Sudden cardiorespiratory arrest following spinal anesthesia

INTRODUCTION

Sudden cardiac arrest after spinal anesthesia is a relatively rare but potentially catastrophic event. The true incidence of cardiac arrest during neuraxial anesthesia is not as low as it was traditionally believed to be; it varies between 1.3 and 18 in 10,000 (1"12). A review by Sprung et al. (1) reported the frequency of arrest for patients during regional anesthesia to be 1.5 per 10,000, which was less than the reported frequency of arrest for patients receiving general anesthesia (5.5 per 10,000). These facts do not necessarily imply greater safety of regional anesthesia. Rather, this may reflect the fact that a wider variety of more complex surgical cases are performed under general anesthesia, and also that there may be a bias toward general anesthesia in the emergency setting. Two large prospective studies designed to evaluate the incidence of complications during spinal anesthesia reported 2 cardiac arrests in 1881 patients (9) and 26 arrests in 40,640 patients (2) for an overall incidence of 7 in 10,000 (0.07%). This incidence is high compared with the rate of one cardiac arrest for every 10,000 cases (0.01%) reported for epidural anesthesia (2). Equally important are the long-term survival and outcome after such events. Auroy et al. (2, 3) reported 29 cardiac arrests among 40,640 spinal and 30,413 epidural anesthetics, only 6 of which were fatal. Importantly, no neurologic deficits were observed in the 23 (79%) patients who survived cardiac arrest. In another large 20-yr prospective study, Kopp et al. (13) showed that median time to arrest was 50 min (range 0-210 min) after last local anesthetic administration (intrathecal/epidural/caudal). Because the time of onset of cardiac arrest cannot be predicted, it is of utmost importance that all the patients who receive neuraxial anesthesia get constant monitoring and vigilance. Also, in order to achieve satisfactory neurologic outcome, resuscitative measures of an advanced cardiac life support protocol have to be promptly initiated.

CASE REPORT

A 64 years old woman (weight 69 kg, height 160 cm, BMI 26.95 kg/m²) was scheduled for elective total hip replacement surgery. Preoperative assessment was unremarkable except for a history of arterial hypertension and hyperlipidemia. Medications consisted of lisinopril, hydrochlorothiazide and atorvastatin. Physical examination, laboratory evaluation and ECG were unremarkable. She was classified as ASA II patient according to the ASA classification. For premedication patient received oral midazolam 7.5 mg. Upon arrival in the operating room, ECG, automated blood pressure cuff and pulse oximeter monitors were applied...
and routine monitoring (with ECG, non-invasive arterial pressure and oxygen saturation) was started. One wide-bore 14-gauge intravenous access was secured and 20-gauge arterial catheter was inserted in left radial artery, according to our local protocol. Arterial blood was drawn for preoperative analysis (blood gases, electrolytes, glucose, lactates). Lab results: pH 7.43, pO2 10.1 kPa, pCO2 5.1 kPa, sO2 95%, HCO3 25.2 mM, sodium 137 mM, potassium 3.5 mM, calcium 1.13 mM, glucose 5.0 mM, lactate 0.9 mM. The preoperative blood pressure was 150/85 mmHg, heart rate was 62 beats/min with sinus rhythm and peripheral oxygen saturation was 99%. Sufentanyl 5 μg was given, and nasal oxygen supplementation at 4 L/min was begun. Fluid preload consisted of 500 mL of Ringer lactate solution. After positioning the patient to the sitting position, 0.5% levobupivacaine 15 mg mixed with morphine hydrochloride 125 μg was injected with a Whitacre 27-gauge spinal needle at L2-L3 interspace. Spinal puncture was uneventful. She was returned to supine position and urinary bladder was catheterised. Ten minutes after the spinal puncture sensory level to pinprick was noted to be at Th8 bilaterally. The patient was then turned to left lateral decubital position and a preparation for surgery had started. For 25 minutes after the intrathecal administration of local anesthetic, patient was fully responsive, oriented, hemodynamically and respiratory stable, with only a slight decrease of arterial blood pressure and heart rate (135/75 mmHg and 55 beats/min, respectively), and oxygen saturation was consistently 100%. Then suddenly the patient becomes unresponsive, apneic, with unmeasurable traces of oxygen saturation and pulseless on carotid artery. P-wave asystole was noted on ECG monitor. She was immediately turned supine, sterile drapes that were already in place were removed, external cardiac massage was started by a surgeon, while airway was easily secured with endotracheal tube. No muscular relaxant was needed for intubation because the patient was flaccid and completely areflectic. After the confirmation of the tube position by auscultation, ventilation with 100% oxygen was asynchronously started. Atropine 0.5 mg and ephedrine sulphate 20 mg were administered, while epinephrine 1 mg, diluted to 1:10,000 solution, was prepared. After approximately one minute of CPR patient status was checked: blood pressure was 125/65 mmHg and stable, heart rate was 78 beats/min with synus rythm, oxygen saturation was 100% and end-tidal CO2 was 4.7 mmHg. Arterial blood was drawn for analysis: pH 7.36, pO2 52.4 kPa, pCO2 6.0 kPa, sO2 100%, HCO3 24.7 mM, sodium 137 mM, potassium 3.8 mM, calcium 1.21 mM, glucose 5.5 mM, lactate 1.0 mM. Inhalational anesthesia with sevoflurane 1.2 vol% was carefully started, and after ten minutes of observation during which patient remained fully hemodynamically and respiratory stable and without a need for inotropic support, a consensus was made to begin with surgery. Invasive arterial blood measurement was started. Surgery lasted for 92 minutes and went uneventful, intraoperative blood loss was 250 mL, diuresis was 500 mL. Patient received our standard intravenous fluid load consisting of Ringer lactate 1000 mL and 6% hydroxyethyl starch 130/0.42/6:1 balanced solution (Tetraspan® 6%, BBraun) 500 mL. General anesthesia was maintained only with sevoflurane 0.8–1.2 vol%. At the end of the surgery patient was awakened and extubated. Postoperative laboratory analysis was normal. She was fully oriented but completely amnestic and unaware of the event. No neurologic deficit or cognitive dysfunction was observed, except for mild confusion and retrograde amnesia. Early recovery and physical rehabilitation on the ward was normal, and the patient was on 4th postoperative day discharged to rehabilitation clinic according to preoperative plan.

**DISCUSSION**

Cardiac arrest fatalities during spinal anesthesia are becoming increasingly recognised by many representative case reports throughout medical science literature of 21st century. As it is described in introduction, although such fatalities were usually described as unexpected or extremely rare, they were actually surprisingly common (1, 2, 3, 13, 14, 46, 50). In a study including 20,000 spinal anesthetics, fatal cardiac arrest was reported in healthy young patients less than 30 years of age (15). A closed claims analysis by Caplan et al. (14) reported 14 arrests with six mortalities in young healthy patients undergoing minor surgical procedures. Of the eight survivors, seven had serious neurological damage. Such a situation should attract attention that many of these fatalities may be avoidable and that the working mechanism should be cleared.

A responsible mechanism for cardiac arrest after spinal anesthesia is of a possible respiratory, cerebral and circulatory origin.

**RESPIRATORY MECHANISM**

It may be considered as a cause for cardiac arrest after spinal anesthesia, because most of those patients also receive a sedative drug that may be responsible for inadequate ventilation. But the evidence for a respiratory etiology is sparse. Spinal anesthesia sensory levels up to Th4 do not lead to hypoventilation; they are actually associated with mild hyperventilation (16, 17). And after the widespread use of pulse oximetry in routine monitoring, it is now difficult to invoke hypoxemia as the primary cause, because cardiac arrests occur in the setting of oxygen saturation readings of 95–100% at the time (18, 19, 20). However, for safety, one should consider that oversedation may increase the risk of bradycardia (14) and the patient may not be able to communicate symptoms of early warning of a vasovagal attack (21). In our patient that wasn’t the case; she received only premedication with hypnotic dose of oral midazolam together with a low-dose sufentanil intravenously, and was fully responsive, with oxygen saturation reading of 100%, until the moment of sudden cardiac arrest.
CEREBRAL MECHANISM

There is some evidence in the older literature that cerebral hypoxia might occur during spinal anaesthesia in some patients (22, 23). More recently, Atallah et al. (26) proved that following spinal anaesthesia and during TURP, some patients developed impaired cerebral oxygenation. Although impaired cerebral oxygenation seems not to be a primary etiology for fatal cardiac arrest during spinal anaesthesia, it can be suggested that a disturbance in cerebral oxygen balance might be a precipitating factor for the occurrence of cardiac arrest in specific surgical situations during spinal anaesthesia. In our patient that wasn’t the case either.

CIRCULATORY MECHANISM

A circulatory etiology for cardiac arrest during spinal anaesthesia is directly or indirectly related to the blockade of sympathetic efferents (25, 26). Local anesthetics injected into subarachnoid space block not only motor and somatic sensory fibres; they also produce preganglionic sympathetic denervation, and the level of sympathetic block is always 2 to 6 segment higher than somatic levels, which are usually assessed by pinprick. Skin discrimination of temperature may be used as a clinical correlate of the level of sympathetic denervation during regional anaesthesia. The cardiac sympathetic outflow emerges from C5 to Th5 levels, with the main supply to the ventricles from Th1 to Th4. A significant part of the chronotropic and inotropic control of the heart and its oxygen demand is mediated through the upper four thoracic spinal segments. The major determinant of heart rate is the balance between sympathetic and parasympathetic systems with the latter predominating. A high thoracic sympathetic blockade covering the cardiac segments (Th1-Th4) produces significant reduction in heart rate. However, high sympathetic blockade may leave some cardiac sympathetic activity intact, since sympathetic responses to hypercarbia are not totally suppressed by high Th1 to Th5 blockade (27, 28).

A more important effect of sympathetic inhibition during spinal or epidural anaesthesia is a significant decrease in venous return due to dilatation of resistance and capacitance vessels (27). Reductions of the right atrial pressure of 36% and 53% have been reported after low and high spinal blocks respectively (17). It is clear that a circulatory etiology underlies the occurrence of severe bradycardia and asystole, given the blockade of sympathetic efferents and the profound decrease in venous return associated with higher levels of spinal sympathetic blockade (29). Those decreases in preload after spinal anaesthesia initiate reflexes that cause severe bradycardia.

Mackey et al. (19) have suggested three working reflexes:

1. The pacemaker stretch reflex: the rate of firing of the pacemaker cells within the myocardium is proportional to the degree of stretch. Decreased venous return results in decreased stretch and a slower heart rate.

2. Low pressure baroreceptors in the right atrium and vena cava when stimulated may cause bradycardia. With progressive hypovolemia, there may be a paradoxical recurrence of baroreceptor discharge, the so-called “collapse firing” (21).

3. The Bezold-Jarisch reflex: mechanoreceptors in the left ventricle when stimulated may cause bradycardia. The Bezold-Jarisch reflex includes reactions triggered by cardiac mechano-receptor activation and it has been used to describe perioperative bradycardia with hypotension (19, 30, 51). Some unmylenated afferent pathways from the inferoposterior wall of the left ventricle pass via the glossopharyngeal and vagus nerves to the brain stem and may respond to decreased venous return due to relative hypovolemia by bradycardia. This bradycardia is a protective reflex that prevents the heart from contracting when relatively empty (31, 37). The Bezold-Jarisch reflex overlaps with the vasovagal syncope (52) as it became a term which includes reactions triggered by cardiac mechanoreceptors and describes perioperative bradycardia with hypotension (19, 31, 32, 37, 46). The Bezold-Jarisch reflex was originally described in 1867 by von Bezold and Hirt (40). They observed that an intravenous injection of veratrum alkaloids cause a profound decrease in blood pressure and heart rate in conjunction with apnea. In the late 1930s Jarisch and Richter confirmed that the depressor effect initially observed by von Bezold was, in fact, a
reflex in origin. In 1947 Dawes et al. (41) finally showed that the reflex apnea was caused by a separate mechanism from that mediating the hemodynamic changes. Today, the term Bezold-Jarisch connotes the reflex as describes by Dawes: bradycardia, vasodilatation, and hypotension resulting from stimulation of cardiac receptors.

We have assumed that in our patient the Bezold-Jarisch reflex, possibly together with the other two cardiac reflexes proposed by Mackey, is responsible for sudden onset of cardiac arrest. Because we have managed to achieve hemodynamic stability in the patient in less than one minute, our presumption is that the circulatory reflex originated from the heart was the main cause of the event, rather than an unexpectedly high sympathetic blockade.

Risk factors for cardiac arrest following spinal anesthesia

John B. Pollard (5) has summarized six risk factors for bradycardia < 50 beats/min during spinal anaesthesia: baseline heart rate < 60 beats/min, ASA physical status I, age < 50 years, sensory level block above T6, prolonged P-R interval and the use of ß-blocking drugs. He holds the opinion that the presence of a single risk factor out of these six, does not make it certain that a patient will experience severe bradycardia or cardiac arrest. However, when two or more of these six factors are present, the patient may be considered at high-risk for bradycardia and cardiac arrest during spinal anaesthesia. Patient in our case report didn’t have any of these warning signs, but had still developed a sudden cardiac arrest. Considering this, a continuous vigilance and monitoring is mandatory, while providing anesthetic care for every patient after spinal anaesthetic.

Possible prevention of cardiac arrest

1. Proper selection of patient and operation

It is strongly advised that it should be appropriate to reconsider the use of spinal anaesthesia for a patient with vagotonia (5). Similarly, it may be prudent not to use spinal anaesthesia when significant blood loss or the use of vasodilators is anticipated (48, 49), because the vasodilatation caused by spinal anaesthesia can make resuscitation ineffective.

2. Fluid support

Although one might assume that maintaining preload during spinal or epidural anaesthesia is a uniform practice of anaesthetists, the literature demonstrates otherwise as many cases with cardiac arrest reported in literature occurred in settings without volume preloading (33, 46).

With decrease of venous return due to hypovolemia or a redistribution of blood volume, blood pressure is initially maintained by vasoconstriction. During progressive and continuing decrease in venous return, there may be a sudden fall in blood pressure, heart rate and peripheral resistance. Restoration of venous return is always urgent as spontaneous recovery from asystole may occur if this is achieved (34, 51). A simple manoeuvre as leg elevation if possible should not be ignored (14, 51). In an original study, Mirt and Vesna (38) compared the use of 12ml/kg Ringer Lactate solution within 20 min before spinal anaesthesia versus within 20 min starting immediately after spinal anaesthesia. In the first group of patients, the cardiac output (CO) increased by 20% after the infusion and returned to baseline value 30 min after spinal block. In the second group, CO increased after spinal block and was still 11.3% above the baseline 30 min after the spinal block. They (38) concluded that the decrease in CO after spinal anaesthesia can be prevented by the infusion of Ringer lactate solution, with CO reaching the highest value while the infusion is running.

3. Pharmacological support

Inspite of emphasizing the importance of volume loading and prompt replacement of fluid loss during spinal anaesthesia, decreases in preload can occur so quickly with altering the patient position, releasing a tourniquet or similar perioperative events that there may not be enough time to give sufficient fluid volumes over several minutes. Here, the administration of pharmacological drug support may be appropriate. In such situations, or when bradycardia is the presenting sign, atropine may decrease the incidence of cardiac arrest during spinal anaesthesia (7, 9). Brown et al. (39) advised vigilance of the anaesthetist and the willingness to utilize iv. atropine (0.4–0.6 mg), ephedrine (25–50 mg) and epinephrine (0.2–0.3 mg) in stepwise escalation of therapy, when bradycardia develops following spinal analgesia. Successful resuscitation was carried out in all settings where atropine was typically used as the first line of therapy (8, 42, 43, 46). Atropine may not be the best single agent if bradycardia is accompanied by vasodilatation as hypotension may persist after the relief of bradycardia by atropine (7).

It may be useful to note that epinephrine and external cardiac massage were also needed in some cases. None of these patients suffered morbidity after the event, reinforcing Caplan et al. (14) suggestion that immediate treatment of bradycardia or asystole under these circumstances is crucial. If initiating the measures of basic and advanced life support is delayed, the mortality becomes extremely high, approaching 90.9% among the patients that arrested in the Thailand prospective registry of 40,271 cases of spinal anaesthesia reported by Charuluxananan et al. (50). It should be clear that bradycardia with heart rate of 50 beats/min is consistent with cardiac sympathetic nerve inhibition associated with mid-thoracic block level (44). However, the development of sudden bradycardia over a few heart beats and the potential for the reversal of bradycardia by postural changes that increase venous return, can only be explained by a vasovagal reaction (21, 44, 52).

When bradycardia is profound or a full cardiac arrest occurs after spinal anaesthesia, the early administration of epinephrine is crucial. The vasodilatation caused by spinal anaesthesia can make cardiopulmonary resusci-
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CARDIOPULMONARY RESUSCITATION

Severe bradycardia (≤40 beats/min), if persistent despite pharmacological therapy, should be managed like asystole by cardiopulmonary resuscitation, comprising its usual principles of basic and advanced life support. Once persistent cardiac arrest occurs, external cardiac massage must be started to ensure circulation of drugs and perfusion of vital organs. Prompt treatment with epinephrine has been emphasized as crucial for successful recovery (5, 13, 33, 35, 46). In our case, we have managed to achieve sustained hemodynamic stability in our patient with external chest compressions, atropine and ephedrine. Epinephrine, although already prepared, was not needed.

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