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# *Diaphragm Function during Anesthesia: Still Crazy after All These Years*

CHRONIC obstructive pulmonary disease (COPD) impairs lung gas exchange. General anesthesia also interferes with gas exchange, even in patients with healthy lungs. Both processes are associated with alterations in the structure of chest wall components such as the diaphragm. Structural abnormalities such as the "flattening" of diaphragm that accompanies lung hyperinflation have attracted considerable recent attention as an important part of the pathophysiology of the severe COPD.[1,2] Indeed, one of the presumed benefits of lung volume reduction surgery for COPD is a reduction of chronic hyperinflation, which produces more normal chest wall configuration. Anesthesia also changes the shape and motion of the chest wall. Given the potential for this "double whammy," one would thus expect (and conventional wisdom holds) that patients with COPD would poorly tolerate general anesthesia. For example, most anesthesiologists have seen recommendations from their medical colleagues that a patient with COPD could safely receive regional, but not general, anesthesia. Although COPD is certainly a risk factor for perioperative pulmonary complications, most of these patients actually do quite well with general anesthesia, usually maintaining adequate gas exchange both intraoperatively and postoperatively.[3–8] In this issue of ANESTHESIOLOGY, Kleinman *et al.*[9] revive a venerable experimental method to investigate why this occurs. Consideration of their results also provides a good excuse to review what we know about how anesthesia affects the function of the diaphragm, arguably the most important skeletal muscle in the body. The last editorial in ANESTHESIOLOGY on the subject, thirteen years ago, rightly characterized the diaphragm as the "elusive muscle."[10] Although we haven't caught it yet, the pursuit continues to be instructive.

Almost 30 yr have passed since Froese and Bryan[11] provided the first direct measurements of how anesthesia affects the shape and motion of the normal human diaphragm. They described how the silhouette of the diaphragm created by fluoroscopy moved during breathing before and after the induction of anesthesia. Based on their interpretation of their results and subsequent studies by others,[12] the following scenario evolved (and has been adopted by many anesthesia texts). Anesthesia, with or without pharmacologic paralysis, produces a cephalad (headward) shift of the end-expiratory position of the diaphragm by reducing normal end-expiratory muscle tone. This shift in the diaphragm reduces the functional residual capacity (FRC) and compresses lung parenchyma in dependent regions, causing atelectasis. This atelectasis significantly contributes to intraoperative gas exchange abnormalities by increasing shunt and may persist into the postoperative period[13] (especially after surgeries that invade the thorax or abdomen), perhaps leading to morbidity such as pneumonia.

Although atelectasis is not the sole cause of abnormal gas exchange during anesthesia, subsequent studies have confirmed its significance.[14] Atelectasis has been observed consistently in nearly every group of anesthetized patients, with one fascinating exception. Gunnarsson *et al.*[8] studied patients with moderate to severe COPD and found that most developed no atelectasis at all during anesthesia. Consistent with this observation, anesthesia did not increase shunt in these patients. They also found that, contrary to earlier observations in normal patients made by this group of investigators, that anesthesia did not affect the end-expiratory position of the diaphragm. They postulated that the hyperinflation accompanying COPD in some way protected these patients from cephalad diaphragm displacement and thus contributed to the relative maintenance of gas exchange.

Although appealing, some aspects of this scenario have been challenged by subsequent studies, now including that of Kleinman *et al.* Consider the effect of anesthesia on end-expiratory chest wall configuration in normal patients. It is little appreciated that Froese and Bryan reported the end-expiratory position of the diaphragm of only two normal subjects, who in fact behaved somewhat differently. The most dependent (*i.e.*, posterior in a supine subject) region of the diaphragm moved cephalad with the induction of anesthesia in both subjects. However, the nondependent region (*i.e.*, anterior) actually moved in the opposite direction (caudad) in one subject, and cephalad in the other. Subsequent studies by several investigators have also found that although anesthesia produces a consistent cephalad shift of dependent diaphragm regions, it causes either no change in position or an actual caudal shift of nondependent regions.[15–20] Kleinman *et al.* noted a similar pattern of results in their normal subjects, although none of the shifts were statistically significant because of consider-

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able variation among subjects. Thus, they confirm that decreases in FRC produced by anesthesia in normal subjects cannot be attributed primarily to changes in diaphragm position. Furthermore, if atelectasis is caused by compression of lung parenchyma by a cephalad displacement of the dependent diaphragm, the greater the displacement, the greater should be the amount of atelectasis. Alas, there is no such correlation.[19,21] Thus, although there is little doubt that atelectasis is caused by anesthesia-induced changes in chest wall shape, the cause is not as simple as a cephalad shift of the diaphragm. For example, anesthesia increases the curvature of the spine,[18,20] which provides an anchor for all other chest wall structures and thus may affect diaphragm and rib cage configuration. Such secondary effects probably explain any changes in diaphragm shape produced by anesthesia, because there is currently little evidence that there is "normal" end-expiratory muscle tone in the diaphragm of awake subjects.[22]

So much for healthy patients; what about the effects of anesthesia on chest wall configuration in patients with COPD? The most striking feature of Kleinman *et al.*'s data is that the overall effects of anesthesia on the position and motion of the diaphragm were remarkably similar in healthy patients and those with COPD (albeit with substantial variability among patients), despite the significant hyperinflation present in the latter. Although other chest wall components such as the rib cage were not studied, it appears that at least the diaphragm of patients with COPD adapts to provide a relatively normal pattern of displacement during active contraction. Further, the regional compliances of the passive diaphragm also appear to be relatively normal in these patients, as evidenced by an unchanged pattern of displacement during positive pressure ventilation. To be sure, there are definite limits to the ability of fluoroscopy to image the complex three-dimensional structure of the diaphragm. However, it appears that any differences in the tendency of normals and patients with COPD to develop atelectasis and shunt with the induction of anesthesia cannot be attributed to striking differences in diaphragm position and motion, as postulated by Gunnarsson *et al.*

What then accounts for the apparent lack of atelectasis formation and relative preservation of gas exchange in patients with COPD? We still don't know, but speculation is one of the privileges of the editorialist. First, airflow obstruction often creates intrinsic positive end-expiratory pressure in patients with COPD, and this may help maintain end-expiratory lung volumes during anesthesia. Second, the pattern of respiratory muscle activation is altered in patients with COPD compared with patients with healthy lungs. In particular, abdominal muscles actively assist expiration in many COPD patient in the supine position, unlike in healthy patients.[23] Removal of this activity by anesthesia with pharmacologic paralysis may promote maintenance of FRC during anesthesia.[20] Finally, it is now apparent that high alveolar oxygen concentrations (presumably used during the induction of anesthesia by Kleinman *et al.*, although this is not specified) promote the development of atelectasis during anesthesia,[24] and it is possible that the ventilation/perfusion mismatch characteristic of COPD or mechanical changes in the lung parenchyma may affect this process. All of these mechanisms could (and should) be evaluated in future studies.

Apart from these insights into applied respiratory physiology, the article of Kleinman *et al.* serves as a reminder that despite the recent (and well-deserved) enthusiasm for the promise of genetic and molecular techniques in science, there are still many interesting questions remaining in patient-oriented research that are amenable to study by readily-applied techniques such as fluoroscopy. Requirements for such studies remain the same as always: intellectual curiosity, the knack for asking the right questions, and the tenacity required to conduct research in clinical settings. Congratulations to these authors for continuing the chase of the elusive muscle.

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## References

1. Butler C: Diaphragmatic changes in emphysema. Am Rev Respir Dis 1976; 114:155–9
2. De Troyer A: Effect of hyperinflation on the diaphragm. Eur Respir J 1997; 10:708–13
3. Milledge JS, Nunn JF: Criteria of fitness for anaesthesia in patients with chronic obstructive lung disease. BMJ 1975; 3:670–3
4. Pietak S, Weenig CS, Hickey R, Fairley HB: Anesthetic effects on ventilation in patients with chronic obstructive pulmonary disease. ANESTHESIOLOGY 1975; 42:160–6
5. Warner DO, Warner MA, Offord KP, Schroeder DR, Maxson P, Scanlon PD: Airway obstruction and perioperative complications in smokers undergoing abdominal surgery. ANESTHESIOLOGY 1999; 90:372–9
6. Wong DH, Weber EC, Schell MJ, Wong AB, Anderson CT, Barker SJ: Factors associated with postoperative pulmonary complications in patients with severe chronic obstructive pulmonary disease. Anesth Analg 1995; 80:276–84
7. Kroenke K, Lawrence VA, Theroux JF, Tuley MR: Operative risk in patients with severe obstructive pulmonary disease. Arch Intern Med 1992; 152:967–71
8. Gunnarsson L, Tokics L, Lundquist H, Brismar B, Strandberg A, Berg B, Hedenstierna G: Chronic obstructive pulmonary disease and anaesthesia: formation of atelectasis and gas exchange impairment. Eur Respir J 1991; 4:1106–16
9. Kleinman BS, Frey K, VanDrunen M, Sheikh T, DiPinto D, Mason R, Smith T: Motion of the diaphragm in patients with chronic obstructive pulmonary disease (COPD) while spontaneously breathing *versus* during positive pressure breathing after anesthesia and neuromuscular blockade. ANESTHESIOLOGY 2002; 97:298–305
10. Froese AB: Anesthesia-paralysis and the diaphragm: In pursuit of an elusive muscle. ANESTHESIOLOGY 1989; 70:887–90
11. Froese AB, Bryan AC: Effects of anesthesia and paralysis on diaphragmatic mechanics in man. ANESTHESIOLOGY 1974; 41:242–55
12. Hedenstierna G, Strandberg A, Brismar B, Lundquist H, Svensson L, Tokics L: Functional residual capacity, thoracoabdominal dimensions, and central blood volume during general anesthesia with muscle paralysis and mechanical ventilation. ANESTHESIOLOGY 1985; 62:247–54
13. Lindberg P, Gunnarsson L, Tokics L, Secher E, Lundquist H, Brismar B, Hedenstierna G: Atelectasis and lung function in the postoperative period. Acta Anaesthesiol Scand 1992; 36:546–53
14. Tokics L, Hedenstierna G, Svensson L, Brismar B, Cederlund T, Lundquist H, Strandberg A: V/Q distribution and correlation to atelectasis in anesthetized paralyzed humans. J Appl Physiol 1996; 81:1822–33
15. Krayer S, Rehder K, Beck KC, Cameron PD, Didier EP, Hoffman EA: Quantification of thoracic volumes by three-dimensional imaging. J Appl Physiol 1987; 62:591–8
16. Krayer S, Rehder K, Vettermann J, Didier EP, Ritman EL: Position and motion of the human diaphragm during anesthesia-paralysis. ANESTHESIOLOGY 1989; 70:891–8

17. Drummond GB, Allan PL, Logan MR: Changes in diaphragmatic position in association with the induction of anaesthesia. Br J Anaesth 1986; 58:1246–51

18. Warner DO, Warner MA, Ritman EL: Human chest wall function while awake and during halothane anesthesia. I. Quiet breathing. ANESTHESIOLOGY 1995; 82:6–19

19. Warner DO, Warner MA, Ritman EL: Atelectasis and chest wall shape during halothane anesthesia. ANESTHESIOLOGY 1996; 85:49–59

20. Warner DO, Warner MA, Ritman EL: Mechanical significance of respiratory muscle activity in humans during halothane anesthesia. ANESTHESIOLOGY 1996; 84:309–21

21. Reber A, Nylund U, Hedenstierna G: Position and shape of the diaphragm: implications for atelectasis formation. Anaesthesia 1998; 53:1054–61

22. Druz WS, Sharp JT: Activity of respiratory muscles in upright and recumbent humans. J Appl Physiol 1981; 51:1552–61

23. Ninane V, Rypens F, Yernault JC, De Troyer A: Abdominal muscle use during breathing in patients with chronic airflow obstruction. Am Rev Respir Dis 1992; 146:16–21

24. Rothen HU, Sporre B, Engberg G, Wegenius G, Reber A, Hedenstierna G: Prevention of atelectasis during general anesthesia. Lancet 1995; 345: 1387–91