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Regional Versus General Anesthesia in Severe Aortic Stenosis: Revisiting Hemodynamic Assumptions

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Dear Editor,

Severe aortic stenosis (AS) remains one of the most challenging pathophysiologic conditions in perioperative medicine. Fixed left ventricular outflow obstruction, concentric hypertrophy, impaired diastolic compliance, and preload dependence render these patients particularly vulnerable to hypotension, tachycardia, and loss of sinus rhythm. Coronary perfusion in severe AS is critically dependent on adequate diastolic pressure and filling time. Even transient reductions in systemic vascular resistance (SVR) or increases in heart rate may precipitate myocardial ischemia or cardiovascular collapse.

Contemporary guidelines from the European Society of Cardiology and the American College of Cardiology emphasize maintenance of sinus rhythm, adequate preload, and preservation of systemic arterial pressure in severe AS.[1,2] These principles are foundational in anesthetic planning, yet the optimal anesthetic technique, regional anesthesia (RA) versus general anesthesia (GA), remains debated.

General Anesthesia: Predictable Control or Hemodynamic Risk?

General anesthesia is frequently favored in severe AS due to concerns regarding sympathetic blockade during neuraxial techniques. GA allows controlled ventilation, invasive monitoring, and titratable anesthetic depth. However, its cardiovascular effects are not negligible.

Propofol produces dose-dependent vasodilation and myocardial depression, leading to decreased SVR and arterial pressure.[3] In severe AS, where coronary perfusion pressure equals aortic diastolic pressure minus left ventricular end-diastolic pressure, hypotension may critically impair subendocardial perfusion.[4] Although etomidate is often regarded as a hemodynamically stable induction agent, its use has declined due to concerns regarding adrenal suppression, particularly in critically ill patients.[5]

Moreover, laryngoscopy and tracheal intubation induce sympathetic activation with tachycardia and hypertension.[6] In AS, tachycardia shortens diastolic filling time and reduces coronary perfusion, increasing myocardial oxygen demand in a hypertrophied ventricle with limited reserve.

Large observational studies have demonstrated that even short durations of intraoperative hypotension are independently associated with myocardial injury and increased mortality.[7,8] These findings are particularly relevant in AS patients, whose coronary perfusion is pressure dependent.

Regional Anesthesia: Reassessing Traditional Contraindications

Neuraxial anesthesia has historically been regarded as relatively contraindicated in severe AS due to the risk of abrupt sympathetic blockade and sudden reductions in SVR. This concern is physiologically sound; spinal anesthesia can produce rapid vasodilation and venous pooling.

However, emerging evidence suggests that carefully titrated neuraxial techniques may not uniformly result in catastrophic instability. Incremental epidural dosing with invasive monitoring and vasopressor readiness has been reported to maintain hemodynamic stability in selected patients.[9] The risk appears to be related more to rapid sympathectomy than to the regional technique.

Peripheral nerve blocks (PNBs) offer an even more attractive alternative. Unlike neuraxial anesthesia, PNBs generally avoid extensive sympathetic blockade. Ultrasound-guided regional techniques may allow extremity or selected surgical procedures to proceed with minimal sedation, thereby preserving spon-

taneous ventilation and intrinsic sympathetic tone. By avoiding induction-related vasodilation and intubation-induced tachycardia, PNBs may theoretically better maintain coronary perfusion pressure and myocardial oxygen balance in severe AS.

Mechanistic insights published in the Journal of the American College of Cardiology highlight that hypertrophied ventricles in AS have impaired microvascular reserve and are particularly susceptible to supply-demand mismatch.[10] Therefore, anesthetic strategies minimizing hypotension and tachycardia may confer physiologic advantages.

Historical data suggest that asymptomatic severe aortic stenosis may not carry the prohibitive surgical risk once traditionally assumed.[11] This evolving understanding invites reconsideration of rigid anesthetic dogma.

Individualized Strategy Rather Than Dichotomy

The debate should not be framed as a binary opposition between RA and GA. Instead, anesthetic management in severe AS must be grounded in preservation of preload, SVR, sinus rhythm, and controlled heart rate. Patients undergoing peripheral procedures may benefit from ultrasound-guided PNBs with minimal sedation. Conversely, major intra-abdominal or thoracic surgery may necessitate GA with invasive arterial monitoring and proactive vasopressor support (e.g., phenylephrine or norepinephrine to maintain SVR). In this context, transesophageal echocardiography (TEE) may provide valuable real time assessment of preload, ventricular function, and valvular hemodynamics, facilitating the prompt detection and targeted management of hemodynamic instability.

In conclusion, severe aortic stenosis demands physiologically guided anesthetic planning rather than adherence to historical contraindications. While abrupt neuraxial sympathectomy may remain hazardous, carefully selected and titrated regional techniques, particularly peripheral nerve blocks, may offer meaningful cardiovascular stability in selected patients. Prospective, condition-specific comparative studies are warranted to refine evidence-based recommendations.

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