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Aortic Stenosis

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Introduction

- Aortic stenosis is becoming more prominent as the average lifespan of humans increases., as statistics are reporting that greater than 4% of Americans and Europeans over the age of 75 years old are directly impacted by some degree of aortic stenosis (Cary & Pearce, 2013).
- In aortic stenosis, narrowing of the aortic valve causes some degree of obstruction in blood flow from the left ventricle to the aorta often brought on by three causes: calcium degeneration seen in aging, congenital bicuspid valve, and inflammatory damage from rheumatic heart disease (McCance et al., 2014).
- As a nurse working in a cardiac intensive care unit frequently caring for patients with aortic stenosis, it is imperative that proper education on the topic of aortic stenosis is performed. Whether it be a community event, doctor's office, or hospital setting, education and awareness of aortic stenosis can lead to saving lives and improved quality of life.

Underlying Pathophysiology

- The most common cause of aortic stenosis is related to calcium build up on the aortic valve leaflets. There are two different mechanisms in which this may occur:
 - 1) "Mechanical stress or disease causes valvular interstitial cells within the valve leaflets to transform from the usual state of maintenance and repair into an activated state in which cell proliferation is increased and myofibroblasts and osteoblasts develop, promoting calcification, osteogenesis, and bone formation" (Cary & Pearce, 2013).
 - 2) "Mechanical stress associated with blood crossing the aortic valve damages the basement membrane of the leaflets, allowing entry and accumulation of T lymphocytes, monocytes, and low-density lipoprotein that then initiate inflammation and oxidation of the lipoprotein" (Cary & Pearce, 2013).
- Calcium deposition that accumulates on the aortic valve leaflets through cellular manifestations "bulge outward toward the aorta and extend to the sinuses of Valsalva, causing restricted leaflet motion and obstruction of left ventricular outflow during systole" (Cary & Pearce, 2013).
- Due to the increased stress placed on a congenital bicuspid aortic valve, aortic stenosis often occurs at an earlier age (Cary & Pearce, 2013).

Significance of Pathophysiology

- Severity of aortic stenosis is equivalent to morbidity and mortality rates. The more severe the aortic stenosis, the higher risk of morbidity and mortality (Obasare et al., 2017).
- According to Treibel et al. (2018), aortic stenosis is a slow and progressive with major impacts on the left ventricle leading to left ventricular hypertrophy (LVH) and "afterload-induced heart failure."
- LVH develops as a compensatory mechanism, in effort to maintain cardiac output and stroke volume, from the increase in afterload that occurs as the aortic valve stenosis worsens in severity. As a result, LVH places additional strain on the heart causing a decrease in the elasticity of the heart muscle during contractions also causing a decrease in blood to the coronary arteries and "increased myocardial workload, oxygen consumption, and mortality" (Cary & Pearce, 2013).
- Decreased blood flow to the coronary arteries places patients at a higher risk for myocardial ischemia and infarction.
- Associated reliance on atrial kick to maintain stroke volume and cardiac output occurs and "loss or compromise of atrial kick as a results of atrial fibrillation, ventricular pacing, and/or intravascular fluid volume overload may precipitate pulmonary congestion, hypotension, and angina" (Cary & Pearce, 2013).

Diagnostic Studies

Study	Purpose
Doppler echocardiography	Estimation of severity of aortic stenosis, left ventricular size, and ejection fraction Estimation of pulmonary pressures, aortic valve gradient, aortic valve area Assessment of thickening of aortic valve leaflet, reduced leaflet motion, reduced valve opening
Cardiac catheterization	Assessment of coronary arteries to determine need for simultaneous coronary artery bypass surgery and aortic valve replacement Direct measurement of left ventricular and ascending aortic pressures to determine aortic valve pressure gradient Determination of left ventricular systolic pump function quantified by measuring left ventricular end-diastolic and end-systolic volumes, and ejection fraction
12-Lead electrocardiography	Evidence of left ventricular hypertrophy: Increased R-wave amplitude of the QRS complex in lead V ₆ , increased S-wave amplitude in lead V ₁ , ST-segment depression and T-wave inversion in leads facing the left ventricle: I, aVL, V ₅ , and V ₆
Chest radiography	Determination of heart size Detection of calcification in the aortic valve (lateral view) With heart failure, enlarged heart size from dilatation of left atrium and left ventricle, venous congestion, and pulmonary edema
Stress testing	Determination of the degree of exercise tolerance Distinguish between asymptomatic and symptomatic aortic stenosis
Brain natriuretic peptide	Determination of severity of increased left ventricular pressure and volume overload Distinction between cardiac and noncardiac dyspnea

^a Based on information from Kurtz and Otto,¹ Mookadam et al.,² and Bergler-Klein.^{3a}

Table 1. Diagnostic Studies in Aortic Stenosis (Cary & Pearce, 2013).

Clinical Manifestations

- Shortness of breath and/or dyspnea on exertion
- Decreased level of activity tolerance
- Fatigue
- Chest pain
- Dizziness/lightheadedness
- Decreased systolic blood pressure
- Pulsus paradoxus
- Heart murmur
- Heart failure

*Some patients may be asymptomatic up to the point of severe aortic stenosis *
(Cary & Pearce, 2013)

Treatment Options

- Surgical Aortic Valve Replacement (SAVR)
 - Standard treatment of choice for patient's with severe aortic stenosis able to tolerate the risks of general anesthesia and the post-operative rehabilitation process following a sternotomy.
 - Surgery involves open sternotomy and cardio-pulmonary bypass with the surgeon cutting out the stenotic aortic valve and permanently placing a new valve deemed the best fit for the patient based off of pre-operative testing.
 - Valve replacement may be done with a bi-leaflet mechanical valve (requires lifelong anticoagulation therapy) or an engineered tissue prosthetic valve made from bovine or porcine pericardial tissue (typically does not require anticoagulation therapy long-term). Valve type is selected based on the age, co-morbidities, and lifestyle of the patient (Dobson, Fairbairn, Plein, & Greenwood, 2015).
- Transcatheter Aortic Valve Replacement (TAVR)
 - Procedural alternative available for patients with severe, often-symptomatic, aortic stenosis deemed to be too high-risk for SAVR.
 - Criteria for TAVR include: severe and symptomatic aortic stenosis, deemed too high risk for SAVR by two physicians, and have a predicted greater than one year survival rate post-procedure (O'Sullivan & Wenaweser, 2017).
 - Performed by interventional cardiologists with a surgeon on stand-by. Using a femoral, subclavian, or apical approach, a new prosthetic tissue valve is deployed by a catheter over the existing stenotic valve while the patient's heart is still beating (Dobson et al., 2015).
- Aortic Valvuloplasty
 - Performed in the cardiac cath lab by an interventional cardiologist. Often done interim in high risk cases until physicians can plan for SAVR vs TAVR or palliatively (Elthaninoff et al., 2014).
- Medical Management
 - No true medication therapy is available to treat or cease the progression of aortic stenosis (Pawade, Newby, & Dweck, 2015).

Implications for Nursing Care

Asymptomatic Aortic Stenosis Management

- Strict blood pressure control- hypertension can place increased workload on a heart with LVH. Vasodilators such as ACE inhibitors or ARBs are the drugs of choice. Beta blockers are used cautiously unless needed for arrhythmia management
- Monitor for hypotension-can induce heart failure in patients with aortic stenosis
- Monitor for orthostatic hypotension
- Balanced ratio between rest and activity
- Encourage good oral care practices to avoid endocarditis on aortic valve
(Cary & Pearce, 2013)

Symptomatic Aortic Stenosis Management

- "For patients who do not have surgical repair, medical management of angina, exertional syncope, and signs and symptoms of heart failure becomes necessary" (Cary & Pearce, 2013).
- Use of a pulmonary artery (PA) catheter is necessary to titrate intravenous (IV) drugs helpful in maintaining adequate preload, afterload, and contractility.
- Diuretic therapy may be used to combat a heart failure exacerbation-monitoring adequate fluid volume to prevent excessive shifts of fluid that could affect a patient's hemodynamic status
(Cary & Pearce, 2013)

Conclusions

- Aortic stenosis is a growing problem as humans are living longer
- Can be potentially fatal if left untreated. "After onset, without surgical intervention, the mean life expectancy is 2 to 3 years" (Cary & Pearce, 2013).
- Diagnostic studies are widely available to help assess the presence and severity of aortic stenosis
- Management of symptoms associated with severe aortic stenosis is critical in an intensive care setting until surgical intervention can occur
- Surgical interventions are available with good outcomes for life expectancy if patients meet criteria
- In an acute care setting, "goals in patients' daily plan of care include balancing rest and activity to maintain oxygen supply and demand and maintaining heart rate, blood pressure, temperature, and fluid volume status within reference ranges" (Cary & Pearce, 2013).

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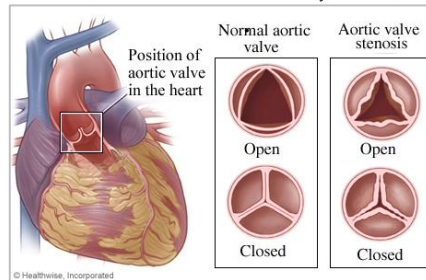


Figure 1. Comparison of normal aortic valve vs. stenotic aortic valve (University of Michigan Frankel Cardiovascular Center, 2018).



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