Tolerance of laparoscopy for resection of phaeochromocytoma

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Summary
We describe two patients who underwent resection of phaeochromocytoma by a laparoscopic approach. Although outcome from surgery was successful, there was marked variability in hormonal and haemodynamic changes. In one patient, despite an infusion of nicardipine, peritoneal insufflation produced a marked increase in catecholamine concentrations associated with transient but intense vasoconstriction, but there was no change in the second patient. In both patients, exsufflation caused no significant haemodynamic changes in spite of the high doses of nicardipine given throughout the procedures. (Br. J. Anaesth. 1996; 77: 795–797)

Key words
Surgery, laparoscopy, Complications, phaeochromocytoma. Carbon dioxide, pneumoperitoneum.

Surgical removal of phaeochromocytoma is a risky procedure because of preoperative cardiovascular changes, particularly during surgical manoeuvres. Preoperative preparation, completed during operation by administration of antihypertensive agents, enables the anaesthetist to largely control the haemodynamic instability. However, acute complications may still occur. The laparoscopic approach described recently seems to be of great interest as it may decrease postoperative morbidity and it allows viewing of the whole of the abdominal cavity in order to reveal possible multiple tumour sites. Nevertheless, as the pneumoperitoneum may induce haemodynamic disturbances, the safety of this technique remains questionable. We report two cases in which we measured the haemodynamic changes induced by pneumoperitoneum during removal of phaeochromocytoma by a laparoscopic approach.

Case reports

PATIENT NO. 1
A 32-yr-old woman (50 kg, 158 cm) was referred with a phaeochromocytoma of the right adrenal gland, manifest clinically as hypertension, sweating and severe headache. Urinary catecholamine concentrations were 34 300 nmol/24 h (normal values 150–1600 nmol). Echocardiography revealed left ventricular hypertrophy with a wall thickness of 17 mm. Systolic arterial pressure (SAP), recorded continuously, averaged 165 mm Hg with peaks of 240 mm Hg. Continuous ECG showed several episodes of supraventricular arrhythmia. Treatment with nicardipine 150 mg day⁻¹ and labetalol 200 mg day⁻¹ for 10 days did not provide satisfactory haemodynamic control.

One hour before surgery and after oral premedication with hydroxyzine 100 mg and alprazolam 0.5 mg, SAP was 202 mm Hg and heart rate 109 beat min⁻¹. Anaesthesia was induced with fentanyl 0.5 mg, midazolam 15 mg and vecuronium 6 mg, and maintained with an infusion of fentanyl 1 mg h⁻¹, midazolam 10 mg h⁻¹ and additional bolus doses of vecuronium. No inhalation agents were used and patient’s lungs were ventilated mechanically with 60% nitrogen in oxygen at a rate of 15 bpm with a tidal volume of 8–10 ml kg⁻¹, maintaining end-expired carbon dioxide concentration at 3.5–4%. After a bolus of 2 mg, an infusion of nicardipine 4 mg h⁻¹ was given to maintain SAP at 140–150 mm Hg.

A pulmonary artery catheter was inserted for continuous measurement of cardiac output (Intellithcath 8F-Catheter and Vigilance Monitor, Baxter, USA); additional monitoring included invasive measurement of arterial pressure, pulse oximetry, capnography and ECG. Blood sampling was hourly, performed to measure blood-gas tensions. Plasma concentrations of catecholamines were measured using high-pressure liquid chromatography (table 1). Normovolaemic haemodilution of 600 ml was undertaken using hydroxyethyl starch and 2000 ml were given to produce a pulmonary capillary wedge pressure of 18 mm Hg. After this 90-min preparation phase, the patient was placed in a left lateral decubitus position of 30°. A carbon dioxide pneumoperitoneum with a pressure of 10 mm Hg was undertaken using hydroxyethyl starch and 2000 ml were given to produce a pulmonary capillary wedge pressure of 18 mm Hg. After this 90-min preparation phase, the patient was placed in a left lateral decubitus position of 30°. A carbon dioxide pneumoperitoneum with a pressure of 10 mm Hg was produced for 10 min and then suddenly, a hypertensive peak (244/131 mm Hg) with a reduction in heart rate was observed, but without any variation in cardiac output (fig. 1). This episode was controlled rapidly with nicardipine 5 mg i.v. followed by an infusion of 6 mg h⁻¹.

The various vascular tumour pedicles were isolated successively using four trocars. Despite continuous administration of nicardipine, vascular
clamping near the tumour caused marked haemodynamic changes. Supplementary bolus doses of nicardipine and bolus doses of esmolol were given when tachycardia or an increase in cardiac output, or both, were observed. After total vascular detachment of the tumour (fig. 1: double arrow), nicardipine was stopped. The tumour (6 cm in diameter) was then extracted via a MacBurney incision. The total preoperative doses of nicardipine and esmolol were 96 and 170 mg, respectively. At the end of the procedure, core body temperature was 36.2°C. Throughout the operation, \( P_{\text{aCO}_2} \) had been maintained at 4.1–4.9 kPa. The postoperative course was uneventful and antihypertensive treatment was not required. Analgesia comprised non-steroidal anti-inflammatory analgesics only. The patient’s trachea was extubated 6 h later in the recovery room and she was transferred to the surgical unit. Oral feeding was resumed at 24 h and the patient was discharged on the third day after operation.

PATIENT NO. 2

A 64-yr-old woman (54 kg, 147 cm) was referred with a phaeochromocytoma of the left adrenal gland. The clinical picture comprised arterial hypertension, sweating, palpitations and Raynaud’s phenomenon. Urinary concentrations of catecholamines were 13 200 nmol/24 h (normal values 150–1600 nmol). Echocardiography revealed left ventricular hypertrophy with a wall thickness of 19 mm with moderate aortic stenosis. Treatment with nicardipine 100 mg day\(^{-1}\) for 20 days stabilized arterial pressure.

One hour before surgery and after oral premedication with hydroxyzine 100 mg and alprazolam 0.5 mg, SAP was 141 mm Hg and heart rate 88 beat \( \text{min}^{-1} \). Anaesthesia was induced with fentanyl 0.4 mg, midazolam 10 mg and vecuronium 6 mg, and maintained by continuous infusion of fentanyl 0.4 mg h\(^{-1}\), midazolam 4 mg h\(^{-1}\) and additional doses of vecuronium. SAP was then stabilized at 90–100 mm Hg without administration of nicardipine. No inhalation agents were used and haemodynamic and ventilatory management, and blood-gas tensions were measured, as described for patient No. 1. Normovolaemic haemodilution of 500 ml was undertaken and 1750 ml were given to produce a pulmonary capillary wedge pressure of 15 mm Hg. The patient was then placed in a right lateral decubitus position of 30°. A bolus of nicardipine 1.5 mg was administered, followed by an infusion of 6 mg h\(^{-1}\). Haemodynamic disturbances caused by tumour manipulation were treated in the same way as those described for patient No. 1. The total preoperative doses of nicardipine and esmolol were 36 and 150 mg, respectively. At the end of the procedure, core body temperature was 35.9°C. Throughout the operation, \( P_{\text{aCO}_2} \) had been maintained at 3.9–4.4 kPa. The postoperative course was uneventful and antihypertensive treatment was not required. Analgesia comprised non-steroidal anti-inflammatory analgesics only. The patient’s trachea was extubated 4 h later in the recovery room and she was transferred to the surgical unit. Oral feeding was resumed at 24 h and the patient was discharged on day 4 after operation.

Discussion

The cardiovascular changes induced by phaeochromocytoma are usually aggravated by the stress of surgery. Classically, arterial hypertension occurs, caused by intense vasoconstriction, often associated with myocardial hyperexcitability and arrhythmias.\(^{24}\) Adrenalectomy using a laparoscopic approach has been proposed with the aim of diminishing the stress of laparotomy and avoiding direct manipulation of

Table 1 Variations in plasma concentration of noradrenaline/adrenaline (nmol litre\(^{-1}\)) immediately before insufflation of the pneumoperitoneum (prePNP), 5 min (PNP), 1 h (PNP +1), 3 h (PNP +3) and 4 h (PNP +4) after insufflation of the pneumoperitoneum, and 30 min after exsufflation (postPNP). (Normal values: noradrenaline < 4 nmol litre\(^{-1}\); adrenaline < 1 nmol litre\(^{-1}\). Dopamine concentrations were normal

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<th>PrePNP</th>
<th>PNP</th>
<th>PNP +1</th>
<th>PNP +3</th>
<th>PNP +4</th>
<th>PostPNP</th>
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<tr>
<td>Patient No. 1</td>
<td>22/2</td>
<td>79/3.2</td>
<td>57/3.6</td>
<td>154/9.8</td>
<td>8/2.3</td>
<td>107/3.3</td>
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<tr>
<td>Patient No. 2</td>
<td>12/1.1</td>
<td>8.7/1.2</td>
<td>10.4/1.3</td>
<td>36/7.6</td>
<td>4.9/5.1</td>
<td>9.1/1.4</td>
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the gland. However, creation of a pneumoperitoneum by carbon dioxide insufflation carries problems as it causes an increase in intra-abdominal pressure and leads to absorption of carbon dioxide. The increase in intra-abdominal pressure causes a decrease in cardiac index by reduction of venous return and an increase in peripheral vascular resistance. Excessive absorption of carbon dioxide may lead to sympathetic stimulation with, in extreme cases, arrhythmias induced by hypercapnia.

Therefore, the use of laparoscopy for pheochromocytoma is only justifiable if it is possible to rigorously control preload, afterload and arterial P\textsubscript{CO\textsubscript{2}}. Our experience in the anaesthetic management of laparoscopy has led us to use calcium-channel blockers to control vasoconstriction associated with pneumoperitoneum. Moreover, with hyperventilation, hypercapnia is generally avoided. The anaesthetic technique used here did not differ from that used for conventional pheochromocytoma surgery. The use of a potent analgesic at a high dose and a benzodiazepine proved to be sufficient to control surgical stress.

Calcium-channel blockers, such as nicardipine, are currently the anti-hypertensive agent of choice during pheochromocytoma resection. Meurisse and colleagues observed good haemodynamic stability during laparoscopic removal of pheochromocytoma. However, these authors admitted that insufflation of the pneumoperitoneum was a critical time. In this study, although the conditions for pneumoperitoneum were exactly the same, the responses observed in the two patients were not comparable. In our first patient, after insufflation, noradrenaline concentrations increased to three times pre-insufflation concentrations, which was probably an indication of intense mechanical stimulation of the tumour by intra-abdominal pressure. Despite preliminary infusion of nicardipine, the immediate consequence was intense vasoconstriction with a marked increase in arterial pressure. This hypertensive bout was nevertheless controlled rapidly by an additional bolus of nicardipine. In spite of the lower doses of nicardipine in our second patient, there was no vasopressor response to the pneumoperitoneum and no variation in catecholamine concentrations was recorded. In contrast, in both patients, insufflation of the pneumoperitoneum had no effect on cardiac output, whereas a 15–20% reduction in cardiac index is usually observed during conventional laparoscopic surgery. The explanation for the stability was probably the preload optimization by monitoring of pulmonary capillary wedge pressure. Recently, Giebler and colleagues performed pheochromocytoma resection using a capnoretroperitoneoscopic technique. Although they were not reported in detail, the haemodynamic changes seem to have been well tolerated.

Tumour manipulation causes marked increases in arterial pressure, heart rate and cardiac output. Administration of nicardipine or esmolol, or both, makes it possible to maintain satisfactory haemodynamic stability. However, considerable release of noradrenaline was observed in patient No. 1. In common with insufflation, successive laparoscopic manipulation may lead to significant tumour stimulation. Total tumour removal leads to a rapid decrease in catecholamine concentrations with a parallel decrease in cardiac output and vascular resistances. However, in both patients, exsufflation caused no significant haemodynamic changes in spite of the large doses of nicardipine.

References