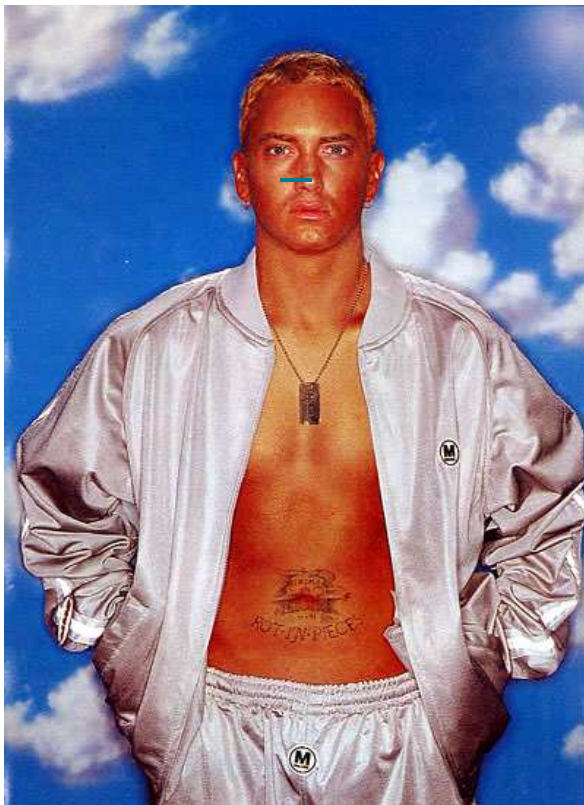




M&M

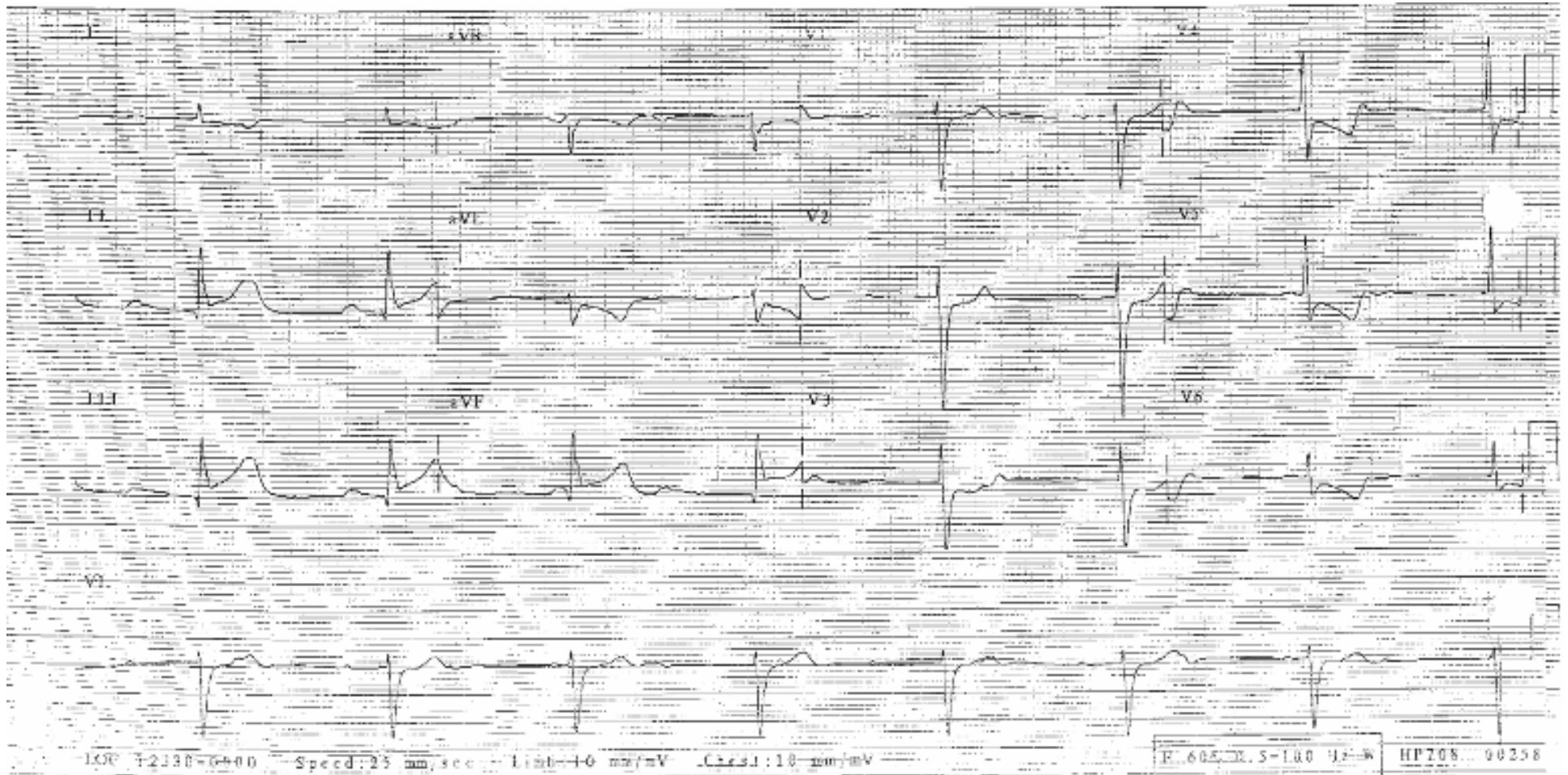
David Stultz, MD
PGY 4 Cardiology
January 22, 2004



Case #1

- 47 YO intoxicated WM
- Syncopal episode, complains of crushing chest pain
- + Tobacco, + FH

Initial EKG



Right Sided EKG



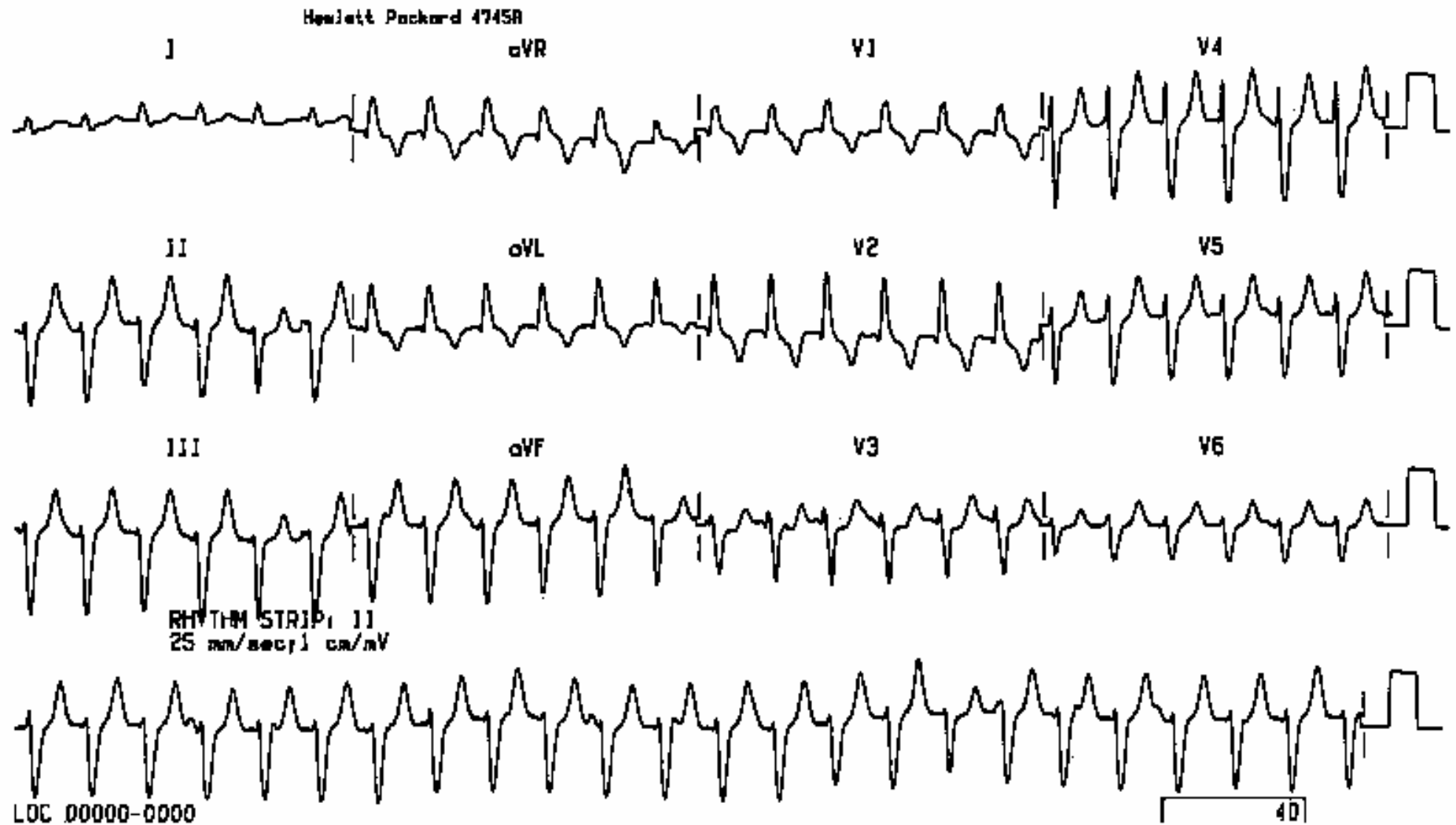
During Cath



2:1 AV block

- Every other P wave is conducted to the ventricles
- 2 to 1 AV block starts after the 5th QRS in this 3 channel recording. The first non-conducted P wave is indicated with an arrow.
- the PR interval of conducted P waves is constant
- in this EKG there is a long PR interval
- 2 to 1 AV block cannot be classified into Mobitz type I or II as we do not know if the 2nd P wave would be conducted with the same or longer PR interval.

Also During Cath



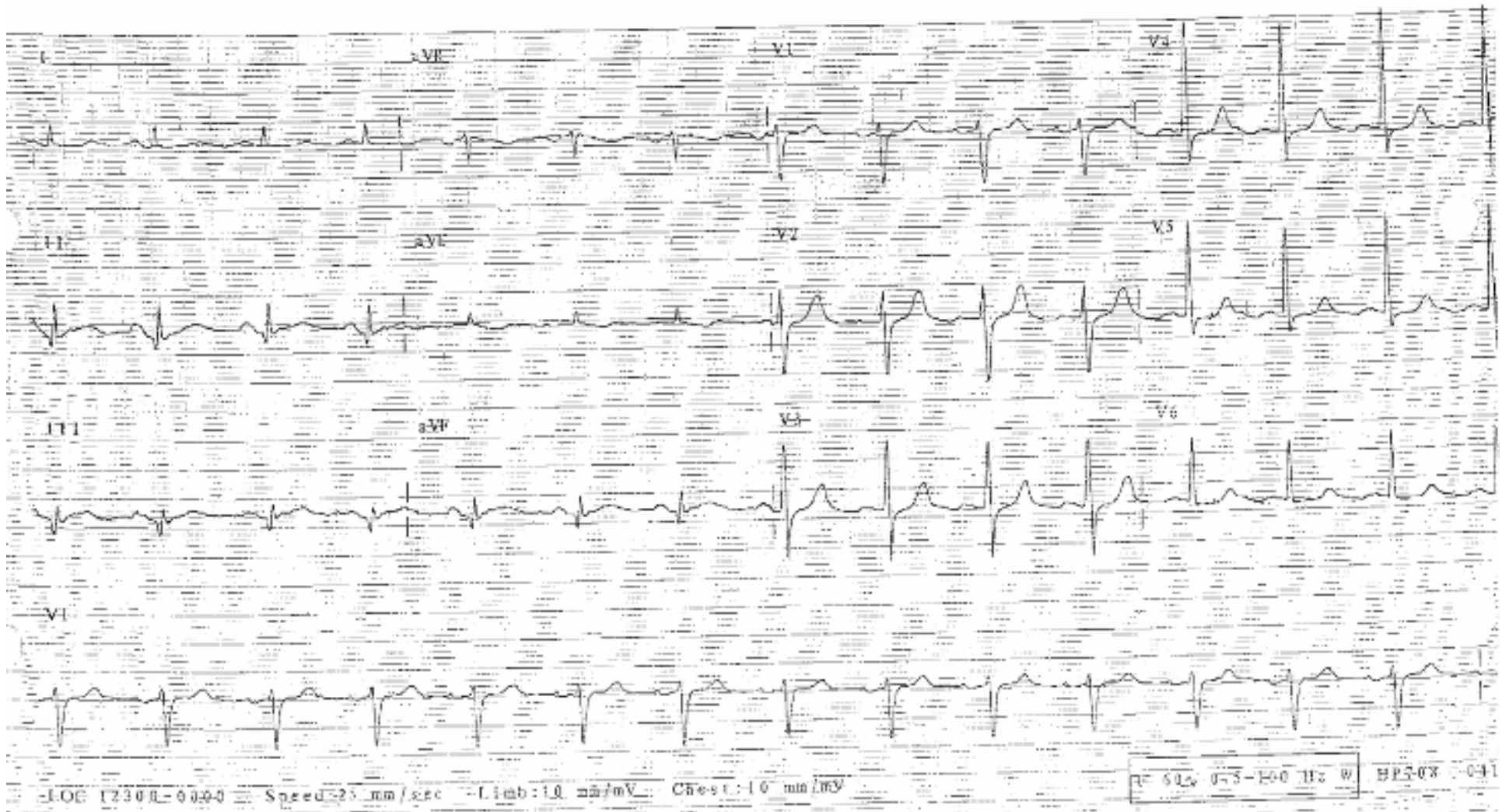
Ventricular Tachycardia

A wide QRS tachycardia is VT until proven otherwise.

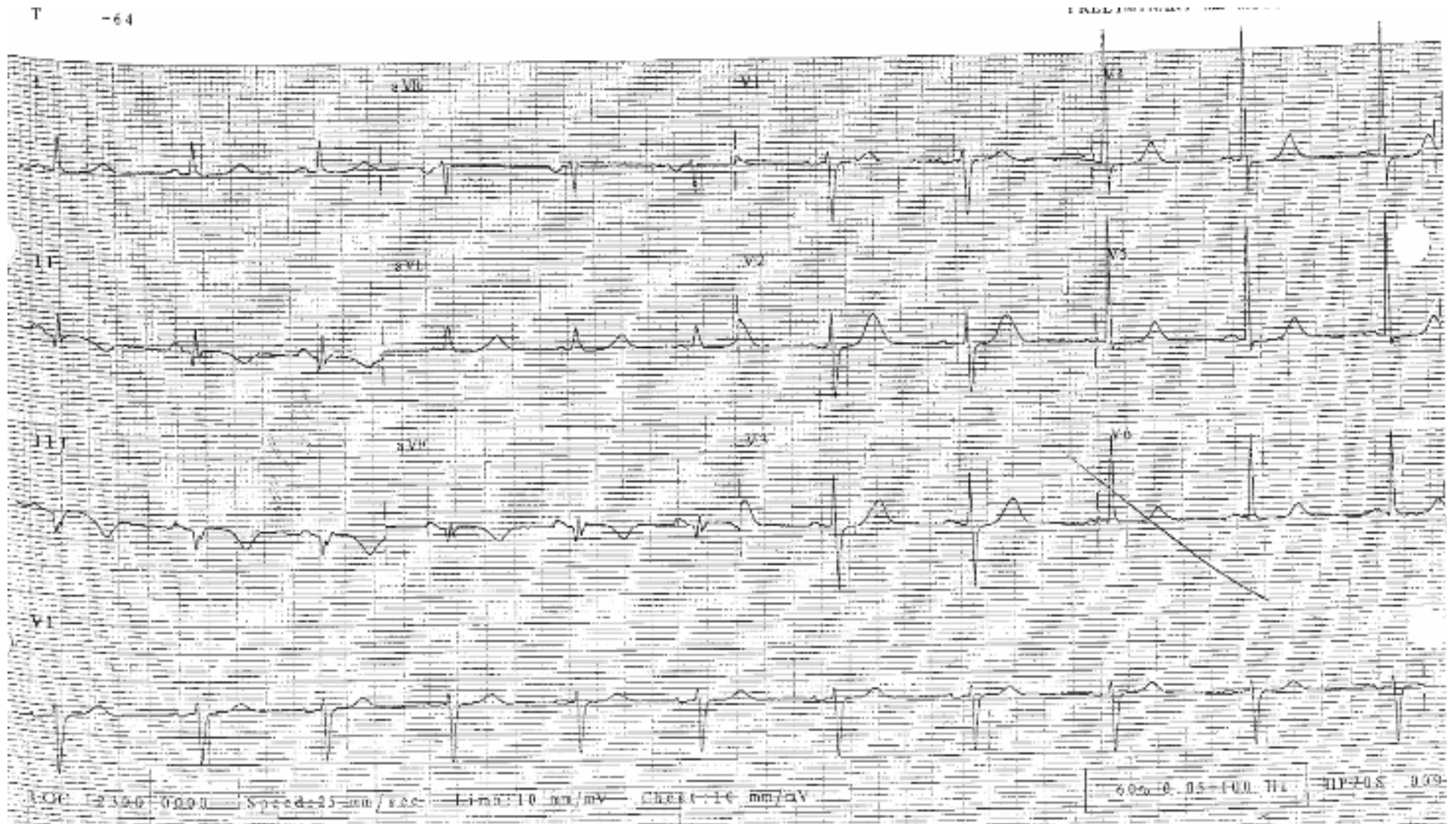
Features suggesting VT include

- evidence of AV dissociation
- independent P waves
- capture or fusion beats
- beat to beat variability of the QRS morphology
- very wide complexes (> 140 ms)
- the same morphology in tachycardia as in ventricular ectopics
- history of ischemic heart disease
- absence of any rS, RS or Rs complexes in the chest leads
- concordance (chest leads all positive or negative)

4 hours after admission About 2 hours post cath



24 hours after admission



48 hours after admission



6 days after admission



RV Infarction

- **Clinical Findings**
 - Normal or depressed right ventricular function
 - Shock
 - Tricuspid regurgitation
 - Ruptured ventricular septum
- **Hemodynamic Measurements**
 - Abnormally elevated right atrial pressure
 - Normal right ventricular and pulmonary artery systolic pressures
 - Increased ratio of right ventricular to left ventricular filling pressure
 - Depressed right ventricular function curve

RV Infarction

- **Cardiac Catheterization**
 - Involvement of right (usually) or left (rarely) circumflex coronary arteries
 - Right ventricular akinesis
- **Differential Diagnosis**
 - Hypotension with acute myocardial infarction
 - Pericardial tamponade
 - Constrictive pericarditis
 - Pulmonary embolus

RV Infarction

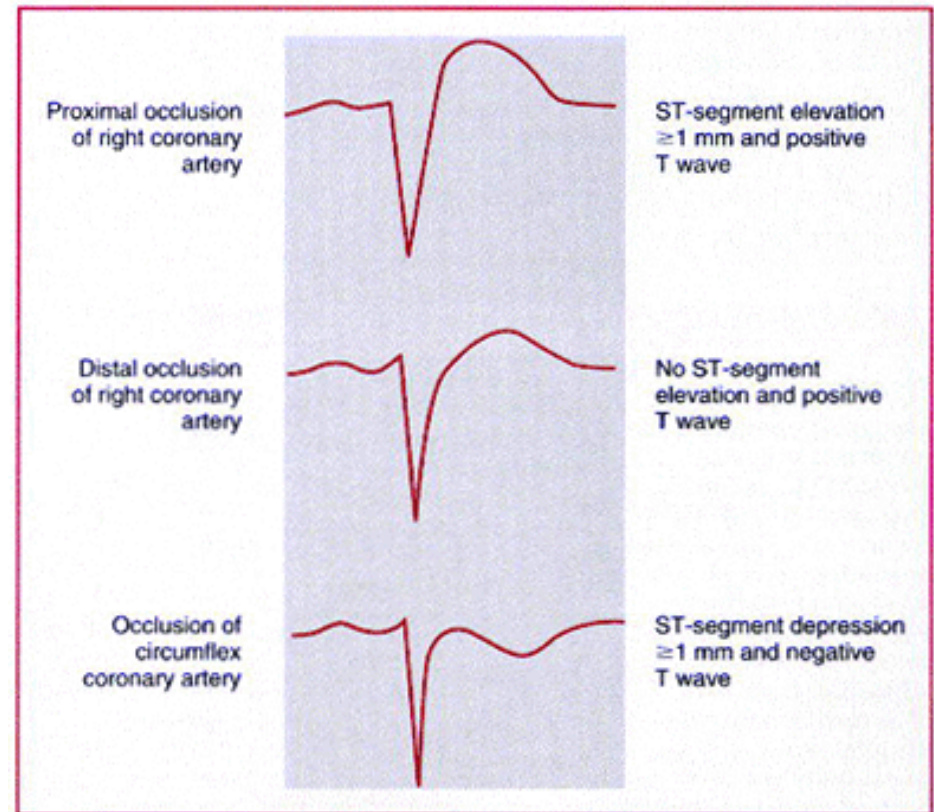
- **Scintigraphy**
 - Uptake in right ventricular free wall
 - Increased right ventricular dimensions and decreased wall motion
- **Echocardiography**
 - Increased right ventricular dimension
 - Absence of pericardial effusion
- **Cardiac Enzymes**
 - Increased magnitude of enzyme values relative to degree of left ventricular dysfunction

Diagnostic features of RV infarct

- Elevated right ventricular filling pressure
- Steep right atrial y descent
- Early diastolic drop and plateau (square root sign) in the right ventricular pressure tracing
- Pulsus Paradoxus (10 mm drop of Systolic with inspiration)
- Kussmaul's sign (Increase in JVP with inspiration)

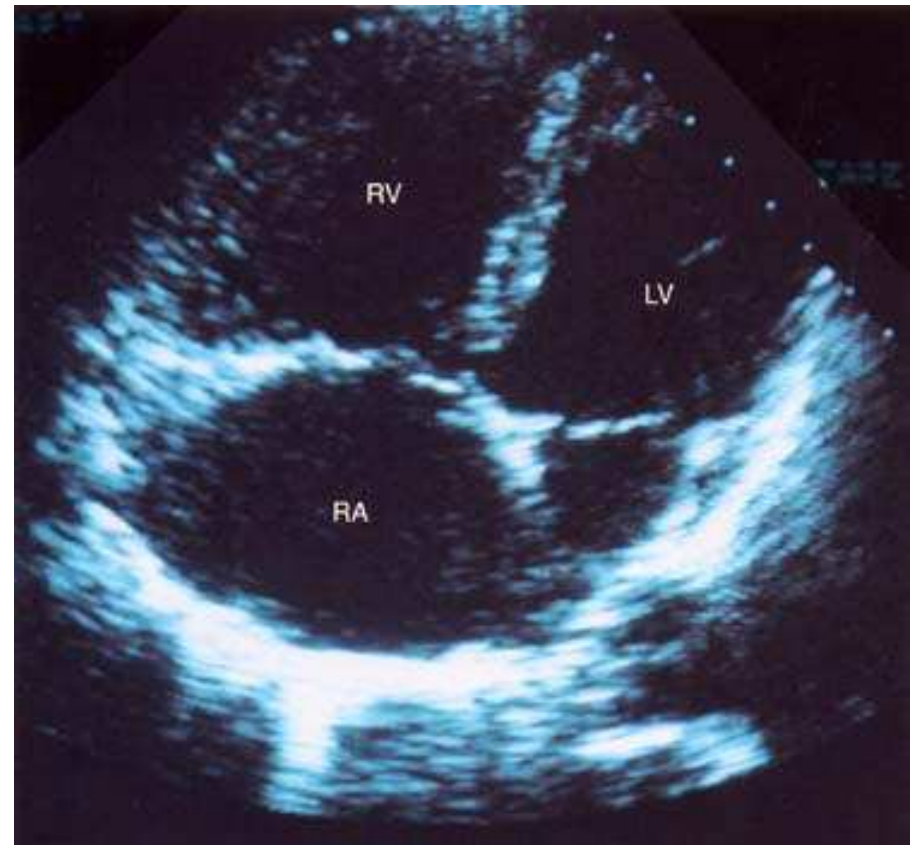
Right Sided EKG (V₄R)

- Proximal RCA – ST elevation 1mm and positive T wave
- Distal RCA – No ST elevation, positive T wave
- Cx – ST depression 1mm and negative T wave



Echocardiography

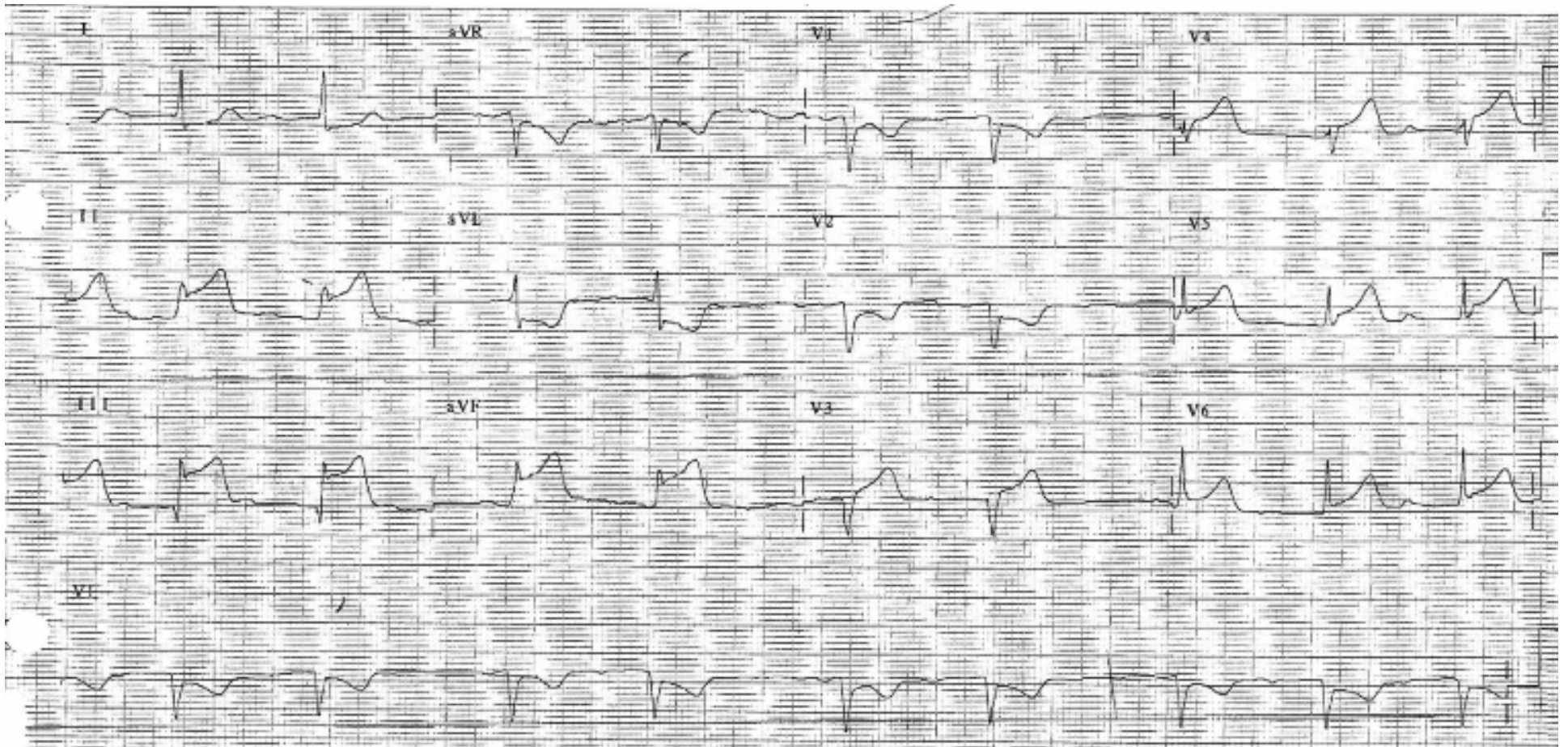
- Dilated RV
- Dilated RA
- Regional wall motion abnormality (Left or Right Ventricle)
- No pericardial fluid



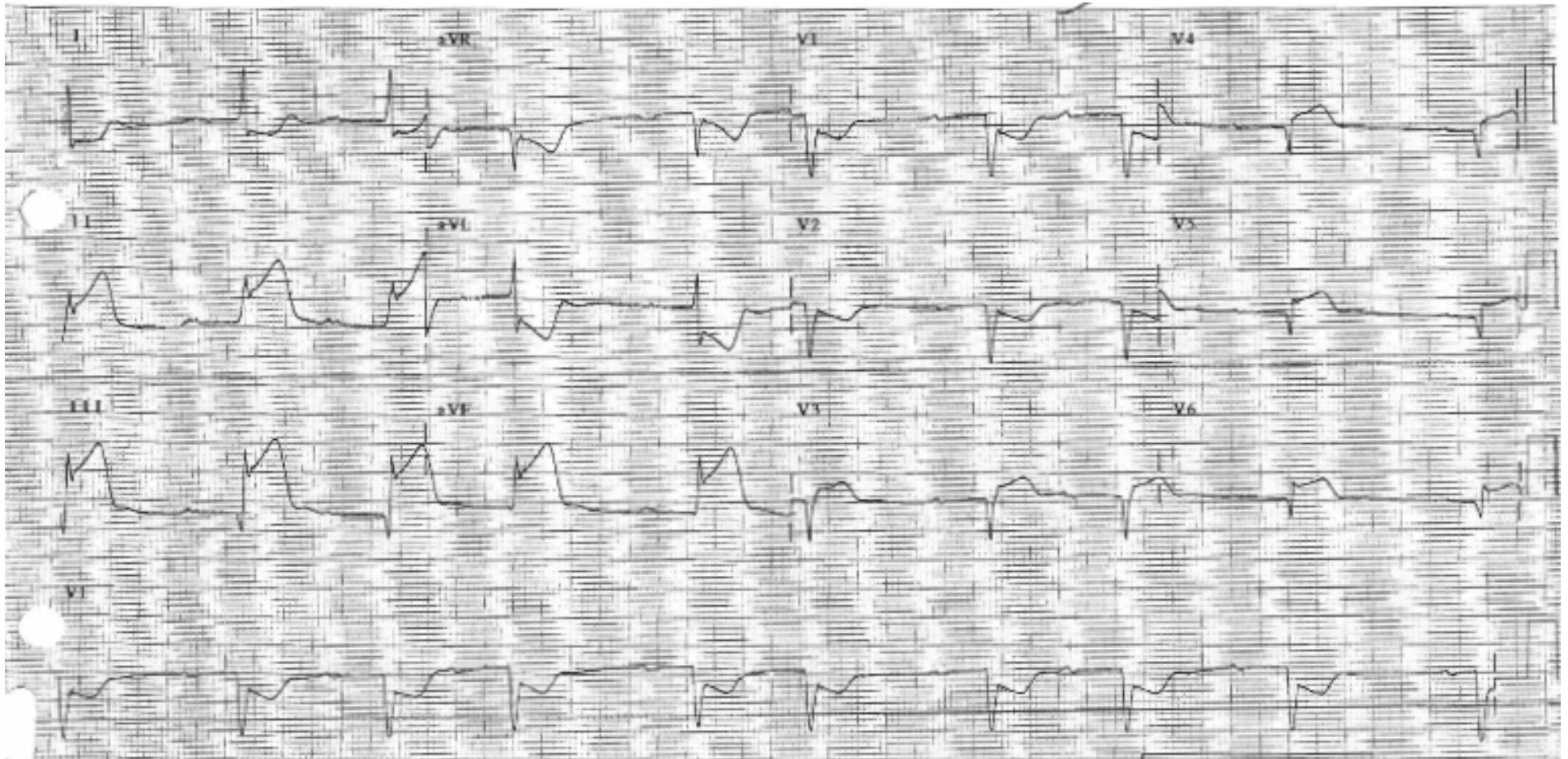
Case 2

- 50 yo AAF with hx HTN, +tob
- Vomiting, diaphoresis, +/- Chest pain

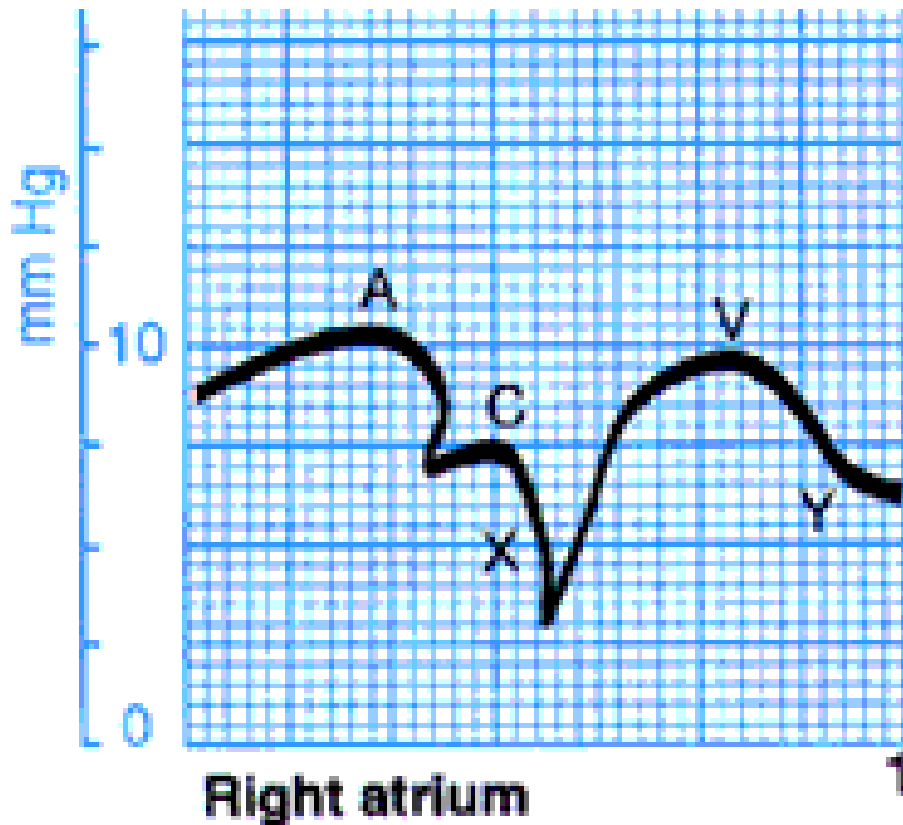
Initial EKG Case 2



Right Sided EKG Case 2



Right Atrial Waveform



- a wave** - RA contraction
elevated in RV failure
- c wave** - tricuspid closure
- v wave** - passive filling of RA during ventricular systole = T wave on ECG
elevated in tricuspid regurgitation
- x descent** - atrial diastole
- x' descent** - RV systole
- y descent** - atrial emptying

Hemodynamics of RV infarct

- Elevated right sided filling pressures
- Equalization of right and left sided diastolic pressures
- Low cardiac output

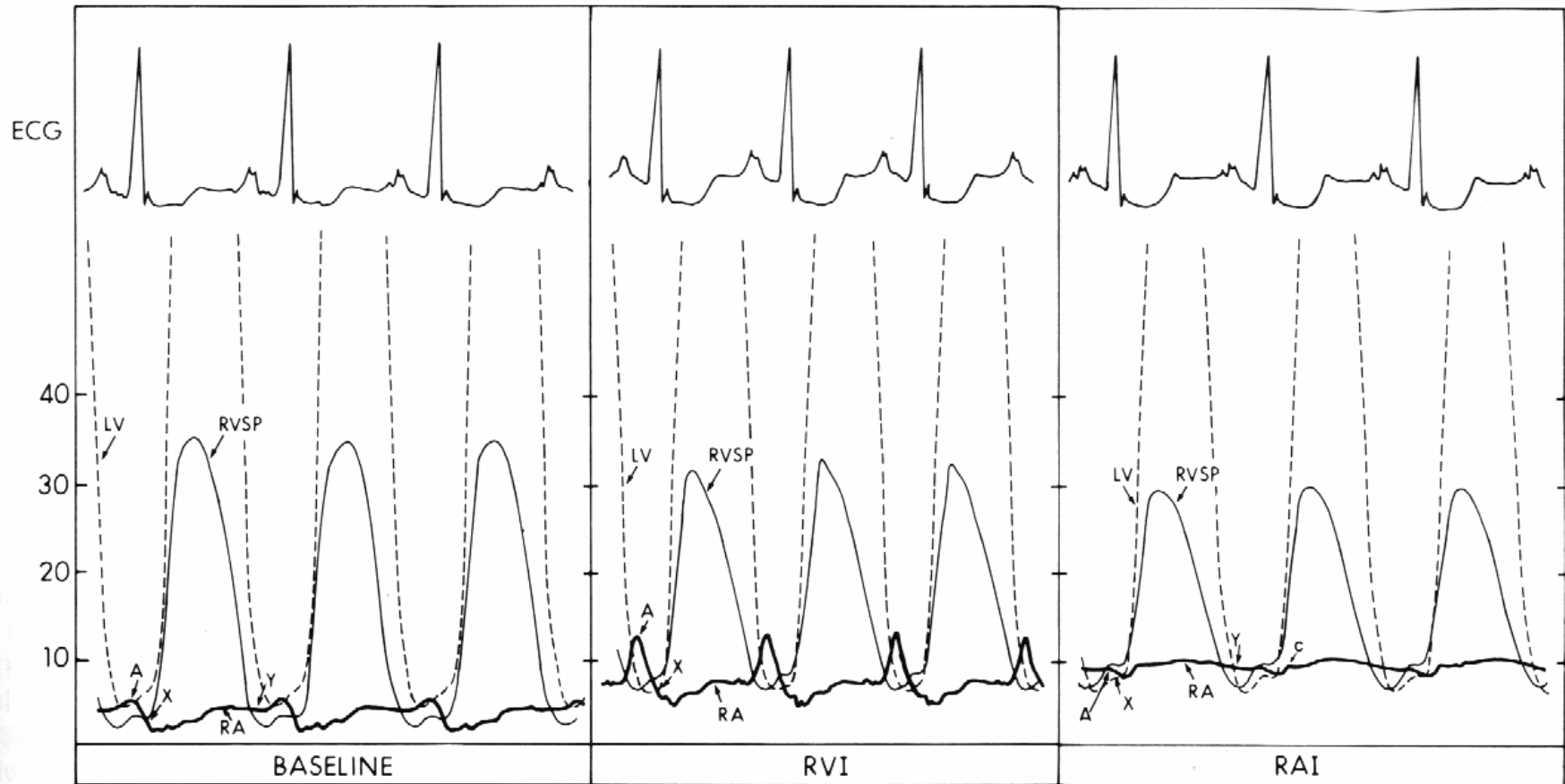
Features to watch

- Y descent (tricuspid opening/passive filling) is a measure of RV compliance
- A wave and X descent - RA contraction typically augmented with stiff RV, but if RCA occlusion is proximal this may be compromised

“W” and “M” patterns

- W wave
 - RCA occlusion distal to RA branches
 - Augmented a waves
 - Steep x descent
- M wave
 - RCA occlusion proximal to RA
 - Depressed a waves
 - Blunted x descent

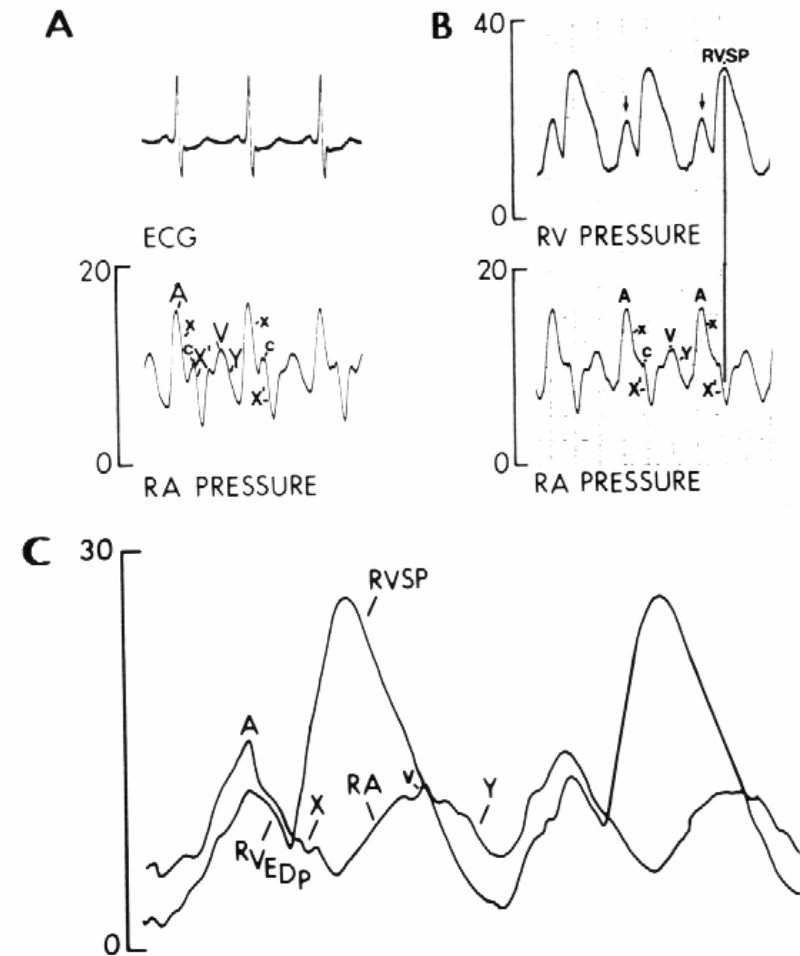
Progression of waveforms



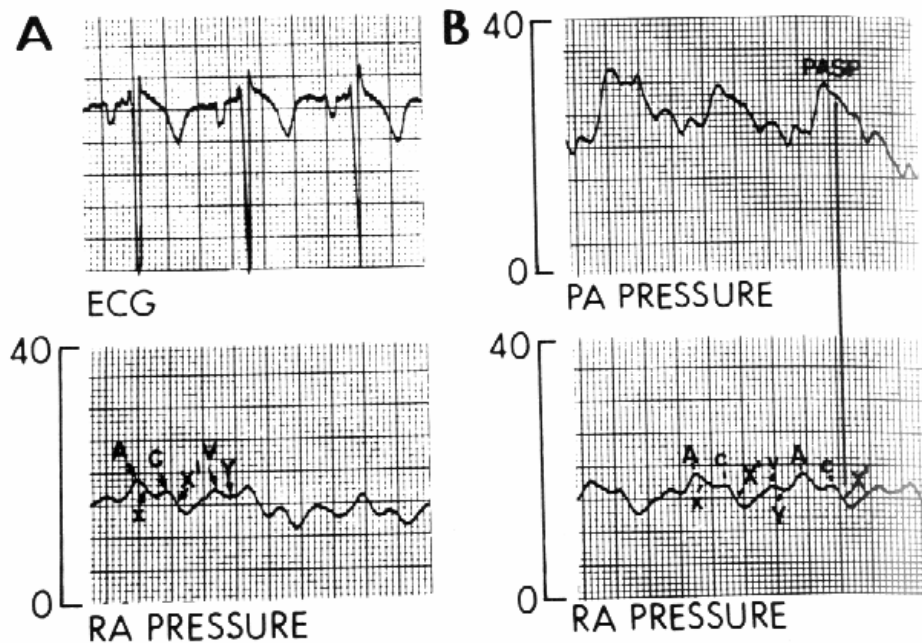
- RVI – augmented a wave and x descent
- RAI – blunted a wave and x descent

Hemodynamics of RV infarct occlusion distal to RA

- “W” pattern formed by prominent a waves and sharp x descent
- X descent is a **SYSTOLIC** event, and correlates with RVSP
- Note elevated RVEDP corresponding with tall a wave

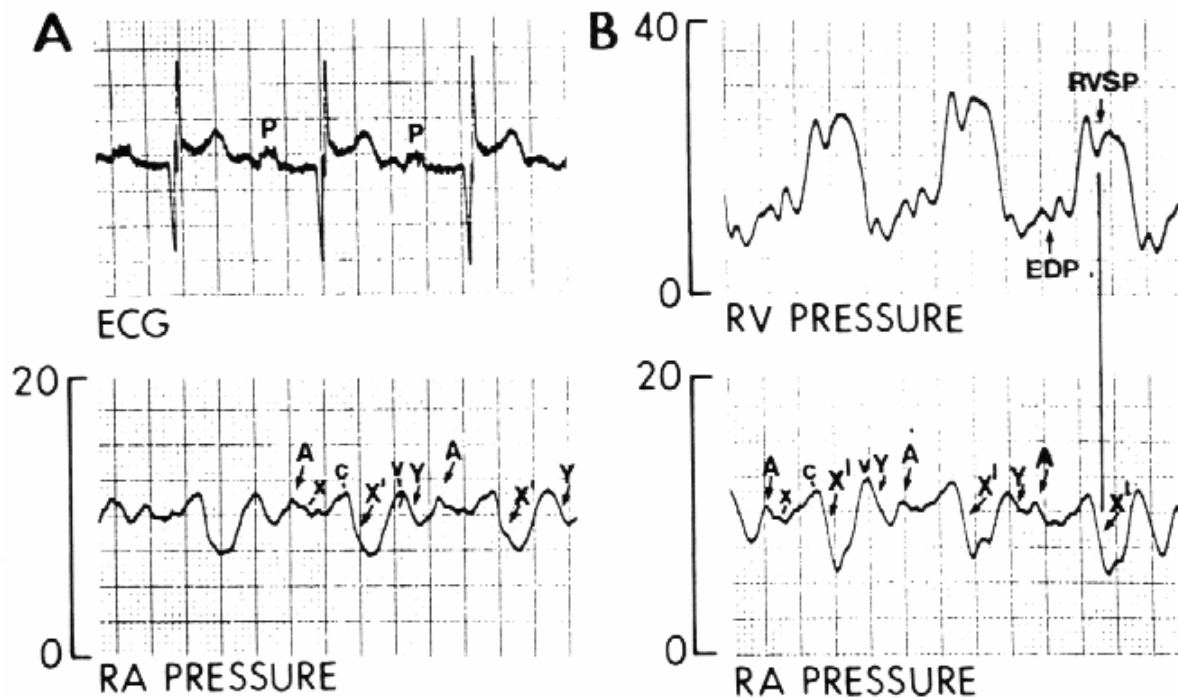


Hemodynamics of RV infarct occlusion proximal to RA



- “M” pattern formed by blunted a wave and x descent
- X’ descent occurs during t wave
- X’ descent occurs during PASP peak (systolic event)

Another “M” pattern



- X descent occurs during t wave
- RV tracing reveals that sharp descent is x
- Diastolic y blunted

Treatment of RV infarct

- **Maintain Right Ventricular Preload**
 - Volume loading (IV normal saline)
 - Avoid use of nitrates and diuretics
 - Maintain AV synchrony
 - AV sequential pacing for symptomatic high-degree heart block unresponsive to atropine
 - Prompt cardioversion for hemodynamically significant SVT
- **Inotropic Support**
 - Dobutamine (if cardiac output fails to increase after volume loading)

Treatment of RV Infarct

- **Reduce Right Ventricular Afterload with Left Ventricular Dysfunction**
 - Intraaortic balloon pump
 - Arterial vasodilators (sodium nitroprusside, hydralazine)
 - ACE inhibitors
- **Reperfusion**
 - Thrombolytic agents
 - Primary PTCA
 - CABG (in selected patients with multivessel disease)

Pearls for RV infarction

- If hypoxia is present, consider PFO with Right → Left shunt due to increased right sided pressures
- Tricuspid Valve replacement/repair can be performed for severe TR in setting of RV infarct

Case #3

- 74 yo F with hx of CABG
- Symptoms of syncope
- Ultimately referred for cath as outpatient
- Hx of DM, CLL, HTN

Cath procedure

- Difficult stick
- R femoral access obtained, above anatomic crease
- No problems during cath
- Perclose procedure performed successfully

15 minutes later...

- Pt discharged, had syncopal event on ride home
- Pt admitted to CCU
- Hgb 10
- CT scan + for retroperitoneal bleed
- Hypotensive
- Attempted L subclavian TLC
- Successful L femoral TLC

Off to surgery

- Surgical consult on board stat
- To OR post cath day 1
- L subclavian 8.5F sheath + swan
- L femoral 6F sheath for art line
- Repair of R external iliac
 - Perclose sutures noted 'nearby'
- Stabilized post op

Medications

- Ativan 1mg
- Demerol 50 mg
- Kcl 20 meq
- Lasix 40mg
- Phenergan 25 mg
- Hydroxyzine 25mg
- Albumin 5%
- Nicardipine IV
- Clonidine 0.3 patch
- Pepcid 20mg bid
- Hydralazine 10mg q6h
- Solumedrol 40mg
- Zaroxolyn 5mg po bid
- Lopressor 50mg bid
- Versed prn
- Morphine prn
- NaCl 75 cc/hr
- Diprivan
- Terazosin

Interesting labs...

	OP	POD 1 0200	POD 1 0700	POD 2	POD 3	POD 4
Na	137	136	133	139	135	136
K	5.2	5.0	4.7	4.6	4.2	3.4
Cl	109	110	112	108	106	104
CO ₂	21	20	12	23	23	23
BUN	25	29	29	35	44	56
Creat	1.5	2.0	2.4	2.8	3.5	4.2
WBC	95.7	157	159	66	36.5	23.9
Hgb	26.3	6.7	9.4	10.5	8.8	10.6
Plt	208	222	203	82	57	48

Uh oh

- On POD #4, on routine assessment pt found to have cold, mottled L leg. Distal pulses previously dopplerable, not able to doppler now.
- L fem 6F arterial line still in place

To the cath lab

- Thrombus proximal and distal to sheath
- SFA 100%
- Deep femoral with decreased flow
- 100% popliteal
- 100% posterior/anterior tibial, peroneal

- To the OR for thrombectomy

What happened

- Underlying CLL
- Cath with difficult stick
- Retroperitoneal bleed
- Acute renal failure
 - ATN due to shock, low Hgb, decreased perfusion
- Thrombocytopenia
- Thrombus formation

Heparin induced thrombocytopenia?

(Well, that's what I'm going to talk about, anyway)

- Two types
 - Nonimmune
 - Benign, self-limited
 - Dose dependent
 - Rarely causes large drop in platelet count or clinical complication
 - 15% of patients treated with heparin
 - Immune mediated
 - 2-3% of all exposed patients
 - Heparin Induced Thrombocytopenia +/- Thrombosis
 - 25-30% mortality rate, 25% amputation rate

Risk factors for HIT

- Initial low platelet count
- Renal impairment
 - Increased risk of death
- Recent cardiovascular surgery
 - Increased risk of amputation

Diagnosing HIT

- Thrombocytopenia
 - Decrease of 50% from baseline
 - Start thinking when there is 30% decline
- Absence of other cause of thrombocytopenia
- Confirmation by heparin associated antibody assay
- Return of normal platelet levels when heparin is discontinued

Clinical Features of HIT

- Usual onset day 5-6 (3-14)
- Nadir of platelets usually 30-60k
- Occurs with all types/methods of heparin
 - Continuous infusion>flushes>heparin coated catheters
 - Bovine heparin>porcine heparin>LMW heparin
- Can start within hours of exposure
- Surgical patients have increased venous risk
- Cardiovascular patients have increased arterial risk
- No relation to gender, age, or inherited clotting disorders

Thrombosis

- 35% of patients with HIT will develop clinically significant thrombosis
- Thrombosis can form anywhere
- Arterial > Venous or Venous > Arterial depending on the study
- DVT, PE, Mesenteric ischemia, CVA, Myocardial infarction

Pathogenesis in a nutshell

- Heparin attaches to Platelet factor 4 (PF4) on platelet surfaces
- IgG Fab antibody binds heparin/PF4 aggregate
 - Fac portion recognizes Fcγ1a receptors on platelets
- This causes the platelet to release PF4
- Heparin/PF4 complexes can form on endothelial surfaces; IgG attaches to this and damages the cell

Lab tests

- Platelet aggregation assay
 - Normal plt + pt serum + heparin evaluated for aggregation
- Serotonin release assay (SRA)
 - Donor plt incubated with radiolabeled serotonin
 - 40-80% sensitive
- ELISA to heparin-PF4 complex
 - False negatives common
- Clinical HIT may have negative lab tests!

Management of HIT

- DC all heparin
 - Flushes, catheters
 - This is not sufficient to prevent thrombosis, however (35% develop thrombosis)
- Direct thrombin inhibitor
 - Lepirudin
 - Argatroban
 - 6 day therapy (up to 14)
 - PTT 1.5-3x baseline
 - New thrombosis/amputation/death (25.6% vs. 38.8% control)

Other meds (not recommended) for HIT

- ASA, dextran, warfarin are not indicated for sole treatments of HIT
- Danaparoid
 - Low molecular weight heparinoid
 - Favorable in vitro activity vs heparin
 - Anecdotal/small series support
 - Unable to monitor, no dosing regimen
- Ancrod – a defibrinating agent that is not recommended

Future meds for HIT

- Hirulog (Angiomax)
 - Currently in development for HIT
- Factor Xa inhibitors
 - Arixtra
- Antiplatelet agents
 - 2b/3a, plavix have limited, but successful, clinical experience

Other management of HIT

- Coumadin may be started if otherwise indicated when platelet counts $>100k$
- Plasmapheresis can be used to filter out IgG antibodies if heparin exposure is unavoidable
- Immunoglobulin can be used in patients refractory to plasmapheresis
- Thrombolysis with urokinase can be used for organ/limb threatening ischemia. Selective thrombolysis causes less trauma than thrombectomy

Risk of heparin use

- Heparin risk is primarily bleeding complications
 - Risk factors:
 - advanced age
 - serious concurrent illness
 - heavy consumption of alcohol
 - concomitant use of aspirin
 - renal failure

Other risks of heparin

- Anaphylaxis
 - 5-10 minute onset
 - Wheezing, chills, fever, tachycardia, diaphoresis
 - Limited patients, have been successfully treated with LMWH without reaction
- Osteoporosis
- Skin reactions (urticaria, necrosis)
- LFT abnormalities
- Eosinophilia
- Hyperkalemia
- Hyperaldosteronism
- Priapism
- Alopecia

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