# Clinical Reports

# Haemodynamic decompensation during Caesarean section

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Lumbar epidural anaesthesia has a well-established safety record for Caesarean section[1]. In the parturient, however, cardiovascular compensation for vasodilatation, hypovolaemia, positional changes, surgical manoeuvres, and other complications of anaesthesia and surgery may be less adequate than in the nonpregnant patient.[1,2]

The present report discusses a sudden cardiovascular collapse in a healthy parturient undergoing an apparently routine Caesarean section under an apparently routine epidural anaesthetic. The need for constant vigilance, and being prepared for any complication, in any parturient, is re-emphasized.

## Case report

A 27-year-old primigravida with an uncomplicated pregnancy presented at term, in active labour, with a breech presentation. A decision was made to perform a Caesarean section, and lumbar epidural anaesthesia was chosen. The patient was prehydrated with 1500 ml of lactated Ringer's solution, and a Racz epidural catheter was inserted through a #17 Tuohy needle in the $L_{3-4}$ interspace, using a loss-of-resistance technique. A test dose of 3 ml 1.5 per cent lidocaine was given, with no apparent complications. Then, over the next 20 minutes, a total of 26 ml of 0.5 per cent bupivicaine was given in 3 ml aliquots. Oxygen was delivered by a face mask at 6 $L \cdot min^{-1}$. The patient was maintained in a 15 degree left lateral decubitus tilt. Blood pressure, measured with a Dinamap automatic cuff, was stable at 120/70 mmHg, and heart rate was stable at 75 $b \cdot min^{-1}$, up to the time of delivery.

Surgery was begun approximately 40 minutes after insertion of the epidural catheter, and 20 minutes after the last dose of bupivicaine. At this time, a level of T6 to pinprick was measured, and surgical anaesthesia was apparently adequate. Due to difficulty in extracting the baby, prolonged epigastric pressure was applied by the surgical team. Approximately 12 minutes after skin incision, and after approximately five minutes of almost continuous fundal pressure, a male infant was delivered, with Apgar scores of 8/9. Immediately after delivery, Oxytocin, 10 IU, was placed in the Ringer's lactate infusion (approximately 1000 ml).

Immediately after delivery, the patient complained of some dyspnoea. Vital signs were stable, and the anaesthetic level to pinprick was rechecked and found to be to T-7. Approximately one minute later, the patient again complained of dyspnoea, demonstrated some purposeless movements of the arms, then suffered an apparent respiratory arrest as well as loss of consciousness. At this time, BP was 30/25 mmHg and HR was 35 $b \cdot min^{-1}$ as measured by the Dinamap, which had been maintained on a one-minute cycle. The carotid pulse, though palpable, was thready, and the radial pulse was unobtainable. Cardiopulmonary rescuscitation was then initiated. The lungs were ventilated with 100 per cent oxygen while cricoid pressure was applied. Succinylcholine, 100 mg was administered, and the trachea was intubated.

Initially, two doses of atropine, 0.4 mg each, and a single dose of ephedrine, 50 mg, were given, with no response of BP and an increase of HR to 45 $b \cdot min^{-1}$ over several minutes. Therefore, epinephrine, 1 mg, was given, with an increase of BP to 40/20 mmHg and an increase of HR to 55 $b \cdot min^{-1}$ over the next few minutes. Thereafter, 0.5 mg more epinephrine was given, producing a BP of 218/185 mmHg and a HR of 185 $b \cdot min^{-1}$. After five minutes, BP was 185/110 mmHg and HR was 150 $b \cdot min^{-1}$. Vecuronium, 10 mg, was then given for

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relaxation, and 100 per cent oxygen maintained. Scopolamine, 0.4 mg, was given for amnesia. Ten minutes later, approximately 20 minutes after the cardiovascular collapse, the vital signs stabilized, with BP of 110/60 mmHg and a HR of 150 $b \cdot min^{-1}$; ECG showed a sinus rhythm throughout. At this time, a right internal jugular venous catheter was inserted. No air was aspirated, and a sample of blood was immediately sent to the pathology laboratory for examination for fetal elements. None was detected, either immediately or after permanent staining.

Surgery was completed approximately 90 minutes after the skin incision. The estimated blood loss was 1000 ml. Fluids administered in the operating room totaled 2000 ml Ringer's lactate, in addition to the 1500 ml preload. The patient was taken to the surgical intensive care unit where mechanical ventilation was maintained. Arterial blood gas analysis showed no evidence of respiratory abnormality, and the patient was awake and apparently neurologically intact. Therefore, she was extubated 12 hours after admission to the unit, and transferred to a regular post partum bed the following day. Follow-up examination showed no sequelae.

## Discussion

In the absence of further laboratory and physiological information, the mechanism of the cardiovascular collapse in our patient must remain speculative. However, there are several possible causes.

Hypovolaemia is unlikely, because of the relatively stable course up to the time of the collapse, the amount of intravenous fluid infused, and the absence of unusually bloody or prolonged surgery. Local anaesthetic toxicity can cause a similar clinical picture.[3] However, the time-course of events in our patient is inconsistent with intravascular injection of bupivicaine. Although the time-course of the non-purposeful movements of the arms which can be interpreted as myoclonic seizures is consistent with systemic absorption of large amounts of local anaesthetic, the severity of the cardiovascular collapse might better account for these movements. Drugs, such as oxytocin, can cause hypotension in the parturient.[4] However, oxytocin was not infused in concentrated amounts and no other drugs were administered. High regional blockade can precipitate both repiratory and cardiovascular complications. However, our observation of a stable analgesic level makes this possibility unlikely.

Air embolism might also occur during difficult extraction of the fetus, due to exposure of traumatized vessels to air. Lack of aspiration of air into the central venous catheter does not rule out this diagnosis, especially because the catheter was not inserted until 20 minutes after the collapse. However, the patient's rapid, complete recovery makes the diagnosis of massive air embolism less likely.

Likewise, the traumatic manipulation of the uterus might expose the vessels to amniotic fluid, leading to amniotic fluid embolism. The unknown location of the central venous catheter, as well as its placement 20 minutes after the collapse, might have decreased the likelihood of detecting fetal elements. Clear amniotic fluid has been shown to have vasoactive properties in the absence of fetal elements.[5,6] The lack of detectable fetal elements in the blood sample, as well as the patient's rapid, complete recovery, make the diagnosis of massive amniotic fluid embolism less likely.

The profound bradycardia which was resistant to moderate doses of atropine raises the possibility of "vagal reflexes." Central hypovolaemia may be caused by surgical manipulations,[7,8] and could well have occurred when profound epigastric pressure was applied continuously. The cardiovascular changes may result, in part, from opposing effects on the sympathetic and parasympathetic nervous systems. The observed effect would depend upon which predominates. At the level of epidural blockade, T6-7, blockade of the cardiac sympathetic nerves would be unlikely. However, blockade of peripheral vasoconstriction might have severely limited the compensation for hypotension due to various causes. Baroreceptor stimulation would be expected to activate the cardiac sympathetic outflow, producing tachycardia. In contrast, left ventricular receptors that respond to hypovolaemia by producing vagal activation have been identified in animals.[9,10] It is believed that the resulting bradycardia might serve as a protective mechanism during profound hypovolaemia by improving diastolic filling. The commonly observed vasovagal syncope, "fainting," in awake young subjects presents with hypotension and bradycardia, with a decrease of cardiac filling pressure before vascular resistance decreases. This bears certain similarity to our case.[11] In a controlled study of volunteers subjected to preanaesthetic hypovolaemia, epidural anaesthesia to T5 produced profound bradycardia and hypotension.[12] In our patient, an additional source of afferent vagal stimulation could have been from the vigorous surgical manipulations, to produce the so-called vagovagal reflex.[13] The Figure illustrates the possible effects of a combination of vagal afferent stimuli resulting from vigorous surgical manipulation: central hypovolaemia due to caval compression, and direct visceral stimulation. These combined afferent stimuli might produce an exaggerated vagal efferent response. This mechanism seems to be one of the most likely causes of our patient's profound bradycardia.

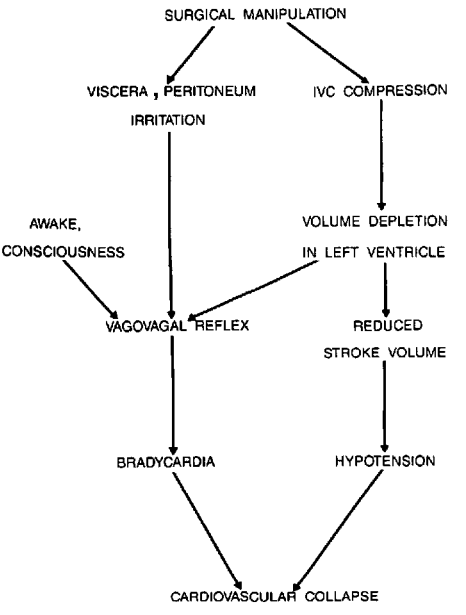


FIGURE Summary of vagal mechanisms that might have contributed to cardiovascular collapse.

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