Review Article

# Barotrauma and pneumothorax

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**Abstract:** Barotrauma is physical damage to body tissues caused by a difference in pressure between a gas space inside, or in contact with the body, and the surrounding fluid. This situation typically occurs when the organism is exposed to a significant change in ambient pressure, such as when a scuba diver, a free-diver or an airplane passenger ascends or descends, or during uncontrolled decompression of a pressure vessel, but it can also happen by a shock wave. Whales and dolphins are also vulnerable to barotrauma if exposed to rapid and excessive changes in diving pressures. In the current review we will focus on barotraumas from definition to treatment.

**Keywords:** Barotrauma; pneumothorax; ICU; ventilation

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## Definition

Intubation and mechanical ventilation are common but aggressive therapeutic manoeuvres in anesthesia or in ICU setting. While human being is created, as other creatures, to breathe with a mechanism based on negative pressures, the patient on mechanical ventilation is ventilated with positive pressures, which is not physiological. Barotrauma must be considered as complication of the use of positive pressures in a tissue, where normal air movement is mainly passive. It is defined as the presence of extraalveolar air in locations, where it is not normally found in patients receiving mechanical ventilation (1-9). The implementation of positive pressure ventilation predisposes an already affected lung—when certain limits are overcome—to injury. This is described as ventilator associated lung injury (VALI) or ventilator induced lung injury (VILI). VALI and VILI can be divided into macrobarotrauma (the form of radiologically detected barotrauma) and microbarotrauma, with diffuse lung injury and possible injury of other organs due to release of inflammatory mediators-biotrauma. According to the etymology, barotrauma refers only to high pressures. Nevertheless, it seems that not only pressure, but high lung volume and lung hyperinflation as well, play a major role in barotrauma aetiology. Volutrauma is the term that describes ultrastructural lung injury due to overdistention occurring during mechanical ventilation. The two terms—barotrauma and volutrauma—reflect the two sides of the same phenomenon: the lung injury due to a large distending volume and/or to a high airway pressure (10-19).

Clinical manifestations of barotrauma include pneumothorax, pulmonary interstitial emphysema (PIE), subcutaneous emphysema, pneumoperitoneum, pneumomediastinum or pneumopericardium, air embolisation, tension lung cysts, and hyperinflated left lower lobe.

When an overdistended alveolus ruptures, air is diffused into the perivascular adventitia, resulting in PIE.

Air can be introduced along the perivascular sheaths into the mediastinum and pneumomediastinum or pneumopericardium are present. When adequate air is accumulated in mediastinum, it is decompressed along cervical fascial planes into the subcutaneous tissues and subcutaneous emphysema is formed. The air can be decompressed both retroperitoneally and intraperitoneally (pneumoperitoneum). In case of mediastinal parietal rupture pneumothorax is appeared.

## Pathophysiology

All barotrauma forms come from rupture of a hyperinflated alveolus and air leak into the surrounding tissues and spaces. This suggests that risk factors predisposing to barotrauma are a severe underlying lung disease, which seriously affects alveoli [ARDS, ALI, chronic obstructive pulmonary disease (COPD) exacerbation, necrotizing infections] and all factors predisposing to hyperinflation: high transpulmonary pressure (airway pressure minus the pleural pressure), high tidal volumes, high intrinsic PEEP. In each case hyperinflation occurs when the inspired air cannot be totally expired (dynamic hyperinflation) (20-29).

All forms of pressure are not equal as barotrauma aetiologic factors. The peak airway pressure (Ppk) is the pressure measured by the ventilator in the major airways and it strongly reflects airway resistance. It increases with hyperinflation, but it has no pathological value in the clinical setting, since it arises from the increased airway resistance and the high inspiratory flow rate and is mainly dissipated in the airways and thus does not reach the alveoli.

In volume control modes of ventilation, when the Ppk reaches its maximum value, there is a rapid drop of the pressure until a plateau is reached (plateau pressure-Ppl). This Ppl is the pressure applied to the small airways and alveoli. The Ppl and more correctly the transpulmonary pressure (Ppl-Ppleural) reflect the compliance of the respiratory system, given the initial PEEP level and the delivered tidal volume and gauge the maximum alveolar pressure and hence maximal alveolar volume. Certainly, the volume is related to the pressure by the compliance characteristics of the lung and chest wall. A patient with emphysema (high lung compliance) and elevated Ppl (35 $cmH_2O$) would be expected to have a large end-expiratory alveolar volume and thus be at risk of barotrauma. A patient with very low compliance (e.g., ARDS) would be expected to have a lower alveolar volume at the same Ppl level (35 $cmH_2O$) and hence to be at lower risk of barotrauma.

In respiratory failure, when PEEP is set, the tidal volume (when using pressure controlled ventilation) or the airway pressure above PEEP (when using volume controlled ventilation) are not adjusted. This may produce high Ppl, which might overdistend vulnerable lung regions. This mechanism is pronounced in the volume controlled mode, when ventilating lungs with low compliance. However, even when Ppl is limited to 30-35 $cmH_2O$, as in the ARDS net study (3), the PEEP level is correlated to the incidence of barotraumas, indicating that the PEEP itself may also be a risk factor (4,5).

Intrinsic PEEP (PEEPi) is the lowest alveolar pressure achieved during the respiratory cycle and reflects the least alveolar volume during inspiration and expiration. It is measured during an end-expiratory hold manoeuvre in a relaxed but not necessarily paralysed patient. During this manoeuvre there is no air flow, and pressures equilibrate within the respiratory system and reflect the mean end expiratory alveolar pressure. There is no value of PEEPi that is predictive of barotrauma, but successive measures of PEEPi give an indication of the evolution of dynamic hyperinflation: increasing PEEPi reflects aggravating hyperinflation (6).

The only measure that has been shown to predict complications of hyperinflation, as barotrauma, is the end inspiratory volume (Vei) (7) that is the volume remaining in the lung above functional residual capacity (FRC). It is measured by collecting total exhaled volume in a paralysed patient over 60 s of apnoea. Vei >20 mL/kg is predictive of complications like hypotension and barotraumas. Assuming that the compliance of the respiratory stays constant, Vei can be calculated as follows: $Vei = (Vt \times Ppl)/(Ppl\text{-}PEEPi)$ (2).

In severe COPD patients, all the above mentioned take a major role in the pathophysiology of hyperinflation. In case of COPD exacerbation, the balance between respiratory workload and respiratory working capacity is difficult to maintain. A small disturbance can lead to vicious circle ending up in barotrauma. An acute pathological event (e.g., infection) can precipitate airway obstruction, which leads to hyperinflation and decreased compliance. These changes provoque further increase in minute ventilation and PEEPi, which in the end tend to a further increase of hyperinflation. In this setting Ppk diverges greatly from Ppl and does not necessarily reflect the risk of alveolar injury, as we mentioned earlier. In this condition gas trapping results in a very high PEEPi, which indicates high alveolar end expiratory volume, with an elevated risk of barotrauma,

 

even with small tidal volumes (30-39).

## Diagnosis and clinical signs

These are not specific in sedated critically ill patients. Symptoms can vary from mild hypoxemia, hypotension, and tachycardia to extremely severe signs, such as profound hypoxemia, cardiovascular collapse and low cardiac output syndrome. Ventilator—patient dysynchrony may be present. Subcutaneous emphysema can be revealed by auscultation and palpation: crepitation in the neck, face, chest or abdomen means subcutaneous emphysema. Usually, this is not associated with hemodynamic disturbances, but this finding should alert the intensivist to the possibility of pneumothorax or tension pneumothorax, which if misinterpreted may be lethal. Other clinical signs are patient discomfort, slight increase in airway pressures or decrease in tidal volumes or an unmeasurable arterial blood pressure and increased venous pressure, together with hugely increased airway pressures that renders adequate ventilation impossible (tension pneumothorax). Subcutaneous emphysema and unilateral absence of breath sounds are also signs of barotrauma (40-49).

Radiographic evaluation is the method of choice to evaluate barotrauma. Subcutaneous emphysema is often a common finding in daily routine clinical imaging. This includes air presence in the surrounding the chest tissues, as thorax, neck, arms, face or even abdomen. Nevertheless, the diagnosis of subcutaneous emphysema remains clinical. PIE, which is more common in infants and less recognized in adults, include small parenchymal cysts, perivascular presence of air (halos), linear streaks of air radiating toward the hilus, pneumatoceles and large sub pleural air collections. PIE as subcutaneous emphysema can lead to pneumothorax.

## Prophylaxis-treatment

In barotrauma, as in most conditions in medicine, the best treatment is prophylaxis, as Hippocrates said many centuries ago. In mechanically ventilated patients with severe underlying disease, the use of lung protective strategies is essential to reduce barotrauma risk. The goal of these strategies is to provide an adequate gas exchange ($SpO_2$ >90%) without causing additional iatrogenic damage to lungs (8,9). The most common factors responsible for barotrauma are firstly high lung volume, associated with both elevated transpulmonary pressure and alveolar hyperinflation and secondly, repeated alveolar collapse and reopening due to low end expiratory volume. First goal of this approach is to "open the lung and keep the lung open." This was described by Lachman (10). It involves recruitment manoeuvres by using pressure control mode of ventilation and increasing slowly the peak inspiratory pressure to 55 $cmH_2O$. The I:E ratio is set 1:1. This type of ventilation is continued for about 2 min, after which the Ppl is lowered to 30 $cmH_2O$. Then PEEP high enough to maintain the alveoli open should be set (15-18 $cmH_2O$) and then is carefully titrated downwards towards the lowest possible value that prevents derecruitment.

Ventilation with a tidal volume of 6 mL/kg ideal body weight has been shown to decrease VILI or VALI. However, the acceptable tidal volume depends on the underlying disease. In severe ARDS with low lung compliance, tidal volume should probably be lower than 6 mL/kg. In less severe conditions Vt can be set above 6 mL/kg.

In severe ARDS, compensation of low Vt by increase of frequency (Fr) is usually safe: Exhalation is much faster, due to low compliance combined with nearly normal resistance. In COPD patients Fr should be set low, in order to allow a long expiration time (Te) to oppose dynamic hyperinflation. These patients can be compensated by a relatively high Vt, when the lung compliance is normal or high (40-49).

In a COPD patient under mechanical ventilation, our major concern is hyperinflation and our goal is to minimize it. The main determinants of hyperinflation are expiration time and tidal volume. Expiration time must be as long as possible and this suggests low respiratory rate and low Ti/Te ratio. The minute ventilation must be relatively low (MV=Vt × RR) but with a high inspiratory flow to achieve an adequate tidal volume in a small period of time. Generally, there is a consensus in the ventilator settings in COPD patients: Minute ventilation <115 mL/kg, tidal volume <8 mL/kg, respiratory rate 10-12/min, inspiratory flow 80-100 L/min. There is no agreement on what ventilatory mode should be initially set.

In the ARDS group, the I/E ratio setting must be high (>1) in order to improve oxygenation and alveolar recruitment. In the COPD group, the I/E must be low, in order to prolong the expiration time and to minimise the PEEPi.

The end inspiratory plateau pressure (Ppl) should be kept under 30 $cmH_2O$. However, as the transpulmonary pressure is more important in lung injury than the Ppl, compliance of the chest wall must be considered, as previously mentioned in pathophysiology.

PEEP contributes to the reopening of collapsed alveoli and opposes alveolar collapse. PEEP increases FRC and improves lung compliance. The application of PEEP is lung protective, since it prevents atelectrauma, caused by cyclic collapse and reopening of unstable alveoli. Unfortunately, PEEP can overdistend other-non collapsed-alveoli, as PEEP increases Ppl and therefore can contribute to VALI. PEEP should be set above the lower inflection point (LIP) at the P-V curve (11). However, LIP does only indicates the pressure at which recruitments srarts, and not the pressure at which it ends or the pressure which is high enough to prevent derecruitment. Therefore, LIP is not ideal for setting PEEP (11,12). PEEPi can be offset by external PEEP. This is true only in patients with expiratory bronchial collapse. In other cases of pulmonary hyperinflation (due to short expiratory time), external PEEP and PEEPi have mainly additive effects and can predispose to barotraumas (13).

If it is not possible to reach normoventilation within the safe parameters as mentioned above, it is recommended to use the so-called permissive hypercapnia, to avoid additional iatrogenic lung damage. We accept high $Pco_2$ levels, even up to 70 mmHg, in order to avoid an aggressive ventilation, which might decrease $Pco_2$, but with a high cost, in terms of VALI. This option is acceptable, when Ph is maintained between above 7.2 and renal function is good enough to counterbalance respiratory acidosis (41,50-57).

## Conclusions

Barotrauma is a complication of positive pressures ventilation and is related not only to pressure but also to volume. Dynamic hyperinflation is the major aetiologic factor of barotrauma. Transpulmonary pressure, tidal volume and PEEPi are the main determinants of hyperinflation. Diagnosis is both clinical and radiological. Pneumothorax or tension pneumothorax must be suspected in all forms of barotrauma as possible dangerous complications. Lung protective strategies are essential to prevent barotrauma. Important parameters include: Ppl <30 $cmH_2O$, Vt: 6-8 mL/kg, carefully titrated PEEP.

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## References

1. Prevention and early detection of complications of critical care. In: Hall JB, Schmidt GA, Wood LD. eds. Principles of Critical Care. 3rd ed. New York: McGraw-Hill, 2005:180.
2. Papaiwannou A, Zarogoulidis P, Porpodis K, et al. Asthma-chronic obstructive pulmonary disease overlap syndrome (ACOS): current literature review. J Thorac Dis 2014;6 Suppl 1:S146-51.
3. The Acute Respiratory Distress Syndrome Network. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. N Engl J Med 2000;342:1301-8.
4. Eisner MD, Thompson BT, Schoenfeld D, et al. Airway pressures and early barotrauma in patients with acute lung injury and acute respiratory distress syndrome. Am J Respir Crit Care Med 2002;165:978-82.
5. International consensus conferences in intensive care medicine. Ventilator-associated lung injury in ARDS. American Thoracic Society, European Society of Intensive Care Medicine, Societé de Réanimation Langue Française. Intensive Care Med 1999;25:1444-52.
6. Slutsky AS. Mechanical ventilation. American College of Chest Physicians' Consensus Conference. Chest 1993;104:1833-59.
7. Tuxen DV, Williams TJ, Scheinkestel CD, et al. Use of a measurement of pulmonary hyperinflation to control the level of mechanical ventilation in patients with acute severe asthma. Am Rev Respir Dis 1992;146:1136-42.
8. Brower RG, Ware LB, Berthiaume Y, et al. Treatment of ARDS. Chest 2001;120:1347-67.
9. Boussarsar M, Thierry G, Jaber S, et al. Relationship between ventilatory settings and barotrauma in the acute respiratory distress syndrome. Intensive Care Med 2002;28:406-13.
10. Lachmann B. Open up the lung and keep the lung open. Intensive Care Med 1992;18:319-21.
11. Maggiore SM, Jonson B, Richard JC, et al. Alveolar derecruitment at decremental positive end-expiratory pressure levels in acute lung injury: comparison with the lower inflection point, oxygenation, and compliance. Am J Respir Crit Care Med 2001;164:795-801.
12. Hickling KG. Best compliance during a decremental, but not incremental, positive end-expiratory pressure trial is related to open-lung positive end-expiratory pressure: a mathematical model of acute respiratory distress syndrome lungs. Am J Respir Crit Care Med 2001;163:69-78.
13. Brochard L. Intrinsic (or auto-) PEEP during controlled mechanical ventilation. Intensive Care Med 2002;28:1376-8.

 

14. Kioumis IP, Zarogoulidis K, Huang H, et al. Pneumothorax in cystic fibrosis. J Thorac Dis. 2014;6:S480-7.
15. Kuhajda I, Zarogoulidis K, Kougioumtzi I, et al. Tube thoracostomy; chest tube implantation and follow up. J Thorac Dis 2014;6:S470-9.
16. Manika K, Kioumis I, Zarogoulidis K, et al. Pneumothorax in sarcoidosis. J Thorac Dis 2014;6:S466-9.
17. Kuhajda I, Zarogoulidis K, Kougioumtzi I, et al. Penetrating trauma. J Thorac Dis 2014;6:S461-5.
18. Visouli AN, Zarogoulidis K, Kougioumtzi I, et al. Catamenial pneumothorax. J Thorac Dis 2014;6:S448-60.
19. Huang Y, Huang H, Li Q, et al. Transbronchial lung biopsy and pneumothorax. J Thorac Dis 2014;6:S443-7.
20. Eirini Terzi, Konstantinos Zarogoulidis, Ioanna Kougioumtzi, et al. Acute respiratory distress syndrome and pneumothorax. J Thorac Dis 2014;6:S435-42.
21. Boskovic T, Stojanovic M, Stanic J, et al. Pneumothorax after transbronchial needle biopsy. J Thorac Dis 2014;6:S427-34.
22. Li Z, Huang H, Li Q, et al. Pneumothorax: observation. J Thorac Dis 2014;6:S421-6.
23. Huang Y, Huang H, Li Q, et al. Approach of the treatment for pneumothorax. J Thorac Dis 2014;6:S416-20.
24. Browning RF, Parrish S, Sarkar S, et al. Bronchoscopic interventions for severe COPD. J Thorac Dis 2014;6:S407-15.
25. Machairiotis N, Kougioumtzi I, Dryllis G, et al. Laparoscopy induced pneumothorax. J Thorac Dis 2014;6:S404-6.
26. Ouellette DR, Parrish S, Browning RF, et al. Unusual causes of pneumothorax. J Thorac Dis 2014;6:S392-403.
27. Parrish S, Browning RF, Turner JF Jr, et al. The role for medical thoracoscopy in pneumothorax. J Thorac Dis 2014;6:S383-91.
28. Terzi E, Zarogoulidis K, Kougioumtzi I, et al. Human immunodeficiency virus infection and pneumothorax. J Thorac Dis 2014;6:S377-82.
29. Zarogoulidis P, Kioumis I, Pitsiou G, et al. Pneumothorax: from definition to diagnosis and treatment. J Thorac Dis 2014;6:S372-6.
30. Tsakiridis K, Mpakas A, Kesisis G, et al. Lung inflammatory response syndrome after cardiac-operations and treatment of lornoxicam. J Thorac Dis 2014;6:S78-98.
31. Tsakiridis K, Zarogoulidis P, Vretzkakis G, et al. Effect of lornoxicam in lung inflammatory response syndrome after operations for cardiac surgery with cardiopulmonary bypass. J Thorac Dis 2014;6:S7-20.
32. Argiriou M, Kolokotron SM, Sakellaridis T, et al. Right heart failure post left ventricular assist device implantation. J Thorac Dis 2014;6:S52-9.
33. Madesis A, Tsakiridis K, Zarogoulidis P, et al. Review of mitral valve insufficiency: repair or replacement. J Thorac Dis 2014;6:S39-51.
34. Siminelakis S, Kakourou A, Batistatou A, et al. Thirteen years follow-up of heart myxoma operated patients: what is the appropriate surgical technique? J Thorac Dis 2014;6:S32-8.
35. Foroulis CN, Kleontas A, Karatzopoulos A, et al. Early reoperation performed for the management of complications in patients undergoing general thoracic surgical procedures. J Thorac Dis 2014;6:S21-31.
36. Nikolaos P, Vasilios L, Efstratios K, et al. Therapeutic modalities for Pancoast tumors. J Thorac Dis 2014;6:S180-93.
37. Koutentakis M, Siminelakis S, Korantzopoulos P, et al. Surgical management of cardiac implantable electronic device infections. J Thorac Dis 2014;6:S173-9.
38. Spyratos D, Zarogoulidis P, Porpodis K, et al. Preoperative evaluation for lung cancer resection. J Thorac Dis 2014;6 :S162-6.
39. Porpodis K, Zarogoulidis P, Spyratos D, et al. Pneumothorax and asthma. J Thorac Dis 2014;6:S152-61.
40. Panagopoulos N, Leivaditis V, Koletsis E, et al. Pancoast tumors: characteristics and preoperative assessment. J Thorac Dis 2014;6:S108-15.
41. Visouli AN, Darwiche K, Mpakas A, et al. Catamenial pneumothorax: a rare entity? Report of 5 cases and review of the literature. J Thorac Dis 2012;4:17-31.
42. Zarogoulidis P, Chatzaki E, Hohenforst-Schmidt W, et al. Management of malignant pleural effusion by suicide gene therapy in advanced stage lung cancer: a case series and literature review. Cancer Gene Ther 2012;19:593-600.
43. Papaioannou M, Pitsiou G, Manika K, et al. COPD assessment test: a simple tool to evaluate disease severity and response to treatment. COPD 2014;11:489-95.
44. Boskovic T, Stanic J, Pena-Karan S, et al. Pneumothorax after transthoracic needle biopsy of lung lesions under CT guidance. J Thorac Dis 2014;6:S99-107.
45. Papaiwannou A, Zarogoulidis P, Porpodis K, et al. Asthma-chronic obstructive pulmonary disease overlap syndrome (ACOS): current literature review. J Thorac Dis 2014;6:S146-51.
46. Zarogoulidis P, Porpodis K, Kioumis I, et al. Experimentation with inhaled bronchodilators and corticosteroids. Int J Pharm 2014;461:411-8.
47. Bai C, Huang H, Yao X, et al. Application of flexible

 

bronchoscopy in inhalation lung injury. Diagn Pathol 2013;8:174.
48. Zarogoulidis P, Kioumis I, Porpodis K, et al. Clinical experimentation with aerosol antibiotics: current and future methods of administration. Drug Des Devel Ther 2013;7:1115-34.
49. Zarogoulidis P, Pataka A, Terzi E, et al. Intensive care unit and lung cancer: when should we intubate? J Thorac Dis 2013;5:S407-12.
50. Hohenforst-Schmidt W, Petermann A, Visouli A, et al. Successful application of extracorporeal membrane oxygenation due to pulmonary hemorrhage secondary to granulomatosis with polyangiitis. Drug Des Devel Ther 2013;7:627-33.
51. Zarogoulidis P, Kontakiotis T, Tsakiridis K, et al. Difficult airway and difficult intubation in postintubation tracheal stenosis: a case report and literature review. Ther Clin Risk Manag 2012;8:279-86.
52. Zarogoulidis P, Tsakiridis K, Kioumis I, et al. Cardiothoracic diseases: basic treatment. J Thorac Dis 2014;6:S1.
53. Kolettas A, Grosomanidis V, Kolettas V, et al. Influence of apnoeic oxygenation in respiratory and circulatory system under general anaesthesia. J Thorac Dis 2014;6:S116-45.
54. Turner JF, Quan W, Zarogoulidis P, et al. A case of pulmonary infiltrates in a patient with colon carcinoma. Case Rep Oncol 2014;7:39-42.
56. Machairiotis N, Stylianaki A, Dryllis G, et al. Extrapelvic endometriosis: a rare entity or an under diagnosed condition? Diagn Pathol 2013;8:194.
56. Tsakiridis K, Zarogoulidis P. An interview between a pulmonologist and a thoracic surgeon-Pleuroscopy: the reappearance of an old definition. J Thorac Dis 2013;5:S449-51.
57. Huang H, Li C, Zarogoulidis P, et al. Endometriosis of the lung: report of a case and literature review. Eur J Med Res 2013;18:13.

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