Barotrauma*
Pathophysiology, Risk Factors, and Prevention

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Since the time of Macklin and Macklin's1 early investigation of malignant interstitial emphysema in spontaneously breathing patients, the setting in which barotrauma occurs has changed. With the advent of mechanical ventilation in respiratory support, barotrauma has become a well recognized and feared complication. Barotrauma as an immediate cause of death may range as high as 13 to 35 percent.2,4 In one series where the diagnosis was delayed, 31 percent died.4

It becomes evident that knowledge of the risk factors that predispose to barotrauma and vigilant anticipation of its occurrence may significantly affect the mortality rate. In addition, the ability to continuously adapt ventilatory management to minimize the risk of barotrauma becomes a necessary ingredient in optimal management of the patient in respiratory failure.

We will examine some of the early work which has clarified the pathophysiology of barotrauma. In addition, the clinical studies will be reviewed where barotrauma is associated with mechanical ventilation, attempting to elucidate the risk factors that predispose the patient to this problem. Finally, we will examine the clinical presentation and further investigate how special techniques and different modes of ventilation can be used in the management of the high risk patient.

Barotrauma is an all inclusive term which may more subtly manifest as pulmonary interstitial emphysema (PIE), pneumomediatinum, pneumoperitoneum, subcutaneous emphysema, or sometimes as a life threatening emergency when pneumothorax or tension pneumothorax develop. Macklin and Macklin1 performed many of the early investigations on the pathophysiology of barotrauma prior to the use of mechanical ventilation.

They were intrigued by the case reports of patients who died of respiratory failure with stiff hyperexpanded lungs, which at autopsy could not be collapsed by pressure and floated when placed in water. Although grossly resembling "bronchitic emphysema," it was later discovered that this condition was secondary to ruptured alveoli and dissection of air along the vascular sheaths passing to the mediastinum and ultimately to subcutaneous tissue and retroperitoneum.5 Death was caused immediately by pneumothorax, as air ruptured through the mediastinal wall, or more insidiously by extrinsic compression of the vascular sheath and interstitium.

Using special techniques, Macklin and Macklin were able to determine that the site of disruption occurred at the common border of the alveolar base and the vascular sheath.

Using a canine model, Polach and Adams6 ruled out the influence of high airway pressure, by itself, as the cause of alveolar rupture. Air was insufflated into the lungs in large volumes, but expansion of the lungs was restricted by a tight thoracic cage bandage. Despite marked elevation in intraalveolar pressure, the gradient between the inside and outside of the alveolus was attenuated by a matched elevation in the supporting pressure on the outside of the chest. Under these circumstances, alveolar overinflation was avoided, and the alveoli did not rupture.

Macklin and Macklin1 surmised that a gradient between the alveolus and the vascular sheath was necessary to produce alveolar rupture and interstitial emphysema. They theorized this gradient can be produced by either (1) alveolar overinflation without a concomitant expansion of the vascular lumen, or (2) reduction in caliber of pulmonary vessels as occurs in states of decreased pulmonary blood flow, i.e., pulmonary embolus. Thus, barotrauma is more likely to occur in those clinical situations which accentuate this gradient.

Caldwell et al7 performed a study in rabbits years later in an attempt to verify the significance of an alveolar-arterial (A-a) pressure gradient in the genesis of perivascular interstitial emphysema. Tracheal, pleural, and pulmonary artery pressures were mea-

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sured, and an A-a pressure gradient was calculated in rabbits subjected to increasing increments of airway pressure. Caldwell et al. confirmed a direct correlation between alveolar distention under high pressure and the incidence of PIE. No relationship, however, was found between the A-a gradient and the development of PIE. The authors argued that the gradient of significance was between the alveolus and the sheath which was not measured. Thus, the widened gradient concept could not be refuted in this study.

Macklin and Macklin also hypothesized, without good experimental evidence at the time, that the alveolar perivascular sheath gradient could be widened by diminished pulmonary blood flow. If this is true, then alveoli at increased volumes and pressure may rupture more easily during periods of low pulmonary blood flow. This idea was examined by Lenaghan et al. in canines. Ten animals were phlebotomized and made hypotensive for an hour before being challenged with increasing increments of airway pressure. Seven of these dogs developed hemorrhagic atelectasis after one hour. The mean tracheal pressure at lung rupture in these dogs was 25.1 mm Hg. Three dogs were hypovolemic and yet were macroscopically unaffected by the degree and duration of the hypotension. Tracheal pressure at lung rupture for these three dogs was 46.4 mm Hg, which was not significantly different from that found in normovolemic, normotensive dogs used in another part of the study. The contribution of low pulmonary blood flow to the risk of barotrauma therefore remains only speculative.

FACTORS PREDISPOSING TO BAROTRAUMA

With the advent of mechanical ventilation, the clinical consequences of barotrauma from positive pressure ventilation became even more important. Contrary to the relatively slow accumulation of interstitial emphysema in spontaneously breathing patients described by Macklin and Macklin, tension pneumothorax, occurring in 30 to 97 percent of all pneumothoraces, may cause a rapid demise in the patient if not addressed immediately. Identifying the high risk population, therefore, becomes clinically imperative.

INFLUENCE OF AIRWAY PRESSURE

Peak airway pressure (PAP) is most frequently cited as the main risk factor which contributes to barotrauma. Although authors disagree about the level of the safe upper limit, generally the higher the level, the greater the probability that barotrauma will occur. Most recently, Petersen and Baer reported a 43 percent incidence of barotrauma, as defined by pneumomediastinum, subcutaneous emphysema, or pneumothorax, in patients who required a PAP above 70 cm H2O. The incidence of barotrauma in patients who required a PAP between 50 to 70 cm H2O was 8 percent. No barotrauma occurred with a PAP less than 50 cm H2O. Woodring examined chest x-ray films of 15 patients with ARDS and found PIE in 13 of 15 (88 percent), ten of whom also had pneumothorax. Twelve of 13 cases of barotrauma occurred at or above a PAP of 40 cm H2O. Pneumothorax occurred at an average PAP of 62 cm H2O. Other reports of clinically safe airway pressures vary from 25 to 50 cm H2O. Therefore, at this point in time, no specific PAP guarantees the absence of risk from barotrauma. It is reasonable, however, to focus our efforts on minimizing the PAP as much as possible.

The mean airway pressure (\(P_{\text{aw}}\)) has been shown to be reduced with the use of intermittent mandatory ventilation (IMV). No relationship has been found, however, between \(P_{\text{aw}}\) and the incidence of barotrauma. On the other hand, in a retrospective study by Mathru and Venus, despite both a higher peak and end-expiratory airway pressure, patients ventilated with IMV-CPAP exhibited an incidence of pulmonary barotrauma (PBT) of only 7 percent compared to 22 percent PBT found in patients using the CMV mode. The authors surmised that fewer mechanical breaths from the ventilator plus less asynchronous breathing using IMV were the major determinants differentiating the groups resulting in less barotrauma with IMV.

In the last 15 years since high frequency jet ventilation (HFJV) has been introduced, it has offered at least theoretic advantages over conventional ventilation. Most are related to the fact that peak airway pressures are reduced with HFJV allowing safe use of this method in patients after lung resection or in patients with bronchopulmonary fistulas. Theoretic advantage is also ascribed to HFJV for its ability to reduce pulmonary barotrauma. However, in a prospective randomized trial comparing IMV with HFJV, despite higher PAP with IMV, the incidence of pulmonary barotrauma was not significantly different between the two groups.

It is difficult at the present time to advocate a particular mode of conventional ventilation. Although present data seem to support the use of IMV in those patients at risk for barotrauma, a prospective trial comparing controlled and/or assisted ventilation with IMV is needed. In those patients requiring extremely high PAP who are at significant risk for barotrauma using conventional ventilation, the use of HFJV is certainly a reasonable alternative if adequate alveolar ventilation and oxygenation can be accomplished.

POSITIVE END-EXPIRATORY PRESSURE

Controversy also surrounds the use of positive end-expiratory pressure (PEEP) and its association with barotrauma. Some early reports, and more recent studies, support the view that the use of PEEP is associated with an increased risk of barotrauma. Oth-
ers, however, have found no such relationship.\textsuperscript{6,4,9,12,31} Petty and Fowler\textsuperscript{22} argue that maximal therapeutic benefits are typically achieved at PEEP levels of 5 to 15 cm H\textsubscript{2}O and that higher levels are risky. Kirby et al.,\textsuperscript{23} however, have countered that if less than 15 cm of PEEP had been used in their series of patients with ARDS, over one half of the survivors would have died, as most showed no improvement in ventilatory status until 20 cm PEEP was used. As suggested by others,\textsuperscript{24} a more reasonable approach may be to titrate the PEEP to a level which permits reduction of FIO\textsubscript{2} to nontoxic levels.

**Influence of Disease State on Development of Barotrauma**

Reports have also featured the risks of barotrauma associated with both necrotizing pneumonia and aspiration of gastric contents. Fleming and Bowen\textsuperscript{25} examined the complications of long-term respiratory support in 125 patients in an army evacuation hospital during the Vietnam War. Bronchopleural leaks occurred in 19 of 128 patients. Seventeen of 19 patients had necrotizing pneumonia due to Staphylococcus, Pseudomonas, or both. The median peak airway pressure at the time of air leak in 17 of 19 patients was only 30 cm H\textsubscript{2}O arguing against PAP as the source of the barotrauma.

DeLatorre et al.,\textsuperscript{26} found only a 4 percent incidence of barotrauma in his prospective evaluation of ventilator patients. However, six of 16 patients with documented aspiration pneumonia developed barotrauma. Gastric acid aspiration has been found to cause frank necrosis extending from the tracheobronchial tree to the periphery of the lungs.\textsuperscript{27}

Finally, there appears to be a high incidence of barotrauma in patients with pre-existing chronic lung disease ranging from 13 to 83 percent\textsuperscript{28,4,8} in different series. These patients, who typically have little pulmonary reserve, may require mechanical ventilation after only a low level pulmonary insult. If the lungs are only segmentally insulted, positive pressure volumes from the ventilator will be distributed to the more compliant areas of those chronically damaged lungs, causing overdistention, and predisposing to barotrauma.

**Making the Diagnosis**

Pneumothorax may present abruptly and manifest as acute respiratory distress. Steir et al.,\textsuperscript{29} report that in 45 of 74 cases, the diagnosis of pneumothorax was made on a clinical basis. A systolic blood pressure less than 90, hyperresonance, diminished breath sounds, and tachycardia were seen in 81 to 94 percent, respectively. The mortality rate was only 7 percent of the 45 cases where the diagnosis was made on clinical basis. In 29 patients where the diagnosis was delayed between 30 minutes and eight hours, 31 percent of these patients died of pneumothorax. In other series where barotrauma was a complication of mechanical ventilation, mortality rates changed from 58 to 77 percent.\textsuperscript{3,9,10,21,31}

It is apparent that anticipation and early recognition of the complication is imperative. One of the early clinical harbingers of life-threatening barotrauma may be pulmonary interstitial emphysema (PIE). This may manifest as parenchymal air cysts, linear air streaking toward the hilum, perivascular air halos, pneumatoceles, or subpleural air. In Woodring's\textsuperscript{32} report of PIE and pneumothorax in severe ARDS, he found PIE appeared within 12 hours prior to pneumothorax in five of ten cases.

In a series of 82 patients with severe respiratory failure, Johnson and Altman\textsuperscript{33} retrospectively identified 17 patients with PIE on chest x-ray film. Nine progressed to mediastinal emphysema, cervical, and subcutaneous emphysema. Eight of these developed tension pneumothorax.

Finding subpleural air cysts on chest x-ray film is a particularly ominous development.\textsuperscript{34} These cysts appear typically at the lung bases but may develop anywhere along the visceral pleural. They are rapidly expanding with continued positive pressure ventilation and have been reported as large as 9 cm. Rolfling et al.,\textsuperscript{35} identified 38 patients with extraalveolar air on chest x-ray film during mechanical ventilation. Five of five patients who demonstrated subpleural air cysts on chest x-ray film developed an ipsilateral tension pneumothorax. Other studies have confirmed the high incidence of this complication when subpleural air cysts have been identified.\textsuperscript{10,37}

Recognition of the early signs of barotrauma requires a high index of suspicion. The PIE may be the initial presentation of barotrauma. This is more easily recognized in opacified lungs when alveolar edema is contrasted with air.\textsuperscript{9} In addition, alerting the reviewing radiologist to the possible presence of PIE in the high risk patients may pay dividends if PIE can be found with a magnified examination of the chest x-ray film.

Finally, subcutaneous emphysema is temporally related to the development of PIE. In one study of 74 patients who developed pneumothorax from continuous mechanical ventilatory support, all 74 also demonstrated subcutaneous emphysema.\textsuperscript{8} It requires only seconds to palpate the cervical, supraclavicular, and anterior chest cage regions. The benefits may be lifesaving.

**Prevention and Treatment**

As in the case of many disease entities, the best treatment for this problem is prevention and early recognition. Once the high risk patient has been identified, it is incumbent on the managing physician to focus efforts on minimizing the risk factors that predispose to barotrauma.

High peak airway pressure is most frequently cited
as the major determinant of risk for barotrauma. High peak airway pressures are usually associated with increased levels of PEEP in patients with ARDS who have noncompliant lungs. One of the easiest ways to decrease PAP is to reduce the tidal volume. Steir et al reported that although by standard protocol tidal volumes of 10 ml/kg were used initially in mechanically ventilated patients, in 62 of 74 patients in their series with pneumothorax, the mean tidal was later calculated to be 18 ml/kg. Some recommend a tidal volume between 5 and 10 ml/kg can be used to minimize the PAP in those patients with ARDS and high levels of PEEP. The optimal tidal volume can also be obtained by construction of a compliance curve.

Often overlooked is the fact that PAP can also be decreased by a reduction in the peak flow which may have been set at an unnecessarily high level and can be lowered without compromising an adequate I:E ratio.

Many patients produce high airway pressure when they “fight the ventilator” or breathe dysynchrnoously with the ventilator. Frequently, this occurs right after intubation and most adapt within a short time afterwards. However, some patients, driven by anxiety or irritable stretch receptors in the lungs, fail to breathe synchronously especially patients receiving asynchronous IMV. Although a canine study failed to demonstrate increased frequency of barotrauma in the asynchronous compared to synchronous mode of IMV, these specimens were not tachypneic, and therefore, were less likely to stack ventilator breaths on spontaneous tidal volumes.

Several other options are available in this type of patient. Assist control (AC) not only reduces the risk of breath stacking, but can also decrease the work of breathing. Ventilatory requirements for most patients will be satisfied by the added ventilatory supplementation assist control provides, and when IMV is changed to assist control, the respiratory rate typically decreases accordingly. Although early reports promoted IMV over controlled mandatory ventilation (CMV) because it avoided respiratory alkalosis, recently, two different studies have failed to demonstrate a clinically significant difference in pH when patients are switched from IMV to AC. Results of these studies performed in stable patients may not be extrapolated to the response seen in newly intubated patients or patients who are driven to breathe by irritable stretch receptors in the lungs. Unabated tachypnea in these patients ventilated with assist control may promote hemodynamic instability and hypocapnia.

In patients with ARDS, because of noncompliant lungs, much greater spontaneous transpulmonary pressures must be generated to move even small tidal volumes. The work of breathing may be prohibitive in the severely compromised patient who has little cardiopulmonary reserve. Therefore, using even synchronized IMV in these patients, although decreasing the risk of barotrauma, may unfavorably affect an already tenuous oxygen supply:demand ratio. Assist control may also be less than optimal. These patients are frequently anxious and tachypneic despite a normal or low PaCO₂. High PAP and increased frequency of mechanically delivered tidal volumes may also place these patients at increased risk for barotrauma.

The most prudent treatment for this type of patient may be paralysis with neuromuscular blocking agents and heavy sedation. Not only will the work of breathing be eliminated, but the PAP should also be decreased when respiratory muscles are relaxed and asynchronous breathing is avoided. Although paralysis may alleviate some major problems with the ventilator patient, it may, by itself, create a new set of difficulties including disconnection from the ventilator, increased risk for pulmonary emboli, and inadequate sedation.

Another group of patients at special risk for barotrauma are those with status asthmaticus. Previous studies have reported mortality as a direct consequence of pneumothorax between 9 and 35 percent. High airway pressures are typically required to overcome severe bronchial obstruction. Because of a variability of obstruction in the different airways, there is a maldistribution of mechanical tidal volumes. This promotes gas trapping and nonuniform alveolar distention. It is understandable, therefore, why these people are at risk of barotrauma.

A new approach promoted separately by Darioli and Perret and Menitove and Goldring is to hypoventilate the patients to minimize the PAP. This is accomplished by a reduction in tidal volume, peak flow, ventilatory rate, and paralysis with pancuronium bromide. At the same time, hyperoxic mixtures are used to ensure adequate arterial saturation. Physiologic pH was maintained with bicarbonate supplementation in Menitove and Goldring’s patients. Prolonged hypercapnia was successfully tolerated in Darioli’s patients as long as the PaCO₂ did not exceed 90 mm Hg. No mortality was experienced in either series.

A variant form of barotrauma occurs in patients with asymmetric lung disease. Because of areas of differential compliance, conventionally delivered mechanical tidal volumes will be distributed in the pathway of the least resistance, which in this case will be to the uninvolved, nondiseased segments of the lungs. This may do little to relieve the hypoxemia and may even make the hypoxemia worse if excess volume is delivered to the normal lung, thereby causing hyperinflation.

Conventional remedies for refractory hypoxemia of increasing PEEP may make hyperinflation worse. Significant alveolar overdistention causes compression of perialveolar capillaries, increases the vascular resist-
ance in that segment, and effectively shunts blood away from these areas. The blood will be redistributed to the nondistended, poorly ventilated, diseased areas of the lungs which decreases the V/Q ratio making the hypoxemia worse.\(^4\) In addition, the V/Q ratio will be increased in the hyperinflated area of the lungs and exacerbate alveolar dead space. We see, therefore, that both ventilation and oxygenation will suffer.

One of the goals of mechanical ventilation is to maintain adequate arterial oxygenation at nontoxic oxygen concentrations. If this cannot be accomplished with the use of PEEP, then what are the alternatives?

Early observations were made that positional changes affected PaO\(_2\) in patients with unilateral lung disease.\(^{35,47}\) Zack et al.\(^6\) performed a prospective study on patients with varying degrees and distribution of lung disease. He found that when the healthy lung was placed in the lateral, dependent position, PaO\(_2\) values improved. This effect was later corroborated by Remolina et al.,\(^{48}\) and in this study, the arterial PaO\(_2\) improvements were much more substantial. Positional changes may therefore be used to improve perfusion in the healthy lung, and thus, improve the V/Q ratio at least on that side. This modification may be adequate to attain satisfactory gas exchange. However, frequently, this adjunct will still be insufficient. Patients who have more profound acute asymmetrical lung disease may not be able to achieve a minimal satisfactory arterial oxygen saturation even at toxic levels of FIO\(_2\).

One solution to this problem presently being developed is the use of independent lung ventilation (ILV).\(^{34,47}\) This is accomplished with a double lumen endotracheal tube and one or two mechanical ventilators. Since conventional ventilation with PEEP may worsen the maldistribution of ventilation and perfusion in asymmetrical lung disease, independently ventilating each lung should minimize this anomalous distribution.

The aim of this therapy is to equalize the difference in compliance and volume between the diseased and healthy lung. Because of differing time constants for expansion between the two lungs, different volumes, pressure, and levels of PEEP are necessary to optimize the functional capacity of each lung.

A variety of ILV modifications have been devised to achieve this goal.\(^{34,47}\) Most recently, Siegel et al.\(^35\) developed a computer-based system capable of monitoring pressure-flow-volume relationships and gas exchange characteristics in each lung independently. From these data, "best PEEP" compliance curves were computed and displayed as needed. This afforded optimal and accurate bedside assessment of disease severity, and adjustments were made accordingly. Although simpler methods of ILV have been devised, this technique is still limited by its demand for technologic sophistication and a certain level of physician expertise in management.

High frequency positive pressure ventilation (HFPPV) as a nonconventional mode of ventilation, may have utility in this disease. Using both convection and diffusion principles of gas exchange, HFPPV may satisfy alveolar ventilation and oxygenation at lower airway pressures\(^{49}\) and may thus avoid the mechanical maldistribution of perfusion induced by conventional ventilation and PEEP. This method used successfully in one case report awaits further trials.\(^{41}\)

**Conclusion**

We have reviewed the animal studies which have elucidated the pathophysiology of barotrauma. In addition, we have listed the known risk factors that predispose to barotrauma and described its variation in presentation and the importance of its recognition.

Special problems in ventilatory management are encountered in the high risk patient with ARDS, status asthmaticus, and asymmetrical lung disease. We have reviewed the techniques presently used for optimal management in these patients.

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