Renal consequences of intraabdominal hypertension in a porcine model. Search for the choice indirect technique for intraabdominal pressure measurement

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Abstract

Objective: To study the effects on the renal system in a porcine model of intraabdominal hypertension, and to determine the indirect technique of choice for determination of the intraabdominal pressure.

Materials and methods: 30 pigs were used and divided into two groups according to increased intraabdominal pressure values (20 mm Hg and 30 mm Hg). In both groups pressures were registered 8 times, summing up to 3 h, with a CO\textsubscript{2} insufflator. Three different measures of the intraabdominal pressure were taken: a direct transperitoneal measure, using a catheter of Jackson-Pratt connected to a pressure transducer, and two indirect measures, a transvesical by means of a Foley to manometer system, and a transgastric by introducing in the stomach a catheter connected to a pressure monitor with electronic hardware. Mean arterial pressure was calculated, along with the cardiac index, production of urine and serum creatinine.

Results: There was a greater correlation between the transvesical and the transperitoneal intraabdominal pressures ($R^2=0.95$). Average transgastric intraabdominal pressure was inferior to the transperitoneal indicator in all taken measurements. The average arterial pressure descended in both groups, with earlier significant differences observed at 30 mm Hg ($p < 0.020$). Urine production was lower at 30 mm Hg compared with the 20 mm Hg group (9.63 ± 1.57 ml versus 3.26 ± 1.73 ml). Serum creatinine increased in both groups being pathological at 30 mm Hg after 1 h 20 min, with existing differences between early pressures ($p < 0.027$).

Conclusions: This study revealed marked renal affection with higher severity at 30 mm Hg pressures. The transvesical technique showed a greater correlation with the direct measurement technique used, defining this as the method of choice for determination of intraabdominal pressure.

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Introduction

Abdominal compartment syndrome (ACS) is a clinical entity characterized by increased intra-abdominal pressure with marked abdominal distension and with potential to cause, if not diagnosed and corrected in time, multiple organ dysfunction syndrome (MODS), and death of the patient.\(^1\)

The clinical manifestations of this syndrome involve mainly the cardiovascular, pulmonary, renal, and gastrointestinal systems, although others such as the neurological one and even the abdominal wall are also included. These systems can be affected by either direct compression, as in the case of pulmonary dysfunction, or by a decrease in perfusion of the various organs.\(^2\)

At kidney level, there is a significant clinical association between IAH and renal impairment, renal failure being one of the main manifestations of ACS.\(^3\) Thus, renal alteration and failure are a common cause of admission in ICU, observing that this function is the most sensitive to the increase of the IAH.\(^4,5\) Since 1913, it has been known that IAH causes a dysfunction in this system. However, it was not until 1947 when Bradley and Bradley\(^6\) studied, for the first time, the effects caused by increased IAP on renal function in humans, demonstrating the decrease in the glomerular filtration rate and the renal plasma flow, decreased urine output being one of the first clinical manifestations of IAH.\(^7\)

Currently, one of the biggest problems is to establish a proper diagnosis to apply treatment as early as possible. In relation to this, and as demonstrated by Sugrue et al.,\(^8\) clinical examination cannot accurately assess the IAP. Similarly, Malbrain et al.\(^9\) observed a poor correlation between abdominal distension and the presence of increased IAP. It is therefore essential to have a reliable measurement technique of the IAP and, this way, act at the right moment. Consequently, the IAP measurement is essential to diagnose the IAH and the consequent ACS, the choice of the technique used being critical for a proper diagnosis. This measure should be performed routinely in patients with a high probability of an IAH, with a measurement frequency of 4 h, with the patient in supine and at the end of expiration.\(^10\) That is why our goal has focused on comparing 2 types of indirect measures observing their correlation with intraperitoneal intra-abdominal pressure to determine which the technique of choice in patients admitted with abdominal hypertension is, and early identifying the renal alterations that occur with increased IAP.

Materials and methods

We used 30 healthy sows, weighing 20–30 kg. The study was approved by the ethics committee of animal experimentation, fulfilling the European regulations on protection of animals used for experimental and other scientific purposes.

After the adaptation and quarantine period, the study began. All the animals received the same anesthetic protocol, administering intraoperative analgesia.

For lifting the IAP, we used a CO\(_2\) insufflator (SCB Thermoditator Karl Storz-Endoskope) using an inflow of 1–2 l/min to reach the desired pressures in each of the groups, 20 or 30 mm Hg.

After reaching the desired pressure in each group, 3 types of IAP measurements were conducted.
Transperitoneal intra-abdominal pressure

It was calculated continuously and directly; to do so, a Jackson-Prat catheter connected to a pressure transducer, and this to a S/STM General Electric Datex-Ohmeda compact anesthesia monitor, was introduced laparoscopically into the abdominal cavity over the right hypochondrium.

Transvesical intra-abdominal pressure

It was obtained intermittently and indirectly; to do so, the animal placed in dorsal decubitus, a cystoscopy assisted catheter was introduced, to which we connected a Foley manometer system (Holtech Medical, Copenhagen, Denmark) between the diuresis bag calibrated with marks in mm Hg from 0 to 40 mm Hg. After placing the system, 20 ml of saline solution were introduced for system purging. The measurement was obtained by placing the zero of the measurement system at the level of the pubic symphysis of the hip with the rest of the system in 90° position relative to the horizontal one of the animal. After opening the existing forceps below the antibacterial filter, one can see it descend, at first, the physiological saline used for purging, and subsequently the urine contained in the system to stabilize and provide measurement of the IAP.

Transgastric intra-abdominal pressure

First, the animal was placed in right lateral decubitus to perform an upper digestive endoscopy to empty the stomach and thus avoid possible interferences. Once empty, a catheter was inserted in the stomach to determine the transgastric intra-abdominal pressure (TG-IAP) continuously. Its proximal end was connected to a pressure monitor with electronic hardware (Spiegelberg CD Pharma) which, after filling the air balloon with a total volume of 0.05–0.1 ml and automatically adjusting the zero, measured in real-time graphically recording the TG-IAP.

For determining the hemodynamic parameters, we used the PICCO® system. Blood samples were removed for determination of serum creatinine, and the production of urine was taken into account.

The means of the monitored variables during the anesthetic procedure were calculated at the following times: T1 after obtaining the appropriate pressure for each group; T2 after 20 min to obtain the proper pressure (stabilization period); T3 after 30 min of T2; T4 after 60 min of T2; T5 after 90 min of T2; T6 after 120 min of T2; T7 after 150 min of T2; and T8 after 180 min of T2.

Statistical analysis

We obtained a descriptive statistics for each variable obtaining the mean ± standard deviation. The comparison between groups 2-2, at each of the times, was performed using the Student’s t-test. The evolution of the parameters was analyzed using a multivariate analysis, repeated measures ANOVA. In the case of the urine output, we took the basal level as 0 and we observed its variation over time. The degree of correlation of the variables determined the

Results

In all the groups we were able to obtain the desired pressure.

The statistical analysis showed statistically significant differences between the transperitoneal intra-abdominal pressure (TP-IAP) and the TG-IAP, in which lower values than in the rest of the pressures were obtained. There were no statistically significant differences between the 2 pressures measured indirectly (Table 1).

Figs. 1 and 2 show that there was a high degree of positive correlation between the 3 IAPs measured in the study, the regression coefficient (R²) being higher between the TP-IAP and the TV-IAP.

Table 2 shows the hemodynamic parameters measured in the 2 groups; in it we can see a progressive decrease in the

![Graph 1](image1.png)

**Figure 1** Degree of correlation and regression line between the measures of the TV-IAP and TP-IAP shown throughout the study time. Pearson correlation coefficient.

Pearson correlation coefficient between every 2 variables. The significance level in all cases was p < 0.05. To carry out the statistical analysis, we used the SPSS 17.0 for Windows; SPP, Chicago, IL, USA.

![Graph 2](image2.png)

**Figure 2** Degree of correlation and regression line between the measures of the TG-IAP and TP-IAP shown throughout the study time. Pearson correlation coefficient.
MAP in both study groups, being earlier under pressures of 30 mm Hg, there being differences between pressures after 50 min following the increase in the pressure. In the cardiac output (CO), no significant changes were observed, we could only observe in the T6 differences between pressures.

Table 3 reflects the values of serum creatinine throughout the study. An increase thereof can be observed in both study groups, there being significant differences between pressures from the T3. In the group of 30 mm Hg, a pathological increase in creatinine can be observed from the T4.

Fig. 3 represents the urine output of each group at each time. The differences between pressures are present from the start of the study, showing a lower urine output at pressures of 30 mm Hg.

Discussion

In the last decade, thanks to the World Society of Abdominal Compartment Syndrome, great progress has been made to better understand both the etiology and pathophysiology of the IAH and ACS.\textsuperscript{11,12} That is why, one of the biggest problems we face is determining the precise point at which to act so that the patient does not develop a MODS. This may

Table 2 Hemodynamic measures collected throughout the study in the IAP 20 and 30 mm Hg groups for 3 h.

<table>
<thead>
<tr>
<th>Hemodynamic measures</th>
<th>Times</th>
<th>IAP group 20 mm Hg/3 h</th>
<th>IAP group 30 mm Hg/3 h</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean blood pressure (mm Hg)</td>
<td>T1</td>
<td>60.26 ± 4.23</td>
<td>59.66 ± 1.38</td>
</tr>
<tr>
<td></td>
<td>T2</td>
<td>57.40 ± 3.73</td>
<td>55.93 ± 1.38</td>
</tr>
<tr>
<td></td>
<td>T3</td>
<td>56.66 ± 7.43</td>
<td>51.53 ± 3.06\textsuperscript{**}</td>
</tr>
<tr>
<td></td>
<td>T4</td>
<td>55.80 ± 8.54</td>
<td>48.66 ± 3.26\textsuperscript{**}</td>
</tr>
<tr>
<td></td>
<td>T5</td>
<td>53.40 ± 9.37</td>
<td>47.33 ± 3.35\textsuperscript{**}</td>
</tr>
<tr>
<td></td>
<td>T6</td>
<td>49.33 ± 8.78</td>
<td>45.86 ± 4.85\textsuperscript{**}</td>
</tr>
<tr>
<td></td>
<td>T7</td>
<td>46.53 ± 8.65</td>
<td>44.20 ± 3.74\textsuperscript{**}</td>
</tr>
<tr>
<td></td>
<td>T8</td>
<td>44.66 ± 7.52\textsuperscript{*}</td>
<td>42.00 ± 4.45\textsuperscript{**}</td>
</tr>
<tr>
<td>Cardiac output (L/min/m\textsuperscript{2})</td>
<td>T1</td>
<td>1.75 ± 0.37</td>
<td>1.81 ± 0.38</td>
</tr>
<tr>
<td></td>
<td>T2</td>
<td>1.30 ± 0.23</td>
<td>1.21 ± 0.39</td>
</tr>
<tr>
<td></td>
<td>T3</td>
<td>1.34 ± 0.25</td>
<td>1.37 ± 0.29</td>
</tr>
<tr>
<td></td>
<td>T4</td>
<td>1.42 ± 0.45</td>
<td>1.51 ± 0.47</td>
</tr>
<tr>
<td></td>
<td>T5</td>
<td>1.50 ± 0.55</td>
<td>1.53 ± 0.54</td>
</tr>
<tr>
<td></td>
<td>T6</td>
<td>1.52 ± 0.57</td>
<td>1.76 ± 0.41\textsuperscript{*}</td>
</tr>
<tr>
<td></td>
<td>T7</td>
<td>1.48 ± 0.53</td>
<td>1.83 ± 0.30</td>
</tr>
<tr>
<td></td>
<td>T8</td>
<td>1.47 ± 0.54</td>
<td>1.77 ± 0.52</td>
</tr>
</tbody>
</table>

\textsuperscript{*} p < 0.05 vs. baseline.

\textsuperscript{**} p < 0.05 vs. groups with IAP.
be related to late diagnoses, poor prognoses, and infectious complications. Therefore, the objectives of this work have focused on early determining renal alterations occurring in the face of increased IAP and determining an indirect technique of choice able to reliably measure the existing IAP in the patient to establish an early diagnosis and proper treatment.

Clinically, renal involvement presents with a progressive decrease in the diuresis as the pressure increases. This oliguria does not respond to fluid therapy, diuretics, or vasoactive drugs. In general, the changes in the IAP affect the production of urine more than the MAP, thus, decreased urine output being one of the first visible signs of IAH. This is in agreement with our results, where we observe a more marked decrease in the urine output in the 30 mm Hg group, finding oliguria without actually occurring anuria at the set times of the study. This may be because a longer study time would be needed for anuria to occur, as demonstrated by long-term studies in pigs from 24 h of study. This is consistent with the pathological increase in creatinine to 30 mm Hg that we observe in our results. Like us, Toens et al. also observed an increase in serum creatinine from 6 h. This circumstance is because they make the first measurement from that time without being able to determine whether that increase occurred before that time. Therefore, we consider important to determine measurements over time of this parameter, since it has been reported that the degree to which serum creatinine changes with regard to a baseline value can reflect changes in the GFG.

A decrease in the CO has been described as the main mechanism of renal function impairment due to a decrease of renal perfusion and an increase of the renin–angiotensin–aldosterone activity. However, both the compression of the vein and the renal parenchyma have also been involved as primary mediators of renal function. This is because as previous studies have shown, normalization of the CO does not improve renal disorders caused by increased IAP. Our results show no alteration in the CO but a renal involvement possibly attributed to the compression of the vessels themselves and the renal parenchyma as a consequence of increased pressure. Thus, we can say that there was renal involvement; however, we consider necessary further studies that correlate the histological alterations, loss of functionality, and changes in the renin–angiotensin–aldosterone system that occurs before renal impairment.

It has been described that an isolated elevation of the IAP should not be considered as a criterion for making decisions for critical patients. Because there are several studies that show that neither the IAH nor the ACS should be diagnosed by means of physical examination several methods have been used to determine the measure of the IAP, both directly and indirectly.

In connection with the measurement systems that have been used directly and invasively to measure the IAP, there are different methods, such as intraperitoneal catheters connected to a pressure transducer, or during laparoscopic surgery using a Veress needle. However, these direct methods are considered too invasive and, therefore, not suitable for critical patients due to increased risk of infection that they might involve, currently being used to validate indirect methods. In our study, we used a Jackson-Prat catheter connected to a pressure transducer as a measure of the IAP directly and continuously. This way, the pressure in the abdomen could be determined, at all times, continuously showing a high correlation with the indirect measured PIA methods.

### Table 3

<table>
<thead>
<tr>
<th>Renal measures</th>
<th>Times</th>
<th>IAP group 20 mm Hg/3 h</th>
<th>IAP group 30 mm Hg/3 h</th>
</tr>
</thead>
<tbody>
<tr>
<td>Creatinine</td>
<td>T1</td>
<td>1.96 ± 0.57</td>
<td>2.34 ± 0.60</td>
</tr>
<tr>
<td></td>
<td>T2</td>
<td>2.05 ± 0.63</td>
<td>2.43 ± 0.62</td>
</tr>
<tr>
<td></td>
<td>T3</td>
<td>2.12 ± 0.59</td>
<td>2.62 ± 0.15</td>
</tr>
<tr>
<td></td>
<td>T4</td>
<td>2.27 ± 0.50</td>
<td>2.81 ± 0.49</td>
</tr>
<tr>
<td></td>
<td>T5</td>
<td>2.41 ± 0.53</td>
<td>2.88 ± 0.54</td>
</tr>
<tr>
<td></td>
<td>T6</td>
<td>2.57 ± 0.56</td>
<td>3.10 ± 0.47</td>
</tr>
<tr>
<td></td>
<td>T7</td>
<td>2.59 ± 0.58</td>
<td>3.17 ± 0.41</td>
</tr>
<tr>
<td></td>
<td>T8</td>
<td>2.69 ± 0.54</td>
<td>3.26 ± 0.44</td>
</tr>
</tbody>
</table>

* p < 0.05 vs. baseline.  
** p < 0.05 vs. IAP groups.

Figure 3 Changes in the urine output during all the study times at the 2 pressures studied: 20 mm Hg (green) and 30 mm Hg (blue). *p < 0.05 vs. baseline; †p < 0.05 vs. groups with IAP.
In relation to the 2 indirect methods used in our study, both have been validated by several authors. Although the measure of the TG-IAP may have advantages over the TV-IAP, being a continuous method, the same as Lee et al. and Malbrain et al., our results show a high degree of correlation between the two pressures, showing a higher correlation between the measure of the IAP directly and the TV-IAP in both study groups. This shows that both pressures are closely related to each other, behaving similarly faced with the increase of the pressure, so we can consider the TV-IAP as the technique of choice in patients admitted to the ICU. Thus, we agree with Malbrain et al. and De Potter et al., on considering that it is an accurate method for estimating the IAP, besides being easy and safe to perform.

Before the results shown, we consider that with elevated IAP continuously at pressures of 30 mm Hg, there begins to be damage to the kidney, the first clinical manifestation being decreased urine output. With regard to the measurement methods, we consider, as the first option, the manometry technique employed in this study because of the advantages that it presents, such as being easy to perform, cost-effective, rapid, and requiring minimal handling.

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**Conflict of interest**

The authors declare that they have no conflict of interest.

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**References**

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