Postoperative treatment of myocardial ischaemia by thoracic extradural local anaesthetic

G. BOCCARA, C. MANN, D. DUCHENNE, Y. POUZERATTE, P. COLSON

Summary
This case report describes an episode of postoperative myocardial ischaemia after total oesophagectomy that was successfully treated by extradural administration of local anaesthetic. Extension of sympathetic blockade in this manner resolved the myocardial ischaemia and haemodynamic disturbances experienced by the patient. (Br. J. Anaesth. 1998; 80: 848–849)

Keywords: myocardial ischaemia postoperative; analgesic techniques extradural; anaesthetics local; sympathetic nervous system block; surgery oesophageal

Thoracic extradural analgesia provides excellent postoperative analgesia after major thoracoabdominal surgery and may have a positive influence on postoperative morbidity. In patients with coronary artery disease, thoracic extradural analgesia with local anaesthetics may enable prevention and treatment of myocardial ischaemia by blocking the cardiac sympathetic nervous system.

This case report describes the influence of thoracic extradural analgesia on postoperative ECG ischaemic episodes after oesophageal resection in a patient with coronary artery disease and chronic obstructive respiratory failure.

Case report
A 55-year-old patient (1.70 m, 74 kg, ASA III), underwent total oesophagectomy without thacotomy, for adenocarcinoma of the lower one-third of the oesophagus.

The patient was a smoker (50 pack-years) and suffered from chronic obstructive pulmonary disease with hypoxia ($P_{aO_2}$ 8.5 kPa on room air) and normocapnia. FEV$_1$ and vital capacity were 46% and 78% of theoretical values respectively. Ultrasonic cardiology showed global hypertrophy without dysfunction of the left ventricle. Coronary arteriography revealed diffuse atheroma with stenosis (40%) of the left anterior descending and circumflex coronary arteries and right major coronary arteries. A Methylene test (0.4%) was positive with spasm of the second segment of the right coronary artery, reversible with intracoronary nitroglycerine. Systolic ejection fraction was 70%, preoperative ECG was normal and the patient had no angina. The patient was taking diltiazem and nicorandil, and inhaled $\beta_2$ sympathomimetics and corticosteroids.

After oral premedication, a thoracic extradural catheter was inserted after puncture at T7–T8. After a test dose of 3 ml and then 7 ml of lidocaine 2% with 1/200 000 adrenaline, analgesia was maintained by continuous extradural infusion (6 ml h$^{-1}$ of a solution of 0.25% bupivacaine and 1 $\mu$g ml$^{-1}$ sufentanil). Haemodynamic monitoring consisted of a pulmonary artery catheter with continuous measurement of cardiac output (Intellicath 8F-Catheter and Vigilance Monitor, Baxter, USA) and a radial artery catheter. The perioperative ST segment was continuously monitored in leads II and V5 (Monitor M066A, Hewlett-Packard, Andover, MA, USA). Thirty min after administration of the extradural local anaesthetic, sensory block tested by the pinprick method was at T2–T12. General anaesthesia was then induced by midazolam 3 mg, propofol 150 mg, sufentanil 25 $\mu$g, and atracurium 45 mg, permitting orotracheal intubation and controlled ventilation with 60% nitrous oxide in oxygen and 0.6–1% expired isoflurane. Ventilatory parameters were stable throughout the procedure. Blood transfusion was not required. Hematocrit was 29% and body temperature was 36.5°C at the end of surgery. In addition, peri- and postoperative diltiazem i.v. 5 mg h$^{-1}$ and nitrates 10 mg day$^{-1}$ transdermally were administered.

Extrusion was possible 1 h after operation in the post-anaesthesia care unit. Analgesia was continued by patient-controlled extradural 0.125% bupivacaine and sufentanil 0.5 $\mu$g ml$^{-1}$ (3 ml h$^{-1}$ background infusion, 2.5 ml bolus, lock-out 12 min). Visual analogue scores (VAS) at rest (0 = no pain, 100 mm = maximum pain) were between 10 and 25.

At 36 h, there was ST segment depression in lead 2 and elevation in V5 (ST segment shift $\geq$ 1 mm, at J point + 60 ms, $\geq$ 1 min duration), with hypertension (200/110 mmHg) and a heart rate of 85 beats min$^{-1}$. There was no angina or acute respiratory failure. The ECG revealed ST segment depression in the apicolateral region. Plasma potassium concentration was normal (4.1 mmol l$^{-1}$). The sensory block was adequate (T6–L2) with a low VAS score at rest without motor block. No analgesic bolus had been requested in the previous hour. In view of the ECG signs of myocardial ischaemia in spite of perioperative diltiazem and nitrate we gave, without effect, diltiazem bolus (20 mg) and sublingual nitrates.
Also, a 6 ml bolus of 0.125% bupivacaine–0.5 μg ml⁻¹ sufentanil solution was administered extradurally. The new sensory block extended to T1–L4 with simultaneous disappearance of repolarization problems on the ST analyser and normalization of blood pressure (130/75 mmHg) and heart rate (60 beats min⁻¹). Specific myocardial enzymes were normal (5% serum creatinine kinase MB isoenzyme and concomitant myoglobin < 80 μg l⁻¹, troponin Ic < 0.5 μg l⁻¹ at 24 h).

Background infusion was increased (6 ml h⁻¹) to maintain sensory block above T4. There were no further complications.

Discussion

We describe an episode of electrocardiographic myocardial ischaemia that appeared 36 h after major thoracoabdominal surgery in a patient with coronary artery disease. This responded to blockade of the cardiac sympathetic nervous system.

Mangano and colleagues showed that episodes of myocardial ischaemia are more frequent and more severe postoperatively compared with preoperatively or perioperatively in patients with high cardiac risk. In addition, only 10–30% of postoperative episodes of myocardial ischaemia are linked to perioperative haemodynamic instability. These authors emphasize the protective effect of postoperative analgesia against episodes of myocardial ischaemia. However, in the present case, effective analgesia did not prevent the episode. The quality of postoperative analgesia was demonstrated by low pain scores (VAS < 30 mm) and presence of a sufficient sensory block (T6–L1).

This episode developed in the face of significant haemodynamic disturbances (hypertension) and resolved probably because blood pressure and heart rate were corrected. We can speculate that sympathetic nervous system block by extradural local anaesthetics induced beneficial peripheral effects (vasodilatation). On the other hand, cardiac sympathectomy by local anaesthetics could influence development of ischaemia if it was a vasospastic phenomenon.

In a patient with coronary artery disease, cardiac sympathetic block (T1–T5) with local anaesthetics leads to a moderate reduction in cardiac output (11%), heart rate (8%) and mean arterial pressure (19%) that is easily compensated by a reduction in myocardial oxygen demand. If the mean arterial pressure remains stable (dMAP < 20%), there is no significant modification of coronary blood flow.

On the other hand, there is an increase in the perfusion of myocardial ischaemic territories, with favourable redistribution of coronary perfusion toward the endocardium to the detriment of the epicardium, and finally, inhibition of poststenotic coronary vasoconstriction.

De Leon-Casasola and colleagues showed that, for a quality of analgesia identical to that achieved with i.v. morphine patient-controlled analgesia after upper abdominal surgery, 5 days of patient-controlled thoracic extradural analgesia with local anaesthetics reduced the incidence of postoperative myocardial ischaemia. Among these episodes of ischaemia, 72% were silent and occurred within 36 h of surgery. In cardiology, cardiac sympathetic blockade of thoracic extradural analgesia with local anaesthetics is proposed for the treatment of coronary artery disease refractory to classical treatment.

References