Unstable Angina: New onset angina effecting activities, Resting angina for 20 minutes+ Increasing angina (duration or intensity)

TIMI SCORING
A THREAT

A ge 65+
T wo + anginal episodes in last 24hrs
H istory of 50% stenosis of coronaries
R isk factors: 3+
E KG, S-T deviation
A spirin taken in last week
T roponins elevated

ANTI-PLATELET AGENTS

Blocks formation of Thromboxane A2 via prostaglandin synthetase block
>Aspirin
ADP receptor blockers (block fibrinogen binding to platelet receptor)
>Clopidogrel (Plavix)
Glycoprotein Ia - IIIb inhibitors (block fibrinogen binding to platelet receptor)
>tirofiban (Aggrastat)
>eptifibatide (Integrilin)

In the first four months after stent placement, patients are given ASA, a beta blocker, and clopidogrel and then the clopidogrel is discontinued. Patients with DM are also placed on an ACEI.

Typical angina CP: all of the following: substernal CP, CP aggravated by exercise or emotion, CP relieved by rest or NTG. Atypical CP has 2 of these 3. Atypical CP in men age 30-39 is 34% probability of CAD and in women age 50-59 is 31%.

Stress testing

Test	Sensitivity	Specificity
Exercise ECG	68	77
Nuclear SPECT perfusion	88	77
Stress echo	76	88
PET Stress	91	82

CHRONIC STABLE ANGINA
All patients receive: asa, statin, beta blocker, long acting nitrate.
If still symptomatic, add a CCB.
If still symptomatic, add renolazine.
If still symptomatic, cath and revascularize if possible.

CAD: if lo EF, DM, or HTN, add an ACE inhibitor.

THERAPY for CAD following acute coronary event

	ASA	BB	nitro	heparin	clopidogrel	GP IIb/IIa inhib	Study
NSTEMI OR UA: timi 0-2	X	X	x	Con-sider	x		Predischarge stress test*
NSTEMI or UA: timi 3+	x	x	X	x	consider	x	Early cath
STEMI	x	x	x		x		Cath

*If EF < 40% or abnormal stress test, then cath.

In the setting of MI: If there is hypotension, JVD & clear lung fields >> RV infarction. Confirm this with right precordial leads.

For CHF, everyone gets a BB and an ACEI (or ARB).

For CHF, indications for HYDRALAZINE and ISOSORBIDE DI-NITRATE are:
*In patients unable to take and ACEI or an ARB.
***In black patients, with NYHA III or IV.**
*In all patients with continued symptoms.

Criteria for aldosterone antagonists: NY Heart 3 or 4: spironolactone. NY Heart 2 with $\leq$ 35% EF.

Criteria for cardiac resynchronization: EF $\leq$ 35%, ventricular dyssynchrony on ECG, sinus rhythm, and NY Heart III or IV while on optimal medical therapy.

HOCUM. AD in 50% of patients so take a family history. Prominent Q waves in the infero-lateral leads simulate an MI. 55% of patients lacking an outflow gradient at rest develop obstruction with exercise.
Contraindications: digoxin and vasodilators (e.g., CCBs and hydralazine).

HOCUM:
Asymptomatic; phenotypic cardiac features: Avoid vigorous excersie.
For stable CHF symptoms and preserved EF: B blockers, verapamil, disopyramide.

DDx of Restrictive Cardiomyopathy:
Non-infiltrative: idiopathic, familial, scleroderma.
Infiltrative: Amuyloidosis, sarcoidosis, hemochromatosis
Myocardial strage disease: Fabry disease (x linked deficiency of a-glactosidase)
Endomyocardial Disorders: Endomyocardial fibrosis, eosinophilic CM (Loffler), doxyrubicin.

Avoid betablockers, verapamil, and diltiazem in WPW.
Prosthetic valves: Warfarin and ASA x 3 months. Then ASA indefinitely.
Mechanical valves: Lifelong warfarin and ASA.

Patients with an unexplained CVA with an aortic atheroma should be treated with a statin and either anti-platelet therapy or warfarin.

ABI: Normal 0.91 – 1.4.

The following are **normal** in pregnancy:
*Increase in Pulse by 25%.
*Mild SOB with exertion.
*Reduced SPB by 10 mmHg
*An S3.
*Mild peripheral edema
*Grade 1 or 2 systolic murmur.

Peripartum cardiomyopathy.
*The leading cause of pregnancy related maternal deaths in the U.S.
*Deaths are caused by CHF, PE, and arrhythmias.
*Rx = betablockers digoxin, hydralazine, nitrates, and diuretics, I.V. immune globulin.

Hydralazine, Labetolol and metoprolol can be safely used in pregnancy.

Anticoagulation in pregnancy

Gestation in weeks	warfarin	UFH	LMWH	Anti-Xa LMWH
Venous thrombolism				
≤ 6	x			
6-12		x	x	
13-37	x	x	x	
37 to term		X		
A fib				
≤ 6	X			
6-12		x	X	
13-37	x	x	X	
37 to term		X		
Mechanical valve				
≤ 6	X			
6-12	x	x		X
13-37	x	x		X
37 to term		X		
Ventricular dysfunction				
≤ 6	X			
6-12		x	X	
13-37	x	x	X	
37 to term		x		

NEUROMUSCULAR SYSTEMS May 24, 2005

System	VOLUNTARY N.S.	AUTONOMIC N.S.	AUTONOMIC N.S.
Effector Anatomy	Skeletal muscle	Viscera & blood vessels	Viscera & blood vessels
Function	Motor	Fight or Flight: Catabolic, Sympathetic anatomically: Midriasis, tachycardia, quiet gut, sweating, increased alertness.	Vegetative-Digestive-Relax: Anabolic, Parasympathetic anatomically: miosis, bradycardia, peristalsis
Transmitter	Ach: Acetylcholine	Norepinephrine (also ATP & neuropeptide Y).* Ach in 1)preganglions, 2) post-ganglion sweat glands, 3)blood vessels to skeletal muscle causing vasodilation.	Ach (Also VIP) in 1)pre-ganglionic neurons, 2) post-ganglionic vegetative effector cells
receptor	Nicotinic receptors	Nicotinic	Muscarinic
Antagonist	Blocked by Tubocurare		Antagonists: atropine, ipratroprium(anti-asthmatic), scopalamine (causes mydriasis, decreases motion sickness, decreases GI motility for irritable bowel), antihistamines; antispasmodics: dicyclomine and hyoscyamine.
Agonist			Agonist: pilocarpine
Other		Adrenal medulla is a pot-ganglionic sympathetic cell that has lost its axon. It secretes Norepinephrine, Epinephrine & dopamine into blood.	

*Acetocholine is in all pre-ganglionic synapses of both PS and Sympathetic NS, in all parasympathetic postganglia, and in some sympathetic post ganglia. Norepinephrine is in the majority of postganglionic transmission, including the ciliary body, sublingual and submaxillary gland, heart, bronchi, bowel, rectum, urinary bladder, blood vessels and hair follicles of lower limb.

Effects of specific drugs on specific adrenergic receptors. January 31, 2004

Drug	Effect	ADRENERGIC RECEPTOR Alhpa1: vaso-const; heart: inc duration of contr'n.(2)	Beta-1: Increase Inotropy & chronotropy	Beta-2. Vascular walls cause vasodilation.	Other	Adverse effects
Phenylephrine (neosynephrine)	^ BP *	YYY				
Norepinephrine (levophed)	^ BP ^ HR	YYY	YYY			
Dopamine, 5 - 15 mcg/kg/min (Intropin)	^ CO some vasodil some vasocons	Y	YYY		YY	
Dopamine, 15+ mcg/kg/min	^ BP	YY				
Dobutamine (Dobutrex)	^ CO Slight v BP		YYY	YYY		
Vasopression						
milrinone (primacor)	vasodilates				phosphodiesterase inhib	ventricular arrhythmias; renal excretion.
nitroprusside (nipride)	vasodilates venous & arterial					cyanide formed; look for neuro changes and metabolic acidosis.
Nitroglycerin	v BP					
Isoproteronel	^ HR v BP		YY	Y		
Epinephrine <1 mcg/min	^ CO	Y	YYY	Y		
Epinephrine > 1 mcg/min	^ CO ^ BP	YYY	YYY			
nessiritide (natrecor)	venous & art vasodilation; diuresis, naturesis.				Inhibits r-a-a system,	
milrinone (primarcor)	vasodilates venous & arterial sm muscle; ^ cardiac cntrlty				phosphodiesterase inhibitor. Decreased C Amp degradation.	
vasopression	volume retains.				inc CAMP; v urine vol.	
fenoldipam						

(1) Dopamnie at 5-15 mcg/kg/min does not enhance renal perfusion.

(2) This is associated with a reflexive decrease in heart rate and cardiac output.

Notes
Ranolazone is anti-anginal and causes selective inhibition of the late sodium channel.
Also positive in:
Right ventricular infarction.
Massive Pulmonary embolism.
Other entities:
Obstruction of vena cava, atrial tumors, tri-cuspid stenosis and CHF, cardiac tamponade (rare)

Pulsus paradoxus: normal fall with inspiration is 12 mmHg.
Pulsus paradoxus is greater than 20. Occurs in **cardiac tamponade.**

Pulsus Paradoxus is paradoxical, as describe by Kussmal, because the apical beat persists while the pulse disappears.

It is the drop in systemic blood pressure with inspiration determined by deflating the cuff slowly and listening first for the point at which the pulse is no longer continuous and then becomes continuous altogether. This difference is normally less than 12 mmHg. With severe asthma and with pericardial tamponade, it is increased to greater than 12 mmHg.

In patients suspected of tampanode (two highly sensitive features are tachycardia and distended neck veins), pulsus paradoxus is highly sensitive for pericardial tamponade, sensitivity of 98%, specificty of 83%, and positive LR of 5.9 and negative LR of 0.03. If it is absent, the patient is very unlikely to have tampanode. (McGee, Evidence Based Physical Diagnosis, 2001). Pulsus paradoxus arises because the space within the pericardium is restricted, and an increase in the size of one of the four chambers of the heart, reduces the size of the others. On inspiration, there is increased filling of the Right Atrium, and the enlarged atrium pushes the interventricular septum into the LV, causing reduced filling and decreased LV pressure and hence decreased BP.

Constrictive Pericarditis: Prominent Y descent. The Y descent occurs in early diastole just after closure of the Aortic & Pulmonic valves (P2). The y descent begins the moment the tricuspid valve opens at the beginning of diastole, causeing the atrium to empty into the ventricle and venous pressure to fally abruptly. The prominent y descent (Friedrich's sign) occurs in 57% to 94% of patients with constrictive pericarditis.
Kussmaul's sign in **constrictive pericarditis**: Kussmaul's sign is the paradoxical increase in venous pressure with inspiration and occurs in 50% of patients with constrictive pericarditis. Constrictive pericarditis is associated with signs of right heart failure.

Other findings in constrictive pericarditis are atrial fibrilation (36-70%), pericardial knock (28-94%), hepatomegaly (87-100%), and ascites (ascites (53-89%).

Table. Duration of clopidogrel therapy. I.e., all stents get 1 year of clopidogrel. Only in chronic stable angina with a bare metal stent is there 1 month.d

Condition	No Stent	Bare metal stent	Drug eluting stent	CABG
Chronic stable angina	Only if ASA is contraindicated	1 month	1 year	Not indicated
UA, NSTEMI or STEMI	1 year	1 year	1 year	1 year

	Tamponade	Constrictive Pericarditis	Restrictive Cardiomyopathy	Right Ventricular MI
Pulsus Paradoxus (1)	Present			
Prominent Y Descent		Present		
Kussmaul's sign (2)		Present		
Pericardial knock		Present		
Heart Sounds		Distant		
Apical Pulse		Distant	Well defined	
Acute LV failure			Frequent	
LVH			Present	
RVH				Yes
Chronic illness		Often present		
Hepatomegaly & Ascites		Often present		
Pericardial calcification		Yes		
Low voltage	Yes	Yes	Yes	
Electrical alternans	Yes			
Echocardiography	Fluid		Increased LV thickness. Diastolic dysfunction(3)	

(1) Pulsus Paradoxus: Fall in system BP with inspiration > 12 mmHg

(2) Kussmaul's Sign: JVD declines during inspiration.

(3) Amyloid gives a speckled appearance of the wall.

Rhythm	Use	Contraindicated
Regular SVT	Carotid Sinus Massage, Adenosine, Verapamil, Diltiazem	
Atrial Fibrillation with aneterograde conduction	Procainamide	Adenosine, CCBs
Wide Complex tachycardia	Procainamide	

Causes of hyperlipidemia: hypothyroidism, nephrotic syndrome, and uremia plus drugs including oral contraceptives, estrogens, thiazides, and beta blockers.

NSAIDs inhibit prostaglandins and are contraindicated in heart failure.
Aspirin is widely prescribed but can also interfere with prostaglandin synthesis. Therefore, **Clopidogrel** (a non-aspirin platelet inhibitor) is preferred in CHF.

Tilt test is the most important new diagnostic tool for syncope.
Neurocardiogenic syncope: The patient is tilted up, and there is an increase in heart rate, but this is not sustained. After 20 minutes, both heart rate and blood pressure may fall.
Autonomic reflexes inappropriately dilate the arterial resistance vessels.
RX for autonomic hypotension is increased salt, fludrocortisone, ephedrine, or support hose.

Therapy for dig toxicity now includes digoxin immune Fab fragments!!

Amiodarone has several adverse effects, CHF, bradycardia, AV Block
Interstitial pneumonitis (lethal),
Hyper or hypo-thyroidism(4%),
Corneal microdeposits (100% in long term Rx with block of vision),
LFTs increased (30%)
Photosensitivity and slate blue skin

Pericarditis has up-sloping ST segment (middle sags up).
MI has down-sloping ST segment (middle tents up).

Pregnancy is absolutely contraindicated:
Primary Pulmonary HPT
Eisenmenger's syndrome.

Long QT Syndrome can present as syncope associated with emotional stress. Treatment is with beta blocker and ICD.

Murmurs and Maneuvers

	INCREASE	DECREASE
VALSALVA or STANDING	HCM	Other Murmurs
SQUATTING or SUPINE	Other Murmurs	HCM
Handgrip	MVP & MR	HCM & Most other murmurs

CLASSIFICATION OF ANTI-ARRYTHMIC DRUGS (Vaughan & Williams, 1984)

Class I : Decreases the Up-slope of the Action Potential. Drug closes Na Channels.

Ia quinidine (autoimmune thrombocytopenia; hearing loss, tinnitus, psychosis), procainamide (pericarditis); Disopyramide (Norpace)

Ib lidocaine

Ic flecainide

Class II: Decrease sympathetic activity. Drug slows sinus rate and slows AV node conduction.
Beta blockers (impotence)

Class III: Prolongs the Action Potential. Drug blocks K channels and prolongs QT interval.
Amiodarone (pulmonary fibrosis, ^,v thyroid, corneal deposits, gray skin, photosensitivity),
Ibutilide (use to convert A Fib)
Dofetilide (to maintain SR in A. Fib)
Bretylium
Sotalol (also is a beta blocker)

Class IV: Slows or blocks inward current (Ca+)
Calcium Channel Blockers: Verapamil (Calan, Isoptin); Diltiazem (Cardizem)

Miscellaneous: Adenosine (Adenocard)

ANTI-COAGULATION IN ATRIAL FIBRILLATION

	History of coagulopathy	CVA risk factors	ASA 325 mg	Warfarin: INR 2.5	W IN
Post cardioveersion for AF, patients to receive warfarin for 4 weeks.				x	
< 60, Lone AF	None	None	X		
< 60 with heart disease but no CHF or HPT and EF must exceed 35%	None	None	X		
> 60 with no heart disease	None	None	X		
Other intermediate risk patients		Yes		X	
Hi risk: RHD, prosthetic valve, prior PE, persistent thrombus on TEE.		NA			

Acute Aortic Regurgitation	Acute Mitral Regurgiation
Nitroprusside (decreases afterload) Chronotrope Inotrope	Nitroprusside (decreases afterload) ± Inotrope
Pacemaker to speed rate	Aortic balloon counterpulsation

	Chronic Aortic Regurgitation	Chronic Mitral Regurgitation	Aortic Stenosis	Mitral Stenosis
Most common causes	endocarditis, RHD, calcific degeneration	Myxomatous degeneration, RHD, endocarditis	Calcific degeneration; if RHD, then look for AR, MR & MS	Usually RHD
HEART SOUNDS	Pan or early diastolic, Hi pitch decrescendo, LSB to occ Apex, Loudest at end exp leaning forward , Hi pulse pressure & diffuse apical imp.	Pan systole, Blowing, Apex to occ Axilla, Increased w/ handgrip (NOT with standing), Diffuse apical impulse. Sometimes a click.	Mid to late systole, Harsh cresc-descresc, Base (2nd Rt ICS) to carotids (occ apex), Decreased w/ handgrip & standing, Decreased A_2 & Delayed pulses	Early or Pan diastole, Decrescendo rumble, Apex to none, L lateral decub position, OS.
Medical RX	Ab'c prophylaxis for endocarditis ACEI or CCB	Ab'c prophylaxis for endocarditis Simple MR: no vasodilator or beta blocker. With HPT or cardiomyopathy: use ACEI or long-acting CCB (amlodipine) **(avoid beta blocker)**	Ab'c prophylaxis for endocarditis. Possibly statins (may decrease progression) (No cardiac meds; go to Repair.)	Ab'c prophylaxis for endocarditis Ab'c prophylaxis for RF for 10 years or until age 40 and continuously to those with exposure (e.g., teachers). Use a **Negative chronotrope (beta blocker) to prolong filling.**
Monitoring	Moderate: Exam & Echo Q yr Severe: Exam & Echo Q 6 mo's	Moderate*: Exam & Echo Q yr Severe: Exam & Echo Q 6 mo's	NL valve area is 3-4 cm^2. Echo for 2-3 cm^2 is Q5yrs, for 1-2 cm^2 is Q2 yrs, for <1cm^2 is Qyr & look at LV function	
Indications for surgery	1) Sx's + LV Dysfctn OR 2) Asx'c + severe AR + ES diam >50mm or ED diam >70mm	1) Severe regurg + Sx's OR 2) Asymptomatic + any of AF, pulmonary HPT, or LV dsyfctn** 3)Earlier if good MV repair is likely.	1) Symptomatic OR 2) Asymptomatic with valve <1 cm^2 plus any of LV Dysfctn**, LVH, hypotension on exer-cise test (caution!), or rapid increase in jet velocity***	??? Apparently, weigh risks of delay (e.g., A.F. from increased LA size) VS potential success of PCT intervention (pliable non-calcified valve with ≤ 2+ MR) VS open heart surgery.

*MR classed as Compensated, Transitional & Decompensated corresponding to ECHO end diastolic diameter in mm: < 60, 65-68, >70,respectively, and to Cath Ejection Fractions of >60%, 53-57%, and < 50% respectively (from: UpToDate).

**LV Dysfunction defined as EF <60% or end systolic diameter > 45 mm.

***Average jet velocity increase is 0.3 m/s/year. A velocity > 3 m/sec represents a poor prognosis.

Anti-clotting agents for acute coronary artery syndromes February 19, 2004

	Mechanism	Indications	adverse effects (other than bleeding)	Dose	met'm
heparin (unfractionated) (Enoxaparin)	inhibits IX, X, XI, & XII via enhancing the binding of anti-thrombin III to serine proteases	Either: UA + TIMI 3+ or UA + ST v or ST v + CPKMB, Trop ^ (1)	HITTS (heparin induced thrombocytopenia and thrombosis syndrome)		Enoxaparin: monitor anti-Xa in ^ creatinine
warfarin	inhibits II, VII, IX & X via inhibition of vit K, a co-factor in their porduction				
ASA (ecotrin)	blocks prostaglandin synthesis and prostaglandin synthetase and thus thromboxane A2 synthesis (2)	As for Heparin		81 mg for prophylaxis 325 mg for prophylaxis post MI	
clopidogrel (Plavix) (a thienopyridine)	blocks ADP receptor on platelets & binding of fibrinogen	Possibly those for heparin		Acute: 300mg loading, then 75 mg qd	Liver
tirofiban (Aggrastat)	Inhibitor of GIIa/IIIb, necessary for bridging platelets.	As for heparin	I.C. bleeding, bradycardia	Acute: 0.4 mcg/kg/min for 30 min then decrease to 0.l V w/ ^ creatinine	Kidney
eptifibatide (Integrilin)	Ditto		I.C. bleeding, bradycardia	acute: 180 mcg/kg load, then 2 mcg/kg/min for 3 days. For thrombolysis, add a 2nd bolus at 10 min.	
Abciximab	binds to GIIa, III b receptors on platelets, preventing aggregation.	Primary antgioplasty without fibrinolytic Rx.	bleeding		Kidney
dipyridamole (Persantine)	inhibits platelet aggregation via accumulation of cyclic AMP and adenosine				

(1) TIMI score: Age 65+, 3+ CAD risk factors (DM, HPT, dislipid, FH, Cig), Documented CAD with 50+ stenosis, ST ^, 2 anginal episodes in past 24 hrs, ASA use in past week, ^ CK-MB or troponin.

(2) ASA promotes vasoconstriction and platelet aggregation.

Comparison of Pharmacologic Stress Agents†

	Dipyridamole	Adenosine	Dobutamine
Chemical	Pyrimidine derivative	Endogenous vasodilator of purine derivative	Synthetic catecholamine
Onset and duration of action, half life	Effect peaks at 7–15 minutes, half-life 30–45 minutes	Immediate, half-life < 5 seconds, effects disappear rapidly after infusion	Onset 1–2 minutes, half-life 2 minutes
Mechanism of action	Blocks reuptake of endogenous adenosine causing coronary vasodilation	Stimulation of adenosine receptor A2a causes coronary vasodilation	Alpha1, beta1, beta2 stimulation increases myocardial O2 demand and secondary vasodilatation
Dose	140 μg/kg/min for 4 min (max 0.56 mg/kg)	140 μg/kg/min for 6 min	5–40 μg/kg/min, depending on heart rate response
Radionuclide injection	7–9 minutes after initiation of infusion	3 minutes in to infusion, infusion continued for further 3 minutes	At peak stress
Hemodynamics	Slight increase in heart rate and slight decrease in blood pressure (BP)	Slight increase in heart rate and slight decrease in BP (adenosine>dipyridamole)	Target heart rate 85 percent of maximum predicted heart rate
Side effects	Occur frequently, but minor	Same as dipyridamole but resolve rapidly heart block more common	Most common chest pain, most serious nonsustained ventricular tachycardia, non fatal myocardial infarction
Contraindications	Bronchospasm, second or third degree AV block or sick sinus syndrome (unless protected by a functioning pacemaker)	Same as dipyridamole	Recent acute coronary syndromes, hemodynamic and electrophysiologic instability

† Data from Muthu Velusamy, MD.

OBSERVATIONS FROM CECIL AND HARRISON QUESTIONS:January 12, 2005

Paradoxical splitting of S2 is associated with CLBB, aortic stenosis, and WPW with a right sided accessory pathway.

Hypertrophic cardiomyopathy is characterized by severe *diastolic* dysfunction.
In hypertrophic cardiomyopathy, the EKG can show LVH and inferior or deep, broad, lateral Q waves mimicking MI, left atrial enlargement, T wave inversions.

Which of the following are the most useful ways to distinguish ventricular tachycardia (VT) from a supraventricular tachycardia (SVT) with aberrancy? Canon A waves clinically or AV dissociation on EKG.

Reduced heart rate variability is associated with increased risk of sudden death following MI.

PR segment depression is specific for pericarditis.

Atrial flutter saw tooth waves are best seen in the inferior leads. Typical atrial flutter is characterized by the absence of an isoelectric interval in the inferior leads and a sawtooth appearance of the flutter waves. The atrial flutter cycle length is typically 200 ms or 300 beats/minute. Because 2:1 conduction commonly occurs at this rate, this tachycardia is usually 150 beats/minute.

Half of patients with AS will have CAD even if asymptomatic. Dyspnea in AS is the symptom associated with the worse prognosis (e.g., compared to syncope, angina, or palpitations).

The amount of alcohol to produce symptomatic cardiomyopathy is estimated to be six drinks a day for 5 to 10 years. For idiopathic dilated cardiomyopathy, steroids have not been proven to improve morbidity or mortality (*Cecil, Ch. 73*)

Renovascular Hypertension: **High-pitched, systolic-diastolic, or continuous abdominal bruits** are strongly suggestive of renovascular obstruction and are found in one half to two thirds of patients. One third of patients have fibromuscular disease, and two thirds have atherosclerotic disease. Patients with fibromuscular dysplasia are younger and less likely to experience cardiovascular complications. Those with atherosclerotic disease tend to be older, have higher systolic BPs, and have more target organ damage than patients with essential hypertension. In patients with suitable lesions, percutaneous revascularization can often be performed at the diagnostic renal arteriography

Causes of pulmonary artery hypotension: Hypoxia; polycythemia. Endothelin is a powerful vasoconstrictive substance and is elevated in some forms of pulmonary hypertension. Prostacyclin is vasodilatory. *(Cecil, Ch. 64*)

What is Epstein's anomaly? An anomalous formation of the Tricuspid valve leading to TC regurgitation.
Correlates of coarctation of the aorta: bicuspid aortic valve,aneurysm of the circle of Willis is less

common.

Duration of time to exercise testing in asymptomatic patients with recent CABAG is 5 years and with recent stent angioplasty is 3 years.

Exercise stress testing is contraindicated in severe aortic stenosis and in severe hypertrophic cardiomyopathy. (*Cecil, Ch. 59*) Exercise-induced hypotension is a poor prognostic indicator, as are severity of ST depression, duration of ST segment depression, early onset of ST segment depression, and poor exercise capacity.

Magnetic resonance imaging of the heart has been used to diagnose hemochromatosis, taking advantage of the paramagnetic properties of iron tissue stores.

ACE inhibitors inhibit kininase II, which breaks down bradykinin, a vasodilator. Higher doses of ACE inhibitors are more effective than low doses. ACE inhibitors should thus be titrated to the doses used in the large congestive heart failure mortality trials. ACE inhibitors can cause hyperkalemia.

Catch 22 syndrome: 22q11 band abnormalities are associated with cardiac defects, abnormal facies, thymic hypoplasia, cleft palate, and hypocalcemia.

Patients with atrial septal defects are at increased risk of stroke.
Atrial arrhythmias are common in ASD.

Indications for pacing during surgery: congenital complete heart block, slow heart rates at baseline, a wide QRS complex, inadequate responses to exercise, and a history of syncope.

Peripheral intravenous catheters can present a significant hazard in patients with cyanotic congenital heart disease because of the increased risk of paradoxical embolism.
Systemic hypotension increases the right-to-left shunt in cyanotic congenital heart disease and may be fatal. Bleeding risk is increased in cyanotic congenital heart disease.

ApoB1/ApoA1 is strongly associated with MI (Yusuf Lancet 2004;364:937).
HDL is believed to promote cholesterol efflux from atherosclerotic plaques and inhibits the oxidation of LDL particles.

Walk-through angina is defined as angina that occurs with exercise but disappears with continued exertion.

Postprandial angina occurs in response to increased oxygen demand in the splanchnic bed after meals.

Nocturnal angina is due to increased venous return as the patient assumes a supine position.
New-onset angina is a subtype of unstable angina.

Treatment of diastolic dysfunction: Beta Blockers; possibly ACEIs. NOT Dig.

Differential diagnosis of Angina: Atherosclerotic coronary artery disease, severe aortic stenosis, aortic insufficiency, hypertrophic cardiomyopathy, and syndrome X, a disorder of the myocardial resistance vessels.

Causes of pericarditis: hydralizine, procainamide, and minoxidil .

Down syndrome is associated with endocardial cushion defects are most common.
Marfan syndrome is associated with aortic aneurysms, aortic regurgitation, aortic dissection, or a floppy mitral valve.
Maternal systemic lupus erythematosus is associated with complete heart block in the offspring. The maternal lupus may not become symptomatic until years after the birth of an infant with congenital complete heart block.
Maternal rubella is associated with patent ductus arteriosus.

An R/S ratio greater than 1 in V_1 and V_2 is indicative of a true posterior myocardial infarction.
T wave inversion throughout the precordium should raise suspicion for a subtotal occlusion in the proximal left anterior descending coronary artery.

Handgrip produces an **increase in systolic BP, left ventricular end-systolic pressure, and end-diastolic pressure**. It is important to instruct the patient not to Valsalva while performing handgrip because this can confuse the response of the murmur to the maneuver. **Squatting** from a standing position produces transient increases in venous return as a result of compression of lower extremity musculature. An increase in systemic vascular resistance and afterload is then produced by compression of the leg arteries. **Standing** from a squatting position produces a reduction in venous return as a result of venous pooling in the legs and in systemic vascular resistance.

Harrison Access Medicine:
RCA occlusion & posterior descending coronary artery lead to Inferior Wall MI. AV block (Mobitz II) & sinus bradycardia.

LAD occlusion leads to AV Block but Greater Hemodynamic instability.

Lidocaine is particularly likely to cause confusion in the elderly patient, for whom a lower dose of the drug should generally be given. Other potential adverse effects of lidocaine include tremor, convulsions, respiratory depression, bradycardia, and hypotension.

The standard approach to ventricular fibrillation or pulseless ventricular tachycardia involves defibrillation with 200 joules, then 300, then 360, followed if needed by epinephrine 1 mg IV push every 3 to 5 min. Persistent ventricular fibrillation or pulseless ventricular tachycardia leads to consideration of amiodarone 300 mg IV push or lidocaine 1.0 to 1.5 mg/kg IV push. In addition, magnesium sulfate 1 to 2 g IV may be given in torsade de pointes or when arrhythmia due to hypomagnesemia is suspected. Procainamide up to 50 mg/min (maximum total 17 mg/kg) is given to patients with intermittent return of a pulse or non-VF rhythm, but then recurrence of VF/VT. A precordial thump may be considered in this setting, but there is insufficient evidence to

recommend its use or avoidance.

Resuscitation of a patient with cardiac arrest is dependent on the rapidity of the initiation of resuscitative efforts, the clinical status of the patient before the arrest, and the mechanism of the event. The most appropriate management of an individual with pulseless ventricular tachycardia consists of an initial 200-J defibrillation. Follow-up shocks at 300 J and 360 J should be attempted if a normal rhythm is not reestablished. The current Advanced Cardiovascular Life Support (ACLS) guidelines call for the administration of either epinephrine or vasopressin, continued CPR, and repeat attempts at defibrillation. Furthermore, in patients with VT intravenous amiodarone has emerged as the optimal antiarrhythmic. Sodium bicarbonate may be given to an individual with a persistent acidosis, but there is no evidence to support this. Lidocaine is a second-line agent and may be given to patients in whom amiodarone has not been successful. Bretylium and procainamide have little or no role in modern ACLS guidelines.

Mitral Valve Prolapse: Any maneuver that reduces left ventricular size, such as standing or the Valsalva maneuver, allows the click and murmur to occur earlier in systole; conversely, conditions that increase left ventricular size, such as squatting or propranolol administration, delay the onset of the click and murmur.

Causes of QT prolongation and Torsades de pointes:

Intracranial events.

Hypomagnesemia (e.g., due to diarrhea).

Hypokalemia.

Drugs::

Anti-arrhythmics: quinidine, sotalol, ibutilide, procainamide, amiodarone

Antiinfective agents (quinolones, erythromycin, clarithromycin, pentamidine)

Antiemetics (droperidol, domperidone)

Antipsychotics (haloperidol, thioridazine)

Other drugs (cisapride, methadone).

Acutely: Rx with Mg and overdrive pacing.

Preventively in congenital long QT use beta blockers.

Osteogenesis imperfecta: brittle bone disease. Blue scleara, hearing loss, short stature, multiple fractures.

loop diuretic: furosemide.

fat pad biopsy: This is for amyloidosis.

The Revised Cardiac Risk Index

Factor	Adjusted Odds Ratio (OR) for Cardiac Complications in Derivation Cohort
1. High-risk surgery	2.8
2. Ischemic heart disease	2.4
3. History of congestive heart failure	1.9
4. History of cerebrovascular disease	3.2
5. Insulin therapy for diabetes mellitus	3.0
6. Preoperative serum creatinine > 2.0 mg/dL	3.0

		Cardiac Complication Rates, %	
Class	Number of Factors	Derivation Cohort	Validation Cohort
I	0	0.5	0.4
II	1	1.3	0.9
III	2	3.6	6.6
IV	3—6	9.1	11.0

Source: Adapted from TH Lee et al. Circulation 100:1043, 1999; with permission.

The abrupt onset of severe hypertension or the onset of any hypertension before the age of 35 or after age 55 should prompt evaluation for renovascular hypertension.

Aortic Aneurysm:
Atherosclerotic
Congenital: Marfan’s, Ehlers-Danlos.
Infections: Syphilis, mycotic aneurysms from BE, TB
Inflammatory: Takayasu's arteritis, giant cell arteritis, seronegative spondyloarthropathies, Reiter's syndrome, ankylosing spondylitis, Rheumatoid arthritis.

Wellen's T waves are deep symmetric T-wave inversions that are seen in either significant left

main coronary artery stenosis or proximal left anterior descending artery stenosis

Acute pericarditis: widespread ST elevation in V2—V6 with reciprocal changes in aVR.
The clinical presentation of acute pericarditis can mimic acute myocardial infarction (MI), aortic dissection, pulmonary embolism, or gastroesophageal reflux disease (GERD). The classic history of acute pericarditis includes sharp substernal pain that **is worse supine and improved upright**. Viruses are usually echo and coxsackie.

This patient presents with classic findings of right-sided heart failure. The differential diagnosis includes pulmonary vascular disease, restrictive cardiomyopathy, constrictive pericarditis, cor pulmonale, and any cause of long-standing left-sided heart failure. A CT or MRI of the chest would assess for pericardial calcifications or parenchymal lung disease. Iron studies for hemochromatosis, fat pad biopsy for amyloidosis, which may restrictive cardiomyopathy. Constrictive pericarditis: TB.

Hypothermia: The ECG shows convex elevation of the J point at the end of the QRS and the beginning of the ST segment in multiple leads (Osborne waves, easily visible in II, III, aVF, and V4—V6). These changes along with QT-interval prolongation.

U Waves: hypokalemia
QT prolongation: see above for causes of Torsades de pointes.

Myoglobin is released from ischemic myocardial cells and appears in serum within hours. It has a very short half-life in serum as it is excreted rapidly in the urine. So it can be used to determine new mycoardial injury.

Eponymous Wellen's T waves are symmetric T-wave inversions in the early precordial leads, which suggest either significant left main stenosis or high left anterior descending stenosis.

Statins can stabilize plaques, cause long term ergress of lipids, and improve vasodilatory tone.

Prinzmetal's, or variant, angina is defined by transient epicardial coronary artery vasospasm with subsequent electrocardiographic abnormalities that include ST-segment elevation or depression. In the majority of patients with this disorder, there is significant atherosclerotic coronary stenosis within at least one major vessel and the spasm occurs within 1 cm of the obstruction. The most common site of focal spasm is the right coronary artery. Epidemiologically, patients with variant angina are younger and have fewer coronary risk factors and lack preceding chronic stable angina. Patients with this as a suspected diagnosis can undergo provocative maneuvers to elicit the electrocardiographic or angiographic changes. Of note, hyperventilation ergonovine, acetylcholine, and other vasoconstrictors have been used. The mainstays of therapy are calcium channel blockers and nitrates to promote vasodilation and prevent spasm. Aspirin is thought to increase the severity of ischemic episodes and is relatively contraindicated.

RX of Acute Ascending aortic dissection is with nitroprusside and a beta blocker (esmolol). Hydralazine is contraindicated.

Increased QT and increased ST segment: hypocalcemia.
U Waves: hypokalemia, use of quinidine, procainamide, and disopyramide.
Increase QRS width: Hyperkalemia.
Short QT interval: Hypercalcemia, Digoxin toxicity.
Bradycardia: Hypercalcemia
Long PR interval: Hypercalcemia.
Wide T wave: Hypercalcemia.

Treatment of hypertensive crisis: The new drug is **fenoldopam** is the best choice. Fenoldopam is an intravenous selective dopamine-1 agonist. It is highly specific for dopamine-1 receptors at 10 times greater affinity than dopamine and does not bind to 1 or 1 receptors. Because of its actions at the dopamine-1 receptor, fenoldopam increases renal blood flow and natriuresis. It has an onset of action within 5 minutes, and no rebound is associated with discontinuation of the continuous infusion. No oral preparation is available.

Nitroprusside is relatively contraindicated in this patient because it increases intracranial pressure.

Causes of high output failure: Paget's, anemia, arteriovenous fistulas, pregnancy, hyperthyroidism, and beriberi. Treatment of paget's with bisphophonates, such as alendronate or risedronate will improve heart failure symptoms.

Melanoma is the tumor most likely to metastasize to the heart, although the most common primaries originating from tumors of the heart are breast and lung owing to the high incidence of these tumors.

Cough syncope is thought to be due to elevated intrathoracic pressure that is transmitted to the inferior and superior vena cavae. Because of the increased pressure on the venous systems, there is a drop in venous return with a concomitant drop in cardiac output. Bradycardia also can contribute to the syncope with increased vagal tone during the event.

Several lifestyle modifications for hypertension:

>Weight reduction to a normal body mass index (18.5 to 24.9) reduces systolic pressure from 5 to 20 mmHg for each 10 kg of weight loss.

>The DASH diet is high in fruits, vegetables, and low-fat dairy products while avoiding saturated and total fats. This diet was proved in the Dietary Approaches to Stop Hypertension study to decrease systolic blood pressure by as much as 14 mmHg.

>Adopting a low-sodium diet with no more than 2.4 g of sodium daily improves BP by 2 to 8 mmHg.

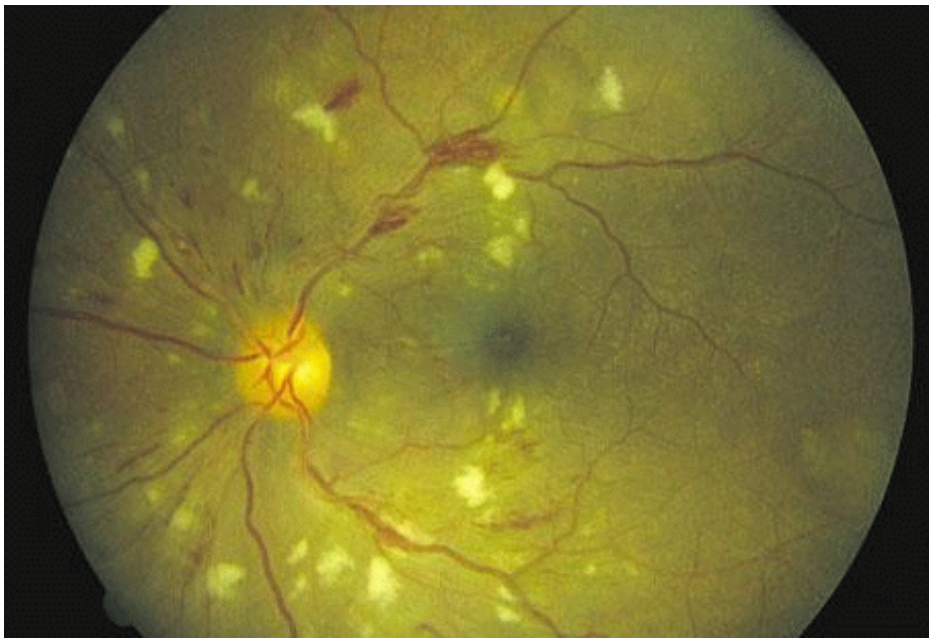
>Physical activity for a minimum of 30 min several days weekly

>Limitation of alcohol beverages to one drink daily in women and two in men.

Shock

An important discriminator in determining distributive shock (e.g., due to sepsis) is systemic resistance, which is calculated as SVR
= 80 * (Systemic Arterial Pressure- Right Atrial Pressure mmHg)/Systemic Blood Flow (L/min). Normal value is 700 - 1600 dynes / cm^2. This value is reduced in distributive shock and increased

in other types of shock. An analogous calculation can be made for pulmonary vascular resistance, PVR, which is normally 20-130.



Retinal emboli can be made up of platelets, cholesterol (Hollenhorst plaque as shown in the question), or calcium. Hollenhorst plaques are generally identified at vessel bifurcation, and their presence suggests that the patient has atherosclerotic plaque proximal to the retinal artery, commonly aortic, heart, great vessel, or carotid. Carotid dopplers are indicated in this case to evaluate for stenosis caused by plaque buildup.

BLS: Basic Life Support:
Sequence for Adults:
*Determine unresponsiveness – shake victim. "Are you OK?" >> unresponsive.
*Call 911
*Look for breathing. >> Not breathing >> Tilt chin (jaw thrust if trauma). >> Deliver two breaths.
*Check for carotid pulse. No pulse. Deliver 15 chest compressions. 1.5 inches deep just below mid-nipple line.
*Repeat 2 - 15, breaths - compressions, at a rate of 100 compressions/minute with a ventilatory rate of about 12 to 14 / minute.
*After one minute, check carotid pulse.
*Press below the cricoid cartilege to prevent regurgitation and inflation of the stomach.
*Continue CPR.

Sequence for Adults with AED immediately available.
*Determine unresponsiveness.
*Call 911.
*Look for breathing. >> Not breathing >> Tilt chin (jaw thrust if trauma). >> Deliver two breaths.
*AED >> Power on >> Attach leads >>Stand clear for analysis >> Stand clear for shock.

For choking responsive adult: FBAO (Foreign Body Airway Obstruction)
*"Are you choking? Can you speak? (If not, then it is severe.)"
*"I can help you."
*Deliver Heimlich: Base of left hand ½ way between umbilicus and xyphoid to give pulses.
*If victim is obese, pregnant, or an infant, the pulses are given to the sternum just above the xyphoid.

For a choking non-responsive adult:
*Grab chin and tongue and open mouth to look for blockage. Do a deep finger sweep blindly in adult (NOT in a child).
*Attempt to deliver breath. If not successful, re-position, and deliver another breath. If still not successful....
*Straddle victim and deliver 4 abdominal pulses.
*Repeat "Belly>Buccal>Blow" until blockage is relieved.

For choking responsive infant
Hold infant in one hand and arm with arm against knee with head downward.
Deliver 5 pulses with heel of hand to back.
Turn over to opposite hand and arm against knee with head downward.
Deliver 5 pulses to sternum.
If infant becomes unresponsive, place infant supine and examine open mouth but DO NOT SWEEP blindly.
Attempt to deliver breath >> fail >> reposition and attempt breath.
Repeat sequence for responsive infant with 5 pulses of heal to back and 5 pulses of fingers to sternum.
Repeat exam of open mouth.
Repeat pulses.

For CPR on infant or child from 1 - 8 years of age, the sequence is as for adults, except do CPR for 1 minute before calling for help due to high probability of respiratory arrest.
AND
The ventilation-compression ratio is 5-1.
For infant, the compressions are given with 2 fingers.
For infant, check the pulse at the brachial artery.
For child 1 - 8, the compressions are given with heal of one hand only.

Pericarditis and/or pericardial effusion, causes
Idiopathic.
Infection: HIV, Hep B, coxsackie and echoviruses. Baceterial MTB Fungal
Autoimmune: Rhematic disease (SLE, RA, scleroderma); Nonrheumatic diseases: IBD, giant cell arteritis, PAN.
Radiation/ Post MI/ Neoplasms/ Cardiac injury
Medications: Procainamide, hydralazine, Pencillin, doxorubicin.
Uremia/ Aorrtic dissection/ Takotsubo CM.
Treatment: ASA 650 Q 4-6HR; Ibuprofen 400-800 Q 6 – 8 Hrs.>>Colchicine>>Prednisone