Effects of increased intra-abdominal pressure on central circulation

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Background. In an experimental model we investigated the effects of a gradual increase in intra-abdominal pressure (IAP) on the central circulation.

Methods. Seven pigs were anaesthetized, mechanically ventilated and instrumented. IAP was gradually increased by 5 mm Hg up to 30 mm Hg by abdominal banding in normovolaemic animals, and then they were made hypovolaemic after blood withdrawal. Right atrial pressure (RAP) and left ventricular end-diastolic pressure (LVEDP) at each step and aortic, femoral and inferior vena cava blood flows were measured. Left ventricular end-diastolic area (LVEDA) was determined using epicardial echocardiography.

Results. Cardiac output maintained at mild IAP was reduced to 76 (24)% of the initial value at 30 mm Hg IAP [mean (SD)] in normovolaemic animals, and 72 (22)% (P<0.001) in hypovolaemic animals. In normovolaemic animals the LVEDA and LVEDP were significantly increased at an IAP of 10 and 15 mm Hg by 26 (24)% and 38 (23)%, respectively. At these IAP values, the difference between the RAP and IAP was positive. When this gradient became negative, that is beyond 15 mm Hg in normovolaemia and for all IAP values in hypovolaemic animals, the LVEDA declined, reaching 78 (16)% and 62 (22)% (P<0.05) of the initial values in normovolaemic and hypovolaemic groups at the highest IAP value.

Conclusions. These results showed that a gradual increase in IAP led to a redistribution of abdominal blood volume towards the thoracic compartment, at IAP lower than 15 mm Hg in normovolaemia, and at its expense at higher values of IAP. In hypovolaemia there was no thoracic compartment gain. Whereas the absolute or transmural RAPs were not informative of the direction of this blood shift, an RAP greater than IAP was associated with an intrathoracic compartment gain.

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Although not new to medicine, the concept of raised intra-abdominal pressure (IAP) attracts growing interest.¹² In an epidemiological study of patients admitted in intensive care units, its prevalence, defined as IAP greater than 12 mm Hg, was 50.5%. An abdominal compartment syndrome defined as an IAP of 20 mm Hg or more was observed in 8.2%.³ Moreover, intra-abdominal hypertension has been shown to be associated with a significant reduction in patient survival.⁴,⁵ This poor outcome has been ascribed to the deleterious effects of raised IAP on perfusion of all organs within the abdomen and on the systemic circulation. These effects have been investigated some years ago in experimental studies leading to the well-established belief that cardiac filling pressure measurements could be misleading in the case of increased IAP.⁶–⁸ In such cases, intracardiac pressure can increase, and thus preload conditions could be...
perceived to be increased, whereas the transmural pressure, and, therefore, the preload conditions are actually decreased.

In this respect, echocardiography is a useful tool to gain some insight on this issue. Such analyses have been achieved in anaesthetized patients undergoing surgical procedures under laparoscopy with carbon dioxide insufflation.9–12 In this context, conflicting results have been observed concerning the circulatory changes induced by abdominal insufflation. These discrepancies could be explained by at least two factors: most often only two levels of IAP were tested, and, moreover, carbon dioxide in the peritoneal cavity could directly stimulate peritoneal endings and trigger vasopressin release,13 precluding any interpretation of the IAP per se.14

The purpose of this study was to reassess the circulatory effects of a progressive increase in IAP using echocardiography in a setting excluding insufflated gases. In an experimental study in pigs, we analysed the circulatory changes induced by increased IAP obtained by progressive mechanical constraint. In order to test the role of total vascular volume, the same sets of experiments were repeated in the same animals made hypovolaemic.

Materials and methods

Animals and anaesthesia

The experiment was conducted in seven pigs (30–35 kg) according to the guidelines of the animal care committee of Claude Bernard University (Lyon, France). Pigs were premedicated with an i.m. injection of ketamine (15 mg kg\(^{-1}\)) and anaesthesia induced by an i.v. injection of propofol (Diprivan 1%, 1 mg kg\(^{-1}\)) followed by continuous infusion (100 \(\mu g\) kg\(^{-1}\) min\(^{-1}\)). After tracheal intubation, anaesthesia was maintained throughout the study with a mixture of propofol (100 \(\mu g\) kg\(^{-1}\) min\(^{-1}\)) and sufentanil (1 \(\mu g\) kg\(^{-1}\) h\(^{-1}\)). All animals underwent mechanical ventilation (Servo ventilator 900C-Siemens-Elema AB, Solna, Sweden) in a controlled mode. The ventilatory frequency was set at 12 min\(^{-1}\), the inspiratory:expiratory ratio at 1:2, the end-expiratory pressure at 0 cm H\(_{2}\)O and the tidal volume was chosen in order to maintain the initial end-tidal partial pressure of carbon dioxide within the normal range. This tidal volume was maintained throughout the study. ECG and body temperature were monitored. Intravascular fluid balance was kept by infusion of lactate Ringer’s solution with the target to obtain an absolute end-expiratory central venous pressure higher than 5 mm Hg in normovolaemic animals.

Surgical preparation

A fluid-filled catheter was placed in the carotid artery to monitor arterial pressure and to perform sequential blood sampling. A pulmonary artery catheter was inserted through the internal jugular vein into the pulmonary artery for measurement of pulmonary artery pressure and pulmonary arterial occlusion pressure. An additional double lumen catheter was inserted into the contralateral internal jugular vein allowing measurement of the right atrial pressure (RAP) and administration of i.v. drugs and fluids. The right femoral artery was exposed and an ultrasound transit-time flow probe (4 mm S series, Transonic System, Ithaca, NY, USA) was placed around the vessel. An 8 cm air-filled latex cylindrical balloon (Marquette, Boissy-St-Léger, France) was placed in the peritoneal cavity via a stab wound to measure abdominal pressure. The unstressed volume was greater than 3 ml and an operating volume of 0.5 ml was used. After median sternotomy and longitudinal pericardiotomy, ultrasound transit-time flow probes were placed around the root of the aorta (14 mm A series, Transonic System, Ithaca, NY, USA) and the inferior vena cava (16 mm S series, Transonic System, Ithaca, NY, USA). The left ventricle (LV) was catheterized by a 20 cm fluid-filled catheter through a purse-string suture via the apex of the LV allowing free movement of the heart. A pericardial well was built by suspending the opened pericardium, which was partially closed. In order to obtain echocardiographic data, the ultrasound transducer of a transoesophageal echocardiographic probe (5 MHz, Sonos 1500, Philips Medical System, Andover, MA, USA) was placed on the epicardium in a position allowing continuous monitoring of the LV in the midpapillary short-axis plane. Pleural drains were inserted and another air-filled balloon was placed in the mediastinal pleural space before the chest was closed airtight. All the vascular lines, the air-filled balloon catheter and a catheter measuring airway pressure at the junction of the tracheal tube were connected to a multichannel recording system (MP100; Biopac System, Santa Barbara, CA, USA). Vascular flows were simultaneously recorded on the same system. Finally, the abdomen was band with a Velcro belt maintained by three inextensible belts. An inflatable large bladder was positioned underneath, allowing IAP to be increased in a progressive manner.

Experimental protocol

Following instrumentation, a 1 h stabilization period was allowed. Under steady-state anaesthesia, respiratory, circulatory and echocardiographic variables were recorded over 5 min and a blood sample was obtained for blood gas analysis and catecholamine content. Thereafter, IAP was gradually increased by 5 mm Hg steps up to 30 mm Hg by inflating the large bladder. Each step lasted 15 min and during the last 5 min the initial recordings were repeated. A new blood sample was withdrawn during the last step for blood gas analysis and catecholamine content. After completion of these recordings, the pressure in the bladder and thus the IAP were released and pigs were bled through the carotid artery to a mean arterial pressure of 60 mm Hg and a new stabilization period was then allowed. As most often the arterial pressure was partially restored, a new withdrawal of blood was necessary to stabilize the mean.
arterial pressure at 60 mm Hg. Thereafter, a new ramp of IAP was performed while the same measurements were obtained.

**Measurements and calculations**

All pressure measurements were continuously performed and averaged over five complete respiratory cycles. The peak inspiratory pressure was recorded at the end of the inspiratory time. The pulmonary arterial occlusion, the pleural and the left ventricular end-diastolic pressures (LVEDP) were measured at end-expiration. All pressure transducers were referenced to the mid-axillary level. Intrathoracic vascular or cardiac pressures were expressed as transmural pressures. They were calculated by subtracting the pleural pressure from the intravascular or cardiac pressures. Conversely, the gradient between RAP and IAP was calculated from absolute values of intravascular pressures. Vascular flows were obtained by digital integration of instantaneous flows over five complete respiratory cycles. Integration of aortic flow was considered as cardiac output. Femoral flow was expressed as a percentage of cardiac output. Systemic vascular resistance was calculated according to the usual formula.

Concentrations of epinephrine and norepinephrine were measured using high-performance liquid chromatography.\(^{15}\)

**Echocardiographic measurements**

Echocardiographic measurements were stored on a videotape and analysed retrospectively by two independent investigators. The mean value of the two measurements performed at the end of the expiratory period was used for statistical analysis. Echocardiography study consisted of an epicardium short-axis view at the midpapillary level for analysis of left ventricular end-diastolic area (LVEDA). End-diastole was defined as the frame corresponding to the largest LV cross-sectional area immediately after the R-wave peak on the ECG. The LV short-axis end-diastolic cross-sectional area was measured by manual planimetry of the area circumscribed by the leading edge of the LV endocardial border. The anterolateral and posteromedial papillary muscles were included within the ventricular area. The inter-user variability was the absolute percent difference in the measurements performed by the two investigators (E.V. and S.D.): this did not exceed 10% for the LVEDA. The inter-user variability of the left ventricular end-systolic area exceeded this threshold, particularly at high values of IAP, and therefore was not reported.

**Statistical analysis**

All results are expressed as means (SD). Data were analysed using a two-way repeated-measures ANOVA followed by a Newman–Keuls post hoc test when ANOVA showed significance to compare mean values. Differences were considered to be statistically significant when \(P\)-value was less than 0.05. Statistical analysis was performed using statistica-software version 6 (Statsoft Inc., Tulsa, OK, USA).

**Results**

The effects of a gradual increase in IAP on circulatory variables are shown in Figure 1. Mean volume of blood initially withdrawn before induction of hypovolaemia was 652 (224) ml and an additional withdrawal of 54 (45) ml was necessary in order to obtain the target arterial pressure of 60 mm Hg.

**Cardiac output and systemic vascular resistance changes after increased IAP**

Irrespective of volaemia, heart rate and mean arterial pressure were not influenced by incremental increases in IAP. In contrast, cardiac output showed no change at low values of IAP but a decrease reaching 76 (24)% in the normovolaemic and 72 (22)% of baseline values in the hypovolaemic groups at the maximal IAP value (30 mm Hg) (\(P<0.001\)). The IAP course evolution of the calculated systemic vascular resistance mirrored cardiac output changes with an increase extending to 60 (25)% and 79 (31)% of the initial value for the normovolaemic and the hypovolaemic groups, respectively.

**Inferior vena cava and right femoral flow changes after increased IAP**

At baseline IAP, the inferior vena cava flow was 64 (4)% and 63 (9)% of the cardiac output for the normovolaemic and hypovolaemic groups, respectively. This percentage remained essentially unchanged at low values of IAP and decreased for values higher than 10 mm Hg. At the highest IAP, these percentages reached 45 (11)% and 26 (18)% (\(P<0.001\)) of the cardiac output for the same groups, respectively. The right femoral flow expressed as a percentage of cardiac output was initially at 7.2 (1.8)% and 8 (2.1)% of the cardiac output for the normovolaemic and hypovolaemic groups, respectively. This steadily decreased with incremental increase in IAP, culminating in 4.7 (1.8)% and 3.4 (2.5)% of the cardiac output for the highest IAP.

**Cardiac and thoraco-pulmonary pressure changes after increased IAP**

In respect of vascular and cardiac pressures, significant changes were only observed in normovolaemic animals. A significant increase in mean arterial pulmonary pressure, pulmonary arterial occlusion pressure and LVEDP was noted at 15 mm Hg of IAP (Table 1). The pressure gradient between the RAP and the IAP became negative soon after the first step of increase in IAP in hypovolaemic animals. In normovolaemic animals this gradient was still positive at 15 mm Hg of IAP (Table 1). The increases in end-expiratory pleural pressures did not reach significance (Table 1). By
contrast, the peak inspiratory airway and the mean airway pressures showed a significant change as soon as IAP was greater than 5 and 10 mm Hg, respectively (Table 1).

Left ventricular dimension changes after increase in IAP

LVEDA showed biphasic changes in normovolaemic animals with an initial increase of 13 (12)% at IAP 5 mm Hg and 26 (24)% at IAP 10 mm Hg followed by a decrease of 78 (16)% of the initial value at 30 mm Hg (P<0.001). In hypovolaemic animals, the initial increase at 5 mm Hg IAP [13 (11)%] was not significant but the decrease reached 62 (22)% (P<0.001) of the baseline value at 30 mm Hg of IAP (Fig. 2).

Ventilatory and neurohumoral effects of increased IAP

Baseline arterial partial pressures of CO₂ were 47.4 (11.3) and 45.6 (7.9) mm Hg for the normovolaemic and hypovolaemic animals, respectively. When the IAP reached 30 mm Hg these significantly increased to 54.7 (7.8) and 57.3 (12.2) mm Hg, respectively. Within the normovolaemic group, the baseline concentrations of norepinephrine and epinephrine were 237 (121) and 98 (80) nmol litre⁻¹, respectively. These increased at 30 mm Hg IAP to 838 (740) and 436 (692) nmol litre⁻¹.

Discussion

The two main findings of the current study are as follows: first, although the mean arterial pressure remained unchanged during gradual increase in IAP up to 30 mm Hg, cardiac output remained stable up to a value of 15 mm Hg and decreased thereafter. The progression of the LVEDA and left ventricular end-diastolic transmural pressure followed a similar pattern. They remained stable or even increased slightly at low IAP and decreased beyond 15 mm Hg of IAP. Second, these decreases occurred for lower values of IAP in hypovolaemic animals and were
more pronounced. Finally, the gradual increase in IAP was associated with a progressive decrease in inferior vena cava flow and a reduction of cardiac output flow through the femoral artery.

Circulatory changes induced by an increased IAP are still controversial. Some authors found a decrease in cardiac output from the very beginning of the increase in IAP, whereas others showed that this decrease was preceded by an unchanged or even a slightly increased cardiac output at low IAP values. Further experimental or clinical echocardiographic studies have failed to be more conclusive. Conflicting results obtained in these studies might be attributable to at least two factors. First, most of these studies have been performed in patients undergoing abdominal surgery under laparoscopy by using carbon dioxide insufflations. These gas insufflations can induce their own circulatory effects, probably as a result of the activation of a neurohumoral vasomotor system. Second, the measures were usually performed with a single value of IAP around 15 mm Hg precluding the analysis of the effect of IAP over a wider range of values. This prompted us to study the loading conditions during gradually increased IAP, using echocardiography in an experimental pig model, and examine the effect of the volaemic status. Banding the abdomen and inflating a large bladder to raise IAP instead of using carbon dioxide insufflations avoided the confounding factors arising from the biochemical properties of an insufflated gas.

In this study, the pattern evolution of left ventricular preload conditions was clearly dependent on volaemia. In the normovolaemic animals, the increase in IAP up to 10 mm Hg brought about an increase in LVEDA, confirmed by that of the transmural LVEDP. These changes indicated a redistribution of the venous volume towards the thoracic compartment as a result of a decrease in abdominal venous capacitance induced by the elevation of IAP. As the amount of blood shifting from the splanchnic area into the thoracic compartment depended on the amount of blood available to be redistributed, hypovolaemic animals did not exhibit any increase in indices of left ventricular preload. For values above 15 mm Hg of IAP, the LVEDA decreased in both groups of animals, indicating an ongoing redistribution at the expense of the thoracic compartment. This is likely to be related to a further increase in abdominal venous resistance, leading to a trapping of blood upstream of the abdominal compartment.

Apart from ventricular surface measurement by echocardiography, no simple means is available in clinical settings to indicate the direction of the blood shift out of the abdominal compartment. RAP, measured as absolute and a transmural value, did not exhibit a clear pattern evolution. Values relative to IAP seemed more informative. Redistribution into the thoracic compartment occurred even though RAP was greater than IAP. As soon as the gradient was reversed, any further increase in IAP induced a reduction of LVEDA. In our study, this blood volume redistribution induced by the increase in IAP occurred independent of the evolution of the inferior vena cava flow. Indeed, in both groups of animals, inferior vena cava flow remained unchanged at low values of IAP and

### Table 1: Intravascular, airway and pleural pressures in animals undergoing incremental increases in IAP. Values are mean (std) from seven pigs undergoing gradual increase in IAP by 5 mm Hg up to 30 mm Hg, while normovolaemic (Normovolaemic) or hypovolaemic (Hypovolaemic). Intravascular or intracardiac pressures are transmural pressures (tm-mm Hg). *P<0.05 vs baseline value. ANOVA showed significance between groups (Normovolaemic vs Hypovolaemic) for all intravascular variables. ANOVA did not show significance between groups for airway and pleural pressures.

<table>
<thead>
<tr>
<th>IAP (mm Hg)</th>
<th>0</th>
<th>5</th>
<th>10</th>
<th>15</th>
<th>20</th>
<th>25</th>
<th>30</th>
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<tr>
<td>Mean pulmonary arterial pressure (tm-mm Hg)</td>
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<tr>
<td>Normovolaemic</td>
<td>26.7 (5.3)</td>
<td>29.3 (1.0)</td>
<td>32.4 (9.1)</td>
<td>34.7 (10.2)*</td>
<td>32.9 (10.6)</td>
<td>28.7 (6.8)</td>
<td>28.7 (8.7)</td>
</tr>
<tr>
<td>Hypovolaemic</td>
<td>20.2 (4.2)</td>
<td>20.7 (4.2)</td>
<td>22.5 (5.0)</td>
<td>22.0 (5.5)</td>
<td>19.2 (6.7)</td>
<td>17.7 (7.2)</td>
<td>16.6 (6.1)</td>
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<td>Pulmonary arterial occlusion pressure (tm-mm Hg)</td>
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<tr>
<td>Normovolaemic</td>
<td>10.3 (2.6)</td>
<td>11.2 (3.1)</td>
<td>13.8 (3.3)</td>
<td>16.9 (4.9)*</td>
<td>13.9 (5.9)</td>
<td>12.1 (4.5)</td>
<td>11.2 (6.5)</td>
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<td>6.0 (3.5)</td>
<td>5.5 (3.5)</td>
<td>5.5 (3.5)</td>
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<tr>
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<td>14.5 (4.5)</td>
<td>15.6 (4.1)</td>
<td>16.6 (6.5)*</td>
<td>12.6 (8.5)</td>
<td>11.0 (9.2)</td>
<td>10.0 (10.4)</td>
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<td>8.2 (5.1)</td>
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<td>4.9 (4.9)</td>
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<td>Peak inspiratory pressure (cm H2O)</td>
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<td>32.3 (6.3)*</td>
<td>34.5 (6.2)*</td>
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<td>6.1 (1.9)</td>
<td>7.0 (1.8)*</td>
<td>7.6 (2.1)*</td>
<td>8.5 (2.1)*</td>
<td>9.3 (2.2)*</td>
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<td>5.1 (1.1)</td>
<td>6.0 (1.3)</td>
<td>6.7 (1.5)*</td>
<td>7.1 (1.5)*</td>
<td>7.6 (1.6)*</td>
<td>8.1 (1.8)*</td>
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<td>End-expiratory pleural pressure (mm Hg)</td>
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<tr>
<td>Normovolaemic</td>
<td>-0.9 (1.3)</td>
<td>-0.7 (1.7)</td>
<td>-0.2 (1.8)</td>
<td>-0.2 (1.9)</td>
<td>0.66 (1.8)</td>
<td>1.0 (1.9)</td>
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<td>0.8 (2.6)</td>
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<td>1.7 (3.2)</td>
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baseline values. ANOVA showed significance between groups for LVEDA and RAP arterial flow to intra-abdominal organs. An increase in IAP has been shown to decrease perfusion and the consecutive reduction in inferior vena likely to be a decrease in the intra-abdominal and hindlimb abdominal venous resistance. The second component was be 2-fold. The first component could be an increase in decreased thereafter. The mechanism of this decrease could be 2-fold. The first component could be an increase in abdominal venous resistance. The second component was likely to be a decrease in the intra-abdominal and hindlimb perfusion and the consecutive reduction in inferior vena cava return. An increase in IAP has been shown to decrease arterial flow to intra-abdominal organs. Decreased femoral flow that we observed, confirmed the contribution of this factor. Beside the sympathetic activation, this mechanical effect could have contributed to the observed increase in systemic vascular resistance.

This study suffered from at least two limitations. First, the increase in IAP was not associated with an increase in abdominal volume as is usually encountered in clinical conditions. Second, the timescale of the study was likely to allow the vascular compartments to reach a new equilibrium when blood was shifted from one to another. Volume exchange usually taking place between the intravascular compartment and the interstitium could be limited during our experiment.

In conclusion, the present study documented LV preload and afterload changes in response to a gradual increase in IAP. In normovolaemic conditions, the decrease in abdominal venous capacitance led to a redistribution of abdominal blood volume towards the thoracic compartment at IAP lower than 15 mm Hg, and at its expense at higher values. In hypovolaemia there was no gain in the thoracic compartment. Whereas the absolute or transmural RAPs were not informative of the direction of this blood shift, a RAP greater than IAP was associated with an intrathoracic compartment gain.

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References

8 Richardson JD, Trinkle JK. Hemodynamic and respiratory alterations with increased intra-abdominal pressure. J Surg Res 1976; 20: 401–4
14 Blobner M, Bogdanski R, Kochs E, et al. Effects of intraabdominally insufflated carbon dioxide and elevated intraabdominal pressure
on splanchnic circulation: an experimental study in pigs. *Anesthesiology* 1998; 89: 475–82