Challenges in the Medical Management of Patients with Aortic Stenosis

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Objectives

• Describe the pathophysiology of AS
• Identify clinical manifestations of AS
• Discuss medical and nursing management of nonsurgical patients
Introduction

• Aortic stenosis (AS)
  - Narrowing of aortic valve orifice
  - → Obstruction of left ventricular outflow
• Calcific aortic valve disease (CAVD)
  - Affects > 4% North Americans and Europeans
  - Increasing in prevalence
Normal Valve Function

• Essential to cardiovascular and cardiopulmonary physiology
• Heart valves ensure forward progression of blood through the heart
• Open & close in response to pressure changes during systole and diastole
Normal Heart & Valve Function

- Open tricuspid & mitral valves in early/mid diastole
- Closed tricuspid & mitral valves in early systole
- Open pulmonic and aortic valves in mid systole
- Closed pulmonic and aortic valves in late systole

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Systolic Contraction & Twist
Aortic Valve A & P

- 3 cup-shaped leaflets
  - Top edge (free margin)
  - Base
- Annulus connects aortic valve to the fibrous skeleton of the heart
Fibrous Skeleton

- Dense fibrous connective tissue
Aortic Valve Anatomy & Physiology

- Commissures join leaflets edge to edge
- Penetrate aortic wall
- Absorb stress of systole and diastole

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Aortic Valve A & P

- Sinus of Valsalva
- Aortic wall bulges outward
- Creates space behind leaflets
3 Sinuses of Valsalva

- Bulge creates space
  - Prevent obstruction to coronary arteries during systole
  - Provide space for blood to pool during diastole for coronary artery filling

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Sinus of Valsalva

- Blood flow outlet narrows
- Blood forced between the Sinus of Valsalva and open cusps, filling coronary arteries
- Backward curling
- Free margins meet
Three Sinuses of Valsalva

- Late systole, backward blood flow fills the cusps outward in
- End systole, cusp’s free margins abut ensuring perfect valve closure
Layers of Leaflet

- **Fibrosa**
- **Ventricularis**
- **Spongiosa**
Aortic valve leaflet A&P

- **Fibrosa** – collagen: distributes pressure load

- **Spongiosa** - glycosaminoglycans, proteoglycans: cushion, minimize friction

- **Ventricularis** - elastic fibers: maintains shape
Aortic Valve Leaflet A&P

- **Valvular interstitial cells (VICS):**
  - Maintain structure & function
  - Inhibit angiogenesis
  - Repair cellular damage

- **Valvular extracellular matrix (VECM):**
  - Collagen fibers
  - Elastin fibers
  - Glycosaminoglycans, proteoglycans
Aortic Stenosis

• Progression from sclerosis to stenosis
  - Sclerosis – mild valve thickening and/or calcification without obstruction
  - Stenosis – increasing obstruction of blood flow and progression of:
    • Leaflet thickening
    • Calcium nodule formation
    • Angiogenesis
  – 10% advance from sclerosis to stenosis
Causes of Aortic Stenosis

- Valve calcification of:
  - Tri-leaflet AV
    - Most common cause of AS
Bicuspid Aortic Valve

• 1-2% of adults More likely to develop AS
• Stenosis occurs earlier
  - 50s to 60s bicuspid
  - 70s to 80s tricuspid
Post-inflammatory Causes of Aortic Stenosis

• Rheumatic heart disease
  – Untreated pharyngeal infections
  – Rare in developed countries
  – Most common cause world-wide

• Prior exposure to radiation
  - 15-20 yrs post exposure
Aortic Valve Calcification

• CAVD is an active cellular biological process
• Not an inevitable consequence of aging
• VICS no longer repair injuries to VECM
  – Alterations of cells within the layers of the AV
• Exact cause is still unclear
Pathophysiology of AS

• Chronic resistance to LV ejection → concentric LV hypertrophy and myocardial fibrosis

• Stronger LV systolic contraction needed to
  - Maintain adequate stroke volume and cardiac output
  - EF is maintained
Concentric vs. Eccentric
Consequences of Concentric Hypertrophy

- Decreased
  - Myocardial elasticity
  - Coronary blood flow
- Increased
  - Myocardial workload
  - Myocardial oxygen consumption
  - Mortality
Consequences of Concentric Hypertrophy

• Increased
  - Diastolic pressure
  - Delayed LV untwisting (relaxation)

• Optimal stroke volume and cardiac output increasingly dependent upon a forceful atrial contraction (atrial kick)
Consequences of Concentric Hypertrophy

• Mitral valve regurgitation
  - Increased LV pressure puts a strain on the mitral valve
  - Increase pressure in the lungs
    • Pulmonary venous hypertension
    • Reactive vasoconstriction of the pulmonary vasculature
2014 AHA/ACC Guideline for the Management of Patients with Valvular Heart Disease

• Stages A-D
Grading of AS

• Aortic jet velocity
• Mean aortic valve pressure gradient
• Aortic valve area
Pressure Gradient

- Peak LV and aortic pressure tracings
- LV pressure higher than aortic pressure
- Grading table uses mean pressure gradient

Wikipedia, Aortic Valve Stenosis
Jet Velocity

- Low velocity (slower) blood flow in the LV outflow tract speeds up as it moves through the narrow, stiffened AV orifice.

Wickimedia Commons, accessed Oct 2014
Clinical Manifestations of AS

• Initial manifestations
  - Decreased exercise tolerance
    • Dyspnea on exertion
    • Exertional dizziness
    • Lightheadedness with exertion

• Late manifestations
  - Angina
  - Syncope
  - Heart failure
Clinical Manifestations of AS

- Jugular vein distension
- Pulmonary rales
- Carotid pulse abnormalities
- Systolic ejection murmur
Carotid Pulse

• *Gentle, careful* palpation
• Pulsus tardus
  - Slowly increasing carotid upstroke
  - Takes longer to reach peak pressure
• Weaker pulse amplitude
Carotid Pulse

• Indication of:
  - Resistance to AV opening
  - Subsequent delay in LV ejection
  - Decreasing volume

• Indications may be masked in the elderly d/t age related changes in arterial compliance and stiffness
Systolic Ejection Murmur

• Crescendo-decrecendo
  - 2\textsuperscript{nd} intercostal space right sternal boarder with bell of stethoscope
• May radiate to carotids
• In elderly, may radiate to the apex
Medical Management of Asymptomatic Patients

- Decrease cardiovascular risk factors
- Monitoring and education
- Medication therapy
Decrease Cardiovascular Risk Factors

- HTN
- Diabetes
- Smoking tobacco
- High cholesterol
- Overweight
- Lack of exercise
Monitoring and Education

- Monitoring
  - F/U visits, echocardiograms

- Education
  - Disease progression
  - Change in exercise tolerance
  - Physical activity
Medication Therapy

• There is currently no known medical therapy to
  – Prevent CAVD
  – Delay the progression of AS
Medication Therapy

• Prophylactic antibiotics
  – Rheumatic AS only

• Treat HTN according to standard GDMT
  – $B$-blockers historically considered unsafe

• Statins??
Guideline Directed Medical Therapy JNC: 8

- ACE Inhibitors (or ARBs)
  - Previously contraindicated
- Beta blockers
- Calcium channel blockers
- Thiazide-type diuretics
Management of Symptomatic Patients

• Surgical options
  - Aortic Valve Replacement (AVR)
  - Transaortic valve replacement (TAVR)
  - Balloon Aortic Valvuloplasty (BAV)
Management of Symptomatic Patients

• Surgical repair is the **only** effective treatment for symptomatic AS
• Mean life expectancy 2-3 years without surgical intervention
• Treat co-morbidities
• Treat symptoms
• Maintain optimal hemodynamics
Management of Symptomatic Patients

- Angina
- Syncope
- Pulmonary congestion
- Acute pulmonary edema
Case Study

• 70yo female w/severe AS and CAD
• NPO after MN CABG/AVR next afternoon
• Developed chest pain at rest
• No PRN order for NTG SL...
  – What do you need to know before proceeding?
  – What is your intervention?
Management of Symptomatic Patients

- Angina
  - Bedrest
  - Oxygen therapy
  - Vasodilators (nitrates)
  - Beta blockers

- Syncope
  - Treat arrhythmia
Case Study, continued

• B/P 108/65, usual B/P 130/70
  – Chest pain unrelieved w/rest and O₂
• IV Nitroglycerin 10mcg/min
  – B/P ↓ 90/60
  – Chest pain unrelieved
• Normal Saline 150cc/hr
• Transferred to ICU
Management of Symptomatic Patients

- **Pulmonary congestion**
  - Digitalis
  - Diuretics
  - ACE Inhibitors or ARB

- **Acute pulmonary edema**
  - Vasodilator therapy
  - Intra-aortic balloon pump (IABP)
Nursing Considerations

• Thorough grasp of the tenuous balance between the narrow range of preload & afterload necessary to maintain forward blood flow & adequate CO

• Extremely sensitive to changes in preload
  – High preload → pulmonary congestion
  – Low preload → low output failure

• Dependent on a strong atrial contraction
Nursing Considerations

• Consider hemodynamics as they relate to signs & symptoms of AS
• Consider hemodynamic effects of medications, treatments, plan of care
• Avoid systemic hypotension
  – Myocardial ischemia
  – Reduced contractility
  – Worsening hypotension
  – Worsening coronary perfusion
Goals in Daily Plan of Care

• Balance rest & activity
  – Maintain HR, B/P, temperature and fluid volume status
• Monitor for indicators of decompensation
  – Hypoxia, arrhythmias, B/P changes, SOB, chest pain, prolonged NPO status
• Identify decompensation *early* to prevent deterioration in clinical status
Patient Education

• Asymptomatic symptom recognition
  – Initial symptoms
    • ↓ exercise tolerance d/t exertional dyspnea or fatigue
    • exertional dizziness
    • exertional lightheadedness
  – Advanced symptoms
    • angina, syncope, HF
Patient Education

• Symptomatic symptom recognition
  – worsening signs and symptoms with prompt reporting
  – balance rest/activity to avoid symptoms

• Impact of medication adherence on cardiac function
Patient Education

• Treatment is improved when patients are educated and involved in
  – Daily weights
  – Signs of decompensation
  – When to call the physician or LIP
  – Changes in elevation with travel
Conclusion

• Symptomatic AS cannot be corrected without surgery
• Medical management of patients with AS, particularly severe symptomatic AS, is challenging
• Nurses must be astute about the tenuous hemodynamic balance of patients with severe AS
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