FIGURE Summary of the anaesthetic course.

hyperthermia was made and the following measures were initiated.

Surgery was completed and the patient's lungs were hyperventilated with 100% oxygen using a vapour-free circuit and ventilator. Pancuronium, fentanyl and diazepam were also given. Dantrolene sodium was given and active cooling with ice packs, gastric lavage, and cold normal saline was given intravenously. Sodium bicarbonate, 300 mmol, was given and a diuresis was forced with frusemide and mannitol. Serial blood samples taken for urea and electrolytes revealed a worsening hyperkalaemia ($K^+ = 7.8 \text{ mmol} \cdot \text{L}^{-1}$) treated with an insulin dextrose infusion.

Within an hour the patient's temperature, metabolic status and serum potassium concentration had returned to normal and he was transferred to the Intensive Care Unit. The remainder of the postoperative course was unremarkable. The highest recorded CPK concentration was $15147 \text{ iU} \cdot \text{L}^{-1}$, and the urine was negative for myoglobin. Muscle biopsy and contracture testing at The Ma-

lignant Hyperthermia Unit, Cork Regional Hospital, Ireland, subsequently confirmed the diagnosis.

Isoflurane has been associated with only one confirmed report of MH¹ where isoflurane was the only trigger agent used. The increase in PETCO₂ preceded a change in body temperature, again demonstrating that capnography is ideal for early detection of MH.^{1,2} Despite the metabolic derangements cardiovascular stability was maintained throughout. Labetalol may have had an effect in blunting the response.

In conclusion isoflurane would appear to be the most likely triggering agent in this patient and PETCO₂ monitoring facilitated early diagnosis and treatment.

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Aortocaval compression

To the Editor:

I am writing to comment on an article entitled "Aortocaval compression in the sitting and lateral decubitus positions during extradural catheter placement in the parturient." The author described aortocaval compression as being more common in the lateral decubitus than in the sitting position during placement of epidural catheters.¹ I think the title of the article is misleading as are the conclusions. The authors detected a decreased cardiac output in patients in the lateral decubitus position. However, they did not measure aortocaval compression (difficult to do) nor did they use any indirect measure to suggest aortocaval compression (increase in venous pressure in the legs or a decrease in systemic blood pressure in the legs). The clinical importance of aortocaval compression relates to the effect on the fetus. No mention is made in the article of the fetal heart rate tracing during insertion of the epidural catheters.

My clinical practice is to insert epidural catheters in the position preferred by the patient unless there is a

particular reason to adopt a different position. I think that insertion of the catheter is then expedited and the effects of position are minimized. I am not convinced that clinically important aortocaval compression is more common in the lateral decubitus position than in the sitting position.

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- 1 Andrews PJD, Ackerman WE, Juneja MM. Aortocaval compression in the sitting and lateral decubitus positions during extradural catheter placement in the parturient. *Can J Anaesth* 1993; 40: 320-4.

Epidural catheter entrapment

To the Editor:

We recently encountered a case of difficulty in removal of an epidural catheter that gave us some insight into a possible mechanism of entrapment.

The patient was a 150 cm tall 26-yr-old para 0 gravida 1 patient weighing about 69 kg, who requested epidural anaesthesia for relief of pain of labour. The initial advancement of a #16 Tuohy needle at interspace L₃₋₄ struck bone. The needle was partially withdrawn and again advanced several times with the tip pointing progressively cephalad until the epidural space was identified by loss of resistance to air. Drugs infused through the catheter provided adequate pain relief for labour and delivery. In the post-partum period, we were unable to remove the catheter despite various manoeuvres such as flexing and twisting the patient's back and injecting refrigerated sterile saline through the catheter. Repeat attempts were made over two days before the catheter was able to be removed on the third day with vigorous traction.

Reviewing the manoeuvres of identifying the epidural space with the Tuohy needle suggests a mechanism of entrapment of the epidural catheter. The proposed mechanism is illustrated in Figures 1 to 3. In Figure 1, initial advancement of the epidural needle has traversed the interspinous ligaments and struck bone at location "X." In Figure 2 the needle is partially withdrawn, angled progressively more cephalad and advanced forming two new tracks. After the epidural needle is advanced through the ligament flavum and a catheter is passed, it is withdrawn. This allows the tissues that had been compressed to recoil

FIGURE 1 A coronal view through the midline at L₃₋₄. The epidural needle is advanced and strikes the base of the spinous process at "X."

FIGURE 2 Left panel: partial retraction of the needle. Middle panel: needle is angulated and displaced cephalad but it still strikes bone. Right panel: needle is again partially retracted, further angulated and displaced cephalad.