CIRCULATORY EFFECTS OF CARBON DIOXIDE INSUFFLATION OF THE PERITONEAL CAVITY FOR LAPAROSCOPY

R. L. MARSHALL, P. J. R. JEBSON, I. T. DAVIE AND D. B. SCOTT

SUMMARY

Measurements of cardiac output, mean arterial pressure, central venous pressure, and heart rate were made in seven patients undergoing laparoscopy. Measurements were made before and after insufflation of the peritoneum with carbon dioxide, and again after intravenous injection of oxprenolol 2 mg. No significant change in cardiac output followed peritoneal insufflation, but there was a significant increase in mean arterial pressure, central venous pressure, and heart rate. Following the administration of oxprenolol, cardiac output, mean arterial pressure, and heart rate all fell, and central venous pressure rose further. Only the change in heart rate was statistically significant.

Blood gas analysis in five patients showed a rise in PaCO₂ and fall in pH after insufflation with carbon dioxide. The implications of these findings are discussed.

Patients undergoing laparoscopy under general anesthesia exhibit well marked respiratory effects if allowed to breathe spontaneously. As the abdomen is distended with gas, movement of the diaphragm is impeded, tending to reduce tidal volume. At the same time the arterial carbon dioxide tension (PaCO₂) rises abruptly, as shown by Hodgson, McClelland, and Newton (1970), especially if carbon dioxide is used to insufflate the abdomen; consequently there is a powerful central stimulation to the respiration. It would appear likely that circulatory changes are also occurring, both in response to the rise in PaCO₂ and as a result of increasing abdominal pressure. Smith and associates (1971) have described the changes seen in arterial pressure, central venous pressure, and heart rate in patients being artificially ventilated during the procedure. We report here the effects on cardiac output in patients breathing spontaneously.

Stephen, Davie, and Scott (1971) showed that the beta-adrenergic blocking agent, oxprenolol, caused circulatory changes when administered during general anaesthesia. On this occasion, the opportunity was taken to observe the changes consequent on its administration when hypercarbia was maximal and catecholamine release might be expected.

METHOD

Seven female patients aged between 21 and 38 were studied. All were in good health, and were undergoing laparoscopy for sterilization by tubal diathermy. All had given consent for the investigations to be carried out. Atropine 0.6 mg was given intramuscularly as premedication. Anaesthesia was induced with thiopentone 500 mg and maintained with a mixture of nitrous oxide 3 l./min and oxygen 1 l./min, to which halothane 1.5% was added from a Fluotec vaporizer. A circle circuit with carbon dioxide absorption was used, and all the patients were allowed to breathe spontaneously. A central venous catheter (E-Z Cath) was passed through a cubital fossa vein.

Arterial blood pressure was measured by sphygmomanometry, and mean arterial pressure calculated as diastolic pressure plus one-third of the pulse pressure. Central venous pressure was monitored using a Sanborn 267AC pressure transducer, the zero reference point being taken as 5 cm below the level of the manubrium sterni. When the position of the tip of the catheter in a central vein had been confirmed by the pressure tracing, electrical damping was used to reduce the respiratory swing.

Cardiac output was measured using the dye-dilution technique. A Waters XP 302 densitometer and earpiece were used to obtain curves resulting from the injection of indocyanine green dye through the central venous catheter. The area under each curve was calculated by the methods of Williams, O'Donovan, and Wood (1966), the calibration being achieved by passing samples of the patient's blood containing known amounts of dye through the glass. 
blood chamber from a Waters cuvette placed between the jaws of the earpiece.

The electrocardiogram was recorded throughout the procedure, and heart rate subsequently counted from the tracing.

After induction of anaesthesia, the patient was placed in a 15° head-down position. Before the commencement of surgery, four recordings of cardiac output were made at two-minute intervals, as a control. Carbon dioxide was then introduced into the peritoneal cavity through a percutaneous (Verres) needle, until the abdomen was sufficiently distended for laparoscopy to be performed. The resulting abdominal pressure was in the range 15-20 cm H$_2$O. This took on average four minutes and the sterilization procedure by tubal diathermy was carried out. After the abdomen was inflated with carbon dioxide, four further recordings of cardiac output were made at two-minute intervals. Oxprenolol (Trasicor) 2 mg was then injected intravenously, over a period of 30 seconds, and three more recordings made, again at intervals of two minutes.

Thus a total of eleven measurements of cardiac output were made for each patient: four during the control period, four after inflation of the abdomen with carbon dioxide, and three after the injection of oxprenolol.

In five of the seven patients, measurements of pH, P$_{CO_2}$, and standard bicarbonate were made on capillary blood just before insufflation with carbon dioxide, and again 8 minutes after the gas had been introduced into the peritoneal cavity.

Statistical analysis was done using Student's $t$-test. In the case of peritoneal insufflation the 4 values taken after insufflation were compared with the 4 control values. With oxprenolol, the last value before the drug was given was compared with the 3 values after its administration. In addition to comparing the mean values, we have compared the maximum changes seen in each patient without regard to time.

**RESULTS**

Carbon dioxide insufflation (Table I).

Cardiac output did not change significantly, rising in some patients and falling in others. The individual results are shown in figure 1.

There was a significant increase in mean arterial pressure, which rose from a mean control value of 69 mm Hg to a mean value of 87 mm Hg ($P<0.05$). The average of the maximum recording in each patient was 91 mm Hg. The individual results are shown in figure 2.

Central venous pressure rose markedly from a mean control value of 8.4 mm Hg to a mean value of 11.4 mm Hg ($P<0.05$). The mean maximum value was 13.8 mm Hg. The individual results are shown in figure 3.

Heart rate also increased significantly from a mean control value of 81 beats/min to a mean value of 97 beats/min ($P<0.01$). The mean maximum value was 101 beats/min. Individual results are shown in figure 4.

The mean increase in P$_{CO_2}$ was 8.4 mm Hg (SD 3.4), and applying the paired $t$-test this change was found to be statistically significant ($P<0.01$). The mean increase in hydrogen ion concentration was 4.53 n.equiv/l. (SD 1.44), which again was statistically significant ($P<0.005$). In no case was there any change in standard bicarbonate before and after peritoneal insufflation.

**Table I.** Cardiac output, mean arterial pressure, central venous pressure and heart rate, before and after peritoneal insufflation with carbon dioxide. The mean values (± SD) from seven individuals are given. The mean maximal change in individual patients without regard to time is also shown. Statistical significance is given for both the mean change and the mean maximal change.

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>Control</th>
<th>CO$_2$ Insufflation</th>
<th>Mean change</th>
<th>Mean max. change</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M (SD)</td>
<td>M (SD)</td>
<td>t P</td>
<td>t P</td>
</tr>
<tr>
<td>8</td>
<td>4.2 (4.4)</td>
<td>4.29 (0.99)</td>
<td>-0.03 (0.75)</td>
<td>&gt;0.9 (1.34)</td>
</tr>
<tr>
<td>-6</td>
<td>4.4 (4.2)</td>
<td>4.1 (4.3)</td>
<td>&gt;0.9 (0.9)</td>
<td>&gt;0.9</td>
</tr>
<tr>
<td>-4</td>
<td>4.4 (4.4)</td>
<td>4.1 (4.3)</td>
<td>17.8 (7.2)</td>
<td>&lt;0.05 (8.0)</td>
</tr>
<tr>
<td>-2</td>
<td>4.2 (4.4)</td>
<td>4.6 (1.07)</td>
<td>(25.7%)</td>
<td>(31.0%)</td>
</tr>
<tr>
<td>Cardiac Output (l/min)</td>
<td>69 (11.5)</td>
<td>81 (14.2)</td>
<td>17.8 (7.2)</td>
<td>&lt;0.05 (8.0)</td>
</tr>
<tr>
<td>Mean arterial pressure (mm Hg)</td>
<td>69 (11.5)</td>
<td>81 (14.2)</td>
<td>17.8 (7.2)</td>
<td>&lt;0.05 (8.0)</td>
</tr>
<tr>
<td>Central venous pressure (mm Hg)</td>
<td>8 (1.8)</td>
<td>10 (2.6)</td>
<td>3.0 (1.5)</td>
<td>&lt;0.05 (2.2)</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>80 (7.7)</td>
<td>81.3 (8.8)</td>
<td>15.7 (4.8)</td>
<td>&lt;0.01 (7.0)</td>
</tr>
</tbody>
</table>

Note: Parentheses indicate percentage change.
Administration of oxprenolol (Table II).

Cardiac output fell slightly in all cases but this did not reach statistical significance, the mean maximum fall being 16%. In 3 cases the cardiac output after oxprenolol was lower than the pre-insufflation values, but in no case was this considered to be of clinical importance.

Mean arterial pressure fell from 91 mm Hg to 84 mm Hg. Central venous pressure rose further in every case to reach a mean maximum of 16 mm Hg. In 2 cases it exceeded 20 mm Hg. Heart rate fell in every case, the mean maximum fall being 15 beats/min. Only the fall in heart rate reached statistical significance (P<0.025).

DISCUSSION

Insufflation of the peritoneal cavity with carbon dioxide is most likely to affect the circulation in two ways. First, the rising PaCO₂, which is especially

Fig. 1. Cardiac output of seven patients before and after peritoneal insufflation with carbon dioxide and after administration of oxprenolol.

Fig. 2. Mean arterial pressure of seven patients before and after peritoneal insufflation with carbon dioxide, and after administration of oxprenolol.

Fig. 3. Central venous pressure of seven patients before and after peritoneal insufflation with carbon dioxide, and after administration of oxprenolol.

Fig. 4. Heart-rate of seven patients before and after peritoneal insufflation with carbon dioxide, and after administration of oxprenolol.
**CIRCULATORY EFFECTS OF CARBON DIOXIDE INSUFFLATION**

TABLE II. Effect of injection of oxprenolol while the peritoneal cavity was inflated with carbon dioxide. The mean (±SD) of the last values before administration of the drug for cardiac output, mean arterial pressure, central venous pressure and heart rate are compared with the mean values of these parameters after administration. The mean maximal change in individual patients without regard to time is also shown. Statistical significance is given for these comparisons.

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>CO₂ insufflation</th>
<th>Oxprenolol</th>
<th>Mean change (SD)</th>
<th>t</th>
<th>P</th>
<th>Mean max. change (SD)</th>
<th>t</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M (SD)</td>
<td>+2</td>
<td>+4</td>
<td>+6</td>
<td></td>
<td>M (SD)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiac output (L/min)</td>
<td>4.6 (1.4)</td>
<td>4.0</td>
<td>4.1</td>
<td>4.2</td>
<td>4.1 (1.12)</td>
<td>-0.50 (0.44)</td>
<td>0.74 (4.0)</td>
<td>&lt;0.5 &gt;0.4 (11%) &lt;0.7 (16%) 1.10 (0.3 &gt;0.2</td>
</tr>
<tr>
<td>Mean arterial pressure (mm Hg)</td>
<td>90.7 (16.0)</td>
<td>86</td>
<td>83</td>
<td>82</td>
<td>83.9 (19.43)</td>
<td>-6.8 (5.5)</td>
<td>0.72 (7.0)</td>
<td>&lt;0.5 &gt;0.4 (7%) &lt;0.4 &gt;0.3 (10%) 0.91 (0.3 &gt;0.2</td>
</tr>
<tr>
<td>Central venous pressure (mm Hg)</td>
<td>13.7 (3.4)</td>
<td>15</td>
<td>16</td>
<td>16</td>
<td>15.8 (3.8)</td>
<td>2.1 (1.3)</td>
<td>1.09 (1.3)</td>
<td>&lt;0.3 &gt;0.2 (13%) &lt;0.3 &gt;0.2 (1.3) 1.23 (0.3 &gt;0.2</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>96.5 (10.3)</td>
<td>86</td>
<td>83</td>
<td>84</td>
<td>84.3 (8.3)</td>
<td>-12.2 (8.5)</td>
<td>2.62 (8.6)</td>
<td>&lt;0.025 (12.6%) &lt;14.9 (15.3%) &lt;0.020 (0.2 &gt;0.2</td>
</tr>
</tbody>
</table>

marked when the patient breathes spontaneously, will have a positive inotropic effect on the myocardium and a pressor effect on the peripheral vessels, probably owing to the release of catecholamines. Secondly, the rise in abdominal pressure will cause a varying degree of obstruction to the flow of blood along the inferior vena cava. Thus these factors will tend to oppose each other.

Our results showed that cardiac output was relatively unaffected, being increased in some patients and decreased in others. Arterial pressure, heart rate, and central venous pressure on the other hand all rose significantly in almost every case. The rise in central venous pressure is not easy to explain but may be analogous to the rise seen when the abdomen is externally compressed e.g., during lumbar puncture to increase the flow of cerebrospinal fluid. That the increase is not totally accounted for by mechanical factors is indicated by the somewhat slow fall observed when the abdomen is deflated. On the other hand the central venous pressure changes rather more rapidly than would be expected if the response was only due to hypercarbia. In two cases an oesophageal balloon was used to determine intrathoracic pressure. No change was observed, though there was naturally an increased respiratory swing. Smith and associates (1971) measured a small increase in intrathoracic pressure presumably due to the increase in the mean airway pressure during artificial ventilation. No increase in pressure should occur if the airway is patent and the patient is breathing spontaneously. However as the diaphragm rises, the lung volume must be decreased. Whether this can cause any change in pulmonary vascular resistance we are unable to say from our data.

The rise in arterial pressure and heart rate probably indicate the catecholamine release effected by hypercarbia. However the external pressure applied to the abdominal portion of the aorta will also be a factor in increasing peripheral resistance.

Smith and associates (1971) have reported essentially similar results to those seen by us in regard to arterial pressure, central venous pressure, and pulse rate even though their patients were supine and artificially ventilated, while ours were head-down and breathing spontaneously. However they thought that cardiac output was consistently increased by about 20% when the abdominal pressure was moderately raised. Our data do not confirm this and it is difficult to understand how raising the pressure within the abdomen can cause more than a very transient increase in venous return.

It should always be remembered that there are pitfalls in observing the cardiovascular state before and during surgery. It is not uncommon to find patients who are anaesthetized but undisturbed showing low values for arterial pressure, heart rate, and cardiac output. They thus appear to be in a state of relative vagal overactivity. A rapid rise in these parameters can occur as soon as painful stimuli occur at operation indicating a shift in parasympathetic/sympathetic balance. Such changes have to be differentiated, if possible, from those occurring in response to specific manoeuvres in the surgical procedure.

Previously we have reported on the changes occurring in the haemodynamics when beta-adrenergic receptor blockers were given during halothane anaes-
thsea (Stephen, Davie, and Scott, 1971). This work showed a fall in cardiac output, heart rate, and arterial pressure, and a rise in central venous pressure. However, the study was designed to show the effects of these drugs in undisturbed patients prior to surgery and it was thought that the results might well be different if the patients were in a state of sympathetic stimulation as occurs at the height of hypercarbia during laparoscopy. In the present study oxprenolol 2.0 mg was given intravenously at just this time. There was a small fall in cardiac output in 6 of the 7 subjects though it rapidly recovered in 3 of them. Heart rate fell, again in 6 out of 7, and a small fall in arterial pressure occurred in 5. Central venous pressure on the other hand rose in all cases from the already high level during the peritoneal insufflation. Indeed in 2 cases it rose to over 20 mm Hg and in 5 it was over 15 mm Hg. Rapid falls always occurred after the abdomen was deflated though central venous pressure seldom reached the pre-laparoscopy level within 6 minutes. These results could be interpreted as indicating that vagal activity is a major factor in the rise of central venous pressure during laparoscopy and that its effect is enhanced by beta-blockade.

ACKNOWLEDGEMENTS

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REFERENCES


LES EFFETS CIRCULATOIRES DE L'INSUFFLATION DE L'ANHYDRO CARBONE DANS LA CAVITE PERITONEALE POUR LAPAROSCOPIE

SOMMAIRE

On mesure le debit cardiaque, la pression artetiere moyenne, la pression veineuse centrale et la frequence cardiaque chez sept patients, subissant une laparoscopie.

Les determinations eurent lieu avant et apres l'insufflation de l'anhydride carbonique dans le peritone, ainsi qu'apres l'injection intraveineuse de 2 mg d'oxprenolol. Aucune modification significative du debit cardiaque ne s'observa apres insufflation peritonale mais il y eut une augmentation significative de la pression artetiere moyenne, de la pression veineuse centrale et de la frequence cardiaque. Apres administration d'oxprenolol il y eut une reduction du debit cardiaque, de la pression artetiere moyenne et de la frequence cardiaque, tandis que la pression veineuse centrale continua a augmenter. L'augmentation de la frequence du coeur seule etait statistiquement significative. L'analyse des gaz sanguins chez 5 patients montra une augmentation de la Paco2 et une reduction du pH apres insufflation de gaz carbonique. L'importance de ces observations est discutee.

KREISLAUFEFFEKTE EINER LAPAROSKOPISCHEN INSUFFLATION DER BAUCHHOHLE MIT KOHLENSTLEXID

ZUSAMMENFASSUNG


EFECTOS CIRCULATORIOS DE LA INSUFICLACION DE ANHIDRIDO CARBONICO EN LA CAVIDAD PERITONEAL PARA LA LAPAROSCOPIA

RESUMEN

Fueron efectuadas mediciones del gasto cardíaco, presión arterial media, presión venosa central y frecuencia cardíaca en siete pacientes sometidos a laparoscopia. Las mediciones fueron practicadas antes y después de insuflación del peritoneo con anhidrido carbónico y otra vez después de la inyección intravenosa de 2 mg de oxprenolol. La insuflación peritoneal no fue seguida por un cambio significativo en el gasto cardíaco, pero hubo un incremento significativo en la presión arterial media, presión venosa central y frecuencia cardíaca. Después de la administración de oxprenolol hubo un descenso en el gasto cardíaco, presión arterial media y frecuencia cardíaca, y la presión venosa central aumentó aún más. Solamente la modificación de la frecuencia cardíaca fue estadisticamente significativa. El análisis de los gases sanguíneos en cinco pacientes mostró un aumento en la PaCO2 y disminución en el pH después de la insuflación con anhidrido carbónico. Son discutidas las implicaciones de estas observaciones.