PRELIMINARY COMMUNICATIONS

Cardiovascular Effects of Peritoneal Insufflation of Carbon Dioxide for Laparoscopy

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Summary

We have investigated, in 13 artificially ventilated and anaesthetized patients, the cardiovascular effects of peritoneal insufflation of carbon dioxide preparatory to laparoscopy. Stepwise increases of intra-abdominal pressure up to a maximum of 25 cm H_2O were accompanied by increases of airway pressure, intrathoracic pressure, central venous pressure, and femoral venous pressure and by signs of cardiovascular stimulation with mild tachycardia and hypertension. End-tidal carbon dioxide tension rose only slightly. The anaesthetic technique used provided good surgical conditions; our results suggest that it does not impose undue strain on the homoeostatic ability of the patient's cardiovascular system.

Introduction

Laparoscopy for clinical purposes was first performed by Jacobaeus in 1910, and, following advances in optical and lighting systems and the work of Steptoe (1965, 1969), this technique is now widely used in gynaecological practice. During the past year we have heard of several cases of acute cardiovascular collapse occurring during laparoscopy, and recent correspondence (Arthure, 1970; Scott, 1970; Herrera-Llerandi, 1971; Loveday, 1971; Muldoon, 1971) has indicated some of its potential hazards.

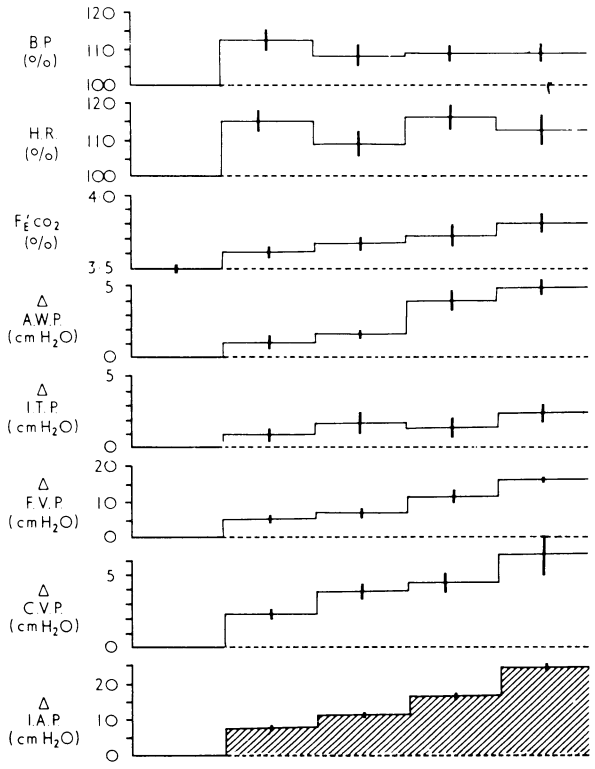
An essential preliminary to laparoscopy is the insufflation of several litres of carbon dioxide into the peritoneal cavity. The respiratory effects of this have been extensively investigated (Hodgson *et al.*, 1970; Seed *et al.*, 1970). The present communication describes some of the effects of the procedure on the cardiovascular system.

Methods

Thirteen patients (average age 23 years) undergoing laparoscopy under general anaesthesia were studied. All had consented to the investigation. After premedication with papaveretum 15 mg and hyoscine 0·3 mg general anaesthesia was induced with thiopentone 250-350 mg followed by endotracheal intubation performed under suxamethonium-induced muscular relaxation. Anaesthesia was maintained with 70% nitrous oxide and 30% oxygen, and the patient was artificially ventilated with a Howell's ventilator at a constant minute volume to give an end-tidal P_{CO_2} of 25 ± 2 mm Hg, muscular relaxation being achieved with alcuronium (0·2 mg/kg body weight initially, supplemented by one-quarter to one-third of this dose when necessary).

With the patient in the horizontal position carbon dioxide was insufflated through a Verres's needle into the peritoneal cavity in aliquots of 1 or 2 litres. After the introduction of each aliquot we measured intra-abdominal pressure (I.A.P.), central venous pressure (C.V.P.), femoral venous pressure (F.V.P.), intrathoracic pressure (I.T.P.), peak airway pressure (A.W.P.), end-tidal CO_2 concentration ($F_{\bar{E}}CO_2$), arterial blood pressure (B.P.), heart rate (H.R.), and rhythm. Intra-abdominal pressure was measured by a saline manometer or a calibrated anaeroid manometer. Central venous pressure and femoral venous pressure were measured via percutaneously-inserted catheters and electromanometers (Devices). Intrathoracic pressure was measured by an oesophageal balloon 8 cm by 1 cm, as described by Milic-Emili *et al.* (1964). Peak airway pressure was measured by an anaeroid manometer and end-tidal CO_2 concentration by a fast-response infrared CO_2 analyser (Hartmann and Braun, URAS III). Heart rate and rhythm were monitored by radiotelemetry to an E.C.G. recorder and rate meter. Arterial blood pressure was measured by sphygmomanometry.

Femoral venous pressure was not measured in patients with a history of venous disorder or who were pregnant or taking oral contraceptives.



Results of investigations in the 13 patients studied.

Results

Each aliquot of carbon dioxide caused an increase in intra-

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abdominal pressure of between 4 and 8 cm H_2O (see Chart). These increases were accompanied by significant rises in central and femoral venous pressures and by smaller (but still significant) rises in intrathoracic and peak airway pressures. There was an increase in heart rate and mean blood pressure of the order of 10%. End-tidal CO_2 concentration rose by 0·39% ($\simeq$ 2 mm Hg). No cardiac dysrhythmias occurred.

Discussion

Our results show that peritoneal insufflation of CO_2 causes a rise in central venous pressure (about one-third as great as the rise of intra-abdominal pressure) accompanied by a smaller rise in intrathoracic pressure, suggesting an increase in right atrial filling pressure. This would be expected to produce an increase in cardiac output (Guyton, 1963) which would be accentuated by the simultaneous increase of arterial P_{CO_2} (Prys-Roberts *et al.*, 1967). The observed increases in pulse rate and mean arterial blood pressure support this contention.

We attempted to measure cardiac output in these patients by dye dilution with indocyanine green and a photoelectric earpiece, and though interpretation of the dye curves was confounded by stasis of blood in the microcirculation of the ear pinna, our findings are consistent with an increase in cardiac output of the order of 20%. Hodgson *et al.* (1970) found a 25% increase in cardiac output in one patient under similar conditions.

Desmond and Gordon (1970) postulated that increased intra-abdominal pressure might cause a reduction in cardiac output by impedance of "venous return," analogous to that which occurs in the supine hypotensive syndrome of pregnancy (Holmes, 1960). We found no evidence for this at intra-abdominal pressures up to 20-25 cm H_2O, and our results suggest that cardiac output may be increased by this procedure. However, our unpublished observations in a smaller number of patients in whom the intra-abdominal pressure was increased above 30 cm H_2O suggest that under these circumstances blood may be dammed back in the legs with a consequent reduction of central venous pressure and presumably of cardiac output.

There are several possible anaesthetic techniques for laparoscopy. The one used in this investigation would seem to be generally satisfactory, since our results show that the patients whom we studied were able to compensate adequately for the stress imposed on their cardiovascular system by laparoscopy. The patients did not receive inhalational anaesthetic agents, such as halothane, which are known to cause cardiovascular depression. They were artificially ventilated so that CO_2 retention was not a problem. They were also well relaxed so that good visualization of the pelvic contents could be obtained without a large increase in intra-abdominal pressure. With this technique the high intra-abdominal pressures used by other workers—for example, up to 50 cm H_2O (Baratz and Karis, 1969), and up to 40 cm H_2O (Steptoe, 1967;)—are unnecessary.

Further studies of the cardiovascular effects of peritoneal insufflation of CO_2, including measurements of cardiac output, are continuing.

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References

Arthure, H. (1970). *British Medical Journal*, 4, 492.
Baratz, R. A., and Karis, J. H. (1969). *Anesthesiology*, 30, 463.
Desmond, J., and Gordon, R. A. (1970). *Canadian Anaesthetists' Society Journal*, 17, 378.
Guyton, A. C. (1963). *Cardiac Output and its Regulation*. Philadelphia, Saunders.
Herrera-Llerandi, R. (1971). *British Medical Journal*, 2, 47.
Hodgson, C., McClelland, R. M. A., and Newton, J. R. (1970). *Anaesthesia*, 25, 382.
Holmes, F. (1960). *Anaesthesia*, 15, 298.
Jacobaeus, H. C. von (1910). *Münchener medizinische Wochenschrift*, 57, 2090.
Loveday, R. (1971). *British Medical Journal*, 1, 348.
Milic-Emili, J., Mead, J., Turner, J. M., and Glauser, E. M. (1964). *Journal of Applied Physiology*, 19, 207.
Muldoon, M. J. (1971). *British Medical Journal*, 1, 51.
Prys-Roberts, C., Kelman, G. R., Greenbaum, R., and Robinson, R. H. (1967). *British Journal of Anaesthesia*, 34, 533.
Seed, R. F., Shakespeare, T. F., and Muldoon, M. J. (1970). *Anaesthesia*, 25, 223.
Steptoe, P. C. (1965). *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 72, 535.
Steptoe, P. C. (1967). In *Laparoscopy in Gynaecology*, p. 32. Edinburgh, Livingstone.
Steptoe, P. C. (1969). *Proceedings of the Royal Society of Medicine*, 62, 439.

MEDICAL MEMORANDA

Adipsia and Hypothermia after Subarachnoid Haemorrhage

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The virtual inaccessibility of the diencephalon to direct study has allowed detailed knowledge of the functions of the hypothalamus in man to accumulate only slowly. Understanding of the pathogenesis of human hypothalamic disease has therefore heavily depended on experimental analysis in animals. During recent years such animal work has resulted in the definition and understanding of several classical problems of the hypothalamus. Jenkins *et al.* (1969) produced biochemical evidence of hypothalamic-pituitary-adrenal disturbance after subarachnoid haemorrhage. The present case showed clinical features attributable to a lesion of the anterior hypothalamus.

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Case Report

A 52-year-old woman with no known history of hypertension had a subarachnoid haemorrhage on 15 March 1969. On admission to hospital she was comatose and had a blood pressure of 180/120. Examination showed no focal neurological signs but she had two large subhyaloid haemorrhages in the left fundus and scattered small haemorrhages in both fundi. Lumbar puncture produced blood-stained cerebrospinal fluid.

Bilateral carotid angiography showed a moderately-sized irregular anterior communicating artery aneurysm filling spontaneously from the left. Bilateral subclavian angiograms were negative. The aneurysm was clipped through a right frontal craniotomy. When