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Spinal-induced hypotension: Incidence, mechanisms, prophylaxis, and management: Summarizing 20 years of research

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Hypotension commonly occurs in parturients undergoing cesarean delivery under spinal anesthesia. This leads to maternal and neonatal adverse outcomes, including maternal nausea and vomiting and fetal acidosis, and might even lead to cardiovascular collapse if not treated. Arterial dilatation and reduction in systemic vascular resistance are the major contributors to spinal-induced hypotension. Therefore, strategies aimed at expanding the intravascular volume with fluid loading or increasing venous return with lower extremities mechanical compression and lateral tilt have had limited effectiveness in the management of spinal-induced hypotension. Vasopressors are therefore the mainstay for the prophylaxis and treatment of spinal-induced hypotension. Phenylephrine is associated with improved neonatal acid-base status and a lower risk of maternal nausea and vomiting compared with ephedrine and is now considered the vasopressor of choice in obstetric patients. This review discusses the various strategies for managing spinal-induced hypotension with a particular emphasis on the optimal use of vasopressors.

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Spinal anesthesia is often the modality of choice for cesarean delivery. It is an effective anesthetic that avoids the risks of general anesthesia in parturients with potentially difficult airways. However, a reliable spinal block has predictable undesirable consequences including maternal hypotension leading to nausea and vomiting, decreased uteroplacental blood flow, and fetal acidosis. In South Africa, more than half the anesthetic deaths in 2011–2013 were related to spinal hypotension [1]. Much research over the last 20 years has improved the management of these adverse sequelae of spinal anesthesia, and yet they remain a persistent challenge to the anesthesia provider. The incidence of hypotension depends on the definition used [2] and on the dose of intrathecal local anesthetics administered [3]. Klohr et al. reported that 15 different definitions of hypotension were used in the literature [2]. With the most commonly used definition of a 20% drop from baseline blood pressure, an incidence of 70–80% is reported [4].

Over the last two decades, considerable changes have occurred in the management of spinal-induced hypotension in the parturient. This review summarizes various strategies that have been investigated over the years including fluid loading, vasopressors, and other methods such as mechanical lower extremity compression, positioning, and $5HT_3$ receptor antagonists. In addition, we discuss the mechanisms of hypotension and suggest areas for further study.

Mechanisms of spinal-induced hypotension

Aortocaval compression was first indicted as a cause for maternal hypotension over 50 years ago. Described in 1957, the theory poses that the gravid uterus compressing the great vessels against the lumbar vertebral bodies impedes the return of blood from the vena cava, resulting in decreased cardiac output [5]. Compression of the aorta also impedes perfusion to the uteroplacental unit. Alternately, an early study from the 1940s suggested that 16–20% of blood volume can be redistributed after spinal blockade to the lower extremities, contributing to its hypotensive effect [6]. Both mechanisms were thought to result in a reduction in central venous pressure, leading to a reduction in cardiac output and resulting in hypotension. Therefore, the mainstay of therapy included strategies such as increasing venous pressure with fluid loading, enhancing venous return with leg wrapping, and avoiding aortocaval compression with left uterine displacement. However, as will be discussed in this review, those strategies were only minimally effective, which challenged the notion that a reduction in cardiac output secondary to those mechanisms is the major factor leading to spinal-induced hypotension. In fact, recent studies assessing hemodynamic parameters have shown that cardiac output, heart rate, and stroke volume increase in the first 15 min following the initiation of spinal anesthesia [7,8]. Concomitantly, a significant decrease in systemic vascular resistance occurs [7], highlighting the fact that loss of arteriolar tone is likely the main mechanism leading to hypotension [9]. Therefore, vasopressors are currently identified as the mainstay for the management of spinal-induced hypotension.

Management strategies

Low-dose spinal anesthesia

The risk of hypotension is related to the dose of intrathecal bupivacaine. Several authors have reported that low-dose spinal anesthesia for cesarean delivery, using doses of 5–7 mg intrathecal bupivacaine, results in a smaller degree of sympathectomy, vasodilation, and hemodynamic changes, including hypotension [10]. Although a smaller dose of intrathecal bupivacaine reduces the risk of hypotension and the ensuing nausea and vomiting, it increases the need for intraoperative analgesic supplementation [3]. It also results in a shorter duration of block and a slower speed of onset [11]. The combined-spinal epidural (CSE) technique, which provides the option to augment the block with the epidural catheter if needed, should therefore be used if a low-dose spinal anesthesia is planned. This allows the administration of epidural local anesthetics to supplement or prolong a block produced by a low intrathecal bupivacaine dose if needed. Roofthooft and Van de Velde recommend prophylactic epidural top-ups if uterus is not closed at 45 min after low-dose spinal injection to prevent breakthrough pain [10]. Some researchers also deliberately administer a low spinal dose with the expectation of extending the block with epidural local anesthetics, a technique that is often described as low-dose sequential CSE technique [12]. Alternatively, the level of the block after low-

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