Anaesthesia for laparoscopic cholecystectomy in a patient with Eisenmenger’s syndrome

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Summary

We describe the management of a patient with Eisenmenger’s syndrome presenting for laparoscopic cholecystectomy. Of prime concern was maintenance of systemic vascular resistance and this was achieved using infusion of noradrenaline started before induction of anaesthesia and continued after operation. Avoidance of other factors that could potentially increase right to left shunt flow contributed to the successful outcome. (Br. J. Anaesth. 1997; 79: 810–812).

Key words
Complications, Eisenmenger’s syndrome, Anaesthesia, general. Surgery, laparoscopy.

In 1897, Eisenmenger first described post-mortem evidence of pulmonary vascular disease in the presence of a large ventricular septal defect and right ventricular hypertrophy.1 This became known as Eisenmenger’s complex. The syndrome was redefined in 1958 by Wood to imply pulmonary hypertension at the systemic level caused by high pulmonary vascular resistance with reversed or bidirectional shunt via a large ventricular septal defect.2 The actual position of the shunt is immaterial. Indeed, Eisenmenger’s syndrome includes any condition in which communication between the pulmonary and systemic circulations gives rise to pulmonary vascular disease causing a right to left shunt.3 Surgical correction of the original defect merely exacerbates the pulmonary hypertension and may precipitate right ventricular failure.

With improved management of congenital cardiac disease, Eisenmenger’s syndrome is becoming very rare. However, these patients occasionally present for incidental non-cardiac surgery. We describe the successful management of a patient with this syndrome presenting for laparoscopic cholecystectomy.

Case report

A 46-yr-old woman was scheduled for laparoscopic cholecystectomy. Cardiac catheterization at 5 yr of age revealed a small ventricular septal defect (VSD) and severe pulmonary hypertension, greater than systemic pressure. In the following years she was admitted on several occasions with pulmonary infections which were treated without mishap. Cardiac catheterization at 26 yr of age showed a VSD with severe pulmonary hypertension and a right to left shunt. The main pulmonary artery pressure was 116/70 mm Hg, aortic pressure was 110/80 mm Hg and pulmonary vascular resistance was approximately 85% of the systemic level. With the patient breathing 100% oxygen, pulmonary vascular resistance decreased by 50% but right ventricular and pulmonary arterial pressures remained unchanged. A year later she underwent a full-term pregnancy with forceps delivery under extradural analgesia and was later transferred to the regional cardiac unit for post-partum monitoring. Apart from a few episodes of desaturation that responded to fluid replacement, recovery was uneventful.

She was in full-time employment at the time of surgery and was not receiving any medication apart from analgesics for gall bladder pain. She complained of shortness of breath on climbing stairs but could manage decorating her own house. On examination she was slim, not obviously cyanosed and showed no clubbing. She had no signs of increased jugular venous pressure, heart rate was 80 beat min−1 and arterial pressure was 120/80 mm Hg. There was right ventricular lift, and auscultation revealed a grade III systolic murmur and loud second sound. The lungs were clear. Preoperative echocardiogram showed a hypertrophied right ventricle with good function and a right to left shunt through a small VSD. The electrocardiogram showed right axis deviation and auscultation revealed a grade III systolic murmur and loud second sound. The lungs were clear. Preoperative echocardiogram showed a hypertrophied right ventricle with good function and a right to left shunt through a small VSD. The electrocardiogram showed right axis deviation with a right ventricular strain pattern and occasional ventricular ectopic beats. Preoperative chest x-ray was reported as normal.

The operation was scheduled for the afternoon and diazepam 10 mg was given in the morning as premedication. Antibiotic prophylaxis for endocarditis included teicoplanin 400 mg and gentamicin 80 mg as she was allergic to penicillin. Heparin was given subcutaneously and continued twice daily.

On arrival in the anaesthetic room, peripheral oxygen saturation (SpO2) was 90% breathing air.

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This was similar to values measured previously on the ward. Breathing oxygen 5 litre min\(^{-1}\) via a face mask, this increased to 99%. Midazolam 2 mg was given and direct arterial and central venous pressure monitoring was established. Before induction, an infusion of noradrenaline 0.05 \(\mu\)g kg\(^{-1}\) min\(^{-1}\) was started. Fentanyl 100 \(\mu\)g and etomidate 18 mg were used to induce anaesthesia and neuromuscular block was achieved with vecuronium. Anaesthesia was maintained with 1–2% enflurane and oxygen enriched air \((F_{\text{O}_2} = 0.4)\) and additional doses of fentanyl to a total of 250 \(\mu\)g.

Shortly after transfer to theatre, arterial pressure decreased from 130/80 to 95/40 mm Hg with a decrease in oxygen saturation to 94%. Infusion of noradrenaline was increased to maintain systemic pressure. There appeared to be a direct correlation between systemic pressure and oxygen saturation and it was necessary to maintain arterial pressure at 160/90 mm Hg to keep oxygen saturation greater than 97%. When surgery was underway this was achieved with low doses of noradrenaline (range 0.03–0.1 \(\mu\)g kg\(^{-1}\) min\(^{-1}\)). The increase in end-tidal carbon dioxide after pneumoperitoneum was countered by increasing the frequency of ventilation rather than tidal volume. Intra-abdominal pressure was maintained at less than 15 mm Hg and a head up tilt of 10° was allowed.

Surgery was uneventful and on completion neuromuscular blocking agents were antagonized with neostigmine and glycopyrronium. The trachea was extubated and she was transferred to the intensive care unit, as planned, for postoperative monitoring. The noradrenaline infusion was weaned gradually to achieve with low doses of noradrenaline (range 0.03–0.1 \(\mu\)g kg\(^{-1}\) min\(^{-1}\)).

Discussion

The theoretical risks of anaesthesia in patients with Eisenmenger’s syndrome are considerable. The dangers of a decrease in systemic vascular resistance and subsequent increase in right to left shunt are well described. However, it has also been suggested that these patients do well with a variety of techniques.\(^4\) Nevertheless, there is agreement that the cornerstone of safe anaesthesia in such patients is maintenance of preoperative levels of systemic vascular resistance.\(^5\) Bird and Strunin anticipated the almost inevitable decrease in systemic vascular resistance at induction by administering a small dose of metaraminol during preoxygenation. Unfortunately, this led to hypertension and bradycardia followed by a sudden decrease in oxygen saturation remedied by administration of atropine.\(^6\) To avoid this we opted for a noradrenaline infusion which provided easy and accurate control of arterial pressure, especially important during different levels of surgical stimulation.

The uninterrupted feedback provided by direct intra-arterial pressure monitoring was essential but we decided not to use a pulmonary artery flotation catheter. We reasoned that the risks of misdirection into the left ventricle and cardiac arrhythmias outweighed the possible benefits in this particular case given the general condition of the patient and good biventricular function identified by echocardiography. The site of communication between the pulmonary and systemic circulations also implied that the data obtained could have been misleading.\(^5\)

An opioid-based premedication has been shown to be safe\(^2\) but we chose a benzodiazepine to avoid the risk of preoperative respiratory depression, hypoventilation and hypoxaemia which might have precipitated a cyanotic crisis. On arrival in the anaesthetic room, \(S_{\text{PO}_2}\) was 90% but delivery of oxygen by a simple face mask with a flow of 5 litre min\(^{-1}\) improved this to 99%. Hypoxaemia caused by right to left shunt is not usually reversible by oxygen therapy. Although pulmonary hypertension in this patient was greater than systemic levels, the improvement in \(S_{\text{PO}_2}\) may be explained by a vasodilator response of the pulmonary vasculature to oxygen with improvement in pulmonary arterial flow and decreased flow across the shunt. This had been demonstrated previously during cardiac catheter studies when this patient was 26 yr old. We were somewhat surprised that this element of reversibility had been maintained.

Problems associated with anaesthesia for laparoscopic procedures pose a higher risk in patients with Eisenmenger’s syndrome. Insufflation of the peritoneal cavity with carbon dioxide causes an increase in end-tidal carbon dioxide. Acidosis, arrhythmias and hypotension may follow, all of which can precipitate a shunt crisis. Equally, efforts to maintain normocapnia are often accompanied by increased pulmonary airway pressures, particularly as intra-abdominal pressure increases. The effect of this is to increase pulmonary vascular resistance further. The situation may be made worse if the Trendelenburg position is adopted during surgery. Fortunately, a slight head-up tilt is often the requirement for cholecystectomy, as in this case. Other possible complications associated with this type of surgery include pneumothorax and gas embolism. The latter is particularly important because of the direct route available, via the shunt, to the systemic circulation.

Early tracheal extubation in these patients is preferable because of the deleterious effect of positive pressure ventilation, and intraoperative
analgesia is thus best provided by short-acting opioids. Although it is not our usual practice to provide patient-controlled analgesia after laparoscopic cholecystectomy we felt that it was justified in this patient in order to minimize stress in the early postoperative period. We were disappointed that it marred an otherwise uneventful immediate postoperative phase and wonder if the intensive care setting may have encouraged over zealous use of the PCA system with subsequent respiratory depression.

The increase in exertional dyspnea noted after operation seemed to be directly related to the decrease in haemoglobin concentration. The fact that symptoms resolved after transfusion strengthened that view. The pathophysiology involved, however, is more contentious. In non-cardiac patients acute anaemia is associated with a decrease in the oxygen carrying capacity of the blood and an initial decrease in oxygen flux. The resultant tissue acidosis causes compensatory peripheral vasodilatation and increase in cardiac output. Anaemia may also contribute to a decrease in blood viscosity which reduces further peripheral vascular resistance. In patients with Eisenmenger's syndrome the dynamics are inevitably more complex. Unfortunately, without the benefit of further invasive investigation in this patient the relative importance of these mechanisms and their effect on flow across the shunt remain speculative.

References