

Unexpected cardiac arrest in spinal anaesthesia

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Spinal anaesthesia has been used for more than a century and is considered as a relatively simple and safe technique. Occasionally unexpected bradycardia and/or asystole may develop during the administration of spinal anaesthesia in apparently healthy and young patients. These situations are often attributed as the consequence of mismanagement of the spinal technique and not due to an intrinsic risk of the technique itself.

Although cardiac arrests were sporadically reported as early as the 40s and 50s of the last century (1-3), it was not until 1988 that CAPLAN *et al.* (4) described 14 unexpected cardiac arrests (UCA) during spinal anaesthesia, collected from the Closed Claims Analysis of the American Society of Anaesthesiologists (ASA). Surprisingly all patients were apparently healthy individuals undergoing minor surgery. Although in some cases previous bradycardia was noticed, in the majority of the cases there were no prodromes. In addition those with UCA showed to be very resistant to apparently well conducted cardio-pulmonary resuscitation with devastating outcomes. The cases extracted from legal files, showed that six patients died in the hospital and among the eight who survived, only one had no neurological sequelae. These cases could be easily defined as Sentinel Events (5) : "An unexpected negative outcome that should not occur under normal situations of medical care".

Since that historical review, more reports were published (6-8). KOPP *et al.* (9) reported 181 cases of UCA registered in the data bank of the Closed Claims Analysis of the American Society of Anaesthesiologists.

In this review, we are including an update of the current knowledge concerning unexpected cardiac arrests during spinal anaesthesia, with emphasis on pathophysiology, prevention and treatment. Extreme bradycardia and cardiac arrest will be considered as synonyms, due to their similarity in pathophysiology.

INCIDENCE OF CARDIAC ARRESTS DURING SPINAL ANAESTHESIA

The reviewed bibliography showed that in the last fifteen years, the incidence of cardiac arrest during spinal anaesthesia varied from 1.50 to 15/10000 (9-13). As these were only the reported cases, the true figure might be much higher. Consequently an anaesthesiologist, who administers spinal anaesthesia on a daily basis, is likely to face this complication at least once in his career.

CONSEQUENCES OF CARDIAC ARRESTS DURING SPINAL ANAESTHESIA

Of the 181 cases, reported in 2001 in the Closed Claim Analysis database, 161 (89%) resulted in neurological damage or death (9). In 91% of cases, neuraxial cardiac arrest resulted in death or brain damage, and the percentage was similar between decades (14). Despite well conducted CPR efforts in the majority of cases, the results of two French reviews by AUROY *et al.* resulted in surprisingly high mortality rate (26 and 30%) (11, 15).

PATHOPHYSIOLOGY

Caplan proposed two possible explanations (4). Sedation was used in several cases, suggesting

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that a misdiagnosed respiratory insufficiency could have been the basis of the problem. After the systematic use of a pulse oximeter it was definitively clear that a potential respiratory insufficiency did not play a determining role. POLLARD (16) concluded that in blocks up to T4 there is no hypoventilation and that in repeated cases of UCA the haemoglobin oxygen saturation were within values of 95-100%. Therefore, if there is any place for hypoxaemia in the onset of cardiac arrest during spinal anaesthesia, it doesn't seem to play a key role.

The second hypothesis raised by Caplan was that some kind of unclear interaction between the sympathetic block and the unrecognized hypovolaemia was present, explaining at least in part, the failure of a well conducted CPR. Therefore he proposed a "central filling" with volume replacement, in addition to early administration of alpha agonists and changes in patient's position, as a way to improve the results.

But how can haemodynamic aetiology be the basis of the problem? Cardiac output (CO) depends on systemic vascular resistance, venous return (preload) and myocardial contractility. Preload is determined by the volaemia status, venous tone, gravity and intrathoracic pressure.

Systemic vascular resistance depends on the vasomotor tone that has: a) myogenic and/or chemical autoregulation; b) hormonal regulation (angiotensine, aldosterone, antidiuretic hormone); and c) vasomotor centre activity regulated through its baroreceptors affected by pain, exercise and stress. Once spinal anaesthesia is administered, all of factors listed above can be disturbed, producing hemodynamic disturbances during spinal anaesthesia (17-20).

In 1993 McCRAE and WILDSMITH (21) described three main mechanisms of hypotension during spinal anaesthesia. Vasodilatation with a diminished venous return and a sudden reduction in CO are often seen. The upper sensory blockade level seems also to be a main cause with the higher the blockade level, the stronger the probability that problems may occur. Nevertheless it is not uncommon to see either high upper sensory blockade levels without haemodynamic problems or an adequate anaesthetic level accompanied by different degrees of hypotension and/or bradycardia. It is important to remember that the sympathetic blockade can be located two to six dermatomes above the upper sensory level (21, 22), a fact that cannot be anticipated due to the unpredictable inter-individual differences. Therefore a T6 dermatome blockade level can block the cardio-accelerator fibres emerging

from T1 to T4 producing brady-arrhythmias. The resulting high sympathetic blockade prevents vasoconstriction of the upper extremities, leaving the anaesthesiologist faced with a "generalized vasodilatation" (23). In addition, as the suprarenal glands receive their innervations from fibres emerging between T8 and L1, studies on animal models with induced cardiac arrest during spinal blockade, have suggested an inhibition of catecholamine release (24).

With a sharp preload reduction due to vasodilatation with relative hypovolemia, reflex bradycardia can occur. This bradycardia is mediated by different mechanisms: central, peripheral and mixed ones. Different authors have described vagal mediated bradycardia (25); firing (stimulation) of baroreceptors in the right auricle and the cava (8); stimulation, due to hypovolaemia, of left ventricle mechanical receptors (Bezold-Jarisch cardiac inhibiting reflex) (26) and the stretching pacemaker reflex: less myocardial distension would produce bradycardia (27). Among all the proposed mechanisms, the Bezold-Jarisch reflex may be the main cause. Is it possible that one or more of these feedback mechanisms lead to bradycardia and collapse and if untreated to cardiac arrest, in "healthy" patients during a spinal blockade? Currently, it is the most accepted etiology.

In fact, four years after Caplan's paper, JACOBSEN *et al.* (25) reproduced similar haemodynamic phenomena in healthy volunteers. The author demonstrated a decrease in the diameter of the left ventricle with bradycardia and severe hypotension in some patients with T 8-9 blockade levels.

RISK POPULATION

If, when a spinal blockade is administered, these patho-physiological phenomena are almost always present, why then, serious haemodynamic complications are so rare? Why do some individuals have these severe complications while the majority of others don't? Preoperative identification of the risk population can represent the first step to prevent and/or treat this entity.

If the initial event is a reduced pre-hydration due to hypovolaemia, unmasked by a sudden vasodilatation, a good approach can be to try to maintain hydration in order to obtain normovolaemia. This is a very common belief. In fact, several authors have stressed on how an adequate preoperative fluid replacement is not as common as believed in the majority of surgical patients (12).

The first point to consider is that hypovolaemia is hard to measure and diagnose and therefore to treat. The second point is that preoperative treatment of hypovolaemia, although important, might not be the key factor in preventing haemodynamic instability during spinal anaesthesia. It seems that a thorough knowledge of reduced venous return, vasodilatation and several reflexes mediated by intrinsic and/or neural mechanisms is missing.

The first risk population is the vagotonic (usually young) individuals. The sympathetic/parasympathetic imbalance, in favour of the latter one, would affect 7% of the population. The main features include bradycardia at rest, possible auricle ventricular blocks of different kinds and the antecedent of asystole post "vagal" discharge (28, 29). Probably, within this group, we also incorporate those individuals with history of car sickness and/or dizziness and "easy" faints.

TARKKILA and ISOLA (30) point out that patients aged less than 50 years are a risk population, which is in agreement with the accepted concept that vagal tone is greater in young people. Paradoxically young patients are frequently classified as low risk ones.

Athletes, characterized by their increased vagal tone, appear to be as another surprisingly risk population. These individuals, usually considered as very low risk patients, must be looked at from a totally different perspective. The athlete, especially the highly competitive one, in addition to the increased vagal tone, occasionally has what has been called the "athletic heart syndrome". Its features include sinus bradycardia, sinus dysrhythmias, first degree and Mobitz I type blockades and alterations in repolarisation (31). Occasionally, in this population UCA has been described during spinal anaesthesia (32).

Another risk population to consider is the patients receiving chronic pharmacological treatment with beta blockers and/or vasodilators for systemic arterial hypertension. Usually classified as ASA physical status 2, it is important to consider that the possibilities for increasing either the heart rate and/or vasoconstricting the peripheral vessels might be impaired (8). In fact, beta blocked patients usually show, at least, a mild baseline bradycardia.

Perhaps when the anaesthesiologist is faced with patients with an increased vagal tone, hypovolaemia and/or some kind of pharmacological vasodilatation, spinal anaesthesia can be a poor choice. This attitude may be especially important when significant bleeding during surgery is possible. Efforts to identify the risk population in the

preoperative period continue without clear cut definitions for clinical use as yet (33-37).

Does the choice of the local anaesthetic solution make any difference? There are few publications concerning this issue. LOTZ *et al.* (38) postulate that hyperbaric solutions can cause the level of the blockade to rise even after 30 minutes post injection and adding vasoconstrictors and alkalination of the drug may predispose to arterial hypotension (21, 33). Although only few references were found, the concept can be important, as it is suggested that the use of common techniques during spinal anaesthesia might have unexpected haemodynamic consequences.

CLINICAL PRESENTATION

UCA is usually described as "sudden", "without prodromes", "progressing in seconds" and/or a chain of events occurring very quickly, even in the hands of experienced anaesthesiologists. Sometimes nausea and/or vomiting are described before the UCA takes place.

Are these sudden onsets or sudden recognitions? It could be possible that an experienced anaesthesiologist, administering spinal anaesthesia to healthy patients on a daily basis, is not the best candidate for an extremely vigilant attitude. The final result of the problem and not its onset may be influenced by this mental attitude.

Nevertheless there are clearly described cases in which the patient was apparently doing well, with an attentive anaesthesiologist, even talking with the patient, when extreme bradycardia and UCA rapidly developed (39). A baseline bradycardia that gets worse is a common finding (8) and bradycardia and auricle ventricular blocks rapidly transforming into total heart block and asystole are described (40). Bradycardia then seems to be a bad "fellow traveller" during a spinal block and, except clear cut contraindications, it should be medically treated.

WHEN DOES OCCUR UNEXPECTED CARDIAC ARREST ?

Although the common belief is that UCA usually occurs within the first 30 minutes, this is not true as demonstrated by many cases in which the complication appeared much later. The possibility of UCA during spinal anaesthesia must be considered as widely distributed across the entire duration of the case, regardless of its duration. LESSER *et al.*

(41), using an automatic record keeping system, described the moment of the appearance of the UCA. Although several of them were installed only 1 minute after administering the spinal, the mean interval to develop UCA was 58 minutes. This finding is worrisome, since in a brief procedure the situation can arise not in the operating room but in the postoperative recovery area. When the latter one does not exist, the situation might be even worse, as UCA can appear on the ward. Although the aetiology of this "deferred" UCA is unknown, it is worth remembering that the sympathetic blockade lasts longer than the sensory and motor blockades.

Interestingly the data of the study of AUROY *et al.* (6) showed that there were less chances of surviving in those with a delayed UCA (42 ± 19 minutes) than in those with a quicker development (17 ± 16 minutes). Again, we don't know the reason of this difference. Is it a problem of surveillance and/or attitude, either in the diagnosis or treatment?

A very intriguing finding is the description of UCA appearing after small postural changes of the patient. LOVSTAD *et al.* (42) report five cases of cardiac arrest 10-70 minutes after the onset of spinal anaesthesia. UCA occurred seconds after position changes, including placing a leg in a holder and turning the patient to the left lateral or prone position. In some of the cases the surgical procedure had already finished. It seems difficult to explain these situations based only on preload changes. May be they are due to reflex phenomena similar to those of autonomic dysfunction or hyperreflexia described in patients with a spinal cord section (43, 44). If this was the case, then the lesson to be learned can be to minimally mobilize patients with spinal anaesthesia.

Finally, and once again, there is the point of the anaesthesiologist's attitude when detecting that something is "going wrong". The best advice is: believe in what is happening in front of you. Disbelief and insecurity are common patterns of this situation and may influence the final outcome. Permanent clinical surveillance and rapid response to changes in the ECG and arterial pressure monitoring data are the clue for success.

TREATMENT ONCE THE COMPLICATION HAS BEEN DETECTED

Treatment must be quick, intensive and multimodal. It is enough to have intra-operative hypotension with bradycardia to rapidly administer atropine plus a vasoconstrictor (e.g. ephedrine). It does not

seem to be wise to administer just one of these drugs and then wait for the result. We have not found the exact amount of arterial pressure decrease that triggers therapy, but in our opinion a 30% decrease in pre-operative systolic blood pressure might be a good figure, especially if accompanied by bradycardia. In addition, volume replacement (including Trendelenburg position), in spite of being important, does not improve the situation when used as the only therapy. If there is no improvement after atropine and vasoconstrictors, intravenous epinephrine must be administered without any delay, as recommended by the SOS ALR group in France (45).

Caplan's review (4) showed that those patients in whom epinephrine was used after 8 minutes of cardiac arrest, had the worse prognosis. In AUROY *et al.*'s review (6), epinephrine was used in less than half of the patients with a 25% mortality. If the patient is in complete cardiac arrest, initiate CPR and prepare for prolonged resuscitation efforts, especially if a high blockade level is present.

FUTURE RESEARCH

Future research should answer several difficult questions: 1) Is there any clear cut identification of risk populations?; 2) What happens during the minutes preceding the cardiac arrest when there are no clinical signs? Is automatic record keeping the answer?

3) What is the adequate timing for sending the patient back to the ward? Is it enough to wait for the recovery of motor and sensory block? Are cardiac arrests developing long after a spinal anaesthetic has been given, really due to the anaesthetic technique?

CONCLUSION

The common mechanism of cardiac arrest during spinal anaesthesia seems to involve still poorly understood relations among vasodilatation, reduced venous return, reduced cardiac output and bradycardia mediated by direct and/or vagal cardiac reflexes.

Potential advantages and disadvantages of spinal anaesthesia in risk populations must be balanced by the anaesthesiologist. Management of volume preloading is important although not the real clue for success. A quick intravenous injection of atropine in case of a maintained mild bradycardia

should be performed without delay, while prompt intravenous epinephrine can make a big difference in the final outcome.

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