Systolic Anterior Motion of the Mitral Valve with Left Ventricular Outflow Tract Obstruction: Three Cases of Acute Perioperative Hypotension in Noncardiac Surgery

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In this report we describe three cases of severe perioperative hypotension in noncardiac surgery patients. As systolic anterior motion of the mitral valve in combination with subaortic left ventricular outflow tract obstruction is an unrecognized cause for hypotension in noncardiac surgery patients, delayed diagnosis can result in erroneous treatment regimen. The aim of the present report is to provide an informative and brief synopsis of the pathophysiological consequences and diagnostic/therapeutic strategies for the perioperative management of systolic anterior motion.

Hypotension is one of the most common perioperative complications during general and regional anesthesia (1–3). It may increase perioperative morbidity and mortality significantly and contribute substantially to adverse postoperative outcome (4,5). A significant relationship between the incidence and severity of intraoperative hypotensive episodes and the evolution of postoperative renal failure and myocardial infarction has been demonstrated (6,7). Moreover, perioperative hypotension was repeatedly identified as an independent risk factor for postoperative multiple organ dysfunction and death in patients undergoing major surgery (8,9).

The underlying causes of perioperative hypotension are multiple, with anesthesia-mediated venodilation and hypovolemia being the most common pathogenic mechanisms (10). Particularly in high-risk patients, other conditions, such as acute cardiac failure or overwhelming vasodilation resulting from systemic inflammation or sepsis, may cause hypotension (11). Further triggers of hypotension and perioperative cardiovascular collapse may include anaphylaxis, arrhythmias, tension pneumothorax, or pericardial tamponade (10).

Aside from these causes, an under-recognized cause of acute and mostly severe perioperative hypotension in noncardiac surgery is the phenomenon of systolic anterior motion (SAM) of one or both mitral valve leaflets, leading to left ventricular outflow obstruction, mitral valve insufficiency, and severe cardiovascular destabilization. Although SAM of the mitral valve is a known phenomenon in cardiology and particularly in cardiac anesthesia, (12) SAM has not been well described in noncardiac anesthesia and may not be as familiar to the majority of anesthesiologists practicing noncardiac anesthesia.

This case series describes three patients with severe perioperative hypotension due to SAM of the mitral valve, and gives an overview of this mechanism of acute cardiovascular collapse during noncardiac surgery.

Case 1

A 69-year-old woman with multiple sclerosis suffered from bilateral femoral neck fracture after falling out of her wheelchair. Except for medically treated chronic arterial hypertension (138/60 (86) mm Hg), the patient had no other history of preexistent, anesthesia-relevant diseases (sinus rhythm, 94 bpm). Volume resuscitation was performed in the preclinical setting and in the emergency room. On the third posttraumatic day, the patient underwent orthopedic surgery. Five minutes after induction of general anesthesia with fentanyl (2 µg/kg), propofol (2.5 mg/kg), and rocuronium (0.5 mg/kg) the patient developed acute hypotension (77/35 (49) mm Hg). Hypotension was initially treated according to

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the institutional protocol with crystalloid infusion and theoadrenaline/Caffedrine injections. Nonetheless, the patient repeatedly developed severe hypotensive episodes. Emergent transesophageal echocardiography (TEE) revealed a hypovolemic and hypercontractile left ventricle (Fig. 1). During another tachycardic episode with a severe decrease in arterial blood pressure, continuous TEE demonstrated SAM of the mitral valve leading to obstruction of the left ventricular outflow tract. In response, colloid solutions were administered and a continuous phenylephrine infusion was initiated (2.35 µg·kg⁻¹·min⁻¹). Tachycardia and ventricular hypercontractility were treated with a sotalol infusion (1.7 µg·kg⁻¹·min⁻¹). Under this therapy, surgery could be finished without further major cardiovascular complications. Postoperatively, the patient was treated uneventfully in the intensive care unit (ICU). After slow withdrawal of phenylephrine and sotalol infusion, the patient was discharged from the ICU to the surgical ward on postoperative day 4.

Case 2

A 64-year-old man suffering from chronic arterial hypertension (125/84 (91) mm Hg under medical treatment) and hyperuricemia underwent cholecystectomy with hepatic resection because of gallbladder carcinoma. Hypovolemia developed because of extensive intraoperative bleeding. Despite infusion of 3 L of crystalloid solution, several acute hypotensive episodes occurred in the context of a sustained sinus tachycardia. After his arterial blood pressure decreased to a nadir of 55/30 (38) mm Hg, TEE was emergently performed and demonstrated a significantly under-filled left ventricle. Under this therapy, surgery could be finished without further major cardiovascular complications. Postoperatively, the patient was treated uneventfully in the intensive care unit (ICU). After slow withdrawal of phenylephrine and sotalol infusion, the patient was discharged from the ICU to the surgical ward on postoperative day 4.

Case 3

A 72-year-old woman underwent total femur prosthesis because of repeated hip prosthesis luxations. The patient chronically suffered from arterial hypertension (155/85 (109) mm Hg, 71 sinus bpm), gastric ulcer disease, and hyperlipidemia. After induction of general anesthesia with fentanyl (5 µg/kg), propofol (2.5 mg/kg), and rocuronium (0.5 mg/kg), the patient developed sustained hypotension (80/35 (50) mm Hg) together with sinus tachycardia (182 bpm). Hypotension could not be adequately treated using crystalloid fluid infusion plus theoadrenaline/Caffedrine injections. After emergently introducing a TEE probe during hypotension, an underfilled, hypercontractile left ventricle exhibiting SAM of the mitral valve leading to left ventricular outflow tract obstruction was revealed as the cause for cardiovascular collapse. Colloid infusion (1000 mL of gelatin) and phenylephrine infusion (3.8 µg·kg⁻¹·min⁻¹) stabilized arterial blood pressure but sinus tachycardia (150 bpm) persisted. Additional sotalol therapy was initiated and decreased her heart rate to ~100 bpm. The patient was postoperatively transferred to the ICU. Stable cardiovascular function allowed for slow withdrawal of phenylephrine and sotalol infusions. On postoperative day 4, the patient was discharged from the ICU with stable cardiopulmonary function.

Discussion

SAM of the mitral valve is the mechanism that describes dynamic left ventricular outflow tract obstruction by anterior motion of either one or both mitral valve leaflets during systole leading to cardiovascular collapse. Narrowing or obstruction of the left ventricular outflow tract may be accompanied by mitral valve regurgitation. Although SAM has first been described in patients with septic and ventricular hypertrophic cardiomyopathy and was once even considered to be pathognomonic for this disease, (13) other cardiac pathologies are much more frequently associated with SAM. For

**Figure 1.** Intraoperative transesophageal echocardiography revealing an asymmetric hypertrophic ventricular septum with systolic anterior motion of the mitral valve (A) and left ventricular outflow obstruction (B).
example, SAM of the mitral valve develops in 5%–10% of patients undergoing mitral valve annuloplasty (14). Left ventricular outflow tract obstruction resulting from septal-mitral valve contact was further reported to complicate acute myocardial infarction as a consequence of compensatory hyperkinesia of the normally perfused, residual myocardium (15). Death from SAM has been shown to occur in severely ill patients even without preexistent cardiac disease (16) and was once even hypothesized to be the terminal pathway of lethal hypovolemia.

The pathophysiology of SAM is believed to include rare predisposing features (primary anomalies of the myocardium or the mitral valve apparatus) and mainly hypercontractility of the left ventricle resulting in rapid blood flow velocities in the outflow tract (17). Thus, parts of the anteriorly displaced mitral valve leaflets extend past their coaption point and protrude into the rapid velocity flow of the left ventricular outflow tract (Fig. 1) (12).

There is agreement that SAM of either one or both mitral valve leaflets is caused by the action of left ventricular flow on protruding parts of the mitral valve. However, the nature of the hemodynamic force is a subject of debate. Initially, investigators suggested that anterior motion is caused by the Venturi mechanism (13). Thereby, rapid velocities in the outflow tract were supposed to lift the mitral valve towards the septum. However, more recent data showed that although Venturi forces are present in the outflow tract during the onset of SAM, their magnitude is much smaller than previously assumed (18).

More current studies indicate that the dominant force acting on the anteriorly displaced mitral valve leaflet is drag that is directly proportional to the velocity of flow in the left ventricular outflow tract and to the angle between the anterior mitral leaflet and the direction of flow in the left ventricular outflow tract (18). The anterior mitral valve leaflet is swept towards the septum by the drag, and as the leaflet approaches the septum, the gradient across the outflow tract rises. The gradient becomes the major hydraulic force acting on the mitral leaflet, encouraging further excursion towards the septum. An amplifying feedback loop is therefore established, facilitating further obstruction (12).

The closer the mitral valve approaches the ventricular septum and the longer the leaflet is in contact with it, the higher is the pressure gradient and the degree of obstruction. The time of onset and the duration of mitral leaflet-septal approach determine the magnitude of the pressure gradient in the left ventricular outflow tract and are used to grade SAM (Table 1) (19).

As a concomitant phenomenon, anterior displacement of the mitral valve during systole often results in relative malcoaption of the valvular leaflets and may thus lead to mitral regurgitation. Typically, the jet of mitral regurgitation is directed laterally or posteriorly (12). Thus, acute mitral regurgitation in SAM of the mitral valve can compound the low output state by further reducing forward left ventricular stroke volume.

Numerous events can trigger SAM of the mitral valve in the perioperative setting. Although preexistent anatomical anomalies of the myocardium or the mitral valve apparatus are rare causes, perioperative SAM of the mitral valve is mostly caused by functional changes in ventricular geometry (i.e., changes in papillary muscle orientation and annular conformation of the mitral valve). Those geometric changes are mainly secondary and result from either absolute or relative decreases in ventricular preload leading to an underfilled, hypercontractile ventricle, in which parts of the mitral valve apparatus are displaced into the left ventricular outflow tract.

Absolute reductions in left ventricular preload in perioperative patients are mostly attributable to hypovolemia or application of venodilating medications, such as most anesthetics. Accordingly, all patients presented in this case series developed SAM after induction of general anesthesia or in the setting of perioperative hypovolemia. However, because neuroaxial anesthesia can also substantially decrease peripheral vascular resistance, and thus ventricular preload, SAM of the mitral valve may complicate not only general anesthesia but also regional anesthetic techniques.

In the perioperative setting, a relative reduction in left ventricular filling is often secondary to increased catecholamine concentrations leading to a hypercontractile, under-filled ventricle (stress, catecholamine infusion, hyperdynamic circulation during the systemic inflammatory response or sepsis). Additionally, relative decreases in ventricular preload depend largely on preexistent patient factors such as ventricular compliance and lusitropy, making patients with hypertrophic cardiomyopathy and diastolic dysfunction more susceptible to develop perioperative SAM (20).

Table 1. Echocardiographic Grading of Systolic Anterior Motion of the Mitral Valve

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
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<tbody>
<tr>
<td>I</td>
<td>No mitral leaflet-septal contact, minimum distance between the mitral valve and the ventricular septum during systole &gt;10 mm</td>
</tr>
<tr>
<td>II</td>
<td>No mitral leaflet-septal contact, minimum distance between the mitral valve and the ventricular septum during systole &lt;10 mm</td>
</tr>
<tr>
<td>III</td>
<td>Brief mitral leaflet-septal contact (&lt;30% of systole time)</td>
</tr>
<tr>
<td>IV</td>
<td>Prolonged mitral leaflet-septal contact (&gt;30% of systole time)</td>
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Considering the underlying mechanic pathology, visualization of anterior motion of one or both mitral valve leaflets by echocardiography is the perioperative diagnostic approach of choice (21,22). Although TEE is superior to other diagnostic methods for diagnosing left ventricular outflow obstruction, (23) transthoracic echocardiography may be a reasonable alternative in the perioperative setting if surgical circumstances allow access to the anterior chest wall (24).

Moreover, echocardiography, and in particular continuous TEE, can reliably identify underlying trigger mechanisms of SAM such as absolute or relative reductions in ventricular preload. Thus, echocardiography can not only diagnose SAM of the mitral valve but also facilitate early treatment of underlying causative factors and it may thereby even prevent development of SAM. This emphasizes the diagnostic importance of echocardiography during severe hypotensive episodes, even in the noncardiac surgical setting.

According to the two pathophysiological different causes of reductions in ventricular preload, perioperative treatment of SAM depends on its underlying etiology. Whereas the first-line therapy in patients presenting with absolute reductions in ventricular preload includes immediate volume substitution and, if needed, vasopressor infusion to ensure vital organ perfusion pressure, the therapeutic approach towards SAM resulting from relative reductions in ventricular preload is more complex and includes the application of β-adrenergic blockers in selective cases. Whereas infusion of β-adrenergic blockers in a hypovolemic patient can severely aggravate cardiovascular failure, cautious application of β-adrenergic blockers may reduce both force of contraction and heart rate, thus increasing left ventricular filling time and volume in patients with hypercontractile ventricles (25).

**Conclusion**

SAM of the mitral valve with consequent left ventricular outflow tract obstruction and mitral regurgitation may be an under-recognized cause of acute perioperative cardiovascular collapse. SAM can be triggered by numerous factors inducing either relative or absolute reductions in left ventricular preload, such as hypovolemia, anesthetic drug-mediated venodilation, and increased catecholamine concentrations. The perioperative diagnostic method of choice is echocardiography, with the transesophageal approach being most suitable. Acute treatment of SAM includes infusion of volume and vasopressors and, in selected patients with relative reductions in ventricular preload, the application of β-adrenergic blockers.

**References**


