Influence of increased abdominal pressure on steady-state cardiac performance

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Influence of increased abdominal pressure on steady-state cardiac performance. J. Appl. Physiol. 86(5): 1651–1656, 1999.—The effect of steady-state increases in abdominal pressure (Pab) on cardiac performance was studied in seven acutely instrumented swine with pneumoperitoneum (PP). The animal was placed on volume-preset ventilation, and PP was created by air insufflation. Cardiac output (CO), right atrial (Pra), left atrial (Pla), pericardial (Ppe), and abdominal inferior vena cava pressures (Pivc) were measured while Pab was increased from baseline to 7.5, 15, and 30 mmHg (PP7.5, PP15, and PP30, respectively). Cardiac function curves of the right and left ventricle (RV and LV, respectively) were compared between baseline and PP30. CO presented biphasic changes, with an initial slight increase at PP7.5 followed by a fall at PP30. A significant discrepancy was observed between Pra and Pivc at PP15 and PP30, consistent with development of a “vascular waterfall.” Transmural Pla (Pla – Ppe) showed parallel changes with CO, whereas transmural Pra (Pra – Ppe) exhibited a sustained increase. The RV cardiac-function curve was more depressed than was that of the LV at PP30; this suggests an increased RV afterload produced by the elevated airway pressure. These results support the hypothesis that our previously proposed concept of abdominal vascular zone conditions (M. Takata, R. A. Wise, and J. L. Robotham. J. Appl. Physiol. 69: 1961–1972, 1990) is also applicable to steady-state hemodynamic analyses. The abdominal zones appear to play an important role in determining CO, with increases in Pab, by modulating systemic venous return and the LV preload. Simultaneous measurements of Pra and Pivc may provide useful information in the hemodynamic care of patients with elevated Pab.

abdominal vascular zone conditions; vascular waterfall; pneumoperitoneum; cardiac output

INCREASES IN ABDOMINAL PRESSURE (Pab) are encountered in a variety of clinical situations, including intra-abdominal or retroperitoneal hemorrhage, bowel obstruction, massive ascites, or use of antishock shrouders. Due to the recent introduction of laparoscopic surgery and intervention, clinicians must now manage on a daily basis patients with elevated Pab. However, the effects of increase in Pab on hemodynamics, particularly its mechanical influence on cardiac performance, have not been fully understood. Although it is generally accepted that cardiac output (CO) decreases at high Pab (2, 4, 5, 11–13, 15, 18, 22), namely >20 mmHg, the effects of lower Pab are controversial. Some investigators found an augmentation of CO at moderately increased Pab (5, 13, 14, 33), whereas others found a consistent decrease (2, 4, 11, 12, 15, 22). A coherent physiological explanation for the diversity of these observations is lacking.

In an attempt to characterize the mechanical effects of increased Pab on venous return, we previously proposed a concept of “abdominal vascular zone conditions” (31), analogous to pulmonary vascular zone conditions (32). Increases in Pab would enhance inferior vena cava (IVC) venous return when right atrial pressure (Pra) significantly exceeded Pab (zone III abdomen) but would reduce IVC venous return when Pra was below Pab (zone II abdomen) due to the collapse of abdominal IVC and the development of a vascular waterfall. These principles were experimentally confirmed in dogs with transient increases in Pab during respiratory maneuvers (28, 30). Because systemic venous return and ventricular preload are the major determinants of CO under conditions with a normally functioning heart (8, 24), the abdominal zone concept may also be critical to define cardiac performance with steady-state increases in Pab. The present study tests the validity of this concept in a steady-state circulatory system and characterizes the mechanical effects of prolonged increases in Pab on ventricular loading and CO by use of an acutely instrumented pig model with pneumoperitoneum.

METHODS

Animal preparation. The study protocol was approved by the Institutional Animal Care and Use Committee. Seven swine, weighing 9–11 kg, were anesthetized with an intramuscular injection of ketamine hydrochloride (20 mg/kg) followed by intraperitoneal injection of pentobarbital sodium (20 mg/kg). A 6.0-mm-ID endotracheal tube was inserted via a tracheostomy and sutured in place. The pigs were ventilated with a time-cycled ventilator (model E 200; Newport Medical Instruments, Newport Beach, CA) at an inspiratory oxygen fraction of 1.0, tidal volume of 10 ml/kg, and positive end-expiratory pressure of 3 cmH2O. Respiratory rate was adjusted to obtain a baseline arterial PCO2 of 30–40 Torr, and minute ventilation was maintained at this rate for the remainder of the trial. Subsequent doses of pentobarbital sodium were given, as needed, and the animals were kept paralyzed with continuous infusion of pancuronium bromide (0.3–0.4 mg·kg−1·h−1). Body temperature was maintained at 37–39°C with the use of a heating pad.

Fluid-filled catheters were placed in the left carotid artery and right atrium via the right external jugular vein to monitor mean arterial pressure (MAP) and Pra. Another fluid-filled catheter was introduced via a femoral vein into the...
IVC and was positioned 3–4 cm below the diaphragm to measure pressure in the abdominal IVC (Pivc). A double-lumen catheter was placed in the left external jugular vein for intravenous infusion and administration of drugs. After a median sternotomy, taking great care not to open the pleural space, an ultrasound transit-time flow probe (8- or 10-mm S series, Transonic Systems, Ithaca, NY) was placed around the root of ascending aorta for measurement of CO. To minimize the errors in flow measurements caused by changes in the cross-sectional area of the aorta or in probe-vessel alignment, we sewed the flow probe to the adventitia of the aorta with 5-0 sutures at the four corners of the probe window (30). A custom-made, air-filled, flat latex balloon (20 × 20 mm) was placed in the pericardial space over the anterolateral wall of the left ventricle (LV) for measurement of pericardial pressure (Ppe). Silk suture loops that were 5-mm long were attached to three corners of the balloon before insertion, and these loops were stitched to the epicardium with 5-0 silk sutures. The operating air volume for the balloon was adjusted to the optimal volume determined by in vitro assessment of the elastic characteristics of the balloon. The fabrication and validation of the flat balloon were described in detail previously (29). A fluid-filled catheter was directly inserted into the left atrium to monitor left atrial pressure (Pla), and the pericardium and sternum were reapproximated. An air-filled, 5-cm, cylindrical latex balloon was placed between the loops of the small intestine within the abdominal cavity, via a small incision in the lower right abdomen, to measure Pab. The unstressed volume of the balloon was 3 ml, and an operating air volume of 0.5 ml was used. A large-bore plastic cannula was inserted in the abdominal cavity for air insufflation, and the abdomen was closed airtight. Airway pressure (Paw) was measured at the connection of the endotracheal tube.

Fluid-filled catheters and air-filled balloons were connected to strain-gauge transducers (model CDX III, Cobe Laboratories, Arvada, CO) and amplifier units (model 2238, San-ei, Tokyo, Japan). The zero reference was obtained at the level of the middle right atrium. Heart rate (HR) was calculated by a tachometer unit (model 1321, San-ei). All pressures and flows were continuously recorded on a microcomputer-based data-acquisition system (MacLab, ADInstruments, Tokyo, Japan).

Series 1: Hemodynamic effects of increased Pab. The plasma-volume status of each animal was controlled by infusion of 5% dextran to maintain Pra between 5 and 6 mmHg before the experiment. Pneumoperitoneum (PP) was created by air insufflation to avoid the effects of carbon dioxide on hemodynamics. Pab was raised stepwise from baseline to 7.5, 15, and 30 mmHg (PP 7.5, PP 15, and PP30, respectively). Hemodynamic variables, including CO, MAP, Pra, Pivc, Pla, Ppe, and Paw, were recorded under each condition. In each step, 10 min were allowed to permit achievement of a quasi-steady state, and the mean values for the following 30 s were used for analyses. The transmural Pra and Pla (Ptram and Platm, respectively), indexes of the preload of the right ventricle (RV) and LV, respectively, were calculated by subtracting Ppe from Pra and Pla, respectively (Ptram = Pra – Ppe, Platm = Pla – Ppe). Statistical comparisons were performed by one-way ANOVA for repeated measures with Scheffé’s test.

Series 2: Effects of increased Pab on cardiac-function curves. The plasma-volume status of each animal was standardized before the experiment, as in series 1. Under baseline Pab conditions, hemodynamic data to generate ventricular-function curves were obtained by drawing 4 ml/kg of blood from the right atrium three times. An adjustment period of 3 min was allowed for stabilization between each step. The blood was returned to the animal, and Pab was increased to 30 mmHg by air insufflation (PP 30). If the CO did not reach 80% of the initial CO (CO0.8) under the baseline Pab condition, infusion of dextran was added as necessary. Hemodynamic data to generate ventricular-function curves at PP 30 were then obtained by an identical procedure (Fig. 1A). MAP, HR, and Paw were recorded just before blood was drawn, both at baseline and PP 30. RV and LV cardiac-function curves at baseline and PP30 were constructed by plotting CO against Pratm and Platm, respectively. As described by Marini et al. (17), data from each animal were fitted to a second-degree polynomial expression as

\[
RV \quad CO = a_1Pratm^2 + b_1Pratm + c_1
\]

\[
LV \quad CO = a_2Platm^2 + b_2Platm + c_2
\]
where \(a\), \(b\), and \(c\) are constants. With these equations, the values of \(P_{Ra_m}\) and \(P_{La_m}\), which correspond to 80, 70, and 60% of the initial \(CO\) at baseline (\(CO_{0.8}\), \(CO_{0.7}\), and \(CO_{0.6}\), respectively), were mathematically estimated in each animal at baseline and at PP30. An example of this analysis is shown in Fig. 1B. The calculated values of \(P_{Ra_m}\) and \(P_{La_m}\), used to make one composite cardiac-function curve from the data of seven animals. The differences in \(P_{Ra_m}\) and \(P_{La_m}\) between baseline and PP30 (\(\Delta P_{Ra_m}\) and \(\Delta P_{La_m}\)) were calculated at \(CO_{0.8}\), \(CO_{0.7}\), and \(CO_{0.6}\) to quantify the degree of rightward shift of cardiac-function curves with increases in Pab. Statistical analyses were performed by one-way ANOVA with Scheffe’s test.

**RESULTS**

**Series 1.** With incremental increases in Pab, \(CO\) presented biphasic changes in all the animals studied, i.e., an initial rise followed by a fall (Table 1). \(CO\) tended to increase at PP7.5, returned toward baseline at PP15, and showed a statistically significant decrease below baseline at PP30 (\(P < 0.01\)). Both \(Pra\) and \(Pla\) increased during PP (\(P < 0.01\)) but stayed less than \(Pab\) at PP15 and PP30. In contrast, \(Pivc\) increased similarly with Pab but not with \(Pra\) (Fig. 2); this produced a substantial pressure gradient between \(Pivc\) and \(Pra\) at PP15 and PP30 (\(P < 0.01\)). \(P_{La_m}\) showed biphasic changes similar to those of \(CO\), with a tendency to increase at PP7.5, followed by a significant decrease below baseline at PP30 (\(P < 0.01\), Fig. 3). However, the changes in \(P_{Ra_m}\) presented a different pattern from those of \(CO\), i.e., \(P_{Ra_m}\) increased at PP15 (\(P < 0.05\)) and never decreased below baseline throughout PP.

**Series 2.** With increases in Pab from baseline to 30 mmHg, HR was essentially unchanged, and MAP showed a small but statistically insignificant increase (Table 2). \(Paw\) was significantly higher at PP30 than at baseline (\(P < 0.01\)) with the volume-preset ventilation.

The composite cardiac-function curves derived from the data of all animals are shown in Fig. 4. The differences in \(P_{Ra_m}\) and \(P_{La_m}\) between baseline and PP30 (\(\Delta P_{Ra_m}\) and \(\Delta P_{La_m}\)) were calculated at \(CO_{0.8}\), \(CO_{0.7}\), and \(CO_{0.6}\) and are illustrated in Fig. 5. \(\Delta P_{Ra_m}\) was always greater than \(\Delta P_{La_m}\) (\(P < 0.01\) at \(CO_{0.7}\) and \(CO_{0.6}\)). This indicates that, with increases in Pab, the cardiac function of the RV was more depressed than that of the LV.

**Table 1. Results of series 1**

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>PP7.5</th>
<th>PP15</th>
<th>PP30</th>
</tr>
</thead>
<tbody>
<tr>
<td>(CO), ml/min</td>
<td>1,052 ± 55</td>
<td>1,107 ± 60</td>
<td>1,023 ± 60</td>
<td>804 ± 40*</td>
</tr>
<tr>
<td>(P_{La}), mmHg</td>
<td>7.1 ± 0.3</td>
<td>9.7 ± 0.4*</td>
<td>10.6 ± 0.5*</td>
<td>9.6 ± 0.5*</td>
</tr>
<tr>
<td>(Pra), mmHg</td>
<td>5.5 ± 0.2</td>
<td>8.2 ± 0.4*</td>
<td>9.7 ± 0.5*</td>
<td>9.4 ± 0.6*</td>
</tr>
<tr>
<td>(Ppe), mmHg</td>
<td>1.4 ± 0.2</td>
<td>3.3 ± 0.2*</td>
<td>4.7 ± 0.2*</td>
<td>5.4 ± 0.6*</td>
</tr>
<tr>
<td>(Paw), mmHg</td>
<td>4.5 ± 0.4</td>
<td>4.7 ± 0.3</td>
<td>6.3 ± 0.4*</td>
<td>8.1 ± 0.6*</td>
</tr>
</tbody>
</table>

Values are means ± SE; \(n = 7\) swine. PP7.5, PP15, PP30, abdominal pressure of 7.5, 15, 30 mmHg, respectively; \(CO\), cardiac output; \(P_{La}\), left atrial pressure; \(Pra\), right atrial pressure; \(Ppe\), pericardiac pressure; \(Paw\), airway pressure. *Significant difference, compared with baseline, by one-way ANOVA for repeated measures with Scheffe’s test, \(P < 0.01\).

**DISCUSSION**

The most important findings in this study were that 1) \(CO\) presented biphasic changes with incremental increases in Pab; 2) \(P_{Ra_m}\), an index of LV preload which is the major determinant of \(CO\), had a similar pattern; and 3) the decrease in \(CO\) was associated with a large pressure gradient in the IVC at the level of the diaphragm. These results suggest that the concept of abdominal vascular zone conditions is also useful to characterize the effects of elevated Pab on steady-state cardiac performance.
We have characterized abdominal vascular zone conditions by the relationship of $P_{ra}$ and $P_{ab}$ (28, 30, 31) in a manner analogous to pulmonary vascular zone conditions (32). The IVC circulation is considered as two venous compartments (an upstream extra-abdominal compartment and a downstream abdominal venous compartment), with a vascular waterfall to the thoracic compartment at the exit. When $P_{ra}$ exceeds $P_{ab}$ (defined as abdominal zone III), the abdominal IVC remains in a normal dilated state. An increase in $P_{ab}$ enhances IVC venous return by discharging blood from the abdominal venous compartment. When $P_{ra}$ is below $P_{ab}$ (defined as abdominal zone II), the abdominal IVC collapses, and a vascular waterfall develops at the level of the diaphragm (6, 20, 27). An increase in $P_{ab}$ causes the abdominal IVC to collapse further, elevating the effective back pressure to venous return at the level of the diaphragm. This impedes blood from the extra-abdominal venous compartment (lower extremities), which is not surrounded by $P_{ab}$, thereby reducing the IVC venous return. The essence of this theory is that the abdominal venous compartment can function either as a capacitor (zone III) or as a collapsible Starling resistor (zone II), depending on the relationship of $P_{ra}$ and $P_{ab}$. The dual nature of the abdominal venous bed, determined by abdominal zone conditions, was experimentally confirmed in dogs with an IVC bypass (31) and in intact open- and closed-chest preparations (28, 30) during respiratory maneuvers produced by phrenic nerve stimulation of 3–8 s.

The IVC venous return accounts for approximately two-thirds of systemic venous return, and systemic venous return and the resultant preload of the heart are the most important determinants of CO under normal conditions (8, 24). Therefore, the above principles, derived from "transient" changes in IVC venous return during respiration, may lend insight into the effects of sustained increases in $P_{ab}$ on steady-state systemic venous return and CO. The results of the experiments in series 1 strongly support this hypothesis. With small increases in $P_{ab}$ up to $P_{P7.5}$, $P_{ra}$ remained higher than $P_{ab}$, with a minimal pressure gradient between $P_{ivc}$ and $P_{ra}$ (i.e., zone III). The increases in $P_{ab}$ should enhance IVC venous return and increase the preload of the heart, as reflected by the increase in $P_{platm}$, resulting in an increase in CO.

Another important finding of this study is that increased $P_{ab}$ levels have different impacts on loading conditions and cardiac performance in the RV and LV. By measuring surface $P_{pe}$ as accurately as possible, we found that the LV preload (estimated by $P_{latm}$) showed similar biphasic changes to CO, whereas the RV preload (estimated by $P_{ratm}$) exhibited a different pattern from CO, showing a sustained increase during PP. It may appear paradoxical that the RV preload increased, despite the decreased systemic venous return. The key to understanding this is in the abdominal zone conditions, which characterize the status of systemic venous return but do not directly determine the RV and LV preload. Because preload is essentially a variable determined by the balance of venous return and cardiac function curves (7), the RV preload should be able to
increase, even with a decreased systemic venous return, if the cardiac-function curve is more depressed in RV than in LV.

Consistent with this analysis, the results of the experiment in series 2 demonstrated that the cardiac-function curve of the RV was indeed more depressed (i.e., shifted toward the right) than was that of the LV. Depression of the cardiac-function curve can result from decreased HR, decreased cardiac contractility, or increased afterload. HR was not affected by the elevated Pab, as shown in Table 2. Although decreases in cardiac contractility due to the elevation of the diaphragm cannot be totally ignored, a recent study (16) that used paired ultrasonic-dimension transducers suggested that LV contractility does not significantly change over a relatively wide range of Pab from 5 to 25 mmHg. It is important to appreciate that volume-preset ventilation, not pressure-preset ventilation, was employed in this study to avoid the effects of hypercarbia and acidemia on hemodynamics. Paw increased as a result of decreased respiratory system compliance produced by the PP (1, 19). This should compress small pulmonary arteries, which results in increased RV afterload (26). High Paw, compressing the small abdominal arteries, should have increased the LV afterload in a similar fashion, but this effect may be partly counterbalanced by the high intrathoracic pressure that reduces LV afterload (3, 21, 23, 25). Thus the different effects of increased Paw on RV and LV performance appear to be better explained by the changes in afterload. Under conditions with volume-preset ventilation, the elevated Pab would impose more afterload on RV than on LV, producing higher right-sided venous pressures.

At a given Pab, the development of a vascular waterfall and zone II abdomen is determined by the level of Pra, which is influenced by various factors such as intravascular volume status, cardiac function, and Ppe or pleural pressures. In the present study, the biphasic changes in CO in accordance with changes in abdominal zone conditions were reproducible by maintaining the initial Pra at a constant and relatively high level (5–6 mmHg). However, as we previously found in dogs with transient respiratory maneuvers, a vascular waterfall did not develop with hypervolemia (28, 30). We have also observed, in pilot studies with dogs with sustained abdominal binding, that even a small increase in Pab could produce a zone II abdomen and decrease CO in animals with normal cardiac function and low Pra. When cardiac function was depressed with propranolol, the abdomen remained longer in zone III, and CO increased with increases in Pab. These observations are consistent with the findings by Kash-tan et al. (13), who reported an increase in CO with Pab as high as 40 mmHg in dogs with high Pra. Similarly, the enhancement of CO by abdominal or lower body compression after open heart surgery (9, 10) would only occur when the Pra is elevated due to cardiac compensation and the abdomen is in a zone III condition. Thus a number of variables, such as the magnitude of the increase in Pab, intravascular volume status, and baseline cardiac function, should also be taken into account to apply the concept of abdominal zones and to predict changes in CO with increased Pab.

Our results suggest a possibly useful notion under clinically relevant conditions with elevated Pab. It is inappropriate to estimate cardiac (LV) preload with right-sided venous pressures when volume-preset ventilation is employed and Paw is increased. Even if there is no LV failure or even if “transmural” RV pressures are estimated by some measure of intrathoracic pressure (e.g., esophageal pressure), the right-sided pressures would no longer reflect the status of systemic venous return or the level of LV preload under such conditions. Instead, because a substantial pressure gradient is consistently observed between Pra and Pivc when the vascular waterfall develops and the abdomen goes from zone III to zone II condition, simultaneous comparison of Pra and Pivc may provide us with critical information as to the abdominal zones, the status of LV preload, and the level of Pab to maximize venous return and CO.

In conclusion, we confirmed that the concept of abdominal vascular zone conditions offers a theoretical framework to interpret the complex hemodynamic responses to steady-state increases in Pab. Increases in Pab are likely to augment CO under a zone III abdominal condition (i.e., Pra > Pab) but to decrease CO under a zone II abdomen (i.e., Pra < Pab), by modulating systemic venous return and the LV preload. The RV preload increased despite the decreased venous return, presumably due to a substantial increase in RV afterload produced by the elevated Paw. Simultaneous measurements of Pra and Pivc may provide an alternative diagnostic strategy in hemodynamic care of patients with elevated Pab.

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