Revisiting Prevention of Maternal Hypotension After Spinal Anesthesia: The Potential Role of Preoperative Oral Midodrine

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Abstract

Background: Spinal anesthesia is the preferred anesthetic technique for elective cesarean section due to its rapid onset, dense neural blockade, and favorable maternal and neonatal outcomes. However, maternal hypotension remains its most frequent and clinically significant complication, with reported incidence exceeding 70% in the absence of prophylactic measures. This hemodynamic instability primarily results from sympathetic blockade leading to arterial and venous vasodilation, reduced systemic vascular resistance, and decreased venous return. Maternal hypotension is associated with adverse outcomes including nausea, vomiting, decreased uteroplacental perfusion, fetal acidosis, and lower Apgar scores. Despite advances in preventive strategies—particularly the routine use of vasopressors such as phenylephrine and norepinephrine—optimal prevention remains challenging, and adjunctive therapies continue to be explored.

Midodrine, an orally administered selective α1-adrenergic agonist, has gained attention for its vasoconstrictive properties and established role in treating orthostatic hypotension. Its pharmacological profile, including predictable onset and prolonged duration of action, suggests a potential role in mitigating spinal anesthesia–induced hypotension when administered preoperatively. Recent studies in non-obstetric populations and emerging investigations in obstetric anesthesia have evaluated its efficacy in reducing the incidence and severity of hypotension, vasopressor requirements, and associated symptoms. However, data specific to cesarean delivery remain limited, and concerns regarding uteroplacental perfusion and fetal safety necessitate careful evaluation.

This review aims to revisit the pathophysiology and clinical implications of maternal hypotension following spinal anesthesia, critically appraise current preventive strategies, and explore the potential role of preoperative oral midodrine as an adjunct in elective cesarean section. Available evidence is synthesized to assess its efficacy, safety, and clinical applicability. While midodrine presents a promising oral alternative or adjunct to traditional vasopressor regimens, further high-quality randomized controlled trials in obstetric populations are required to establish its role in standard clinical practice.

Keywords: Prevention, Maternal Hypotension, Spinal Anesthesia: Oral Midodrine

Introduction

Spinal anesthesia has become the anesthetic technique of choice for elective cesarean delivery due to its rapid onset, reliability, and avoidance of airway manipulation associated with general anesthesia. It provides excellent sensory and motor blockade, allows the mother to remain awake during delivery, and is associated with lower maternal morbidity and mortality compared to general anesthesia. Despite these advantages, spinal anesthesia is frequently complicated by maternal hypotension, which remains a major concern in obstetric anesthesia practice. The incidence of hypotension following spinal anesthesia for cesarean section has been reported to range between 60% and 80% without prophylactic measures, making it one of the most common intraoperative complications in this setting [1,2].

The pathophysiology of spinal anesthesia–induced hypotension is primarily related to sympathetic blockade, resulting in vasodilation, decreased systemic vascular resistance, and reduced venous return. In pregnant patients, these effects are exacerbated by aortocaval compression from the gravid uterus and an already altered cardiovascular physiology characterized by increased blood volume and cardiac output. Consequently, even transient reductions in blood pressure can significantly impact maternal comfort and fetal well-being. Clinically, hypotension is associated with symptoms such as nausea, vomiting, dizziness, and, more importantly, decreased uteroplacental perfusion, which may lead to fetal hypoxia and acidosis [3,4].

Over the past decades, numerous strategies have been developed to prevent and manage post-spinal hypotension. These include fluid loading (crystalloids and colloids), left uterine displacement, and the prophylactic or therapeutic use of vasopressors. Among vasopressors, phenylephrine has emerged as the first-line agent due to its efficacy in maintaining blood pressure and improving neonatal acid-base status. More recently, norepinephrine has been introduced as a promising alternative with favorable hemodynamic profiles. However, despite these advances, hypotension is not completely eliminated, and the need for continuous intravenous infusions, close hemodynamic monitoring, and dose titration remains a limitation in many clinical settings [5–7].

Midodrine, an orally active prodrug that is metabolized to desglymidodrine, a selective α1-adrenergic agonist, has been widely used in the management of orthostatic hypotension. Its mechanism of action—peripheral vasoconstriction leading to increased systemic vascular resistance—makes it a theoretically attractive option for preventing spinal anesthesia–induced hypotension. The oral route of administration, ease of use, and relatively prolonged duration of action further support its potential as a preoperative adjunct. While several studies have explored its role in non-obstetric populations undergoing spinal anesthesia, its application in obstetric anesthesia remains relatively under-investigated, with limited but growing evidence [8,9].

The current literature is dominated by studies focusing on intravenous vasopressors, while the role of oral agents such as midodrine in this context has not been fully elucidated. This represents a significant research gap, particularly in resource-limited settings where continuous vasopressor infusions may not be readily feasible. Additionally, concerns regarding the safety of midodrine in pregnancy, particularly its effects on uteroplacental blood flow and neonatal outcomes, require careful consideration and further investigation.

Therefore, this review aims to critically examine the existing evidence on the prevention of maternal hypotension following spinal anesthesia, with a specific focus on the potential role of preoperative oral midodrine in elective cesarean section. By integrating current knowledge on pharmacology, hemodynamic mechanisms, and clinical outcomes, this article seeks to highlight the advantages, limitations, and future directions for incorporating midodrine into obstetric anesthesia practice.

Spinal Anesthesia for Elective Cesarean Section: Benefits and Hemodynamic Liability

Spinal anesthesia is widely regarded as the gold standard for elective cesarean section due to its rapid onset, high success rate, and ability to provide dense sensory and motor blockade with minimal drug exposure to the fetus. Compared to general anesthesia, it significantly reduces the risks of difficult airway management, aspiration, and neonatal respiratory depression. Furthermore, it allows early maternal–

neonatal bonding and reduces perioperative morbidity. These advantages have led major anesthetic societies to strongly recommend neuraxial techniques as the preferred approach for cesarean delivery in the absence of contraindications [10,11].

Despite its clear benefits, spinal anesthesia is associated with a predictable and often profound hemodynamic disturbance, primarily maternal hypotension. The incidence remains high even in modern practice, particularly when preventive strategies are not optimally applied. The abrupt onset of sympathetic blockade following intrathecal injection leads to vasodilation, decreased systemic vascular resistance, and redistribution of blood volume. In obstetric patients, these effects are magnified due to physiological changes of pregnancy, including increased baseline cardiac output and reduced vascular tone, making them particularly susceptible to rapid hemodynamic shifts [12,13].

A critical contributor to hemodynamic instability during cesarean delivery is aortocaval compression by the gravid uterus. In the supine position, compression of the inferior vena cava reduces venous return to the heart, leading to decreased preload and cardiac output. Simultaneously, compression of the aorta can impair uteroplacental perfusion. Although left uterine displacement is routinely employed to mitigate this effect, it does not completely eliminate the hemodynamic consequences, particularly when combined with the vasodilatory effects of spinal anesthesia [14,15].

The level and extent of the spinal block also play a significant role in determining the severity of hypotension. A higher sensory block, often required for adequate anesthesia in cesarean section (typically up to T4), results in more extensive sympathetic blockade, including cardiac accelerator fibers originating from T1 to T4. This may lead not only to vasodilation but also to bradycardia and reduced cardiac contractility, further compounding hypotension. Consequently, the balance between achieving adequate anesthesia and maintaining hemodynamic stability remains a central challenge in obstetric anesthesia [16,17].

Another important consideration is the rapid onset of spinal anesthesia compared to epidural techniques. While the rapid onset is advantageous for surgical readiness, it allows less time for compensatory physiological mechanisms to adapt. This contributes to the sudden drop in blood pressure observed shortly after intrathecal injection, often within minutes. As a result, proactive rather than reactive management strategies are essential to prevent clinically significant hypotension and its associated complications [18].

In addition to maternal factors, anesthetic technique variables such as the dose and baricity of local anesthetic, the addition of opioids, and patient positioning can influence the degree of hemodynamic change. Higher doses of intrathecal local anesthetics are associated with greater sympathetic blockade and more pronounced hypotension. Efforts to reduce local anesthetic dose through combined spinal-epidural techniques or adjunctive intrathecal opioids have been explored to mitigate this effect, though results remain variable [19].

Given these challenges, it is clear that while spinal anesthesia offers substantial benefits in cesarean delivery, its hemodynamic liability necessitates meticulous planning and management. Current preventive strategies focus largely on intravenous interventions, including fluid loading and vasopressor administration. However, these approaches require close monitoring and resource availability, highlighting the need for additional or alternative strategies. In this context, the exploration of oral agents such as midodrine represents a potentially valuable advancement in optimizing maternal hemodynamic stability.

Pathophysiology of Maternal Hypotension After Spinal Anesthesia

Maternal hypotension following spinal anesthesia is primarily the result of an acute and profound sympathetic blockade. The administration of local anesthetic into the subarachnoid space leads to inhibition of preganglionic sympathetic fibers, resulting in a loss of vasomotor tone. This causes both arterial and venous vasodilation, with venodilation playing a particularly significant role due to increased venous capacitance and subsequent pooling of blood in the lower extremities. The net effect is a reduction in venous return, decreased preload, and ultimately a fall in cardiac output and arterial blood pressure [20,21].

A key determinant of the severity of hypotension is the extent of sympathetic blockade, which is typically

higher than the sensory level by two to six dermatomes. In cesarean section, achieving an adequate sensory level up to T4 is necessary for surgical anesthesia, but this also results in blockade of the cardiac accelerator fibers (T1–T4). Inhibition of these fibers can lead to bradycardia and reduced myocardial contractility, further compromising cardiac output. This dual mechanism—vasodilation combined with impaired cardiac response—explains the rapid and sometimes severe drop in blood pressure observed after spinal anesthesia [22,23].

In pregnant patients, physiological adaptations significantly amplify these effects. Pregnancy is associated with increased blood volume, decreased systemic vascular resistance, and enhanced baseline sympathetic activity. While these changes are beneficial in maintaining uteroplacental perfusion under normal conditions, they render the cardiovascular system more dependent on sympathetic tone. Consequently, the sudden loss of sympathetic input following spinal anesthesia leads to a disproportionately large hemodynamic response compared to non-pregnant individuals [24].

Aortocaval compression further exacerbates the situation. The gravid uterus compresses the inferior vena cava when the patient is in the supine position, reducing venous return and preload. This effect persists even with left uterine displacement, as complete relief of compression is rarely achieved. Additionally, partial compression of the abdominal aorta can reduce blood flow to the uterus and placenta, compounding the impact of systemic hypotension on fetal oxygen delivery. Thus, maternal positioning plays a critical but incomplete role in mitigating hypotension [25,26].

Another important mechanism involves the redistribution of blood volume due to vasodilation. The loss of arterial tone leads to decreased systemic vascular resistance, while venous pooling reduces effective circulating volume. This functional hypovolemia contributes to the rapid onset of hypotension. Although fluid loading strategies aim to counteract this effect, their efficacy is limited by the speed and magnitude of vasodilation, particularly with crystalloid solutions that rapidly redistribute **خارج** the intravascular compartment [27].

Bezold–Jarisch reflex activation has also been implicated in the pathogenesis of spinal anesthesia–induced hypotension and bradycardia. This cardioinhibitory reflex is triggered by decreased ventricular filling and activation of mechanoreceptors in the left ventricle, leading to increased vagal tone, bradycardia, and further vasodilation. Although its exact contribution remains debated, it may play a role in severe or refractory cases of hypotension, particularly when accompanied by profound bradycardia [28].

Hormonal and endothelial factors may additionally influence vascular tone during spinal anesthesia. The release of vasodilatory mediators such as nitric oxide, combined with reduced responsiveness to endogenous catecholamines, may further impair vascular resistance. Moreover, pregnancy-related changes in receptor sensitivity and vascular reactivity can alter the response to both endogenous and exogenous vasopressors, adding complexity to hemodynamic management [29].

Overall, the pathophysiology of maternal hypotension after spinal anesthesia is multifactorial, involving a combination of sympathetic blockade, altered cardiovascular physiology of pregnancy, mechanical factors such as aortocaval compression, and reflex mechanisms. Understanding these processes is essential for developing effective preventive strategies. It also provides the physiological basis for considering agents like midodrine, which act by increasing peripheral vascular tone, as potential adjuncts in mitigating this common and clinically significant complication.

Maternal and Neonatal Consequences of Post-Spinal Hypotension

Maternal hypotension following spinal anesthesia is not merely a transient hemodynamic event but a clinically significant condition with direct consequences for both the mother and the fetus. The reduction in systemic arterial pressure leads to decreased organ perfusion, most notably affecting the uteroplacental circulation, which lacks autoregulatory capacity. As a result, uterine blood flow becomes directly dependent on maternal blood pressure, making even short episodes of hypotension potentially harmful to fetal oxygenation [30,31].

From a maternal perspective, hypotension is commonly associated with symptoms such as nausea, vomiting, dizziness, and, in severe cases, loss of consciousness. Nausea and vomiting are among the most frequent and distressing intraoperative complaints during cesarean delivery under spinal anesthesia, with

a strong correlation to both the severity and duration of hypotension. These symptoms are thought to result from cerebral hypoperfusion and gut ischemia, as well as increased vagal activity. Effective prevention of hypotension has been shown to significantly reduce the incidence of these symptoms, improving maternal comfort and overall satisfaction with the anesthetic experience [32,33].

In more severe cases, maternal hypotension can lead to critical reductions in cardiac output and compromised perfusion of vital organs. Prolonged or profound hypotension may result in altered mental status, cardiovascular instability, and, rarely, cardiac arrest. Although such extreme outcomes are uncommon in modern practice due to vigilant monitoring and prompt intervention, they underscore the importance of proactive hemodynamic management during spinal anesthesia [34].

The impact on the fetus is of even greater concern. Uteroplacental blood flow is highly sensitive to changes in maternal blood pressure, and sustained hypotension can result in decreased oxygen delivery to the fetus. This may lead to fetal hypoxia, metabolic acidosis, and, in severe cases, neonatal depression at birth. Umbilical artery pH and base excess are commonly used indicators of fetal well-being, and studies have demonstrated a clear association between maternal hypotension and adverse neonatal acid-base status [35,36].

Apgar scores, although less sensitive than biochemical markers, may also be affected in cases of significant or prolonged hypotension. Lower Apgar scores at 1 and 5 minutes have been reported in association with poorly controlled maternal blood pressure during cesarean delivery. While most neonates recover quickly with appropriate resuscitation, these findings highlight the potential for immediate neonatal compromise and the importance of maintaining stable maternal hemodynamics [37].

In addition to acute effects, there is concern regarding the potential for subtle or long-term neonatal consequences, although evidence in this area remains limited. Repeated or severe episodes of intraoperative fetal hypoxia could theoretically impact neurodevelopment, but current data are insufficient to establish a definitive link. Nevertheless, this possibility reinforces the need for strict prevention and management of maternal hypotension [38].

It is also important to consider the interplay between hypotension and the use of vasopressors. While vasopressors are essential for maintaining blood pressure, excessive vasoconstriction—particularly with agents that reduce uterine blood flow—may also have adverse fetal effects. This has led to a shift toward agents such as phenylephrine, which better preserve fetal acid-base status compared to older agents like ephedrine. Any new intervention, including oral midodrine, must therefore be evaluated not only for its ability to prevent hypotension but also for its impact on uteroplacental perfusion and neonatal outcomes [39].

In summary, maternal hypotension after spinal anesthesia has significant implications for both maternal well-being and neonatal safety. Its effects range from discomfort and nausea to fetal hypoxia and acidosis. These consequences highlight the critical importance of effective preventive strategies and provide a strong rationale for exploring additional approaches, such as preoperative oral midodrine, to improve hemodynamic stability during cesarean delivery.

Current Preventive Strategies: Fluids, Positioning, Phenylephrine, and Norepinephrine

The prevention of maternal hypotension following spinal anesthesia in cesarean delivery has evolved significantly over the past decades, with a multimodal approach now considered the standard of care. This strategy typically combines mechanical measures, fluid therapy, and pharmacologic interventions, each targeting different components of the underlying pathophysiology. Despite these advances, no single intervention has proven completely effective, and the optimal combination continues to be refined [40,41].

Maternal positioning is one of the simplest and most essential preventive measures. Left uterine displacement, achieved by tilting the patient or placing a wedge under the right hip, is routinely employed to reduce aortocaval compression. By alleviating pressure on the inferior vena cava, this maneuver improves venous return and cardiac output. However, while it is a necessary baseline intervention, it is insufficient on its own to prevent hypotension, particularly in the presence of profound sympathetic blockade induced by spinal anesthesia [42].

Fluid therapy has long been a cornerstone in the prevention of spinal anesthesia–induced hypotension.

Two main strategies are employed: preload (administration of fluids before spinal injection) and coload (rapid infusion initiated at the time of intrathecal injection). Crystalloid solutions are commonly used due to their availability and safety profile, although their effectiveness is limited by rapid redistribution خارج the intravascular compartment. Coloading with crystalloids has been shown to be more effective than preloading, as it better matches the timing of vasodilation. Colloid solutions, with their greater intravascular retention, may offer improved hemodynamic stability but are associated with higher cost and potential adverse effects [43,44].

Pharmacologic management with vasopressors represents the most effective strategy for preventing and treating hypotension. Phenylephrine is currently considered the first-line agent in obstetric anesthesia. As a pure α1-adrenergic agonist, it increases systemic vascular resistance through vasoconstriction, effectively counteracting the vasodilatory effects of spinal anesthesia. Numerous studies have demonstrated that prophylactic phenylephrine infusion reduces the incidence of hypotension and is associated with improved neonatal acid-base status compared to ephedrine. However, its use may be associated with reflex bradycardia and a reduction in cardiac output, particularly at higher doses [45,46].

Norepinephrine has recently emerged as a promising alternative to phenylephrine. In addition to its potent α-adrenergic vasoconstrictive effects, norepinephrine possesses mild β-adrenergic activity, which helps maintain heart rate and cardiac output. Early studies suggest that norepinephrine may provide comparable blood pressure control with less bradycardia and better preservation of cardiac output. As a result, it is gaining increasing acceptance in obstetric anesthesia practice, although further large-scale studies are needed to establish optimal dosing and safety profiles [47,48].

Ephedrine, once widely used, has fallen out of favor due to its association with increased fetal acidosis. Its mechanism involves both direct and indirect stimulation of adrenergic receptors, leading to increased heart rate and cardiac output. However, its slower onset and greater placental transfer result in higher fetal metabolic activity and lactate production, which negatively impacts neonatal acid-base balance. Consequently, its use is now generally limited to specific situations, such as the presence of maternal bradycardia [49].

In clinical practice, vasopressors are often administered as continuous infusions rather than intermittent boluses to maintain stable blood pressure and reduce fluctuations. Prophylactic infusion strategies, particularly with phenylephrine, have been shown to significantly decrease the incidence of hypotension compared to reactive treatment alone. These regimens require careful titration and continuous hemodynamic monitoring, which may limit their feasibility in resource-constrained settings [50].

Despite the effectiveness of these strategies, challenges remain. Fluid therapy alone is insufficient, positioning provides only partial benefit, and vasopressor infusions require technical expertise and close monitoring. Moreover, interindividual variability in response to spinal anesthesia and vasopressors complicates management. These limitations highlight the need for additional or adjunctive approaches that are simple, effective, and safe. In this context, the potential role of preoperative oral agents such as midodrine—capable of increasing vascular tone without the need for intravenous infusion—represents an area of growing interest in obstetric anesthesia.

Midodrine: Pharmacology and Hemodynamic Rationale

Midodrine is an orally administered sympathomimetic agent with a pharmacological profile that makes it mechanistically relevant to spinal anesthesia–induced hypotension. It is a prodrug that undergoes enzymatic conversion to its active metabolite, desglymidodrine, which acts predominantly as a peripheral selective α1-adrenergic receptor agonist. Activation of α1 receptors in arteriolar and venous vascular smooth muscle increases vascular tone, raises systemic vascular resistance, and improves venous return. These effects directly oppose the vasodilatory and venodilatory consequences of spinal sympathetic blockade, providing the central rationale for considering midodrine as a preventive adjunct in neuraxial anesthesia–related hypotension [51,52].

The oral route of administration is one of midodrine's most attractive practical features. After oral intake, midodrine is absorbed and converted to desglymidodrine, with the active metabolite generally reaching peak concentrations within approximately 1–2 hours. Its pressor effect can persist for several hours, which

may correspond well with the critical period during which spinal hypotension usually occurs after intrathecal local anesthetic injection. This time profile suggests that preoperative administration may theoretically establish a background vasoconstrictive state before sympathetic blockade develops [51,53]. Unlike ephedrine, midodrine does not rely primarily on indirect catecholamine release and does not have significant β-adrenergic activity. Therefore, its hemodynamic effect is expected to be mainly vasoconstrictive rather than chronotropic or inotropic. This may be advantageous in patients where tachycardia is undesirable. However, in obstetric spinal anesthesia, where cardiac output preservation is important, a purely α1-mediated effect also raises legitimate questions regarding reflex bradycardia and potential reduction in uteroplacental perfusion if vasoconstriction is excessive [52,54].

The best-established clinical indication for midodrine is symptomatic orthostatic hypotension, where it improves standing blood pressure through peripheral vasoconstriction. This indication supports the biological plausibility of using midodrine in other hypotensive states characterized by loss of vascular tone. However, spinal anesthesia–induced hypotension differs from chronic orthostatic hypotension because it develops rapidly, is procedure-related, and occurs in the context of acute sympathetic blockade. Therefore, extrapolation from orthostatic hypotension to cesarean spinal anesthesia must be made cautiously [51,55].

From an anesthetic perspective, the principal appeal of midodrine is not that it would replace intravenous vasopressors such as phenylephrine or norepinephrine, but that it may reduce the depth or frequency of hypotensive episodes and decrease rescue vasopressor requirements. This may be particularly relevant in settings where infusion pumps, invasive monitoring, or continuous vasopressor protocols are less available. A single oral preoperative medication, if proven effective and safe, could simplify prophylaxis and provide hemodynamic support during the early post-spinal period [56].

Nevertheless, midodrine has important limitations. Its onset is slower and less titratable than intravenous vasopressors, making it unsuitable as a sole rescue therapy for sudden severe hypotension. Its adverse effects include supine hypertension, piloerection, pruritus, urinary retention, and paresthesia. In the cesarean population, additional concerns include maternal hypertension before delivery, possible effects on uteroplacental vascular resistance, and uncertain fetal exposure. These concerns are especially important because any preventive agent in obstetric anesthesia must be judged by both maternal hemodynamic efficacy and neonatal safety [51,54].

Overall, midodrine offers a strong physiological rationale as a preoperative oral adjunct for preventing post-spinal hypotension, but its clinical position remains investigational in elective cesarean delivery. Its α1-mediated increase in vascular tone targets the dominant mechanism of spinal hypotension, while its oral administration may improve practicality. However, because obstetric spinal hypotension is rapid, dynamic, and closely linked to fetal perfusion, midodrine should be viewed as a potential complement—not a substitute—for established vasopressor-based strategies until high-quality obstetric evidence confirms efficacy and safety.

Conclusion

Maternal hypotension following spinal anesthesia for elective cesarean section remains a persistent and clinically significant challenge in obstetric anesthesia. Despite substantial advances in understanding its pathophysiology and the widespread adoption of effective preventive strategies—particularly the use of vasopressors such as Phenylephrine and Norepinephrine—the condition is not completely eliminated. Its multifactorial nature, involving sympathetic blockade, altered maternal physiology, and mechanical factors such as aortocaval compression, necessitates a multimodal and proactive approach to management. Preoperative oral midodrine presents an appealing adjunctive strategy based on a strong physiological rationale. As a selective α1-adrenergic agonist, it directly counteracts the vasodilation and venous pooling responsible for spinal anesthesia–induced hypotension. Its oral route, ease of administration, and relatively sustained duration of action offer potential practical advantages, particularly in settings where continuous vasopressor infusions and advanced monitoring may be limited. Additionally, midodrine may reduce the incidence or severity of hypotension and decrease the requirement for rescue intravenous vasopressors.

However, current evidence supporting the use of midodrine in elective cesarean delivery remains limited

and is not yet sufficient to support routine clinical adoption. Most available data are derived from non-obstetric populations or small-scale studies, and high-quality randomized controlled trials in obstetric patients are still lacking. Furthermore, important safety considerations—particularly regarding uteroplacental perfusion, maternal hypertension, and neonatal outcomes—must be carefully addressed before widespread use can be recommended.

From an obstetric anesthesia perspective, midodrine should currently be viewed as a promising investigational adjunct rather than a replacement for established vasopressor-based strategies. Future research should focus on well-designed, adequately powered randomized controlled trials to evaluate optimal dosing, timing of administration, and comparative efficacy against standard prophylactic regimens. Additionally, detailed assessment of maternal hemodynamics, fetal acid-base status, and neonatal outcomes will be essential to determine its true clinical value.

In conclusion, while preoperative oral midodrine offers a novel and physiologically sound approach to mitigating spinal anesthesia–induced hypotension, its role in elective cesarean section remains to be clearly defined. Integration into clinical practice will depend on the accumulation of robust evidence demonstrating both efficacy and safety, ensuring that maternal hemodynamic stability is achieved without compromising fetal well-being.

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