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Perioperative Nitroprusside Infusion in a Patient With Severe Aortic Stenosis: Another Component of Afterload Reduction Uncovered

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ABSTRACT

Background: The traditional goals of perioperative management of severe aortic stenosis are based on maintaining a high systemic vascular resistance (SVR) to allow for good coronary perfusion.

Case Report: An 87-year-old male presented with septic arthritis of a prosthetic knee joint. Arthroplasty, implant removal, and joint washout were planned as surgical intervention. His comorbidities included severe aortic stenosis (peak/mean gradient 109/60 mmHg, aortic valve area of 0.80 cm^2), new onset mitral regurgitation secondary to a flail posterior mitral valve leaflet, and a new third-degree conduction block. A nitroprusside infusion was initiated 72 hours preoperatively and continued throughout the intraoperative period and postoperative intensive care stay. This novel use of nitroprusside improved cardiac output and forward flow through the stenotic aortic valve.

Conclusions: To our knowledge, the use of nitroprusside infusion during the intraoperative period in patients with severe aortic stenosis undergoing noncardiac surgery has not been described previously. Although contrary to the traditional goal of maintaining a high SVR, this important pharmacological intervention optimizes cardiac indices during the perioperative period in these patients.

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INTRODUCTION

Aortic stenosis is the most common valvular heart disease in the elderly. Reported incidence is between 2%-4% of the population older than 60,[1] and the classic symptom triad is syncope, angina, and dyspnea with varying degrees of exertion.[2] Better medical management of this condition means that more patients with coexisting aortic stenosis present for noncardiac surgery. Literature shows a 17.3% risk of cardiac complications and a 13% risk of mortality during noncardiac surgeries in elderly patients with aortic stenosis.[3] Consequently, such patients demand that anesthesiologists possess a precise understanding of the hemodynamic consequences and the interaction of modern anesthetic agents with the stenotic valve.

Traditional management of such patients in the perioperative period is based on the following principles:[3]

- Maintain normal sinus rhythm with a low normal heart rate to allow for adequate ejection time.
- Maintain a high systemic vascular resistance (SVR) for adequate coronary perfusion.
- Maintain good intravascular volume to allow for filling of the noncompliant left ventricle.

Therefore, anesthetic techniques for patients with low fixed cardiac output must avoid significant afterload reduction, hypotension, and tachycardia at all costs.[3]

CASE REPORT

An 87-year-old male presented with septic arthritis of a prosthetic left knee joint. Cultures of samples from the joint cavity grew *Streptococcus pneumoniae*. Removal of the prosthetic joint and washout of the joint cavity with an antibiotic spacer were scheduled to treat the sepsis. Comorbidities included severe aortic stenosis with a peak/mean gradient of 109/60 mmHg and a calculated aortic valve area of 0.80 cm^2. The patient also had new onset moderate (2+) mitral regurgitation because of a flail posterior mitral leaflet

and a new third-degree atrioventricular block complicated by endocarditis with a root abscess.

Because of the patient's complex cardiac issues, he was admitted to the coronary intensive care unit at our institution for optimization of his cardiac status prior to the knee surgery. On admission, his blood pressure was 180/100 mmHg and remained consistently on the higher side of normal. A baseline echocardiogram showed a moderate (2+) tricuspid regurgitation and a right ventricular end-systolic pressure of 70 mmHg, consistent with moderately severe pulmonary hypertension.

Invasive hemodynamic monitoring with a radial arterial line and a Swan-Ganz catheter was initiated. The baseline hemodynamic data from the Swan-Ganz catheter showed right atrial pressure of 18 mmHg, pulmonary artery pressure of 58/22 mmHg, mixed venous oxygen saturation of 58%, calculated cardiac output of 3.8 L, cardiac index of 1.6 L/min/m^2, and SVR of 2,300 dynes/cm^2.

A nitroprusside infusion was initiated 72 hours prior to surgery at 30 μg/min and titrated upward. The patient's calculated cardiac output using the thermodilution method gradually improved with the slow upward titration of the nitroprusside to 120 μg/min. The patient's high pulmonary artery pressures and systemic blood pressures also trended toward normal as the nitroprusside infusion increased. Seeing the favorable hemodynamic response to the infusion, the anesthesiology team decided to continue the nitroprusside while inducing general anesthesia. A cardiac surgery team and a cardiopulmonary bypass team were kept on standby. General anesthesia was induced with titrated increments of midazolam, fentanyl, and etomidate. Rocuronium was the muscle relaxant used for securing the airway. The patient tolerated the induction and the background infusion of nitroprusside. During the intraoperative period, cardiac output (measured with the Swan-Ganz in situ) was maintained, and stable mean arterial pressures were achieved with gradual titration of the nitroprusside infusion. Postoperatively, the patient was extubated and maintained on the nitroprusside for another 72 hours before it was gradually tapered off.

DISCUSSION

Traditionally, anesthetic management for a patient with aortic stenosis undergoing noncardiac surgery focused on maintaining a high SVR to ensure coronary perfusion in the face of low fixed cardiac output. The use of an arterial and venous dilator such as an intravenous nitroprusside infusion might be considered contradictory and potentially deleterious in such a case. However, our group at the Cleveland Clinic has extensively studied the perioperative use of nitroprusside in critically ill patients with aortic stenosis. Initial studies from our institution showed promising outcomes with an improvement in cardiac index in patients with severe aortic stenosis presenting with congestive cardiac failure.[4,5] Khot et al,[5] in a prospective study, determined the response to intravenous nitroprusside in 25 patients with severe aortic stenosis and left ventricular systolic dysfunction. Included in this study were patients admitted to the intensive care unit for invasive hemodynamic monitoring for heart failure and who had a depressed ejection fraction ($\leq$0.35), severe aortic stenosis (aortic valve area $\leq$1 cm^2), and a depressed cardiac index ($\leq$2.2 L/min/m^2). The results showed, after 24 hours of nitroprusside infusion, a significant ($P<0.001$) increase in cardiac index (2.52 $\pm$ 0.55 L/min/m^2) in patients with severe aortic stenosis compared with baseline. Nitroprusside was well tolerated and did not have significant side effects. Khot et al[5] hypothesized that the favorable response to nitroprusside was based on the dynamic, variable component of the pathophysiology of aortic stenosis.

The low fixed cardiac output state in patients with aortic stenosis sets up an elevated adrenergic state, increased afterload, and increased left ventricular wall tension that are all a part of this dynamic component of the disease pathophysiology. This hyperadrenergic compensatory state ultimately puts excessive stress on the noncompliant left ventricle wall and exacerbates and worsens congestive heart failure. Nitroprusside infusion counteracts the increased afterload and wall tension of this increased adrenergic state and consequently increases cardiac output and forward flow.

Nitroglycerin may have similar beneficial effects on the pulmonary artery and systemic arterial pressures. However, because nitroglycerin is predominantly a venodilator, it decreases preload and would in fact have a deleterious effect on cardiac output. In addition, the reflex tachycardia that ensues would preclude adequate ejection time, which is essential in patients with aortic stenosis.

To further corroborate these findings in our patient, we compared a baseline transthoracic echocardiogram taken prior to the initiation of the infusion with ones taken during the infusion period and after he was weaned from the nitroprusside infusion (Table). We found a definite decrease in the peak/mean gradient and the velocity across the left ventricular outflow track and the aortic valve during the infusion period. This decrease translated into improved cardiac output and forward flow during the perioperative period. The end result was improved end organ perfusion and stable hemodynamics during surgery. Although the coexisting mitral regurgitation would have also responded favorably to the

Table. Echocardiographic Parameters at Baseline Compared With Those During and After Weaning From the Nitroprusside Infusion

Measurements	Baseline	Nitroprusside Infusion	Nitroprusside Weaned
Aortic valve area	0.80 cm^2	0.80 cm^2	101 cm^2
Dimensionless velocity index*	0.26	0.18	0.31
Peak/mean gradient	109/60 mmHg	70/30 mmHg	102/55 mmHg
Left ventricular outflow tract mean velocity	93.5 cm/sec	45 cm/sec	109 cm/sec
Aortic valve mean velocity	361 cm/sec	245 cm/sec	350.7 cm/sec

*Dimensionless velocity index calculated as left ventricular outflow tract mean velocity/aortic valve mean velocity.

systemic vasodilator and afterload reduction, our patient's regurgitant lesion was graded as a moderate 2+ and therefore the predominant effect on the cardiac physiology was based on the severe aortic stenosis.

However, we do not recommend blanket use of nitroprusside in all patients with severe aortic stenosis. Hemodynamically stable (normotensive or hypertensive) patients with severe aortic stenosis are the best candidates for nitroprusside therapy and receive the most benefits. Graded titration and monitoring hemodynamic indices and responses to the nitroprusside are prerequisites in all cases.

CONCLUSION

Our case report highlights the beneficial effects of perioperative nitroprusside infusion on forward flow and valvular gradients in a patient with severe aortic stenosis presenting for noncardiac surgery. To our knowledge, the use of this agent during the intraoperative period in noncardiac surgery patients with severe aortic stenosis has not been described previously. This usage presents a challenging scenario for nitroprusside infusion because the intravenous and inhalation anesthetic agents have a complex interplay with cardiac physiology and add to systemic vasodilatory effects. Though contradictory to the traditional view of a raised SVR maintaining perfusion in aortic stenosis patients, nitroprusside improves cardiac function in patients with decompensated heart failure resulting from severe left ventricular systolic dysfunction and severe aortic stenosis. Perioperative physicians and anesthesiologists must consider the nitroprusside infusion as a bridge to aortic valve replacement and to oral vasodilator therapy and during an acute insult such as a noncardiac surgery in these critically ill patients.

REFERENCES

1. Stewart BF, Siscovick D, Lind BK, et al. Clinical factors associated with calcific aortic valve disease. Cardiovascular Health Study. *J Am Coll Cardiol*. 1997 Mar 1;29(3):630-634.
2. Grimard BH, Larson JM. Aortic stenosis: diagnosis and treatment. *Am Fam Physician*. 2008 Sep 15;78(6):717-724.
3. Christ M, Sharkova Y, Geldner G, Maisch B. Preoperative and perioperative care for patients with suspected or established aortic stenosis facing noncardiac surgery. *Chest*. 2005 Oct;128(4): 2944-2953.
4. Skjodt NM, Townsend DR. Best evidence in critical care medicine: treatment: vasodilatation with nitroprusside in symptomatic patients with severe aortic stenosis and congestive heart failure improves cardiac index. *Can J Anaesth*. 2004 Dec;51(10):1042-1044.
5. Khot UN, Novaro GM, Popović ZB, et al. Nitroprusside in critically ill patients with left ventricular dysfunction and aortic stenosis. *N Engl J Med*. 2003 May 1;348(18):1756-1763.

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