Reversal of Adverse Hemodynamic Effects of Pneumoperitoneum by Pressure Equilibration

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Hypothesis: The creation of positive-pressure pneumoperitoneum during laparoscopic operations can lead to adverse hemodynamic changes, mainly decreased cardiac output. We hypothesized that pneumatic compression sleeves worn on the legs during pneumoperitoneum could abolish the pressure gradient between the abdominal cavity and the legs and so eliminate these adverse hemodynamic changes.

Design: Prospective, randomized, controlled clinical trial with an additional calibration group.

Setting: A regional referral center.

Patients: Forty-five consecutive patients undergoing laparoscopic cholecystectomy who developed hemodynamic changes on induction of positive-pressure pneumoperitoneum were randomized to 3 groups.

Interventions: Low-pressure, nonsequential pneumatic compression sleeves, wrapped around the legs, were used to equilibrate the pressure gradient in the study group and to gradually exceed it in the calibration group. In the control group, no sleeves were used.

Main Outcome Measures: Transesophageal Doppler cardiac output, stroke volume, and systemic vascular resistance were monitored noninvasively.

Results: The creation of positive-pressure pneumoperitoneum caused a significant decrease of cardiac output and stroke volume and increased systemic vascular resistance. In the experimental groups of patients, pressurizing the sleeves to the pneumoperitoneal pressure caused a significant increase of cardiac output (from 4.82 to 6.74 L/min), increased stroke volume, and decreased systemic vascular resistance ($P < .001$). This was not seen in the control group. Additional gradual pressure increase in the sleeves of the calibration group produced no further improvement. Releasing the pressure abolished the hemodynamic advantages.

Conclusions: Applying pressure on the legs equivalent to the positive-pressure pneumoperitoneum improves hemodynamic performance during pneumoperitoneum by nullifying the pressure gradient that is responsible for the adverse consequences. This might be of major practical value, especially for cardiac patients undergoing prolonged laparoscopic operations.

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During recent years, laparoscopic surgery has revolutionized the operative approach in almost all aspects of general surgery, and several laparoscopic procedures have become the gold standard because of their well-known advantages. However, the creation of positive-pressure pneumoperitoneum (PPP), which is essential for abdominal laparoscopic operations, may cause adverse physiological effects. Many human and animal studies have demonstrated diverse hemodynamic changes during PPP that were caused by the increased intra-abdominal pressure and hypercarbia. Their main cardiovascular manifestations include reduced venous return, increased systemic vascular resistance (SVR), and decreased cardiac output (CO), as well as decreased visceral perfusion. These may have clinical implications, especially for elderly and cardiac patients undergoing prolonged laparoscopic operations.

Various remedies that minimize these adverse effects have been proposed. They include the use of inert gas for abdominal insufflation, gasless surgery, low-pressure insufflation, and the use of vasodilatory drugs for improvement of cardiac function.

A mechanical solution using an intermittent sequential pneumatic compression device on the legs was suggested by Bickel. This device was later successfully implemented on a small group of patients. However, it used relatively high pressure necessitating a rather complex apparatus, and its effect on patients suffering from congestive heart failure has not been tested.
In this article, we examine a different approach, whereby pressure is applied not to force blood to the heart but just to abolish the pressure gradient between the abdominal cavity and the lower extremities. This is accomplished with low-pressure nonsequential pneumatic compression sleeves wrapped around the legs and inflated to a constant pressure that is identical to the intraabdominal pressure. Our objectives were to test the clinical validity of the pressure-equilibration concept as a means to counteract the mechanisms that create the adverse hemodynamic effects during PPP and to examine the effectiveness of this device.

STUDY POPULATION AND DESIGN

A total of 63 consecutive patients undergoing elective laparoscopic cholecystectomy for symptomatic cholelithiasis were enrolled in this prospective, randomized study. The patients were all in good health, classified as American Society of Anesthesiology categories I and II. The patients gave their informed consent to participate in the study, which was approved by the local research ethics committee.

Prior to anesthesia, the patients were randomly assigned (using a randomized table) to 3 experimental groups, contingent on their developing a significantly (>10%) decreased CO during induction of PPP. Eighteen patients were excluded because they developed no such hemodynamic changes, and 45 were eventually included: 20 in the experimental study group, fitted with the pneumatic sleeves inflated to the pressure of the PPP; 15 in the control group, in whom pneumatic sleeves were not used; and 10 in the calibration group, in whom pressure in the sleeves was manipulated and eventually exceeded the equilibration values. The calibration group tested the extent of pressure increase needed to gain the maximal advantage of counteracting the PPP and to clarify the pathophysiological mechanism of PPP in affecting hemodynamics. In addition to the above, comparisons between consecutive surgical phases were controlled within the same patient.

ANESTHESIA AND PNEUMOPERITONEUM

General anesthesia was administered to all patients according to the same protocol. Induction was performed using fentanyl (3 µg/kg), propofol (2 mg/kg) or thiopentone (4 mg/kg), and succinylcholine (1 mg/kg) following preoxygenation (2 minutes) by mask. Anesthesia was maintained with nitrous oxide in oxygen, isoflurane, atracurium, and fentanyl. Ventilation was mechanically controlled at a frequency and tidal volume sufficient to maintain normocapnia (end-tidal carbon dioxide between 35 and 38 mm Hg). Intraoperative crystalloid infusion was basically maintained at 7 mL/kg per hour. Carbon dioxide PPP was maintained automatically at 14 mm Hg at an insufflation rate of 1 to 1.5 L/min. Patient monitoring included electrocardiography, noninvasive blood pressure measurements, pulse oximetry, capnography, peripheral nerve stimulation for assessment of depth of anesthesia, and quantification of urinary output.

CARDIAC FUNCTION

Cardiac output and stroke volume (SV) were measured every 3 minutes by a transesophageal Doppler ultrasonographic apparatus (ODM II, Cardio Q Doppler Monitor; Deltex Medical, Chichester, England) with single-use 4-MHz sterile probes (Deltex Medical). Peripheral venous pressure was measured through the brachial vein instead of invasive central venous pressure monitoring. Systemic vascular resistance and mean arterial pressure were calculated according to standard physiological equations (using peripheral venous pressure for derivation of SVR).

LOW-PRESSURE PNEUMATIC SLEEVES

Each leg was wrapped with a pneumatic sleeve from the foot to the groin. Every sleeve contained 3 pneumatic cells (Mego Afek, Kibbutz Afek, Israel) individually connected to a common tube enabling simultaneous insufflation by a digitally controlled apparatus. In patients from the experimental group who displayed decreased CO upon induction of PPP, manipulation of the sleeves started 9 to 12 minutes later, with inflation pressure identical to the intraabdominal pressure. When the anti-Trendelenburg position was ordered, a new sleeve pressure was set, equivalent (in millimeters of mercury) to the intraabdominal pressure plus the hydrostatic pressure of the pooled blood contained in the lower limb's veins, calculated from half the maximal descent of the feet (in centimeters of water) to preserve the pressure equilibrium. In the calibration group, after a few minutes of pressure equilibration, the pneumoflator pressure in the sleeves was gradually increased, in several 3-minute increments, to 10 mm Hg above the PPP. All hemodynamic parameters were recorded, starting with the induction of anesthesia and continuing through the induction of PPP, the manipulation of the pneumatic sleeves (for approximately 15 minutes), the deflation of the sleeves toward the end of surgery, the evacuation of the PPP, and the end of anesthesia.

STATISTICAL ANALYSIS

The data were analyzed with SPSS software, version 11.0 (SPSS Inc, Chicago, Ill) and sample-power software, version 1.2 (SPSS Inc). Data are expressed as mean ± SD, and the significance level was set at P < .05.

The sample size was estimated using the following assumptions: a CO mean difference of 2 L/min; σ = 2.5 L/min; α error = .05 and β error = 10; and normal distribution.

The basic characteristics of the study and control groups were compared by the χ² or Fisher exact test for categorical variables and the Wilcoxon rank sum test for continuous variables. Paired t tests or Wilcoxon rank sum tests were used to compare changes in the values of the hemodynamic parameters during the stages of surgery, when each patient served as his or her own control. The Wilcoxon rank sum test was used to compare the initial hemodynamic parameters between the study and control groups. The sign test was applied to assess the significance of variation in the numbers of patients who demonstrated significant hemodynamic changes (defined as those >10%) between consecutive stages of the procedure.

RESULTS

The experimental and control groups were matched and were similar in all aspects compared (Table 1).

The induction of PPP caused a significant mean ± SD decrease of CO, from 7.03 ± 2.34 to 4.82 ± 1.88 L/min in the experimental study group, as well as in the control group, from 6.58 ± 2.17 to 5.1 ± 1.99 L/min. The mean ± SD SV behaved similarly, decreasing from 94.2 ± 33.9 to 70 ± 24.9 mL in the study group and from 94 ± 29.2 mL.
to 77.4±29.9 in the control group. During this period, the mean±SD SVR increased correspondingly from 1065.0±483.7 to 1432.1±502.7 dyne·second·cm⁻⁵ and from 1035.3±400.5 to 1614.0±929.5 dyne·second·cm⁻⁵. All these changes were statistically significant (P<.001).

Following 12 minutes of PPP, the activation of the low-pressure pneumatic sleeves in the experimental group caused a significant mean±SD increase of the CO to 6.74±1.88 L/min, representing a recovery of 96% of the pre-PPP value (Figure 1). Similarly, the mean±SD SV increased to 91.8±24.8 mL (Figure 2), and the SVR decreased to 1064.2±272.7 dyne·second·cm⁻⁵, very similar to the initial level (Figure 3). All these differences were significant (P<.001).

All 20 patients in this group exhibited a substantial (>10%) decrease in CO following PPP that was reversed in all of them by the pneumatic sleeves. Similar significant differences concerning SV and SVR were demonstrated (data not shown). Conversely, in the control group, only 1 patient exhibited a spontaneous increase in the CO during the extended PPP, in 5 patients the CO remained at the level established after the induction of PPP, and in 9 the CO continued to decrease. Similar changes were noted in the control group concerning the SV, with only 1 spontaneous increase, and in all the SVR remained elevated until the evacuation of PPP.

When the pneumatic sleeves were decompressed, the mean±SD CO decreased significantly, in 18 of the 20 patients of the experimental group, to 5.66±1.88 L/min, the mean±SD SV decreased to 77.5±21.7 mL, and the mean±SD SVR increased to 1280±538.4 dyne·second·cm⁻⁵ (P<.001 for each of these variables). Releasing the PPP restored those hemodynamic improvements in 16 patients. In the control group, terminating the PPP caused significantly increased CO in 10 patients; the mean±SD SV increased to 86.17±24.9 mL, and the mean±SD SVR decreased to 1270±502.1 dyne·second·cm⁻⁵ (Figures 1-3).

In both groups, we have not identified a significant change in pulse or mean arterial pressure in most stages of surgery, except (in mean arterial pressure) immediately following anesthesia (Table 2). However, a significant increase in the peripheral venous pressure was observed during the PPP phase in both groups, and it was reversed by the termination of the PPP.

Additional studies with the calibration group demonstrated that gradual pressurization of the sleeves, up to 10 mm Hg above equilibration values, has not significantly changed the CO or the SV beyond the improvement gained by the inflation of the sleeves to the PPP equilibration pressure.

**COMMENT**

In our study, it was demonstrated that elimination of the PPP-generated intravascular pressure gradient between the abdominal cavity and the lower limb, by using low-pressure, nonsequential compression pneumatic sleeves,
led to significant hemodynamic improvement in patients developing decreased CO.

The possibility of hemodynamic derangement following PPP in minimally invasive surgery is a factor still limiting its widespread use. Although rare during relatively short and elective laparoscopic surgery in a relatively healthy population, a potential threat of cardiovascular deterioration exists during prolonged operations with high-risk patients.\textsuperscript{19,21}

Although the study and control groups were small, the numbers were sufficient to demonstrate a clear and significant difference between them. Adding the calibration group, the effect of equilibration was actually validated in 30 patients.

The hemodynamic effects of PPP were not uniform in our study, as was also demonstrated in other studies in which the CO was found to be decreased or unchanged.\textsuperscript{12,30-34} It is well known that several factors, like the baseline hemodynamic function and the volume status of the patients, might be of influence. However, patients in whom decreased CO was not found following PPP (29% of the enrolled patients) were excluded in our study, thus focusing on patients whose hemodynamic changes could have clinical consequences.\textsuperscript{10,30-34} A priori, we were not able to predict which patient would demonstrate hemodynamic deterioration during PPP.

The effect of low-pressure pneumatic sleeves was probably mediated by the reversal of the effects of PPP, thus elevating SV and decreasing SVR, eventually augmenting CO by decreasing afterload and perhaps increasing preload.

We consider the physiologic effects of pressure equilibration to be similar to the cardiovascular changes that occur during thermoneutral (35°C) water head-out immersion. When the upper part of the body is above water, the intrapulmonary pressure is equal to the atmospheric pressure, as the subject breathes the outside air. The immersed body is under the influence of the atmospheric plus hydrostatic pressure (which is directly proportional to the vertical distance from the water surface). The increased transdiaphragmatic pressure is sufficient to draw additional blood into the thorax, thus increasing venous return. By redistributing blood from the peripheral dependent regions to the thorax, immersion causes enhanced diastolic filling of the right atrium and ventricle, evoking the Starling mechanism, thus increasing SV and CO.\textsuperscript{15,38}

In our experimental group, the abdominal cavity and lower limbs were under atmospheric pressure plus the pressure equivalent to PPP, created by the insufflated carbon dioxide and the pneumatic sleeves, and additional pressure on the legs in case of anti-Trendelenburg positioning. This counterbalanced the gravitational effects on blood distribution, thus mimicking the effect of head-out immersion, in which the higher venous blood pressure in the peripheral dependent areas is counterbalanced by the higher hydrostatic pressure. In other words, the previous pressure gradient between the peritoneal cavity and the legs has become a new transdiaphragmatic pressure gradient between the lower torso (beneath the diaphragm) and the pleural cavity above it.

Although this analogy is not perfect, because the patients in our study were supine and subjected to artificial ventilation, thus decreasing the changes in transdiaphragmatic pressure, it offers a mechanism that can explain the increased SV and CO that were observed in our patients. In both situations, decreased SVR can be explained by decreased sympathetic nervous activity, controlled by baroreflexes associated with the central shift of blood volume.\textsuperscript{36}

The hemodynamic improvement following activation of the pneumatic sleeves almost approached the baseline values recorded before inducing PPP. However, those initial parameters are probably lower than during wakefulness because of the effect of anesthesia.\textsuperscript{39,40}

The ODM II esophageal Doppler, although usable only during anesthesia, provides continuous and reliable recordings and is easy to manipulate. Such recordings rule out the possibility of missing temporary unexpected he-
Table 2. Values of Hemodynamic Variables During Stages of Surgery in the Experimental and Control Groups

<table>
<thead>
<tr>
<th>Stage</th>
<th>Mean ± SD MAP, mm Hg</th>
<th>Mean ± SD PVP, mm Hg</th>
<th>Mean ± SD Pulse, Beats/min</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Experimental</td>
<td>Control</td>
<td>Experimental</td>
</tr>
<tr>
<td>Anesthesia</td>
<td>91.6 ± 11.2</td>
<td>89.2 ± 9.0</td>
<td>10.4 ± 2.9</td>
</tr>
<tr>
<td>PPP</td>
<td>94.0 ± 8.8</td>
<td>99.8 ± 11.6*</td>
<td>12.0 ± 3.4*</td>
</tr>
<tr>
<td>PPP continued</td>
<td>98.6 ± 11.4</td>
<td>99.0 ± 10.3</td>
<td>11.4 ± 3.8</td>
</tr>
<tr>
<td>Sleeves</td>
<td>97.3 ± 10.6</td>
<td>NA</td>
<td>12.1 ± 3.5</td>
</tr>
<tr>
<td>Sleeves continued</td>
<td>94.9 ± 8.2</td>
<td>NA</td>
<td>12.4 ± 3.5</td>
</tr>
<tr>
<td>Sleeves off</td>
<td>92.5 ± 7.9</td>
<td>NA</td>
<td>11.5 ± 3.1*</td>
</tr>
<tr>
<td>End PPP</td>
<td>94.4 ± 6.5</td>
<td>94.7 ± 6.3</td>
<td>10.6 ± 2.8*</td>
</tr>
</tbody>
</table>

Abbreviations: MAP, mean arterial pressure; NA, not applicable; PPP, positive-pressure pneumoperitoneum; PVP, peripheral venous pressure.

*Significantly different from the previous stage (P<.005).

modynamic changes. Based on this, and through a comparison with the control group, we have demonstrated that reversal of deranged hemodynamic changes was affected by the elimination of the pressure gradient and did not occur spontaneously, as was suggested by others. Additional increase of the pressure within the sleeves above equilibration pressure in the calibration group resulted in no additional hemodynamic improvement, supporting our assumption.

A practical application of the concept might be the application of elastic stockings along the legs in the proper tension to achieve similar advantages. Additional studies are necessary to validate this option. However, although we did not measure femoral venous flow during manipulation of our device, the advantages of the constant pressure of elastic stockings in prevention of venous stasis are well known.

Besides the different physiological mechanisms exerted, this method differs from intermittent sequential compression of the lower limbs in the feasibility and simplicity of the present device, its simple operation, cost-effectiveness, and the requirement of much lower pressure.

In conclusion, we demonstrated that a simple pneumatic device based on pressure equilibration eliminates the pressure gradient between the abdominal cavity and the lower limbs and is capable of almost reversing the hemodynamic changes during PPP. This should be of importance for patients at risk for developing cardiac complications during prolonged laparoscopic operations.

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