Using Ventilator and Cardiovascular Graphics in the Patient Who Is Hemodynamically Unstable

Bryant A Murphy MD and Charles G Durbin Jr MD FAARC

The interaction of a mechanical ventilator and the human cardiovascular system is complex. One of the most important effects of positive-pressure ventilation (PPV) is that it can decrease venous return. PPV also alters right- and left-ventricular ejection. Increased lung volume increases right-ventricular size by increasing pulmonary vascular resistance, causing intraventricular cardiac-septum shift, and decreasing left-ventricular filling. Increased intrathoracic pressure reduces afterload on the LV and increases ejection of blood from the LV. Understanding and managing these complex and often opposing interactions in critically ill patients is facilitated by analysis of hemodynamic and ventilator waveforms at the bedside. The relationship of PPV to changes in the arterial pressure waveform gives important information regarding appropriate fluid and vasopressor treatment. This article focuses on effects of respiratory pressures on hemodynamics and considers how cardiac pressures can be transmitted to the airway and cause ventilator malfunction. *Key words: positive pressure ventilation, cardiac output, hemodynamics, systolic pressure variation, venous return, auto-triggering, arterial pressure.* [Respir Care 2005;50(2):262–273. © 2005 Daedalus Enterprises]

Introduction

Initiating or increasing positive-pressure ventilation (PPV) or positive end-expiratory pressure (PEEP) in un-stable patients frequently results in a dramatic fall in blood pressure, with ensuing organ ischemia or, in the most extreme case, cardiac arrest. This is primarily due to the profound influence that intrathoracic pressure has on venous return (VR) or preload of the right heart, leading to a fall in left heart output. On the other hand, patients who are fluid overloaded and in congestive heart failure often

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benefit markedly from PEEP or PPV and may dramatically improve following its application. This effect is mediated primarily by reduction of afterload on the left heart.

The heart and lungs are closely linked in the thorax and through the vascular system. The right and left heart are mechanically linked within a rigid pericardium. The heart, lungs, and their separate circulations influence each other in the normal healthy state in many complex ways. Under conditions of disease, the implications of these interactions are very far-reaching. Understanding the physiology of these interactions and initiating appropriate therapy is essential for safe treatment of an unstable, critically ill patient who requires and benefits from PPV. Waveform analysis, both measured from the airway and the vascular system, is an important part of the process leading to understanding and proper management of these complex interactions. This article will consider the basic physiologic principles of heart and lung interactions and how examination of waveforms can contribute to understanding and managing this complex system.

Venous Return and Intrathoracic Pressure Variation

Under most circumstances, blood flow into the right atrium is controlled by the pressure gradient between the venous system and the right atrium. The baseline venous driving pressure, called by physiologists the *mean systemic pressure* or the *static recoil pressure of the circulation*, can be measured in humans only during cardiac arrest (when the arterial and venous pressures are equal) or immediately following death. This pressure has been estimated at 6–10 mm Hg in normal humans, a value that has been confirmed in animal experiments. Control of the distribution of blood between the arterial system and venous compartments is the major way the body adjusts to instantaneous demands for increases in cardiac output (CO), which is driven by organ and tissue demands. Venous blood volume varies widely but averages 60–75% of the total blood volume, depending on the state of hydration, autonomic tone, and CO demands.

The equality of the amount of blood ejected from both the left ventricle (LV) and right ventricle (RV) and the VR is carefully maintained. Though small variations in stroke volume may occur between individual beats, under all circumstances, VR is exactly equal to CO when averaged over a few heart beats and over time. This must be so or blood would accumulate in the lungs, causing pulmonary edema, or in the periphery, causing shock. Changes in intrapleural pressure that occur during quiet, spontaneous breathing cause small oscillations in VR, which in turn cause cyclical changes in CO from each of the ventricles. PPV often causes larger fluctuations in intrapleural pressure, markedly affecting VR and CO. Though the major effects of PPV on cardiac function are mediated through

effects on VR, transmission of airway pressure to the cardiac chambers and large vessels in the chest induces additional and often opposite effects on ventricular output (see below). To analyze and understand the diverse and complicated interactions between heart and lung, we will consider the influence of mechanical ventilation and PEEP on the circulation as it affects several functions: filling of the right heart, ejection of blood from the RV into the pulmonary vessels, filling of the left heart, and ejection of blood from the left heart. We will also discuss the constraints placed on filling of the left heart by the right heart because both ventricles are contained within a fibrous, nondistensible pericardium. Finally, we will discuss how cardiac contractions can generate airway pressure changes that can affect mechanical ventilation.

Influence of PPV and PEEP on Venous Return

The major mechanical interactions between the respiratory and circulatory system are produced by pressure changes in the thorax (pleural pressure) and changes in lung volume. As mentioned above, the major influence of pleural pressure on cardiac function is mediated by its effect on VR. VR is the amount of blood flow over time into the right atrium. Clinically, VR cannot be measured directly, but is often inferred from venous and atrial pressures, and must be equal to CO.

A normal right atrial pressure (P_{RA}) is considered a surrogate for the adequacy of VR. P_{RA} is more correctly viewed as an estimate of preload of the RV (the volume of the RV just prior to contraction during systole). Actually, in a strict sense, preload really relates to the degree of stretch (or length) of the ventricular muscle fibers at the onset of ventricular contraction. Since fiber length is impossible to measure in intact humans, ventricular volume is often substituted for this measurement. Chamber measurements are also difficult to obtain clinically, and pressures are commonly used to represent size changes induced by fluid therapy. However, using a pressure to estimate the volume of a cardiac chamber is not correct, because the relationship between pressure and volume (compliance) is not a linear relationship and may change acutely and unpredictably. Additionally, changes in cardiac inotropy influence this compliance relationship, as does ischemia. That said, on a short-term basis, there is usually at least a directional relationship between VR and P_{RA}, such that a rise in P_{RA} indicates an increase in VR, and probably in RV chamber size. Conversely, a fall in P_{RA} is usually associated with a corresponding fall in VR. That directional correlation between VR and P_{RA} is clinically helpful during acute volume-replacement in hypovolemic patients, but P_{RA} is not a direct or absolute measure of VR. During cardiac relaxation (diastole), right atrial filling will occur only if the mean systemic pressure ex-
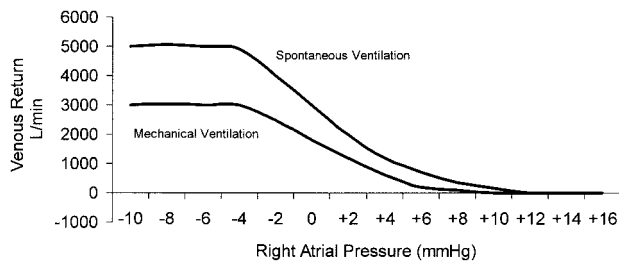
Fig. 1. Waveforms representing instantaneous measurements during a single cardiac cycle. Venous return is affected by the size of the vena cavae as well as by the right atrial pressure during cardiac filling. Positive-pressure ventilation reduces the maximum flow by its effect on cardiac output, and reduces the rate of atrial filling, because of intrathoracic caval compression. This functional obstruction causes atrial filling to cease at a lower atrial pressure.
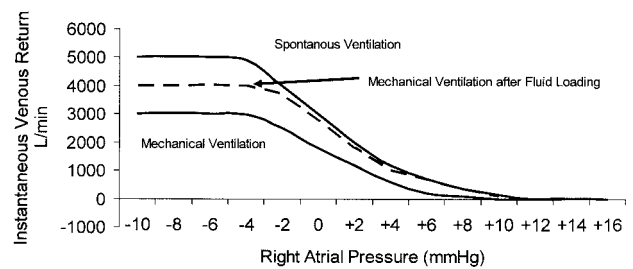

Fig. 2. Fluid loading restores some of the lost cardiac output and venous return by increasing the gradient from mean systemic pressure to right atrial pressure. Atrial filling through the compressed cavae is improved by this mechanism as well.

ceeds P_{RA}. The rate of blood flow into the right atrium is influenced by the pressure gradient between mean systemic pressure and P_{RA} and is not linear over the time during which atrial filling occurs. The complexity of the circulatory systems (systemic, pulmonary, venous) and their complex interactions make analysis of individual components impossible within intact animals. Most of what we know is derived by carefully controlling conditions in isolated animal preparations of a single part of a single system. Our understanding of factors that affect VR has been achieved on that basis, rather than by study of intact animals.

In addition to the upper limit placed on VR by P_{RA}, the vena cavae also offer resistance to flow into the thorax, creating an upper limit to the rate the right atrium can be filled. Under all conditions, filling of the right atrium ceases when P_{RA} equals mean systemic pressure. Maximum flow to the right atrium is limited by the caval size and collapsibility and can be reduced if the cavae are compressed by increased intrathoracic pressure. Figure 1 illustrates the complex relationship between caval size, VR, and P_{RA}. VR is constant over a wide range of P_{RA}, where the cavae flow is limited by mean systemic pressure and then flow falls linearly as P_{RA} rises. Under physiologic conditions, the constant-flow portion of this curve is controlled by the amount of blood available to return to the heart. During this part of the curve the cavae can be seen to intermittently collapse, as the pressure gradient between the right atrium and cavae does not affect blood flow. This upper limit of flow occurs because there is a limitation in available venous blood (ie, the CO from the left heart). Further reduction of P_{RA} causes collapse of the cavae, which identifies the limitation of flow. The down-sloped portion of the VR curve seen with increasing P_{RA} is a marker of the resistance imposed by the fully distended cavae. During this period the cavae are fully distended, there is no collapse, and filling is directly related to the pressure gradient between the right atrium and mean systemic pressure. The slope of this portion of the VR curve is related to the

imposed resistance or size of the cavae. In the presence of increased intrathoracic pressure the cavae are compressed, and both the maximum flow and the onset of the flow limitation on atrial filling is reduced.[1] This is illustrated in the lower curve in Figure 1. To some degree, this limitation to right-atrium filling can be overcome by an increase in venous pressure or mean systemic pressure.[2] This increase can occur with increased venous volume (volume infusion), increased venous tone (drug administration or increase in intrinsic autonomic tone), or increased abdominal pressure. The effect of increasing mean systemic pressure with volume infusion is illustrated in Figure 2, in which the fall in VR (and CO) is balanced by a higher pressure gradient and restoration of right-atrium filling.

Transmural Pressures

Up to this point in the discussion, we have considered only the effects PPV and PEEP on VR, mediated by effects on pressure gradients and caval size. However, airway pressures are transmitted to intrathoracic structures to a variable degree. Clinically, vascular pressures are usually referenced to atmospheric pressure, and the pressure surrounding the vascular structure is assumed to be atmospheric. During conditions of increased intrathoracic pressure, conventionally measured cardiac chamber pressures will reflect some of the intrathoracic pressure, so the transmural pressure (the pressure across the wall) will be lower than the measured pressure. What this means is that the chamber size at the same measured pressure will be smaller during PPV, and thus the effectiveness of ventricular contraction (the Frank-Starling mechanism) will be less. Echocardiography has confirmed this change in the pressure-volume relationship induced by increased intrathoracic pressure. Though using echocardiography might overcome the problem of interpretation of the pressure transmission problem during PPV, it is impractical to continuously measure chamber filling with this method, and vascular pressures remain the usual method to estimate cardiac filling during clinical care.

Measurement of esophageal pressure can be helpful in estimating the contribution of intrathoracic pressure to reducing transmural pressure. Though esophageal pressure measurement is helpful in estimating the average increase in vascular pressure due to transmitted airway pressure during PPV and PEEP (or decreased during negative-pressure ventilation), the distribution of that pressure is not uniform throughout the thorax, and regional differences can make large differences in specific chamber or vessel effects that cannot be easily identified.[3] In general, the less compliant the lung, the less the pressure is transmitted.

Effect of PPV and PEEP on RV Ejection

Though increased pleural pressure from PPV will decrease right-atrium filling by reducing VR, the increased thoracic pressure will facilitate RV output by reducing pulmonary vascular resistance (PVR) and by directly compressing the RV by lung expansion during inhalation. These effects counteract the reduction in RV-filling and help explain why most patients do not suffer severe cardiac compromise when initiating PPV (if they are not hypovolemic). Cardiac output, specifically that from the RV, is less reduced than would be expected from the reduction of VR caused by the rise in intrathoracic pressure.[4] Another important effect of PPV on right-heart function is its effect on PVR. When the lung is inflated from a collapsed state, PVR declines because of unkinking of large pulmonary vessels. At a point somewhere above functional residual capacity, PVR reaches a nadir and then begins to rise because of overstretching of alveolar capillaries in apical lung regions (West's zone 1) and forcing of blood into dependent regions, increasing vascular pressures (West's zone 3). These changes leading to initially reducing PVR and then increasing PVR during lung inflation modulate the effects of reduced VR on RV output. Cyclic changes in RV output directly influence LV filling during ventilation.

These complicated relationships during PPV were investigated by Theres et al, with instrumented, lightly anesthetized pigs.[5] They measured instantaneous inferior venacaval flow (representing VR) and pulmonary artery blood flow (RV cardiac output [RVCO]) as well as transmural chamber pressures in the animals. They averaged the measured values during many cardiac cycles and correlated the average values to the respiratory cycle. As was expected, they found that a fall in VR preceded the fall in RVCO. Figure 3 shows the pressure and flow changes during a ventilatory cycle. The changes in VR were coincident with changes in airway pressure. A slight recovery in VR begins before the breath is terminated, when airway pressure falls slightly from its peak. Those animals had normal lungs, and it is expected that airway pressure changes would be substantially transmitted to the thoracic cavity. RVCO is maintained after the fall in VR because of RV
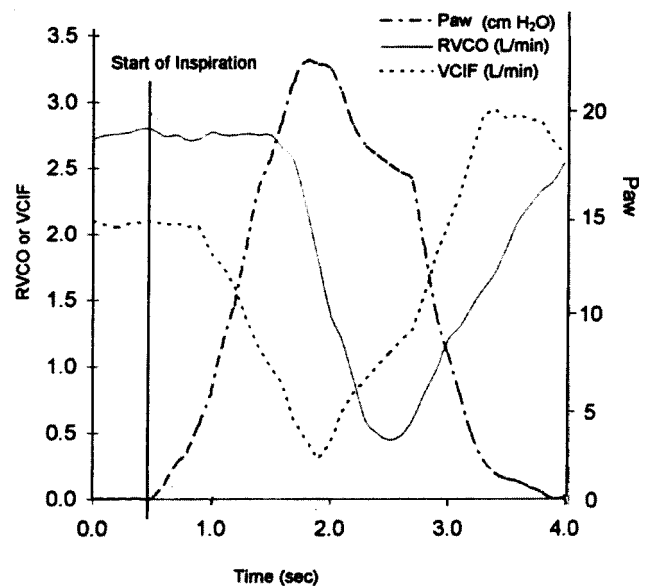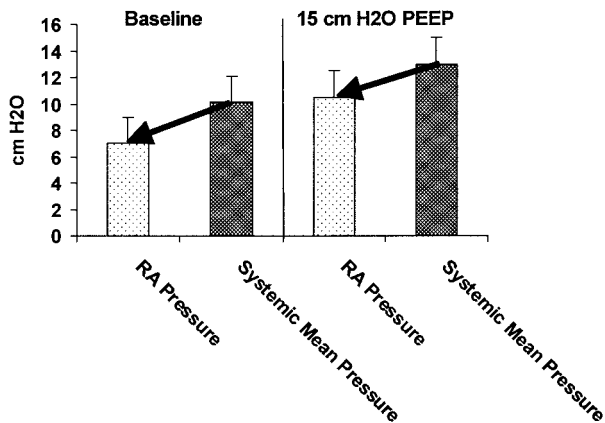

Fig. 3. Phase-related changes in venacaval inflow (VCIF) and right ventricular cardiac output (RVCO) during mechanical ventilation in lightly anesthetized instrumented pigs. Shortly after inspiration begins, vena caval flow falls linearly as airway pressure rises. Shortly after the fall in venous return begins, a fall in RVCO occurs, which reaches a nadir shortly after airway pressure begins to fall in early exhalation. Venous return rises above baseline as exhalation continues. This phasic change in venous return is identical and reciprocal to the changes in airway pressure, which is presumably transmitted directly to the thoracic cavity. P_{aw} = airway pressure. (From Reference 5, with permission.)

compression and reduction in PVR. However, this is not sustained throughout the ventilatory cycle, and RVCO falls during continued lung expansion. On exhalation, VR overshoots baseline because of a rise in mean systemic pressure during obstructed thoracic inflow. RVCO recovery is delayed behind VR recovery, reflecting the delay necessary to fill the atrium, then the ventricle. With that same model, Theres et al found that adding PEEP caused an absolute fall in VR and CO, but the timing of the cyclical changes during each breath was identical to those during mechanical ventilation without PEEP. RV transmural pressures were maintained unchanged with fluid infusion during the experiment, but ventricular size and geometry were not assessed.

The effect of PEEP on the driving pressure for VR was investigated in humans during testing of implantable defibrillation devices.[6] The gradient between P_{RA} and mean systemic pressure was measured. Airway and vascular pressures were monitored; mean systemic pressure was determined 7.5 seconds following induced fibrillation and corrected for the flow that would have occurred with complete equilibration between arterial and venous systems in the absence of changes in venous tone. These measurements were repeated during apnea in 14 patients, with PEEP of

Fig. 4. Changes in right atrial and systemic mean pressure with applied positive end-expiratory pressure (PEEP) of 15 cm H_2O. The arrows indicate the gradient of right-ventricular filling under each condition. RA = right atrial. (Adapted from data in Reference 6.)

zero and PEEP of 15 cm H_2O. Echocardiographic analysis was used in several patients and a decrease in LV stroke volume and CO of 23% was seen with the application of PEEP. Right atrial pressure rose with PEEP, as did the mean systemic pressure. The gradient between P_{RA} and mean systemic pressure was maintained unchanged during the application of PEEP in this experiment (Fig. 4). The patients were hydrated but not acutely fluid-loaded during the experiment. They had relatively normal pulmonary compliance, and transmural pressures were not calculated. The explanation and interpretation of that data is: PEEP raises measured P_{RA} but does not increase transmural pressure and actually reduces atrial and ventricular size. PEEP also causes an instantaneous rise in mean systemic pressure, probably due to abdominal visceral vein compression, preserving the pressure gradient for VR to the right atrium. However, CO is reduced because of the effect of PEEP on RV function and filling. It would be helpful to know if maintaining the transmural pressure during PEEP application would restore the lost CO, but this has not been tested. The bottom line from this experiment is that mean systemic pressure rises with PEEP and PPV, and this counteracts some of the expected fall in VR due to increased thoracic pressure. This protective mechanism explains why only a few patients, usually those who are hypovolemic, suffer important cardiac compromise during the application of PEEP.

In summary, as far as ventilation and cardiac chamber pressures are concerned, VR changes instantaneously with P_{RA} changes during the cardiac cycle as well as with changes interposed by thoracic pressure during breathing with PEEP. With spontaneous breathing, inspiration is associated with an initial increase in venous flow, which continuously falls

as P_{RA} rises. Flow ceases when P_{RA} reaches the driving pressure, mean systemic pressure. During PPV, vena caval compression may additionally limit VR at all levels of P_{RA}. However, VR will increase with an increase in intraabdominal pressure, which will increase venous pressure as well as venous flow. This occurs with PPV because of compression of the abdominal viscera by diaphragm descent, as well as from the backup of venous blood caused by the decreased outflow from the abdominal veins. Thus, the expected large fall of VR and CO from PPV (or PEEP) is less than would occur if intrathoracic pressure alone were elevated.

The interrelationship between lung volume, P_{O_2}, CO, and PVR is even more complicated. The state of venous oxygenation directly influences pulmonary vascular tone. Arterial hypoxemia, which leads to venous desaturation, profoundly increases PVR. Alveolar hypoxemia also causes vasoconstriction. On a local basis, this mechanism is effective at redirecting blood to better-ventilated areas of lung and improving ventilation/perfusion matching—so-called hypoxic pulmonary vasoconstriction. Each of these effects may profoundly influence RV ejection during mechanical ventilation and the contribution of each is impossible to assess in any individual case.

In summary, during PPV, RV output is reduced because of a reduction in VR from a restriction of RV filling and a functional reduction in blood flow through the venae cavae into the chest. RV ejection is facilitated early during lung inflation, because of reduction in RV afterload and by direct compression of the RV from the expanding lungs surrounding the heart. At peak inflation, PVR may be elevated and RV ejection is inhibited.[7] During exhalation, most of these effects are reversed: VR recovers (and overshoots) and total CO is close to baseline without PPV. The effect of PEEP on RV output is similar to PPV, except the effects extend throughout the respiratory cycle and do not recede during exhalation.

Effects of PPV and PEEP on LV Function

PEEP and PPV can improve patients with pulmonary edema. Up to this point, we have discussed the reduction in CO from reduced VR due to PPV and PEEP. In the face of fluid overload, this effect can improve lung edema by reducing an elevated VR and atrial pressure, redistributing alveolar fluid, increasing pulmonary compliance, and reducing the work of breathing. In addition, an important effect of PEEP and PPV is facilitating LV ejection.[8]

The RV output directly influences left atrial and LV filling and output. Averaged over a few heart beats, the outputs from both right and left heart must be identical. The intrathoracic pressure effects described above affect the left heart through effects on the right heart. In addition, transmural effects on the LV and thoracic aorta contribute

to altering LV stroke output. These pressure effects consist of reducing the size of the LV, by compression of the chamber by lung expansion and compressing and reducing the size of the aorta and reducing aortic transmural pressure. The first effect makes difficult the interpretation of LV filling pressure during PPV and PEEP and affects the apparent relationship of vascular pressure to chamber size; that is, it creates an apparent change in compliance of the ventricle. Unlike the RV, the LV is a muscular chamber that is designed to eject blood against a high pressure and afterload. PPV and PEEP reduce the LV afterload by compressing the aorta, facilitating ventricular emptying, and increasing stroke volume, at the same degree of filling. Since LV filling is reduced by the RV reduced filling, this afterload reduction helps maintain CO during PPV and PEEP.

In addition to these direct effects, in an intact human there are autonomic nervous system responses that tend to maintain CO to meet the tissue needs under all circumstances, including institution of PPV. The primary response to an inadequate CO is an increase in heart rate. This is the most rapid and effective way to restore CO. The beat-to-beat variation in heart rate is a measure of autonomic (parasympathetic) activity and health. Beat-to-beat heart rate variation is a normal response to the instantaneous and cyclical differences in RV and LV stroke output. Later responses to decreased CO include increasing venous tone, which raises mean systemic pressure, which increases VR, and diversion of blood from less important organs such as kidneys, gut, and skin to preserve flow to vital organs, including the brain and heart. Hormonal responses include secretion of adrenal hormones, cortisol, and aldosterone; these hormones affect salt and water conservation in the kidney and gut, causing blood volume expansion over a longer period. Another important salt-influencing hormone is released from the cardiac atria: atrial natriuretic peptide. This hormone causes sodium to be excreted from the kidney and thus acts as an aldosterone antagonist in the case of fluid overload in the face of heart failure with atrial distention. Right atrium distention may occur with PPV because of the increased PVR that can occur. High levels of natriuretic peptides may be a marker of RV failure due to PPV, PEEP, or pulmonary hypertension of any cause.[9,10]

PPV and LV Filling Pressure Estimation

Adequacy of LV filling is usually estimated clinically by measuring pulmonary artery occlusion pressure (PAOP), using a Swan-Ganz catheter. PPV and PEEP affect the accuracy and interpretation of this vascular pressure measurement. Because of the partial and unpredictable transmission of airway pressures, wedge pressure is measured at end-exhalation when airway pressure influence is least. If a no-flow situation can be obtained and no extrinsic

PEEP has been applied, the measurement of wedge pressure is considered "accurate." During spontaneous ventilation, wedge pressure and LV end-diastolic volume have a predictable relationship (in the absence of mitral valve disease or primary pulmonary hypertension). However, the effect of PEEP (added and intrinsic) may still be present at end-exhalation during PPV. Correction of the wedge pressure by measuring and subtracting esophageal pressure can be done with an esophageal pressure-monitoring device. The popularity of such measurements has waned, and even with that measurement, it must be recognized that pleural pressure is not transmitted uniformly throughout the chest, making direct subtraction from the wedge pressure suspect. Below we will consider 3 other approaches that have been suggested to estimate the "true" PAOP when PEEP is present and an esophageal balloon is not used: (1) measuring the wedge pressure after disconnection from the ventilator circuit, (2) determining a pressure-transmission ratio and using it to correct the wedge pressure, and (3) if the PEEP exceeds 10 cm H_2O, using central venous pressure to estimate LV end-diastolic volume.

Disconnecting the patient from the ventilator should remove the effects of both PPV and PEEP from the thorax and allow a "true" PAOP to be determined.[11] The technique suggested is to make the measurement from a strip-chart recording at end-exhalation, 2–5 seconds after disconnection from the circuit. If intrinsic PEEP is present, 2–5 seconds may be inadequate; some patients may require as long as 15–20 seconds for the intrinsic PEEP to dissipate. In that case, pharmacologic paralysis would be necessary to achieve apnea. Ventilator flow-time waveforms should allow detection of intrinsic PEEP and predict in which patients ventilator-disconnection will not provide an accurate PAOP measurement. An argument against using the disconnect technique is that the physiologic changes that occur during the measurement compromise the patient's gas exchange, and even if a "true" PAOP off ventilation is determined, it is cardiac filling on the ventilator that needs to be assessed. The first argument can be overcome by calculating an airway pressure transmission ratio and correcting the PAOP obtained during ventilation by that ratio.[12] This correlates well with the disconnect technique in patients without intrinsic PEEP, and does not require interruption of applied airway pressure.

The airway transmission ratio is determined by measuring the change in airway pressure during a tidal breath and the respiratory variation in PAOP. The measurements are performed with the catheter in wedged position and over several ventilator breaths. From ventilator waveforms the change in airway pressure is calculated as plateau pressure minus pressure at end-exhalation. The respiratory induced wedge pressure change is calculated as the maximum mean PAOP minus the minimum mean PAOP during a ventilator breath. The transmission ratio is the respiratory change
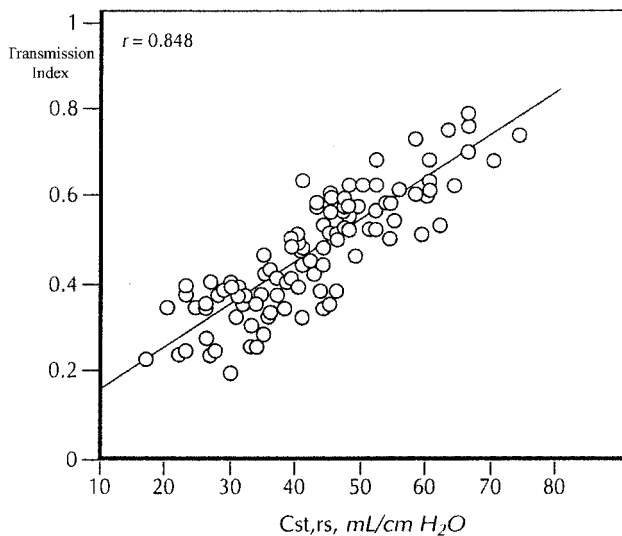
Fig. 5. Pulmonary compliance is the major predictor of the transmission coefficient for pleural pressure. $C_{st,rs}$ = static compliance of the respiratory system. (From Reference 13, with permission.)

in wedge pressure divided by the change in airway pressure. The "true" PAOP pressure is:

$$P_{end-ex} \times (1 - PT_{PAOP} \times PAOP)$$

in which P_{end-ex} is the pressure at end-exhalation, and PT_{PAOP} is the PAOP pressure-transmission-ratio. The transmission ratio is not dependent on tidal volume and correlates directly with pulmonary compliance. As expected, more compliant lungs transmit more of the applied airway pressure to the vascular space. This is illustrated in Figure 5, in which pulmonary compliance and the transmission ratio are plotted for many different patients.[13] Though the "corrected" PAOP can be calculated, the relationship between that number and actual chamber size remains unpredictable and the "correct" chamber size for optimum cardiac function in the face of elevated thoracic pressures is not known.

P_{RA} or central venous pressure may be a more accurate indicator of LV end-diastolic volume if PEEP of ≥ 10 cm H_2O is used. This was demonstrated in a classic study performed by Jardin et al, in which ventricular catheters were placed in patients with acute respiratory distress syndrome and the effects of fluid loading and increasing PEEP were investigated.[7] As seen in Figure 6, there was a linear relationship between LV end-diastolic volume and central venous pressure, but not between LV end-diastolic volume and wedge pressure.

In summary, wedge pressure is often used to estimate LV end-diastolic volume. Determination of a true wedge pressure is difficult because of unpredictable airway pressure-transmission to vascular structures. Several techniques

have been suggested to minimize or correct for those effects. Measurement of end-exhalation pressure is standard and is facilitated by using ventilator waveforms to properly identify the appropriate measurement point in the respiratory cycle. However, the complicated nature of the interactions between the ventilator, lung, and heart make difficult the application of even the "true" or corrected wedge pressure to a clinical situation. The basic assumption that there is a predictable relationship between LV end-diastolic volume to LV volume is altered by PPV and PEEP in complex ways.

Effect of Ventricular Interdependence During PPV With PEEP

Since the right and left heart are contained within a relatively rigid pericardium, changes in the shape and size of one ventricle will change the size and shape of the other ventricle. This is very important in cases of right-heart volume-overload, where the interventricular septum may shift to the left as the RV becomes more spherical and intrudes on the LV. The effect of this intrusion or septal shift is 2-fold: (1) the LV chamber size at the same pressure (LV end-diastolic volume) will be smaller and stroke output less, and (2) the normally spherical shape of the LV will be distorted, and contraction less efficient.[14] The combined effects of the intrusion are that LV apparent compliance and contractility are reduced. The echocardiographic study by Jardin et al dramatically demonstrated septal shift from increasing PEEP and RV overload.[15] Since the RV is not a pressure pumping chamber, dilation occurs with modest increases in afterload increase due to increased PVR.

Arterial Systolic Pressure Variation During Mechanical Ventilation

Putting together all the factors discussed above, it is apparent that ventilation induces alterations in ventricular output between heart beats, varying periodically during the respiratory cycle. Some effects of PPV increase ventricular output, but most effects tend to reduce ventricular filling or output by reducing VR and changing ventricular geometry. Even without mechanical ventilation, there is a cyclical variation in stroke output from the left heart related to the changes in intrathoracic pressures. When an arterial catheter is in place, this changing stroke output can be seen in arterial pressure that fluctuates periodically and coincident with the respiratory cycle. Compared to spontaneous ventilation, mechanical ventilation changes the phase of the arterial pressure changes, because inspiration is associated with a fall in arterial pressure during spontaneous inhalation, and with a rise during mechanically applied breaths. The magnitude of the arterial pressure changes has been suggested as a clinical way of assessing
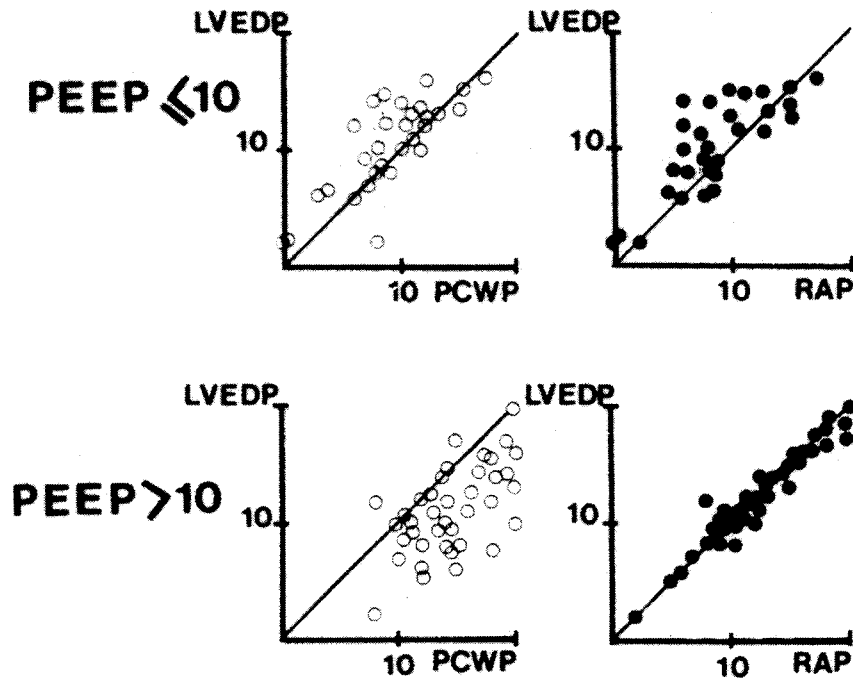
Fig. 6. If positive end-expiratory pressure (PEEP) is > 10 cm H_2O, right atrial pressure (RAP) is a more accurate reflection of left-ventricular end-diastolic pressure (LVEDP) than is pulmonary artery occlusion pressure. PCWP = pulmonary-capillary wedge pressure. (From Reference 15, with permission.)
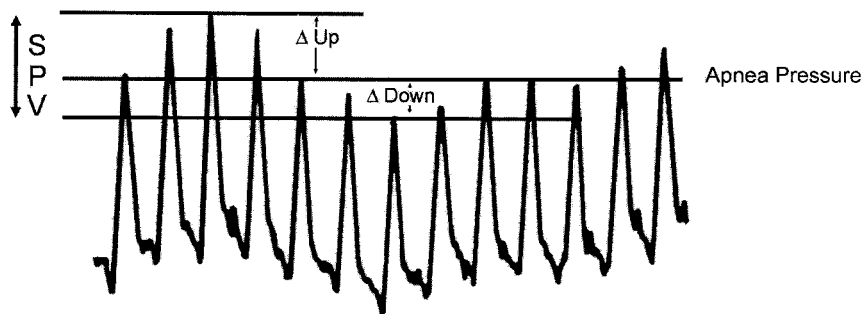

Fig. 7. A radial-artery pressure waveform that shows how systolic pressure variation (SPV) is divided into an increase in blood pressure during inhalation (during positive-pressure ventilation), followed by a decrease in blood pressure as inhalation continues and exhalation occurs. These are conventionally called "delta up" (the rise from apnea pressure during ventilation) and "delta down" (the decrease from apnea pressure to the lowest point in the respiratory cycle).

whether VR is adequate during PPV. Also, it has been suggested that the change in arterial pressure variation occurring during volume infusion could be used as a method to determine when fluid administration has had its maximum effect in restoring reduced CO due to PPV.

The systolic pressure variation (SPV) during mechanical ventilation can be divided into 2 components: the upward component during early inhalation and the downward component that follows inhalation and occurs later. These components are termed the delta up and delta down (Fig. 7). The magnitude of delta down is related to the magnitude of reduction in VR produced by PPV. Observation of changes in the degree of delta down and SPV is often used clinically as an indicator of volume status during fluid administration. A few issues about this approach should be understood. Because the effects of PPV on LV stroke volume are complex and include more than just reduction of VR, restoration of VR with volume infusion will not completely ameliorate the fall in stroke output and CO that occurs with PPV and PEEP. Thus, the SPV will not return completely to normal with volume expansion. Probably the most important consideration in understanding the problems with interpreting the SPV is that most of the effects of PPV and PEEP on CO are mediated through
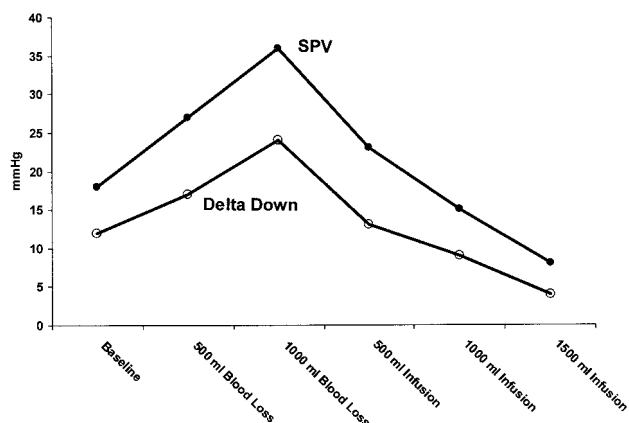
Fig. 8. Systolic pressure variation (SPV) and delta down (see Fig. 7) in a patient undergoing sequential blood removal (graded hemorrhage) and reinfusion of fluid during volume-controlled mechanical ventilation. The magnitude of the change is identical with the 2 variables. (Adapted from data in Reference 20.)
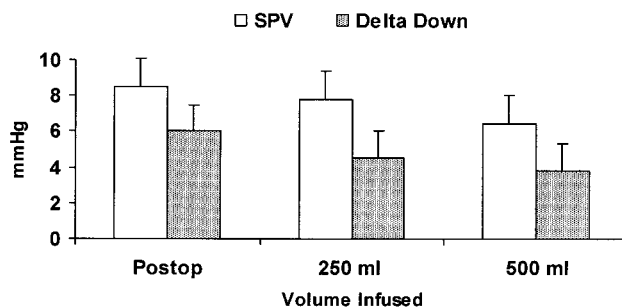

Fig. 9. A decrease in systolic pressure variation (SPV) and delta down (see Fig. 7) occurred in patients undergoing volume infusion following aortic surgery. (Adapted from data in Reference 21.)

Table 1. Hemodynamic Response to Volume Loading Among Septic Patients*

Variable	Responders (mean ± SD)	Nonresponders (mean ± SD)	p
PAOP (mm Hg)	10 ± 4	12 ± 3	0.10
Diastolic area index (cm^2/m^2)	9.1 ± 2.9	12.3 ± 3.5	0.005
SPV (mmHg)	15.4 ± 4	8 ± 3	0.0001
delta down	11 ± 4	4 ± 2	0.0001

*Among septic patients, a positive hemodynamic response to volume loading is best predicted by systolic pressure variation (SPV) and delta down (see Fig. 7).
PAOP = pulmonary artery occlusion pressure
(Data from Reference 22.)

transmission of airway pressure to the pleural space.[13,16,17] Since pleural pressure is not usually measured, and the degree of transmission of airway pressure is not known, factors such as tidal volume, lung compliance, breathing pattern, heart rate and rhythm, autonomic activity,[18] and administered medications will all affect the magnitude of the SPV and delta down.[19]

An interesting study by Rooke et al of surgical patients undergoing hemodilution (a technique to minimize blood transfusion, during which there is intentional removal of blood at the beginning of a major operation and reinfusion after surgical blood loss is controlled) demonstrated a close correlation between increased SPV and delta down induced by volume-controlled ventilation during controlled hemorrhage.[20] That change reverted during volume replacement (Fig. 8). Rooke et al concluded that an SPV of ≤ 5 mm Hg and a delta down of ≤ 2 mm Hg predicted adequate intravascular volume restoration. They also noted that there was considerable individual variation in SPV and that during spontaneous ventilation the SPV was much reduced and changes did not correlate with volume changes.

Another study of SPV and delta down suggested that surgery patients exhibiting a large SPV or delta down were more likely to have reduced LV end-diastolic volume and to respond to fluids with increased cardiac filling and output. Coriat et al demonstrated that this was true in patients following aortic surgery.[21] They studied 21 patients with transesophageal echocardiography, thermodilution CO measurements, and radial artery catheters. SPV and delta down were compared with other measures of preload, and the accepted standard, LV end-diastolic volume calculated from the echocardiograms during PPV (10–12 mL/kg, 10–14 breaths/min, using volume-controlled ventilation with no PEEP). Fluid bolus administration with 5% albu-

min was used to create a cardiac pressure-volume curve. The best predictor of LV end-diastolic volume for the entire group of measurements was the delta down, which had a correlation coefficient of 0.83. SPV was also a very good predictor, and surprisingly, there was no predictable relationship between PAOP and LV end-diastolic volume. During volume loading there was a direct correlation between SPV and delta down and volume change (Fig. 9). Tavernier et al[22] demonstrated the value of monitoring SPV and delta down to optimize septic patients. Using a combination of techniques, including echocardiography, they demonstrated excellent prediction of volume responsiveness between SPV and delta down and CO, stroke volume, and LV end-diastolic volume estimate. Using a cutoff value of 5 mm Hg for delta down produced the best prediction model of fluid bolus responders and nonresponders who were septic. The delta down was much better than the end-diastolic area index or PAOP for predicting which patients would or would not respond to 500-mL volume infusion (Table 1). Figure 10 shows the response of a typical patient to volume infusions.

In the absence of an indwelling arterial catheter, the plethysmogram determined by the pulse oximeter may offer a noninvasive method of determining SPV.[23] The specific technology and amplification techniques used affect

Fig. 11. Receiver operating characteristics for delta down (see Fig. 7), left ventricular (LV) end-diastolic area index, and pulmonary artery occlusion pressure (PAOP) as predictors of fluid responsiveness. (Adapted from data in Reference 22.)
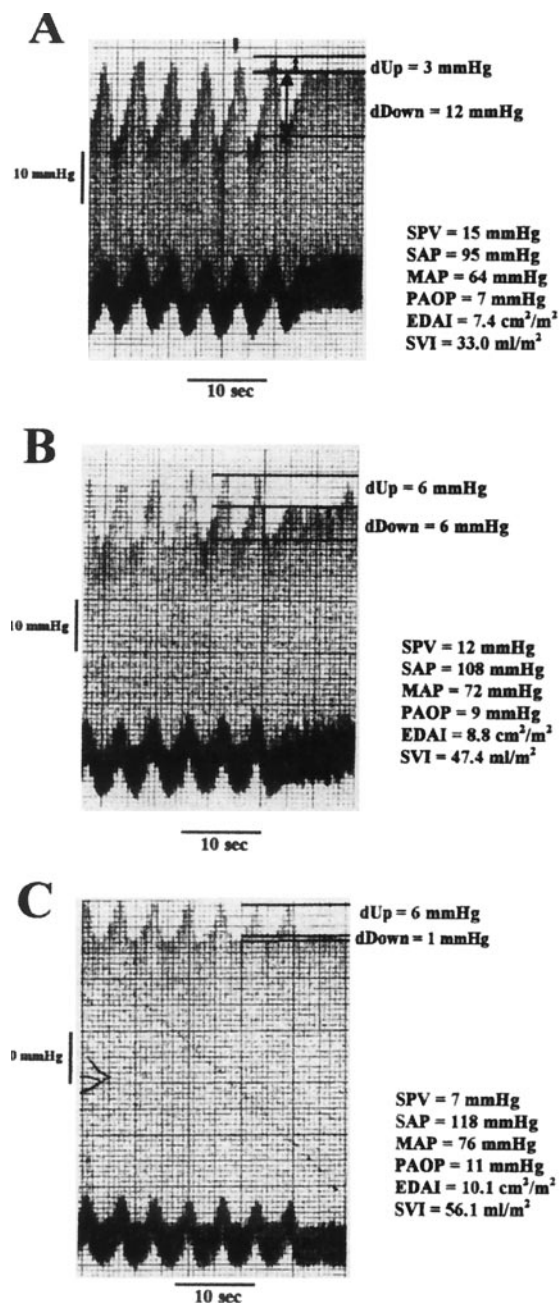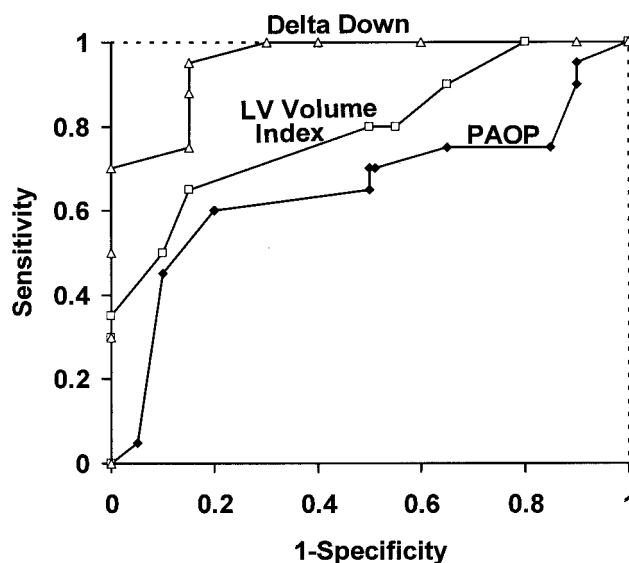

Fig. 10. Arterial pressure waveforms from the resuscitation of a typical patient who is septic, hypotensive, and receiving fluids. A: Prior to beginning resuscitation. B: After some volume has been infused. C: After fluid resuscitation is complete. The reduction in systolic pressure variation (SPV), delta up and delta down (see Fig. 7), increase in blood pressure, cardiac chamber size, stroke index, and pulmonary-artery occlusion pressure (PAOP) are reported in each panel as the resuscitation proceeds. dDown = delta down. SAP = systolic arterial pressure. MAP = mean arterial pressure. EDAI = end-diastolic area index. SVI = stroke volume index. (From Reference 22, with permission.)

the variation observed. Clinical evaluation of this approach requires additional study.[24,25]

The studies described above, and other optimistic reports[26–28] that suggested the utility of monitoring SPV and delta down for managing volume administration in critically ill patients were followed by reports of failure of this approach to be helpful.[29–31] The complexity of effects of changes in intrathoracic pressure described earlier probably account for the variety of results reported. In clinically applying the effects of mechanical ventilation on the SPV and delta down, it is important to remember that PPV has important effects on VR, RV and LV afterload, direct ventricular compression, and ventricular interdependence. It is not surprising that consideration of just one measurement (eg, delta down) for management of a hemodynamically unstable patient will often prove inadequate. As a composite variable, however, SPV or delta down integrates many of these changes and has proven superior to other measurements, including PAOP (Fig. 11).

Cardiac Pressures Are Transmitted to the Airway

Most of our discussion about heart-lung interactions so far has related to the effects of PPV on cardiovascular function. The opposite is occasionally true: cardiac oscillations can affect respiratory function. This is most often an issue during patient-triggered ventilation. Ventilator auto-triggering can occur when the trigger sensitivity is too low and random movements of the patient or the airway circuit may trigger a ventilator-supported breath, even though the patient's respiratory muscles are not contracting. That

situation is usually easily identified by careful patient observation and inspection of airway pressure and flow waveforms. However, the margin between auto-triggering and minimizing the work necessary to trigger a breath is small, especially when the patient's efforts are minuscule. With the advent of even more sensitive flow-triggering, the risk of unrecognized auto-triggering has increased.

We reported a case of a brain-dead patient maintaining normal gas exchange for a period of 4 hours while receiving pressure support ventilation with no backup rate.[32] We believe this was due to breaths being triggered by cardiac contractions that caused gas flow sufficient to trigger the ventilator (a Puritan Bennett 840 using flow-triggering, initially set at a sensitivity of 0.5 L/min). Even when the sensitivity was reduced to 2 L/min, occasional cardiac triggering continued. This and similar cases in our institution caused us to abandon the most sensitive trigger settings when ventilating patients who may be brain-dead or who have severe muscle weakness.

Imanaka et al investigated the factors that predispose to cardiac oscillations causing cardiac triggering.[33] They studied post-cardiac-surgery patients who were given neuromuscular blockers sufficient to suppress all voluntary movement, including diaphragm movement. Despite being totally paralyzed, 20% of these patients experienced auto-triggering. Though the sensitivity of the ventilator was important, both pressure-triggered and flow-triggered modes exhibited auto-triggering. Several patients triggered at pressure sensitivity greater than 2 cm H_2O and flow sensitivity of 4 L/min. The pressure and flow waveforms clearly showed that cardiac oscillations were causing important waves (Fig. 12). Not every cardiac-induced pulsation triggered a breath, and it is not clear why some were sensed and others ignored.

All the ventilators and all the partial-support modes tested demonstrated cardiac triggering in some patients. In evaluating patient factors that may lead to cardiac triggering, Imanaka et al compared those that auto-triggered to those that did not. The groups differed in several important ways. The cardiac-triggering group had slightly higher LV and RV filling pressures, higher cardiac indices, and lower systemic resistance. Arterial and pulmonary pressures were the same in the 2 groups. Considering pulmonary factors, the cardiac-triggering group had higher pulmonary compliance but identical airflow resistance. The auto-triggering group also had higher thoracic time constants. The combination of a hyperdynamic circulation and a long respiratory time constant seems to predict cardiac triggering. Neither pressure nor flow triggering is immune from cardiac triggering, and conventional levels of trigger sensitivity did not prevent cardiac triggering in patients who had the cardiac and respiratory risk factors for auto-triggering. Thus, with current-generation ventilators in partial support modes, clinicians must use caution when evaluat-
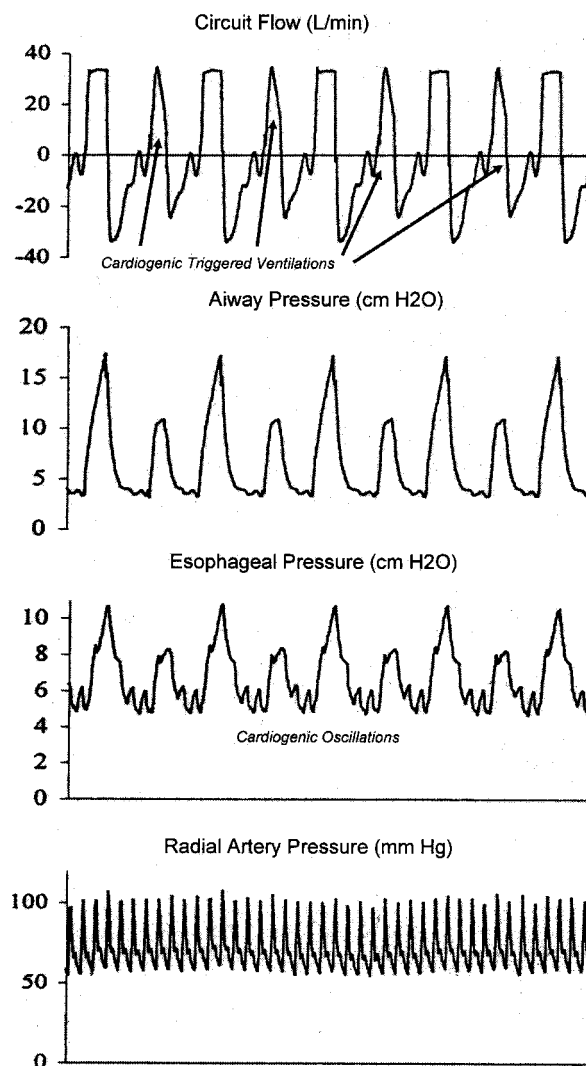

Fig. 12. Cardiac contractions triggering intermittent ventilations in a paralyzed patient. (Adapted from data in Reference 33.)

ing for spontaneous breathing. This is especially important for documenting absence of breathing in brain-dead patients being considered for organ donation.

Summary

Most of the effects of mechanical ventilation and PEEP on cardiac function are mediated through changes in intrathoracic pressure. Reduction in VR is an important effect of raised intrathoracic pressure, which accounts for a marked reduction in cardiac output. Other less important effects that reduce cardiac output during mechanical ventilation are: reduced RV stroke output due to increased pulmonary vascular resistance; increased size of the RV; decreased size of the LV because of encroachment of the enlarged RV with septal shift; and reduced LV filling due to reduced RV stroke volume.

PPV directly compresses the RV, reduces the afterload of the LV, and helps restore some of the lost CO. Because some of the applied airway pressure is directly transmitted to the vascular structures, actual transmural cardiac chamber pressures are difficult to estimate. The combined effects of airway pressure on cardiac chambers and stroke output can be estimated by observing the changes in systemic arterial pressure during mechanical ventilation. A normal delta down (with a reasonable ventilation pressure) predicts adequate fluid status. Occasionally, cardiac pressure auto-triggers the ventilator; this is more likely with a patient who has a hyperdynamic heart and normal lungs.

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Discussion

Benditt: Pepe and Marini[1] found that blood pressure decreased with intrinsic PEEP, but we've always thought that it was decreased venous return. Do you still think that that's the explanation for hypotension with intrinsic PEEP in ventilated patients?

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Durbin: I think we don't see hypotension more frequently because intrinsic PEEP increases abdominal pressure, which augments venous return to some degree. But I still think reduced venous return is the primary mechanism of the hypotension.

Campbell: Clinically, when you are manipulating PEEP and you're curious as to the effect on hemodynamics, we know that the point that we want to get to is volume. You didn't mention anything about some of the newer hemodynamic monitors that measure volume and may be more immune to some of those airway pressure manipulations. Are you currently using those, or do you have any opinion about the use of measuring volume as opposed to measuring pressure?

Durbin: Are you referring to intrathoracic blood volume measurements using electrical impedance, or do you mean using a transthoracic Doppler?

Campbell: Like end-diastolic volume. I think Edwards has an end-diastolic volume index on one of their hemodynamic monitors.

Durbin: I don't have any first-hand experience with it. I think it makes intrinsic sense that it would be closer to the actual number that you're interested in, but I suspect it won't be any more helpful than some of the simpler tools.

Blanch: I have a practical question. Hemodynamics are very important when patients with ARDS [acute respiratory distress syndrome] present with acute right heart failure. Therefore, what tool would you use to monitor those patients?

Durbin: I advocate the Swan-Ganz catheter, because I don't have confidence in any of the other tools. I will occasionally use the echocardiogram to confirm or refute, at one point in time, the relationships between pressure and volume, but the Swan-Ganz catheter can continuously measure cardiac output. It's possible that we could replace the Swan-Ganz with a noninvasive technique, but cardiac output and volume loading are the 2 things that I use to manipulate the hemodynamics in the ARDS patient. I'm waiting for publication of the study on the high-volume/low-volume treatment of ARDS patients, to know where I ought to set my limits on fluids, but I'm still probably going to be looking at filling pressures and cardiac output as my end points, especially with a patient who's critically ill.

The other half of your question was whether I use other techniques such as the echocardiogram. Currently, the echocardiogram isn't a continuous monitor; it's useful only for intermittent measurement of cardiac chamber size and contractility estimation. I know there are some people who will leave the echo probe in the patient and repeat the cardiac evaluation frequently. It's interesting how quickly the wedge pressure or the cardiac output changes and how little change you see in the echocardiograph. That is probably because of changes in heart rate.

The stroke volume is probably a more consistent measure to look at cardiac function. Stroke volume times heart rate is cardiac output. Unfortunately, stroke volume is more difficult to determine with echocardiography. Global filling and function is not too hard to evaluate, but actual stroke volume is less reliably estimated.

So, with a really sick patient, I put in the echocardiographic probe (transesophageally), take a measurement, calibrate the wedge pressure to the chamber size, do an intervention, and then repeat the echocardiogram. I continue to follow cardiac output more or less continuously with the Swan-Ganz catheter.

Benditt: I'm fascinated by the idea of the thorax kind of pumping the abdomen. In patients with spinal cord injuries we use an abdominal binder frequently, and I'm starting to wonder whether maybe part of the blood pressure change that we see with the abdominal binder may not be secondary to this effect of almost like pumping the liver blood. An interesting idea. Maybe we should be using abdominal binders for hypotension.

Durbin: I point out that the changes with PEEP are a little different than those with positive-pressure ventilation. With intermittent positive-pressure ventilation there truly is an abdominal pumping action. With PEEP the effect seen in one model was to acutely raise the mean systemic pressure. Applying PEEP decreases cardiac output. Cardiac output may or may not decrease with positive-pressure ventilation, but it certainly does decrease with PEEP. That PEEP decrease is not entirely restored with simple volume loading, and it certainly isn't restored by compressing the abdomen. In the old days we did similar things to improve blood pressure by raising the patient's legs, trying to augment venous return. Initially, it looks like you're doing something useful, but after 5–6 minutes you find out you're really not, and blood pressure falls back to baseline. So I'm not sure if there is a role for abdominal compression. I know it's more important to achieving stability than I initially thought, but I don't know what role it should have clinically.