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Spinal Anesthesia Complications: A Comprehensive Review

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ABSTRACT

Spinal anesthesia (SA) is considered a safe procedure, but it may have some side effects including hypotension and bradycardia. Sympathetic fiber blockade and vasodilation are the main causes of hypotension. SA is performed more frequent in elderly patients despite the higher risk of hypotension and its consequences. The percentage of hypotension in elderly patients is estimated to be over 70%[1].

For prevention of hypotensive side effect may have risk of hypervolemia or myocardial ischemia in elderly population. Hypotension and bradycardia both may arise from Bezold-Jarish reflex (BJR), spinal anesthesia causes decrease in preload that stimulates BJR which may be mediated by peripheral serotonin receptors (5HT-3)[2]. These receptors are located peripherally as cardiac chemoreceptors on the cardiac vagal afferent and centrally as chemoreceptor trigger zone[3].

Shivering is a common association with spinal anesthesia during intra and postoperative periods. The causes of shivering are not clearly understood it may be due to union of some mechanisms including modulation of thermoregulatory thresholds, decreasing body core temperature, body heat distribution changes and the cooling effect of the injected fluids into neuraxis[4].

While patients feel very uncomfortable due to shivering, it causes monitors artifacts and increases the postoperative pain, heart rate,oxygen consumption and metabolic rate.These effects may lead to myocardial ischemia, hypoxemia, hypercarbia and lactic acidosis [5].

1. HYPOTENSION

Block of pre-ganglionic sympathetic fibers located in the subarachnoid space during subarachnoid anesthesia causes decreases in systemic vascular resistance, secondary to arteriolar and venous dilation with subsequent blood pooling in the body areas under the spinal block inducing systemic hypotension **[6].**

High levels of sensory anesthesia and increasing age appeared to be the two main risk factors for the development of hypotension **[7].**

Decreased cardiac reserves, structural changes in the arterioles and changes in the autonomic nervous system with increasing age may also play a role. Marked hypotension may be especially harmful to elderly patients with limited cardiac reserve **[8].**Moreover, spinal anesthesia induced hypotension increases the risk of nausea and vomiting, altered mental status, and aspiration **[9].**Intraoperative hypotension has been

associated with increased risk of myocardial damage and acute kidney injury after surgery**[10].**

A range of strategies including intravenous fluids and vasopressor drugs have been used to minimize or prevent spinal anesthesia induced hypotension (SAIH) **[11].**Intravenous fluid administration as an intervention to combat SAIH is still a matter of discrepancy regarding type of fluid: either crystalloid or colloid and timing of start of fluid therapy whether pre-load or co-load **[12].**

Vasopressors are routinely used to counteract hypotension after neuraxial anesthesia. The understanding of the mechanism of hypotension and the choice of vasopressor has evolved over the years, but there is no definitive evidence showing absolute clinical benefit of one vasopressor over the other **[13].**

2. BRADYCARDIA

The common mechanism of bradycardia under subarachnoid block in addition to sympathetic blockade and decreased venous return is postulated as parasympathetic over-dominance leading to a decrease in right arterial pressure and pressure in the great veins as they enter the right atrium lead to bradycardia **[14]**.

Baseline heart rates less than 60 beats/minute and current therapy with beta-adrenergic–blocking drugs also increase the risk factors for bradycardia **[15].**

During spinal anesthesia, a sudden decrease in ventricular volume (an empty ventricle) coupled with a vigorous ventricular contraction leads to activation of the mechanoreceptors, and subsequently increased vagal tone and decreased sympathetic activity as the heart perceives itself to be full. Other possible mechanisms of bradycardia during spinal anesthesia include excessive sedation, preexisting autonomic dysfunction, heart block, vasovagal reaction **[16]**.

3. HYPOTHERMIA AND SHIVERING

A decrease in body temperature is commonly encountered after spinal anesthesia. Subarachnoid local anesthetic administration blocks all afferents of skin temperature that patients are unable to manage the decrease in core temperature. Vasodilation due to sympathetic blockade increases skin blood flow, which decrease the body's core temperature in a reliable manner. In preparing the skin for surgery with antiseptic solutions, especially when performed on a large area, evaporation from surgical field and irrigation solutions, or fluid infusion at a higher rate, may also contribute to hypothermia during surgery. A decrease in core temperature may initiate shivering, which increases oxygen consumption. Special care should be exerted to decrease this physiologic stress, especially in elderly patients **[17].**

Shivering like tremor in patients given neuroaxial anesthesia is always preceded with core hypothermia and vasoconstriction (above thelevel of the block)**[18].**

Core hypothermia less than (36°C) is common in patients who have undergone spinal anesthesia, and may be associated with surgical wound infection, decreased immunity, coagulopathy, an increased incidence of cardiac morbidity, postoperative shivering, prolongation of hospital stay and prolonged effects of anesthetic drugs **[19].**

The local anesthetic blocks the inhibitory pathway in the brain and thus produces excitatory signs such as shivering**[20]**.It is uncomfortable for the patients and may interfere with monitoring of electrocardiogram, blood pressure (BP) and oxygen saturation. It also increases oxygen consumption, lactic acid and carbon dioxide production **[21]**.

Preventive pharmacologic therapies are alternative treatment for post-operative shivering **[22].** Non-pharmacologic approaches such as maintaining room temperature, warmers and air blankets **[23]** and hot intravenous infusions have variable effect **[24]**.

4. POSTDURAL PUNCTURE HEADACHE "PDPH"

PDPH is a troublesome complication, mostly observed in middle-aged women and the obstetric population. Lower body mass index, previous PDPH and the presence of chronic headaches are other risk factors. PDPH also decreases with age, which may be related to changes in the composition of cerebral content, which increase on cerebrospinal fluid [CSF] that may compensate and prevents its occurrence. PDPH requires differentiation from other causes of headache **[25].**

It typically occurs in the fronto-occipital region and initiates when moving from the supine position to sitting or standing up. It may vary from mild to severe and the type of pain may be dull, throbbing or burning. Vertigo, nausea and vomiting might be observed due to PDPH in some patients. Headache typically appears on the second day following the dural puncture and can range from lasting one to four days **[26]**.

The leaking of CSF across the dural hole may initiate PDPH. This is explained by the following mechanisms: a decrease in intracranial pressure causes the traction of pain sensitive cranial structures and the depletion of CSF volume may induce compensatory cerebral vasodilatation **[27]**.

5. NEUROLOGIC INJURY

Persistent parasthesias and limited motor weakness are the most common injuries, although paraplegia and diffuse injury to cauda equina roots (cauda equina syndrome) occur rarely. Injury may result from direct needle trauma to the spinal cord or spinal nerves, from spinal cord ischemia, from accidental injection of neurotoxic drugs or chemicals, from introduction of bacteria into the subarachnoid or epidural space, or very rarely from epidural hematoma **[28]**.

The mechanism by which local anesthetics produce cauda equina syndrome is not yet clear; however, in vitro evidence suggests that local anesthetics can produce damage by depolarizing neurons and increasing intracellular calcium concentrations **[29]**.

Other studies demonstrate that local anesthetics can cause neuronal injury by damaging neuronal plasma membranes through detergent like action **[30]**, or by activation of phospholipase-C which results in a decrease in membrane-cytoskeleton adhesion. It is also unclear as yet whether adjuncts added to local anesthetics (e.g., epinephrine) contribute to cauda equina syndrome **[31]**.Cauda equina syndrome is a relatively rare condition, comprising around 2–6% **[32]**.

6. INFECTIOUS COMPLICATIONS

Although bacterial meningitis following neuraxial anesthesia is an uncommon complication, in cases where it occurs it may result in severe complications, including permanent neurologic disability and death. The presence of a fever and neurologic disturbance may provide a differentiation from PDPH. Epidural abscess is generally caused by skin flora; the bacteria most frequently involved is staph aureus. It is therefore prudent to initiate treatment with synthetic penicillin, even in the absence of a positive culture. Other less common causes of infection are aerobic, anaerobic streptococci and anaerobic gram-negative bacilli. The incidence of meningitis varies between 1 in 50,000 and mostly occurs as a result of airborne pathogens. The exact mechanism for how the microorganism reaches the spinal cord remains controversial. It may occur during preparation or performing the block, with a droplet from medical personal is the predominant source. Infection is more likely to occur in streptococci in most of the cases, emphasizing the need for strict adherence to precautions while performing spinal anesthesia**[33].**

7. TOTAL SPINAL ANESTHESIA

Total spinal anesthesia occurs when local anesthetic spreads high enough to block the entire spinal cord and occasionally the brainstem during spinal anesthesia. Profound hypotension and bradycardia are common secondary to complete sympathetic blockade. Respiratory arrest may occur as a result of respiratory muscle paralysis or dysfunction of brainstem respiratory control centers**[34]**. Management includes vasopressors, atropine, and fluids as necessary to support the cardiovascular system, plus oxygen and controlled ventilation. If the cardiovascular and respiratory consequences are managed appropriately, total spinal block will resolve without sequelae **[35]**.

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