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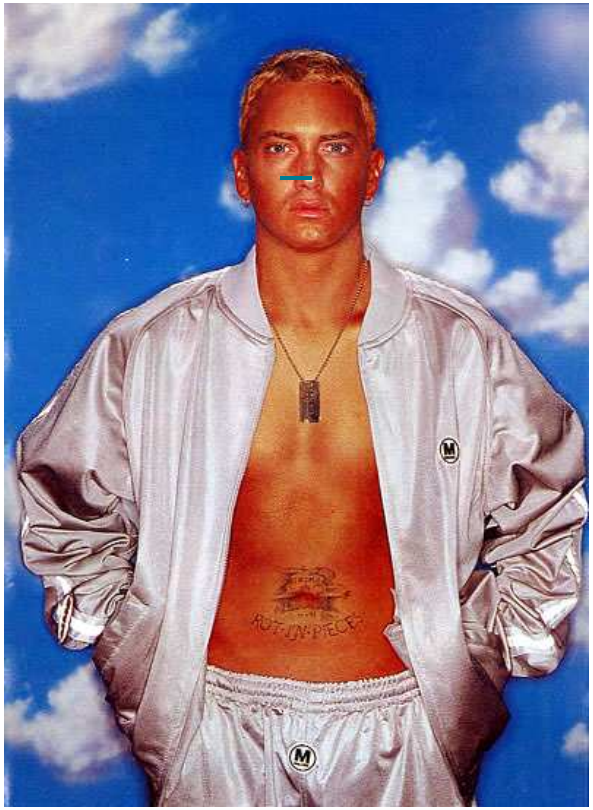
M&M

David Stultz, MD
PGY 4 Cardiology
January 22, 2004



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Case #1

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

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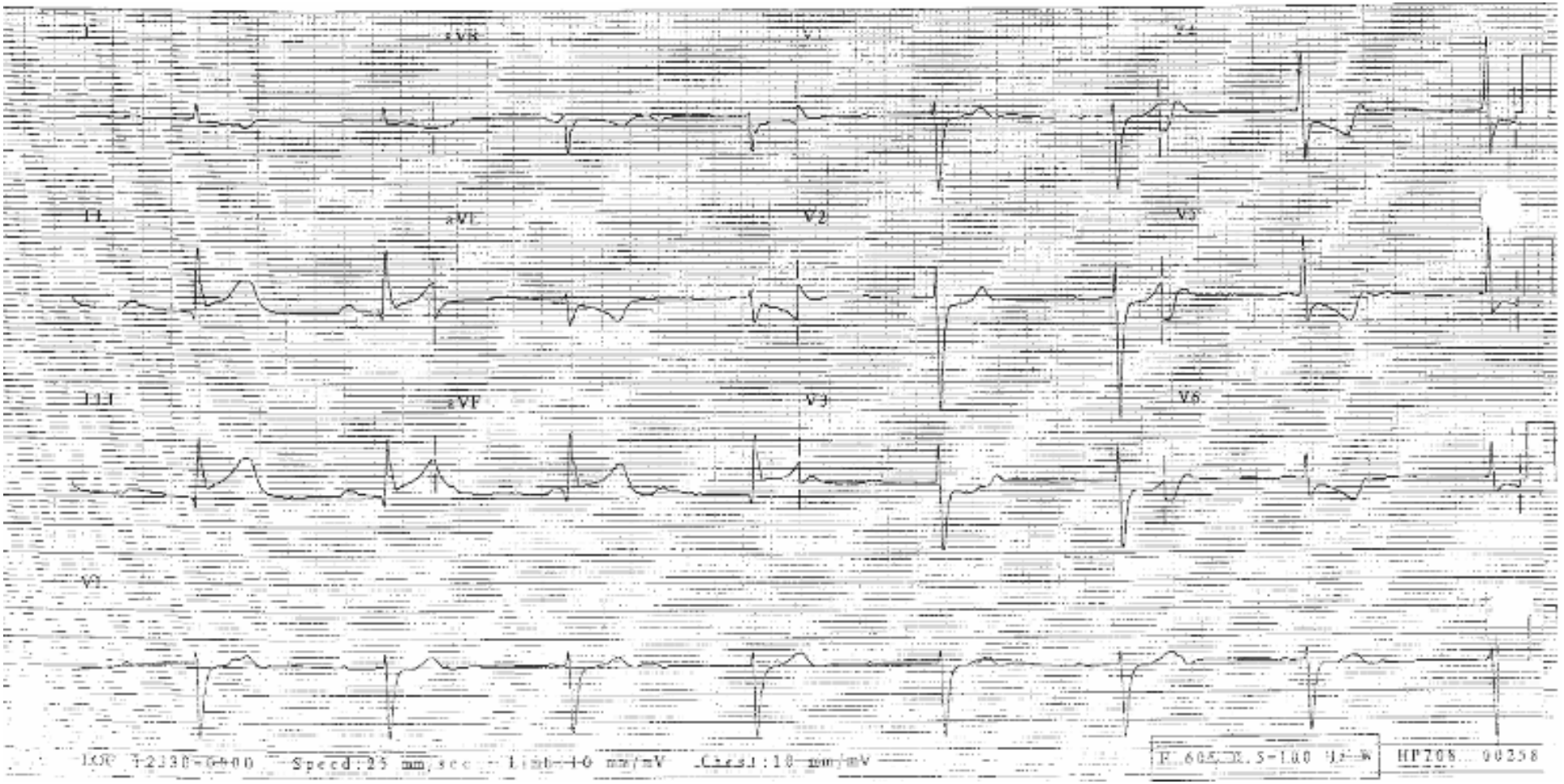
Initial EKG



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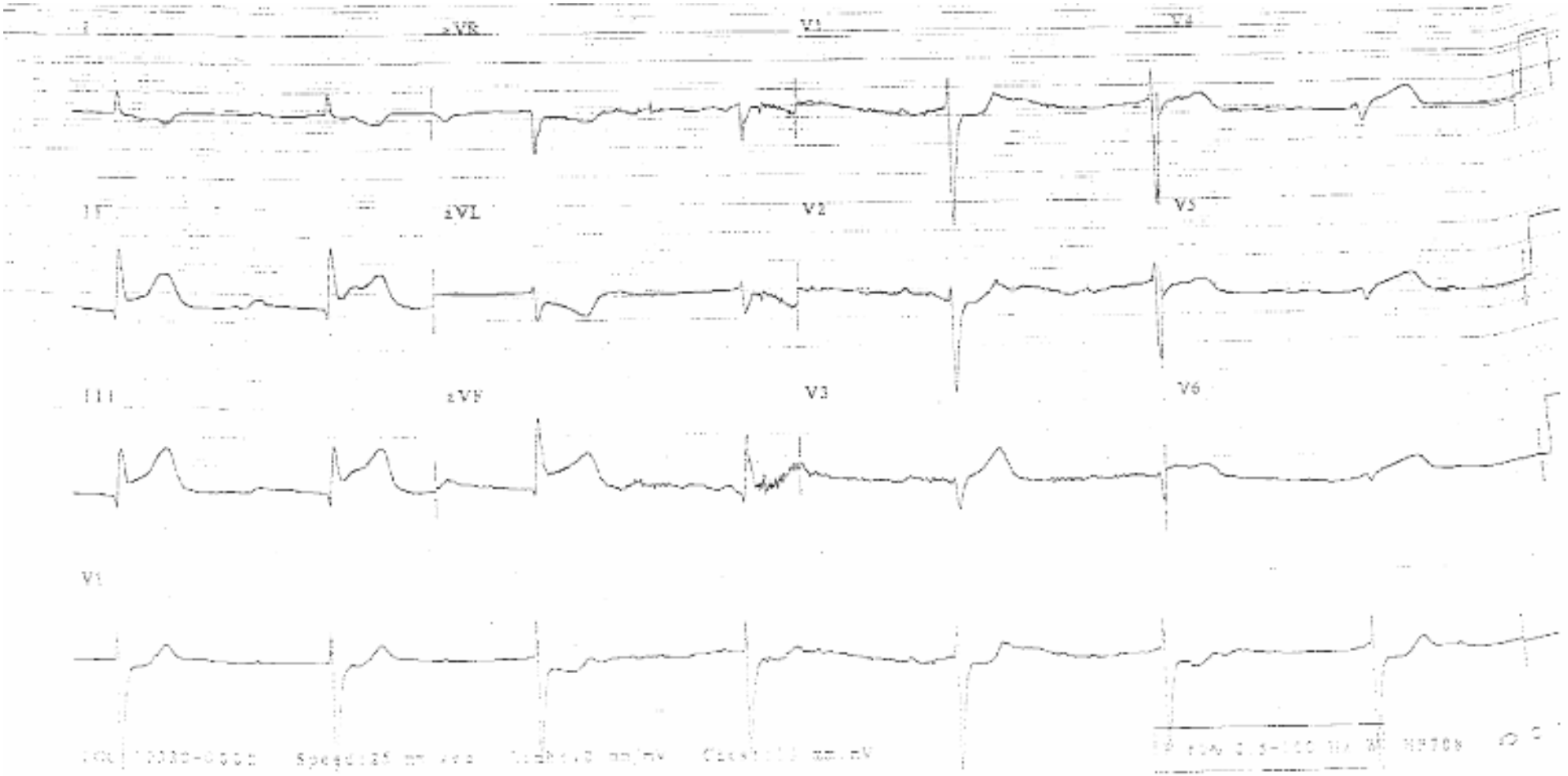
Right Sided EKG



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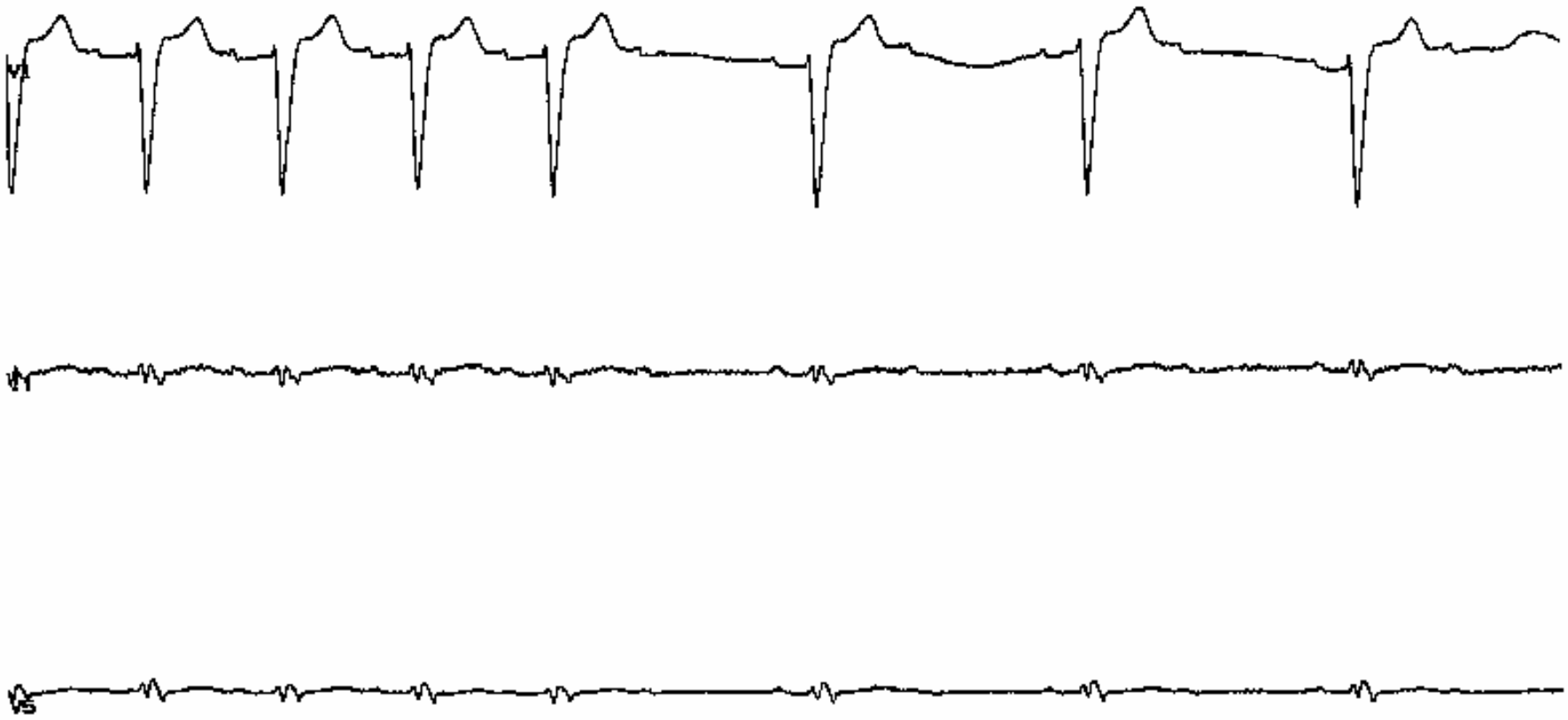
During Cath



http://www.ecglibrary.com/2_1avb.html

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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.

http://www.ecglibrary.com/2_1avb.html

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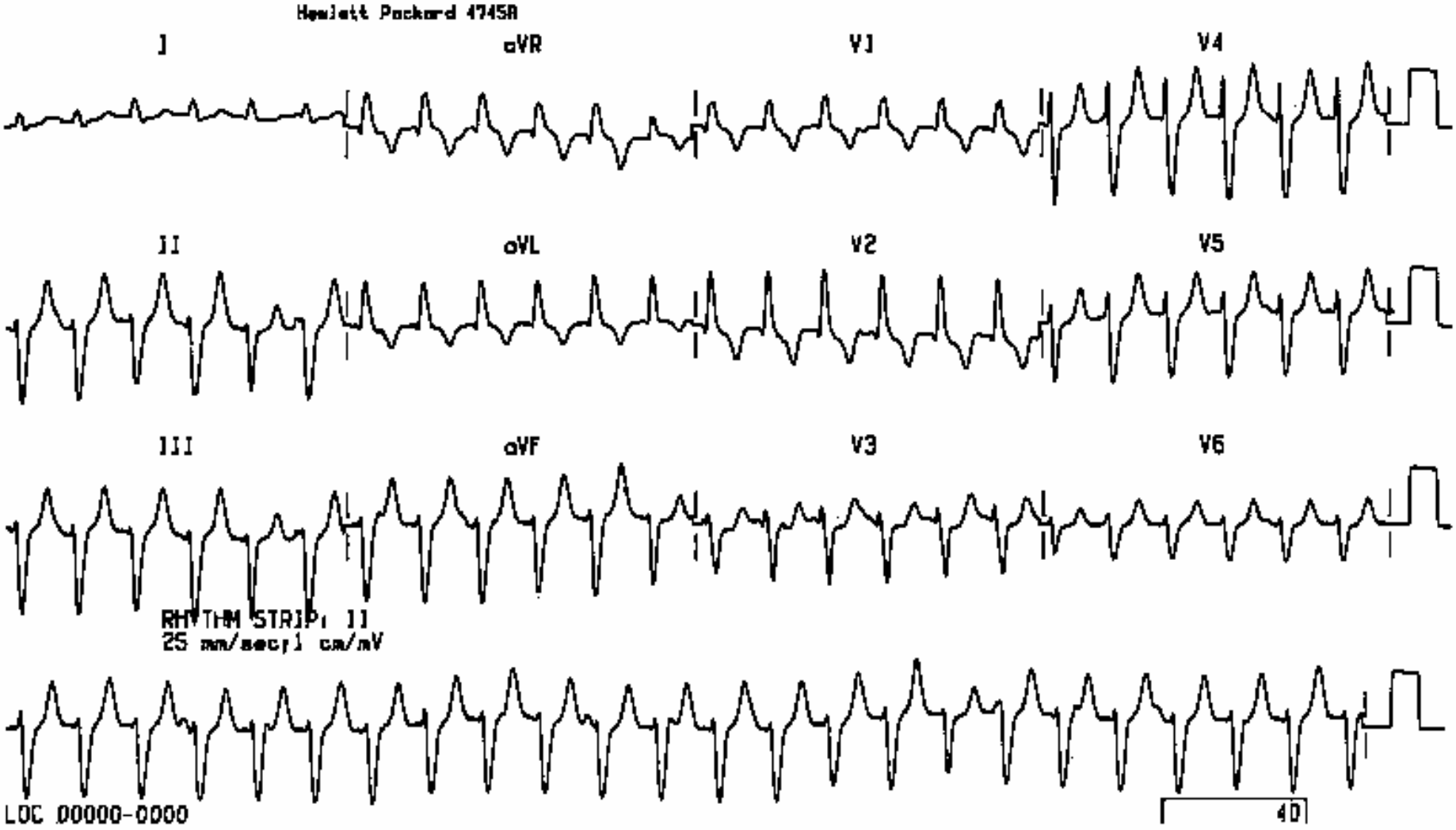
Also During Cath



http://www.ecglibrary.com/vtavd1.html

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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)

http://www.ecglibrary.com/vtavd1.html

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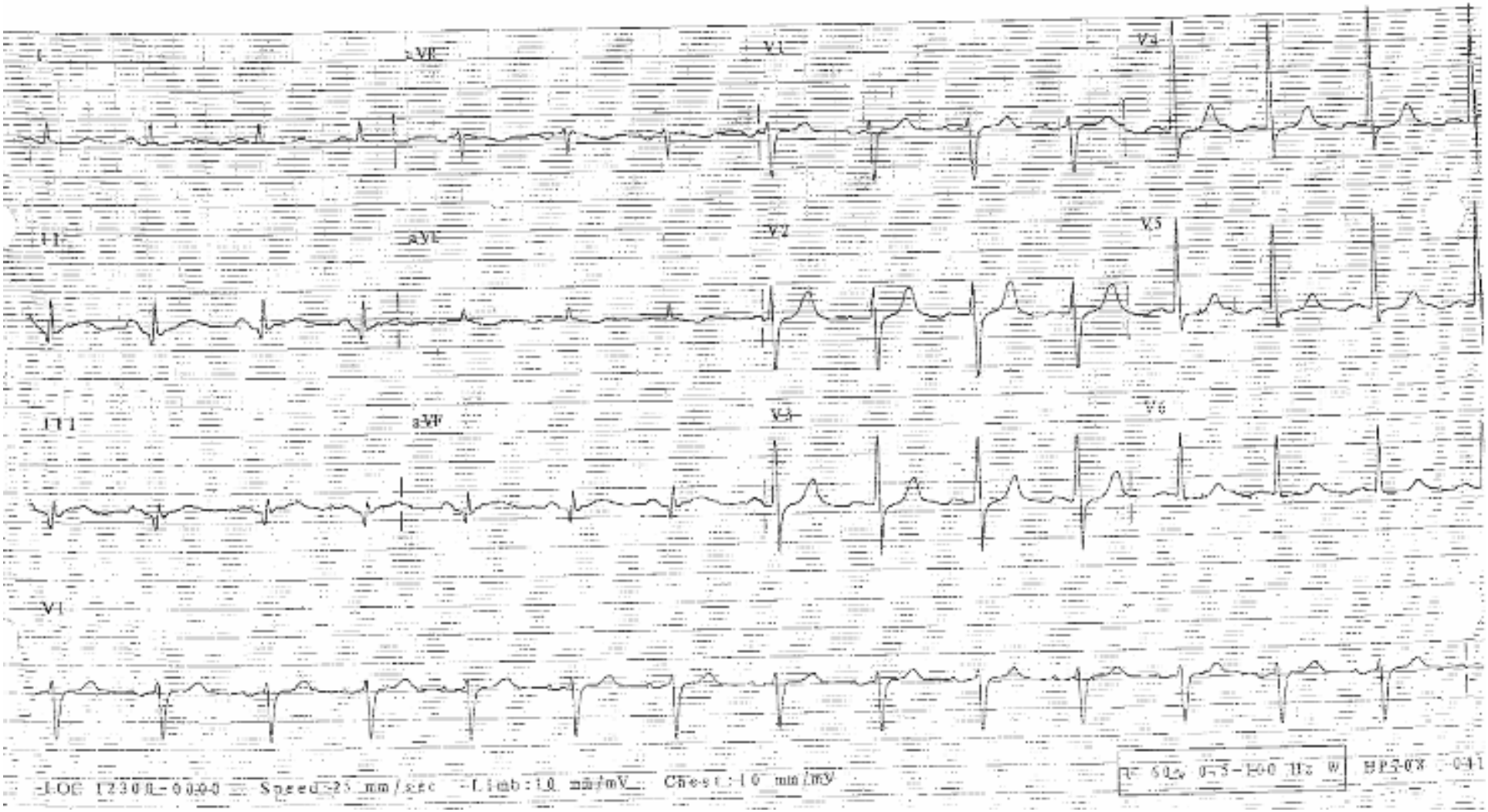
4 hours after admission
About 2 hours post cath



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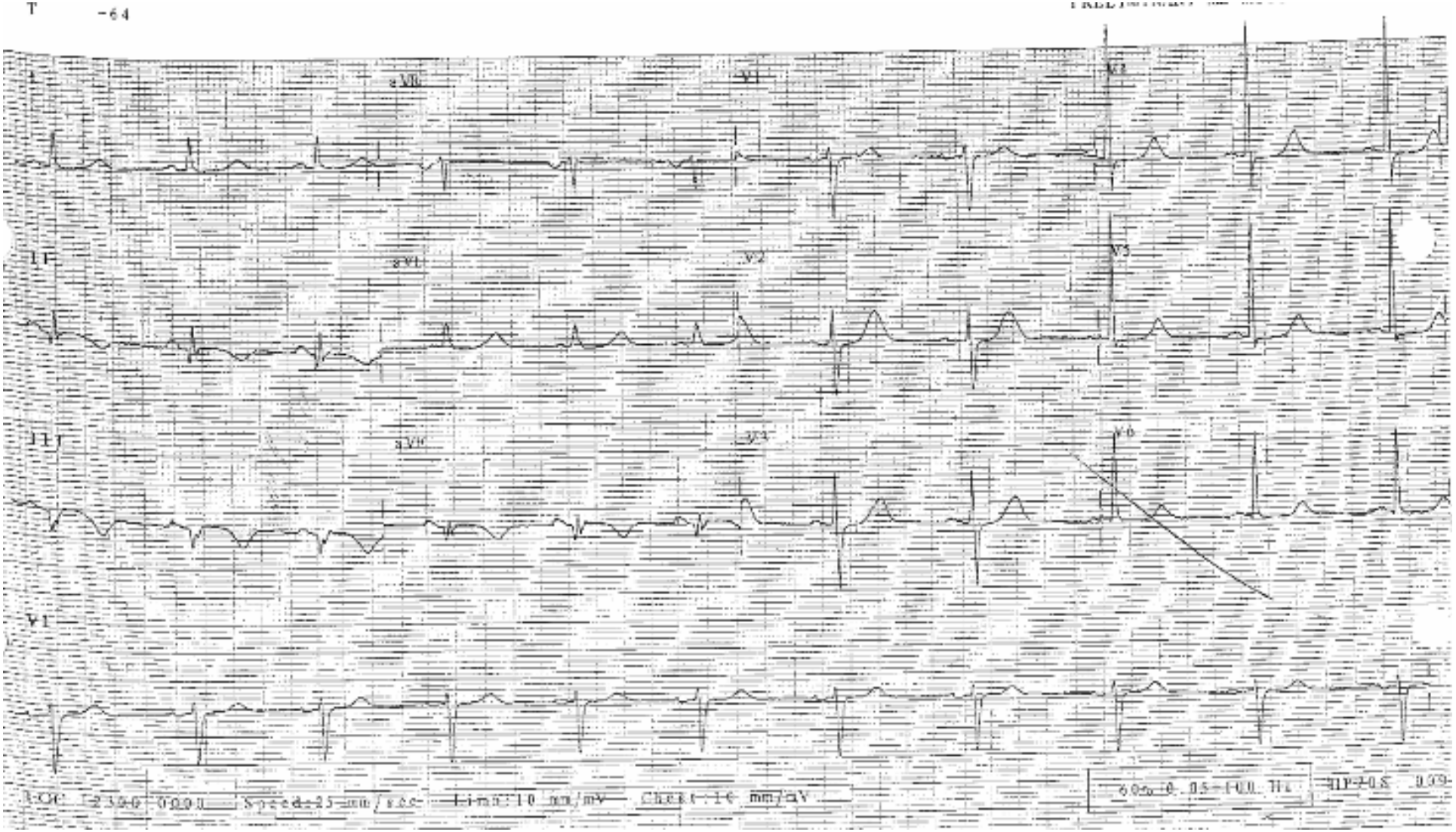
24 hours after admission



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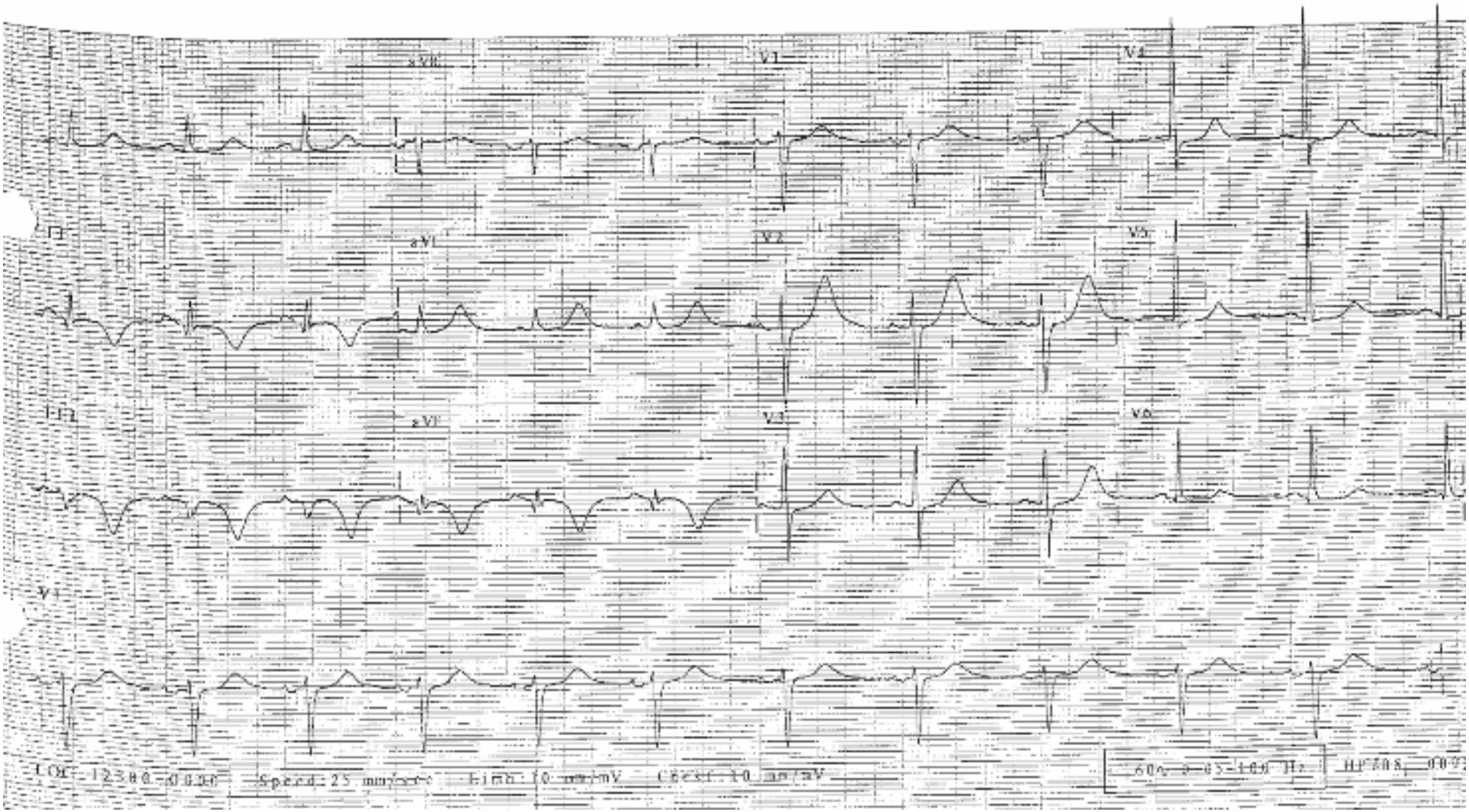
48 hours after admission



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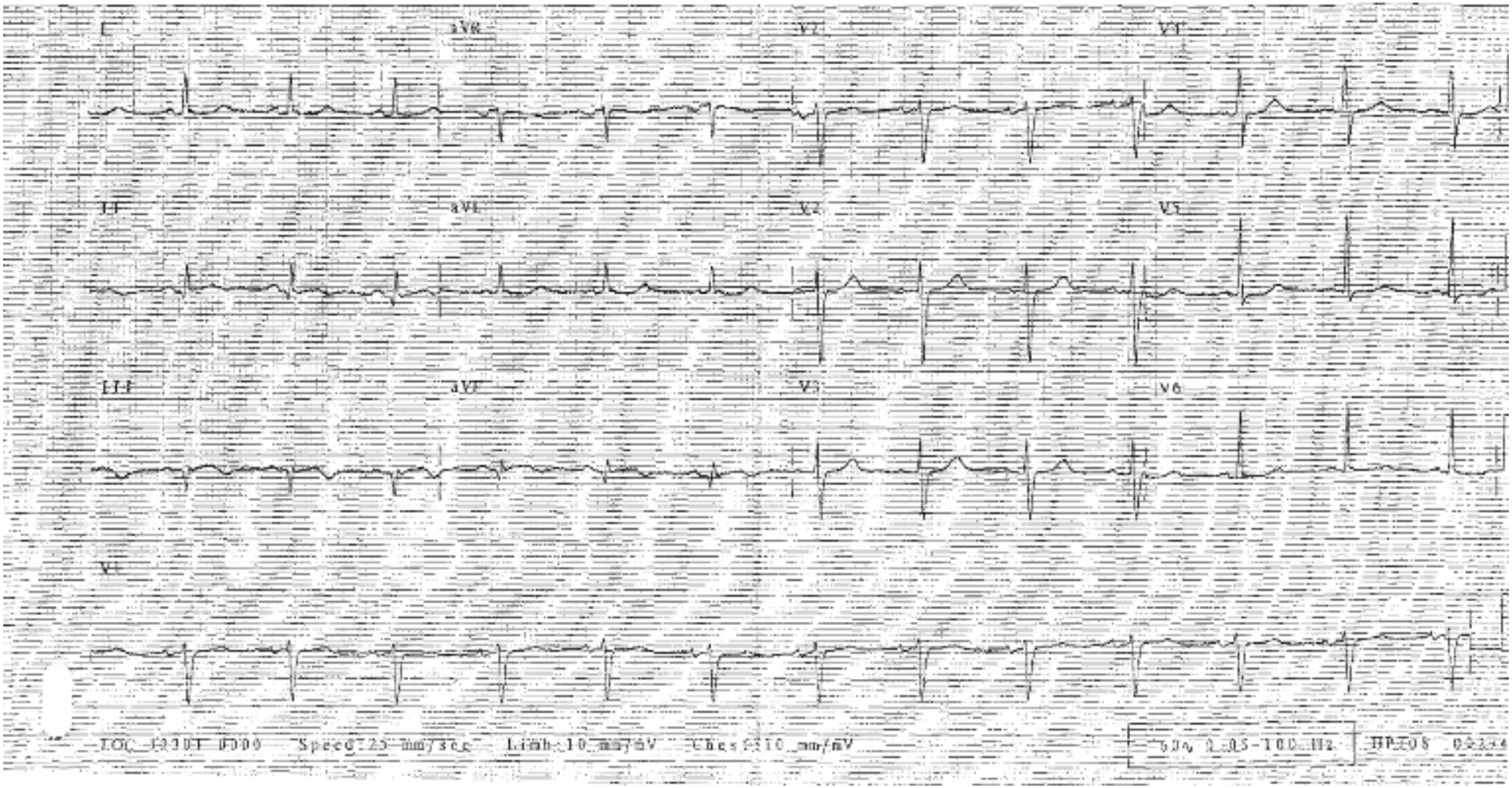
6 days after admission



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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 presure
  - Depressed right ventricular function curve

Braunwald, 1180.

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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

Braunwald, 1180.

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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

Braunwald, 1180.

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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)

Braunwald, 1180-81.

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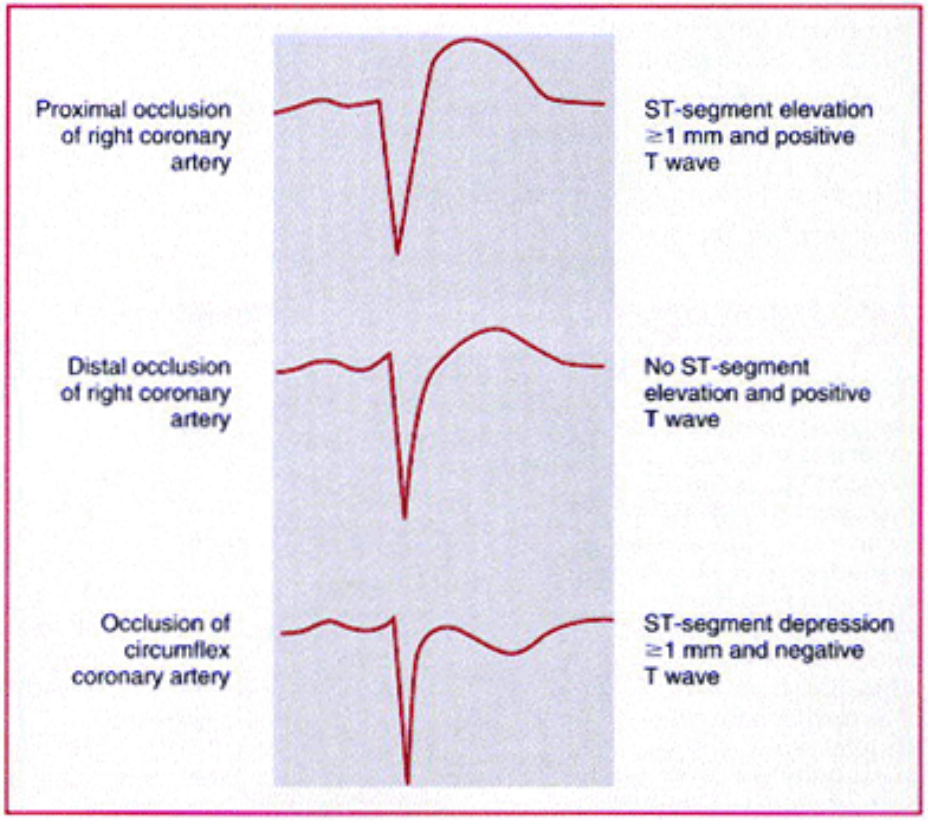
Right Sided EKG (V_4R)

- 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



Braunwald, 1180.
Wellens

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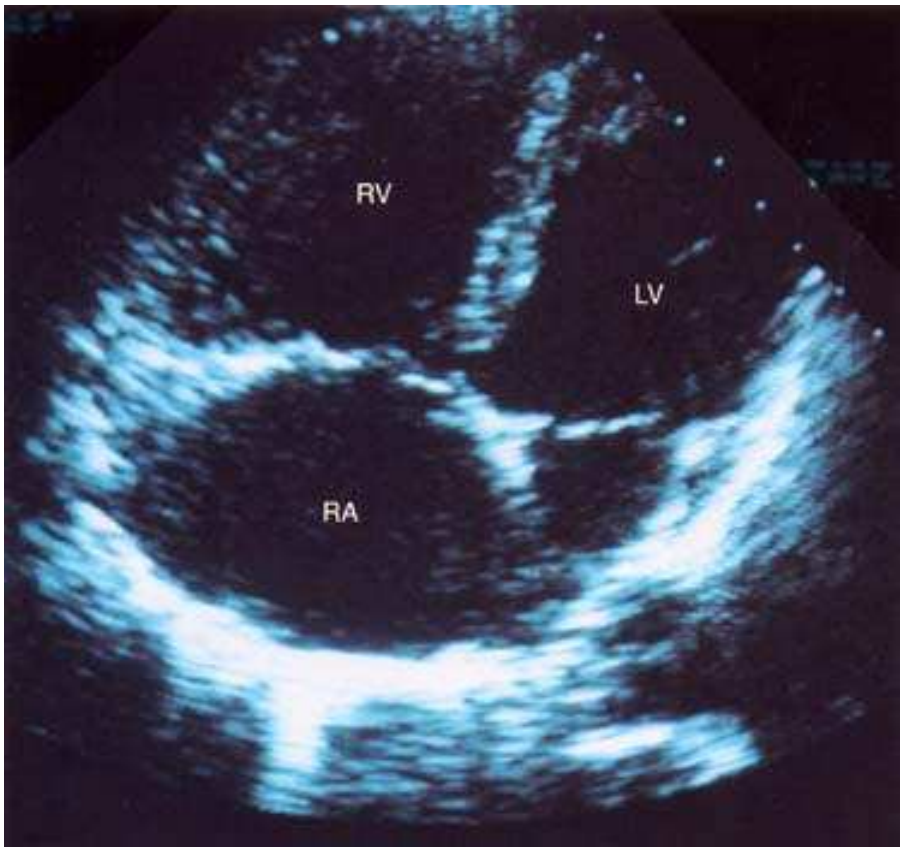
Echocardiography

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



Braunwald, 1181

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Case 2

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

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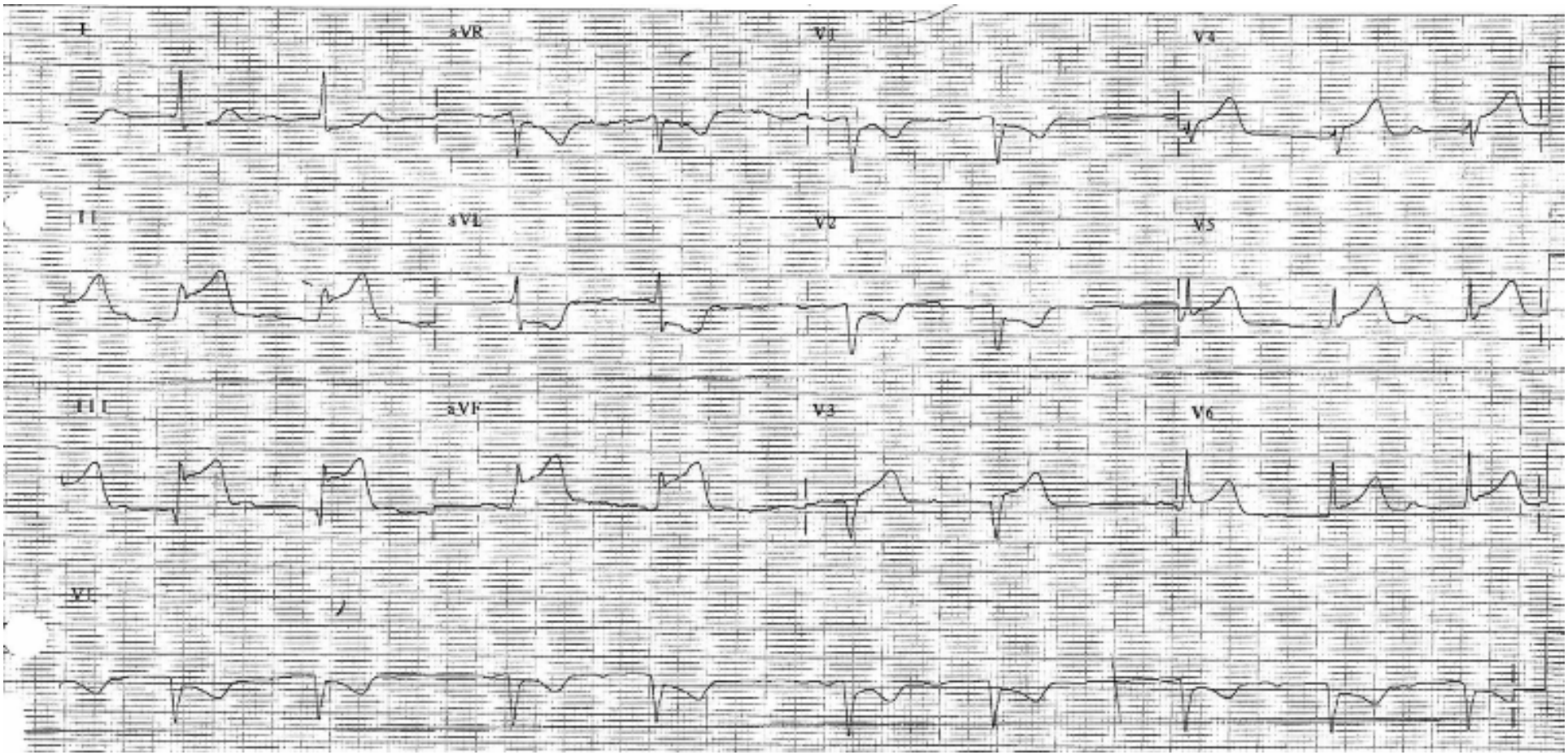
Initial EKG Case 2



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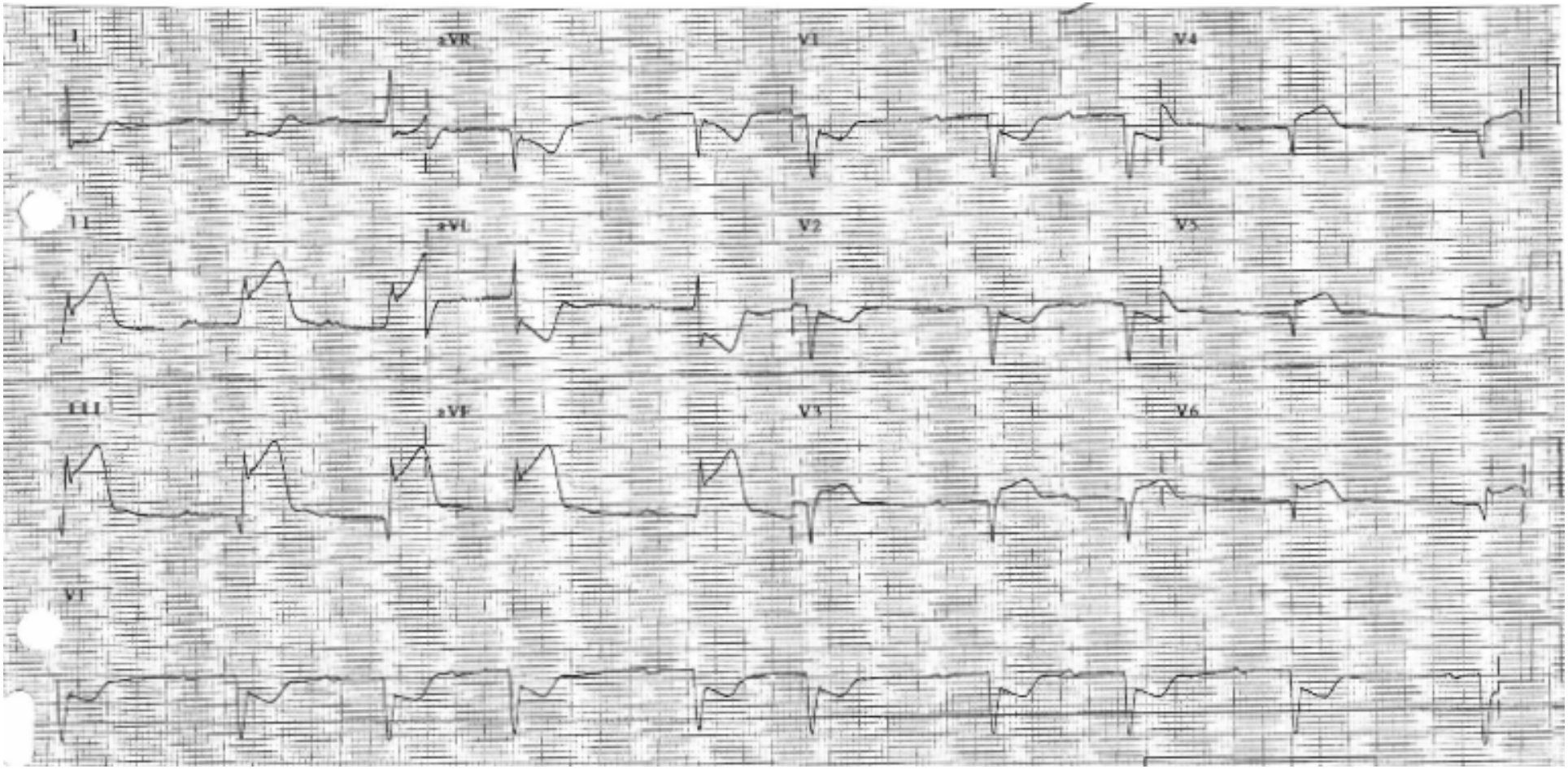
Right Sided EKG Case 2



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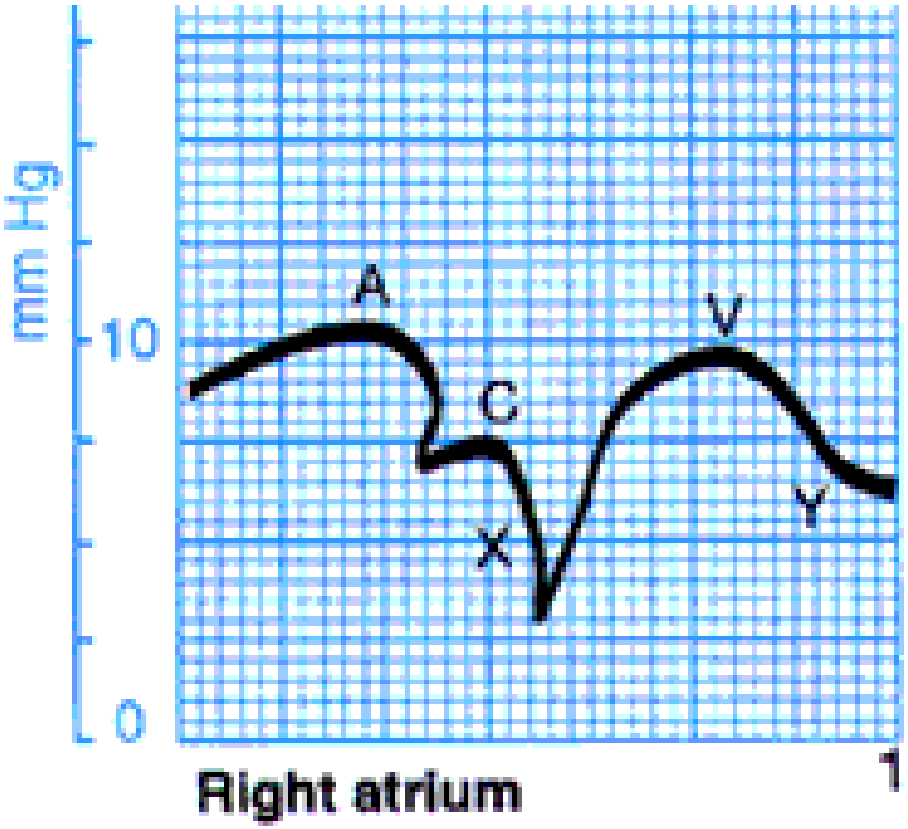
Right Atrial Waveform



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

http://www.staff.vu.edu.au/CriticalCare/Critical%20Care/lecture2_notes.htm

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Hemodynamics of RV infarct

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

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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

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“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

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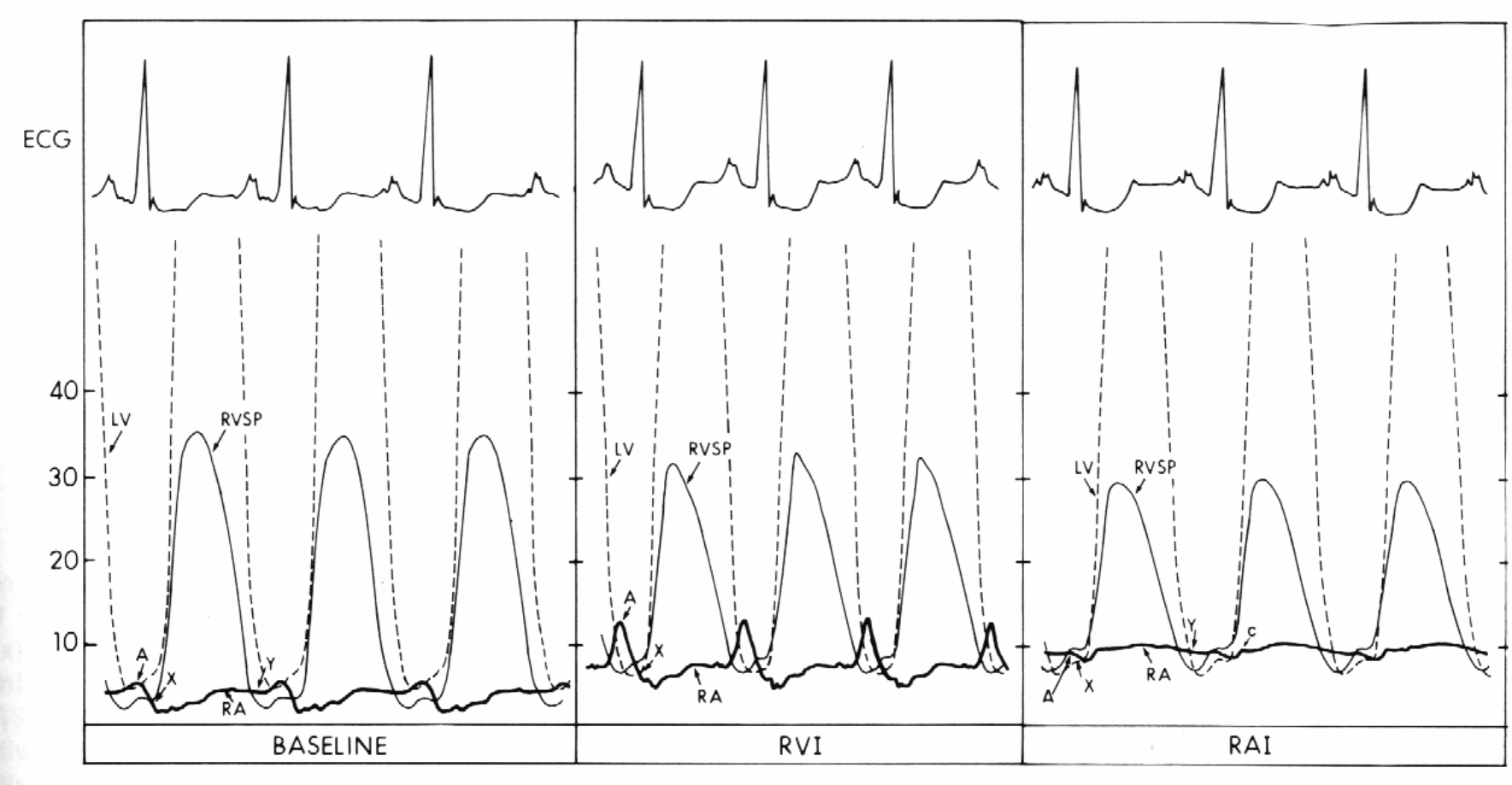
Progression of waveforms



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

Kern, 275

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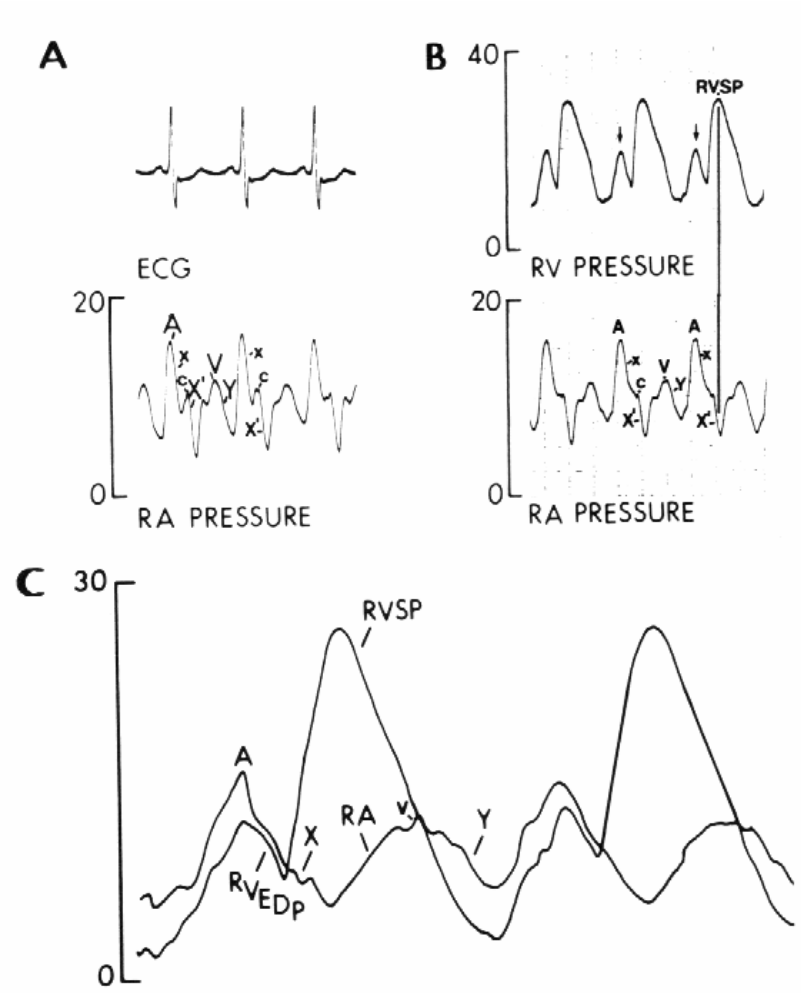
Hemodynamics of RV infarct occlusion distal to RA

- "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



Kern, 276

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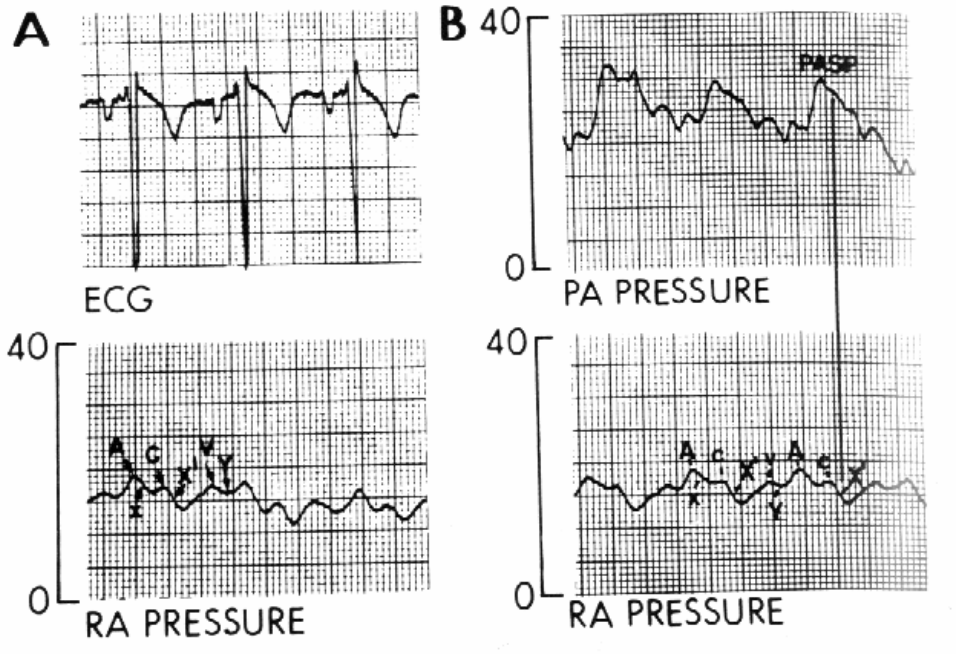
Hemodynamics of RV infarct occlusion proximal to RA



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

Kern, 276

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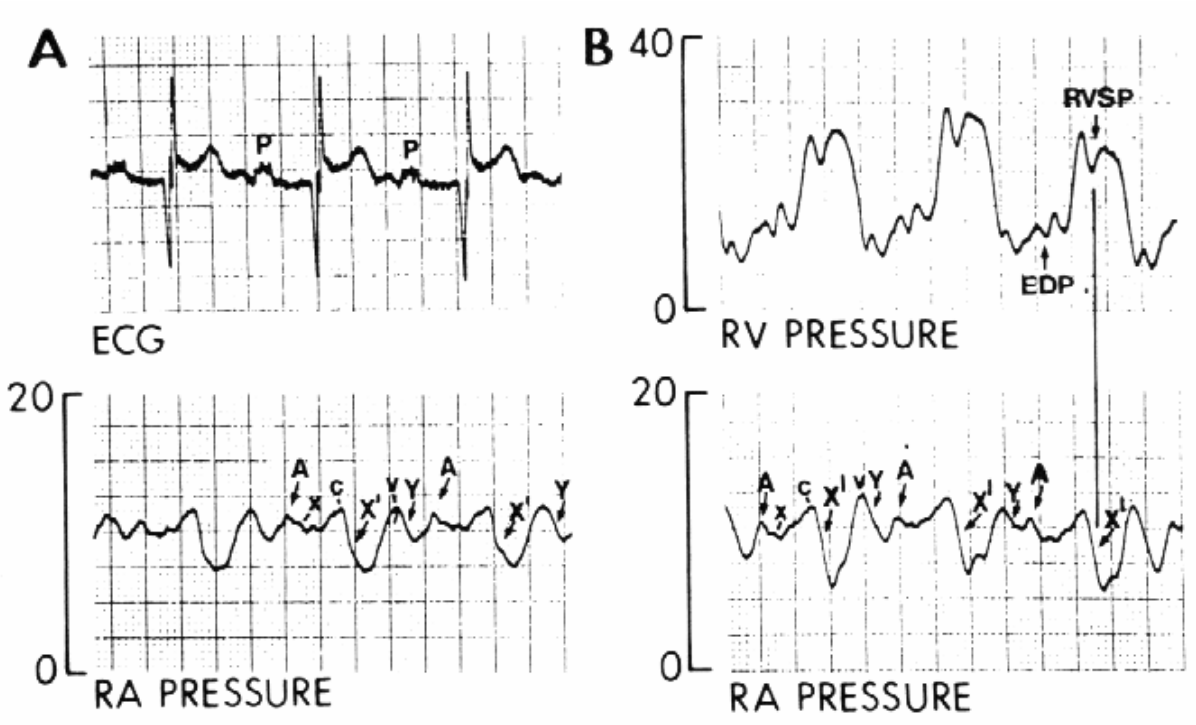
Another “M” pattern



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

Kern, 277

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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)

Braunwald, 1182

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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)

Braunwald, 1182

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Pearls for RV infarction

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

Braunwald, 1180-82

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Case #3

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

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Cath procedure

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

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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

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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

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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

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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**

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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

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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

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What happened

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

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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

Braunwald
Walenga, 262

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Risk factors for HIT

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

Kelton, et al.

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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

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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

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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

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Pathogenesis in a nutshell

- Heparin attaches to Platelet factor 4 (PF4) on platelet surfaces
- IgG Fab antibody binds heparin/PF4 aggregate
  - Fac portion recognizes Fcγ**II**a 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

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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!

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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 thombosis/amputation/death (25.6% vs. 38.8% control)

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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

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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

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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

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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

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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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