NURSING 210
ADVANCED CARDIAC UNIT 2
2.3, 2.4, 2.5

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VASCULAR DISORDERS

- Arterial or Venous
- Ischemia or Excessive accumulation of waste and fluid
PREVENTION

The First Treatment of Choice for Peripheral Arterial Disease
PERIPHERAL ARTERIAL DISEASE (PAD)

- Pain
- Pallor
- Pulselessness
- Paresthesia
- Paralysis
- Poikilothermia

Arterial Occlusions
6 “P’s”
Peripheral Arterial Disease (PAD)

Location of arterial lesions
Collateral Circulation
TREATMENT

- Angioplasty (PTA)
- Laser-Assisted Angioplasty
- Peripheral Atherectomy
- Intravascular Stents
- Amputation
- Bypass
Peripheral Arterial Disease (PAD)

Bypass
Peripheral Arterial Disease Post-op

- Nursing assessments for:
  - Pulse
  - Temperature
  - Color
  - Graft Patency
ANEURYSMS

- Causes
- Locations
- Signs and Symptoms
Aneurysms

50-75% change in size of the vessel is concerning
A

Artery

Fusiform area

B

Sacculated area

C

Torn intima

Blood flow

D

Ruptured area

Blood flow
Dissecting Aneurysm
VENOUS DISORDERS

Acute & Chronic
THROMBUS FORMATION

1. Venous stasis
2. Hypercoagulability
3. Injury
Thrombophlebitis

- **Risk factors**
  - Bed Rest
  - General Surgery
  - Leg Trauma
  - Previous venous insufficiency
  - Obesity
  - Oral Contraceptives
  - Malignancy
Venous Thrombosis
Venous Thrombosis

Veins recanalize
Venous Thrombosis

Veins may recanalize but valves are destroyed

Chronic venous insufficiency

Increased Venous pressure

Varicosities

Venous ulcer
Venous Thrombosis

Venous recanalize

Veins may recanalize but valves are destroyed

- Chronic venous insufficiency
- Increased Venous pressure
- Varicosities
- Venous ulcer

Many veins may become obstructed

- Increased distal venous pressure
- Fluid stasis
- Edema
- Venous gangrene
Venous Thrombosis

Veins may recanalize but valves are destroyed

Chronic venous insufficiency
Increased Venous pressure
Varicosities
Venous ulcer

Many veins may become obstructed

Increased distal venous pressure
Fluid stasis
Edema
Venous gangrene

Thrombi may cause PE
THROMBOPHLEBITIS

- Risk factors
- Problems
- Prevention

1. Venous stasis
2. Hypercoagulability
3. Injury
TREATMENT

- Medical
- Surgical
Chronic Venous Insufficiency

- Patient Education
- Prevention
- Decrease Risk Factors
  - Daily hygiene
  - Foot care
  - Elevation
  - Avoid Standing
  - Inspection
VENOUS STASIS ULCERATION

- Treatment:
  - Patient education
  - Prevention & inspection
  - Dressings
  - Debridement
  - Surgery
COMPRESSIVE STOCKINGS

- Proper Measurement
- Wear Proper size with appropriate pressure gradient
- Apply before getting out of bed
- Put on properly
MEDICATIONS

- Vasodilators
- Antihemorrhheologic
- Anticoagulants
- Thrombolytics
RAYNAUDS DISEASE

- PVD- vascular dysfunction
Taber’s defines heart failure as...

“The inability of the heart to circulate blood effectively enough to meet the body's metabolic needs”.
SYMPTOMS OF HEART FAILURE

- Shortness of breath (dyspnea)
- Fatigue
- Edema – associated with fluid overload
- Lightheadedness
- Hypoxia
- Orthopnea
- Cognitive changes
RESPONSE
COMPENSATION

Sympathetic Nervous System

Increase Heart Rate
Increase force of contraction

Increase Vascular Tone
Peripheral Vasoconstriction

Kidneys ??
TRUE OR FALSE?

Increased sympathetic stimulation will result in increased oxygen Demand.
PULMONARY EDEMA

Normal

Increase Hydrostatic Pressure

Lymphatic flow increases
LEFT-SIDED HEART FAILURE

- CAD (Coronary Artery Disease)
- Hypertension
- Valvular Disease
- Cardiomyopathy
- Myocardial Infarction
RIGHT-SIDED HEART FAILURE

- Left-Sided Failure
- Cor Pulmonale
- Chronic Airway Limitations (CAL) (COPD)
- MI
RIGHT SIDED FAILURE SIGNS AND SYMPTOMS

- Jugular Venous Distention (JVD)
- Enlarged Liver (Hepatomegaly)
- Ascites
- Anorexia
- Dependent edema
- Polyuria at night
HEART FAILURE TREATMENT

\[ \text{CO} = \text{HR} \times \text{SV} \]

- Heart Rate
- Preload
- Afterload
- Cardiac muscle contractility
Medication use in congestive heart failure

- Diuretics: 38.8%
- ACE inhibitors: 19.4%
- Positive inotropes agents: 14.0%
- Beta blockers: 9.9%
- K+ supplements: 5.0%
- Other: 12.9%

ACE = angiotensin-converting enzyme.
RHEUMATIC HEART DISEASE

Results in inflammation in all layers of the heart muscle.

- Impaired contraction
- Thickening of pericardium
- Valvular damage
Rheumatic heart disease

- Prosthetic cardiac valve
- Valvular dysfunction

Congenital heart disease

Degenerative heart disease

Damage to endothelium

Introduction of pathogen

Bacteremia

Platelet-fibrin-bacteria mass forms on valve = Vegetation

- Embolization
- Vegetative growth
- Scarring, perforation of leaflets
Infective Endocarditis

- Bacteria, Fungi, Chlamydiases, Rickettsiae
- Results in valvular damage
- Infiltration
- Sepsis
- Hemodynamic compromise
- Heart block or failure
MITRAL REGURGITATION

A  Cusp  Orifice

Normal valve (open)

B  Fused cusps  Orifice

Normal valve (closed)

C

Stenosed valve (open)

D

Stenosed valve (closed)

Cephalic vein

Stenosed mitral valve

Mitral valve does not close completely
GRADING OF HEART MURMURS

I  Faint: heard after being “tuned in”
II Faint: usually heard
III Moderately loud
IV loud
V Very Loud: need stethoscope
VI Very loud: heard w/o stethoscope
MANAGEMENT OF VALVULAR HEART DISEASE

- Accurate diagnosis of etiology & severity
- LV systolic function is a primary factor in determining optimal therapy
- Prevent endocarditis
- Optimization of preload & afterload
- Treatment/prevention of complications
- Surgery
CARDIOMYOPATHIES

- Dilated
- Restrictive
- Hypertrophic
Normal Heart

Enlarged Heart
Cardiac Tamponade

Signs and Symptoms

- Cough
- Muffled heart sounds
- Prominent neck veins
- Pulses Paradoxus
To ECG

Sternum
Pericardium
Xyphoid

Abdominal cavity
Heart

Three-way stopcock

Aspiration syringe with 1% xylocaine
CONGENITAL HEART DEFECTS
CONGENITAL HEART DEFECTS

- VSD
- ASD
- PDA
- Coarctation of the aorta
- Aortic stenosis
- Pulmonic stenosis
- Tetralogy of Fallot
ANGINA PECTORIS

- Stable
- Unstable (Acute Coronary Syndromes)
  - Preinfarction
  - Variant (Prinzmetal)
  - Crescendo
TREATMENT PLAN

- **MEDICATIONS**
  - Nitro-Dur Patch
  - Propranolol
  - ASA

- **PATIENT EDUCATION**
  - Exercise
  - Follow up plans
  - What is an emergency
Chest Pain/Discomfort

“The evaluation of chest pain/discomfort is to diagnose or exclude those entities associated with the greatest risk of death”
Quality - dull/squeezing
Region - radiation
  - changing
  - right arm/left arm
Severity/Setting
  - rest vs exertion
  - after meals
  - scale of pain
Q-R-S-T-A-A-A

- **Time**
  - sudden/gradual onset

- **Alleviators**
  - Position

- **Aggravators**
  - food/position/exertion/people
  - constant/episodic

- **Associated Symptoms**
  - SOB, cough, temp, nausea, diarrhea
Angina Pectoris
Substernal or retrosternal pain spreading across chest. May radiate to inside of arm, neck, or jaws.

Myocardial Infarction
Substernal pain or pain over precordium. May spread widely throughout chest. Painful disability of shoulders and hands may be present.

Pericarditis
Substernal pain or pain to the left of sternum. May be felt in epigastrium and may be referred to neck, arms, and back.

Pain of Pulmonary Origin
Pain arises from inferior portion of pleura. May be referred to costal margins or upper abdomen. Patient may be able to localize the pain.

Esophageal Pain
(Hiatus Hernia, Reflux Esophagitis)
Substernal pain. May be projected around chest to shoulders.

Anxiety
Pain over left chest. May be variable. Does not radiate. Assess for hyperventilation, sighing respiration, palpitations. Patient may complain of numbness and tingling of hands and mouth.
STAGES OF MI
Effects of Myocardial Ischemia, Injury and Infarction on ECG

- **Zone of ischemia**
- **Zone of injury**
- **Zone of infarction**

**Myocardial ischemia** causes ST segment depression with or without T wave inversion as result of altered repolarization.

**Myocardial injury** causes ST segment elevation with or without loss of R wave.

**Myocardial infarction** causes deep Q waves as result of absence of depolarization current from dead tissue and receding currents from opposite side of heart.
Transmural Infarction

Before coronary occlusion

Onset and first several hours

First day

Heart muscle normal

Subendocardial injury and myocardial ischemia. No cell death (infarction) yet

Ischemia and injury extend to epicardial surface. Subendocardial muscle dying in area of most severe injury

R wave normal or nearly normal

T wave peaked

R wave amplitude diminishing

Normal ECG

ST segment elevated

ST elevation more marked
Remodeling

--- First and second days ---

Transmural infarction nearly complete. Some ischemia and injury may be present at borders

- R wave gone or nearly gone
- T wave inversion beginning
- Significant Q wave
- ST elevation may decrease

--- After 2 or 3 days ---

Transmural infarction complete

- No R wave
- Marked Q wave
- ST may be at baseline

--- Weeks or months ---

Infarcted tissue replaced by fibrous scar, sometimes bulging (ventricular aneurysm)

- Some R wave may return
- Deep T wave inversion
- Significant Q wave usually persists
- ST elevation may persist if aneurysm develops

Key:
- Myocardial ischemia
- Myocardial injury
- Myocardial death (infarction)
- Fibrosis
EKG

Your first Response?
Bp 80/42
CARDIAC SHOCK
CATH LAB

PTCA
Percutaneous Transluminal Coronary Angioplasty
C. Coronal Artery Bypass Grafting
A: Atheromatous lesion
B: Cutter positioned against lesion
C: Balloon inflated
D: Nose cone
E: Cutter is advanced, tissue is shaved off
F: Tissue is deposited in nose cone
G: Smooth lesion after DCA
BYPASS SURGERY

- Saphenous Vein - Greater or lesser
- Internal Mammary Artery
- Radial Artery (2nd or 3rd CABG)
- Cryopreserved Saphenous Veins
THE END