Asystole during spinal anaesthesia after change from Trendelenburg to horizontal position

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We present a case in which progressive bradycardia and sudden cardiac arrest developed in a 26-year-old healthy woman with an ovarian cyst during spinal anaesthesia, immediately after the patient was moved from the 15° Trendelenburg to the supine horizontal position. We postulate that a decrease in venous return was the main cause of the cardiac arrest. It should be stressed again that close monitoring during spinal anaesthesia is essential when the patient is subjected to postural changes.

Sinus bradycardia after spinal anaesthesia is frequently encountered in ordinary clinical settings.[1-3] However, the incidence of cardiac arrest during spinal anaesthesia is extremely low.[4] Bradycardia during spinal anaesthesia has been ascribed to either an interruption of the cardiac accelerator fibres or a decrease in venous return, or to both.[1-3] Recently, it has been suggested that cardiac vagal tone is enhanced mainly through a decrease in the venous return during lumbar epidural blockade.[5] Therefore, venous return is of importance in the regulation of heart rate, particularly during regional anaesthesia. In this report, we present a case in which progressive bradycardia and asystole developed during spinal anaesthesia, immediately after the patient was moved from the Trendelenburg to the supine horizontal position.

Case report

A 26-year-old, 46 kg, 156 cm woman was scheduled to undergo an oophorectomy for an ovarian cyst, under spinal anaesthesia. She had no specific findings in her medical history except that she had suffered from lightheadedness several times and syncope once in the past. However, the cause of the episodic faintness had not been defined. A preoperative electrocardiogram revealed a sinus arrhythmia with a rate of 53 beats · min^{-1}. Preoperative haemoglobin, haematocrit and blood sugar were 11.0 g · dl^{-1}, 34.4 per cent and 173 mg · dl^{-1}, respectively. Positive ketone bodies were found on urinalysis, but other preoperative laboratory data were within normal limits.

The patient had fasted preoperatively for six hours. Premedication consisted of hydroxyzine 100 mg IM one hour before the start of anaesthesia. On arrival in the Operating Room, her blood pressure was 116/67 mmHg, heart rate 63 beats · min^{-1}, and respiratory rate 14 breaths · min^{-1}. She was well sedated, but lucid. A 16-gauge venous cannula was placed for administration of lactated Ringer's solution. Following sterile preparation of the skin, a 25-gauge spinal needle was inserted in the L_{3-4} intervertebral space while the patient was lying in the right lateral position. After a free flow of clear cerebrospinal fluid was confirmed, 2.4 ml of solution of 0.5 per cent tetracaine in ten per cent dextrose to which 3.7 mg of phenylephrine had been added, was injected over 5 sec. No adverse reaction was observed during the subarachnoid injection. The patient was immediately placed in the supine horizontal position. At that time, her blood pressure and heart rate were 90/34 mmHg and 53 beats · min^{-1}. Because sensory blockade was found only below T_{10}, approximately three minutes after the tetracaine injection, the head of the operating table was lowered by about 15 degrees in order to extend the analgesia. During this period in the Trendelenburg position, the blood pressure and heart rate showed little change. The patient did not complain of any nausea or discomfort. About 500 ml of lactated Ringer's solution had been infused up to this time. Because an upper level of analgesia to T_5 was obtained five minutes after the induction of anaesthesia, the table was returned to the previous horizontal position. Shortly thereafter, the heart rate progressively decreased from 54 to 30 beats · min^{-1}, then standstill of the heart occurred. The patient's

Key words

ANAESTHESIA, REGIONAL: spinal; COMPLICATIONS: cardiac arrest; POSITION: Trendelenburg, supine horizontal.

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eyeballs rolled upward and consciousness was lost. Closed cardiac massage was performed while the lungs were ventilated with pure oxygen by mask. Atropine 0.5 mg and ephedrine 10 mg were given IV immediately. After about 30 seconds the heart regained a normal sinus rhythm and spontaneous respiration also appeared. The blood pressure increased to 144/76 mmHg, and the heart rate increased to 93 beats·min^{-1}, one minute after the initiation of resuscitation. The patient regained consciousness, became well oriented, and responded to verbal commands. The upper level of analgesia to pinprick was at T_5. The remaining one-hour anaesthetic course was uneventful.

The removed ovarian cyst weighed about 220 g. The patient was discharged two weeks after the operation without any sequelae. Detailed cardiovascular tests were carried out one month after the operation. Postural changes in blood pressure were not observed. Additionally, no abnormality was proved by means of a Holter electrocardiogram, an echocardiogram, and a Master's exercise test. There was no evidence of autonomic neuropathy in the analysis of beat-to-beat variations in the heart rate when the patient breathed deeply or was tilted head-up. A mild glucose intolerance was ascertained by a glucose tolerance test, and an increased free fatty acid was noted (1260 μEq·L^{-1}; normal range 170–590). The patient was diagnosed as having borderline diabetes mellitus, requiring no special treatment.

Discussion

Although the cause of cardiac arrest in this patient can not be defined conclusively, it is reasonable to assume that moving the patient from the Trendelenburg to the supine horizontal position contributed mainly to this circulatory catastrophe.

Bradycardia as one of the characteristic features during spinal anaesthesia has been attributed to either preganglionic block of the cardiac accelerator fibres or a decrease in the venous return to the heart, or to both.[1–3] It is presumed that the upper level of sensory blockade at T_5 in this case may indicate an almost complete interruption of the cardiac efferent sympathetic nerves, according to Chamberlain and Chamberlain's recent demonstration that the mean sympathetic-sensory differential with tetracaine anaesthesia is more than six segments.[6] In addition, Baron et al.[5] observed a resetting of the baroreflex due to decreased venous return after lumbar epidural anaesthesia in humans. It has also been shown in elderly men that the heart rate and catecholamine responses to the hypotension induced by a 30-degree head-up tilt are impaired.[7] These findings may indicate an inadequate compensatory mechanism of circulation after regional anaesthesia. The rhythm of the heart can be thus affected by venous return[1,2] which is in turn influenced by postural changes. Accordingly, it seems likely that this progressive bradycardia and asystole could be induced chiefly by an abrupt decrease in venous return, secondary to the postural change from the Trendelenburg to the horizontal position.

Although the size of the removed ovarian cyst in our case appeared too small to obstruct the inferior vena cava, large intraabdominal masses such as a gravid uterus and pelvic tumours can induce the "supine hypotensive syndrome," secondary to a decrease in venous return, particularly after regional blockade.[8,9] Likewise, an inadvertent upward extension of the motor blockade leading to respiratory insufficiency can be excluded as the cause of the cardiac arrest, since adequate spontaneous respiration and the T_5 level of analgesia were confirmed both before and after the resuscitation. It is clear that the patient should have been immediately placed in the head-down position, because venous return seems to play a critical role in the regulation of heart rate, especially in the presence of sympathetic denervation.

It has been shown that respiratory sinus arrhythmia reflects an adaptation of the cardiac frequency to the respiratory fluctuations of the venous return,[10] and that parasympathetic influences predominate in the regulation of resting heart rate.[11] Therefore, the patient's preoperative electrocardiogram showing sinus arrhythmia and bradycardia likely indicates that our patient might already have been in a vagal dominant state and her heart rate might be extremely vulnerable to changes in venous return. The history of episodic faintness may suggest a vagus-mediated syncope, through either pronounced bradycardia or transient asystole.

There are many reports that diabetic neuropathy affects the heart, resulting in a predominantly vagal denervation.[12,13] As a result, tachycardia and little beat-to-beat variation (RR interval) in the heart rate become manifest in the symptomatic or even the asymptomatic diabetic. However, our patient exhibited no abnormal finding at the time of monitoring of the RR interval, when she breathed deeply or was tilted head-up. The neuropathy due to diabetes mellitus should preferentially counteract the vagal-mediated circulatory responses, thereby making this unlikely as the cause of the cardiac arrest. Finally, the history of episodic light-headedness could be related to idiopathic hypertrophic subaortic stenosis,[14] which, however, was not evident on a postoperative echocardiogram.

In summary, we described a case in which a sudden cardiac arrest occurred immediately after the patient was moved from the 15-degree head-down to the horizontal position during spinal anaesthesia. A decrease in venous return was presumably the main causative factor for this event. It must be stressed again that close monitoring during spinal anaesthesia is essential, particularly when

patients with evidence of enhanced vagal tone are subjected to postural changes.

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Résumé

On présente le cas d'une bradycardie progressive et d'un arrêt cardiaque lors d'une anesthésie rachidienne chez une patiente âgée de 26 ans présentant un kyste ovarien survenant immédiatement après changement de position d'un Trendelenbourg de 15° à une position horizontale. On pense que la diminution du retour veineux est la cause principale de l'arrêt cardiaque. On doit insister sur le fait qu'une surveillance étroite est nécessaire lors d'une rachi anesthésie quand le patient est asujetti à des changements de position.