Aortic Stenosis

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**Introduction**

A recent influx of patients undergoing the transcatheter aortic valve replacement (TAVR) procedure sparked interest in the pathophysiologic process of Aortic Stenosis (AS), especially in the geriatric population. AS is most asymptomatic until later stages of the disease, which delays treatment and interventions leading to poor outcomes and prognosis, especially for those who are not surgical candidates. The TAVR procedure has allowed patients who are otherwise not candidates for open heart surgery under valve replacement in a low risk fashion, prolonging life and improving quality of life. AS causes an increase in afterload, decreasing output of blood from the left ventricle. This process can lead to an array of complications including left sided heart failure. The underlying pathophysiological process of AS will be outlined in this presentation.

**Subjective Findings**

- Decreased breath (SOB) upon exertion or at rest with severe AS
- Secondary to left ventricular heart failure and subsequent pulmonary congestion
- Signs that may be related to increased workload on the left ventricle and a subsequent increase in oxygen consumption
- Typically manifested by activity and relieved with rest
- Syncope related to a decrease in cardiac output and a subsequent decrease in cerebral perfusion
- May be precipitated by high left ventricular pressures causing acute baroreceptor activated vasodilation leading to decreased cardiac output
- Palpitations related to a decrease in atrial fibrillation or sinus tachycardia
- Increased fatigability and decline in activity

**Objective Findings**

- Systolic ejection murmur upon auscultation of heart tones, best heard between the second and third intercostal space on right sternum border, occurring after S1 and ending before S2
- Pulmonary crackles upon auscultation secondary to congestive heart failure and pulmonary congestion
- Jugular vein distention may be present in severe AS
- Tachypnea, or other cardiac aneurysms such as arterial fibrillation (AF) or Aortoatrial blocks may be present
- Hypertension
- Decreased oxygen saturation
- Other Hemodynamic changes are outlined in the table below.

(Cary & Pearce, 2013)

**Grading AS**

- Graded as mild, moderate or severe (Kurtz & Otto, 2010)
- Three patterns are used in diagnosis, which are assessed through Doppler echocardiogram (Kurtz & Otto, 2010)
  - Aortic jet velocity
  - Mean aortic valve pressure gradient
  - Aortic valve area
- According to Cary and Pearce (2013), “Aortic jet velocity is a direct measurement of the severity of stenosis and is the strongest predictor of clinical outcome” (p. 62-69).
- AS is considered hemodynamically significant when the aortic valve area is less than 1.0 cm²
- Otto (2010) defines severe AS as “the degree of valve obstruction at which symptoms might result from the valve obstruction” (p. 286).
- Grading specifications based on findings from echocardiogram are outlined below (Cary & Pearce, 2013).

<table>
<thead>
<tr>
<th>Grading</th>
<th>Aortic jet velocity</th>
<th>Mean aortic valve pressure gradient</th>
<th>Aortic valve area</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severe</td>
<td>&gt;4.0 m/s</td>
<td>&gt;50 mmHg</td>
<td>&lt;1.0 cm²</td>
</tr>
<tr>
<td>Moderate</td>
<td>2.5-3.9 m/s</td>
<td>20-50 mmHg</td>
<td>1.0-1.9 cm²</td>
</tr>
<tr>
<td>Mild</td>
<td>1.0-2.4 m/s</td>
<td>5-20 mmHg</td>
<td>&gt;1.9 cm²</td>
</tr>
</tbody>
</table>

(Cary & Pearce, 2013)

**Significance of Pathophysiology**

- Pathophysiology of AS can lead to decompenstation of left ventricle because of increased workload and oxygen demand, leading to heart failure and pulmonary congestion (Cary & Pearce, 2013).
- Severe AS can lead to life threatening symptoms such as angina, syncope, and cardiac arrest (Traylor, 2013).
- Understanding of pathophysiological process of AS is key to medical management
  - Possible use of antinflammatory drugs to decrease calcification and remodeling (Gould, Simmons & Ansell, 2013)
  - Anti-anginal agents to decrease cholesterol and subsequent reduction in obstructed LUS (Gould, Simmons & Ansell, 2013)
  - Vasodilators, diuretics, and possibly beta blockers helps to reduce left ventricular workload and decrease shear and stress on valvular tissue (Cary & Pearce, 2013)
- Surgical replacement with beta blockers, as not to potentiate systemic heart failure.
- Surgical replacement of open chest or TAVR. Poor prognosis for medically managed patients (Traylor, 2013).

**Underlying Pathophysiological Process**

- Multiple pathological processes are thought to play a role in the development of aortic stenosis
- Biomechanical factors
  - Creation of increased pressure and shear on the valve cusps, producing inflammation and extracellular remodeling
- Lipid accumulation
  - Oligodendrocytes engulfs by macrophages from foam cells. These, along with T lymphocytes, activate proinflammatory cytokines, which promote apoptosis and tissue remodeling with subsequent fibrosis and calcification.
- Calcification
  - Valvular interstitial cells (VICS) differentiate and proliferate into osteoblasts, induced by a combination of mechanical forces, inflammatory markers and growth factors, which replace normal tissue with dense collagen and bone, leaving the valve stiff and non-compliant.
- (Gould, Simmons & Ansell, 2013)

**Normal Valve**

**Stenotic Valve**

**Implications for Nursing Care**

- The nurse caring for the patient must be highly aware of the pathophysiological process of AS, as well as treatments and possible complications (Cary & Pearce, 2013).
- Deep understanding of stress range of pretest and afterload to maintain adequate cardiac output (Gould, Simmons & Ansell, 2013).
- Maintenance of activity to balance oxygen supply and demand (Cary & Pearce, 2013).
- Thorough focused assessment of patient including, but not limited to, auscultation of heart tones and breath sounds, presence of jugular venous distention, syncope or dizziness, arrhythmias, heart rate and rhythm, and presence of edema are crucial in the identification and differentiation of possible complications (Cary & Pearce, 2013).
- Clinical awareness of decompenstation of patient including decreased oxygen saturation, change in mental status, confusion, tachypnea, cardiomyopathy, and shortness of breath is crucial (Gould, Simmons & Ansell, 2013).
- Angio aneurysm with rashes, red, and uterine therapy or associated with-hypertension may prompt intra aortic balloon pump therapy to augment coronary perfusion and decrease myocardial workload (Cary & Pearce, 2013).
- Education must be provided to patients diagnosed with aortic stenosis regarding recognition of warning signs and symptoms and prompt reporting of these changes to their health care provider (Cary & Pearce, 2013).

**Conclusion**

AS is a disease that affects roughly one and a half million people in the United States alone (Ozkan, 2010). Aortic Calcific Stenosis is the most common type, and multiple pathophysiological processes contribute to the development of this disease. Mechanic stress on the valve over time can cause inflammation and remodeling of the tissue, rendering the valve dysfunctional. Oligodendrocytes that accumulates in the valvular tissue activate the immune system and other biochemical substances that promote apoptosis of valvular cells, fibrosis, and calcification. Also, both of these processes can cause differentiation of VICS into osteoblasts, replacing normal tissue with dense collagen and bone leaving the valve stiff and non-compliant (Gould, Simmons & Ansell, 2013). Understanding the underlying pathophysiological of AS can help health care providers better care for these patients, as well as develop future treatments that may decrease mortality and improve outcomes. The nurse must have a firm grasp on the process of this disease in order to effectively care for the patient in the acute care setting. Through assessment, knowing signs and symptoms of AS, as well as being able to identify the decompenstation of the patient is key to successful care of a patient with AS. Surgical intervention is the gold standard of treatment for AS, as medically managed patients have extremely poor outcomes. The TAVR procedure has helped those who are not typically surgical candidates undergo valve replacement, improving outcomes and quality of life in many patients (Traylor, 2013). Further research on the pathophysiological process of AS can hopefully guide the development of pharmacological advances that may help improve outcomes and decrease mortality rates in medically managed patients with AS.

**References**


**Additional Sources**


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