

## CASE REPORTS

# Cardiac arrest after Caesarean section under subarachnoid block

T. J. SCULL AND F. CARLI

### Summary

Cardiac arrest occurred on arrival in the recovery room after emergency Caesarean section under subarachnoid block. The patient was resuscitated successfully and recovered with no adverse effects. The current literature is reviewed and the pathophysiological mechanisms involved in the aetiology of cardiac arrest under subarachnoid block are discussed. Early use of adrenaline to treat severe bradycardia or hypotension is recommended. (*Br. J. Anaesth.* 1996; **77**: 274–276)

### Key words

Anaesthesia, obstetric. Complications, cardiac arrest. Anaesthetic techniques, subarachnoid.

Subarachnoid anaesthesia is a widely practised technique for both elective and emergency procedures. It is recommended for both its efficacy and safety [1, 2]. Haemodynamic instability and cardiac arrest have been reported in healthy patients [3]. Occasional case reports [3–5] and a series of patients experiencing cardiac arrest [6] have been reported in the American literature, but there is little in the European journals. We report a case of cardiac arrest, occurring in a parturient after a Caesarean section under subarachnoid block.

### Case report

A 31-yr-old Afro-Caribbean woman (weight 76 kg, height 1.68 m), ASA I, presented at term in spontaneous labour. This was her second pregnancy; her first had resulted in a vaginal delivery and the course of the second had been uneventful.

On arrival in the delivery suite she was in active labour, her cervix was dilated 6 cm and her membranes were bulging. These were ruptured and fetal monitoring, via a scalp electrode, commenced. Over the next 8 h, progress was slow, dilatation increasing to 8 cm. The baby showed signs of distress which were manifested as type 2 decelerations, and it was decided to deliver the baby by Caesarean section.

Subarachnoid anaesthesia was commenced using a 27-gauge Quincke needle with 0.75% hyperbaric bupivacaine 2 ml and preservative-free morphine 0.25 mg. Bilateral sensory block to temperature from T2 to S5 was obtained. After positioning the patient in the supine position, with left lateral wedging, Caesarean section was carried out, and a boy (3585 g)

with Apgar scores of 9 at both 1 and 5 min was delivered. Arterial pressure, ECG and oxygen saturation were monitored throughout the procedure. Systolic arterial pressure was 110–120 mm Hg and heart rate 70–110 beat  $\text{min}^{-1}$ . Saline 0.9% (2 litre) and three boluses of ephedrine 5 mg were required to maintain stability. After delivery, an infusion of Syntocinon 20 u. in normal saline 1 litre was started. The duration of Caesarean section was 1 h 35 min and estimated blood loss was 700 ml. The patient's vital signs before leaving the operating theatre were: arterial pressure 110/60 mm Hg, heart rate 105 beat  $\text{min}^{-1}$  and  $\text{SpO}_2$  96%. The patient was then transferred to the adjacent recovery room in a semi-reclined position, fully conscious, conversing and holding her baby. As monitoring was being re-established, she suddenly became unresponsive, with no palpable pulse and no respiratory effort.

Cardiopulmonary resuscitation was initiated and her trachea was intubated within 2 min. ECG revealed the initial rhythm to be ventricular fibrillation and she received four dc shocks, adrenaline 2 mg i.v., naloxone 0.4 mg i.v. and lignocaine 100 mg i.v. At 9 min after cardiac arrest her rhythm had converted to sinus tachycardia, with a strong pulse. She was then transferred to the intensive care unit.

A series of investigations was carried out to identify the cause; ECG revealed a sinus tachycardia with no acute changes, echocardiogram showed good left ventricular function (ejection fraction 55–60%), with normal valvular morphology and function. Serum electrolyte concentrations were within normal limits, except for a low serum magnesium concentration of 0.25 mmol  $\text{litre}^{-1}$  (normal 0.75–1.25 mmol  $\text{litre}^{-1}$ ) obtained at the time of cardiac arrest. A CT scan revealed minimal cerebral oedema, and a ventilation–perfusion scan of the lungs was normal.

The patient's lungs were ventilated for 20 h and she remained haemodynamically stable without need of inotropic support. After tracheal extubation she was confused, with no memory of the preceding events. Her cognitive function returned to normal over the following week. Despite extensive investi-

T. J. SCULL\*, MB, CHB, FRCA, F. CARLI, MD, MPHIL, FRCA, Department of Anaesthesia, McGill University, Royal Victoria Hospital, 687 Pine Ave West, Montreal, Quebec, Canada H3A 1A1. Accepted for publication: February 28, 1996.

\*Present address: Nuffield Department of Anaesthesia, John Radcliffe Hospital, Oxford.

Correspondence to F.C.

gation, no obvious cause of the arrest was found, other than the low magnesium concentration, the aetiology of which was unclear. In the postoperative period she developed a wound infection that necessitated a prolonged hospital stay. She was discharged home 25 days after her section and remains well.

## Discussion

Cardiac arrest during subarachnoid anaesthesia is an uncommon but well reported phenomenon. Bradycardia with resultant hypotension is thought to occur by two mechanisms. The first involves block of the cardioacceleratory sympathetic fibres, which may occur with a sensory block as low as T10, as it has been shown that the sensory-sympathetic differential may be up to six segments [7]. This allows unopposed parasympathetic input with a negative chronotropic effect. The other mechanism is a manifestation of decreased venous return, which may trigger reflexes mediated by caval and atrial receptors [8], and the pacemaker stretch reflex [9]. Acute reductions in venous return have also been reported to activate the Bezold-Jarish reflex with resultant bradycardia [10].

Excessive bradycardia progressing to asystole has been well documented recently [3, 4]. In some reports [3–5], patients responded promptly to atropine, ephedrine and external cardiac massage, cardiac output and consciousness being rapidly restored with no sequelae after the event. The series of patients reported by Caplan and colleagues [6], as a result of closed claims analysis, exhibited a different outcome and possibly a different pathogenesis. These patients showed marked similarities; all were young (mean age 35 yr), healthy (ASA I or II), had high sensory block, and all experienced a poor outcome; six died and seven were severely neurologically damaged. Close scrutiny of these cases implicated the use of sedation with opioids or benzodiazepines, with a resulting respiratory insufficiency, to be possible causative factors in 50% of the cases [6, 12]. In addition, another 22 cases from closed claims analysis have recently been reported [11]. These patients had similar characteristics and outcomes to the initial series of 13. Pulse oximetry failed to demonstrate desaturation and it was concluded that “one or more circulatory mechanisms played an important role in these events.”

Our patient had some factors in common with the “Caplan groups”. The patient was young, haemodynamically stable, with a high sensory block, but outcome was good. Cases of cardiac arrest after subarachnoid block that required protracted resuscitation and had a good outcome are rare. Perhaps this represents a lack of reporting or indicates that a high sympathetic block in some way complicates successful resuscitation.

Detailed analysis of the closed claim cases reveals the importance of warning signs of the forthcoming arrest, usually bradycardia or hypotension in the preceding 1–2 min. Our case was in transfer to recovery during this phase, with no monitoring in place and it is therefore not possible to comment on the presence or absence of these signs.

We believe that the patient suffered a primary cardiac arrest. In the period immediately after the arrest she received treatment with i.v. naloxone, but we do not feel that respiratory depression secondary to intrathecal opioids played any aetiological role in the chain of events. It may be significant that our patient experienced the cardiac arrest shortly after repositioning had occurred. In this unit it is customary to roll the patient from side to side in order to clean the abdomen, and then to transfer the patient to the recovery area in a semi-reclined position, approximately 30° head-up. These positional changes may have resulted in alterations in venous return and triggering of the above-mentioned reflexes, causing bradycardia and hypotension. Blood loss in the presence of sympathetic block may lead to hypotension, however only 700 ml was lost over a 1–1.5-h period, and adequate replacement was achieved.

Hypomagnesaemia at the time of the arrest was the only electrolytic abnormality. This is associated with cardiac arrhythmias, and is known to prolong QT intervals [12]. In addition to cardiac signs, patients usually complain of weakness and tremors and exhibit muscle fasciculation. The patient had none of these signs or symptoms, which makes it difficult to conclude that this was the cause.

The series of patients reported by Caplan and colleagues [6] did not receive adrenaline until approximately 8 min had passed after recognition of the cardiac arrest; a perfusing rhythm was re-established within approximately 3 min of this treatment. We feel that the early use of adrenaline and the prompt resuscitative efforts were important factors in the positive outcome of our patient.

We would like to support the approach advocated by Caplan and colleagues that the potent  $\alpha$  and  $\beta$  agonist adrenaline be used early in the treatment of extreme bradycardia and hypotension in patients with high sympathetic block, especially if initial treatment with ephedrine or atropine has been ineffective.

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