous back-pressure thereafter: More rapid lysis of venous thrombus by exogenous plasminogen activators should leave the valves in better functional condition).

Blood clot is readily disrupted (mechanically) while in its earliest, soft-gel stage: Perhaps it becomes "fluidized" by agitation in the man-ner of a thixotropic gel, or maybe such energy input simply disaggregates clumps of platelets not yet tightly bound by fibrin. Undoubtedly, clot lysis factors from passing blood can help remove clot; however, if passing normal blood could rapidly dissolve clot, there would be no mural clot in aneurysms (where turbulence and activated blood clotting and lysis factors presumably maintain a balance between clot deposition and clot resorption).

Whatever the mechanism, disrupted fresh clot apparently dissolves readily on resumption of full arterial flow, while any "reasonable" amount of agitation has little effect on fully polymerized fibrin (wit-ness the currently-utilized, hemostatic "fibrin glue"). Thus, even a considerable build-up of "jellied whole blood" on the inner wall of a preclotted dacron graft will soon vanish after institution of full arterial flow, without apparent distal embolization. Similarly, following an hour of aortic occlusion (during repair of an abdominal aortic aneurysm, using no heparin), there was often a delay of many hours before the reappearance of bounding peripheral pulses ("spasm" we said, but heparin mostly prevents this delayed return of pulses due to "spasm").

If a fresh, red, "whole blood" clot can usually be relied upon to liquify in one or more days (whether intravascular or extravascular in location), one can reasonably conclude that such clot contains the "seeds of its own early dissolution": Clots or emboli that become "chronic" (usually greyish or dark, shrunken and firm) must somehow be different. Not uncommonly, such grey, persistently solid clot represents the lateral deposition of activated platelets and fibrin from a moving blood stream (rather than jellied whole blood); but regardless of its origin, present location, or constitution, "old" thrombus or embolus must be deficient in fibrinolytic activity (simply because it lasted long enough to become old). A clot deficient in fibrinolytic activity will require supplementation from surrounding tissues or exter-nal sources if it is to achieve timely dissolution.

A large "old" clot has far less exposed surface per given volume than a small embolus, so one might also expect the larger older thrombus to last longer: Indeed, the superficial femoral artery rarely, if ever, reopens by spontaneous clot dissolution, while a radial or coronary artery frequently does so. This suggests that endothelium and passing blood can often overcome fibrinolysin deficiencies within smaller clots.

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It is noteworthy that venous and right-heart thrombi and emboli dissolve more reliably than arterial or left-sided ones, even when large: While one could claim that this was due to greater fibrinolytic activity in the walls of veins, it is equally likely that venous clots dissolve more reliably because they are almost always composed of "fresh whole blood." Dissolution of clot within an entirely blocked and stagnant vessel is presumably based upon increased fibrinolytic activity within this clotted vessel; a fibrinolytic state must persist until the entire column of blood has been liquified and flushed out (as flow is resumed): Otherwise, if stagnation of flow is thrombogenic (as common experience leads us to conclude), one could not explain any vessel ever reopening after it became occluded by thrombus. The effects of stagnation of flow are evident during arterial thrombosis, which is often limited to a segment of vessel located between major branches that continue to provide collateral flow.

It is likely that an atherosclerotic artery (as the blocked superficial femoral artery is usually found to be) has diminished endothelial fibrinolytic activity, increasingly thrombogenic surfaces (especially at ulcerated plaques), turbulent flow (past irregular surface deposits and stenoses), and mural clot deposits rather than fresh whole blood thrombi: These factors are uncommon on the venous side, so the usual persistence of clot in larger arteries (but not large veins, except following venous clipping or ligation) is most likely due, at least in part, to such differences.

Given enough time, it appears, any thrombus will dissolve. Perhaps the fate of each clot is determined not only by relative deposition and dissolution rates, but also by the state and fate of surrounding and distal tissues, as well as the rate of local fibrous tissue ingrowth. Although the undisturbed, atheromatous, superficial femoral artery remains blocked once it has become occluded, it can still be reopened by a proximal streptokinase infusion, which favorably alters the dynamics of "old" or "recent" clot buildup and dissolution (although an excess of streptokinase supposedly prevents plasmin formation).

Larger emboli (old retracted clot in larger peripheral arteries) are resistant to surface attack by normally circulating fibrinolysins. A large, old, embolic clot already has a small exposed surface area compared to its volume: In addition, the surface area exposed to circulating fibrinolysins is limited to the proximal plus distal cross-sectional area of the blocked vessel, regardless of the intraarterial volume of such clot.

Prior to the availability of streptokinase infusions, an embolic blockage of a large vessel was usually considered a surgical emergency, unless one was willing to accept permanent occlusion (occasionally an "old" clot might still move distally, however, due to clot shrinkage, surface dissolution, vessel dilation or whatever). We now have a choice, especially in arteries such as the popliteal or tibial, where embolectomy is likely to be less effective than thrombolysins. Plasmin activation, by streptokinase, urokinase or tissue plasminogen activator, is alleged to be most useful within five days of thrombosis or embolus, and less effective after ten days (it is currently considered unsafe within ten days of an operation, which limits its applicability after surgery).

Summary: Continued arterial obstruction can be expected following an embolic occlusion by "organized" clot (as the length of time required for dissolution of such "mature" clot allows continuing stagnation to lead to ongoing local intravascular thrombosis). Although any clotted hematoma within a body cavity or living soft tissue will eventually dissolve (if not walled off or constantly replenished), loss of peripheral tissue viability and fibrous ingrowth are additional time-related factors that may overwhelm "natural" clot dissolution when mature, embolic, intraarterial clot is involved.

Streptokinase or tissue plasminogen activator infusions can favorably affect the dynamics of clot deposition and dissolution: Although their usefulness and optimal methods for application are still being clarified, they are alleged to be especially helpful with deep venous thrombosis (a 3 or 4 day course is supposed to help prevent destruction of venous valves). A single dose of warfarin (which stops production of "vitamin K dependent" protein-clotting factors), given before discontinuing streptokinase, is normally sufficient to establish an anticoagulated state following streptokinase depolymerization of circulating protein-clotting factors (according to the literature).

Case Report: Thrombosed Popliteal Aneurysm

A 75 year old male had successful bypass graft (Gore-Tex) of a huge popliteal aneurysm that had obstructed venous return and caused phlebitis of the lower leg: Surgery was also recommended for the opposite popliteal aneurysm, which finally thrombosed two years later (causing twelve hours of severe ischemic symptoms, for which medical attention was not sought). Another year later, recurrent ischemia with impending gangrene (on the non-operated side) led to an angiogram that showed no runoff below the knee on that side. Streptokinase infusion relieved this recurrent, severe ischemia within hours, although it had no effect on the year-old organized "mural" clot within the aneurysm.

The patient remained well and ambulatory for another year, an angiogram still showing one open collateral vessel below the knee on this non-operated side. A second episode of thrombosis was again treated successfully with streptokinase. Eventually he required emergency bypass for recurrent ischemia, which was unsuccessful and led to amputation (once again suggesting the long-term value of elective bypass for significant popliteal aneurysm, even when such aneurysm may pursue a most indolent course).

Case Report: Ongoing Femoral Artery Embolectomies

A 60 year-old bulldozer operator with a known (apical) left-ventricular aneurysm entered the hospital with a severely ischemic leg. Femoral embolectomy (for removal of grey-white, laminated, "old organized" clot) restored his pulses. The following day he developed acute aortic occlusion. Repeat embolectomy (bifemoral) extracted larger amounts of the same material with good return of pulses. Loss of pulses was again noted while moving the patient from the operating table. Repeat embolectomy was performed at once, and also again within 30 minutes. Total volume of clot removed was hundreds of cc.

The patient did well thereafter, presumably having finally excavated all of the laminated clot from his left ventricular aneurysm (fortunately these large chunks went to his distal aorta and iliacs, not to aortic arch branches or visceral vessels). He was back driving his bulldozer within 10 days.

One wonders if streptokinase-induced fibrinolysis of emboli will inadvertently also increase the normally low "release rate" of such large mural thrombi from ventricular or abdominal aortic aneurysms. (Surprisingly many of the intracavitary thrombi visualized soon after anterior myocardial infarct will disappear without apparent embolism; another indication of the dynamic state of intravascular coagulation, or possibly of the ability of the body to accept some emboli without complaint.)

Miscellaneous Observations (Children)

Pediatric patients bleed less, heal more quickly, usually calcify homograft valves more rapidly and sometimes develop severe, resistant, post-traumatic arterial spasm that can lead to arterial thrombosis and tissue necrosis: Perhaps regional infusions of heparin or vasodilators (e.g. "a calcium blocker" or nitroglycerine), or the use of thrombolytic agents (if indicated by angiography) will decrease the likelihood or extent of this unusual but dreaded complication.

Compression Stops Blood Loss

Bleeding can always be stopped if external counterpressure can be applied effectively (by gauze pad, hemostat, pneumatic compression, tourniquet or whatever). Ongoing blood loss into or from organs such as kidney, liver, spleen, pancreas, brain or lung often presents difficulty in this regard, as direct pressure on these parenchymal organs usually causes further tissue disruption and bleeding, especially when their capsule has been torn - for only their capsule, vessels and ducts have useful tensile strength (and often not much). However, it is reported that even severe liver rupture can frequently be treated by an initial gauze packing, with elective repair 12 to 24 hours later, in a warm, stabilized, often no-longer-bleeding patient who now has normal coagulation parameters (see below).

Retroperitoneal bleeding, especially from the pelvic area, can be exceedingly difficult to control by any direct means (except possibly proximal vascular ligation or intraarterial embolization), especially when bleeding is associated with significant pelvic injury or tumor. On rare occasion, bleeding from a bulky, friable malignancy is only controllable by scooping out the entire lesion and placing packs under pressure, or by vascular embolization. One usually prefers to avoid entering a large retroperitoneal hematoma secondary to massive blunt trauma; on the other hand, penetrating injury that causes a large retroperitoneal hematoma is more likely to require direct surgical control of the bleeding vessel.

External compression of legs and lower torso (as by a pneumatic counterpressure garment) increases the functional blood volume and improves venous return by reducing peripheral venous blood storage. Such a compressive exoskeleton also controls blood loss and prevents further damage by stabilizing disrupted tissues during patient transport and evaluation. Not uncommonly, such a garment will only be loosened over the abdominal area (for access) when "the time has come to cut," for a rapid drop in venous return and arterial pressure can accompany the vascular bed expansion and increased bleeding that often results from deflation of the MAST suit, especially with a still-hypovolemic patient.

Perhaps an inflatable envelope to slip over an injured liver or spleen might stop bleeding here in similar fashion, again without causing necrosis of underlying tissue. As mentioned, surgeons have traditionally "packed" large gauze pads into or about bleeding areas to stem blood loss that seemed about to become fatal; high levels of positive end-expiratory pressure (PEEP) also can sometimes control or reduce mediastinal blood loss (by utilizing tightly inflated lungs to apply mediastinal compression).

Positive End-Expiratory Pressure Reduces Bleeding Following Cardiopulmonary Bypass

We have been favorably impressed with the use of PEEP to slow early postoperative bleeding and diminish mediastinal blood clot accumulations: 10-20 cm positive end-expiratory pressure is routinely applied as soon as the sternal halves have been reapproximated, and this is usually maintained for at least 4-6 hours thereafter. When well tolerated, such PEEP confirms an adequate blood volume, for a "small" blood volume would tend to accumulate peripherally in otherwise empty veins, rather than enter the chest against this sustained, above-atmospheric pressure (especially when the patient is not

yet moving, and thus not "muscle-pumping" venous blood toward the heart). If not well tolerated, PEEP is decreased or discontinued promptly until further volume replacement can be carried out, then gradually reinstituted.

Once a patient enters the intensive care unit (still on breathing assistance), any significant mediastinal widening or chest tube drainage of clotting blood is considered to be an indication for "More PEEP!" (rarely even up to 35 cm water back-pressure, which is the maximum setting available on our current respirators). Any decrease in tolerance by such a patient for PEEP (a drop in BP and pulse, for example) may signify ongoing blood loss and possibly cardiac compression by clot. Hypovolemia early after heart surgery (especially if due to intra-pericardial blood accumulation - see also Chapter 15) may be associated with bradycardia and dysrhythmia - unlike the normal response to blood loss which is tachycardia.

PEEP thus helps the surgeon identify and maintain a "full" vascular system, which is helpful and reassuring information and an especially desirable state early after heart surgery. Mild hypervolemia has little if any risk at this stage, for unnecessary fluid will be excreted rapidly while the patient is still on ventilatory assistance. In addition, red cell survival is diminished after the trauma of cardiopulmonary bypass, so progressive anemia is anticipated for the first week or two after surgery: Thus any transfused blood is likely to be helpful (which, of course, is not a plea for unnecessary transfusion; it merely implies that if you gave a little too much blood acutely, it can still be useful).

In the meanwhile, any acutely detrimental effects of fluid overload can be reversed by PEEP. Thus, during acute fluid losses and resuscitation efforts, volume infusions can reasonably be based primarily upon the need to maintain an adequate peripheral blood pressure. Low-dose dopamine, or some other boost to arterial pressure and renal blood flow, can then be applied to the patient who is obviously "full," yet still not perfusing.

Pulmonary artery and central-venous monitoring and infusion catheters can aid in the evaluation of an unstable patient, but their placement and analysis should never delay or interfere with essential blood replacement during an emergency. Our anesthesiologists sometimes prefer to transfuse directly into a large vein through the large introducing sheath for the pulmonary artery balloon catheter, and then to float the balloon catheter into position later, if it still seems likely that "fine-tuning" will be required (when there no longer is need for massive volume replacement).

In any case, the results reported on blood samples taken from an acutely unstable, bleeding patient are often irrelevant (even if reported promptly) and accurate wedge-pressure measurements can not only be difficult to take and interpret in an emergency, but they often distract from necessary resuscitative efforts as well. In addition, a number of deaths have been reported from catheter-induced dysrhythmia or pulmonary artery perforation by the balloon in wedge position, especially in the heparinized patient on bypass (if the catheter becomes stiffer with hypothermia, it could damage a peripheral pulmonary artery more readily when the heart is pushed into the awkward positions required during surgery).

The 170 micron "blood transfusion" filter is supposedly preferable to one with smaller pores, as large flows can be hard to push through the latter; allegedly one can induce activated clotting factors and distal-side-of-filter aggregates when using smaller pore filters for transfusion. As this apparently is not a significant factor when heparinized blood is forced through the even smaller pore size (20-40 micron) filter during cardiopulmonary bypass, it is again evident that citrate does not "block" clot

activation as well as heparin.

In some cases with poor coagulability of blood or generalized oozing, high PEEP (15-25 cm water) has helped us avoid reoperation: It may stop mediastinal blood loss practically at once, or it may have no apparent effect (especially on a brisk arterial bleeder in an unstable patient early after heart surgery). One can vary the PEEP setting continuously (along with intravenous antihypertensives or pressors or additional blood volume) to help maintain blood pressure in the desirable range, using PEEP as another "control" to help "fine tune" the acutely unstable, early postoperative patient.

The only complaint I have been able to associate with high PEEP (and that was really high, 35 cm of water applied for about eight hours continuously to avoid reoperation) was volunteered by one patient who stated that he was short of breath because his lungs felt "too big." This symptom disappeared within several days, but it certainly was understandable. We routinely maintain 5 cm water PEEP until the patient is extubated, usually about 12 hours after heart surgery, as this seems to reduce postoperative atelectasis while on the respirator.

An occasional patient will not tolerate PEEP above a certain level, regardless of blood volume. If the patient seems stable at 15 cm PEEP but cannot tolerate 20 cm PEEP, it could well be that the higher pressure has an adverse effect on heart position (kinking vein grafts, altered right heart afterload, left ventricle geometry or whatever) and should be avoided. PEEP control of mediastinal hemorrhage is merely another (not necessarily the best) treatment option.

Summary: Bush pilots say, "Always leave yourself an out": It is nice to be aware of alternate methods of patient management, even if you personally find them distasteful. (Reportedly, a 10 cm PEEP application postoperatively has no effect on postoperative mediastinal blood loss; this finding does not conflict with our experience of apparent benefit at higher PEEP pressures.)

Packs Control Bleeding

Undisturbed clot (under a gauze pad, or on a raw surface) supports continued hemostasis (by adhesion, clot shrinkage and chemical factors that induce spasm and thrombosis of divided vessel ends). For these reasons, a gauze pad usually need not be applied at anywhere close to the arterial systolic pressure in order to slow or stop bleeding (which is why large gauze packs, crammed into the chest, abdomen or other space, can sometimes control otherwise "uncontrollable" blood loss.

At times, such pads will only need to remain in place for five or ten minutes or an hour; at other times, one may be forced to leave them in place for one or two days while the patient stabilizes under intensive care (and coagulation factors return to normal). If bleeding persists (but at a diminished, replaceable rate after such packing), the chest wound can be left open (to provide space and easy egress for accumulating blood, and to minimize compression or displacement of lungs and heart by clot): The patient is then returned to intensive care (with a loose Betadine-soaked dressing covered by larger, Betadine--soaked towels until reoperation). Even when covered in this fashion, an open body cavity will noticeably increase the patient's overall heat loss, so an unpleasantly hot ICU room and warming blankets may be essential.

On the other hand, abdominal closure usually helps keep viscera and such large hemostatic pads in position: Acute compression of abdominal organs only becomes risky if it jeopardizes venous return or visceral viability (see Chapter 1), unlike the hollow viscera of the chest which must also move and

expand if the patient is to survive. In addition, of course, abdominal viscera are likely to hang out from the open abdominal wound, while heart and lungs are more tightly tethered within the chest: Although intestine that hangs out untended can become dry, dirty, congested and even dead, a properly exteriorized ileum or colon certainly does well as an ileostomy or colostomy. It seems that mucosa resists drying, while a dry serosa may lead to loss of the local "trans-serosal" blood supply.

Operative removal of packs from the chest or abdomen is usually scheduled for a day or so after their placement (or sooner, if necessitated by increasing symptoms due either to the packs or enlargement of adjacent blood clot). Some surgeons try to pack with long thin gauze pads that can be drawn out through a small opening left in the neck or abdomen, thus avoiding reexploration: Perhaps reinspection and irrigation (followed by appropriate wound closure) is unnecessary, but it is usually very reassuring.

Kelly Clamps Also May Be Left in Place

At nephrectomy, tuberculous kidney vessels were sometimes too friable to withstand ligation. If unintended vessel amputation occurred during attempted ligation, the remaining vessel length might barely be adequate to allow the surgeon to return the patient to his hospital ward bed with large Kelly clamp handles protruding from the partially open flank incision. Those clamps were generally left in place for about 10 days (meanwhile the patient was encouraged to "Lie Still!"): Thereafter, the clamps were "eased off very carefully." At present, the "delicate" application of multiple large Hemaclips is frequently safer than any attempt to ligate friable vessels deep in a wound (and also far easier than the Kelly clamp ritual described above).

Operative Deaths from Blood Loss

Unfortunately, it is not uncommon for a somewhat hypotensive individual (with rupturing abdominal aneurysm, gunshot, lacerated liver, or pelvic or retroperitonal bleeding) to survive one or two hours of emergency transport and evaluation, only to die thereafter during surgery, of possibly avoidable events related to blood loss. In such a "Bleeding Emergency!" the preoperative routine should include establishment of safe routes for massive blood transfusion: Clearly these routes must bypass any injury or potential operative site (if practical), so that transfused blood reaches the heart through open, unobstructed, uninjured veins (and is not simply blocked, or worse yet, spilled, a few cm farther along the course of an injured vein).

A bladder catheter will be an essential monitoring device during and after resuscitation. Mannitol and Lasix should be used freely (see Chapter 14); tetanus toxoid is administered to injured patients as soon as possible. Antibiotics are almost always indicated when severe injury and blood loss are associated with surgery.

We are told that hypothermia alone can impair platelet function and cause other coagulation defects (that obviously will not be detected on any routine analysis of coagulation factors). Hypothermia also can endanger tissue oxygenation by increasing the affinity of red blood cells for oxygen (apparently this is especially true in "older" stored red cells that are depleted in 2,3,DPG): However, the acidosis that results from poor tissue perfusion or from any decrease in citrate and lactate metabolism - as after multiple transfusions of a hypothermic patient - will improve oxygen release from hypothermic red cells.

It is also reported that a decrease in serum calcium accompanies hypothermia, and that this accentuates the depression of myocardium by low temperature. Hypothermia and acidosis both cause potassium to leak from the cell, which in turn makes hypocalcemia more of a myocardial depressant. While acidosis is a common terminal event, some authorities point out that "alkalinizing" may be harmful, for citrate is soon metabolized, so the massively transfused patient usually becomes alkalotic anyhow, after adequate resuscitation. Allegedly it is only those patients who cannot be resuscitated that stay acidotic; such patients will respond to more blood and better perfusion, but not to persistent administration of sodium bicarbonate (which has numerous other adverse effects in higher doses).

You will note a lot of "theory" above; however, as a practical matter, one should adequately replace massive blood loss, check urine output, prefer fresh blood for multiple transfusions, avoid elevated serum potassium levels, give calcium chloride if indicated and keep the patient warm. High serum citrate levels in a hypothermic, hypotensive, hypovolemic, multiply transfused patient may result in a high serum calcium with a low "ionized calcium"; the latter being the "effective" form of calcium. Citrate elimination occurs rapidly with restoration of normal blood flow and hepatic perfusion. Similarly an elevated serum potassium should be considered in terms of body potassium balance, good perfusion will usually decrease serum potassium levels rapidly (as can diuretics, insulin and glucose infusions, certain resins given by enema, and dialysis).

Arrangements required to prevent and correct hypothermia include warming blankets, a warm operating room, a heated nebulizer for the anesthesia ventilator, administration of body temperature blood and electrolyte solutions, and large quantities of hot, sterile saline for irrigation of the peritoneal or pleural cavities (if open) or even for irrigation down the nasogastric tube. (I have never tried to rewarm a patient's stomach like this, but it could have some minor beneficial effect on overall body temperature, or at least on nearby heart and liver metabolism; see Chapter 3.) Vigorous transfusion can often be delayed as long as the arterial blood pressure remains stable and above the "minimal perfusion pressure," to help conserve the patient's own remaining blood until surgery can be undertaken.

If at all possible, preparations are completed so that many blood transfusions are readily available, and arrangements are made for "lots of help pumping blood," especially during induction of anesthesia (when cardiac output often drops precipitously due to muscle relaxation, generalized vasodilation and interference with venous return by positive-pressure ventilation) and while the abdomen or chest are "opened" (for such cavity decompression often accelerates bleeding, even before exploratory manipulations can dislodge clot).

Access

There is no sense in exchanging a critically compromised patient's remaining measurable blood pressure for the better exposure that might be gained through turning him to the lateral position for thoracotomy. If the lateral position is unlikely to be tolerated, perform an anterior thoracotomy at the level indicated. Excellent exposure is possible with a submammary incision and the chest retractor opened widely. (Ribs break and rip loose and it looks terrible, but closure is simple and this chest incision also heals well.)

Do not let inaccessibility of bleeding disturb you: Simply "Go for it!." Even if clavicle must be cut or removed, or an ugly scar extends onto the breast, or pectoral muscle must be divided high, or a bullet through the lung apex and subclavian artery means that the bleeding bullet hole and hemothorax are best approached by some unusual route: Just do whatever is reasonably required to prevent death by

blood loss. It is well to remember, however, that standard incisions became that way for a reason (for example, multiple divided rib ends are very difficult to retract for good exposure: Better access is usually achieved between ribs, sometimes at more than one level).

Although proximal control of a bleeding vessel may soon become critical for hemostasis or to facilitate repair, direct local pressure (perhaps through an enlarged bullet wound onto the subclavian vessels) can meanwhile allow survival, by control or reduction of the ongoing, unacceptable blood loss; someone sturdy and smart enough to understand the importance of maintaining continuous compression should be assigned to this task. An additional, more appropriate incision may then be necessary for satisfactory repair. Just remember, "We don't shoot 'em, we just fix 'em" (and some are rather "messed-up" before we get 'em). In other words, a patient cannot always expect a standard repair of a substandard stab or bullet wound.

It is well to review possible emergency approaches to bleeding from various sites before the problem presents. As a general rule, for example, the lower the pressure within a lacerated vessel or heart chamber, the less blood will be lost, and also the less likely it is that the first sutures placed will "tear out" and enlarge the problem defect. Close coordination with a superb anesthesiologist, who is able to regulate blood pressure "on request," will make you appear far more skilled than the surgeon who simply does "his own thing" without realizing that he is but "one among equals" on the surgical team. The competent and forewarned anesthesiologist will be ready to administer nitroprusside, nitroglycerin, isuprel, calcium, dopamine and epinephrine in timely fashion.

We often drop the arterial pressure to 50 or 60 mm mercury, for example, when placing or tying an important suture in friable aorta; a quick flush of nitroglycerin has proven very safe and satisfactory for this. Another technique that is recommended for left ventricle repairs - if cardiopulmonary bypass is not readily available - is momentary inflow occlusion (I have not tried that, and would worry about air entry into a partially emptied left ventricle). In any case, vasodilator therapy has certainly been lifesaving for many of our patients (including those having their descending thoracic aorta cross-clamped without a shunt or cardiopulmonary bypass).

Specifically worth consideration before an emergency arises is how best to gain access to an injured subclavian artery in an unstable patient who has just been shot or stabbed. The subclavian is mentioned again because I have encountered numerous unnecessary deaths of patients who entered the hospital alive "but bleeding bad," and then died during mental or mechanical delays induced by the fact that the area of injury might be most accessible via either a posterior or anterior intercostal approach, or on the other hand, an incision above, through or below the clavicle, or perhaps a sternotomy...

For this "mental exercise," one should recall that the innominate and right subclavian arteries are more or less anterior and readily accessible by a sternal-split, as is the ascending aorta. On the other hand, the ductus, left subclavian artery and distal aorta arch are more or less posterior structures that are poorly accessible by this approach. The proximal left subclavian artery can be reached by an anterior third interspace thoracotomy or a posterolateral fourth or fifth interspace incision (but it is not uncommon for a bleeding, unstable patient to "arrest" when he is turned for posterolateral thoracotomy, so this becomes another argument for the anterior approach).

The mid-portion of the left subclavian artery is accessible from above the clavicle after dividing neck muscles to clavicle and first rib (watch for the phrenic nerve): A distal left or right subclavian artery can be approached from below the clavicle, through the pectoral muscle or anterior axillary fold,

as during axillofemoral bypass (have a care for brachial plexus nerves). If necessary, a partial or subtotal clavicle resection is quick and well tolerated. When approaching the right innominate and subclavian arteries by a sternal split and neck incision above the clavicle, watch for the recurrent laryngeal nerve. Intraluminal balloon-tipped (Fogarty) catheters can be a great help in obtaining early proximal and distal control of blood loss from any point on either side of the vessel injury.

Young surgeons are taught to preserve the left innominate vein during sternotomy: This was not always so. In the early years of heart surgery, the left innominate vein was commonly ligated during sternotomy (without observable sequellae, so not every damaged vessel need be repaired, nor should undue time be wasted in dissecting out the left innominate vein).

Blood Loss Must be Coordinated with Blood Replacement

Cardiac arrest due to loss of essential blood volume is far more easily prevented than reversed. An empty thoracic aorta can be hard to differentiate from esophagus, but by that time it usually does not matter whether (or even which structure) you finally cross-clamp.

The two biggest errors made by surgeons operating upon severely injured patients are:

1) Trying to see the source of blood loss clearly before stopping the blood loss, and

2) Not coordinating "technically necessary" blood loss (during visualization and repair) with actual (not the "desired" or "possible") blood replacement by the anesthesiologist.

One will often be unable to see the source of severe bleeding clearly (without first reducing or controlling that bleeding) until the patient is dead. Surgical manipulation prior to control of bleeding usually increases the severity of blood loss. Severe blood loss simply cannot be replaced in timely fashion by any anesthesiologist (plus any number of helpers) unless it is intermittent; so the surgeon must avoid "tunnel vision" and restrict surgical blood loss to sustainable levels. This often means that one must alternate between exploration (or repair) and "time out for compression" of the bleeding area or its vascular supply.

Even some highly skilled surgeons are simply not emotionally suited to succeed with this type of problem, and they should recognize that fact and avoid being repeatedly responsible for unnecessary deaths. One objective of this chapter is to establish the need for "common sense." Until wide experience has been gained, proper "surgical arrogance" - a calm and confident approach in the face of extreme adversity - must be based upon meticulous mental preparation (such as "Now what would I do if... ?"). Undoubtedly, most well-trained surgeons could repair most injuries, given enough time and sterile textbooks, but patients are often "long gone" by then.

It is usually most reasonable to make a long midline incision for abdominal exploration, then rapidly lift out small intestine and clot to gain exposure of the injured or ruptured vessel. Simple palpation (deep within an abdomen still filled with blood and clot) can often identify the source of bleeding most speedily (ruptured liver, kidney, spleen, aneurysm, whatever). Remember, in a true emergency, one need not see the spleen to resect it: Not only can all abdominal and thoracic organs safely be lifted toward (their major connections to heart and great vessels in) the midline, but access to retroperitoneal vessels and organs is often improved by rapid mobilization of overlying viscera toward the rnidline as well.

Spleen: If, on palpation, spleen feels torn and presumably is the source of ongoing, life-threatening blood loss, simply loosen and remove the spleen from the left upper quadrant with one smooth "scoop and pull" motion that brings it up into the long midline incision. The splenic pedicle is then gently compressed between fingers until its vessels can be identified and secured individually. (Although splenic preservation is always an appropriate consideration, it becomes increasingly irrelevant as the blood pressure approaches zero.)

Minimal bleeding from the raw splenic bed (including greater curvature of stomach) is swiftly packed and easily corrected later (any splenic capsule left behind is of no importance). Undamaged tail of pancreas is often mobilized with spleen to the midline in this fashion. Although pancreas is rarely, if ever, harmed during such mobilization, it can easily be damaged by large clamps and ligatures placed hastily across the splenic pedicle thereafter, when the need for haste usually has passed (several injuries to multiple organs can certainly contribute to a "belly full of blood," but patients with severe bleeding from more than one source usually do not make it to the hospital).

Liver: A ruptured right lobe of liver is easily compressed and simultaneously brought into view (toward the midline incision) by the support of many lap pads jammed behind it in the right upper quadrant (delete one pad when the blood pressure suddenly drops due to kinking or compression of the inferior vena cava). Any bleeding within liver parenchyma can usually be controlled by gently squeezing the liver between the two hands of that same sturdy reliable assistant, while the anesthesiologist again catches up with surgical blood loss.

The surgeon sees blood loss, while the anesthesiologist only measures its effect: Common sense and common courtesy suggest that the surgeon must keep the anesthesiologist closely informed on actual and impending changes of blood volume and blood pressure. It may be his digital readout, but your patient will benefit if the busy (or dozing) anesthesiologist has his attention politely drawn to important or acute changes (it is much easier to win if you don't get behind).

The surgeon should develop his personal monitoring skills by briefly but frequently palpating a nearby significant artery and comparing his estimate of blood pressure to the value displayed on the arterial pressure readout. Then, on those too frequent occasions "when the bottom falls out," he can readily determine if the apparent hypotension is "for real" or simply represents a technical malfunction. (All too commonly, an ill but normotensive patient is subjected to vigorous, unnecessary and hazardous pressure-elevating-therapy on the basis of some monitoring defect.)

Blood loss from the liver can often be diminished to sustainable levels by the "Pringle maneuver" (a vascular clamp placed across the porta hepatis vessels and common duct). Allegedly this interruption of hepatic arterial and portal venous inflow can be tolerated for at least an hour at normothermia: Suggestions for improving the ischemic tolerance of liver include massive doses of steroids prior to "Pringling," and also local organ hypothermia (however, the latter seems hazardous for the usual injured patient who is already in "impending hypothermic cardiac arrest," and I have not seen local hypothermia utilized under these circumstances).

Large volumes of dark blood issuing from deep to the liver suggests injury to a hepatic vein or the inferior vena cava. Attempts to gain adequate exposure for repair here are likely to result in exsanguination, or to require cross-clamping of the inferior vena cava (which can fatally diminish venous return). A suggested method for inferior vena caval bypass during hepatic vein or inferior caval repair involves placing an internal stent (an appropriately side-holed, #36 chest tube) through the right

atrial appendage, with tourniquets placed on both sides of the hepatic-level caval lacerations (just above the renal veins intraabdominally and also around the intrapericardial inferior vena cava).

A large sterile endotracheal tube inserted through the right atrial appendage reportedly is also effective if it can be passed beyond the I.V.C. laceration and the cuff balloon then inflated (to block the cava around the endotracheal tube, just above the renal veins) : Proximal exclusion of the hepatic venous portion of the cava again depends upon an intrapericardial tourniquet around the inferior cava and its indwelling "endotracheal" tube.

This tube also must have an extra side-hole created to allow blood egress within the atrium: The open superior end of the tube can be clamped just outside of the atrial appendage. Caval and hepatic venous repair can then be carried out without loss of visibility or patient exsanguination (apparently over half of these patients still die during surgery, however).

Direct control of intrahepatic bleeding can be achieved by capsule cautery and then the "finger-fracture" method (liver parenchyma is crushed between thumb and finger to carry the dissection to the site of bleeding: Meanwhile, vessels and ducts encountered en route can be sutured with fine prolene or controlled by hemostatic clips). Final debridement of dead tissue is carried out after release of the vascular clamp on the porta hepatis: Surface ooze usually responds to gauze packing (with or without oxidized cellulose or other preparations that stimulate blood coagulation). Living omentum is then placed against all raw liver surfaces and the area widely drained.

Reportedly, one can successfully tamponade a penetrating knife or bullet wound of liver simply by pulling several Penrose drains into or past the bleeding site (attached to a soft rubber catheter, for ease in handling). This "fill-the-hole-with-drains" method allegedly allows rapid control of bleeding without major dissection into (and resection of) liver; it utilizes as many large Penrose drains as needed to achieve tamponade: These are then left protruding from an abdominal drain site, loosened at about one week, and removed as indicated by the drainage of bile and old blood, often after many weeks.

Abdominal Aorta: Rarely, a ruptured abdominal aorta is best controlled initially by plugging the proximal aorta with an exploring intraluminal finger (which both controls bleeding and guides the infra renal-artery aortic vascular cross-clamp into proper position). A vascular clamp placed "superficial-to-deep" can safely be used to control either common iliac artery without separating the iliac vein from the artery (formerly an occasional cause of significant "technical" blood loss), but avoid including ureters when clamping iliac vessels more distally.

Fogarty has developed an intraluminal inflatable blocker for aorta as well as more peripheral vessels (this sounds like an excellent idea, but I have not used it). One gathers that emergency room thoracotomy solely for cross-clamping of the thoracic aorta (in order to control intraabdominal blood loss) does not greatly improve the likelihood of patient survival: On the other hand, an emergency thoracotomy undertaken to remedy intrathoracic blood loss is reportedly more likely to be successful.

A Time to Pack and Run Blood

While trying to identify and control a source of major intraabdominal blood loss, the surgeon inevitably "stirs up" bleeding through manipulation of damaged tissue and removal of obscuring blood and clots. Often, by the time such ongoing blood loss can be remedied, the patient has been resuscitated with great volumes of salt solution and bank blood and will no longer have the "right stuff" within his

vascular tree; his blood now runs cold, dilute and incoagulable. At that point, despite commendable progress in control of "surgical" blood loss, the "medical" blood loss (a "red tide") usually rises dramatically, and the anesthesiologist then falls increasingly behind in volume replacement.

Hypovolemia and hypothermia often lead to hypotension and then death of the severely injured patient, even with "everything repaired." Hypothermia following injury is due to many adverse factors, such as large volumes of room temperature (or even colder) fluids given intravenously, a combination of direct (conductive, radiant and convective) and evaporative heat losses, and presumably a marked diminution in metabolic heat production as well.

Correction of such a hypothermia can be very difficult or even impossible (despite hot saline irrigations, warming blankets, warmed intravenous fluids, heated nebulizer on the anesthesia machine, and an unpleasantly warm operating room), except by early completion of the procedure and wound closure (or by instituting cardiopulmonary bypass). Although we have all survived many episodes of cold skin without measurable change in our core temperature, heat loss from an open body cavity affects the patient's core temperature directly, so cold viscera usually means cold blood and a cold heart.

Conclusion: In any event, when faced by a cold, bleeding, oliguric patient at surgery, consider that this may be the time to "pack" and stabilize, or even "get out" (when that is a "viable" option). Elective reexploration of a warm, stable patient, a day or two later, is an entirely different matter; gentle removal of packs may then be all that is required.

Blood

Bank blood binds calcium chemically, thereby further lowering the ionized serum calcium levels - and trauma plus major surgery is a tremendous stress - so intermittent calcium replacement and large intravenous doses of solumedrol are probably both justified during large-volume blood replacement (of course, if calcium chloride is delivered through a blood administration set, it can clot any bank blood still in the tubing).

A diminishing urine output after major trauma and blood loss is often the last stop before acute renal failure, so with any decrease in urine production (regardless of estimated blood volume), mannitol is usually indicated to decrease the likelihood of renal shutdown secondary to tissue and blood cell destruction, protein denaturation, tubular blockage, renal interstitial edema or circulating nephrotoxins (that could result from trauma, preexisting disease, pressor drugs, blood warmers, blood pumps or perhaps even adverse interactions between infused units of blood).

Hemolytic transfusion reactions are alleged to be another rare cause for uncontrollable intraoperative oozing and hypotension following blood transfusion. Supposedly this bleeding results from disseminated intravascular coagulation caused by release of thromboplastic material from disrupted red cells which depletes the circulating blood of platelets and coagulant factors. Allegedly less than 50 cc of mismatched blood can cause such a massive reaction.

Obviously, if totally disseminated "true" intravascular coagulation really occurred, the patient would die almost instantly of blocked vessels: While life continues, therefore, a balance must exist between clotting and clot lysis, so that any relative increase in clotting can consume vital coagulation factors without actually closing off vital parts of the circulation (which may be somewhat protected by

a comparable increase in clot lysis factors).

Modern blood-banking safeguards make a severe hemolytic transfusion reaction very unlikely. We have, however, encountered a rare, delayed hemolytic reaction, of the sort seen 3-7 days after transfusion. Allegedly this occurs when prior sensitization causes recently transfused blood to become incompatible some days after it was given.

Recommended treatment for any acute hemolytic transfusion reaction includes fresh frozen plasma and platelets for the coagulant deficits, sodium bicarbonate to avoid an acid urine in which red cell stromal precipitation is more likely, volume loading with Ringers Lactate, vigorous diuresis, and steroids in large doses (to treat the acute pulmonary hypertension and peripheral hypotension).

I have no experience with this problem, although many years ago I did have one postoperative patient who received blood intended for someone else, but by pure chance, that "wrong" blood was compatible with the patient who received it (even though I did not order it, he did not need it, and the hospital may even not have charged for it).

The more common febrile and allergic blood transfusion reactions are also due to prior sensitization, but only to donor white cells, or occasionally platelets or blood proteins; or very rarely, bacterial contamination: Administration of blood as "packed cells" can decrease such (usually minor) non-hemolytic allergic reactions; any fever, rash, chills or pruritus normally responds to prompt discontinu-ation of transfusion plus antihistamine treatment.

A rare, more severe allergic, or even anaphylactic, response to transfusion may require epinephrine and steroid therapy. Although repeat analysis of the patient's blood and donor blood-bag content has never been helpful in my experience, it is a good precaution against missing a serious bacteremia. The incidence of allergic reactions to transfused blood allegedly remains elevated after a first sensitization event.

Disseminated intravascular coagulation can result from many injuries or illnesses: For example, if blood is simply exposed to temperatures over 40°C, it may be hemolysed and plasma proteins denatured, blood cells clumped and toxic plasma proteases activated; simple tissue injury, blood transfusion "mismatch," ischemia (or low blood pressure), autotransfusion of salvaged blood after exposure to pleura, peritoneum or an air fluid interface; all of these occurrences have been alleged to release tissue thromboplastin, activate the clotting cascade and lead to "diffuse microvascular clotting" (whatever that is and wherever that may occur. I would hazard a guess that the pulmonary vessels probably "get it" first.)

Treatment of disseminated intravascular coagulation may range from complete anticoagulation with heparin to attempted restoration of losses in blood volume and necessary components of the coagulation cascade. Obviously a restoration of depleted blood coagulation factors will be required if bleeding is the major manifestation of this disorder, while heparinization would be preferred if the patient is endangered by thrombosis (these patients show a drop in both platelets and fibrinogen, along with elevated levels of fibrin "split products").

Epsilon amino caproic acid may apparently be infused to neutralize circulating fibrinolysins (including streptokinase), or even applied locally to a bleeding surface wound or needle puncture site in such a patient. When I think that a patient maybe developing "DIC," I request urgent consultation, if

only to share my confusion.

Case Report: Postoperative Disseminated Intravascular Coagulation

A 37 year old native Alaskan female, mother of 11, underwent a straightforward pericardiectomy for severe chronic (tuberculous) constrictive pericarditis. Being just out of residency training, I attributed the remarkable lack of bleeding from cut surfaces at surgery to my recently perfected surgical skills. Postoperatively the patient did well for some hours before developing peripheral venous distension, agitation, shortness of breath, and increasing rales and rhonchi.

Assuming that her obvious hypervolemia was due to release of the overfilled, chronically obstructed venous blood pool into the circulation, I attempted phlebotomy, but the needle clotted several times as I drew blood. In desperation, as she was progressively becoming terminal and I had fixated on the need to reduce her blood volume, I incised her external jugular vein under local anesthesia and tried to bleed her into a sterile basin. The blood clotted in mid air and fell to the basin in long blue strands. She died shortly after I gave up this futile effort.

It was not easy to explain this outcome to her husband and eleven children of all ages, especially so soon after reporting my "successful" surgery. Autopsy revealed widespread thrombosis of arteries and veins, as well as mural thrombi within all heart chambers.

Conclusion: She certainly should have been heparinized. Phlebotomy did prove lifesaving for the next postpericardiectomy patient who developed early postoperative pulmonary edema: Since then, effective diuretics and volume-controlled respirator care have essentially outmoded "blood-letting" for postoperative hypervolemia (but phlebotomy remains an effective remedy for the unusual case or circumstance).

Intra-Arterial Transfusions

The unstable, badly injured trauma victim, or patient with ruptured abdominal aneurysm, is often the center of a reddish whirlwind of activity, devoted mostly to removal of large quantities of warm "recently perfect" blood from the operative field and return of as much "barely adequate" blood as possible (along with a host of side-effects of these efforts). Although far greater volumes of blood are routinely removed and infused intra-arterially during cardiopulmonary bypass, that scene is usually so placid that the uninitiated observer might wonder if the relaxed perfusionist and drowsing anesthesiologist should not be replaced by an inexpensive computer.

Clearly, appropriate planning and routine preparation can allow total extracorporeal support of circulation and ventilation during removal and autotransfusion of large volumes of temperature-controlled well-oxygenated blood at necessary flow rates and pressures. Why is this not possible with the (unscheduled) injured patient?

At least four reasons are commonly cited:

1) Heparin may cause fatal bleeding into brain or spinal cord if given soon after injury to these structures, and heparin is still required to achieve total cardiopulmonary bypass (although reportedly, the Bio-medicus pump can be used to maintain heparinless bypass of the descending thoracic aorta during repair of aortic disruptions if the flow is kept above one liter per minute and the patient is

decannulated promptly thereafter);

2) Significant tissue injury or an interval of low blood pressure with poor perfusion can both lead to consumption of coagulation factors, making it difficult to restore normal coagulation after extracorporeal bypass is discontinued;

3) One hesitates to recirculate "possibly contaminated" blood (e.g. with opened intestine, or while resecting a malignancy);

4) Cardiopulmonary bypass is a "subspecialty technique" (developed by, and thus only available to, cardiac surgeons).

Impending exsanguination can occasionally override these theoretical constraints at present: It seems likely that further advances in oxygenator technology will soon allow heparinless bypass for circulatory support of severely injured patients during all types of surgery. It could eventually even become standard policy to place all injured patients who are "bleeding out" from any cause on cardiopulmonary bypass. Certainly, equipment cost should not be a factor, as a few transfusions avoided can result in savings equal to the cost of the "bypass disposables" that are required.

It may be that compartmentalization of surgical specialties has prevented appropriate dissemination of cardiopulmonary bypass technology and techniques into general surgery: Although such technology could soon become available to non-cardiac surgeons who have appropriate background, training and skills, the occasional application of cardiopulmonary bypass in general surgery might best be "intermediated" for now by anesthesiologists who have become especially adept at cardiac surgery and "intensive care."

Anticoagulants

The few anticoagulants available vary in their effectiveness (even between different individuals), in their safety and controllability, in their applicability to arterial and venous clotting processes, in their mode of action, in the mechanism of and possibility for early neutralization or reversal, and in their adverse or cumulative interaction with many other drugs and even foods. Anticoagulants are so widely distributed in nature that one would anticipate the discovery and clinical utilization of more specific and useful drugs in the future.

More than two decades ago, hirudin (an extract of mashed leeches) lost its market share to slaughterhouse heparin; might hirudin now deserve another look? Similarly, during certain months, tundra mosquitoes outweigh all other Alaskan animal life combined: These mosquitoes are exceptionally easy to trap (e.g. within a tent) and their stingers never "clot off": Indeed, they may well be the next great untapped source of anticoagulant (as well as animal protein and other useful byproducts).

Coagulation Disorders

Coagulation disorders occur with far greater frequency after cardiopulmonary bypass than after thoracotomy alone: As cardiac surgery has matured, we have gained increasing experience with such states. Those coagulation problems following trauma that are not simply consequent to dilution, hypothermia or consumption coagulopathy (the latter is further evidence of poor prognosis in a badly

injured patient), have similarities to some of the post-perfusion problems presented below.

After Bypass

Common causes of "coagulopathy-type" bleeding after cardiopulmonary bypass have included "blood bashing," heparin, hypotension and tissue hypoxia, thermal injury to blood or patient, chemical injury to blood or patient, and "blood dyscrasia" (whatever that is).

Blood bashing is becoming less of a problem. With every oxygenator improvement, we encounter fewer episodes of significant postoperative bleeding. Noticeable darkening of urine due to hemolysis (a gross indicator of overall blood trauma) is now infrequent, regardless of the duration of perfusion. Several of the 20-40 micron arterial-line blood filters that are available also seem well tolerated by the circulating blood. A principal remaining cause of mechanical blood injury is the "pump sucker," which should always be used at its lowest practical setting (and avoid "sucking up" clots or pericardial fluid, for both can allegedly initiate clotting in the cardiopulmonary bypass system).

At the time when "standardized" heparin dosages were still in use for cardiopulmonary bypass, postoperative bleeding problems were not uncommon (with reoperation for bleeding required in perhaps five percent of all patients). There was a definite decrease of this complication when regular intraoperative reappraisal of "activated clotting times" was instituted; these tests suggested that we had often given insufficient heparin (which led to increased activation and consumption of clotting factors within the oxygenator and filters). Another apparent decrease in postoperative bleeding problems was seen in our own practice, when we switched from a bubble oxygenator to the Cobe membrane (Cobe Laboratories, Denver, Colorado).

Inadequate neutralization of heparin by protamine can result in "moderate" early postoperative blood loss (protamine is strongly basic and binds ionically with strongly acidic heparin). In the occasional case where such bleeding persists until "residual free heparin" is reported by the laboratory, additional protamine can terminate the oozing. Reportedly, protamine is eliminated from the circulation more rapidly than heparin, which can apparently lead to what is known as "heparin rebound," an hour or two after protamine administration: That also responds to administration of additional protamine, if such seems indicated (but I have not recognized this as a clinical problem).

An occasional patient will suffer a marked drop or even "loss" of blood pressure during or just after protamine administration, which "response" (as well as true protamine anaphylaxis) is allegedly more common in patients allergic to fish. Apparently, a return to full heparinization and one or two grams of intravenous calcium chloride will usually counteract this "protamine crash," with a rapid return to cardiopulmonary bypass support often needed in the interim.

It is probably best to let this "rescue" heparin wear off spontaneously, rather than risk a second adverse protamine response. We now administer protamine over a period of five minutes or more, and add a gram of calcium chloride to the protamine solution; these modifications appear to have nearly eliminated hypotension secondary to protamine administration (excessive doses of protamine allegedly may have an adverse effect on plasma fibrinogen and platelet function as well).

As mentioned above, the modern arterial-line blood filter is not destructive to properly anticoagulated blood. Arterial line filtration at 20 to 40 microns remains crucial for reduction of those intraoperative showers of microemboli that were so often associated with diffuse cerebral damage and

postoperative psychoses. I strongly believe that cardiopulmonary bypass should not be performed without appropriate arterial-line filtration.

Many patients who survived heart surgery "without any complications" in the pre-arterial-line-filter days "never really got over it" or "Just ain't what they used to be." In fact, many of our finest "cures" and dramatic "saves" were returned to their relatives and jobs as non-caring incompetents or even "zombies."

The prompt disappearance of almost all "postoperative cerebral problems" upon institution of adequate arterial filtration has impressed experienced surgeons from numerous surgical centers. (The literature used to have frequent articles on how windows, calendars, clocks or psychiatrists were necessary to keep our patients from becoming psychotic after heart surgery - that has all subsided now, except from a few centers that have not yet "caught on" that filters are important.)

Other Causes of Coagulopathy

Prolonged hypotension, tissue hypoxia, a long pump run, numerous transfusions (perhaps ten or more), heparinization for bypass during thoracic aneurysm repair and over-rapid rewarming, all can render blood less coagulable. The increasingly efficient "integral heat exchangers" for rewarming the patient's blood require especially close monitoring to avoid overheating of blood (and consequent protein denaturation, with great likelihood of severe postoperative bleeding and clotting problems). A rewarming water temperature of 40°C (104°F) should never be exceeded.

Incidentally, air bubbles are more likely to form within the patient's arterial circulation during cooling of the patient, and within the oxygenator during rewarming. Cold blood binds more oxygen (both to hemoglobin and in solution), so fully-oxygenated cold blood, suddenly rewarmed (on entry into a warm brain, for instance) is theoretically more likely to allow oxygen bubbles to come out of solution. It seems clear, therefore, that neither cooling nor rewarming should be rushed, and that excessive oxygenation of the circulating blood adds unnecessary risk to routine cardiopulmonary bypass.

As for blood dyscrasia or chemical insults leading to coagulopathy; preoperative aspirin with von Willebrand's Disease can cause severe and continuing postoperative blood loss. One should probably also avoid adding aminoglycoside antibiotics directly to the bypass system (we only add Prostaphlin or Keflin) to prevent undesirable neutralization of heparin during the pump run (anecdotal evidence indicates that entire extracorporeal circuits have clotted following addition of these antibiotics).

We generally take a "shotgun approach" to post-bypass bleeding and clotting abnormalities, giving fresh frozen plasma for prolonged prothrombin or partial thromboplastin times. On rare or desperate occasions, we have supplemented the fresh frozen plasma with an apparently very effective, commercial, lyophilized, blood plasma product (Conyne). Platelets are given for bleeding when the platelet count is low or if the bleeding patient has been on aspirin. (It is alleged that no functioning platelets remain in whole blood that has been stored for over 72 hours.)

Each platelet pack should raise the patient's platelet count by at least 5000 platelets/mm3, (some report an increase of 10,000 or more platelets per unit given: This may be difficult to determine in a patient who is bleeding actively) so we use 6-12 platelet units at a time, when these seem indicated (in order to bring the platelet count back toward 100,000 per cubic mm when it is necessary to correct

generalized small vessel ooze or bleeding). We do not give platelets prophylactically for a low platelet count in the absence of bleeding, except when significant blood loss can be anticipated (following replacement of an acutely dissected aorta, for example).

If one simply cannot stop generalized oozing, and sternotomy closure causes (or obviously would cause) tamponade, or if the heart will not tolerate chest closure (due to cardiac enlargement, lung stiffening from pulmonary edema, or other cause), we have on rare occasion returned the patient to the intensive care unit with chest retractor in place and gauze surgical "lap" packs stuffed about bleeding areas as indicated (see also discussion earlier this chapter). Obviously chest tubes are not needed to drain such an open chest, and povidone-iodine-solution-soaked towels are laid across the whole area to prevent bacterial contamination. A silastic rubber sheet can be laid over the heart and lungs to shield them from abrasion by the chest retractor blades and sternal edges until final sternal closure can be achieved.

On rare occasion, a thin silastic sheet (laid loosely over the heart and great vessels beneath the sternal approximation) can also expedite sternotomy closure. I used such a sheet once to "shoe-horn" a distended heart beneath the sternum when the heart otherwise could not tolerate such closure. The patient died later so I cannot discuss long term results, but I have since read that silastic-rubber-coated fabrics may cause a dense fibrous reaction with increased risk of cardiac compromise and infection. So if used in this fashion, the fabric should probably be removed when the patient has stabilized. Such a silastic sheet also protected a huge arch aneurysm from abrasion by sternum and wires (when consulted about closing a child who had been explored for presumed malignancy) pending transfer to Crawford in Houston for successful repair (see also Chapter 10).

Case Report: Coagulopathy After Repair of a Ruptured Thoracic Aneurysm

A 70 year-old male was transferred from a small rural hospital complaining of severe chest pain of two days duration. Chest roentgenograms demonstrated a huge left chest mass displacing and occluding his left main bronchus. Angiogram revealed a free rupture of his descending thoracic aorta aneurysm into the mediastinum. Exploration and graft was carried out (using femoral vein to femoral artery, partial cardiopulmonary bypass). A long rent in the aneurysm wall was found to be tamponaded by esophagus. Although intact, the esophagus seemed thin walled and bluish (which, under the circumstances, did not surprise us).

Bleeding after repair was excessive, and multiple lap pads were packed into the chest during closure to improve hemostasis. Reexploration became necessary (for recurrent blockage of the left main bronchus by lap pads and clot) about twelve hours later. Esophagus was again noted blue but intact. Lap pads were removed as hemostasis was now satisfactory.

For three days, all seemed well (in fact, a conference on thoracic aneurysms was even scheduled); then the patient developed a small trickle of blood from the corner of his mouth (a well known "bad sign"). Esophagoscopy revealed gangrenous esophagus, which was removed bluntly through a transabdominal and cervical approach. This third major operation was followed by copious irrigations of 1:4 dilution, stock Betadine solution into the chest and grafted aorta area from both above and below. The patient soon became stuporous and died a week later: At first we thought he died of sepsis and "hepatorenal failure," but bacterial cultures were all negative and his serum iodine level was enormously elevated (presumably due to excessive absorption of Betadine through the widely exposed pleural surfaces).

Conclusion: I made several errors in the management of this patient. In the first place, perfusion pressure within an esophageal wall while it tamponades an aortic tear must be zero; I should, therefore, have anticipated esophageal necrosis, knowing that the esophagus had been exposed to aortic pressure for two days prior to this admission. A thin, blue esophagus should at least lead to early and frequent evaluation by esophagoscopy (or possibly even prophylactic esophagectomy). Secondly, it probably would have been much better to reopen the chest for the third time, in order to achieve optimal debridement and irrigation around the graft.

Finally, Betadine irrigation of a large, normally absorptive, pleural surface should be avoided. An appropriate antibiotic or hypochlorite irrigant solution (such as Dakin's solution) would likely have been as effective for local sterilization of peri-esophageal surfaces, including the aortic graft (and also far less toxic). In retrospect (and having since seen other reports of similar deaths), I now suspect that this patient actually died of a severe metabolic acidosis brought on by iodine poisoning.

Case Report: Intentional Iatrogenic Consumption Coagulopathy

A tall, slender, 32 year old male entered the emergency room with an apparent acute aortic dissection and hypotension: Sonography confirmed ascending aortic arch dissection and pericardial tamponade. Composite graft replacement of aortic valve and ascending aorta was carried out urgently, with reimplantation of the coronaries.

Despite snug closure of the aneurysm wall over the graft, aortic blood loss remained massive through many suture holes, so the aneurysm wall was anastomosed to the right atrial appendage. External bleeding from the aorta was considerably diminished by this decompressive left-to-right shunt, but unfortunately it still remained excessive, so the atrial appendage was then disconnected and protamine given.

A ten cc vial of topical thrombin (labeled "not for injection") was then injected at several sites into the blood-filled space between the aortic graft and the aneurysm wall: That space promptly clotted, terminating the uncontrollable blood loss. Thereafter, the patient's blood became incoagulable, due to consumption of "all available clotting factors" (the platelet count fell to about 1000 platelets per cubic mm. In an earlier, even more desperate case, the topical thrombin injection was equally effective, and platelets fell to 7000 per cubic mm. I am aware of others who have resorted to this "when all else fails, inject thrombin" ploy, also with successful outcome).

In any event, being now faced with incoagulable blood, this patient's mediastinum was firmly packed with lap pads to control the persistent ooze from "everywhere else," and the patient was taken to the intensive care unit with his sternum widely open and the chest retractor still in place, bleeding moderately under the povidone-iodine soaked towels that covered his anterior chest. After much fresh frozen plasma and blood, platelets were given as soon as they became available, and the ongoing blood loss soon subsided. The patient was returned to surgery on the first postoperative day for elective removal of retractor and packs: Routine sternotomy closure followed placement of chest tubes. He was discharged after a ten day course of prophylactic antibiotics, and has done well over the ensuing three years.

I have since learned of a simple modification by Helseth's group of the original Bentall and DeBono composite-graft operative technique, which can greatly improve hemostasis during such procedures: It consists of a second, circumferential, running, over-and-over, 2-0 prolene suture line,

which brings graft and aorta together in water-tight fashion just above the site of valve insertion (proximal to where the new coronary orifices will be placed). Conveniently, the premanufactured composite graft (containing a Bjork-Shiley valve) flares outward just above the valve insertion, in order to join onto the larger, tubular prosthesis: The junction "shoulder" thus formed becomes a perfect, supravalvular, "second annulus" (which can prevent any minor perivalvular leakage from becoming a troublesome stream or even raging red torrent).

Reoperation for Post-Bypass Bleeding

A #32 silastic substernal chest tube will often evacuate all of the blood being pumped out from an arterial bleeder on the inner sternal periosteum (especially with high PEEP and a frequently milked chest tube). Such "freshly shed" blood tends to clot within and obstruct the drainage tubing. However, if the tubing can be kept open by meticulous attention, a sticky layer of fibrin will accumulate on the inner surface of the clear plastic chest tubing: This thin pink or grey deposit signifies "fresh blood loss."

When blood loss is fully drained in this immediate fashion, there will be no mediastinal widening on chest roentgenogram, and only minimal physiologic derangement as long as all external blood loss is currently replaced. This situation usually allows adequate time for correction of any abnormalities of blood clotting, so the decision to reoperate will be based upon how much blood the surgeon and blood bank intend to replace (as well as whether the ongoing blood loss seems steady or slowing).

If surgery is "required," it can be scheduled whenever the operating room, anesthesiologist and O.R. nurses become available. In other words, unless rapidly "bleeding out," this patient may be transported to the operating room on a semi-urgent basis without undue risk, once a decision to reexplore has been made.

With worrisome bleeding soon after sternotomy closure, however, and an increasingly precarious patient who shows progressive mediastinal widening on consecutive roentgenograms, a surgeon should "stand by": The patient with developing cardiac tamponade must remain heavily sedated on a ventilator (morphine, 5 or 10 mg is given intravenously whenever needed, or even "just in case") and also paralyzed (by Pavulon 10 cc, or other muscle relaxant, given intravenously), to prevent the sudden (possibly final) decline in output from an already compressed heart that can be precipitated by coughing against an endotracheal tube (or even just straining to help while being transferred to the operating table).

If such a patient is deteriorating despite Dopamine, with pulse unstable and systolic pressure often below 100 mm Hg, it is probably safer to reopen the sternotomy immediately in the intensive care unit than to risk transport while "in extremis." (Sterile wire scissors are kept taped to the bed of every post-sternotomy patient for use in this eventuality).

On Reopening a Sternotomy in Bed to Relieve Acute Tamponade

Use sterile gloves, lots of Betadine solution, and place sterile, Betadine-moistened towels around the incision. Sterile gown and mask are not necessary, but care is taken to avoid significant contamination of the sternotomy wound. There is no risk of iodine toxicity from povidone-iodine absorption here, for current blood loss and later copious intraoperative saline irrigations will soon wash any Betadine overflow out of the chest.

Access to a sterile chest retractor is desirable: As the sternum is cranked open, one must separate the sutured-together edges of pericardium carefully (by feeling under clot, if necessary) in order to relieve any remaining intrapericardial tamponade. Great care must be taken to avoid injury to internal mammary artery and saphenous vein bypass grafts, both during such an initial emergency pericardial opening and later in the course of clot removal.

On rare occasion, a briskly bleeding saphenous vein graft side-branch may require prompt control by hemostat or hemaclip, but correction of other than the immediately-obvious major vessel bleeding is usually best deferred until good lighting, sterile conditions, proper instruments, adequate suction, lots of sterile warm saline and competent surgical personnel become available in the operating room: Place a Betadine-soaked sterile towel over the open wound during transfer to surgery and leave the chest retractor "cranked open" (unless this seems inappropriate or impractical).

Once sternum and pericardium have been opened to decompress the heart, the patient is generally in better condition for transport, despite a chest still overflowing with blood and clot: Blood transfusions are meanwhile continued as indicated by the patient's appearance and blood pressure. It is well to remember that the blood now welling up out of the newly opened chest was probably lost and replaced some time ago, and that the patient just now being relieved of cardiac tamponade may well have an overfilled venous reservoir, and thus not need more blood unless the bleeding persists.

Infection has not occurred in any of the eight or ten patients that we had to reopen in bed with such haste (perhaps due to luck, lots of Betadine on the skin, prophylactic antibiotics such as Prostaphlin or Keflin, complete removal of clot during meticulous intraoperative hemostasis, and copious warm-saline irrigation of the farthest reaches of pericardium and any opened pleural cavities.

One tends to regard fluid that drains through any chest tube as a reasonable sample of "what is going on" inside. Thus, fresh bloody drainage is considered a worrisome sign of continued active blood loss, while gradual or intermittent cessation of such drainage suggests that the acute blood loss is almost over; later drainage of old liquified clot is then viewed as a sign that all may soon be well. Unfortunately, it is not always quite that simple.

While ongoing but intermittent drainage of fresh blood (and clotting within the tube) could signify intermittent bleeding, more likely it represents continued active postoperative blood loss in the face of suboptimal chest tube location and function. For example, moderate bleeding from a saphenous vein graft may soon build a large mass of clot and fresh blood, deep down within the pericardium (and thus relatively far from the substernal mediastinal tube). Within hours, some of that "older" clot may begin to dissolve or liquify, regardless of whether active bleeding has stopped. Under these circumstances, a mediastinal tube might drain fresh blood intermittently, or "old" liquified blood "off and on" (between episodes of fresh bloody drainage), or the tube may simply cease to function if it becomes blocked or buried in clot.

Summary: Only with an ideal tube location, superb chest tube care, and minor to moderate postoperative blood loss, will the chest tube output provide a reliable, real-time sample of intrapleural blood loss.

How To Evaluate Drainage of Defibrinated "Old Blood"

At times, one may encounter gradual widening of the mediastinum on routine postoperative chest

roentgenograms, despite minimal chest tube drainage during the early postoperative hours: Then a worrisome increase in drainage of non-clotting (bright or dark red) blood is reported, often in "gushes" that seem associated with changes of position (or PEEP setting).

This entirely-liquid blood leaves no fibrin precipitate; in fact, it rapidly dissolves any such coating that remains on the (inner) chest tubing surface, as it flows easily on down to the water seal bottle. Despite these worrisome gushes, the patient in question (who may have received two or more units of blood and varying amounts of Ringer's Lactate in the first 6 or 8 hours postoperatively) now seems quite stable in pulse and blood pressure, with a slowly rising hematocrit as well.

If the chest roentgenogram is repeated at this point, it will show decreased mediastinal widening (provided the patient is still intubated and on the same amount of PEEP). With or without such chest film, however, this entirely liquid blood clearly is not "freshly shed"; rather, it is only just now appearing as a consequence of dissolution of clot that formed some hours ago during the actual blood loss. Obviously then, this liquid drainage of old blood, even when copious or quite red, is no reason to transfuse an otherwise stable patient.

Conclusions: (A) "Freshly shed" blood will "coat and clot" inside of the chest tubing; in fact, it must constantly be milked along toward the water seal bottle if tube function is to be preserved. Even with excellent chest tube management, the actual blood loss often greatly exceeds the current volume of chest drainage. Therefore, blood volume replacement must always be based upon a current evaluation of the patient, and not merely ordered on the basis of recent chest tube drainage, or the extent of mediastinal widening seen on roentgenogram.

It is well to remember that apparent but not real widening of the mediastinum can also result from lower lobe atelectasis, slight rotation of the patient, and higher diaphragm position (consequent to an "expiratory" film or discontinuation of ventilatory support): All of these changes may closely mimic the roentgenographic shadow cast by mediastinal accumulations of fresh blood or clot.

(B) On the other hand, "old" (or "lysed" or "defibrinated") blood flows easily out of the chest tube, does not coat the tubing with fibrin (it even dissolves any fibrin already deposited there), requires only occasional milking of the tubing (to push along a dark and shrunken clot), is not associated with a drop in blood pressure or hematocrit and (although it closely resembles "real circulating blood") is not recently from within the circulation: Blood volume replacement (to make up for this "old" but freshly externalized "blood loss") is therefore not indicated. In fact, evacuation of this "old blood" by any means can only improve heart and lung function, so "let it flow."

Autotransfusion of Major Hemothorax

Significant blood loss into the chest is common; adequate facilities for control of such hemorrhage are not always instantly available. A patient's own warm and unclotted blood will always be the ideal fluid replacement during major blood loss, pending cessation (or surgical control) of bleeding. Liquid blood accumulating within the chest has been reinfused into thousands of patients.

More than one autotransfusion system is currently marketed, and many others have been devised and utilized successfully. Autotransfusion is limited only by the clinical state of the patient and the

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condition and volume of blood being drained (it must be liquid and preferably not grossly contaminated by bacteria, other foreign matter or malignant cells).

A simple, emergency autotransfusion setup might well be kept available for immediate use in any rural clinic or general hospital. Using ordinary, inexpensive equipment, several of our patients have received rapid, successful autotransfusion of major hemothorax with excellent hemodynamic stabilization. The basic idea (not at all original) is to disconnect the chest drainage bottle from the chest tube when this bottle fills with blood, and to then hang this bottle for immediate intravenous reinfusion, while another empty chest bottle is connected to the chest tube for continued blood collection.

Materials for Autotransfusion of Hemothorax

1) Standard materials for inserting a chest tube.

2) A chest tube (No. 28 or No. 32 French for an adult).

3) Two of the Bentley (or any other) simple water seal pleural drainage bottles (Bentley Labs, Irvine, CA.) Please Note: I have a conflict of interest here, as I worked with Bentley Labs and Tector on the design and marketing of this unit (which has a lot of useful features that have been described elsewhere in this text).

4) Sterile saline for the chest bottle "water seal" (200 cc to prime each Bentley bottle); this prime will not be necessary in the presence of copious bleeding into the chest, or during positive-pressure controlled breathing (for free intrapleural blood then flows out easily to create its own "water seal").

5) Anticoagulant for the chest bottle: Heparin in a multidose vial (1000 U/cc), or Citrate, Phosphate, Dextrose (C-P-D) solution, as preferred.

6) A one cc syringe and needle for adding Heparin to the chest bottle.

7) A No. 20 French chest tube, with the flared end appropriately trimmed to fit tightly into the chest bottle air vent and the other end cut off to allow insertion of a blood filter (or blood pump).

8) A multiple transfusion blood filter (optional).

9) A hand-squeezed blood pump set (McGaw Labs).

Technique

The chest tube is inserted and secured, taking care not to spill already accumulated blood during tube placement. The chest tubing of a water seal chest drainage bottle is then connected to the chest tube. Heparin (or other anticoagulant) is added to the chest bottle, which is swirled occasionally during collection to disperse anticoagulant and prevent clotting.

When a chest bottle is nearly full of blood (over 2 liters), it is replaced by an empty bottle, and the full bottle is hung for intravenous return of its content through a large (#14 or #16) intravenous cannula or needle (Fig. 9). Blood is easily pumped into the patient's vein (via the trimmed #20 Argyle chest tube inserted into the chest bottle air vent and sequentially connected to the blood filter and hand pump).

One should loop the open, disconnected chest tubing (without kinking it) over the top of an intravenous pole to avoid a mess when the chest bottle is first inverted. (This chest tubing must remain unobstructed so that air may continue to enter the chest bottle as its contained fluid is pumped out into the patient.)

One can return 2 liters of blood in ten to twenty minutes by this method. As with any rigid intravenous bottle, care must be taken to avoid pumping air into the patient from a nearly empty bottle. When it has been emptied down to its "prime" (the air vent enters the Bentley chest bottle far enough to prevent loss of the last 200 cc), the hanging chest drainage bottle can be taken down and reused as a chest bottle while the second bottle is hung for autotransfusion. Although this setup is simple, it is best to practice with used equipment and water, and possibly even to label and leave a "sample setup" hanging (in order to minimize confusion at a time of great turmoil).
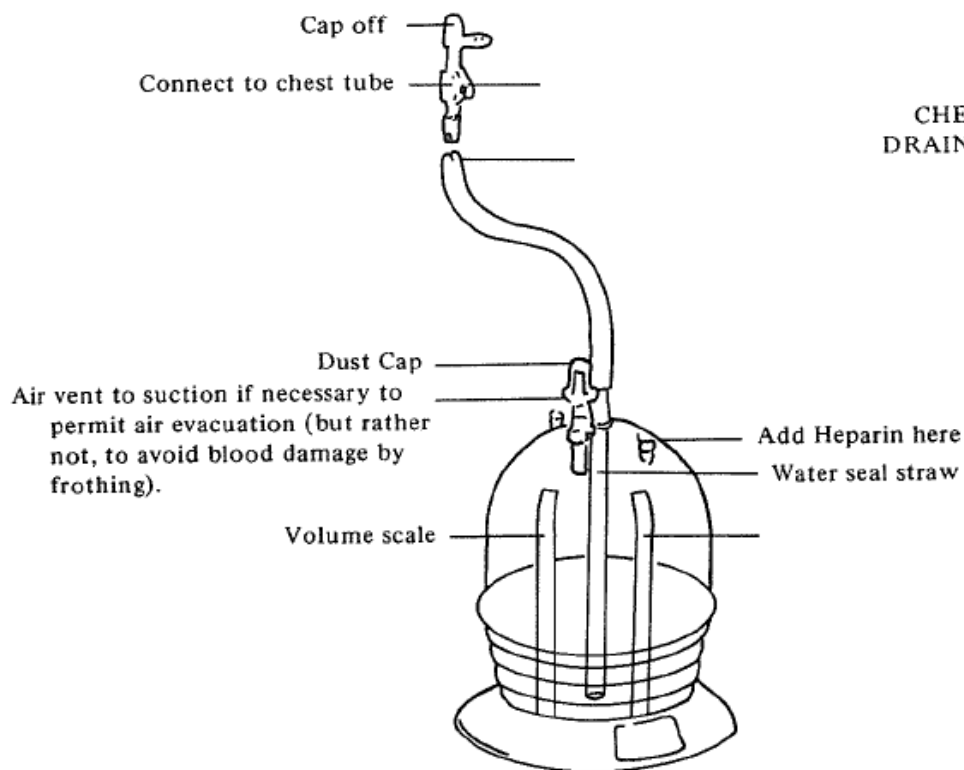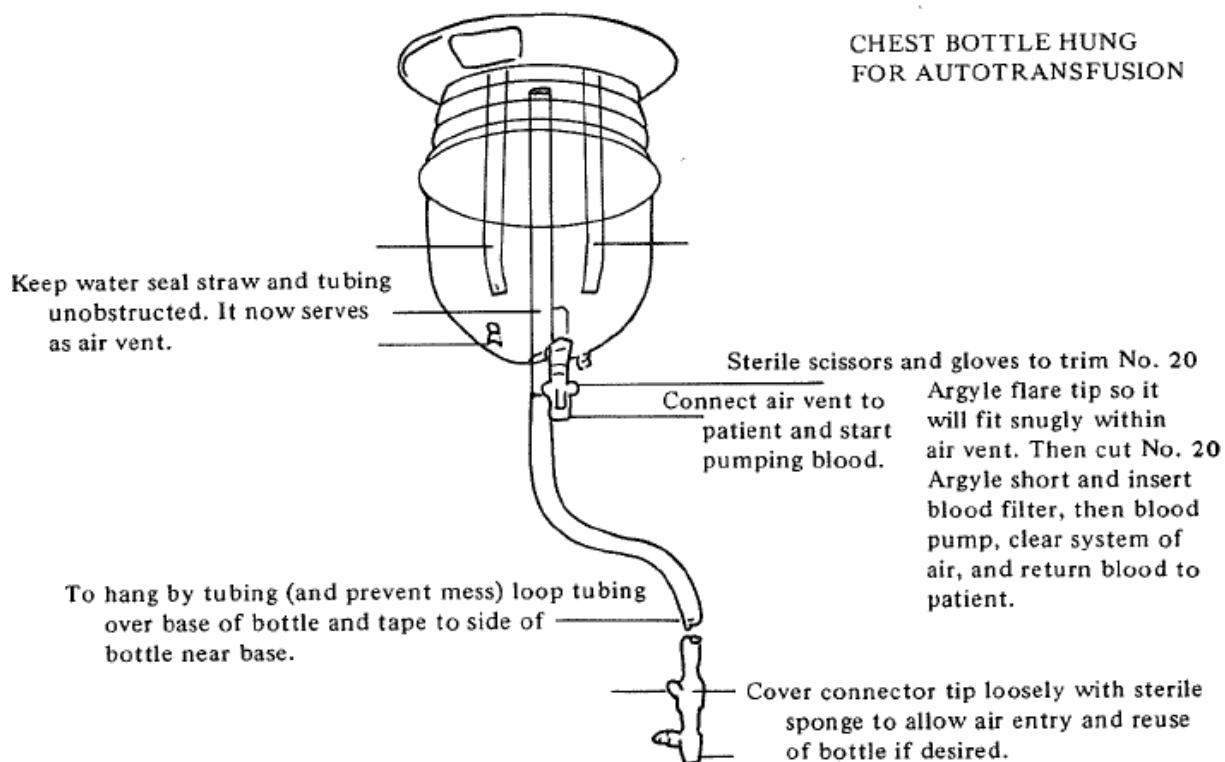
CHEST BOTTLE HUNG FOR AUTOTRANSFUSION

Keep water seal straw and tubing unobstructed. It now serves as air vent.

Connect air vent to patient and start pumping blood.

Sterile scissors and gloves to trim No. 20 Argyle flare tip so it will fit snugly within air vent. Then cut No. 20 Argyle short and insert blood filter, then blood pump, clear system of air, and return blood to patient.

To hang by tubing (and prevent mess) loop tubing over base of bottle and tape to side of bottle near base.

Cover connector tip loosely with sterile sponge to allow air entry and reuse of bottle if desired.

Cap off

Connect to chest tube

CHEST BOTTLE DRAINING CHEST

Dust Cap

Air vent to suction if necessary to permit air evacuation (but rather not, to avoid blood damage by frothing).

Add Heparin here

Water seal straw

Volume scale

Figure 9.

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Case Reports:

With this method, we have autotransfused several patients (with good hemodynamic results in all), using about 1500 U of Heparin per bottle.

PATIENT 1 had massive chest and brain injuries and was autotransfused with over 6 liters of blood in about an hour, using three or four chest bottles. Such blood replacement stabilized his circulation and allowed evaluation and emergency room thoracotomy for ongoing severe intrathoracic blood loss. The source of blood was a torn pulmonary artery. (Later death was due to preexisting brain damage.)

PATIENT 2 with multiple rib fractures, had autotransfusion of a 2 liter hemothorax and excellent stabilization of his circulation. No further transfusions or chest surgery were required (complete recovery after exploratory laparotomy).

PATIENT 3 suffered a lacerated left anterior descending coronary artery at pericardiocentesis (during resuscitation in the cardiac catheterization lab). Successful vessel repair was achieved on the beating heart in the operating room (with cardiopulmonary bypass unavailable). This patient received a five liter autotransfusion during his one-hour stabilization (as well as other transfusions) and showed complete recovery, without electrocardiographic or demonstrable angiographic abnormality. He later underwent aortic valve replacement elsewhere (before we initiated our heart surgery program).

Discussion: If blood for autotransfusion is liquid and not grossly contaminated, it allows an immediate return of warm, properly cross-matched blood to the critically injured patient. In these three episodes, it prevented any delay for "cross-match," as well as hypothermia from cold blood and other problems often associated with significant infusions of bank blood: It also reduced blood transfusion costs significantly. Although no debris was seen on the blood filters used, it may be wise to keep a good transfusion filter in the intravenous infusion line.

Patient 2 was injured about twelve hours prior to transportation to our facility, and his intrapleural blood was free within the thorax for a number of hours. However, it was liquid and drained rapidly on chest tube insertion. When it remained warm, red and liquid for several minutes, it was reinfused without anticoagulant, while bank blood was still being cross-matched.

I do not know if we infused unphysiologic concentrations of activated clotting and clot lysis factors into his blood stream or not, but his own defibrinated blood certainly worked well in this case (in our view, we simply accelerated the normal resorption of his hemothorax). During any delayed autotransfusion of this sort, one must watch for volume overload, even when only returning the patient's own blood (see Chapter 14).

Patient 3 was interesting in that we transfused 2 liters of blood for every 1.5 liters brought out by chest tube drainage, due to ongoing intrathoracic clot accumulation during the one hour delay before an operating room became available.

A modification of the above-described (PDS-100) Bentley water seal chest drainage bottle by Tector (the PDS-200), permits ready reinfusion of ordinary quantities of postoperative chest drainage: This is easily drawn out of the standing chest bottle via an accessory straw (normally kept capped), directly into a standard glass "blood transfusion" vacuum bottle. Hopefully, such an approach can further reduce the need for postoperative blood transfusions. The reinfusion of defibrinated or liquified

blood has apparently been tolerated without complication by many of Tector's postoperative heart surgery patients, including myself.

Unlike Thucydides, who wrote for eternity, this piece of writing is designed to meet the needs of an immediate public, and I will feel amply rewarded if it achieves that purpose over the next several years. AvH