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Central Venous and Bladder Pressure Reflect Transdiaphragmatic Pressure During Pressure Support Ventilation*

Sarah Chieveley-Williams, MD; Lila Dinner, MD; Anna Puddicombe, RGN; Debbie Field, Msc; A. T. Lovell, MD; and John C. Goldstone, MD

Study objectives: To determine whether the change in bladder pressure (Pblad) and central venous pressure (Pcvp) may reflect the changes in esophageal pressure (Pes) and gastric pressure (Pgas) when inspiratory pressure support (IPS) is altered.

Design: Prospective clinical study.

Setting: The ICUs of a teaching hospital.

Patients: Ten patients currently receiving IPS ventilation via a tracheostomy or an endotracheal tube who already had bladder and central venous catheters in situ.

Measurements and results: Airway pressure, Pes, Pgas, Pcvp, Pblad, and flow were measured at the original IPS setting. IPS then was reduced by 5-cm H2O increments until IPS was zero or was at the minimum pressure that could be tolerated by each patient. At each level of IPS, pressures and flow were measured at steady-state breathing. The maximum pressure difference for each pressure during inspiration was calculated. We found that the \( \Delta P_{\text{blad}} \) correlated closely with the \( \Delta P_{\text{gas}} \) \( r = 0.904 \) and that the \( \Delta P_{\text{es}} \) correlated with the \( \Delta P_{\text{cvp}} \) \( r = 0.951 \). When the \( \Delta P_{\text{cvp}} - \Delta P_{\text{blad}} \) was compared with the transdiaphragmatic pressure for each patient as the IPS was altered, the correlation coefficients varied from 0.952 to 0.999.

Conclusion: Although absolute values for the \( P_{\text{cvp}} \) during mechanical ventilation do not always reflect the \( P_{\text{es}} \), useful information can be obtained from this route. In individual patients, the two sites of measurement followed each other when IPS was changed, enabling a bedside assessment of the response to reducing respiratory support.

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Key words: central venous pressure; esophageal pressure; mechanical ventilation; monitoring of respiratory muscle function; transdiaphragmatic pressure

Abbreviations: IPS = inspiratory pressure support; Paw = airway pressure; Pblad = bladder pressure; Pcvp = central venous pressure; Pdi = transdiaphragmatic pressure; Pes = esophageal pressure; Pgas = gastric pressure; Ppl = pleural pressure

Although respiratory muscle function and strength are important, monitoring of the diaphragm is invasive and is performed infrequently in critically ill patients. During inspiration, the diaphragm descends, raising intra-abdominal pressure and reducing intrathoracic pressure.

To quantify diaphragmatic strength, the pressure difference across the diaphragm is measured from the addition of gastric pressure (Pgas) and esophageal pressure (Pes), and termed transdiaphragmatic pressure (Pdi). While Pes and Pgas are the standard methods of measuring Pdi, pressure changes during respiration are transmitted to other structures in the mediastinum and abdomen.

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Fleming and colleagues measured central venous pressure (Pcvp) and Pes in healthy volunteers during inspiratory efforts against a closed airway. They concluded that measurements of Pcvp reflect pleural pressure (Ppl) changes and found that the ratio of the ΔPcvp to the ΔPes was close to unity. The use of intravesical pressure as a measure of intra-abdominal pressure has been investigated by other research groups, who have validated it against Pes and abdominal pressure. Intravesical pressure was shown to be an accurate reflection of both directly measured intra-abdominal pressure and intragastic pressure.

During inspiratory pressure support (IPS) ventilation, the respiratory muscles contract and initiate ventilation. When IPS is maximal, little effort is required, and when IPS is reduced, respiratory muscle effort is increased. We have studied patients receiving IPS to test whether Pcvp and bladder pressure (Pblad) reflect Pes and Pgas, and to investigate whether useful information can be obtained from the ΔPblad – ΔPcvp. Furthermore, we have investigated whether the ΔPblad – ΔPcvp can monitor changes when IPS is altered.

**Materials and Methods**

Prior to the study, written informed consent was obtained from the patient for a protocol that had been approved previously by the institutional review committee. Ten patients were enrolled into the study, comprising 6 male and 4 female patients. All patients were recovering from critical illnesses and were then being weaned from mechanical ventilation, which entailed daily assessments during periods of breathing through a simple circuit. Table 1 provides a summary of the demographic information for the patients.

At the time of the study, all patients were breathing spontaneously with IPS ventilation via a cuffed oral endotracheal tube or a cuffed tracheostomy. All patients had both a bladder catheter and a central venous catheter in situ as part of their medical care. The central venous catheter entered either the internal jugular or subclavian vein with the distal end located radiographically in the superior vena cava.

<table>
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<th>Subject</th>
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</thead>
<tbody>
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<td>F</td>
<td>COPD, laparotomy</td>
</tr>
<tr>
<td>2</td>
<td>47</td>
<td>F</td>
<td>Intracerebral bleed, aspiration pneumonia</td>
</tr>
<tr>
<td>3</td>
<td>28</td>
<td>M</td>
<td>Pancreatitis</td>
</tr>
<tr>
<td>4</td>
<td>66</td>
<td>M</td>
<td>Coronary artery bypass grafts, ARDS</td>
</tr>
<tr>
<td>5</td>
<td>61</td>
<td>M</td>
<td>Abdominal sepsis, fascitis of the umbilicus</td>
</tr>
<tr>
<td>6</td>
<td>65</td>
<td>M</td>
<td>Coronary artery bypass grafts</td>
</tr>
<tr>
<td>7</td>
<td>55</td>
<td>F</td>
<td>Pancreatitis</td>
</tr>
<tr>
<td>8</td>
<td>68</td>
<td>M</td>
<td>COPD</td>
</tr>
<tr>
<td>9</td>
<td>68</td>
<td>M</td>
<td>Coronary artery bypass grafts, ARDS</td>
</tr>
<tr>
<td>10</td>
<td>78</td>
<td>F</td>
<td>Pneumonia</td>
</tr>
</tbody>
</table>

*F = female; M = male.

**Measurements**

Airway pressure (Paw), Pes, Pcvp, Pgas, and Pblad, as well as inspiratory flow at the airway, were measured for each patient. Airflow was measured with a heated pneumotachograph (Hans Rudolph; Kansas City, MO), and air volume was obtained from the integrated flow signal.

Pes and Pgas were measured using 10-cm air-filled balloon catheters (P.K. Morgan; Kent, UK) that were passed transnasally into the patient under local anesthesia. Two milliliters of air was injected into the gastric balloon, and 0.5 mL of air were passed into the esophageal balloon. The correct placement of the esophageal catheter was confirmed according to the method described by Baydur et al. Each catheter was connected to a differential pressure transducer (Medex Medical; Lancaster, UK).

Pcvp was measured from the distal port of a triple-lumen central venous catheter (Arrow; London, UK) connected to a differential pressure transducer (Medex Medical) that was filled with a 0.9% saline solution. The Pblad was measured by draining the bladder and instilling 50 mL sterile 0.9% saline solution to ensure a fluid-filled system, as described by Collee et al. A 21-gauge needle was inserted through the catheter sampling membrane, and the Pblad was transduced using a 0.9% saline solution-filled differential pressure transducer (Medex Medical).

Pressure and flow were calibrated prior to each study using a water manometer and an air flowmeter (Platon; London, UK). All measurements were recorded on a microprocessor system (Macintosh; Apple; Cupertino, CA). The signals were recorded at 100 Hz, were stored, and were analyzed using virtual instruments developed within a software package (MacLab, version 3.6.1; ADInstruments; East Sussex, UK).

**Protocol**

Before commencing recordings, the patient was made comfortable in a semi-recumbent position of approximately 45°, and secretions were cleared from the airway to prevent coughing during data collection.

A 1-min recording of all data was made at the original level of IPS. The level of IPS then was reduced by 5-cm H2O decrements, and 1-min recordings were made at each level of IPS until the patient was breathing with only 5 cm H2O positive end-expiratory pressure. At each level of IPS, recordings were made after 10 to 20 min to allow a period of equilibration. During the protocol, each patient was observed for signs of respiratory distress (ie, high respiratory rate, fall in oxygen saturation, or cardiac arrhythmia). If these conditions developed, the study would be terminated and the patient would be returned to the original level of ventilatory support.

**Statistical Analysis**

Prior to analysis, the Pcvp signal was averaged because the cardiac component of the Pcvp added noise to the respiratory waveforms.

We used the onset of inspiration, which was defined as the first negative deflection in the flow signal, to act as a trigger for a software signal averager. Because the heart rate is independent of the respiratory rate, the effect of averaging ± 30 breaths together is that the respiratory signal is reinforced while the cardiac signal is reduced.

The maximal changes in Pgas, Pblad, Pes, and Pcvp during inspiration (between the points of zero flow) at each level of pressure support were measured. Pdi (ie, the ΔPgas – ΔPes) and the ΔPblad – ΔPcvp then were calculated for each patient at each level of IPS.

Pdi and ΔPblad – ΔPcvp were compared using the method..
described by Bland and Altman and were correlated for each individual at varying levels of pressure support. The results are presented as the mean difference ± SD for both methods of measurement.

**RESULTS**

During the study protocol, no patient developed signs of severe respiratory distress. Table 2 shows the changes in values for Pes, Pcvp, Pgas, and Pblad for each patient at the minimum level of pressure support tolerated, together with the ratio of the ΔPes to the ΔPcvp and the ΔPgas to the ΔPblad. The ΔPes/ΔPcvp ratio varied between 0.8 and 2.1 and the ΔPgas/ΔPblad ratio varied between 0.6 and 1.3 at the minimum level of pressure support.

As pressure support was reduced, we calculated the slope of the ΔPes to the ΔPcvp for each patient. The mean slope of the ΔPes to the ΔPcvp was 1.1, ranging from 0.8 to 1.8 for individual patients. The correlation coefficient for each patient varied from 0.775 to 0.998. A Bland-Altman plot of the ΔPes and the ΔPcvp shows that the mean difference between the measurements is −1.0 cm H₂O (SD, 4.1 cm H₂O) and that the limits of agreement range from 7.1 to −9.1 cm H₂O.

Pgas and Pblad changes also were compared as ventilatory support was reduced. The slope of the line of the ΔPblad to the ΔPgas varied from 0.4 to 1.3 for individual patients. The correlation coefficient of the ΔPblad to the ΔPgas during IPS reduction for each patient varied from 0.877 to 0.987. When compared as a Bland-Altman plot, the mean difference between the ΔPgas and the ΔPblad was −0.4 cm H₂O (SD, 0.9 cm H₂O).

Table 3 shows Pdi (ie, ΔPgas − ΔPes) and ΔPblad − ΔPcvp, together with the Pdi/ΔPblad − ΔPcvp ratio for each patient at the minimum level of IPS tolerated. Figure 1 shows Pdi and ΔPblad − ΔPcvp plotted for each patient as pressure support was varied. The slope of the line of Pdi to ΔPblad − ΔPcvp was 1.05 for the entire group, varying from 0.4 to 1.1 for individual patients. However, in all patients the relationship between Pdi and ΔPblad − ΔPcvp was linear, with correlation coefficients varying from 0.906 to 0.999 for each individual (Table 4). When Pdi and ΔPblad − ΔPcvp were compared using a Bland-Altman plot, the mean difference between the two measurements was −0.2 cm H₂O (SD, 4.0 cm H₂O), giving limits of agreement from −8.2 to 7.8 cm H₂O.

Figure 2 shows how Pdi and ΔPblad − ΔPcvp, the values for which were normalized as the percent change from baseline values at the lowest levels of IPS, varied for each patient as IPS was changed. Except at very low levels of pressure support, Pdi,

### Table 2—Comparison Between ΔPes/ΔPcvp Ratio and ΔPgas/ΔPblad Ratio at Minimum Tolerated IPS

<table>
<thead>
<tr>
<th>Subject</th>
<th>ΔPcvp, cm H₂O</th>
<th>ΔPes, cm H₂O</th>
<th>ΔPgas, cm H₂O</th>
<th>ΔPblad, cm H₂O</th>
<th>ΔPgas/ΔPblad Ratio</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>−7.0</td>
<td>−7.1</td>
<td>1.01</td>
<td>3.9</td>
<td>6.5</td>
</tr>
<tr>
<td>2</td>
<td>−9.2</td>
<td>−15.6</td>
<td>1.7</td>
<td>5.8</td>
<td>4.2</td>
</tr>
<tr>
<td>3</td>
<td>−32.1</td>
<td>−34.8</td>
<td>1.08</td>
<td>6.2</td>
<td>6.6</td>
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<tr>
<td>4</td>
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<tr>
<td>5</td>
<td>−6.48</td>
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<td>3.4</td>
<td>3.7</td>
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<tr>
<td>6</td>
<td>−12.9</td>
<td>−12.4</td>
<td>0.96</td>
<td>12.1</td>
<td>12.2</td>
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<tr>
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<td>1.8</td>
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<td>−11.9</td>
<td>0.71</td>
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### Table 3—Pdi and ΔPblad − ΔPcvp for All Subjects at Minimum Tolerated IPS

<table>
<thead>
<tr>
<th>Subject</th>
<th>Pdi, cm H₂O</th>
<th>ΔPblad − ΔPcvp, cm H₂O</th>
<th>Pdi/ΔPblad − ΔPcvp Ratio</th>
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<td>11.0</td>
<td>13.5</td>
<td>0.81</td>
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<td>21.4</td>
<td>13.3</td>
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<tr>
<td>3</td>
<td>41.0</td>
<td>38.7</td>
<td>1.06</td>
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<tr>
<td>4</td>
<td>12.8</td>
<td>14.3</td>
<td>0.90</td>
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<tr>
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<td>11.2</td>
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<td>1.10</td>
</tr>
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<td>6</td>
<td>24.5</td>
<td>25.1</td>
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<tr>
<td>7</td>
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<td>31.9</td>
<td>1.03</td>
</tr>
<tr>
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<td>1.96</td>
</tr>
<tr>
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<td>44.2</td>
<td>53.3</td>
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</tr>
<tr>
<td>10</td>
<td>12.9</td>
<td>17.9</td>
<td>0.72</td>
</tr>
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</table>
when measured by both methods, was reduced as ventilatory support increased, and in all cases ΔPblad – ΔPcvp mirrored the changes induced in Pdi.

**DISCUSSION**

In the present study, changes in Pcvp are easily detected during mechanical ventilation and vary from a positive deflection when the respiratory muscles are completely inactive to pronounced negative swings during large inspiratory efforts. In all cases during this study, the negative deflections were greatest at the lowest levels of pressure support and increased in size as pressure support was reduced. The ΔPcvp reflected the ΔPes, and when the data are normalized the agreement is striking.

This finding may be useful at the bedside, as many patients receiving pressure support have continuous Pcvp monitoring, and it is easy to detect the impact of reducing pressure support in these patients. For example, when IPS was reduced in patient 7, the pressure swing increased fivefold, indicating a substantial increase in respiratory muscle effort. Pdi increased simultaneously from 7.8 to 32.8 cm H₂O. Furthermore, the pattern of minimal respiratory effort can be detected either when the ΔPcvp is positive or minimally negative. In these cases, pressure support can be reduced and the response noted.

The agreement between the two sites of measuring transpleural pressure was in some cases not accurate enough to use the two measurements interchangeably. While all the patients with high or low pressure swings could be identified from the ΔPcvp, subtle differences could not be detected in all cases.

There are several reasons that might contribute to the lack of concordance between the measurements of the ΔPes and the ΔPcvp. First, Pcvp was measured with a fluid-filled catheter system in comparison to the air-filled esophageal catheter system. While it is accepted that fluid-filled systems have better frequency responses than air-filled systems, Asher and colleagues did not demonstrate large differences when air-filled and fluid-filled systems were compared. This, therefore, would account for only very minor differences between Pes and Pcvp.

The relative positions of the esophageal and central venous catheters may be of more significance. Milic-Emili and coworkers compared Pes and Paw at a variety of catheter positions within the esophagus. When close to the diaphragm, Pes reflected transpulmonary pressure accurately. As the catheter was withdrawn, there was a greater variability between Pes and Paw. The position of our esophageal catheters was confirmed by the method described by Baydur et al and was in the middle third of the esophagus. In every case, the central venous catheter tip was more cephalad than the esophageal balloon, and this may account for some of the lack of concordance between the two measurements. However, such an effect, if constant, would still allow valuable trend data to be obtained from the Pcvp within an individual.

In the patients who demonstrated the extremes of the ΔPes/ΔPcvp ratio, their underlying pathologies may have contributed to the discrepancy between the two measurements. In both adults and neonates Pes is augmented at high lung volumes by the effect of excess gas decompression, resulting in Pes overestimating Ppl. In subject 8, who had COPD, the ΔPes/ΔPcvp ratio was 2.14. It is probable that this patient had significant air-trapping and an increased functional residual capacity. Therefore, Pes may be augmented, increasing the ΔPes/ΔPcvp ratio during dynamic breathing. Conversely, in patients with consolidated or wet lungs (and therefore reduced lung volumes), Pes may be an underestimate of Ppl. The resultant ΔPes/ΔPcvp ratio may be less than unity, as demonstrated by subjects 9 and 10, who had ratios of 0.78 and 0.71, respectively. These two effects may be cancelled by dual pathology. For example, subject 1 had COPD and had undergone a laparotomy, with the first condition increasing lung volume and the latter decreasing it. The resultant ΔPes/ΔPcvp ratio was 1.01.

While the absolute ΔPes might not equal the ΔPcvp in every case, trends in ΔPcvp can be compared usefully with ΔPes, as indicated by the correlation coefficient of the ΔPes/ΔPcvp ratio when IPS is adjusted.

In 1965, Comroe suggested that an intrathoracic vein with its thin wall is capable of transmitting intrapleural pressure and might therefore be an acceptable alternative to the esophagus for transpulmonary pressure measurement. Femelae et al studied...
ied healthy adults using three identical fluid-filled systems to record mouth pressure, Pes, and Pcvp. They found that in most instances valid measurements of Ppl could be obtained from Pes or Pcvp. An x-y plot of the ΔPes/ΔPcvp ratio fell close to the line of identity.

**Figure 2.** The percentage change in Pdi and ΔPblad − ΔPcvp with changing IPS for each patient.
Walling and Savege\textsuperscript{12} suggested that Pcvp was more reliable than Pes in reflecting a change in Ppl in the anesthetized supine patient. Pes was found to be marginally greater than Pcvp, a finding confirmed in our study by most patients. They hypothesized that this was due to the weight of the mediastinal contents on the esophagus in the supine position. However, in their study, as in ours, dissimilar catheter systems were used, the central venous catheter system being fluid-filled and the esophageal system being air-filled.

We tested the frequency response of the fluid-filled Pcvp system \textit{in vitro} prior to the study and found it to be satisfactory at 13.5 Hz compared to 15 Hz for the air-filled system. However, the patients studied had central venous lines that had been \textit{in situ} for several days, and it is possible that the frequency response characteristics of the system were altered by thrombus, drug crystals in the line, or the catheter lying next to the vessel wall.

Removing the cardiac waveform from the Pcvp trace was straightforward using time synchronization, with clear waves related to inspiration visible on all traces. Although there was greater overall variability between Pgas and Pblad values in our study, this had only a minor effect during the calculation of Pdi since intrathoracic pressure changes made a much greater contribution to the end result. The study by Collee et al\textsuperscript{2} used identical fluid-filled systems for both the stomach and bladder. Their results were similar to ours, with Pgas changes being approximately 2.5 cm H$_2$O above or below intravesical pressure.

In conclusion, Pblad and Pcvp provided reflections of respiratory muscle effort when pressure support was reduced. We suggest that the change in Pcvp might be used as a guide to diaphragmatic contraction during IPS reduction, thus providing a rapid assessment when an esophageal balloon catheter is not \textit{in situ}. The $\Delta$Pblad/$\Delta$Pcvp ratio may be a useful additional assessment and warrants further investigation during the ventilation-weaning process.

\section*{References}
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