Wenckebach type heart block following spinal anaesthesia for Caesarean section

B.F. Matta MB BA DA FRCA^{anaes}, P. Magee MB BA DA FRCA^{anaes}

A case is described of complete heart block during spinal anaesthesia for Caesarean section in a fit 23 yr-old woman. This developed shortly after the institution of the block, with the height of the block below T₅ and in the absence of hypotension. The patient was resuscitated successfully with vagolytic and alpha-agonist drugs. A Wenckebach block persisted for a short period postoperatively. The importance of instituting monitoring before the beginning of anaesthesia and the immediate availability of atropine and alpha-agonists before the initiation of spinal anaesthesia is stressed.

Cette observation décrit l'apparition d'un bloc auriculoventriculaire complet pendant la rachianesthésie d'une femme de 23 ans en bonne santé subissant une césarienne. Le bloc a fait son apparition tôt après la mise en marche d'une anesthésie sans réaction hypotensive dont le niveau est demeuré sous T₅. La patiente a été réanimée efficacement par un vagolytique et un agoniste alpha. Un bloc de Wenckebach a persisté pour une courte période en postopératoire. Les auteurs soulignent l'importance de la mise en place du monitoring et de la disponibilité immédiate de l'atropine et d'un agoniste alpha avant l'initiation d'une anesthésie rachidienne.

Key words

ANAESTHETIC TECHNIQUES: spinal;
COMPLICATIONS: heart block;
HEART: arrhythmia, heart block.

From the Sir Humphry Davy Department of Anaesthesia, Bristol Royal Infirmary, Bristol.

Address correspondence to: Dr. B.F. Matta, Department of Anesthesiology, RN-10, University of Washington, Seattle, Washington 98195, U.S.A.

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Case history

A 23-yr-old primigravida with transverse lie was booked for an elective Caesarean section at 39 wk of pregnancy. She had an otherwise normal pregnancy. The only past medical history of relevance was that at seven years of age she had a heart murmur, which required no treatment and had disappeared by 14 yr. Routine preoperative clinical assessment was uneventful, and haematological and biochemical investigations were normal. The patient requested a regional anaesthetic. Routine antacid prophylaxis (oral ranitidine 300 mg and sodium citrate 15 ml) was given at 2200 the night before and at 0800 on the morning of delivery.

Electrocardiography, non-invasive blood pressure monitoring and pulse oximetry were attached to the patient. A 14G intravenous cannula was inserted in the left forearm. Before the block the patient received Ringer's lactate 1000 ml, and an infusion of ephedrine 50 mg in normal saline 500 ml was started and titrated according to the blood pressure. The patient was turned on to her left side and a 25G Whitaker spinal needle was introduced at the L_{2/3} interspace. When free flow of CSF was confirmed, 2.5 ml heavy bupivacaine 0.5% were injected slowly. The blood pressure was 130/80 mmHg. Immediately after injection, the patient complained of pins and needles in her left leg. She was turned on to her back with 15° left lateral tilt and no head-down tilt. She was given oxygen via a mask at 4 L · min⁻¹. The BP remained stable at 130/90 mmHg and the ECG showed sinus rhythm (100 beats · min⁻¹) with a normal PR interval.

About two minutes after injection, when the level of block tested with cold spray was T₅, the patient felt faint and dizzy and became cyanosed. The oximeter was not giving a reading as the patient was agitated and was moving around. The BP at the time was 125/90 mmHg. The ECG showed a sinus bradycardia of 30 bpm that progressed to complete heart block. Atropine, 0.6 mg, was given iv and the ephedrine infusion was speeded up. There was an immediate increase in the heart rate up to 140 bpm

FIGURE Wenckebach block.

and the BP increased to 180/100 mmHg. The upper level of blocks remained at T₅.

The lady was reassured and delivery proceeded without further incident. A healthy infant was delivered with normal one and five minute Apgar scores. The patient's husband later said that the patient had a tendency to faint when she became excited. Approximately two hours after the initiation of the spinal anaesthetic, a 12 lead ECG showed progressive prolongation of the P-R interval in a Wenckebach type heart block (Figure). Cardiorespiratory examination was normal. A 12 lead ECG 24 hr after the incident was normal. An echocardiograph and a 24 hr Holter tape were also normal. A cardiologist did not recommend any further treatment.

Discussion

There have been several reports of severe bradycardia, sometimes with cardiac arrest after spinal anaesthesia.^{1,2,4} The bradycardia that occurs with spinal anaesthesia is slow in onset, and easily treated. Suggested causes are the loss of cardiac sympathetic stimulation and a decreased venous return.³ Caplan *et al.*⁴ reviewed 14 cases of cardiac arrest that occurred during spinal anaesthesia and concluded that there is a poorly understood potential for sudden cardiac arrest in healthy patients. In most of their cases, cardiac arrest had a better neurological outcome if there was prompt augmentation of central venous filling by positional change and potent alpha-agonist drugs. This may have improved organ perfusion and shortened the period of the cardiac arrest. In our case, the patient was resuscitated promptly with vagolytic and alpha-agonist drugs, presumably resulting in an increase in venous return and heart rate. The heart block that resulted was followed by a Wenckebach type block postoperatively, which persisted for several hours.

The bradycardia was not caused by hypotension, as the patient's BP remained stable, 125/90 mmHg immediately before the patient complained of dizziness. It is unlikely that it was caused by high blockade, as the sensory level

remained unchanged at T₅. It may have been a vasovagal attack, but these usually resolve quickly and do not persist for several hours.⁵ It is possible that the spinal anaesthetic may have brought to light an undiagnosed disorder of cardiac conduction⁶ that was transient and hence not picked up on later tests. The reason for the heart block and Wenckebach block that followed remains unclear.

We would like to stress the importance of instituting monitoring before the beginning of anaesthesia and the immediate availability of atropine and alpha-agonists before the initiation of spinal anaesthesia.

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