Esophageal and Gastric Pressure Measurements

Joshua O Benditt MD

The measurement of esophageal and gastric pressures with balloon-tipped catheters has been used with great success over the past half century to delineate the physiology of the mechanical respiratory system. Pleural pressure and abdominal pressure values estimated from esophageal and gastric pressure measurements allow analysis of lung and chest wall compliance, as well as work of breathing, respiratory muscle function, and the presence of diaphragm paralysis. Although much of the use of these measurement techniques has been in the clinical laboratory, to improve the understanding of basic physiologic mechanisms, the techniques have also been used in clinical situations to diagnose diaphragm paralysis, assess the work of breathing during mechanical ventilation, and estimate pulmonary compliance. In this article I review the historical background, physiology, placement techniques, and potential clinical applications of esophageal and gastric pressure measurements. In addition, I will briefly review the measurement of bladder pressure, which is a related topic. *Key words: esophageal pressure, gastric pressure, pleural pressure, work of breathing, diaphragm paralysis, lung compliance, chest wall compliance, pressure time index.* [Respir Care 2005;50(1):68–75. © 2005 Daedalus Enterprises]

Introduction

Monitoring ventilation is one of the critical functions of the modern intensive care unit (ICU), and there are many methods for assessing ventilation in the ICU. To understand airflow and ventilation in humans, we must understand the pressures generated by components of the respiratory system. These pressures, which generate airflow in the human respiratory system, are complex. Bedside inspection of ventilation and respiratory pattern, and assessment of easily measured airway pressures are often adequate for understanding respiratory physiology and

Joshua O Benditt MD is affiliated with the Division of Pulmonary and Critical Care Medicine, University of Washington, Seattle, Washington.

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Correspondence: Joshua O Benditt MD, Division of Pulmonary and Critical Care Medicine, University of Washington School of Medicine, Box 356522, 1959 NE Pacific Street, Seattle WA 98195-8673. E-mail: benditt@u.washington.edu.

pathophysiology. However, for a detailed understanding of the physiology of the mechanical respiratory system and, on occasion, to best deliver treatment for respiratory failure, a more detailed assessment of pressures within the respiratory system is necessary. Esophageal and gastric pressure measurement is one technique available to gain that more in-depth evaluation. This article details the use of those measurements, with an emphasis on potential clinical applications.

Historical Development

The mechanics of breathing have intrigued scientific observers for centuries. In ancient times it was believed that the thorax was expanded by an actively expanding lung. Later, Galen (circa 150 AD) first understood that the lungs were expanded by the outward movement of the thorax. It was many more centuries, however, before the first scientific recording of the elastic properties of the lung was performed, by Carson, a Scottish physician, who in 1817 attached a water manometer to the trachea of a recently killed animal and noted an increase in the tracheal pressure when the chest was opened, which he attributed to the elasticity of the lung.[1] Similar measurements were later performed with humans by Donders, who realized that there were pressure fluctuations within the pleural space.[2] The first pleural pressure measurement is attributed to Ludwig, who in 1847 made recordings using a water-filled balloon inserted into the intrapleural space of an experimental animal. The balloon was connected to a mercury manometer.[3] In 1900, Aron recorded the first direct pleural pressure measurement in a human with emphysema, who was being treated with suction drainage with a chest tube.[4] It was not, however, until the mid-20th century that a less invasive method for estimating the pleural pressure was developed that allowed more routine laboratory and clinical assessment of the detailed respiratory mechanics.[5] This allowed for the accumulation of large amounts of human in vivo data and clear descriptions of the actions of the respiratory muscles and the elastic properties of the lungs during the 1950s and 1960s. Measurements of esophageal and gastric pressure have been used intermittently in clinical practice since that time.

Techniques

Physiologic Background

The lung and chest wall are 3-dimensional mechanical structures that can change in volume under the influence of pressures applied naturally by the respiratory muscles or artificially by applying positive pressure to the airway (ie, positive-pressure ventilation) or negative pressure external to the chest wall (ie, negative-pressure ventilation,
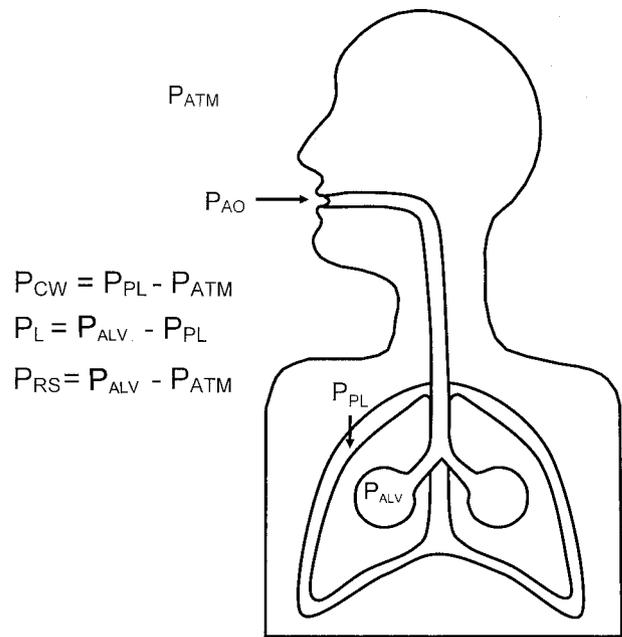

$$P_{CW} = P_{PL} - P_{ATM}$$
$$P_L = P_{ALV} - P_{PL}$$
$$P_{RS} = P_{ALV} - P_{ATM}$$

Fig. 1. Illustration of and equations for trans-pulmonary (P_L), trans-chest wall (P_{CW}), and trans-respiratory system (P_{RS}) pressures. P_{pl} = pleural pressure. P_{atm} = atmospheric pressure. P_{alv} = alveolar pressure. P_{AO} = pressure at the airway opening.

such as the "iron lung"). The lung and chest wall move together, conjoined by the pleural space, which is in fact only a potential space. The pressure in the pleural space is denoted P_{pl}, and at rest in the upright human it is generally slightly negative, because the lung is a passive structure that is elastic and has a tendency to recoil to a smaller volume than the respiratory system combination (lung and chest wall together). The lung is prevented from collapsing because of the tendency of the chest wall to recoil outwards and the negative value of P_{pl}. At the end of a relaxed exhalation (to functional residual capacity) and with the mouth open, the alveolar pressure (P_{alv}), the pressure at the airway opening (P_{AO}), and the atmospheric pressure (P_{atm}) are equal. Thus, at functional residual capacity with the mouth open, the distending pressure of the lung (P_L) is equal to the pressure inside the lung P_{alv} (which in this case is equal to P_{atm}) minus the pressure in the pleural space P_{pl} (Fig. 1). The importance of this is that the distending pressure across the lung (transpulmonary pressure) determines the volume of the lung. Changes in distending pressure result in changes in lung volume and therefore ventilation. Thus, to understand ventilation—a primary objective in respiratory medicine—we must understand and be able to measure P_{pl} and P_{alv}. This will in turn allow us to calculate the all-important distending pressure of the lung, chest wall, and respiratory system.

As noted above, P_{alv} is measured by assessing P_{AO} during a static maneuver when, with an open glottis and uninterrupted airway, $P_{alv} = P_{AO} = P_{atm}$. We can easily
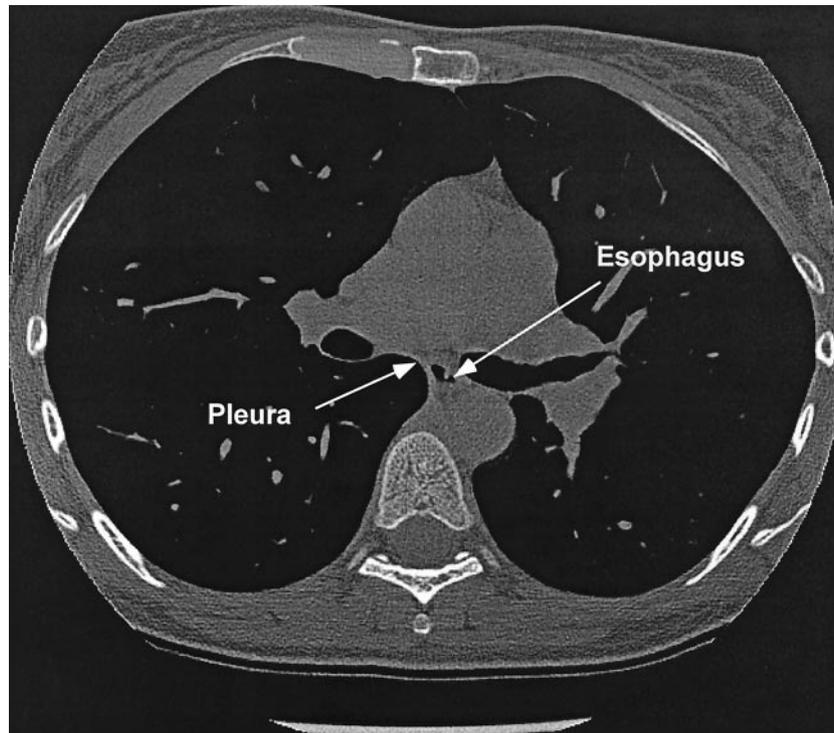
Fig. 2. Computed tomogram of the chest, showing the proximity of the esophagus to the pleural space.

measure P_{atm}, and by convention, P_{atm} is said to equal a pressure of zero. P_{pl} is measurable directly only by placing a catheter in the pleural space, which is not usually possible in clinical practice. Fortunately, the pressure in the lower one third of the esophagus (P_{es}) closely approximates the pressure in the adjacent pleura[6–8] when the subject is in the upright posture. Figure 2 shows the reason for this; it is a cross-sectional computed tomogram view of the thorax, which shows the close proximity of the esophagus to the pleural space. Because the body of the esophagus is essentially a passive structure (except during a swallow), able to transmit pressure from the adjacent pleural space (P_{pl}) to the measurement catheter in the esophagus, P_{es} is a reasonably close surrogate for P_{pl} in a human being in the upright posture.[6,8] This does not necessarily hold true in the supine posture, in which the mediastinum may compress the esophagus, and compression of the posterior and inferior portions of the lung can create large regional differences in pleural pressure.[9,10]

In addition to the measurement of P_{es}, it is also possible to measure the gastric pressure (P_{ga}) by placing another catheter more distally, in the stomach. P_{ga} closely approximates the pressure in the abdominal cavity. With accurate measurements of P_{pl} and abdominal cavity pressure, a wide variety of useful measurements of the mechanical respiratory system can be determined. I will discuss below some of the more clinically important of these measurements, which include: (1) lung and chest wall compliances, (2)

work of breathing (WOB), (3) respiratory muscle performance, and (4) transmural cardiac distending pressures.

Technique of Catheter Placement

Figure 3 shows a diagram of the devices required for placing and recording measurements from an esophageal catheter. The components include the balloon catheter, pressure transducer, and a recording device (either a computer or strip-chart recorder).[7]

The catheters are commercially available but can be easily manufactured in the laboratory. The device consists of a thin polyethylene catheter with multiple small holes in the distal 5–7 cm of its length (Figure 4). The distal end of the catheter is then placed in a 10-cm latex balloon that prevents the holes in the catheter from being occluded by esophageal tissue and maintains a column of air within and around the catheter, in order to measure pressure in the surrounding structure. The proximal end of the catheter is attached to the pressure transducers and recording equipment.

The balloon catheter (or catheters) is passed through the nares into the posterior pharynx. At this point the subject is instructed to swallow (if spontaneously breathing) and the catheter is passed into the esophagus and then into the stomach. The catheter is attached to the transducer/recorder system, and 2.0 mL of air is injected into the balloon. Then 1.5 mL of air is withdrawn, to leave 0.5 mL of air in the system to partially inflate the balloon and the catheter. The
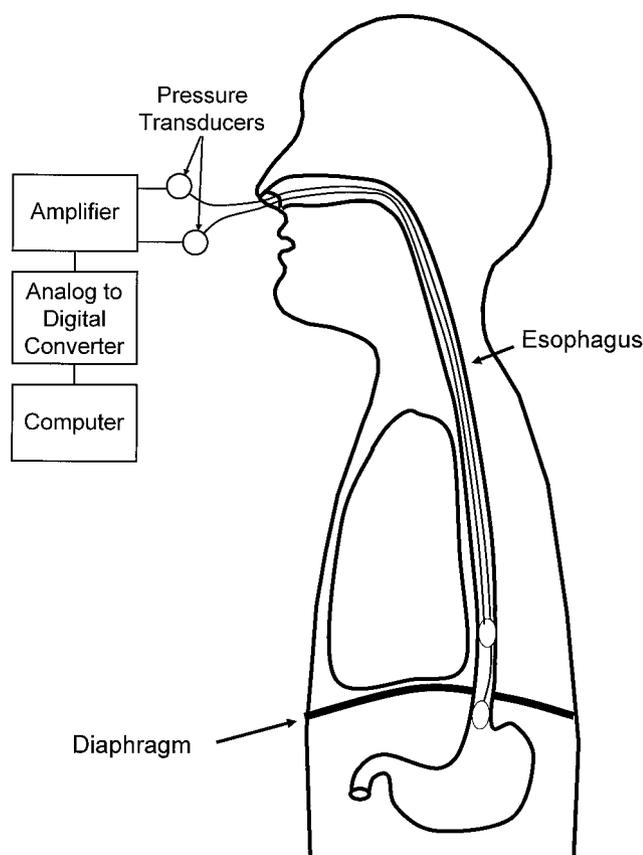
Fig. 3. Diagram of equipment required for recording pressure signals from esophageal and gastric balloon catheters. Note catheter positioning in relationship to the diaphragm.

Fig. 4. Commercially available balloon catheter (part #140912–032–000, Sensor Medics, Yorba Linda, California).

presence of a positive pressure deflection during inspiration indicates that the balloon is located in the stomach, if the diaphragm is functioning. The catheter is then slowly withdrawn into the esophagus, where the pressure reads negative during inspiration. The catheter is then withdrawn

another 10 cm after the initial negative deflection, to ensure that the entire catheter is within the esophagus. The catheter will be posterior to the heart, and cardiac pulsations appear on the waveform. The catheter tip will be approximately 35–45 cm from the nares.[7] It is helpful to mark the catheter at 10-cm intervals prior to placement, and some commercially made devices are pre-measured and marked. If a gastric balloon is being placed, the same procedure is followed, but the catheter is not withdrawn and 2.0 mL of air is added to the system. If diaphragm paralysis is present, the gastric pressure may not be positive during inspiration and so the gastric catheter tip will have to be placed beyond the point where cardiac pulsations are seen, or at least to 45 cm from the nares.

To assure that the esophageal catheter is in the correct position, a dynamic "occlusion test" is performed to assure that P_{es} is changing in concert with P_{AO}. In this test the subject makes inspiratory and expiratory efforts against a closed airway.[11,12] Equivalence of P_{AO} and P_{es} over a range of pressures during respiratory effort is believed to ensure the accuracy of the P_{es} measurement.

Measurements and Clinical Applications

Compliance Measurements

Compliance is a measure of the distensibility of a mechanical structure. It is calculated by dividing the change in volume of that structure (ΔV) by the change in applied pressure (ΔP):

$$\text{Compliance} = \Delta\text{ Volume}/\Delta\text{ Pressure} \tag{1}$$

or

$$C = \Delta V/\Delta P \tag{2}$$

In the ICU it is common to measure the compliance of the total respiratory system (C_{RS}), which is calculated as:

$$C_{RS} = V_T/(P_{AO}\text{ end-inhalation} - P_{AO}\text{ end-exhalation}) \tag{3}$$

in which C_{RS} is the resistance of the respiratory system, V_T is tidal volume, and P_{AO} is the pressure at the airway opening. Because we cannot measure P_{alv} directly, this is done by recording static airway pressure (P_{AO}) measurements using values displayed by the ventilator at the end of expiration and the end of inspiration. During a static maneuver with an open airway between ventilator tube and alveolus, $P_{AO} = P_{alv}$.

However, the use of the esophageal balloon catheter allows us to divide the compliance of the respiratory sys-

tem (C_{RS}) into its components of lung compliance (C_L) and chest wall compliance (C_{CW}) The calculations are:

$$C_L = V_T/(P_{AO} - P_{es}) \text{ end-inhalation } -$$

$$(P_{AO} - P_{es}) \text{ end-exhalation} \quad (4)$$

$$C_{CW} = V_T/(P_{es} - P_{atm}) \text{ end-inhalation } -$$

$$(P_{es} - P_{atm}) \text{ end-exhalation} \quad (5)$$

This can be extremely important clinically, as we are most often interested in lung pathology changes over time, and therefore we are interested in changes in C_L rather than changes in C_{CW}, which often occur but are usually not clinically important. For example, if we rely on C_{RS} measured at the bedside to follow changes in the severity of a patient's acute respiratory distress syndrome, we may see changes in the value that do not reflect changes in C_L but may reflect changes in C_{CW} incurred by changes in edema in the chest wall soft tissue structures, abdominal distention, paralytic agents, or even simple changes in patient position. In an upright human the normal value for compliance of the chest wall and the lung is approximately 200 mL/cm H_2O. The compliance of the respiratory system is approximately 100 mL/cm H_2O.

Work of Breathing Measurement

The WOB is often substantially elevated in individuals with illness that requires ICU admission. Techniques for measuring the WOB have been available for nearly a century.[3] With the advent of novel modes of mechanical ventilation, much interest has developed in the WOB imposed by various ventilation modes and devices. Several commercial devices for measuring WOB have been used in the clinical setting, although their popularity has declined recently.[13,14]

From classic physics, work in a 2-dimensional system is equal to the force applied to an object multiplied by the distance the object travels. That is, work = force × distance, or W = F × D.

However, in the 3 dimensions that apply in the respiratory system, work now becomes the pressure applied to yield a change in the volume of the system, or

$$W = P \times V = \int_0^V P \times dv \quad (6)$$

in which $\int_0^V P$ is the integral of the pressure across the respiratory system, as a function of volume, and dv is the change in the volume of the respiratory system. Work performed on the lung and chest wall can be depicted graphically as areas under the active inflation and deflation pressure-volume curves as they relate to passive pressure-volume curves of those structures. In this situation

Table 1. Weaning Outcome Predicted by Work of Breathing Measurements

Study	Ventilator-Dependent		Ventilator-Independent	
	Work/L (J/L)	Work/min (J/min)	Work/L (J/L)	Work/min (J/min)
Fiastro et al[16]	NM	> 15.88	< 1.27	≤ 15.68
Henning et al[17]	NM	> 16.66	NM	< 9.80
Proctor et al[18]	NM	> 13.13	NM	< 13.13
Peters et al[19]	≥ 0.98	≥ 9.80	< 0.98	< 9.80

NM = not measured

work is expressed as L × cm H_2O. In practice, work is often expressed in the form of joules. One joule equals the work when 10 cm H_2O is applied to 1 L of gas. Campbell refined earlier analyses and developed the Campbell diagram, which revolutionized the analysis of WOB and allowed partitioning of WOB into its elastic, resistive, inspiratory, expiratory, lung, and chest wall components.[15] By using an esophageal balloon, it is possible to partition the WOB into components and to identify how much work the patient is actually performing. Work is most often described in joules, and work units are often presented in 2 ways: J/min and J/L of gas.

Several commercial devices (eg, CP-100, Bicore Monitoring Systems, Irvine, California, and Ventrak, Novametrix Medical Systems, Wallingford, Connecticut) marketed in the 1990s were designed to measure WOB in real time in mechanically ventilated patients.[13,14] One of the intended uses for these devices was the assessment of a minimum "cutoff" level for WOB as a predictor for ventilator dependence. The hypothesis is that spontaneous ventilation without mechanical assistance is not possible for prolonged periods. Table 1 shows the results of 4 such studies, in which the WOB was studied in groups of ventilated patients, some of whom were weaned from ventilation and others of whom were not.[16-19] The WOB was used a predictor for identifying which individuals could be weaned from mechanical ventilation. Unfortunately, all the "cutoff" points in these studies were determined post-hoc and there was a great deal of overlap among the patient groups that were and were not weanable from mechanical ventilation. No study has prospectively looked at WOB as a determinate of weaning failure and ventilator dependence. Use of these devices in the ICU has decreased substantially in the past decade.

Clearly, measurement of WOB in an investigational setting can be quite accurate and has greatly aided our understanding of disease processes and mechanical ventilation. For example Marini et al elegantly demonstrated, using the measurement principals described above, that substantial respiratory muscle work often occurred during conventional me-

chanical ventilation when inspiratory flows were not set high enough to account for the increased velocity of respiratory muscle contraction in states of elevated respiratory drive.[20]

Tension-Time Index and Pressure-Time Product

Measurement of the mechanical WOB can underestimate true energy costs to the subject, because of energy expended during isometric contraction and the amount of time spent in contraction. A measure that appears to more closely approximate the oxygen cost of breathing is the pressure-time product,[21,22] which is calculated as the product of the time spent in muscle contraction during inspiration as a percent of the total respiratory cycle time and the pressure generated by the muscle during inspiratory contraction. The pressure measurement most often used in this calculation is P_{es}. For patients receiving volume-controlled ventilation, in which the tidal volume is predetermined, the calculations are straightforward. Unfortunately, with pressure-support ventilation the calculations are made more difficult, because lung volume and inspiratory flow can vary from breath to breath. Jubran and Tobin[23] recently developed a modified method for calculating the upper and lower bounds of the pressure-time product for patients on pressure-support ventilation.

In addition, a tension-time index specifically designed for the diaphragm has been developed, in which esophageal and gastric balloons allow calculation of transdiaphragmatic pressure (P_{di}), which is calculated as $P_{ga} - P_{es}$. Thus, the tension-time index for the diaphragm is the product of the total respiratory cycle time and P_{di}/maximum P_{di}. Bellemare et al[24] noted that if the tension-time index for the diaphragm value exceeded 0.15, the diaphragm was likely to rapidly fatigue and be unable to maintain contraction. The tension-time index for the diaphragm correlates well with measures of oxygen consumed by the diaphragm.[21,22,24]

Respiratory Muscle Function

Assessment of respiratory muscle function is improved greatly with esophageal and gastric pressure measurement. In large part this is because the diaphragm, the major muscle of inspiratory function, is inaccessible to direct clinical assessment. Measurement of P_{es} and P_{ga} allows calculation of P_{di} according to the formula $P_{di} = P_{ga} - P_{es}$. Measurements of diaphragm force-generation can be made in relative isolation from intercostals, accessory muscles, and elastic recoil of the chest wall. Davis et al have suggested that P_{di} should be used as a routine clinical measurement in patients with suspected diaphragm weakness or paralysis.[25]

The measurement of maximum P_{di} can be obtained volitionally by having the patient inspire as forcefully as possible against a closed airway, which is known as the Mueller maneuver,[26] or by having the patient sniff force-
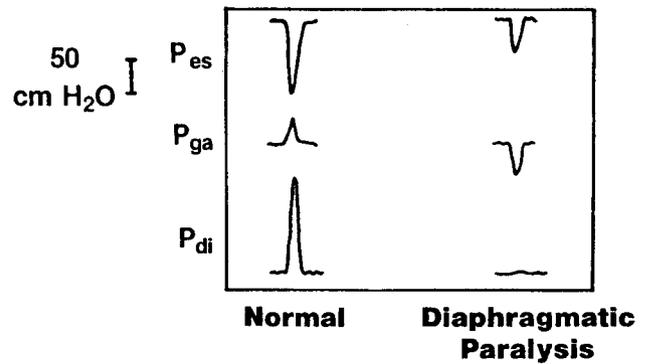

Fig. 5. Esophageal and gastric balloon catheter waveforms in a normal individual and a subject with diaphragm paralysis. P_{es} = esophageal pressure. P_{ga} = gastric pressure. P_{di} = diaphragmatic pressure. (From Reference 32, with permission.)

fully.[27] Sniff P_{di} appears to generate higher and more reproducible values and is preferred by some institutions for routine measurement.[27–31] In the laboratory setting a maneuver known as the "Mueller-expulsive" can also measure maximum P_{di} and appears to generate higher values. This is a difficult maneuver for patients to accomplish and therefore is infrequently used in the clinical setting.

It is also possible to measure maximum P_{di} without relying on patient volition, by stimulating the phrenic nerve with electrical or magnetic stimulators. It must be noted that the volume at which the maximal P_{di} maneuver is initiated is very important, because the diaphragm shortens progressively as lung volume increases and is able to generate less force as it shortens. Maximum pressure-generation occurs at residual volume, although it is common practice to measure maximum P_{di} at functional residual capacity. The normal range for P_{di} depends on size, gender, body position, and the initial volume of the respiratory system during the maneuver, but a normal P_{di} for an adult is around 100 cm H_2O.

Bilateral diaphragm paralysis can also be assessed with the use of esophageal and gastric balloon catheters. Although fluoroscopy is often performed in attempts to diagnose this disorder, the results can be misleading and can lead to a false negative test.[25] The finding of $P_{di} = 0$ during an inspiratory maneuver is diagnostic of bilateral diaphragm leaflet paralysis[32] (Fig. 5) and this may be the only reliable method to arrive at that diagnosis.

Left-Atrial Distending Pressure

Left-atrial distending pressure is an important determinant of left-ventricular end-diastolic dimensions and performance of the left ventricle. Left-ventricular distending pressure is equal to the left-atrial end-diastolic pressure minus the pressure immediately external to the left atrium,

which is the pericardial pressure. In clinical practice, left-atrial end-diastolic pressure is estimated by measuring the pulmonary artery occlusion ("wedge") pressure. Pericardial pressure is essentially impossible to measure in routine clinical practice. Because of the proximity of the esophagus to the pericardium, it had been thought that using P_{es} to estimate pericardial pressure would be possible. However, in situations where the measurement of pericardial pressure is of particular importance, as during the application of PEEP, P_{es} has been shown not to correlate well with pericardial pressure and may not be accurate in estimating left-atrial distending pressure.

Marini et al studied 8 mongrel dogs and found that P_{es} did not correlate directly with measured pericardial pressure.[33] During the administration of PEEP in the supine position, the heart was elevated and shifted to the left. They concluded that this elevation moved the weight of the heart off the esophagus, decreasing P_{es} and causing an underestimation of pericardial pressure. They found similar elevation of the heart by PEEP in 3 human subjects in the supine position as well. Kingma et al noted a similar underestimation of pericardial pressure by P_{es}.[34] Thus, although we do not have extensive data from humans, it cannot be recommended that P_{es} be routinely used to measure pericardial pressure and left-atrial distending pressure.

Measurement of Intra-Abdominal Pressure With a Bladder Catheter

Measurement of intra-abdominal pressure is helpful for assessing diaphragm function, as described in the preceding paragraphs. In addition, intra-abdominal pressure measurement is important when considering disease states in which there is pathologic elevation of the pressure below the diaphragm, known as abdominal compartment syndrome. In that situation the pressure is markedly elevated in the closed intra-abdominal compartment, and this leads to decreased perfusion of intra-abdominal organs, which can threaten their viability. The mortality of untreated abdominal compartment syndrome has been reported to range from 42% to 100%.[35] The leading cause of abdominal compartment syndrome is massive volume resuscitation, which is often required following trauma, surgery, or catastrophic medical illness.

Elevation of intra-abdominal pressure can have detrimental effects not only on the abdominal organs but also on the heart and lungs. Elevation of the diaphragm can cause direct cardiac compression, which reduces ventricular compliance.[36] Elevated intra-abdominal pressure also can lead to impaired venous return, by compressing the vena cava within the abdomen. Elevated intra-abdominal pressure is transmitted across the diaphragm and can lead to increases in intrathoracic pressure, which can artificially elevate intravascular and intracardiac pressure measure-
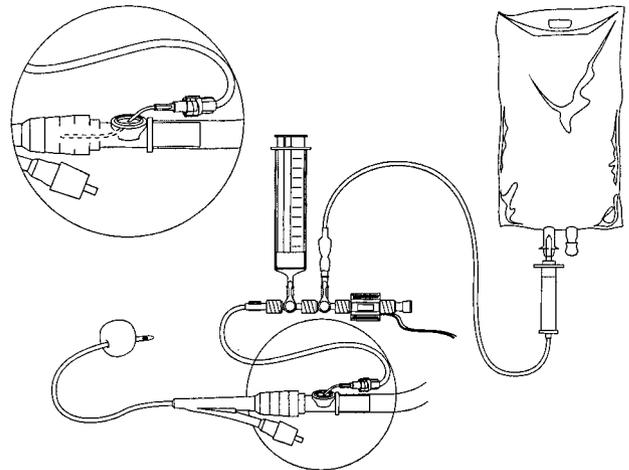

Fig. 6. This closed system for measuring bladder pressure includes a 1,000-mL bag of normal saline, a 60-mL Luer-Lok syringe, a segment of pressure tubing, and a disposable pressure transducer. An 18-gauge angiocatheter is inserted into the culture-aspiration port of the urinary drainage tubing and the needle is removed, leaving the plastic infusion catheter in place. (From Reference 38, with permission.)

ments, including pulmonary-capillary wedge pressure. In addition, in mechanically ventilated patients, airway pressures are increased. Compression of the lung, atelectasis, and pulmonary dysfunction can occur.[37]

Physical examination and radiologic testing is not effective in diagnosing abdominal compartment syndrome. Measurement of intra-abdominal pressure can be performed by assessing P_{ga}. However, measurement of bladder pressure is an easy and reliable method for assessing intra-abdominal pressure (Fig. 6).[38] The technique uses an indwelling bladder catheter to measure intra-abdominal pressure across the bladder wall. There is a strong correlation between bladder pressure and intra-abdominal pressure in humans and animals.[39]

Summary

Esophageal and gastric pressure measurements have been extremely helpful in understanding the physiology of the respiratory system during spontaneous breathing and mechanical ventilation. The measurements can be helpful in some clinical situations. Measurements of WOB and pressure-time index can be performed in clinical situations, but certainly are not routinely warranted. Measurement of P_{di} for the diagnosis of complete diaphragm paralysis is the accepted standard test. The measurement of (relatively easily measured) bladder pressure can be very useful in the assessment of potentially devastating intra-abdominal compartment syndrome.

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Discussion

MacIntyre: In the late 1980s and early 1990s, these esophageal and gastric pressure measurement devices were thought to be, as you put it, the Swan-Ganz catheter equivalent for the pulmonologists, and they were going to give us all kinds of information. And they did give us lots of data, but I think the problem was that the data didn't help us make decisions. For instance, the work of breathing, as you pointed out, is only one determinant of a ventilator-dependent patient. The work only looks at the loads; it doesn't look at the capabilities, the cardiac function, the muscle function, or the nutritional status. So it's only

one discrete variable. Indeed, that's probably why the tension time index is a little bit better—because it considers muscle function as well.

The role of this device, I think, in the chest wall issues is perhaps more important. I think perhaps we're not using esophageal pressure measurement as much as we should; there are a lot of obese and edematous patients, as you pointed out. With all this new emphasis to do compliance curves, pressure-volume curves, and plateau pressure measurements to protect the lung, this issue of chest wall compliance becomes, in my opinion, very important. So esophageal pressure might actually find more utility in that environment. And, you're right, they do not sell stand-alone systems today, but one ventilator manufacturer has it as a feature on one of their devices, so you don't necessarily have to go to eBay to get it.

Benditt: I do think that chest wall compliance is going to be very important. I get into little arguments with the ARDS Network folks when they're talking about plateau pressure levels less than 30 cm H_2O, and that is the *respiratory system* plateau pressure, not the *lung* plateau pressure. It's always bothered me a little bit that there was no clear evaluation of how much the chest wall was contributing to these pressures, and I can imagine a huge or very, very edematous ICU patient in whom the chest wall would make a big contribution but the lung is in good shape, or a little thin COPD [chronic obstructive pulmonary disease] patient who maybe has developed ARDS but in whom it may not be contributing, and I've always thought it would be great to get a balloon down those people so we could really measure the *lung* pressure, just that variable, not the total respiratory system pressure. I agree that it may be more useful.

Bigatello: Regarding the partition of lung and chest wall during the measurement of compliance, I think there is an important technical question. When you put in an esophageal balloon, or when you look at an occlusion pressure at end-expiration, you can measure transpulmonary pressure and use it for the measurement of compliance that way, by subtracting the pressure at the airway minus the esophageal pressure. However, as you have pointed out, the esophageal pressure measurements are not that reliable; measuring *changes* rather than absolute values of esophageal pressure is much more reliable. So is this the technique you use and do you think it is the correct one? Or would you rather do 2 measurements of esophageal pressures—sort of a chest wall chord compliance—and measure chest wall compliance that way, then calculate in reverse the lung compliance?

Benditt: That's a great point, and I think it underlines the fact that in uncooperative patients, when you can't do the dynamic occlusion method, generating an absolute value for esophageal pressure is difficult, and that is very important for chest wall compliance. In terms of using a sort of a "delta" [ie, change in esophageal pressure] and back-calculating, I haven't done that myself, and so I can't attest to its accuracy. But I can see the logic behind that.

Hess: How do you use the esophageal pressure, then, to correct the wedge pressure, if the esophageal pressure does not reflect the absolute pleural pressure?

Benditt: That's a big question. Basically, I try to ensure, as much as possible, the correct positioning of the catheters; I look for cardiac pulsations in the balloon. I'm the stingiest about filling it with only 0.5 mL, which I think is very important. Baydur et al[1] described the "dynamic occlusion

technique" for assessing the accuracy of the relationship between airway and esophageal pressure changes known, but that technique does not ensure that the absolute value is correct. So far there are no really good studies on how to predict juxtacardiac pressure from esophageal pressure in humans.

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Hess: So you're not taking a number and subtracting that from the wedge pressure?

Benditt: I'm not subtracting the esophageal pressure from the wedge pressure. I'm saying, yes, there's a very large, positive, integral, pleural pressure.

Hess: And what makes it even more confusing is that you're measuring it in cm H_2O, whereas the vascular pressure values are in mm Hg.

Benditt: Right. You have to correct for that.

Durbin: My talk will address some of these issues in heart/lung interactions. This was a nice lead-in to that. The questions Dean [Hess] asked I'd like to answer in 2 ways. First, the pressure is helpful in understanding the cardiovascular system effects only if you know the geometry and the size of the ventricle. It's really end-diastolic volume and geometry that we're interested in. Pressure is a surrogate (and a very poor one) for volume, so even having an accurate, corrected number for wedge pressure doesn't help you understand the cardiac physiology in many conditions. Second, there are methods for looking at pleural pressure distribution from the lung to the cardiovas-

cular system. If the thorax were considered homogeneous or not homogeneous, these effects could be considered in the model as well.

The cardiovascular system, which is complicated enough, with the corrections you've brought up, becomes even more complicated when placed inside a human being, where interactions with the nervous system and corrections are occurring continually. It probably isn't worth the effort to try to do what you're suggesting, other than to recognize where obvious errors do exist. It may be more important to look at the outcome of an intervention. For instance, the effect on cardiac output of giving a fluid bolus is more important than the change in wedge or corrected wedge pressure.

The clinical impact of these pressure interactions creates variations in heart volume that cause respiratory-induced systolic and diastolic blood pressure variations (so called "delta down" and "delta up"), which are reflected in direct arterial-pressure waveforms. These pressure-induced changes may actually be better indicators of an individual patient's responses to therapy.

Nilsestuen: I want to comment on the usefulness of having esophageal pressure waveforms. In all the articles I reviewed in preparation for the patient-ventilator-synchrony discussion, esophageal pressure was almost always used as the evaluative tool in clinical situations, to look at trigger function, inspiratory rise time, and termination criteria; so it has been very useful in the evaluation of patient-ventilator synchrony. It is unfortunate that esophageal pressure measurement is no longer commercially available, except in combination with the Avea ventilator [Viasys Healthcare, Conshohocken, Pennsylvania].

Hess: What about the use of respiratory variation and the central venous pressure as inflection of pleural pressure? Scott [Harris] and Luca [Bigatello] will tell you that that is sort of our "poor man's" way of looking at these things sometimes in the ICU.

Benditt: I'll leave that discussion to Dr Durbin.