The Pathophysiology, Diagnosis and Management Strategies for Flail Chest Injury and Pulmonary Contusion: A Review

John (Hans) W. Schweiger, MD, FCCP

John (Hans) W. Schweiger, MD is the recipient of the IARS 2000 Anesthesiology Teaching Recognition Award. Dr. Schweiger's award-winning manuscript is included in the IARS 2001 Review Course Lectures Book. Dr. Schweiger will be presenting a Tutorial titled, "Current Management of Flail Chest" at the IARS 75th Clinical and Scientific Congress on Sunday, March 18 at 7:00 am.

Learning Objectives:

1. Understand the pathophysiology of flail chest injury and pulmonary contusion.
2. Accurately diagnose blunt, non-penetrating chest injuries.
3. Identify areas of controversy and review evidence regarding management options.
4. Discuss novel approaches to peri-operative ventilatory support and oxygen therapy.
5. Summarize therapeutic recommendations for management of flail chest injury and pulmonary contusion.

Until World War II, flail chest injury and pulmonary contusion received little attention in the medical literature. During the 1950s and 60s, improved initial resuscitation, triage, and transportation of trauma victims allowed more patients to survive the immediate injuries and present to hospital. Gradually, the significant morbidity and mortality associated with nonpenetrating pulmonary injuries began to be recognized in the medical literature. Blunt chest trauma contributes to nearly one-third of all annual admissions to US trauma centers, and remains a major factor in trauma-related mortality (1). It ranks third in frequency, behind major central nervous system and extremity injuries, as a consequence of nonpenetrating trauma. More than two-thirds of cases occur as a result of motor vehicle accidents; the remainder result from crush injuries, falls and/or blast injuries (2). The reported incidence of flail chest resulting from thoracic trauma ranges from 12% to 25% (3,4). Pulmonary contusion follows rib fractures as the most common injury resulting from blunt chest trauma, and occurs in 40% to 60% of these victims (3,4). Over the past 50 yr, we have gained much understanding of the pathophysiology, diagnosis, and management of nonpenetrating chest trauma. Nevertheless, considerable misunderstanding persists in the medical literature, and contributes to less than optimal patient care. With the advent of the anesthesiologist as "perioperative" physician, blunt chest trauma provides a unique opportunity to participate in the pre- and intraoperative resuscitation and stabilization of the trauma victim, as well as supervise postoperative therapy in the intensive care unit (ICU) and acute/chronic pain management. This report will examine the evolution in thought leading to current management strategies, and briefly review both conventional and controversial therapeutic interventions.

Pathophysiology of Flail Chest and Pulmonary Contusion

Blunt chest trauma frequently disrupts the structural integrity of the chest wall. It also may result in significant injury to the underlying lung parenchyma. As a consequence, the injured victim may develop arterial hypoxemia, paradoxical chest wall motion and acute respiratory failure. The exact mechanism by which multiple rib fractures, with or without acute lung injury, interfere with normal gas exchange has not been fully described. A flail chest results when several ribs are fractured at two or more sites or when the ribs become disarticulated from their cartilaginous attachments with the sternum. Multiple rib fractures may precipitate respiratory impairment characterized by paradoxical motion of the flail segment and increased work of breathing. Paradoxical chest wall motion manifests as a "caving in" of the flail segment on inspiration, followed by a "bulging out" on exhalation. These movements are dyssynchronous with the remainder of the thorax and its normal relationship to diaphragmatic movement. During spontaneous inspiration, a flail occurs as a consequence of atmospheric pressure on the surface of the chest pushing inward on the unattached segment as the pressure within the pleural space decreases. Thereafter, the flail moves outward as exhalation generates an increase in pleural pressure. The flail segment itself is usually not detrimental, unless a bony fragment of the fractured rib

lacerates the lung parenchyma and causes a pneumothorax. The intense pain, muscle spasm and increased elastic recoil of the lung associated with the injury can, however, frequently cause the patient to develop 1) a rapid, shallow breathing pattern, 2) an involuntarily "splinting" of the injured hemithorax, 3) an inability to clear secretions, and 4) an inability to generate an effective cough. Often, these factors may predispose the patient to develop pneumonia.

In 1952, Jensen (5) reintroduced a hypothesis, first introduced by German physicians, that flail chest causes acute respiratory insufficiency as a result of a "pendelluft" effect. He theorized that inspiration might generate a pressure gradient directed toward the healthy hemithorax. As a result, during exhalation decreased intrathoracic pressure would be generated within the injured hemithorax. It was postulated that an inefficient pendulum-like motion of gas would occur in the distal airways and interfere with the normal to-and-fro movement of gas in the conducting airways, resulting in rebreathing and inefficient alveolar gas exchange. Management of a flail chest concentrated on the mechanical dysfunction of the chest wall, and treatment was aimed at external stabilization of the flail segment to minimize the pendelluft effect (6). Subsequent work by Maloney et al. (7) and Duff et al. (8) discredited this theory. They demonstrated normal ventilation within the injured hemithorax despite the paradoxical chest wall motion.

A paradigm shift occurred in our understanding of blunt chest trauma after it was realized that injury to the lung parenchyma, not abnormalities in chest wall mechanics, predisposed patients to acute respiratory failure. An impact with sufficient force to fracture multiple ribs routinely transmits sufficient energy to the chest wall to traumatize the underlying lung parenchyma. This premise has been supported by numerous studies demonstrating a high correlation between flail chest and pulmonary contusion (4,9). As a result of the acute lung injury and subsequent decrease in lung compliance, the work of breathing required to maintain normal alveolar ventilation increases dramatically. Although the increase in work of breathing is difficult to quantify, it results from the increased elastic recoil of the lung, and impairs the patient's ability to expand the injured lung fully during inspiration. Garzon et al. (10,11) studied pulmonary mechanics in patients with lung contusion, and found that lung compliance was decreased in nearly all patients. They found that lung compliance did not return to bas-line values until several weeks after the injury. Moreover, airway resistance was also increased for almost a week. Lung contusion causes a decrease in the patient's functional residual capacity (FRC) and a concomitant decrease in lung compliance. The decrease in FRC necessitates an increase in the magnitude of the pleural pressure swings and results in increased work of breathing. Subsequently, the paradoxical movement of the flail segment will be accentuated.

The contused area of lung is characterized by disruption of the alveolar architecture, interstitial and intraalveolar hemorrhage, and atelectasis. Microscopic analysis demonstrates that disruption of the lung parenchyma begins to occur within minutes of the initial injury, as a result of hemorrhage into the alveolar and interstitial spaces (12). Within 1–2 h, the injured lung shows evidence of pulmonary edema as the capillary endothelium begins to leak. Edema accumulates in the interstitial space, but later involves the alveoli themselves. In response to the injury, mono- and polymorphonuclear cells infiltrate the affected area. After 18 to 24 h, the lung's architecture is distorted by an accumulation of cellular debris, hemorrhage, and inflammatory cells. The inflammatory response also triggers the production of a proteinaceous exudate which, if it accumulates in large quantities, may interfere with alveolar-capillary gas exchange. Eventual return of normal lung architecture and recovery lung function may take several weeks.

Few consistent predictors of morbidity and mortality have been identified in patients with flail chest and pulmonary contusion. Major premorbid factors include the victim's age, past medical history, and baseline health status before the injury (13). The injury severity score (ISS) reflects the severity of all associated traumatic injuries, as well as the volume of blood transfused during resuscitation (14). In regards to mortality related to blunt chest trauma, a higher ISS score was found to correlate with major trauma to the central nervous system and long bone fractures. An ISS score >30 in patients >44 yr old or an ISS >40 in patients <44 yr old was associated with a higher mortality. Some investigators have found significance in the volume of blood transfused, whereas others have not reported any predictive value, arguing that increased blood transfusion simply reflects the severity of associated injuries (1).

Diagnosis of Flail Chest and Pulmonary Contusion

The diagnosis of flail chest and pulmonary contusion is contingent on the clinician's index of suspicion. Accurate diagnosis of blunt chest trauma is important so that therapeutic interventions may be instituted in a timely manner. Whenever possible, information describing the mechanism of injury should be obtained from emergency personnel. Appropriate interventions help to prevent, or at least to minimize, the occurrence of pulmonary complications such as atelectasis, retained secretions, tracheobronchitis, pneumonia, and/or acute respiratory failure.

A diagnosis of flail chest can be made after careful examination and inspection of the chest wall during

several respiratory cycles. The physical examination should begin by gently exposing the patient's torso in a well-lit room, and inspecting the chest wall for bruising, hematoma, bony deformities, and/or areas of paradoxical chest wall motion. Direct palpation of the thorax may elicit crepitus and/or pain, which will help to localize the site of injury. In some cases, initial inspection of the thorax may not reveal a flail segment, particularly if the patient is employing a rapid, shallow breathing pattern and generating small tidal volumes. Landcasper et al. (15) found that flail chest injury was documented in only 78% of patients on initial examination, and delayed in 22% of cases from 1 to 10 days after injury. A chest radiograph may prove helpful if it can direct inspection toward an area of previously unidentified rib fractures. However, it may be of little help in detecting cartilaginous injuries or in those patients with poorly calcified ribs.

As previously mentioned, much of the morbidity and mortality associated with blunt chest trauma is related to injury of the underlying lung parenchyma. Therefore, it is equally important to diagnose the presence and extent of pulmonary contusion correctly, so that appropriate and expeditious treatment can be instituted. An eventual diagnosis, made on the basis of radiologic pulmonary edema, is one that has occurred far too late. Clinical signs of pulmonary contusion include dyspnea, tachypnea, intercostal muscle retraction, and the use of accessory muscles of respiration. If the patient is allowed to breathe room air, pulse oximetry can provide early diagnostic information because oxyhemoglobin saturation is frequently decreased. Arterial hypoxemia may then be confirmed by arterial blood analysis. The routine administration of supplemental oxygen, during the initial evaluation, may mask a considerable ventilation/perfusion (V/Q) inequality responsible for arterial hypoxemia. Early V/Q mismatch can best be observed when the patient is allowed to breathe room air or a low Fio_2. Failure to diagnosis this V/Q inequality accurately may lead the physician to 1) overestimate the patient's pulmonary reserve, 2) delay investigation into the reason for the oxyhemoglobin desaturation, and 3) withhold appropriate therapy to restore the FRC and lung compliance toward normal.

A chest radiograph may prove useful as an adjunct for diagnosis, but its sensitivity is not very high; it has been reported to be as low as 70% (16). Unfortunately, it is frequently difficult to obtain a good quality chest film as a result of the patient's immobilization and/or inability to perform deep inspiration. The diagnosis of pulmonary contusion should be entertained when a new, focal infiltrate occurs underneath an area of multiple rib fractures. Radiographic abnormalities consistent with pulmonary contusion may lag several hours behind deterioration in the patient's clinical condition. Erickson et al. (17) demonstrated that radiographic evidence of pulmonary contusion developed in only 33% of traumatized monkeys 30 min after injury, but focal infiltrates subsequently developed in 77% of the animals after 4 h. For many years, it was believed that there was an association between the size of the flail segment, and the incidence of subsequent respiratory failure. Clark et al. (1), however, found that neither the number of rib fractures nor the size of the flail segment accurately predicted the occurrence of subsequent respiratory failure.

The utility of arterial blood analysis in the evaluation of pulmonary contusion should not be underestimated, especially when obtained with the patient breathing room air (18). Most patients with blunt chest trauma demonstrate a progressive decrease in Pao_2 and $Paco_2$, and a concomitant increase in pH_a reflecting respiratory alkalosis. It has been documented that arterial hypoxemia frequently precedes the development of any radiographic abnormalities (19). Interestingly, Pao_2 does not correlate with the extent of lung contusion, as hypoxic pulmonary vasoconstriction within the injured lung tends to limit blood flow to contused areas (20). The Pao_2/Fio_2 ratio may be a sensitive marker of ultimate mortality, but its usefulness has only been supported once the pulmonary contusion has evolved. Kollmorgen et al., found no difference in the initial Pao_2/Fio_2 ratios between survivors and non-survivors of pulmonary contusion, but noted a subsequent difference after the initial resuscitation period (21). A Pao_2/Fio_2 ratio <300 was found to indicate an increased risk of developing acute respiratory failure. Moreover, analysis of the arterial blood is useful in evaluating the patient's response to treatment modalities.

Van Eeden et al. (22) proposed that V/Q scans be obtained to evaluate nonpenetrating chest injury. In comparison with chest radiographs, V/Q studies were able to detect abnormalities much earlier after chest trauma. When performed serially, V/Q studies may also prove useful for guiding therapy. At present, however, the cost and labor intensity of the procedure has precluded its widespread application in clinical practice. Wagner et al. (23) have demonstrated that the opacities seen on plain chest radiographs after pulmonary contusion are frequently caused by hemorrhage into the alveoli from disruption of small blood vessels. They have described a method to quantify the amount of airspace filling on computed tomographic scan so that the extent of lung injury can be compared between patients and their response to treatment can be evaluated.

Management of Flail Chest and Pulmonary Contusion

Before 1950, patients with flail chest were routinely treated by application of external splints (i.e., combinations of adhesive straps and sandbags), operative

fixation, and/or external traction (24). In 1952, Jensen (25) described the use of a breathing apparatus for application of continuous positive airway pressure (CPAP) in blunt chest injury to minimize the flail segment's paradoxical motion. He submerged the exhaust port of a ventilator under 4 cm of water so that the pleural pressure would be maintained above atmospheric pressure throughout the respiratory cycle. In 1956, Avery and Mörch (26) utilized a mechanical ventilator to generate positive pressure, and create "internal pneumatic stabilization" of the flail segment. This heralded an era in which conventional treatment for flail chest and pulmonary contusion routinely included early tracheal intubation or tracheostomy tube placement and application of positive-pressure mechanical ventilation. Unfortunately, the morbidity and mortality associated with blunt chest trauma was not improved with these interventions, and may have actually been increased (27).

In 1975, Trinkle et al. (28) challenged the need for early intubation and conventional mechanical ventilation. They suggested that the use of "selective" management of flail chest injury could decrease overall morbidity and mortality. One group of patients was treated with the conventional protocol previously described, whereas another comparable group of patients received supportive treatment for their pulmonary contusion. In the latter group, the tracheas were intubated and the lungs mechanically ventilated only when the Pao_2 could not be maintained >60 mm Hg breathing room air or > 80 mm Hg breathing supplemental oxygen. All of these patients were treated with IV morphine and intercostal nerve blocks, fluid restriction, colloid therapy, diuretics, and vigorous pulmonary toilet (e.g., intermittent positive pressure breathing and frequent nasotracheal suctioning). Patients receiving conventional treatment with intubation and mechanical ventilation spent more days in the ICU and demonstrated a higher complication and mortality rate than did those receiving only supportive treatment. The higher complication and mortality rates associated with intubation/tracheotomy and mechanical ventilation were attributed to an increased incidence of pneumonia, sepsis, multisystem organ dysfunction, and tracheal injury. Subsequent investigators have supported the use of selective management strategies by confirming lower morbidity and mortality rates, as well as decreased length of stay in the ICU and hospital (29,30). The most identifiable cause of mortality in intubated patients has been tracheobronchitis and pneumonia, with infection rates as high as 60%, and subsequent sepsis and multisystem organ failure. Although some patients with blunt chest injury and multisystem trauma may ultimately require tracheal intubation and mechanical ventilation, the vast majority of victims with isolated pulmonary contusion do not.

CPAP. As soon as flail chest and pulmonary contusion are diagnosed, CPAP therapy should be applied by face mask. An optimal level of CPAP is defined as the airway pressure required to minimize the elastic work of breathing and minimize the pulmonary venous admixture, without causing adverse hemodynamic effects. Application of CPAP should not be delayed for several hours while the pulmonary contusion evolves and gas exchange deteriorates (31). Covelli et al. (32) demonstrated the efficacy of ≥10 cm H_2O CPAP by face mask in 35 patients treated for progressive arterial hypoxemia, with all improving their Pao_2/Fio_2 ratio within the first hour of therapy. Oxygen delivery improved in each patient, although five patients ultimately required tracheal intubation and titration of CPAP to higher levels. They concluded that face mask CPAP was safe and effective for treatment of arterial hypoxemia in spontaneously breathing patients with early progressive respiratory insufficiency.

APRV. In those patients who require tracheal intubation because of impending respiratory failure, airway pressure release ventilation (APRV) is the logical mode of providing partial ventilatory support (33). APRV was first introduced by Downs and Stock in 1985 (34). It was designed to augment alveolar ventilation in those patients who require assistance, despite the reduction in overall respiratory work of breathing with CPAP. During APRV, the change in lung volumes, which augment effective alveolar ventilation, are produced by intermittently releasing the "optimal" CPAP level (e.g., P-high = 20 cm H_2O for 5 sec) briefly down to a lower CPAP level (e.g., P-low = 8 cm H_2O for 1 sec).

Compared with controlled mechanical ventilation, the major advantage of partial ventilatory support with spontaneous breathing is improved V/Q matching at the level of the alveoli. Putensen et al. (35) demonstrated that spontaneous breathing superimposed on mechanical ventilation contributed to improved V/Q matching and increased systemic blood flow. Moreover, the spontaneous contribution to mechanically assisted breaths obtained during pressure support ventilation was not sufficient to counteract the maldistribution seen in V/Q during positive pressure ventilation. Sydow et al. (36) compared APRV with volume-controlled/inverse ratio ventilation (VC-IRV), and found that APRV resulted in lower sedation requirement, more spontaneous breathing, and achieved greater oxygen delivery than VC-IRV. In addition, peak airway pressures were considerably lower during APRV, and decreased further within 24 h. The $AaDO_2/Fio_2$ ratio and venous admixture were also both improved with APRV. They concluded that the progressive recruitment of collapsed alveoli during spontaneous breathing with APRV resulted in more efficient oxygenation and ventilation of patients with moderate to severe acute lung injury than with VC-IRV.

Thereafter, Rathgeber et al. (37) reported on 596 adult patients with normal preoperative pulmonary function who underwent cardiothoracic surgery. They compared three modes of postoperative ventilatory support (i.e., Controlled Mandatory Ventilation (CMV), Intermittent Mandatory Ventilation (IMV), and Biphasic Intermittent Positive Airway Pressure (APRV/BIPAP), to determine their effect on duration of intubation, analgesic/sedative requirements, and pulmonary gas exchange. Patients receiving APRV required significantly shorter periods of intubation than did those in either the CMV or IMV groups. Those individuals breathing with APRV required lower doses of post-operative sedation less frequently, as well as lower cumulative doses of opioids, than did patients receiving CMV or IMV. As a direct consequence, $Paco_2$ levels were lower in patients breathing with APRV, and the duration of mechanical ventilation shorter than in those on CMV or IMV. They concluded that APRV is a convincing alternative to both CMV and IMV, and the ability of APRV to facilitate spontaneous breathing a distinct advantage.

Supplemental Oxygen. Oxygen administration is frequently utilized in patients with flail chest and pulmonary contusion, and its effects incorrectly assumed to be benign. To obtain maximal information from the initial oxyhemoglobin saturation with pulse oximetry or arterial blood analysis, data must be obtained with the patient breathing room air. Obviously, if the patient is in extremis, then supplemental oxygen should be administered to temporize the situation while a definitive diagnosis is made. More commonly, however, the routine administration of supplemental oxygen merely masks the considerable V/Q mismatch within the lung. This tends to delay definitive therapeutic intervention (e.g., application of CPAP) that can effectively reverse the primary pathology. After the initial evaluation, supplemental oxygen may be administered by nasal cannula, or face mask, but at as low an Fio_2 as feasible to maintain an oxyhemoglobin saturation >90%. Such a low level of Fio_2 is desirable to avoid the detrimental effects of oxygen which include 1) absorption atelectasis, 2) interference with hypoxic pulmonary vasoconstriction in injured lung areas, 3) formation of free radical species, 4) interference with normal mucociliary clearance, and 5) detrimental effects on the ability of type II alveolar pneumocytes to produce surfactant.

Pulmonary Hygiene/Monitoring. Vigorous pulmonary hygiene (e.g., chest physiotherapy, incentive spirometry, and nasotracheal suctioning of secretions) is thought to facilitate recovery, but conclusive evidence remains elusive. Nevertheless, much of the morbidity associated with pulmonary contusion can be attributed to ineffective pulmonary hygiene and inadequate monitoring of the patient's deteriorating pulmonary status. During the first 48 h after injury, the patient's respiratory status tends to be vulnerable to the progressive nature of the pulmonary contusion and therefore mandates vigilant monitoring. Continuous pulse oximetry is required as well as regular documentation of the patient's respiratory rate and inspiratory effort. Intermittent arterial blood samples should be obtained to monitor acid/base status (i.e., pH_a and base deficit/excess), Pao_2, and $Paco_2$, but do not relieve the physician and nursing staff of their obligation to observe the patient closely. Selective management of traumatized patients does not necessarily require ICU admission, but rather careful in-hospital observation and follow-up. Fiberoptic bronchoscopy may be utilized to clear thick secretions/mucous plugs from the airways and to obtain quantitative bronchoalveolar lavage and protected-brush biopsy specimens.

Fluid Management. Numerous animal studies have investigated the effects of fluid administration on the evolution of pulmonary contusion. Trinkle et al. (38) demonstrated that administration of crystalloid solutions increased the size of lung contusion and lung weight. Both the extent of contusion and lung weight increased when the IV infusion rate was accelerated. Fulton et al. (39) found that the administration of large quantities of crystalloid increased areas of congestion and atelectasis in the noncontused lung. They attributed this finding to an increase in capillary hydrostatic pressure despite normal left atrial pressures and left ventricular function. Earlier, Fulton and Peter (40) had reported an initial increase in pulmonary blood flow, and decrease in PVR after IV fluid administration. They also demonstrated a close relationship between shunt fraction (Qs/Qt) and pulmonary artery pressure (PAP), and their data imply that pulmonary artery (PA) catheterization would be useful during the initial phases of fluid resuscitation. Richardson et al. (41) observed a decrease in Pao_2 with evolving pulmonary contusion when the rate of crystalloid infusion was increased. They also found an increase in contusion size and lung weight, as well as the extent of pulmonary edema within the injured lung after the crystalloid was administered. Kollmorgen et al. (22) observed a correlation between mortality and the total volume of IV fluid used during resuscitation. They were, however, unable to determine whether excessive volume resuscitation exacerbated underlying lung injury or whether patients with more severe injuries required greater volume resuscitation. Tranbaugh et al. (42) found that crystalloid resuscitation decreased plasma oncotic pressure by hemodilution but did not increase the amount of extravascular lung water or pulmonary edema formation. Bongard and Lewis (43) did not observe any relationship between plasma oncotic pressure and Pao_2/Fio_2 ratio in patients with flail chest and pulmonary contusion. After

an initial 10 days of treatment, however, the Pao_2/Fio_2 ratio was increased only in those patients who ultimately survived.

Although the administration of IV crystalloid solutions alters the histology of the lung, compelling evidence in favor of colloid administration in humans is still lacking (44). Current evidence suggests that either crystalloid or colloid solutions may be safely administered as long as they are given at a rate sufficient to achieve adequate mean arterial blood pressure and vital organ perfusion. Indeed, the quantity of volume resuscitation, rather than the type of IV fluids, is a more important determinant in ultimate outcome. Therefore, the net input and output of IV fluids must be closely monitored and the insertion of a PA catheter strongly considered for measurement of left-sided (pulmonary artery occlusion pressure) cardiac filling pressures to avoid excessive fluid administration. Although placement of a CVP monitor may at first seem to be an acceptable alternative, the effects of positive pressure ventilation on PVR and right-sided filling pressures makes accurate determination of data unreliable. Moreover, the CVP does not allow the clinician to determine the intrapulmonary shunt fraction or follow the ventilation-perfusion index, as can be achieved with the PA catheter. The routine administration of diuretics should be discouraged unless their use is guided by data obtained from the PA catheter. Unnecessary diuretic therapy increases the cost of therapy, and may result in adverse consequences such as electrolyte abnormalities, cardiac dysrhythmias, and hypovolemia.

Corticosteroid Administration. In the past, the use of steroids in the treatment of pulmonary contusion was advocated. Franz et al. (45) demonstrated that the administration of methylprednisolone 30 min after injury decreased the size of experimental pulmonary contusion and the weight of the injured lung. Unfortunately, the cytoprotective effect on lung architecture did not result in any improvement on arterial blood analysis. Pulmonary infection is among the most dreaded of complications in patients with pulmonary contusion, especially those requiring mechanical ventilation. Moreover, pneumonia and sepsis are among the most frequent causes of death. Richardson et al. (46) found that steroid treatment impaired the clearance of bacteria in dogs who received 30 mg/kg methylprednisolone. Bacterial clearance was depressed in both contused and noncontused lungs. Lucas et al. (47) observed no improvement in pulmonary function in 114 injured patients after steroid administration, when compared with a group not treated with steroids. Those patients who did receive steroids demonstrated a decrease in Pao_2, but increases in CVP, intrapulmonary shunt fraction, number of days on a ventilator, and perhaps most importantly, the death rate. They concluded that steroids neither prevented nor ameliorated pulmonary dysfunction after blunt chest injury. Thus, present evidence does not justify the routine administration of steroids to patients with pulmonary contusion, and their use may actually worsen outcome.

Epidural Analgesia. Effective pain control strategies are essential to facilitate adequate coughing and tracheobronchial hygiene, increase spontaneous tidal volume, and decrease splinting of the chest wall. Conventional pain control for multiple rib fractures has consisted of IV/IV patient-controlled analgesia narcotics, and/or intercostal nerve blocks with local anesthetic agents. Parenteral opioids rarely provide complete analgesia, or relieve associated muscle spasm. Moreover, in high doses they may adversely affect the patient's ventilatory drive and result in profound respiratory acidosis. Intercostal nerve blocks may be effective, but usually have a shorter duration of action. They require repeated placement and expose the patient to the risk of hemo- or pneumothorax with each application.

The use of continuous epidural analgesia has been shown to circumvent many of these problems, and to offer effective analgesia after blunt chest trauma. Dittman et al. (48) investigated the effects of epidural bupivacaine on pulmonary function and gas exchange. They reported an increase in dynamic lung compliance, functional residual capacity, Pao_2, spontaneous tidal volume, and vital capacity within 30 min of achieving adequate epidural analgesia. Pao_2 and vital capacity continued to increase throughout the first day of epidural administration. In addition, there was a decrease in airway resistance, paradoxical chest wall movement, and an improvement in the patient's ability to cough.

Mackersie et al. (49) reported on the efficacy of continuous epidural fentanyl administration (CEFA) as an alternative to the use of local anesthetics. Theoretical advantages of fentanyl given via the epidural space include a lower incidence of sympathectomy and hypotension, avoidance of motor blockade, and less tachyphylaxis. They found that 95% of patients obtained adequate analgesia with CEFA, and did not require additional administration of IV opioids. Maximal inspiratory pressure and vital capacity improved after successful CEFA, suggesting normalization of respiratory mechanics. Pruritus was the most commonly seen side effect, whereas urinary retention, nausea/vomiting, and backache occurred less frequently. Another important finding was the absence of any appreciable changes in pre- and postepidural fentanyl $Paco_2$ levels, suggesting a low risk of respiratory depression. This is an advantage of fentanyl compared with longer-acting opioids, which may predispose the patient to delayed respiratory depression.

Bollinger and Van Eeden (50) studied the treatment of multiple rib fractures in nonpenetrating chest

injury. They randomly allocated patients to one of two treatment protocols: a CPAP mask combined with epidural analgesia or tracheal intubation and conventional mechanical ventilation with positive end-expiratory pressure. They observed that patients treated with CPAP and adequate epidural analgesia experienced a lower infection/pneumonia rate and required fewer days in the ICU and the hospital.

Summary

In an editorial written in 1978, Downs (51) observed that "the clinician must recognize the manifestations of a variety of forms of chest injury, and must apply the appropriate therapy based on a knowledge of the involved pathophysiological conditions." Over 20 yr later, this remains prudent advice to the anesthesiologist responsible for the initial evaluation and management of trauma patients with flail chest and pulmonary contusion. Multiple rib fractures may precipitate a paradoxical breathing pattern and increase work of breathing, but are rarely the primary cause of acute respiratory failure. Instead, the severity of the underlying lung injury should determine the appropriate therapeutic approach. If the lung is not injured, or only mildly contused, then little therapy is necessary except for adequate pain management of the rib fractures. In contrast, if the lung is severely contused, scientific evidence supports a selective approach to management with avoidance of tracheal intubation and mechanical ventilation, whenever possible.

Because deleterious effects on gas exchange and pulmonary function occur within minutes of injury, the early application of CPAP with a face mask should be instituted. CPAP ameliorates the V/Q mismatch associated with pulmonary contusion, enhances functional residual capacity, and increases compliance of the injured lung. CPAP minimizes the extent of the paradoxical movement of the flail segment, and therefore improves the respiratory mechanics of the disrupted thorax. CPAP should be applied continuously throughout the patient's respiratory cycle to facilitate the patient's comfort and enhance the efficiency of gas exchange and spontaneous ventilation. Routinely, CPAP allows the patient to achieve hemoglobin saturations >90%, while receiving the lowest possible level of supplemental oxygen (i.e., Fio_2 <0.30). These cumulative effects frequently make tracheal intubation and mechanical ventilation unnecessary, thereby minimizing the incidence of pulmonary infection as well as any associated morbidity and mortality.

In those patients requiring ventilatory support, APRV has been shown to be equal or superior to standard modes (e.g., CMV or IMV). Patients breathing with APRV require shorter periods of intubation, less frequent sedation, and lower cumulative doses of both opioids and sedative-hypnotics. As a consequence, $Paco_2$ tends to be lower with APRV, and the duration of mechanical ventilation shorter than with CMV or IMV. APRV allows delivery of an optimal level of CPAP, yet facilitates gas exchange and the removal of CO_2. Because APRV facilitates spontaneous breathing throughout the entire respiratory cycle, it can maintain systemic oxygen delivery without any impairment in the patient's cardiovascular performance.

In addition to the early application of face mask CPAP or APRV, judicious fluid management is necessary. Actual blood loss must be replaced with an equivalent amount of autologous blood to maintain acceptable oxygen delivery and assure the function of vital organs. There is no overwhelming evidence that either crystalloid or colloid solutions are superior, however, crystalloids do appear to be more cost-effective. Thus, crystalloids should be favored during the initial phase of resuscitation. More important than the type of IV fluid is the amount of fluid administered. The rate of administration must be guided by data derived from invasive hemodynamic monitoring (i.e., A-line and PA catheter), rather than by any arbitrary or universal guidelines. If euvolemia and adequate end-organ perfusion are to be maintained, then a PA catheter is essential for measurement of pulmonary artery occlusion pressure and calculation of PVR. Furthermore, the use of a PA catheter is helpful in the ventilatory management of the patient with pulmonary contusion, as calculation of oxygen delivery and intrapulmonary shunt fraction allow determination of the optimal CPAP level. A CVP catheter may provide erroneous information, particularly in patients receiving positive pressure ventilation. Misinterpretation of CVP data may therefore hinder, rather than aid, the anesthesiologist in guiding appropriate fluid and ventilatory therapy.

Diuretics should not be administered to achieve a net negative fluid balance without a PA catheter in place because severe electrolyte abnormalities and unanticipated hypovolemia may result. At present, steroids should not be administered to patients with pulmonary contusion because the increased risk of infection outweighs any theoretical advantage of decreased interstitial edema. Finally, the patient's pain must be alleviated early in the course of their hospitalization and an evaluation must be performed for any contraindications to regional analgesia. An epidural catheter should be placed in all eligible candidates, and conventional therapy (e.g., IV/IV patient-controlled analgesia with opioids) utilized only when patients are not candidates for, or refuse, regional anesthesia. Thus, through a combination of proper diagnosis and timely intervention with selective management strategies, a reduction can be achieved in the morbidity and mortality trauma victims with flail chest injury and pulmonary contusion.

References

1. Clark GC, Schecter WP, Trunkey DD. Variables affecting outcome in blunt chest trauma: flail chest vs. pulmonary contusion. J Trauma 1988;28:298–304.
2. DeMuth WE, Smith JM. Pulmonary contusion. Am J Surg 1965; 109:819–23.
3. Poole GE, Myers RT. Morbidity and mortality rates in major blunt trauma to the upper chest. Ann Surg 1981;193:70–5.
4. LoCicero J, Mattox KL. Epidemiology of chest trauma. Surg Clin North Am 1989;69:15–9.
5. Jensen NK. Recovery of pulmonary function after crushing injuries of the chest. Chest 1952;22:319–46.
6. Moore BP. Operative stabilization of nonpenetrating chest injuries. J Thorac Cardiovasc Surg 1975;70:619–30.
7. Maloney JV, Schmutzer KJ, Raschke E. Paradoxical respiration and 'pendelluft'. J Thorac Cardiovasc Surg 1961;41:291–8.
8. Duff JH, Goldstein M, McLean APH, et al. Flail chest: a clinical review and physiological study. J Trauma 1968;8:63–74.
9. Fulton RL, Peter ET, Wilson JN. The pathophysiology and treatment of pulmonary contusions. J Trauma 1970;10:719–30.
10. Grazon AA, Gourin A, Seltzer B, et al. Severe blunt chest trauma: studies of pulmonary mechanics and blood gases. Ann Thorac Surg 1966;2:629–39.
11. Grazon AA, Seltzer B, Karlson KE. Physiotherapy of crushed chest injuries. Ann Surg 1968;168:128–36.
12. Fulton RL, Peter ET. The progressive nature of pulmonary contusion. Surgery 1970;67:499–506.
13. Stellin G. Survival in trauma victims with pulmonary contusion. Am Surg 1991;57:780–4.
14. Baker SP, O'Neill, Haddon W, Long WB. The injury severity score: a method for describing patients with multiple injuries and evaluating emergency care. J Trauma 1974;14:187–96.
15. Landercasper J, Cogbill TH, Strutt PJ. Delayed diagnosis of flail chest. Crit Care Med 1990;57:780–4.
16. Erickson U, Johansson L, Nylen O, Saldeen T. Radiographic findings in pulmonary insufficiency following non-penetrating trauma of the chest. Scand J Thorac Cardiovasc Surg 1972;6: 278–86.
17. Erickson DR, Shinozaki T, Beekman E, Davis JH. Relationship of arterial blood gas and pulmonary radiographs to the degree of pulmonary damage in experimental pulmonary contusion. J Trauma 1971;11:689–93.
18. Wise AJ, Topuzlu C, Mills EL, Page HG. The importance of serial blood gas determinations in blunt chest trauma. J Thorac Cardiovasc Surg 1968;56:520–4.
19. Okies JE, Pricher FJ, Guinn GA. Pulmonary alterations in the acutely injured patient. Ann Thorac Surg 1972;13:420–6.
20. Wagner RB, Sliviko B, Jamieson PM, et al. Effect of lung contusion on pulmonary hemodynamics. Ann Thorac Surg 1991;52: 51–58.
21. Kollmorgen DR, Murray KA, Sullivan JJ, et al. Predictors of mortality in pulmonary contusion. Am J Surg 1994;168:659–64.
22. Van Eeden SF, Klopper JF, Alheit B, Bardin PG. Ventilation-perfusion imaging in evaluating regional lung function in non-penetrating injury to the chest. Chest 1989;95:632–38.
23. Wagner RB, Crawford WO, Schimpf PP, et al. Quantitation and pattern of parenchymal lung injury in blunt chest trauma: diagnostic and therapeutic implications. J Comput Tomogr 1988; 12:270–81.
24. Kinsella TJ, Bronson RG. Skeletal traction in the treatment of traumatic anterior thoracic instability. Minn Med 1949;32: 486–500.
25. Jensen NK. Recovery of pulmonary function after crushing injuries of the chest. Chest 1952;22:319–46.
26. Avery EA, Mörch ET, Benson DW. Critically crushed chests; a new method of treatment with continuous mechanical hyperventilation to produce alkalotic apnea and intenal pneumatic stabilization. J Thorac Surg 1956;32:291–309.
27. Schaal MA, Fischer RP, Perry JF. The unchanged mortality of flail chest injuries. J Trauma 1979;19:492–6.
28. Trinkle JK, Richardson JD, Franz JL, et al. Management of flail chest without mechanical ventilation. Ann Thorac Surg 1975;19: 355–63.
29. Shackford SR, Virgilio RW, Peters RM. Selective use of ventilator therapy in flail chest injury. J Thorac Cardiovasc Surg 1981; 81:194–201.
30. Richardson JD, Adams L, Flint L. Selective management of flail chest and pulmonary contusion. Ann Surg 1982;196:481–7.
31. Kirby RR, Downs JB, Civetta IM, et al. High level positive end-expiratory pressure (PEEP) in acute respiratory insufficiency. Chest 1975;76:156–63.
32. Covelli HD, Weled BJ, Beekman JF. Efficacy of CPAP administered by face mask. Chest 1982;81:147–50.
33. Downs JB, Stock MC. APRV: a new concept in ventilatory support. Crit Care Med 1987;15:459–61.
34. Stock MC, Downs JB, Frolicher DA. Airway pressure release ventilation. Crit Care Med 1987;15:462–6.
35. Putensen C, Räsänen J, Lopez FA, Downs JB. Effect of interfacing between spontaneous breathing and mechanical cycles on the ventilation-perfusion distribution in canine lung injury. Anesthesiology 1994;81:921–30.
36. Sydow M, Burchardi H, Ephraim E, et al. Long-term effects of two different ventilatory modes on oxygenation in acute lung injury. Am J Respir Crit Care Med 1994;149:1550–6.
37. Rathgeber J, Schorn B, Falk V, et al. The influence of controlled mandatory ventilation (CMV), intermittent mandatory ventilation (IMV) and biphasic intermittent positive airway pressure (BIPAP) on duration of intubation and consumption of analgesics and sedatives. Eur J Anaesthesiol 1997;14:576–82.
38. Trinkle JK, Furman RW, Hinshae MA, et al. Pulmonary contusion; pathogenesis and effect of various resuscitative measures. Ann Thorac Surg 1973;16:568–73.
39. Fulton RL, Peter ET. Compositional and histologic effects of fluid therapy following pulmonary contusion. J Trauma 1974; 14:783–90.
40. Fulton RL, Peter ET. Physiologic effects of fluid therapy after pulmonary contusion. Am J Surg 1973;126:773–7.
41. Richardson JD, Franz JL, Grover FL, Trinkle JK. Pulmonary contusion and hemorrhage; crystalloid versus colloid replacement. J Surg Res 1974;16:330–6.
42. Tranbaugh RF, Eblings VB, Christensen J, Lewis FR. Determinants of pulmonary interstitial fluid accumulation after trauma. J Trauma 1982;22:820–6.
43. Bongard FS, Lewis FR. Crystalloid resuscitation of patients with pulmonary contusion. Am J Surg 1984;148:145–51.
44. Virgilio RW, Rice CL, Smith DE, et al. Crystalloid vs. colloid resuscitation: is one better? A randomized clinical study. Surgery 1979;85:129–39.
45. Franz JL, Richardson JD, Grover FL, Trinkle JK. Effect of methylprednisolone sodium succinate on experimental pulmonary contusion. J Thorac Cardiovasc Surg 1974;68:842–4.
46. Richardson JD, Woods D, Johanson WG, Trinkle JK. Lung bacterial clearance following pulmonary contusion. Surgery 1979; 86:730–5.
47. Lucas CE, Ledgerwood AM. Pulmonary response of massive steroids in seriously injured patients. Ann Surg 198;194:256–61.
48. Dittman M, Keller R, Wolff G. A rationale for epidural analgesia in the treatment of multiple rib fractures. Int Care Med 1978;4: 193–7.
49. Mackersie RC, Shackford SR, Hoyt DB, Karagianes TG. Continuous epidural fentanyl analgesia; ventilatory function improvement with routine use in treatment of blunt chest injury. J Trauma 1987;27:1207–12.
50. Bollinger CT, Van Eeden SF. Treatment of multiple rib fractures; randomized controlled trial comparing ventilatory with non-ventilatory management. Chest 1990;97:943–8.
51. Downs JB. Flail chest syndrome and pulmonary contusion. Arch Surg 1978;113:903.