Balancing Blood Pressure: Understanding Hemodynamic Fluctuations in Spinal Anesthesia

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Authors

Mohammad Reza Nateghi[1], AboTaleb Saremi[1*], Bahareh Abbasi[1,2], Elham Karimi MansoorAbad[1]

1- Sarem Gynecology, Obstetrics and Infertility Research Center, Sarem Women's Hospital, Iran University of Medical Sciences (IUMS), Tehran, Iran
2- Department of Medical Genetics, National Institute of Genetic Engineering and Biotechnology (NIGEB), Tehran, Iran

Corresponding Authors: : AboTaleb Saremi; Sarem Fertility & Infertility Research Center (SAFIR), Sarem Women's Hospital, Iran University of Medical Sciences (IUMS), Tehran, Iran. Address: Sarem Women Hospital, Basij Square, Phase 3, Ekbatan Town, Tehran, Iran. Postal code: 1396956111, Phone: +98 (21) 44670888, Fax: +98 (21) 44670432.

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ABSTRACT

Spinal anesthesia is a widely utilized technique in various surgical procedures, yet it presents the potential for hemodynamic fluctuations, manifesting as hypotension and hypertension. Understanding the underlying mechanisms and predisposing factors for these fluctuations is essential for optimizing patient care and outcomes especially in obstetrics settings. Hypotension during spinal anesthesia primarily results from sympathetic blockade-induced vasodilation and reduced systemic vascular resistance. Factors influencing its occurrence include the sensory block level, anesthesia onset speed, intravenous fluid volume and type, and baseline blood pressure. Hypertension may arise from sympathetic stimulation due to pain or anxiety during the procedure. Mechanisms contributing to hypertension encompass the renin-angiotensin-aldosterone system activation and increased norepinephrine release. The individual's baseline sympathetic tone, level of sympathetic blockade, and preexisting conditions, particularly in hypertensive patients, can influence these hemodynamic responses. Obstetric patients face additional challenges due to aortocaval compression in the supine position during pregnancy. Spinal anesthesia-induced hemodynamic fluctuations, encompassing both hypotension and hypertension, require careful consideration. Effective management strategies include fluid preloading, vasopressor administration for hypotension, and anxiolytic medications or short-acting antihypertensive agents for hypertension. Further research is needed to refine these strategies and enhance patient safety during spinal anesthesia, especially in populations with specific susceptibilities, such as hypertensive and obstetric patients.

Keywords: Spinal Anesthesia; Hemodynamic Fluctuations; Hypotension; Hypertension; Obstetric Patients

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Introduction

Spinal anesthesia is a commonly used technique in various surgical procedures, including cesarean sections and lumbar disc surgery. However, it is associated with the risk of both hypotension (low blood pressure) and hypertension (high blood pressure) [1-4]. Hypotension is a well-known complication of spinal anesthesia and occurs due to sympathetic blockade, resulting in vasodilation and decreased systemic vascular resistance. In this review, we delve into the intricacies of spinal anesthesia-induced hypotension and hypertension. We explore the physiological responses, potential causative factors, and the multifaceted mechanisms that contribute to these hemodynamic fluctuations. Furthermore, we shed light on the specific patient populations, such as obstetric patients and hypertensive individuals, who may be at an increased risk of experiencing blood pressure irregularities during spinal anesthesia.

Spinal Anesthesia and Hypotension

Several factors contribute to the occurrence of hypotension, including the level of sensory block, the speed of onset of anesthesia, the volume and type of intravenous fluids administered, and the patient's baseline blood pressure [3, 5-7]. In controlled hypertensive patients, the incidence of hypotension following spinal anesthesia may be higher compared to normotensive patients [2]. Additionally, patients on antihypertensive medications, such as calcium channel blockers and β-blockers, may experience a greater decrease in blood pressure during spinal anesthesia [8]. Hypotension during spinal anesthesia can have adverse effects on organ systems, and prompt recognition and treatment are crucial to prevent complications [8]. On the other hand, hypertension following spinal anesthesia is less common but can occur in certain situations. It may be a result of sympathetic stimulation due to pain or anxiety during the procedure. In patients with preexisting hypertension, the sympathetic response to spinal anesthesia may lead to an increase in blood pressure [2, 3]. The sympathetic response to spinal anesthesia can lead to an increase in blood pressure through various mechanisms. When spinal anesthesia is administered, sympathetic nerve fibers are blocked, resulting in a decrease in sympathetic tone. However, in some cases, there can be a compensatory sympathetic response that leads to an increase in blood pressure [9]. One possible explanation for this increase in blood pressure is the activation of the renin-angiotensin-aldosterone system (RAAS). Spinal anesthesia can cause a decrease in blood pressure, which is sensed by baroreceptors in the carotid sinus and aortic arch. This triggers the release of renin from the kidneys, leading to the production of angiotensin II, a potent vasoconstrictor. Angiotensin II acts on the smooth muscle cells of blood vessels, causing vasoconstriction and an increase in blood pressure [10]. Another mechanism that may contribute to the increase in blood pressure is the release of norepinephrine from sympathetic nerve terminals. Spinal anesthesia can lead to a decrease in sympathetic outflow, which in turn can cause an increase in the release of norepinephrine. Norepinephrine acts on alpha-adrenergic receptors in blood vessels, causing vasoconstriction and an increase in blood pressure [9, 11]. Furthermore, the sympathetic response to spinal anesthesia can also be influenced by factors such as the patient's baseline sympathetic tone, the level of sympathetic blockade achieved with the anesthesia, and the presence of preexisting conditions such as hypertension. Patients with higher baseline sympathetic tone or preexisting hypertension may be more prone to experiencing an increase in blood pressure following spinal anesthesia [12]. It is important to note that the increase in blood pressure following spinal anesthesia is not always observed and can vary among individuals. Factors such as the dose and type of anesthesia used, the patient's physiological response, and the presence of other medications or comorbidities can influence the magnitude of the sympathetic response and its effect on blood pressure [1, 13]. The sympathetic response to spinal anesthesia can lead to an increase in blood pressure through mechanisms such as activation of the renin-angiotensin-aldosterone system and the release of norepinephrine. The magnitude of this response can vary among individuals and is influenced by factors such as baseline sympathetic tone, the level of sympathetic blockade achieved, and the presence of preexisting conditions [2, 9].

Spinal Anesthesia and Hypotension in Hypertensive Patients

Hypertensive patients are more prone to hypotension due to spinal anesthesia due to several factors related to their underlying condition and the physiological response to anesthesia. One possible explanation is the impaired supraspinal control over spinal sympathetic centers in patients with spinal cord injury. Autonomic dysreflexia, characterized by paroxysmal hypertension, can occur in patients with spinal cord injury due to the disconnection of spinal sympathetic centers from supraspinal control. This dysregulation of sympathetic activity can make hypertensive patients more susceptible to fluctuations in blood pressure, including hypotension induced by spinal anesthesia [11]. In the case of controlled hypertensive patients,

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increased sympathetic activity and norepinephrine levels, along with decreased parasympathetic activity, may contribute to their increased susceptibility to hypotension during spinal anesthesia. This imbalance in autonomic activity can result in persistent sympathetic stimulation and a loss of elasticity in the arterial wall, making hypertensive patients more prone to hypotensive episodes [3, 11-15]. Furthermore, the use of antihypertensive medications in hypertensive patients can also influence their response to spinal anesthesia. Different antihypertensive agents can have varying effects on the cardiovascular system, which can alter the compensatory mechanisms that regulate blood pressure during anesthesia. Calcium channel blockers and β-blockers, for example, have been associated with a higher incidence of hypotension during spinal anesthesia [16]. Additionally, the age-related changes in cardiovascular function and the presence of comorbidities commonly seen in hypertensive patients can further contribute to their increased susceptibility to hypotension during spinal anesthesia. Elderly patients, in particular, may experience hemodynamic instability during intubation and extubation, which can increase the risk of vascular events and further exacerbate the hypotensive response to anesthesia [17]. It is important to note that the occurrence of hypotension during spinal anesthesia in hypertensive patients is still a topic of debate, and there may be conflicting reports regarding its incidence and severity. Factors such as the specific patient population, the type and dose of anesthesia used, and the management strategies employed can influence the outcomes [18-21].

Spinal Anesthesia and Hypotension in Obstetric Cases

Obstetric patients are more prone to hypotension due to spinal anesthesia for several reasons. The physiological changes that occur during pregnancy, combined with the effects of spinal anesthesia, contribute to this increased susceptibility [22]. One factor is the supine position of the pregnant patient during the procedure. When a pregnant woman lies flat on her back, there is a risk of aortocaval compression, which occurs when the weight of the uterus compresses the inferior vena cava and reduces venous return to the heart. This compression can lead to a decrease in cardiac output and subsequent hypotension Additionally, the compression of the aorta can reduce blood flow to the placenta, potentially compromising fetal well-being [23]. Spinal anesthesia itself can cause sympathetic blockade, resulting in vasodilation and a decrease in systemic vascular resistance. This reduction in vascular resistance can further contribute to hypotension The sympathetic blockade also affects the baroreceptor reflex, which plays a role in regulating blood pressure. The decreased sympathetic tone can impair the compensatory response to changes in blood pressure, making obstetric patients more susceptible to hypotension [24]. Furthermore, hormonal changes during pregnancy, such as increased levels of progesterone and decreased levels of angiotensin II, can lead to systemic vasodilation and lower blood pressure. These hormonal changes, combined with the effects of spinal anesthesia, can further exacerbate the risk of hypotension in obstetric patients [25].

Spinal Anesthesia and Hypertension in Obstetric Cases

Physiological changes that occur during pregnancy, including increased blood volume and cardiac output, which can lead to higher blood pressure levels. Additionally, certain pregnancy-related complications, such as pregnancy-induced hypertension or preeclampsia, can further increase the risk of hypertension during spinal anesthesia [9]. Up to 12% of obstetric patients with pregnancy-related complications meet the criteria for thrombocytopenia in pregnancy, with approximately 1% having moderate to severe disease Thrombocytopenia can pose challenges when deciding whether to proceed with a neuraxial procedure, such as spinal anesthesia, in affected patients [26]. Spinal anesthesia causes sympathetic blockade, resulting in vasodilation and a decrease in systemic vascular resistance. However, in some cases, there can be a compensatory sympathetic response that leads to vasoconstriction and an increase in blood pressure [27]. The management of hypotension and hypertension during spinal anesthesia involves various strategies. To prevent hypotension, preloading with intravenous fluids, such as crystalloids or colloids, is commonly used. Studies have shown that colloid solutions, such as hydroxyethyl starch (HES), may be more effective in maintaining blood pressure compared to crystalloids[1]. In addition to fluid management, vasopressor medications, such as ephedrine or phenylephrine, may be administered to treat or prevent hypotension [4, 14].

Conclusion

In conclusion, spinal anesthesia can lead to both hypotension and hypertension. Hypotension is a more common complication and is primarily caused by sympathetic blockade and vasodilation. Various factors, including patient characteristics and the type of anesthesia, can influence the occurrence of hypotension. Hypertension during spinal anesthesia is less common and may be a result of sympathetic stimulation. The management of hypotension and hypertension involves fluid management and the use of vasopressor or antihypertensive medications, respectively. Further research is needed to optimize the management of hypertension during spinal anesthesia.

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Ethical Issue
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Conflict of interests
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Authors' ORCID
AboTaleb Saremi
https://orcid.org/0000-0003-4191-6624

Mohammad Reza Nateghi
https://orcid.org/0000-0001-5754-0516.

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