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Pulmonary Contusion: An Update on Recent Advances in Clinical Management

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Abstract Pulmonary contusion is a common finding after blunt chest trauma. The physiologic consequences of alveolar hemorrhage and pulmonary parenchymal destruction typically manifest themselves within hours of injury and usually resolve within approximately 7 days. Clinical symptoms, including respiratory distress with hypoxemia and hypercarbia, peak at about 72 h after injury. The timely diagnosis of pulmonary contusion requires a high degree of clinical suspicion when a patient presents with trauma caused by an appropriate mechanism of injury. The clinical diagnosis of acute parenchymal lung injury is usually confirmed by thoracic computed tomography, which is both highly sensitive in identifying pulmonary contusion and highly predictive of the need for subsequent mechanical ventilation. Management of pulmonary contusion is primarily supportive. Associated complications such as pneumonia, acute respiratory distress syndrome, and long-term pulmonary disability, however, are frequent sequelae of these injuries.

Abbreviations

ARDS	Acute respiratory distress syndrome
CI	Confidence interval
CIREN	Crash Injury Research and Engineering Network
CT	Computed tomography
Delta V	Change in velocity
ECMO	Extracorporeal membrane oxygenation
FDG	Fluorodeoxyglucose
FIO_2	Fraction of inspired oxygen
ICU	Intensive care unit
ISS	Injury Severity Score
NPPV	Noninvasive positive pressure ventilation
OR	Odds ratio
PaO_2	Arterial partial pressure of oxygen
PEEP	Positive end-expiratory pressure
PET	Positron emission tomography
ROC	curve Receiver operating characteristic curve

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Introduction

Pulmonary contusion is a common result of major trauma [1]. A review of data from the Trauma Registry at University Hospital in San Antonio, TX indicated that 1722 (27%) of 6332 patients with multiple traumatic injuries and an Injury Severity Score (ISS) of 15 or higher had pulmonary contusions. Although thoracic injuries among children are uncommon, 50% of such lesions involve pulmonary contusion [2].

Among civilian trauma populations, falls and rapid deceleration after vehicular crashes are the predominant mechanisms of lung injury. Recent reports from the Crash Injury Research and Engineering Network (CIREN) [3] demonstrated that two significant predictors of pulmonary contusion are an instantaneous change in velocity (delta V) of more than 45 mph (odds ratio [OR] = 1.9) and a frontal crash into a fixed object (OR = 1.8) [3]. Near-side lateral impact during a vehicular crash has also been implicated as an important mechanism leading to this lesion [3, 4]. In the combat setting, the shock wave produced by explosions and high-velocity projectiles may cause serious trauma to the pulmonary parenchyma.

The clinical manifestations of lung injuries may be insidious; respiratory difficulty and radiographic findings may become evident hours after injury. Patients who sustain pulmonary contusions are at a higher risk than other trauma patients of subsequent adverse events, including pneumonia and acute respiratory distress syndrome (ARDS), and long-term respiratory disability may result. Although for children the mechanism of injury differs from that for adults and generally involves being struck by a motor vehicle as a pedestrian, the outcomes of pulmonary contusions appear to be similar for both age groups [5]. Patients who have experienced trauma involving high-energy transfer should be evaluated for pulmonary contusion because prompt diagnosis and intervention may improve outcome.

History

The initial reports of pulmonary contusion focused on the description of severe lung injury with an "intact thorax" [6]. The first description of pulmonary trauma without associated chest wall injury has been attributed to Morgagni in 1761; the initial commentary in the English medical literature was written by R. W. Smith of Dublin in 1840 [6]. In these early reports, the occurrence of serious lung injury in the absence of associated bony trauma was attributed to the elasticity of the chest wall of younger patients. As Laurent [7] commented in 1883, "A 16-year-old boy was injured when a wheel passed over his chest, when he fell while attempting to jump on a moving horse cart. At autopsy, there was no external evidence of chest wall injury, but both lungs were severely contused with small lacerations. Beer was noted in the stomach!" With the exception of scattered case reports, however, little else was written about lung trauma until the beginning of the twentieth century.

In describing their experiences with injured soldiers from the battlefields of World War I, several authors reported that soldiers in proximity to the detonation of high explosives sometimes died as a result of pulmonary complications. Initially, these deaths were attributed to a variety of causes, including inhalation of toxic gases, sudden vacuum effect in gas-containing parts of the body, and even injury to the nervous system [8–10]. In 1918 and 1919, Hooker [11] conducted landmark studies by exposing animals to artillery blasts; these studies were the first to demonstrate that lung hemorrhage was responsible for the respiratory dysfunction observed after blast injuries. When war once again broke out in Europe in 1939, the World War I experiences were reviewed in anticipation of similar mechanisms of injury during World War II. Reports from the Spanish Civil War and World War I described soldiers found dead on the battlefield with no apparent external injury but with severe pulmonary hemorrhage at autopsy [12–14]. During the bombing of Britain in World War II, both civilians and soldiers who were close to explosions sustained severe "respiratory embarrassment" [15–19]. The prevalence of thoracic injuries during air raids prompted new investigations of blast-induced pulmonary injury.

At that time, considerable controversy existed as to whether the observed pulmonary pathology was due to the effect of suction (negative-pressure wave) on the lungs, the excessive distension of the lungs by air (positive-pressure wave), or the direct impact of the blast on the chest wall itself. Zuckerman [20] conducted elegant experiments designed to determine the cause of lung injury in animals placed at various distances from explosive charges. He found that the most common lesion was pulmonary hemorrhage and that the severity of injury was directly related to the distance of the subject from the epicenter of the blast. A key observation of his study was that protective garments appeared to help to lessen the severity of lung injuries caused by blast mechanisms. This finding supported the theory that lung injury after explosive blasts is due primarily to the direct impact of the pressure wave on the chest wall [20, 21]. Other investigators subsequently demonstrated that protective gear could also prevent lung injury after blast exposure in a submerged environment [22, 23].

Severe battlefield lung injuries proved common during World War II [24]. Burford and Burbank [25] described the occurrence of "wet lung" among Allied soldiers with chest injuries. These investigators examined 114 patients within 72 h of injury and found two distinct phenomena: the injured lung produced more interstitial and intra-alveolar fluid than the normal lung, and the injured lung appeared less able to rid itself of accumulated fluid. Burbank et al. [25, 26] attributed their findings to vigorous fluid resuscitation of patients who had experienced lung injury as the result of thoracic trauma.

In contrast to descriptions of pulmonary contusion associated with military experiences, descriptions of pulmonary contusion related to civilian trauma were sparse before the 1960s. In 1965, Demuth [27] was among the first to suggest that pulmonary contusion was a common occurrence after civilian thoracic trauma. He reported pulmonary contusions in 47 patients, which constituted 10% of all thoracic injuries treated over a 10-year period in a rural area of Pennsylvania. With an increasing recognition of the incidence of this pulmonary lesion, investigators turned their attention to the establishment of better diagnostic and predictive markers for pulmonary contusion. In 1965, Reid and Baird [28] reported their 2-year experience with 33 patients presenting with crushed chest injuries.

They hypothesized that direct damage to the lung itself (rather than the associated bony injury) was the primary cause of respiratory dysfunction, and they proposed that arterial blood gas measurements could be used to classify the severity of the resulting respiratory derangements.

By the end of the 1960s, the typical pathophysiology and plain radiographic findings associated with pulmonary contusion had been well delineated [28–31]. The experience of military surgeons during the Vietnam conflict provided further details about the role of high-velocity missiles and blast injuries as causative mechanisms for pulmonary contusion. The use by these surgeons of more sophisticated laboratory and monitoring techniques [32–34] provided the foundation upon which most of today's management techniques for pulmonary contusion are based.

Pathophysiology and pathology

Clemedson [35] described the primary causes of pulmonary contusion after World War II. Three basic phenomena appear to contribute to the incidence of these lung injuries. The first is the spalling effect, the shearing or bursting effect that can occur at the interface between a gas and a liquid (or between other media with large differences in density). This phenomenon is visible when a shock wave from a depth charge reaches the surface of the ocean, resulting in a "spray dome." When air-containing organs such as the lung are exposed to such forces, the spalling effect may result in the disruption of the alveolus at the point of its initial contact with the shock wave. The second phenomenon is the inertial effect that occurs when low-density alveolar tissue is stripped from heavier hilar tissues because the two types of tissue accelerate at different rates in response to the blast. The third phenomenon is the implosion effect that results from rebound or overexpansion of gas bubbles as pressure waves pass. In addition to these three basic effects, the pulmonary parenchyma may also be torn by excessive distension.

Air- and gas-containing portions of the body, such as the eardrums, the lungs, and the hollow viscera, are particularly vulnerable to the direct effects of blasts. The lungs may also be injured as a result of interaction with the surrounding thoracic cage. The action of external mechanical forces on the bony and cartilaginous outer structures can cause mechanical tearing of lung tissue or direct laceration due to displacement of fractured ribs or chest wall compression. An example of blast effect on the lung occurred in 1967 when the destroyer *Eliat* was sunk by Egyptian missile boats near Port-Said. Sailors were apparently floating in the ocean when a second missile struck the sinking ship, injuring 32 of them. Nineteen men had both lung and bowel injuries, while eight had isolated pulmonary lesions [36].

Parenchymal lung injury leads to pathophysiologic changes, the severity of which depends on the extent of injury; it may also lead to respiratory failure. Bleeding into uninvolved lung segments may cause bronchospasm and may compromise alveolar function. Associated increases in the production of mucus, decreases in the clearance of mucus from the airways, and decreases in the production of surfactant by injured alveolar tissues may also contribute to pulmonary dysfunction [37].

Segmental lung damage can result in ventilation/perfusion mismatch, elevations in intrapulmonary shunt, increases in lung fluid, and loss of lung compliance [38]. These pathologic changes manifest themselves clinically in the form of hypoxemia, hypercarbia, and increased work of breathing [30]. Patients may present with rapid respiratory rate, rhonchi or wheezes, or even hemoptysis. Associated changes may not be visible on plain chest radiographs until 4–6 h after injury, and these changes may not accurately reflect the extent to which the pulmonary tissue is affected [29]. In general, the respiratory derangements associated with pulmonary contusions resolve within 3–5 days, but delayed deterioration may occur. Late pulmonary dysfunction may also occur after contusion as a result of the local inflammatory response to injury and sequestered blood, the systemic inflammatory responses related to associated injuries, or the development of nosocomial pneumonia [37].

Severe pulmonary hemorrhage, or "hepatization of the lung," was the typical anatomic finding in lung contusions examined by Hooker [11]. Pathologic findings at autopsy have revealed lesions characterized by diffuse hemorrhage: phrenicocostal sinus contusions with related liver damage, rib markings, massive hilar contusions, or hemorrhagic spots (Fig. 1) [18, 39]. Animal model studies have identified a characteristic pattern of lesion progression after injury [40, 41]. This progression is marked by initial interstitial hemorrhage, followed after 1–2 h by the onset of interstitial edema. These animal models have demonstrated that the architecture of the lung appears to be preserved but that the infiltration of monocytes and neutrophils is apparent at early intervals. At 24 h after injury, protein, red blood cells, and massive accumulations of inflammatory cells are found in the air spaces, along with fibrin and loss of normal architecture as massive edema increases. By 48 h after injury, large amounts of fibrin and cell debris and large numbers of type II alveolar cell-derived granulocytes, neutrophils, and macrophages have accumulated. At this stage, the lymphatic vessels appear to be dilated and filled with protein. By 7–10 days after traumatic lung injury, healing is almost complete in these animals, with little residual scarring [42].

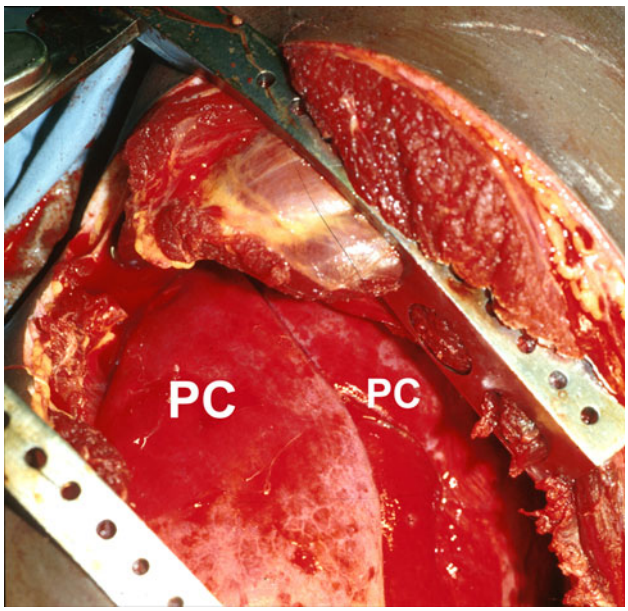

Fig. 1 Gross anatomic picture of left hemithorax during emergency thoracotomy. Pulmonary contusion (PC) noted involving multiple lobes of the lung

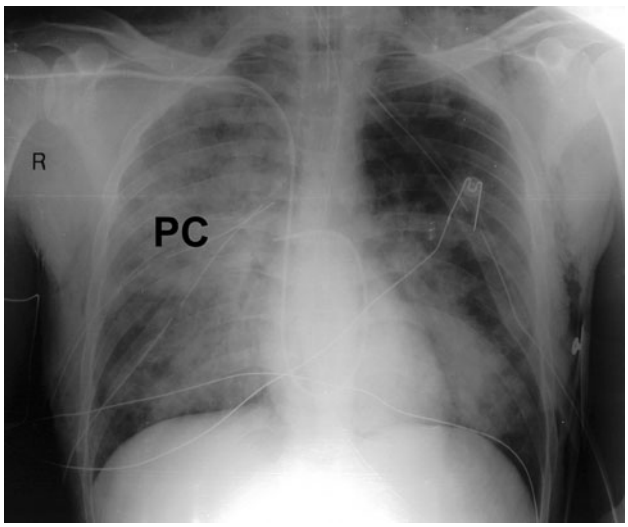

Fig. 2 Chest radiograph demonstrating large pulmonary contusion (PC) involving right lung

After pulmonary contusion, acute pathologic changes are associated with important physiologic alterations in hemodynamics [43]. Using computed tomography (CT) to determine the degree of injury and correlating this quantification with estimates of pulmonary vascular resistance and shunt fraction, investigators have found that some patients experience serious pulmonary vasoconstriction after injury. It has been postulated that this finding reflects a compensatory mechanism that limits perfusion to traumatized parenchyma, thereby minimizing any increase in shunt fraction. Some patients ("nonreactors") do not exhibit this response but instead, for unknown reasons, exhibit unchecked increases in shunt fraction [43].

Imaging

Early reports of plain radiography findings associated with pulmonary contusion described characteristic consolidation patterns consistent with "traumatic pneumonia" (Fig. 2), typically appearing within 4–6 h after injury [20, 44] and "vanishing" within a few days [45]. There appears to be a relationship between the extent of abnormalities seen on chest radiograph and the severity of clinical findings [46–48]. In primate models of injury, the findings of chest radiography have been noted to underestimate the actual degree of injury as determined at autopsy [49]. In the clinical setting, the exclusive use of plain chest radiographic imaging often contributes to the underdiagnosis of pulmonary contusion: only 47% of lesions are detected at the time of admission, whereas 92% are seen 24 h after injury [50]. In addition, it should be noted that enlargement of lung contusions on X-ray during the first 24 h is generally a negative prognostic sign. Furthermore, the degree of contusion can be hard to separate clinically from the effects of aspiration, fluid overload, transfusion (e.g., TRALI), and embolization. Interpretation of CT scan images can lead to confusion in differentiating pulmonary collapse/consolidation from aspiration with direct contusion, and hemothorax can mimic intrapulmonary hemorrhage. It should be noted that the CT scan may identify radiographic abnormalities that are deemed pulmonary contusions which do not lead to any respiratory dysfunction. These lesions therefore are of minimal clinical relevance and may be deemed asymptomatic. Some authors have suggested therefore that the latest generation of CT scanners may be overly sensitive in delineating this lesion and that those patients with pulmonary contusion identified by "CT-only" appear to have a better outcome than those lung lesions noted on both plain radiographs and CT [51].

Attempts to use chest radiography findings to create scoring systems that can predict outcome after traumatic lung injury have been limited by the lack of sensitivity. Therefore, chest radiography findings have been replaced in such scoring systems by thoracic CT findings (Fig. 3), which have proved highly accurate. A comparison of the findings of plain radiography and CT of the chest in a dog model found that initial radiographs did not demonstrate the lesion in two-thirds of the cases. In contrast, 100% of these lesions were discernable on initial CT scans. An assessment of clinical accuracy showed that radiographs obtained even 6 h after injury failed to demonstrate pulmonary contusion in 21% of chest trauma patients. In

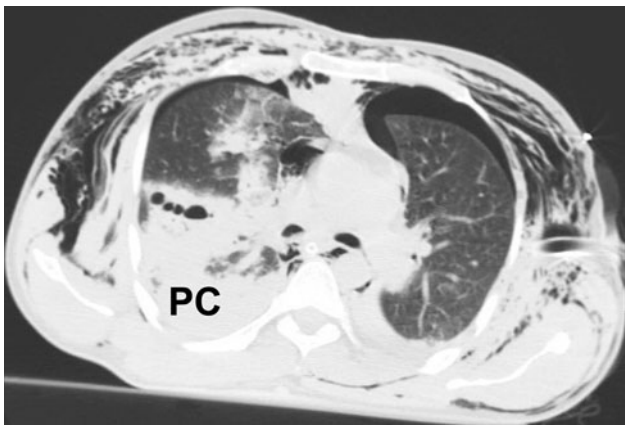

Fig. 3 Thoracic CT scan demonstrating opacification representing pulmonary contusion (PC) of right lung. Subcutaneous emphysema and pneumothoraces are seen bilaterally

addition, the findings of chest radiography contributed much more frequently to the underestimation of the extent of lesions than did the findings of CT scans [52]. In a CT study of 103 patients with blunt thoracic trauma, 33 had pulmonary contusions that were not visible on a plain chest radiograph. A unique finding of chest CT scans is subpleural sparing, which can be seen in 95% of children with pulmonary contusion but is not seen in children with atelectasis or pneumonia [53]. Chest radiographs also grossly underestimate the degree of ventilation/perfusion mismatch, which can be better estimated with nuclear imaging studies [54].

Wagner et al. [55, 56] demonstrated that CT scans can effectively stratify pulmonary contusion injuries on the basis of the type and severity of the lesion. Using this classification system, the authors could predict the patient's need for ventilatory support by determining the percentage of airspace consolidation that was apparent on CT scans. When more than 28% of the airspace was involved, all patients required subsequent mechanical ventilation. The ability of CT to discern the severity of pulmonary contusion has been confirmed by other investigators [57, 58]. Spiral CT allows the clinician to predict reliably which patients will experience severe hypoxemia. Miller et al. [58] assessed 49 patients with pulmonary contusions (35 bilateral). The lesions were classified according to contusion volume as moderate (volume <20%; $n = 32$) or severe (volume ≥20%; $n = 17$). The average contusion volume was 18% (range = 5-55%). The moderate and severe groups were similar in ISS (moderate, 26.5; severe, 23.3; $P = 0.33$), admission blood pressure (moderate, 129 mmHg; severe, 131 mmHg; $P = 0.90$), and the ratio of the arterial partial pressure of oxygen (PaO_2) to the fraction of inspired oxygen (FIO_2) at the time of admission (PaO_2/FIO_2; moderate, 255; severe, 197; $P = 0.14$). However, the rate of ARDS in the moderate contusion group (22%) was much lower than that in the severe group (82%; $P < 0.001$).

Other investigators have found similar relationships between the amount of lung volume injured, as demonstrated on CT scans, and the extent of pulmonary derangements [59]. Contemporary researchers have proposed the use of magnetic resonance imaging [60], nuclear imaging [61], and even ultrasonography [62] for the diagnosis of pulmonary contusion. For example, some investigators have found that chest ultrasonography, well known to be extremely user dependent, is highly sensitive and specific in diagnosing pulmonary contusion [63]. They support the use of this imaging method in both the emergency center [63] and the intensive care unit (ICU) [64]. Because of the high accuracy and reproducibility of CT and its ready availability at most trauma centers, this imaging technique is currently the standard of care for the diagnosis and risk stratification of pulmonary contusions [56–58]. However, there may be no need to get a CT scan for a patient with a mild pulmonary contusion and no physiologic compromise.

Management

Initial interventions

The management of pulmonary contusion is primarily supportive. Beginning in the prehospital setting, supplemental oxygen and rapid assessment of airway and breathing should be undertaken as recommended by standard trauma protocols. Because pulmonary contusions may be associated with severe hypoxemia, patient transport can be hazardous. During prehospital transfer, some of these patients may benefit from being placed so that the obviously injured hemothorax is in a dependent position; this positioning limits bleeding into the contralateral, less injured lung. However, such positioning may worsen hypoxemia because the effect of gravity may increase blood flow to the injured lung and cause a greater ventilation/perfusion mismatch.

After arriving at the hospital, the patient should be rapidly examined and treated in accordance with modern principles of trauma care. Very rarely, patients with unilateral pulmonary contusions accompanied by massive intratracheal bleeding or severe air leaks may require early selective bronchial intubation [65]. Alternatively, patients with diffuse lung contusions may require endobronchial blockers, which have been used successfully to control hemoptysis [66]. Bronchial blockers can help control bleeding into the uninjured lung, provide tamponade at bleeding sites, and decrease the likelihood of air embolization. They eliminate the need for the risky procedure of changing endotracheal tubes when a severely injured

patient requires one-lung or dual-ventilator management. The use of one-lung ventilation for a patient with unilateral thoracic injuries improves oxygenation and reduces the ventilation/perfusion mismatch while maintaining airway pressures within a safe range [67]. Rarely, dual-lung ventilation and even lobectomy may be needed for patients with very severe pulmonary injuries [68]. Severe hypoxemia or hemodynamic instability after pulmonary contusions may preclude the routine use of whole-body CT for trauma patients. In such circumstances, ultrasonography or diagnostic peritoneal lavage should be used to exclude abdominal injuries, and transesophageal echocardiography should be used to exclude aortic injuries.

Optimization of oxygenation and ventilation

Aggressive pulmonary toilet, meticulous fluid management, and control of chest wall pain are paramount in the treatment of all patients with pulmonary contusion. In cases of severe injury, intensive monitoring is also essential for ensuring adequate organ perfusion and oxygenation. For patients with severe unilateral lung lesions, arterial desaturation may be minimized by the use of a kinetic or rotating bed [69]. We have observed that rotating patients with an uninjured contralateral hemithorax into the dependent position can improve oxygenation, possibly by increasing blood flow to the uninjured or "good" lung and optimizing the ventilation/perfusion relationship. The use of constantly rotating beds has been shown to significantly reduce the incidence of pneumonia and the number of ventilator days after major thoracic trauma [70]. If specialty rotating beds are not available, intermittent prone positioning (instituted by some authors for intervals totaling 8 h per day) [71] significantly increases oxygenation parameters after severe to moderate traumatic lung injury.

Patients should undergo intubation only if they exhibit respiratory difficulties [72]. In addition, noninvasive positive-pressure ventilation (NPPV) may be appropriate for selected patients with pulmonary contusion and hypoxemia. Antonelli et al. [73] performed a prospective multicenter evaluation of 2,770 patients with hypoxemic acute respiratory failure. Of these, 354 met the criteria for NPPV and did not initially undergo intubation. Of the patients who underwent NPPV, 70% did not require any intubation; the greatest success in avoiding intubation was observed in patients with cardiogenic pulmonary edema (90 of 99 patients, 90%) or pulmonary contusion (59 of 72 patients, 82%). About 10% of patients with acute lung injury (ALI) ($PaO_2/FIO_2 = 200$-300) may avoid intubation if treated with NPPV [74].

Underlying pulmonary disease or associated injuries may dictate the need for ventilatory support in certain patients. When required, mechanical ventilation strategies should be tailored to optimize oxygenation while minimizing the potential for secondary lung injury. Several strategies have been used successfully, including those that have emphasized the routine use of alveolar recruitment maneuvers [75] and permissive hypercapnia in difficult cases [76]. For patients with ALI or ARDS after pulmonary contusion, lung recruitment maneuvers (increasing intrinsic and extrinsic positive end-expiratory pressure [PEEP] to open previously closed lung units; using high-frequency inverse ratio ventilation) have been shown to increase arterial oxygenation, normally aerated lung volume, and total lung volume while decreasing the amount of collapsed lung tissue [75]. The successful use of high-frequency oscillatory ventilation for patients with severe pulmonary contusions has also been reported [77, 78].

Interestingly, direct damage to the lung after trauma alters the composition of surfactant. A study that used daily bronchial alveolar lavage to analyze unilateral pulmonary contusions in 18 patients found that total phospholipid concentrations and the percentage contents of phosphatidylcholine and sphingomyelin were significantly higher in the contused lungs than in the normal lungs [79]. Subsequently, a small prospective randomized pilot study ($n = 8$ per arm) found that adjunctive exogenous bovine surfactant replacement (single dose, 200 mg/kg) improved oxygenation and decreased ventilatory requirements in patients with pulmonary contusions and hypoxemia. [80]. Any ventilation strategy or adjunctive therapy should optimize oxygenation while minimizing the risk of additional ventilator-associated barotrauma [81]. When oxygenation is not possible, as may be the case for patients with ARDS after pulmonary contusion in spite of the use of typical heroic measures (high-frequency ventilation, prone positioning, nitric oxide inhalation, prostaglandin infusion), extracorporeal membrane oxygenation (ECMO) has occasionally been life-saving [82].

Pain control and restoration of pulmonary mechanics

The pain associated with an injury to the thoracic wall may contribute substantially to hypoventilation in patients with pulmonary contusion. Aggressive attempts to alleviate discomfort and restore unhindered pulmonary mechanics are mandatory. Regional analgesia is preferred to establish pain control whenever possible as oral or intravenous medications alone typically do not successfully alleviate the severe pain associated with rib fractures. Delay in alleviating chest wall pain may lead to hypoventilation, atelectasis, and subsequent respiratory deterioration. Intrapleural, paraspinal, or intercostal block may be a viable option, but the results of several studies suggest that epidural catheters provide optimal pain relief and may improve outcome [83, 84]. Unfortunately, many trauma patients have spinal

fractures or other injuries that preclude the use of epidural catheters. For example, Bulger et al. [83] studied 408 patients admitted with more than three rib fractures. Of these, 282 (69%) met exclusion criteria, 80 could not consent, and 46 were prospectively randomly assigned to receive epidural pain relief ($n = 22$) or intravenous opioids ($n = 24$). The latter two groups were similar in mean age, injury severity score, sex, chest Abbreviated Injury Scale, and mean number of rib fractures. The epidural group had more flail segments (38 vs. 21%, $P = 0.20$) and pulmonary contusions (59 vs. 38%, $P = 0.14$) and required more chest tubes (95 vs. 71%, $P = 0.03$). However, in spite of this difference between groups in the number of direct pulmonary injuries, the rate of pneumonia was 18% for patients in the epidural group and 38% for patients in the intravenous opioid group. When the analysis was adjusted for direct pulmonary injury, the risk of pneumonia was higher for the opioid group (OR = 6.0; 95% confidence interval [CI] = 1.0-35.0; $P = 0.05$). When the analysis was stratified for the presence of pulmonary contusion, the opioid group exhibited a twofold increase in the number of ventilator days required (incident rate ratio = 2.0; 95% CI = 1.6-2.6; $P < 0.001$). It therefore appears that a subset of patients with chest wall injury (approximately one-third of severe chest wall injuries) are candidates for and benefit from epidural analgesia. The use of catheter-based delivery systems for continuous regional analgesia after thoracic surgery is an option that warrants additional investigation [84].

Early experiences with operative chest wall fixation of flail chest in the setting of pulmonary contusions were not favorable [85]. However, the results of subsequent prospective investigations suggest that surgical stabilization may be beneficial in restoring chest wall mechanics and optimizing the subsequent ventilator course of patients with flail chest and pulmonary contusions. Tanaka et al. [86] prospectively compared the clinical effectiveness of surgical stabilization with that of routine ventilator management in 37 patients with severe flail chest injuries requiring prolonged ventilatory support. On day 1 after injury, patients were randomly assigned to surgical stabilization with Judet struts ($n = 18$) or internal pneumatic stabilization ($n = 19$). The two groups were similar in ISS, chest Abbreviated Injury Score, number of rib fractures, severity of lung contusion, and PaO_2/FIO_2 at the time of admission. Compared to the routine care group, the surgical stabilization group experienced a shorter ventilatory period (10.8 ± 3.4 days vs. 18.3 ± 7.4 days), a shorter stay in the ICU (16.5 ± 7.4 days vs. 26.8 ± 13.2 days), and a lower incidence of pneumonia (24% vs. 77%); all differences were statistically significant at the level of $P < 0.05$. The number of patients who had returned to full-time employment by 6 months after injury was also significantly higher in the surgical stabilization group (11 of 18 patients vs. 1 of 19 patients). These authors recommended surgical intervention for patients with severe flail chest injuries. Certainly, large prospective studies are required before routine use of rib plating can be advocated.

Irrespective of the modality, an aggressive approach toward combating the effects of pain on pulmonary mechanics should be undertaken in all cases. Adequate pain control allows the critical care team to better institute chest physiotherapy and early mobilization which are believed to help lower the likelihood of mechanical ventilation. Richardson et al. [87] found that aggressive critical care management with such approaches for patients with severe chest trauma reduced the need for intubation from 100 to 23%, with an associated improvement in the survival rate from 60 to 93.5%.

Resuscitation

Fluid management has been a controversial issue since Burford and Burbank [25] attributed the incidence of "wet lung" in World War II soldiers with thoracic injuries to overzealous fluid administration. The results of studies using animal models have been inconclusive regarding the potential adverse effects of infused fluids [88–94], and relatively few human clinical trials have addressed the impact of resuscitation on pulmonary contusion. Collins et al. [95] studied casualties from the Vietnam War and found that the volume of blood transfused was associated with the incidence of hypoxemia among soldiers who had sustained thoracic injuries, but only among those with evidence of direct lung trauma. A subsequent confirmatory study measured lung water in 16 civilian trauma patients presenting with shock (mean arterial pressure < 40 mmHg) and found that lung water was increased only in the patients with pulmonary contusions, not in those with hemorrhagic shock in the absence of pulmonary trauma [96].

Other investigators, however, have found no association between the amount of fluid resuscitation and the degree of pulmonary dysfunction or the need for ventilatory support after lung contusion [97, 98]. Subsequently, compensatory pulmonary mechanisms such as increased lymph drainage in models of hemorrhagic shock have been proposed as an explanation for many of the findings associated with the course of pulmonary contusion. Unfortunately, experimental data in this area have proved contradictory: some authors have found evidence of increased pulmonary capillary permeability (and increased lung water) in patients who have undergone resuscitation for hemorrhagic shock [99], but others have not [100].

As our understanding of hemorrhagic shock continues to evolve, innovative resuscitative approaches have emerged.

Hypertonic saline, for example, has been shown to effectively restore perfusion after hemorrhagic shock, and the volume requirement is smaller than that for traditional high-volume isotonic alternatives or blood product-based approaches [101]. The results of studies using animal models of hemorrhage suggested that the use of hypertonic saline, when compared to the use of isotonic fluids, may decrease the amount of edema in the brain [102] and the visceral organs [103, 104]. The results of studies using hypertonic saline in animal models of lung injury, however, have proved contradictory. Although some investigators have found that hypertonic saline resuscitation for rats with pulmonary contusion decreases the amount of pulmonary inflammation and increases cellular protection [105], others have failed to detect any benefit [106]. In our series of experiments using pigs subjected to pulmonary contusion and hemorrhage, small-volume hypertonic saline resuscitation failed to reduce the magnitude of lung injury or to provide substantial physiologic benefit over isotonic solutions [107]. No clinical studies have demonstrated a pulmonary physiologic benefit from the use of hypertonic saline after thoracic injury. Recently, a large prospective trial evaluating the value of hypertonic saline resuscitation in trauma was terminated due to lack of apparent efficacy (E. Bulger, personal communication).

Red blood cell substitutes, like hypertonic saline, can restore perfusion after hemorrhagic shock even though limited fluid volumes are used [108]. As is true for hypertonic saline, however, the benefits of red blood cell substitutes for patients with thoracic injury have not been defined. Using a porcine model of pulmonary contusion and hemorrhage, we [109] found that resuscitation with a hemoglobin-based blood substitute led to pulmonary hypertension, larger pulmonary contusions, and stiffer lungs.

Arginine vasopressin has emerged as a promising treatment adjunct for vasodilatory shock. Using a swine model, we [110] examined the therapeutic potential of administering vasopressin to animals subjected to severe chest trauma and pulmonary contusion. Remarkably, the early use of vasopressin was associated with a decrease in the mortality rate, a reduction in fluid requirements, and an improvement in pulmonary function. Clinical trials examining the effects of administering vasopressin specifically to trauma patients, including those with pulmonary contusion, are ongoing.

Antibiotics and steroids

No studies have demonstrated a benefit from the prophylactic use of antibiotics after pulmonary contusion. Certainly, the indiscriminate use of antimicrobial agents should be avoided so that the development of bacterial resistance can be limited. Similarly, no clinical findings suggest that corticosteroids are useful for patients with pulmonary contusion. The results of some animal studies suggest that steroid use may ameliorate hypoxemia and decrease the size of the contusion [111], but others have failed to find such benefits [112]. The use of steroids may, in fact, contribute to decreased bacterial clearance [94] and subsequent increases in the risk of pneumonia. It has been suggested that steroids decrease pulmonary vascular resistance after injury, but the clinical benefit of this effect has not been established [113, 114]. Currently, corticosteroids have no role in the management of pulmonary contusion.

Outcome

Overall outcomes after pulmonary contusion may be affected by the mechanism and pattern of injury. Israeli investigators studied 154 patients who were injured as a result of 17 bombing attacks; 28 (18.2%) of these patients sustained blast lung injuries. Patients with penetrating head injury and those with injuries to four or more body areas were significantly more likely to suffer from blast lung injury (OR = 3.47 and 4.12, respectively; $P < 0.05$) than were other victims of bombing attacks [115]. Experiences with civilian victims of blast injury suggest that although most patients with serious contusions due to this mechanism of injury will typically require mechanical ventilation, late deterioration of pulmonary status is rare, death attributable to lung injury among those who survive the initial blast is unusual, and timely diagnosis and correct treatment will produce excellent outcomes [116, 117].

The evolution of care for patients with pulmonary contusion has resulted in considerable improvement in outcomes. In spite of medical advances during the 1950s and 1960s, the use of ventilators, antibiotics, and monitoring failed to reduce the adverse outcomes associated with pulmonary contusions. Historically, the mortality rate after these lung injuries was approximately 40% [27, 118, 119]. Although the use of ventilators markedly decreased the number of early deaths due to respiratory failure, it was associated with delayed mortality due to sepsis and with tracheostomy complications [120]. Before the 1970s, all patients with crushed chest injuries underwent mechanical ventilation. Trinkle et al. [72] were among the first to implement a selective intubation protocol for thoracic trauma patients with respiratory compromise. They reported that 427 patients with blunt chest trauma underwent selective intubation, whereas 328 such patients were managed initially without intubation (only 10 of these patients eventually required ventilator support). The mortality rate in this study was 6.5%.

Pulmonary contusion does, however, increase the risk of morbidity for victims of trauma. The results of animal and clinical studies support the notion that pulmonary contusion decreases the clearance of bacteria [94] and predisposes patients to pulmonary infection [121]. For patients with severe thoracic wall injury or flail chest, pulmonary parenchymal injury markedly increases both the likelihood of intubation (from 20 to 50%) [87] and the associated mortality rate (from 16 to 42%) [122]. The severity of contusion predicts the need for ventilatory support after thoracic trauma [55]. Pulmonary contusion has been shown repeatedly to be an important risk factor for ARDS [123–125]. In one early study, pulmonary contusion led to ARDS in 38% of 50 multitrauma patients [126]. A recent study found that ARDS developed in 200 (5%) of 4397 patients with blunt trauma. The strongest predictors of the development of ARDS were an ISS higher than 25 (area under the receiver operating characteristic [ROC] curve, 0.72) and pulmonary contusion (area under the ROC curve, 0.68) [123]. Of note, intramedullary nailing of the femur or tibia does not appear to increase the risk of respiratory dysfunction for multitrauma patients with pulmonary contusions [127]. An interesting new imaging procedure, 18F-fluorodeoxyglucose positron emission tomography (FDG PET), has facilitated the determination of which patients with pulmonary contusions are likely to experience ARDS. The patients who subsequently develop ARDS demonstrate diffuse FDG uptake, whereas other thoracic trauma patients with pulmonary contusions do not [128].

Post-traumatic empyema is a rare but serious sequela of pulmonary contusion. In one study, empyema was found in only 71 (3%) of 2261 thoracic trauma patients. The most important risk factors for empyema were retained hemothorax (OR = 5.50; $P < 0.001$) and pulmonary contusion (OR = 3.06; $P < 0.001$) [129].

The long-term consequences of pulmonary contusion have not been clearly defined. Kishikawa et al. [130] investigated the impact of lung trauma on patients with flail chest who had ($n = 12$) or did not have ($n = 9$) pulmonary contusion. Before discharge and 6 months after injury, patients underwent both spirometry and blood gas analysis. No residual pulmonary derangements were found after flail chest alone, but persistent abnormalities in functional residual capacity (approaching closing volume) and oxygenation were detected after lung contusion. Patients with pulmonary contusion, but not those with flail chest alone, frequently exhibited disabling dyspnea. Subsequent chest CT scans revealed fibrosis in the lungs of pulmonary contusion patients with dyspnea [130]. Leone et al. [131] performed long-term follow-up on 55 patients with multiple trauma associated with blunt chest trauma. Six months after injury they noted that pulmonary function tests were impaired, and physical function was decreased in 70% of patients, resulting in reduced pulmonary-specific quality of life. On the other hand, two Israeli studies found that most patients with blast lung injury will either experience no long-term sequelae [97] or regain good function within 12 months after injury [131]. Eleven survivors of severe blast injury to the lung were examined 1 year after injury and were found to have normal lung function and complete resolution of pulmonary parenchymal abnormalities, as shown by chest radiographs [132]. Among children with pulmonary contusion, long-term follow-up (4.5 ± 1.0 years) revealed normal lung function and normal appearance on chest images [133].

Summary

Pulmonary contusion is common after mechanisms of injury that impart substantial kinetic energy to the thorax. Hemorrhage into the lung substance leads to pulmonary pathophysiologic changes that worsen for 24–48 h and then generally resolve by 7 days after injury. The diagnosis of pulmonary contusion is confirmed by pulmonary dysfunction and radiographic findings. Computed tomography of the chest is clearly superior to plain radiography in identifying a pulmonary contusion and may be helpful in predicting the need for mechanical ventilation and the likelihood of pneumonia or ARDS. Management of patients with pulmonary contusion is supportive and requires judicious fluid administration, control of the pain associated with bony thoracic injuries, and careful hemodynamic monitoring. In spite of presently available diagnostic and therapeutic interventions, pulmonary contusions are associated with substantial morbidity.

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