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Aortic Stenosis

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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 mostly asymptomatic until later stages of the disease, which delays treatment and intervention 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 undergo valve replacement in a low risk fashion, prolonging life and improving quality of life. AS causes an increase in afterload, decreasing outflow of blood from the left ventricle. This process can lead to an array of complications including left sided heart failure. The underlying pathophysiologic process of AS will be outlined in this presentation.

Facts about AS

- 1.5 million suffer from AS in the U.S, with calcific AS being the most common
- Severe AS has a 5 year survival rate of 3% without surgical intervention, which is lower than many metastatic cancers
- AS is usually seen in those greater than 75 years of age
- Only about half of those who suffer from severe AS are symptomatic.

(Chen, 2014)

Grading AS

- Graded as mild, moderate or severe (Kurtz & Otto, 2010).
- Three parameters are used in diagnosis, which are assessed through Doppler Echocardiogram (Kurtz & Ottp, 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. 63-34).
- AS is considered hemodynamically significant when the aortic valve area is less than 1.0 cm^2.
- Otto (2010) defines severe AS as, "the degree of valve obstruction at which symptoms might be caused by valve obstruction" (p. 280).
- Grading specifications based on findings from echocardiogram are outlined below (Cary & Pearce, 2013).

Grade	Aortic jet velocity, m/s	Mean aortic valve pressure gradient, mm Hg	Aortic valve area, cm^2
Mild	<3.0	<25	1.5
Moderate	3-4	25-40	1.0-1.5
Severe	>4	>40	<1.0

[a] Based on information from Bonow et al.[1]

Subjective Findings

- Shortness of breath (SOB) upon exertion or at rest with severe AS
  - Secondary to left ventricular heart failure and subsequent pulmonary congestion
- Angina
  - May be related to increased workload on the left ventricle and a subsequent increase in oxygen consumption
  - Typically exacerbated by activity and relieved with rest
- Syncope
  - Likely 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
  - Likely a sensation resulting from atrial fibrillation or sinus tachycardia
- Increased fatigability and decline in activity

(Cary & Pearce, 2013).

Objective Findings

- Systolic ejection murmur upon auscultation of heart tones, best heard between 2nd intercostal space on right sternal 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
- Tachycardia, or other cardiac arrhythmias such as atrial fibrillation (Afib) or Atrioventricular blocks may be present
- Hypotension
- Decreased oxygen saturation
- Other Hemodynamic changes are outlined in the table below.

(Cary & Pearce, 2013).

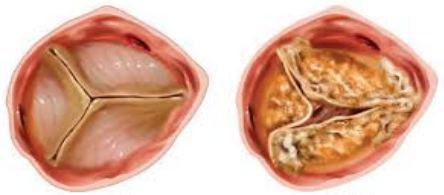
Parameter	Definition		
Stroke volume (SV)	Volume of blood ejected from the ventricle with each contraction		
Cardiac output (CO)	Volume of blood ejected from the heart per minute CO=heart rate (per minute) x SV		
Preload	Volume of blood in the ventricle at end diastole (producing a stretch of ventricular muscle cells)		
Afterload	Resistance the heart must overcome to eject blood from the ventricle		
Systemic vascular resistance (SVR)	Resistance to blood flow in all systemic vasculature		
	Reflects	Normal range	Effects of moderate to severe aortic stenosis
Right atrial pressure	Right ventricular preload	2-7 mm Hg	Increases
Pulmonary artery (PA) pressure	Pressures in the pulmonary vasculature	Systolic 15-30 mm Hg Diastolic 4-12 mm Hg	Increases when PA systolic pressure >80 mm Hg (severe pulmonary hypertension)
Pulmonary artery occlusion pressure	Mean left atrial pressure (indirect reflection of LV preload)	2-12 mm Hg	May increase
Left ventricular pressure (LVP)	LV afterload (systolic) LV preload (diastolic)	Systolic 90-140 mm Hg Diastolic 5-12 mm Hg	Increases
Aortic pressure (AP)	SVR and preload	Systolic 90-140 mm Hg Diastolic 60-90 mm Hg	Decreased preload causes decreases in LVP and AP, increased SVR Increased preload causes increased LVP to maintain AP
Systemic vascular resistance (SVR)	LV afterload	700-1600 dynes · sec · cm^{-5}	Increases
Pulmonary vascular resistance	Resistance to blood flow in pulmonary vasculature	20-130 dynes · sec · cm^{-5}	Increases
Cardiac output/reading	Volume of blood ejected from the heart per minute	5-8 L/min	Decreases

[a] Based on information from Otto and Bonow.[1]

Underlying Pathophysiologic Process

- Multiple pathological processes are thought to play a role in the development of aortic stenosis
- Biomechanical forces
  - Create an increase in stretch and shear on the valve cusps, producing inflammation and extracellular remodeling
- Lipid accumulation
  - Oxidized LDL engulfed by macrophages create 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 myofibroblasts, 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 & Anseth, 2013).



Normal Valve

Stenotic Valve

Significance of Pathophysiology

- Pathophysiology of AS can lead to decompensation 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 angina, syncope, and cardiac arrest (Towler, 2013).
- Understanding of pathophysiological process of AS is key to medical management
  - Possible use of anti-inflammatory drugs to decrease calcification and remodeling (Gould, Simmons & Anseth, 2013).
  - Anti-lipid agents to decrease cholesterol and subsequent reduction in oxidized LDL (Gould, Simmons & Anseth, 2013).
  - Vasodilators, diuretics, and possibly beta blockers help to reduce left ventricular workload and decrease shear and stress on valvular tissue (Cary & Pearce, 2013).
    - Special consideration with beta blockers, as to not potentiate systolic heart failure.
- Surgical replacement of aortic valve gold standard treatment (open chest or TAVR). Poor prognosis for medically managed patients (Towler, 2013).

Implications for Nursing Care

- The nurse caring for the patient must be highly aware of the pathophysiologic process of AS, as well as treatment and possible complications (Cary & Pearce, 2013).
- Deep understanding of strict range of preload and afterload to maintain adequate cardiac output (Gould, Simmons & Anseth, 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 vein distention, cyanotic or dusky appearance, hemodynamics, heart rate and rhythm, and presence of edema are crucial in the identification and differentiation of possible complications (Cary & Pearce, 2013).
- Clinical awareness of decompensation of patient including decreased oxygen saturations, shortness of breath, angina, hypotension, cardiac arrhythmias, and chest pain is crucial (Gould, Simmons & Anseth, 2013).
- Angina unrelieved with oxygen, rest, and nitrate therapy or associated with hypotension 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 worsening 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 (Chen, 2014). Calcific Aortic Stenosis is the most common type, and multiple pathophysiologic processes contribute to the development of this disease. Mechanical stress on the valve over time can cause inflammation and remodeling of the tissue, rendering the valve dysfunctional. Oxidized lipids that accumulate 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 combined can cause differentiation of VIC's into myofibroblasts, replacing normal tissue with dense collagen and bone leaving the valve stiff and non-compliant (Gould, Simmons & Anseth, 2013). Understanding the underlying pathophysiology 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. Thorough assessment, knowing signs and symptoms of AS, as well as being able to identify the decompensating 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 (Towler, 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.

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