

Pt Management & Problems of the CV System - Part 1 Cheat Sheet by Maria K (mkravatz) via cheatography.com/71404/cs/18105/

Physiological Changes with Age

Cardiac Valves: stiffen, calcify, degenerate = expect murmurs ("swish")

Conduction System: coronary arteries get poor blood = necrosis, arrhythmias; lose pacemaker cells, lose conduction, fat in SA node, coming from ectopic muscle

Left Ventricle: atrophies, stiffens, enlarges, becomes less distensible, dec. SV & CO, dec. EF, most noticeable w/ physical activity

Aorta & Large Arteries: thicken, stiffen, less distensible = pumps harder (inc. HR) & inc. systemic vascular resistance

Baroreceptors: located in carotid arteries; help regulate BP; less sensitive w/ age; most noticeable w/ position changes

Framingham Heart Study (1948): Landmark study done in Framingham, MA looking at cardiac risk and what we can do--modifiable & non-modif. risk factors

- 5,209 subjects (mean age 47) & offsprings
- Established the CV risk profile!

Assessment: Psychosocial

Ask about...

Occupation?

Insurance?

Support system?

Pets at home?

Hobbies that may help?

* Patients won't get better if they're stressed!

Assessment: Modifiable & Non-Modif. Risk

MODIFIABLE RISK FACTORS

Age: symptoms start by 40yo, unlikely to survive MI if <30yo b/c collateral circulation

Ethnicity: more prevalent in non-Hispanics, death rate higher in African Amer. (HTN)

Heredity: HTN, inc. lipids, DM, obesity

Gender: men > women until menopause, childbearing women have 25% chance, women >40yo & after menopause > men (r/t heart size & collateral circulation)

NON-MODIFIABLE RISK FACTORS

BP: biggest problem = insidious - take meds if needed

HLD: goals - **total cholesterol** < 200; **HDL** > 50, **LDL** < 70 - take meds if needed

Smoking: temp of vape = hyperplasia, asthma-like symptoms; causes 21% of CVD deaths; carcinogenic; inc. epic & norepi = heart works harder, vasoconstriction & dec. circulation, C monoxide = inc. vessel perm.

Physical Inactivity: "new smoking", exercise inc. collateral circulation

Obesity: extra burden on heart

Personal Factors: stress, psych. response

Collateral circulation: inc. angiogenesis; adding vessels to supply cardiac circulation

Obese: BMI >30 / Morbid Obese: BMI >45 Super Morbid Obese: BMI >65

Assessment: Subjective & Objective Data

SUBJECTIVE DATA (History of Symptoms)

Chest Pain: (activity w/) onset? location? severity? type? precipitating factors? other Sx? may c/o nausea, indigestion

- Causes: cardiac (myocardial), pulm., m/s

Dyspnea or SOB: often assoc. w/ left side heart pain, dec. perfusion, orthopnic

Palpitations: usually PAC, c/o rapid HR = dec. EF & CO (caffeine)

Fatigue: mild to severe, may attribute to getting older (compare to daily activity)

 $\textbf{Extremity Pain} \colon \text{arm (may be R), jaw}$

Syncope: if issue w/ CO

Weight Gain: fluid, daily wt, anasarca

OBJECTIVE DATA

General Appearance: AAOx3?, posture - Restlessness assoc. w/ change in O2

Vital Signs: BP? HTN < 130/80, check BP bilat., may see a paradoxical change in BP

Heart Sounds: S1, S2; may hear S3 & S4, murmurs, clicks

Cyanosis & JVD: pallor; JVD = R-sided HF (cor pulmonale), seen w/ OSA; = give Lasix

Subjective Data: Ask for chief complaint (usually CP), PMH, current health

- Dehydrated = lose H20 & electrolytes

Objective Data:

Pulse Pressure: SBP - DBP; normally 30-40
- Closer (~20): r/t vasc. resistance = dec. CO &

- Widened (~40): r/t slow HR, atherosclerosis,

inc. w/ age



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Diagnostic Studies

SERUM CARDIAC ENZYMES (SERUM MARKERS) OR CARDIAC BIOMARKERS

Troponin: GOLD STANDARD OF CP; appears 2-4 hr after damage to myocardial muscle, inc. further depending on damage

CK-MB: r/t cardiac muscle; detected 2-4 hr after damage, elevated 72 hr max

CK-MM: r/t skeletal muscle

CK-BB: r/t brain tissue

Myoglobin: byproduct of muscle breakdown, appears in 2-4 hr, then dec.; affects kidneys; rhabdomyolysis

BNP: r/t stretch of heart; correlates + w/ HF; secreted by ventricles r/t stress

CRP: non-specific inflammatory marker; correlates + w/ atherosclerosis; good for determining severity of disease process

Myeloperoxidase: leukocyte enzyme r/t plaque instability and enzyme production

Ischemia Modified Albumin: circulating albumin touches ischemic tissues

Homocysteine: get from eating meat (in amino acids), linked to disease development

Serum Lipids: correlates + w/ intravascular plaques

COAGULATION STUDIES

Unfractionated Heparin: if elevated, give protamine sulfate

APTT

 $\mbox{\bf PT/INR}\!:$ if elevated, give vitamin K

Why do coagulation studies? To know if pt is anti-coagulated in case of procedure

Antidotes

- * Coumadin = vitamin K
- * Many newer generation anti-coagulants don't have antidotes! = Give cryoprecipitate

More Diagnostic Studies	
OTHER	
EKG	shows issues r/t heart rhythm; 12-lead EKG w/ age 40yo+
Telemetry	continuously monitoring EKG, ambulatory
Holter Monitor	ambulatory type, pt takes it home & writes down what they do to compare it to the rhythm
X-Ray	shows enlargement, fluid; pulmonary edema r/t CHF?
STRESS, NUCLEAR, & ULTRASOUND TESTS	
Exercise Stress Test	look at BP and HR w/ inc. exercise and inc. myocardial O2 demand
Nuclear Perfusion Imaging	stress test & blood flow through the heart
Echocardi ogram	shows wall movement, overall ventilatory performance; can tell how badly heart was damaged

Serum Electrolytes & the Heart		
K	biggest electrolyte r/t heart	
	Hypokalemia: inc. electrical instability, a fib, digoxin toxicity	
	Hyperkalemia: P-wave issues, bradycardia, asystole, ventricle issues; give Kayexalate, insulin (IVP 10 units) + D50; give Lasix	
Na	r/t CHF	
	Hyponatremia	
	Hypernatremia	
Ca	Hypocalcemia	
	Hypercalcemia	
Mg	Hypomagnesemia	
	Hypermagnesemia	
Р	Hypophosphatemia	
	Hyperphosphatemia	
Insu	Insulin: K follows glucose into cells	

C

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TTE

TEE

2-D

3-D (better)

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