

Cardiopulmonary Arrest in Spinal Anesthesia

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Summary: Limongi JAG, Lins RSAM – Cardiopulmonary Arrest in Spinal Anesthesia.

Background and objectives: Spinal anesthesia is an integral part of the daily routine of countless anesthesiologists. It is considered to be a safe procedure, although some complications related to this technique, among them the most feared is cardiopulmonary arrest (cardiac arrest, CA), do exist. The real incidence of CA related to spinal anesthesia, as well as its etiology, is not known and has motivated this review article.

Contents: Articles published in the last twenty years in Medline indexed journals and in a textbook were reviewed. The objective of the present review was to identify the incidence of spinal block anesthesia-related CA and the etiology of those cases. We also tried to identify possible risk factors. Finally, treatment strategies described in the literature were reviewed in order to determine the best conduct when facing a case of CA during spinal anesthesia.

Conclusions: The incidence of spinal anesthesia-related CA varies, and it seems to be lower when compared to that of general anesthesia. In the past, it was believed that CA was due to hypoxemia related especially to excessive sedation. However, nowadays, it is known that the etiology of CA during spinal block anesthesia is related to cardiocirculatory factors, mainly a reduction of preload resulting from sympathetic blockade. Other factors that increase the risk of developing CA also exist. Among those factors, the following should be mentioned: changes in patient positioning and hypovolemia. It is very important to institute treatment as soon as possible. Besides a vagolytic agent, early use of a sympathomimetic drug, especially adrenaline, is also recommended to minimize damage to the patient.

Keywords: Anesthesia, Spinal; Bradycardia; Heart arrest; Intraoperative Complications.

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INTRODUCTION

Spinal anesthesia is an important anesthetic technique in anesthesiology. This is due to the elevated success rate, pre-visibility, and patient satisfaction, beside the low rate of complications associated with this procedure. However, although it is considered a safe technique, it is not devoid of risks or adverse events ^{1,2}.

Among possible spinal block anesthesia-related complications, cardiopulmonary arrest (cardiac arrest, CA) is the most serious. Although considered to be rare, data that attempt to determine its real incidence are very contradictory ³⁻⁵.

Most publications on the subject define cardiac arrest during spinal anesthesia as the sudden development of bradycardia or asystole in the presence of a spinal block, which requires resuscitation maneuvers (chest compressions, drugs, and/or defibrillation) in hemodynamically stable patients ^{4,6,7}.

In order to identify the incidence, etiology, predisposing factors, and types of prevention and treatment of CA during spinal anesthesia, a review of articles published in the last twenty years in the Medline index and in a textbook was undertaken.

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Incidence

The first cases of cardiac arrest, as an inexplicable complication of spinal block anesthesia, were reported in the decade of 1940, when the interest in the subject arose since it involved, in most cases, young and healthy patients ⁵.

To determine the frequency of cardiac arrest during spinal anesthesia, investigators observed a remarkably heterogeneous incidence, ranging from 1.3 to 18 cases in 10,000 spinal anesthetics ^{5,8-10}.

In a study of Auroy et al. ⁸, the incidence of spinal anesthesia-related CA (6.4 ± 1.2 in 10,000) was significantly higher when compared to that incidence during epidural anesthesia and peripheral blockades together (1.0 ± 0.4 in 10,000 anesthetics). Pollard et al. ⁴ observed similar incidence of CA during spinal anesthesia (0.03%) and epidural anesthesia (0.01%) ⁸.

When the incidence of CA during spinal block anesthesia is compared to that of general anesthesia, the majority of investigators observed that CA is more common during general anesthesia ¹¹⁻¹³. However, a study by Biboulet et al. ¹⁴ demonstrated that this complication is more common during spinal anesthesia than during general anesthesia.

Chan et al. ¹⁵ observed a rate of 0.51% of deaths during the first 14 hours after anesthesia, of which 98% were observed during general or combined anesthesia and 2% during regional anesthesia. Braz et al. ¹⁶ observed that the distribution of CA according to the type of anesthesia was 12.7 times greater for general anesthesia when compared to other blocks.

To justify the higher incidence of CA during general anesthesia, the authors suggested that this type of anesthesia is more common in complex, high-risk surgeries in severely ill

patients. On the other hand, they mention the increased knowledge on the physiology of spinal anesthesia, together with the use of less toxic local anesthetics and improved monitoring, contributed to reduce the incidence of CA during spinal anesthesia.¹⁶

Etiology

In 1988, Caplan et al.¹⁷ published an analysis of the results of an American Society of Anesthesiologists (ASA) project that evaluated anesthesia-related complications. The development of 14 episodes of unexpected CA during spinal anesthesia in healthy patients (physical status I and II according to ASA classification) was observed. Regarding the characteristics of those episodes, the highest level of sensorial blockade achieved was T4, and 12 out of 14 patients were sedated and not adequately monitored. The prognosis of those patients was guarded. Despite cardiopulmonary resuscitation, six deaths were observed, and out of eight survivors, only one was able to recover enough to take care of himself, although he remained with mild cognitive deficit¹⁷.

When investigating physiological changes related to spinal anesthesia, Butterworth¹⁸ observed that blockade levels in mid-thoracic segments without paralysis of the phrenic nerve produced little or no changes in tidal volume, respiratory rate, minute-ventilation, or partial blood gas pressure. Phrenic nerve paralysis is a rare cause of apnea during spinal anesthesia, even when sensorial blockade extends up to cervical dermatomes^{4,5,10}.

After pulse oximetry became an integral part of basic monitoring, some authors did not observed desaturation before the development of CA. According to Lovstad et al.³, cases of primary asystole can develop in the absence of respiratory depression or hypoxemia induced by sedative drugs. Koop et al.⁵ and Liguori et al.¹⁹ observed that, in several episodes of documented CA, recordings immediately before and after the event did not indicate cases of arterial oxygen saturation below 90%. Besides, those authors demonstrated that many patients who developed CA had not received sedative drugs^{3,10,19,20}.

In the absence of evidence of a respiratory etiology for CA episodes during spinal anesthesia, alternative mechanisms to justify the development of this complication were investigated. Observing the frequent development of bradycardia during spinal block anesthesia, a cardiocirculatory etiology for those events was investigated⁴.

The effects of spinal block anesthesia on the cardiovascular system typically include a reduction of blood pressure and central venous pressure. Those effects are direct and indirectly related to the sympathetic nervous system blockade promoted by spinal anesthesia. Since the level of sympathetic blockade extends two to six dermatomes above the sensorial blockade, a patient with sensorial blockade in T4 can have a blockade of all his cardioaccelerator fibers (T1-T4), resulting in progressive reduction in heart rate. Sympathetic blockade at the T1 level or above results in increased vagal tonus, which causes nega-

tive inotropic, chronotropic, and dromotropic changes without opposition from the sympathetic nervous system. It should be emphasized that the tendency is to allow the start of the surgery when an adequate level of blockade for the procedure is achieved, approximately 5 to 10 minutes after injection of the local anesthetic into the subarachnoid space. However, after spinal anesthesia, the level of the blockade achieved 10 minutes after skin puncture does not correspond to the final blockade, and it depends on factors like baricity of the local anesthetic and patient position, although other factors can be involved^{3,4,38,21-25}.

Severe peripheral vasodilation, with redistribution of blood for limbs and splanchnic beds, leading to a significant reduction in venous blood return to the heart, which results on a significant reduction in preload, is the most important consequence of the loss of sympathetic tonus. With low levels of sensorial blockade (below T4), reduction in right atrial pressure can be observed in approximately 36% of the cases, while with higher blockades this reduction is more significant, reaching up to 53% of the cases^{1,4,23,26,27}.

The reduction in preload and blockade of cardioaccelerator fibers cause a reduction in heart rate. In 1992, Carpenter et al.¹ observed an incidence of heart rate below 50 bpm in 13% of patients undergoing any surgical procedure under spinal anesthesia. Since those two variable usually coexist, it is difficult to separate the individual contribution of each one for the development of bradycardia. However, according to Carpenter, sympathetic denervation in itself, in the presence of adequate preload, seems to induce only a 10% reduction in heart rate when compared to baseline levels. Thus, the reduction in preload seems to be a more important cause of reduction in heart rate during spinal anesthesia^{1,5}.

The reduction in preload can trigger three reflexes that result in severe bradycardia, and even asystole. First, an intracardiac reflex, is related to receptors in pacemaker cells. Reduction in venous return to the heart leads to a reduction in atrial filling, with consequent reduction in stretching of pacemaker cells, which, in turn, promotes reduction in heart rate^{3-5,19,20,22,26}. The second reflex is attributed to mechanoreceptors, in the right atrium and ventricle, and baroreceptors, in the right atrium and vena cava. The third reflex is determined by mechanoreceptors in the inferoposterior wall of the left ventricle, triggering the Bezold-Jarish reflex in which stimulation of those receptors increases activity in the parasympathetic nervous system and inhibits the activity of the sympathetic nervous system, producing bradycardia, systemic vasodilation, and hypotension. Usually, the reduction in LV end-diastolic volume results in reduction in the activity of those receptors, but a fast reduction in ventricular volume can stimulate and increase activity in those receptors, leading to bradycardia. Those three reflexes have in common the fact that the effector arms of each one involves an increase in vagal tonus^{3-5,19-22,26,28}.

In approximately 7% of the population, the activity of the sympathetic and parasympathetic nervous systems shows an imbalance. This situation, in which the function of the parasympathetic nervous system predominates, is known as "vagotony". Those individuals have a higher tendency to develop vagotonic

manifestations, such as nausea, pallor, bradycardia, hypotension, and syncope, stimulated by physical or emotional stress. Those individuals are at risk of developing severe bradycardia, and even asystole, in situations in which vagal predominance and reduced atrial filling pressures coexist, such as in spinal anesthesia. With the change in balance between the sympathetic and parasympathetic nervous systems due to spinal anesthesia, situations that cause an increase in parasympathetic tonus, such as fear and viscus traction, result in clinically significant vagal effects, such as bradycardia, first- or second-degree atrioventricular (AV) block, and even complete AV block. Fear, pain, and postural changes (for example the sitting position that accentuates the reduction in venous return) can contribute for the development of vasovagal reactions. Anxiety is also one of the factors capable of triggering those reactions, and if patients with propensity to develop them are exposed to emotional stress during spinal anesthesia, a vasovagal reaction might occur, which might vary from a transitory reaction to an episode of CA. Thus, not using a tranquilizer can contribute for the development of those reactions ^{4,20,29,20}.

Although the mechanism through which spinal anesthesia induces bradycardia or asystole are not completely known, it is established that the final pathway is the absolute or relative increase in activity of the parasympathetic nervous system. Therefore, patients with increased vagal tonus are at an elevated risk of developing those complications ^{4,23}.

With the sympathetic blocked produced by spinal block anesthesia and the consequent predominance of the parasympathetic nervous system, additional vagal stimulation can lead to a decrease in heart rate, cardiac output, blood pressure, and systemic vascular resistance, prolonging or even causing severe bradyarrhythmias, and particularly progression of first degree AV to second degree AV block ^{4,29}.

Jordi et al. ²³ observed the development of first degree AV block with evolution to complete AV block and asystole in patients undergoing spinal anesthesia with sensorial blockade at the level of T3. According to the authors, although the episode of asystole was immediately reversed, a retrospective analysis of the Holter indicated persistent first degree AV block for approximately six hours after anesthesia. Development of a 1st degree AV block can be a predictive sign for the development of complete AV block and asystole. However, the difficulty to diagnose 1st degree AV block in a cardioscope limits the applicability of this finding ^{5,24}.

If the development of bradycardia is considered a cardiovascular response typical of a high subarachnoid blockade, on the other hand, the presence of significant bradycardia, although less common, usually precedes episodes of CA during spinal anesthesia. Auroy observed that all cases of CA were preceded by bradycardia ^{3,9}.

RISK FACTORS

Although the development of CA during spinal anesthesia is considered the final step of a spectrum of manifestations

that start with bradycardia, establishing an association among factors related to its development can help identify patients who would be more prone to develop CA during spinal block anesthesia ⁴.

According to Carpenter et al. ¹, the risk of patients with baseline heart rate below 60 bpm to develop bradycardia during spinal anesthesia is five times higher. The risk of patients classified as physical status ASA I, typically young individuals with increased vagal tonus, to develop bradycardia, is approximately three times higher than patients with physical status ASA III and IV. However, the level of the sensorial blockade considered an important predictive factor for the development of bradycardia showed low correlation with the development of bradycardia when compared with three other factors mentioned earlier – blockade above T5 increased only twice the risk of developing bradycardia. On the other hand, according to Pollard et al. ⁴, a blockade level above T6 is considered an important risk factors for the development of bradycardia. The same authors also mention age below 50 years and the presence of AV as risk factors for the development of moderate bradycardia during spinal anesthesia ^{1,5,21}.

According to the findings of Pollard et al. ⁴, in approximately half of the cases of CA, at least two of the risk factors for the development of bradycardia were present. The presence of one single factor does not mean patients will develop bradycardia and asystole when they undergo spinal block anesthesia, but when two or more of those factors are present, the patient should be considered of a high risk for the development of bradycardia and CA during spinal anesthesia. The development of bradycardia can represent one event prior to progression from a sinus rhythm to asystole, allowing identification of a case of imminent CA.

Studies carried out to evaluate the development of complications during spinal anesthesia showed that, regarding the type of surgical procedure, the development of CA is more common during total hip arthroplasty. Auroy et al. ^{8,9} stated that this type of surgery is associated with a higher risk of cardiac arrest evolving to death, and that the development of CA has two incidence peaks: the first, early, shortly after anesthesia, due the administration of excessive doses of local anesthetic in a previously hypovolemic patient (secondary to preoperative fasting, malnutrition, dehydration, use of diuretics and/or vasodilators, among other causes) and the second, late, related to surgical events, such as bleeding, changes in patient positioning, an placement of bone cement, among others. Thus, to prevent those events, it is recommended that the level of the blockade should be limited to T6 and hemodynamic reserves should be evaluated, monitoring cardiovascular function and the degree of bleeding, to reduce preoperative morbidity and mortality. It should be mentioned that the level of sensorial blockade in elderly patients is usually higher than that of young adults with the same dose of local anesthetic.

According to Biboulet et al. ¹⁴, doses as low as 5 mg of bupivacaine, hyper- or isobaric, can cause a sensorial blockade reaching up to T2-T4. Thus, overdose of local anesthetic using the subarachnoid route is a known cause of CA in elderly patients. ^{8,9,31,32}

PREVENTION AND TREATMENT

The development of acute bradycardia and asystole during spinal block anesthesia can be, for the most part, prevented. For such, the maintenance of adequate preload, by the administration of fluids to compensate volume losses, is fundamental, besides taking into consideration changes in preload related to changes in patient positioning, removal of tourniquets, and other perioperative events. When a sudden reduction in preload is expected, rapid volume administration and, if possible, placing the patient in a head down position, can be useful. However, volume replacement in itself might not be enough to reverse vagal symptoms, and it is oftentimes necessary to administer atropine or vasopressors. Since bradycardia can be the only manifestation of an increased vagal tone in response to a reduction in preload during spinal anesthesia, early treatment (HR < 60 bpm) should be considered in patients with multiple risk factors. Brown et al.³³ reported episodes of bradycardia during spinal anesthesia and that the administration of atropine (0.4-0.6 mg) prevented evolution to CA^{4,19,26,34}.

Treatment of bradycardia depends on understanding its pathophysiological mechanisms. Most episodes of vagus-mediated syncope show fast and spontaneous resolution; therefore, treatment might not be necessary. However, in the presence of severe bradycardia and CA, adequate and early treatment should be instituted, and a vagolytic drug that reduces morbidity in cases of CA during spinal anesthesia, should be instituted^{4,29,35}.

The presence of extensive sympathetic blockade leads to significant peripheral vasodilation, which promotes increased peripheral blood flow, with the consequent reduction of cerebral blood flow during cardiopulmonary resuscitation¹⁷.

Cardiopulmonary resuscitation (CPR) is more difficult in the presence of spinal block anesthesia due to a secondary reduction in preload³³. Rosenberg et al.³⁶, when investigating episodes of induced CA in dogs undergoing spinal anesthesia with hyperbaric bupivacaine, observed a reduction in coronary perfusion pressure, hindering CPR, since a coronary perfusion pressure of at least 15 mmHg is necessary for effective CPR.

In order to increase coronary perfusion pressure, the use of a potent sympathomimetic agent is the first treatment option. Alpha-adrenergic stimulation promotes vasoconstriction, which increases peripheral vascular resistance, diastolic pressure, and coronary perfusion pressure, besides improving cerebral blood flow. However, the use of predominantly or purely α -adrenergic agents can lead to a reduction in heart rate and cardiac output as consequence of the increased afterload. On the other hand, β -adrenergic stimulation opposes the negative inotropic and chronotropic effects secondary to vagal stimulation. Thus, the use of mixed agents (α - and β -adrenergic) is effective in increasing blood pressure; however, this action is due, mainly, to the increase in heart rate and cardiac output, with less significant increase in systemic vascular resistance^{21,29}.

Rosenberg et al.³⁷ also observed, in another occasion, that spinal anesthesia produces significant suppression of adrenal

gland function, with the consequent reduction in circulating levels of norepinephrine, and probably epinephrine, in response to maximal stress, such as an episode of CA, and this catecholamine deficiency is an important mechanism to explain the development of refractory CPR during spinal anesthesia.

Ephedrine is one of the sympathomimetic agents used more often. However, it is probable that, in doses commonly used (25-50 mg), this drug is a weak α -agonist, and it is not effective in an extensive sympathetic blockade¹⁷.

In the presence of profound bradycardia during spinal anesthesia, early administration of adrenaline, a more potent α -agonist, can be critical for maintenance of coronary perfusion pressure and decrease the duration of cerebral ischemia and the degree of neurologic damage. The same author believes that the chronotropic effect of adrenaline seems to be more effective than that of ephedrine in preventing progression of bradycardia to asystole^{4,17,26}. Administration of adrenaline seems to be critical for successful resuscitation, especially when adequate response after administration of atropine and ephedrine is not observed.

According to Rosenberg et al.³⁶, the dose of adrenaline necessary to maintain coronary perfusion pressure between 15 to 20 mmHg during spinal block anesthesia ranges from 0.01 to 0.1 mg/kg. Pollard et al.²⁶ recommend 0.2 to 0.3 mg of adrenaline to treat significant bradycardia. If it progresses to CA, usual doses of resuscitation protocols should be administered as soon as possible. The use of adrenaline does not preclude administration of other drugs, especially since adrenaline is not a vagolytic agent. Pollard et al.³⁸ propose a stepwise treatment: atropine (0.4-0.6 mg), ephedrine (25-50 mg), and adrenaline (0.2-0.3 mg)^{4,17,33}.

Evolution of the knowledge on the pathophysiology of bradycardia or CA during spinal anesthesia, the use of modern and less toxic local anesthetics, as well as the identification of adequate treatment, lead to improvements in the prognosis of those patients. In the last twenty years, some studies demonstrated recovery without sequelae of patients who developed bradycardia or asystole during spinal block anesthesia when early treatment was instituted. However, the possibility of a tragic outcome does exist, such as the one reported by Lovstad et al.³ in 2000, in which a 17-year old patient, ASA I, underwent knee arthroplasty under spinal anesthesia and, 25 minutes after the blockade, she developed CA, preceded by bradycardia that evolved to death even after adequate resuscitation.

CONCLUSION

Currently, spinal anesthesia figures among the most used anesthetic techniques. Without any question, it is very safe. However, it is not devoid of complications, some of them severe.

Although the development of bradycardia is predictable, since it is a cardiovascular response to sympathetic blockade provoked by spinal block anesthesia, one should not forget that the possibility of acute evolution to CA is real, and that, among the adverse events related to this procedure, this is

the most feared complication. The severity of those events increases because they usually involve healthy young patients undergoing elective surgeries.

Initially, it was believed that CA during spinal anesthesia was related to hypoxemia secondary to excessive sedation. However, with improvement of monitoring, several authors observed that several episodes of CA were not preceded by a reduction in arterial oxygen saturation. They also observed that, in many of those cases, patients did not receive sedative drugs. From then on, possible cardiovascular causes for the development of CA were investigated. Although several factors can be involved, it is known that the sympathetic blockade provoked by spinal anesthesia causes significant peripheral vasodilation with significant reduction in preload, which seems to be the founding stone in the etiology of those cases. In case of sympathetic blockade, exacerbated activation of the parasympathetic nervous system is observed. Those effects combined result in significant bradycardia and even asystole.

Among other possible causes for those events, we should mention: abrupt changes in patient position and hypovolemia (common in elderly patients and in urgent cases), which seem to aggravate the effects resulting from reduced preload.

Based on those possible causes of cardiac arrest during spinal anesthesia, it is possible to identify patients at higher

risk, such as hypovolemic patients, those treated with beta-blockers, and the so-called "vagotonic" patients (those with a natural predominance of the parasympathetic nervous system, which manifests by heart rate below 50 bpm). In those patients, besides the administration of adequate volume, one should consider early administration of parasympatholytic agents associated or not to sympathomimetic agents, in the presence of moderate bradycardia (HR < 60 bpm).

Early diagnosis and, especially, effective and aggressive treatment are necessary when facing significant bradycardia and/or CA during spinal block anesthesia to improve prognosis of those patients. Several authors have stressed the importance of early use of adrenaline, since the sympathetic blockade causes significant vasodilation, which might make CPR difficult.

Thus, the knowledge of the physiologic changes caused by spinal block anesthesia and its complications, as well as adequate patient selection, respecting the contraindications of the procedure, is extremely important. When a decision to use spinal anesthesia is made, adequate monitoring and constant vigilance are of paramount importance. If a patient develops CA, adequate treatment should be instituted immediately. With adequate management, CPR can be successful, with complete patient recovery.

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Resumen: Limongi JAG, Lins RSAM – Parada Cardiorrespiratoria en Raquianestesia.

Justificativa y objetivos: La raquianestesia forma parte del cotidiano de innumerables anestesiólogos. Se le considera bastante segura, aunque existan algunas complicaciones relacionadas con esa técnica, entre las cuales la más temida es el apareamiento de la parada cardiorrespiratoria (PCR). El apareamiento real de PCR relacionado con la raquianestesia, como también su etiología, todavía no han quedado completamente elucidados, lo que ha motivado la realización de este artículo.

Contenido: Se revisaron artículos publicados en los últimos veinte años en revistas indexadas al Medline y un libro de texto. El propósito de esta revisión fue identificar el apareamiento de PCR relacionado con la anestesia subaracnoidea y la etiología de esos casos. Se buscó también identificar eventuales factores de riesgo. Finalmente, las estrategias de tratamiento descritas en la literatura se revisaron para determinar la mejor conducta frente a un caso de PCR en el transcurso del bloqueo espinal.

Conclusiones: El apareamiento de PCR relacionado con la raquianestesia es bastante variable, y parece ser menor cuando se le compara con la anestesia general. Antiguamente se creía que la PCR provenía de la hipoxemia, relacionada, principalmente, con la sedación excesiva. Sin embargo, hoy se sabe que la PCR en el transcurso de un bloqueo subaracnoideo, tiene una etiología cardiocirculatoria, que se relaciona, principalmente, con la reducción de la precarga resultante del bloqueo simpático. Existen también otros factores que aumentan el riesgo para que la PCR se desarrolle, entre los cuales merecen ser destacados: las alteraciones en el posicionamiento del paciente y la hipovolemia. Con relación al tratamiento, ha quedado bien documentado que lo más importante es que sea instituido rápidamente. Además de un agente vagolítico, debemos utilizar rápidamente un simpaticomimético, en especial la adrenalina, para minimizar los daños al paciente.

Descriptor: COMPLICACIONES: parada cardiorrespiratoria; TÉCNICAS ANESTÉSICAS, Regional: raquianestesia.