Heart Failure with Systolic Dysfunction: A Case and Points

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I have no conflicts of interest related to this presentation or topic.

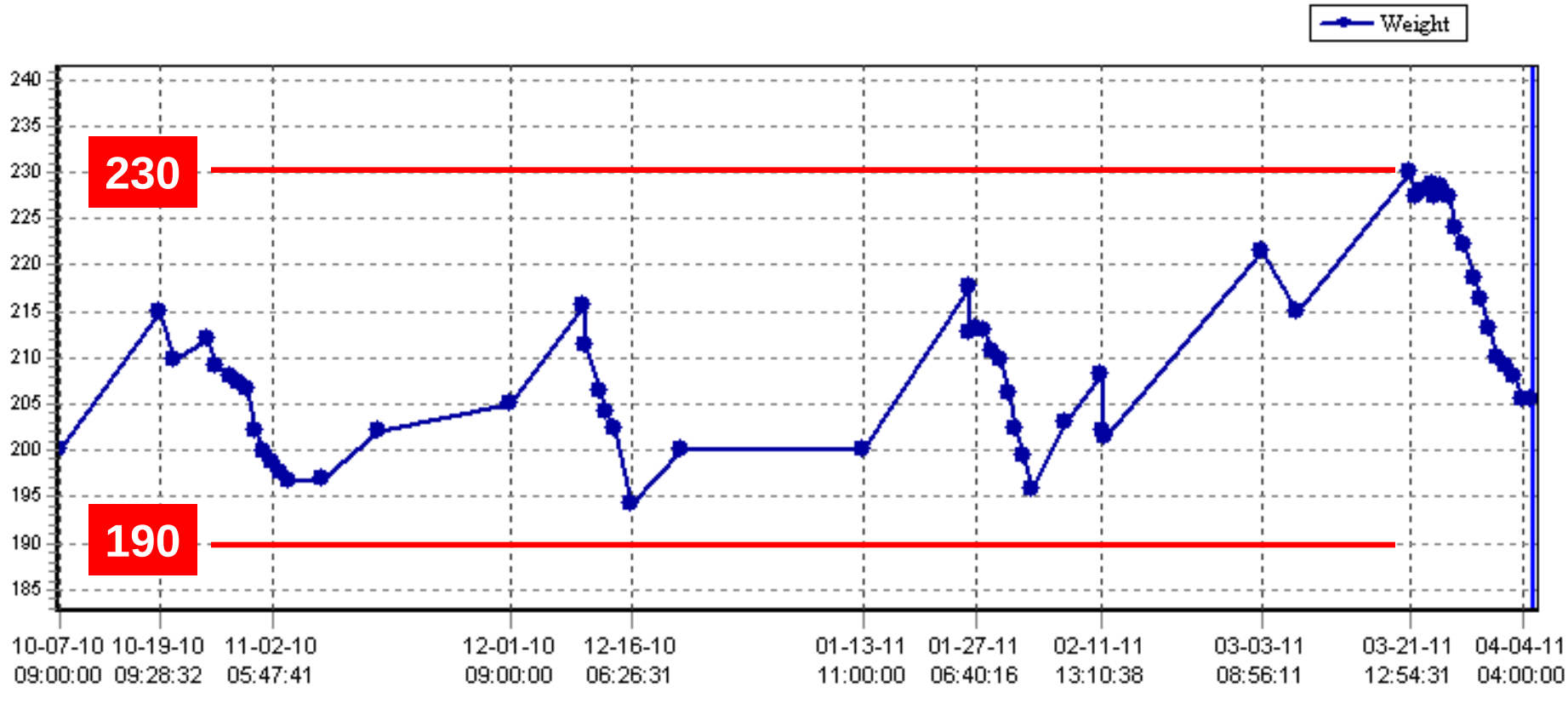
Case Presentation

- 78 yo man admitted with increasing dyspnea, orthopnea, edema, and abdominal pain for one week. Claims medication and dietary compliance. No fever or cough. Meds: ASA 81, Furosemide 120 bid, Lisinopril 5. Wt 230, BP 103/61, R 20, P 78.

Case Presentation - 2

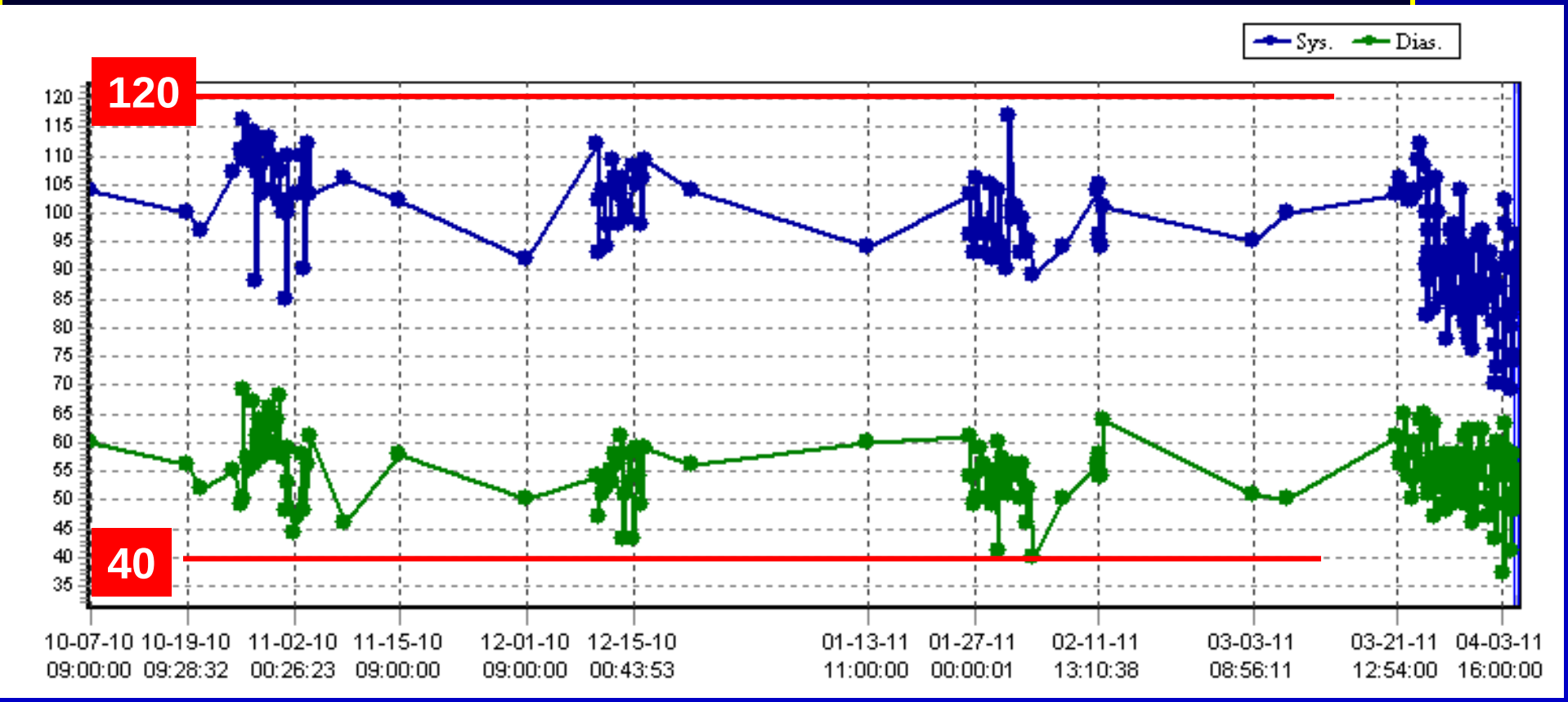
- Hx Hypertension and alcohol use
- 2000 - “systolic dysfunction”
- ICD placed 2003
- SCD-Heft trial participant
- June 2008 EF 19%

- Admissions
  - Nov 12, 2009
  - Apr 27, 2009
  - Nov 4, 2010
  - Dec 16, 2010
  - Feb 03, 2011
  - Feb 12, 2011
  - Apr 13, 2011

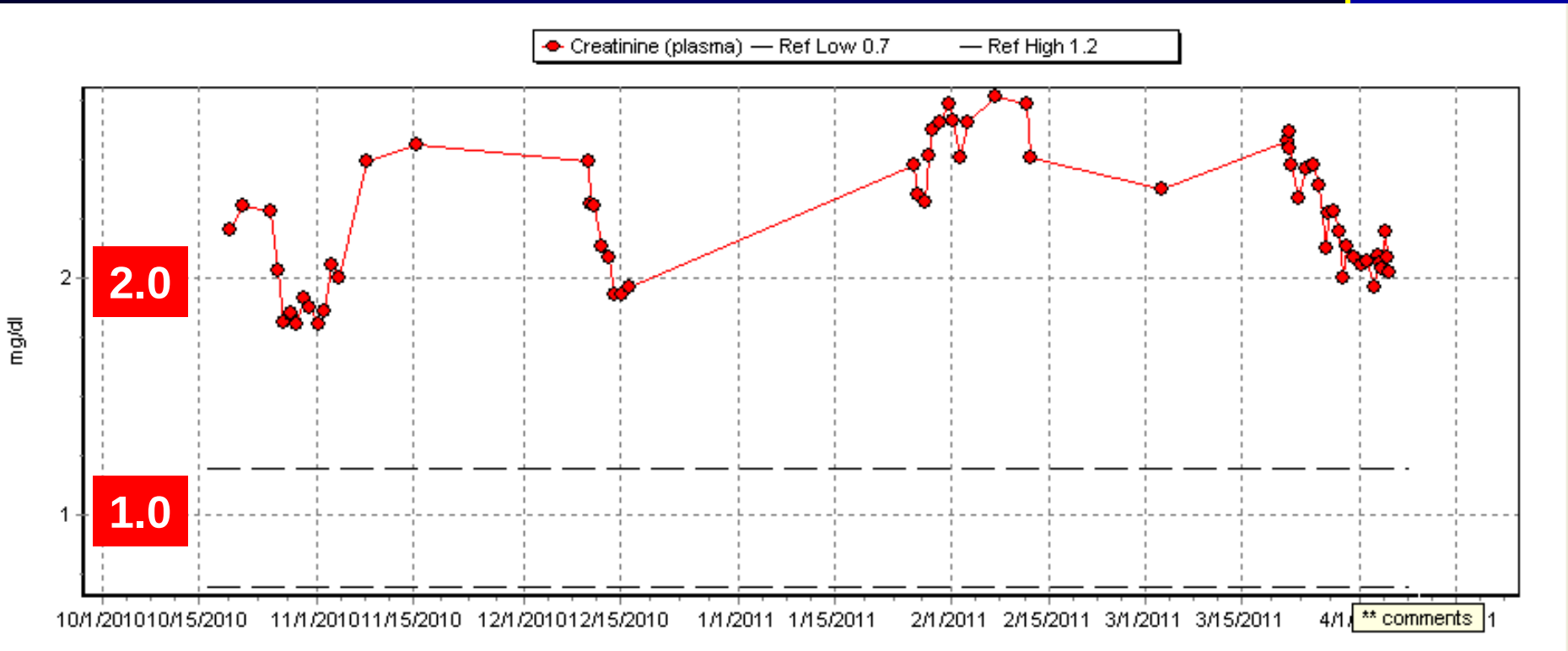


6 Month Weight Trends

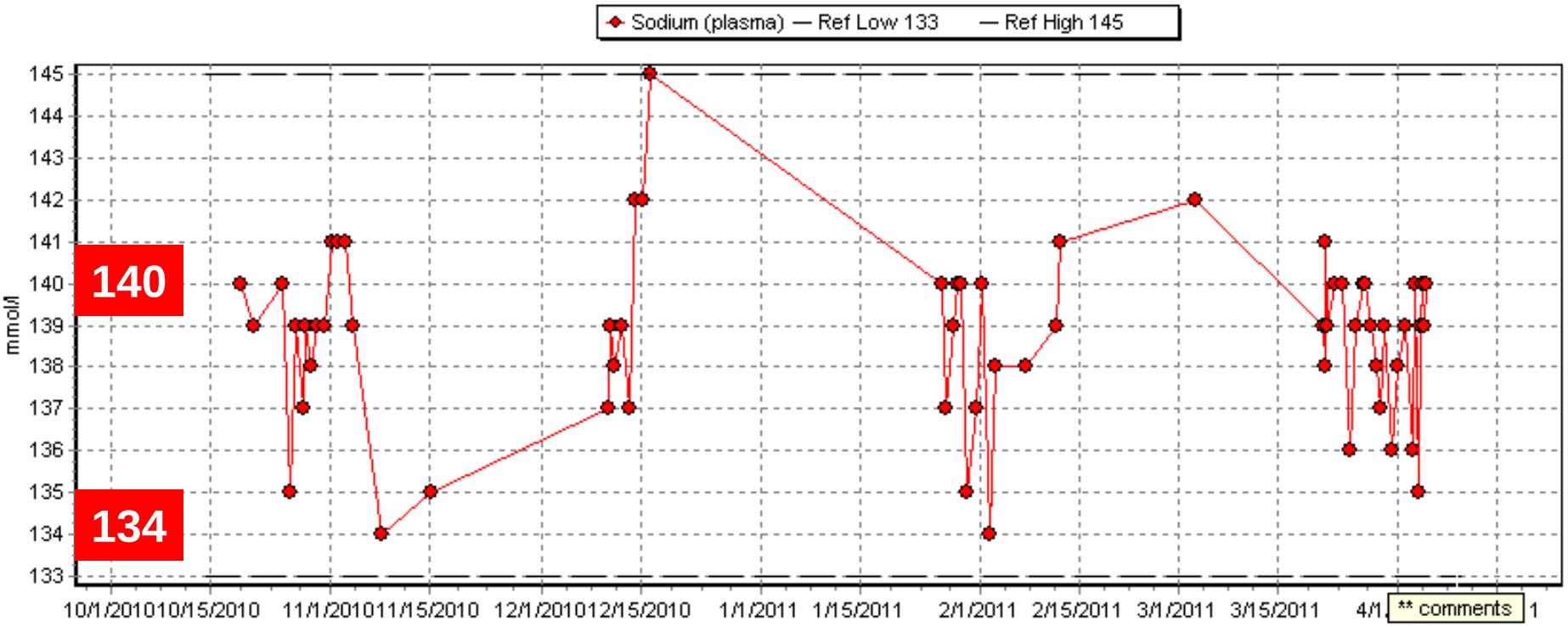
6 Month BP Trends



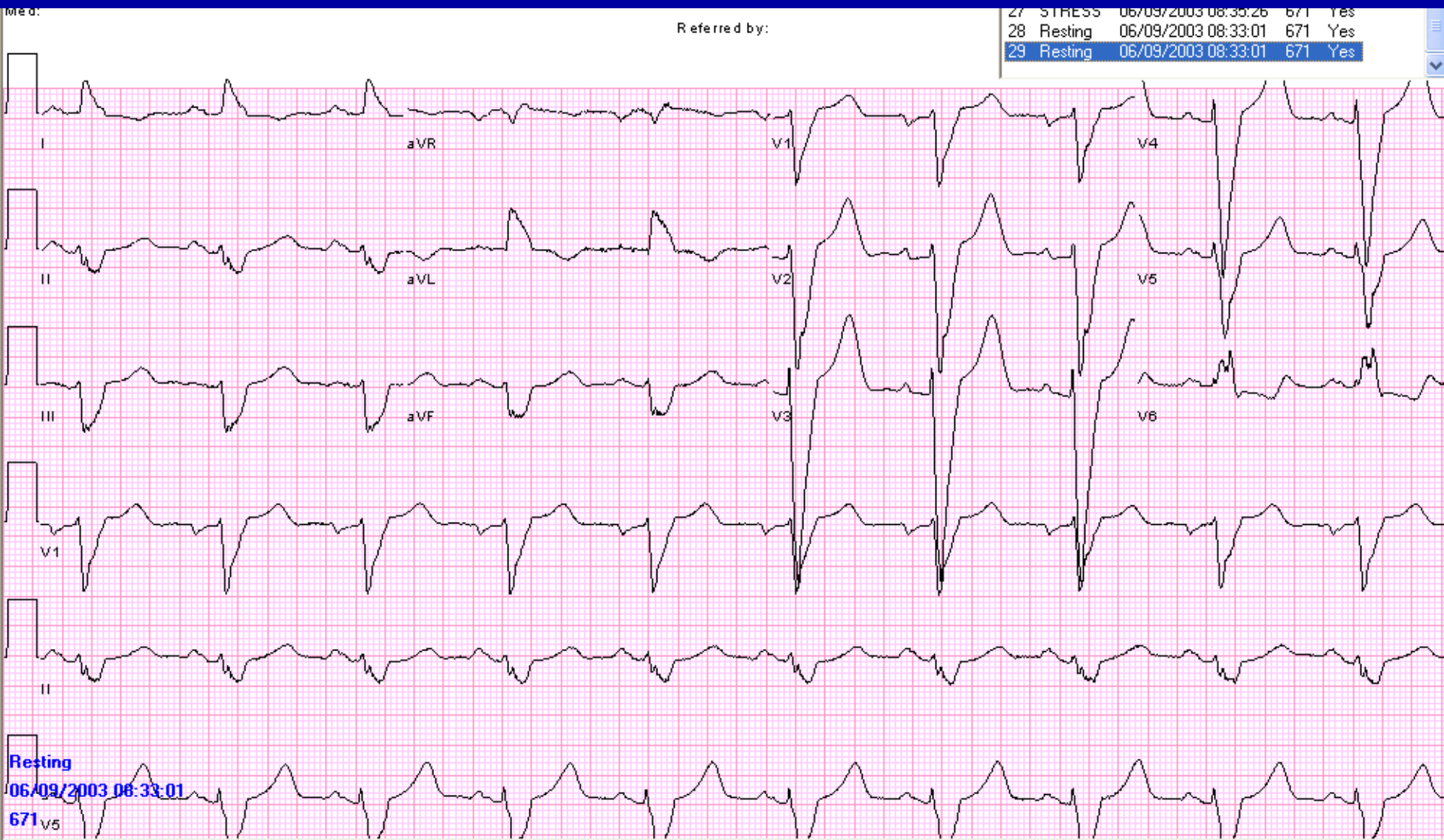
6 Month Creatinine Trends



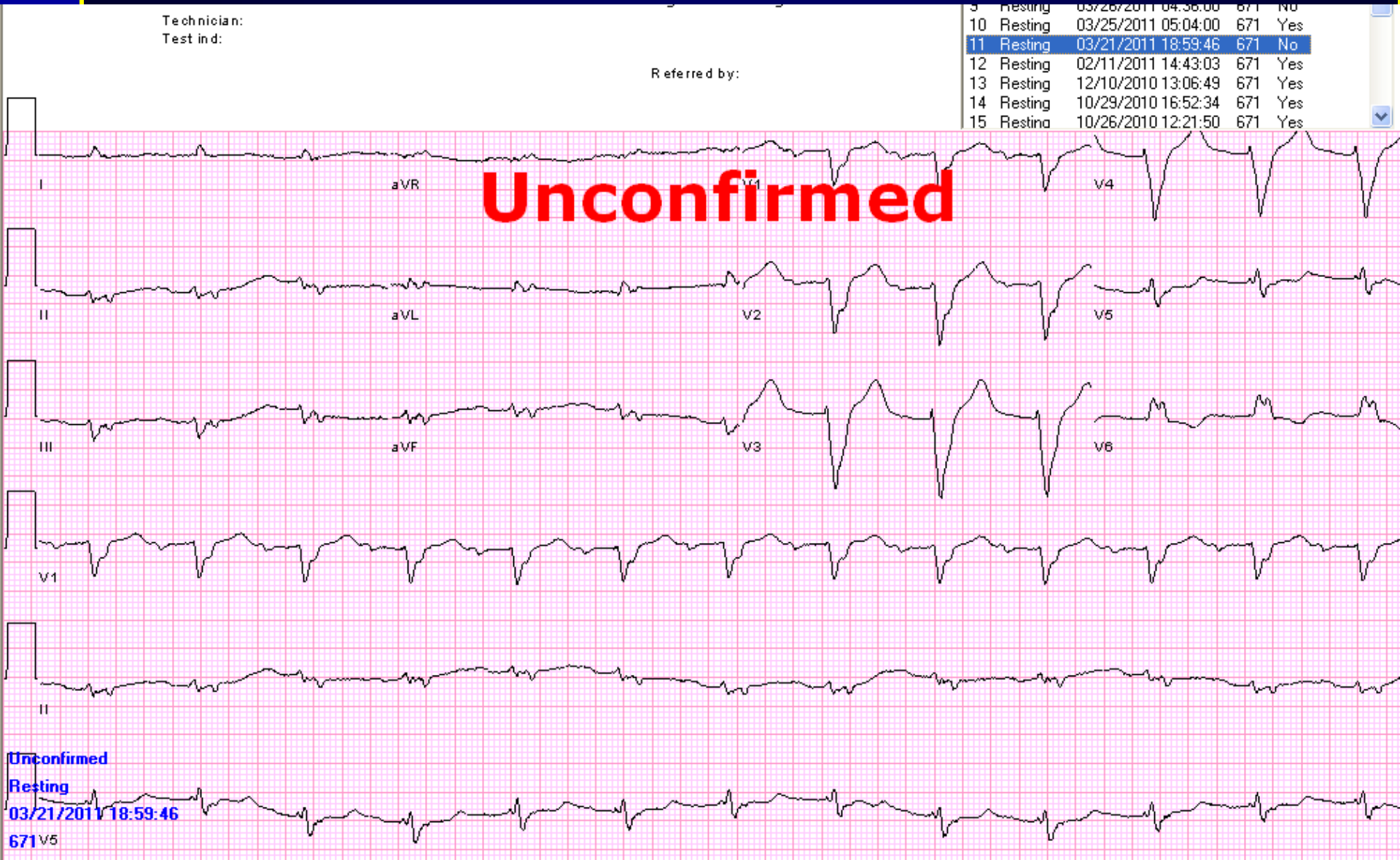
6 Month Sodium Trends



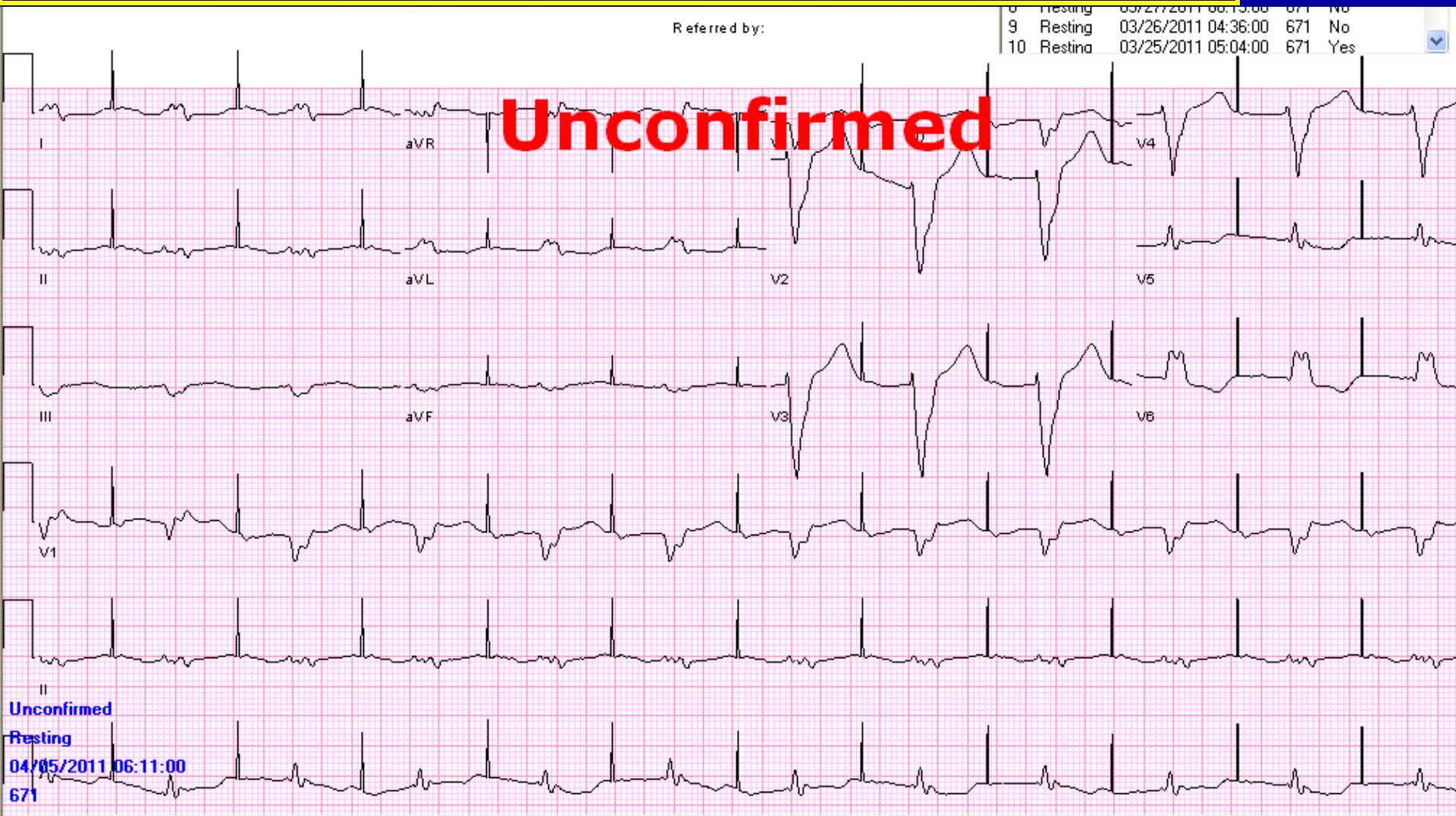
ECG 2003; QRS 0.16 sec

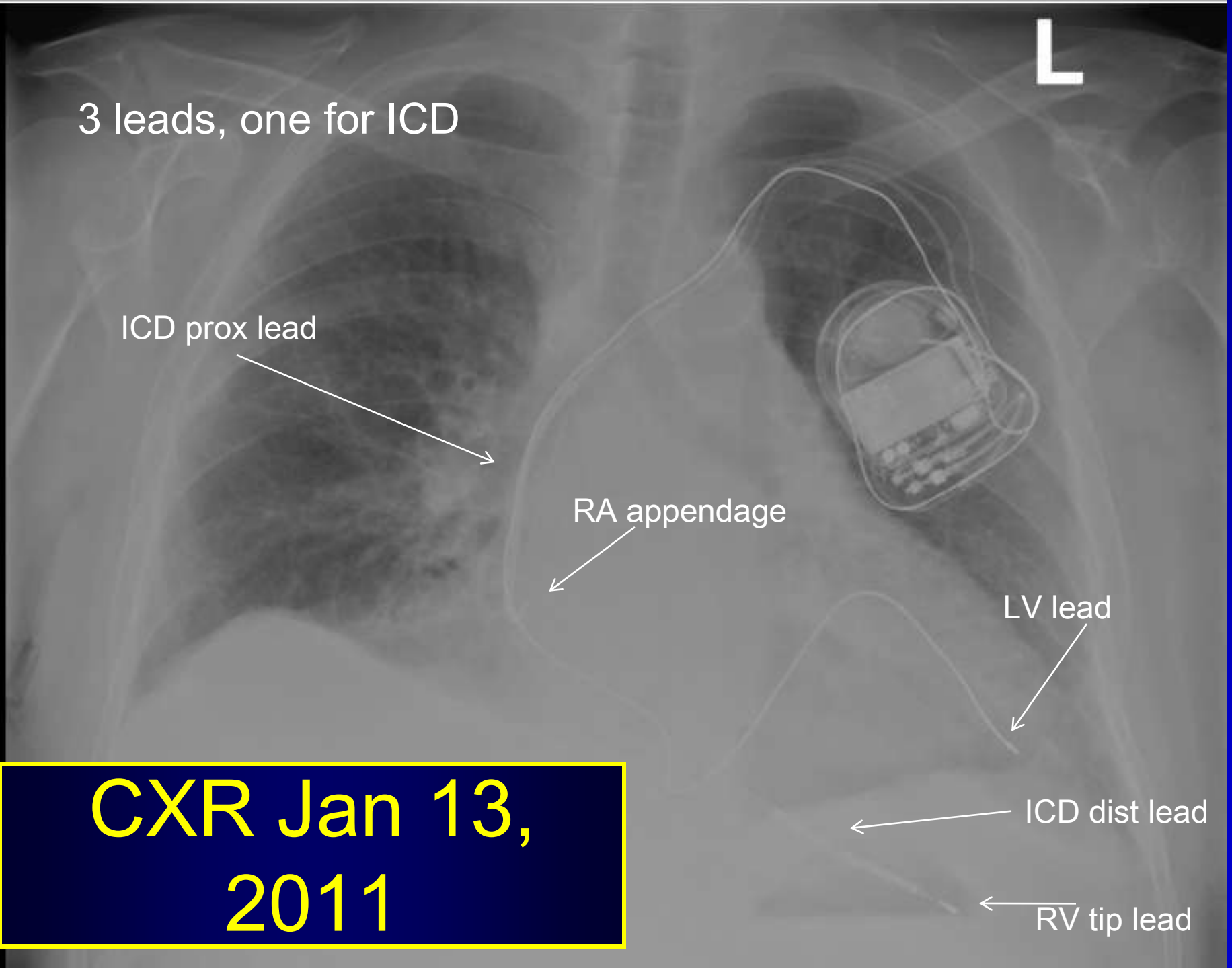


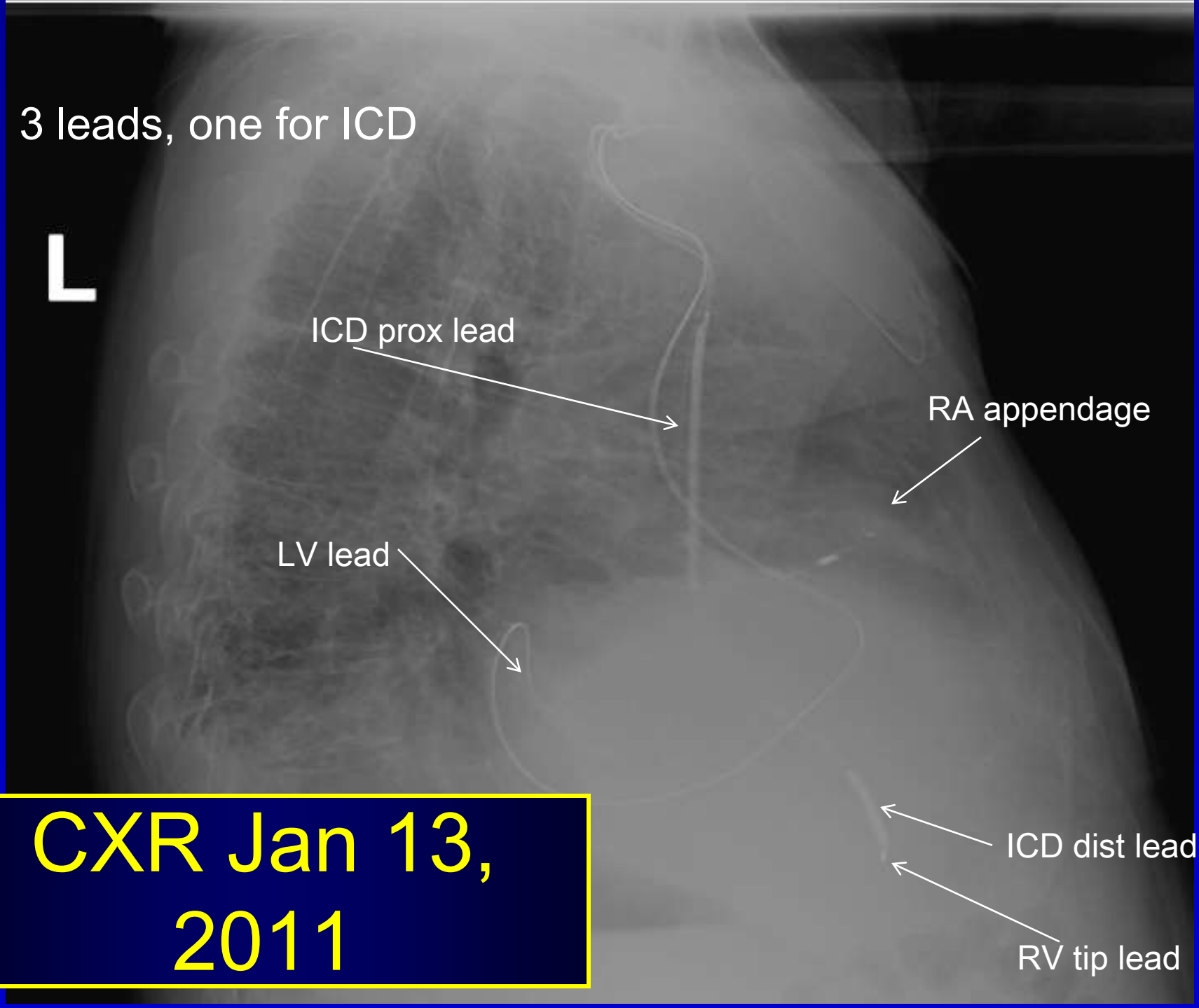
ECG March 21, 2011; QRS 0.17 sec



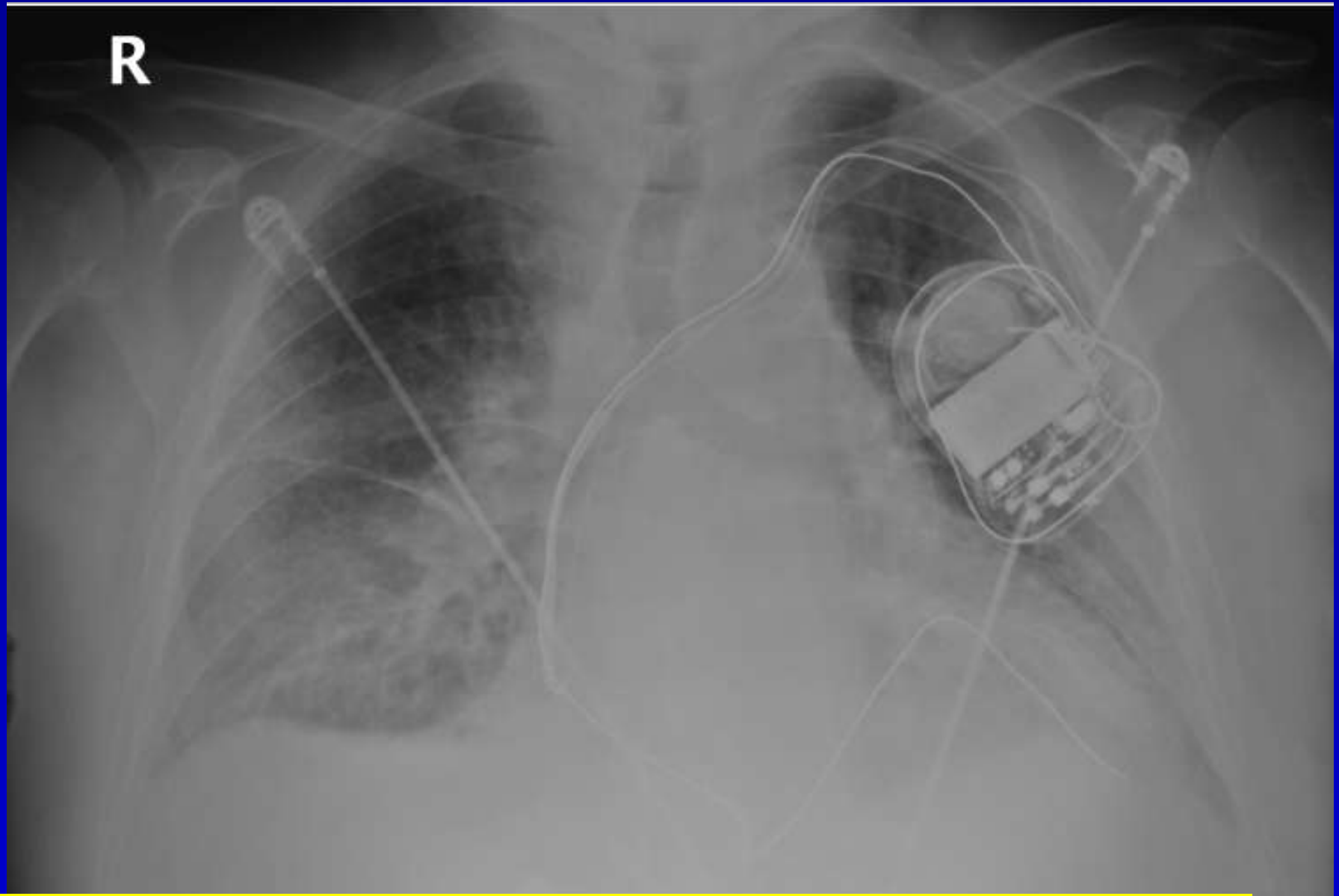
ECG Apr 5, 2011; QRS 0.20 sec




L
3 leads, one for ICD
ICD prox lead
RA appendage
LV lead
ICD dist lead
RV tip lead
CXR Jan 13, 2011



CXR Jan 13, 2011



CXR March 21, 2011

CXR March 25, 2011

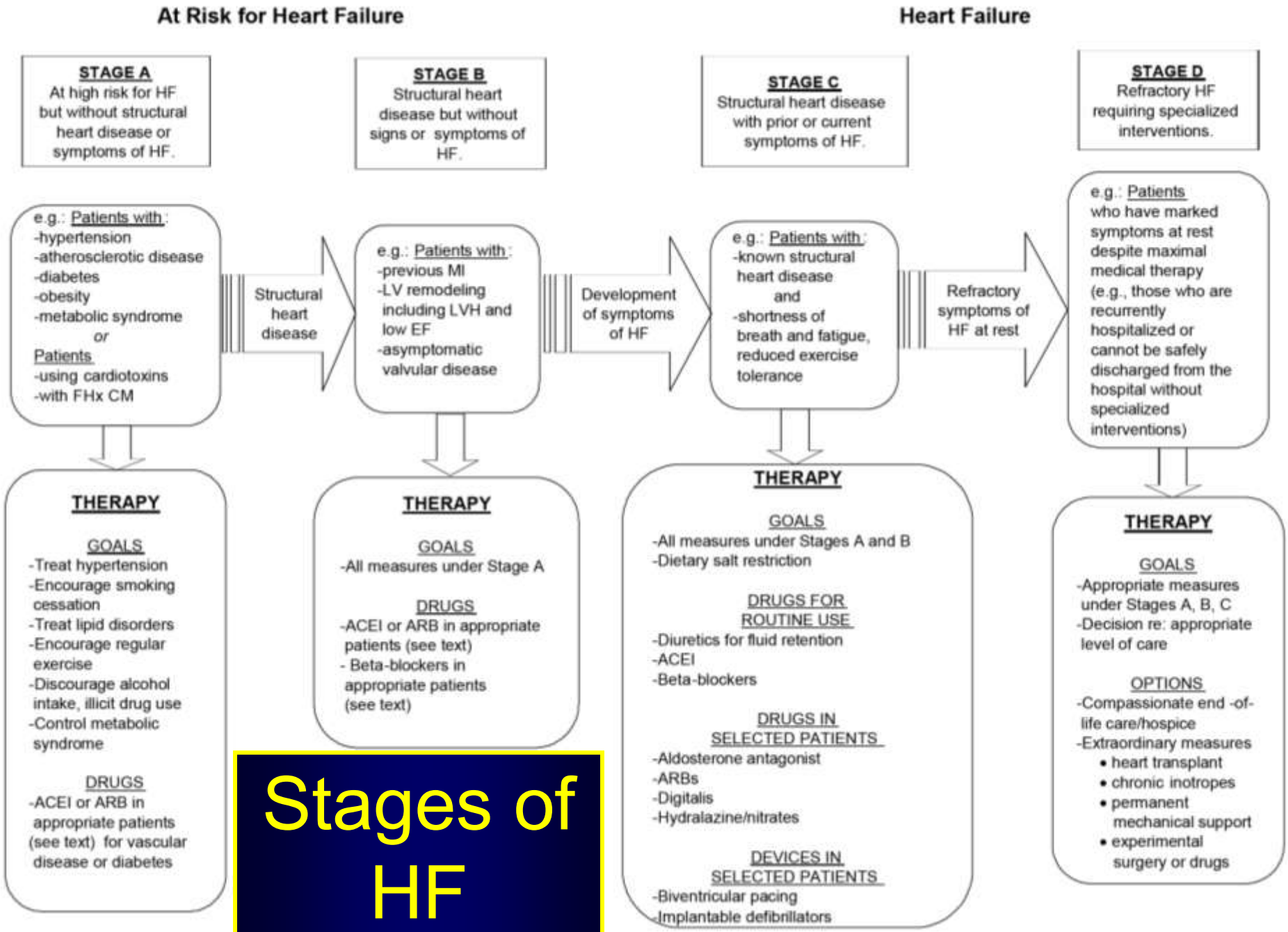
Points from our Patient

- Heart failure can smolder for years
- There are important therapeutic options to help patients with heart failure
- These options have limitations
  - Hypotension
  - Inoperative LV lead
  - Renal insufficiency
- There is plenty of room for progress in management of this clinical problem

Definition: Heart Failure

- Syndrome of dyspnea or exercise intolerance or fluid retention resulting from the inability of the heart to provide output adequate for the needs of the body at a normal filling pressure
- Since some patients may not have fluid overload, the term congestive heart failure is not now favored
- May result from great vessel or pericardial disease or valvular disease, but most are LV dysfunction (systolic or diastolic)

ACC/AHA Guideline update for the diagnosis and management of chronic heart failure in the adult. Update 2009.


At Risk for Heart Failure
Heart Failure
STAGE A
At high risk for HF but without structural heart disease or symptoms of HF.
STAGE B
Structural heart disease but without signs or symptoms of HF.
STAGE C
Structural heart disease with prior or current symptoms of HF.
STAGE D
Refractory HF requiring specialized interventions.
e.g.: Patients with:
-hypertension
-atherosclerotic disease
-diabetes
-obesity
-metabolic syndrome
or
Patients
-using cardiotoxins
-with FHx CM
Structural heart disease
e.g.: Patients with:
-previous MI
-LV remodeling including LVH and low EF
-asymptomatic valvular disease
Development of symptoms of HF
e.g.: Patients with:
-known structural heart disease
and
-shortness of breath and fatigue, reduced exercise tolerance
Refractory symptoms of HF at rest
e.g.: Patients who have marked symptoms at rest despite maximal medical therapy (e.g., those who are recurrently hospitalized or cannot be safely discharged from the hospital without specialized interventions)
THERAPY
GOALS
-Treat hypertension
-Encourage smoking cessation
-Treat lipid disorders
-Encourage regular exercise
-Discourage alcohol intake, illicit drug use
-Control metabolic syndrome
DRUGS
-ACEI or ARB in appropriate patients (see text) for vascular disease or diabetes
THERAPY
GOALS
-All measures under Stage A
DRUGS
-ACEI or ARB in appropriate patients (see text)
- Beta-blockers in appropriate patients (see text)
THERAPY
GOALS
-All measures under Stages A and B
-Dietary salt restriction
DRUGS FOR ROUTINE USE
-Diuretics for fluid retention
-ACEI
-Beta-blockers
DRUGS IN SELECTED PATIENTS
-Aldosterone antagonist
-ARBs
-Digitalis
-Hydralazine/nitrates
DEVICES IN SELECTED PATIENTS
-Biventricular pacing
-Implantable defibrillators
THERAPY
GOALS
-Appropriate measures under Stages A, B, C
-Decision re: appropriate level of care
OPTIONS
-Compassionate end -of-life care/hospice
-Extraordinary measures
• heart transplant
• chronic inotropes
• permanent mechanical support
• experimental surgery or drugs
Stages of HF

Heart Failure in the US

- Leading cause of hospitalization in patients >65 yo
- Accounts for over 5% of health care budget
- 5-year mortality remains >50%

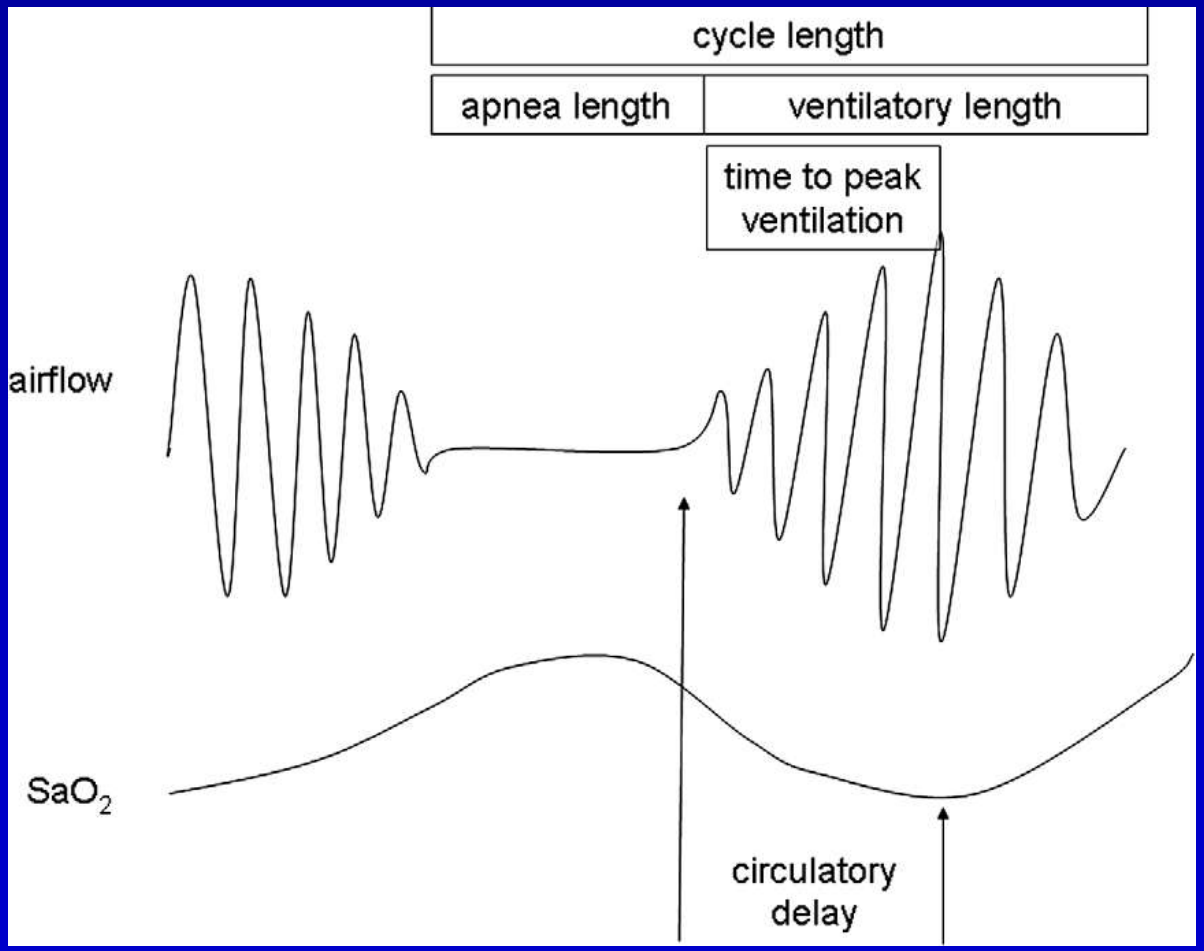
Sharma B et al. Med Clin N Am. 2010; 94:447-464.

Topics in Heart Failure

- Bedside assessment in heart failure
  - Cheyne-Stokes respiration
  - JVP evaluation; S3
- Medical treatments
  - Standard
  - Special
- Device-based therapies
  - ICD, CRT
- The end-stage patient

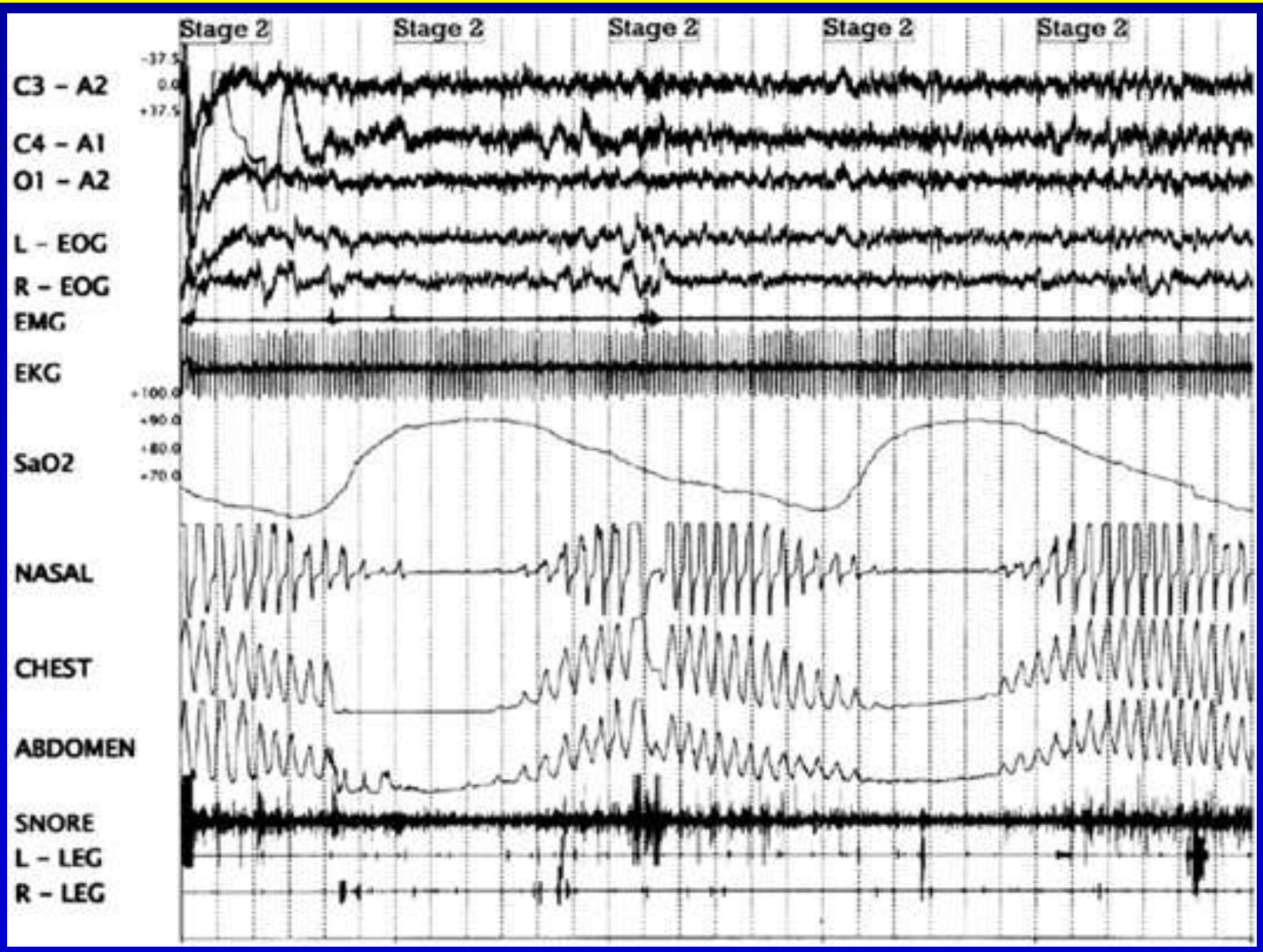
Cheyne Stokes Respiration

- Cheyne Stokes respiration (CSR) is a gradual cyclic alternation between hyperpnea and hypopnea (periodic breathing)



Wedewardt J et al. Sleep Medicine. 2010; 11:137-142.

Polysomnogram in CSR



Sharma B et al. Med Clin N Am. 2010;94:447-464.

Cheyne Stokes Respiration

- Cheyne-Stokes respiration (CSR) is most likely to be seen in patients with CNS disease (damage to respiratory centers) and in patients with heart failure
- In heart failure, a patient may have standard obstructive sleep apnea or CSR or both
- Probably 25-35% of HF patients have CSR and a similar number have OSA

Sharma B et al. Med Clin N Am. 2010;94:447-464.

Physiology of CSR

- The cause of CSR is no longer accepted to be slow circulation time from low cardiac output, rather instability of ventilatory control systems with increased chemoresponsiveness promoting hyperventilation and hypocapnea
- The cycle of hyperpnea and hypopnea varies from about 45 seconds to about 2 minutes
- In general, the worse the LV function (ejection fraction) the longer the cycle

Wedewardt J et al. Sleep Medicine. 2010; 11:137-142.

CSR in Heart Failure

- CSR is an adverse prognostic sign
- CSR is associated with more severe NYHA class, in patients with atrial fibrillation, awake hypocapnia ($PaCO_2$ <36 mm Hg), nocturnal ventricular arrhythmias and LVEF<20%

Treatment of CSR in Heart Failure – No Outcomes Data

- First step, optimize medical therapy, this will reduce severity of CSR
- Treat OSA if present (CPAP may improve CSR but a large study was negative)
- More sophisticated methods of ventilation might be beneficial

- Nocturnal oxygen therapy
- Supplemental CO2 therapy (add dead space)
- Theophylline
- Acetazolamide
- Atrial overdrive pacing
- LV assist device
- Cardiac resynchronization therapy

Assessment of Jugular Venous Pressure

- ACC Guidelines include assessment of jugular venous pressure as recommended in diagnosis and management of patients with known or suspected heart failure
- Elevated JVP is an adverse sign for survival
- Normal JVP is a key criterion for readiness for discharge from hospital from ADHF admission
- MKSAP says physicians are not reliably accurate in estimating jugular venous pressure
- Recommendation: Practice assessing JVP!

S3

Milestones in Heart Failure Treatment

- Bloodletting
- Southey's Tubes
- 1785 – Foxglove - Digitalis
- 1920 – Organomercurial diuretics
- 1958 – Thiazide diuretics
- 1960s – Loop diuretics
- 1967 – Heart transplantation
- 1987 – ACE-I Enalapril in CONSENSUS-1
- 1993-4 – Beta blockade
- 2000 – ARB
- 1999, 2003 – Aldosterone antagonists
- 2002 – ICD therapy (implantable defibrillator)
- 2004 – Hydralazine-nitrate comb'n in African-Americans
- 2005 – CRT therapy (biventricular pacemaker)

"Nearly 70,000 Americans die needlessly each year because they are not given optimal heart failure therapy"

Los Angeles Times, June 6, 2011

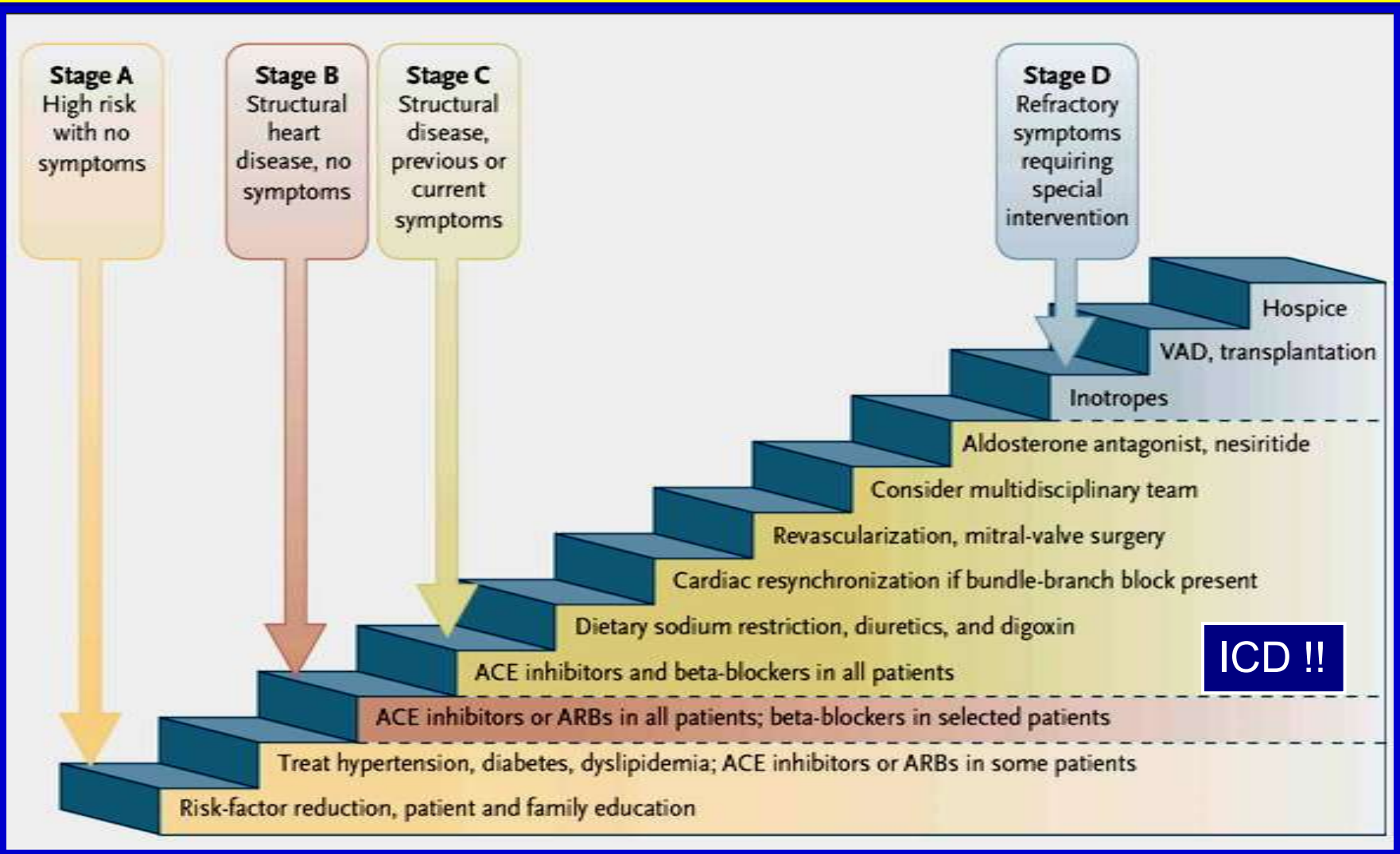
- The estimated number of lives that could be saved by wide implementation of each therapy:
-
  -- Aldosterone antagonists, 21,407.
  -- Beta blockers, 12,922.
  -- Angiotensin-converting enzyme inhibitors, 6,516.
  -- Hydralazine/isosorbide dinitrate, 6,655.
  -- Cardiac resynchronization therapy, 8,317.
  -- Implantable cardioverter-defibrillators, 12,179.

Fonarow GC et al. Am Heart J. 2011;161:1024-30.

Therapy to Improve Outcomes

- Diuretics relieve symptoms (no outcomes data)
- ACE inhibitor improves outcomes
- Beta blocker improves outcomes
- Aldosterone antagonists improve outcomes
- Hydralazine-isosorbide dinitrate improves outcomes
- Cardiac resynchronization improves outcomes
- Implantable cardioverter/defibrillator improves outcomes

Heart Failure Stage and Treatment



Jessup M et al. N Engl J Med. 2003;348:2007; AHA/ACC Guidelines, 2009

Management of Congestion in Heart Failure

- Diuretics are necessary for symptoms of congestion from sodium and water retention – loop diuretics and distal tubular diuretics – diuretic resistance is a problem
- Moderate sodium restriction 3-4 gm/da
- No fluid restriction unless refractory or hyponatremia
- Other options – ultrafiltration, renal replacement therapy

Diuretics in Management of CHF

Table 5. Intravenous Diuretic Medications Useful for the Treatment of Severe Heart Failure

Drug	Initial Dose	Maximum Single Dose
Loop Diuretics		
Bumetanide	1.0 mg	4 to 8 mg
Furosemide	40 mg	160 to 200 mg
Torsemide	10 mg	100 to 200 mg
Thiazide Diuretics		
Chlorothiazide	500 mg	1000 mg
Sequential Nephron Blockade		
Chlorothiazide	500 to 1000 mg (IV) once or twice plus loop diuretics once; multiple doses per day	
Metozalone (as Zaroxolyn or Diulo)	2.5 to 5 mg PO once or twice daily with loop diuretic	
IV Infusions		
Bumetanide	1-mg IV load then 0.5 to 2 mg per hour infusion	
Furosemide	40-mg IV load then 10 to 40 mg per hour infusion	
Torsemide	20-mg IV load then 5 to 20 mg per hour infusion	

IV indicates intravenous; kg, kilograms; mg, milligrams; and PO, by mouth.

ACC/AHA Heart Failure Guideline 2009, p. e25

Advice in Diuretic Use

- Outpatients: goal of 0.5-1.0 kg/da weight loss, goal is to eliminate fluid retention (normal JVP and no edema)
- Observe for electrolyte imbalances or hypotension or azotemia and manage these issues but maintain diuresis “until fluid retention is eliminated, even if this strategy results in mild or moderate decreases in blood pressure or renal function, as long as the patient remains asymptomatic”
- Once euvolemic, maintain diuretic use

ACC/AHA Heart Failure Guideline 2009, p. e25

Mechanisms of Diuretic Resistance

- Increased proximal sodium reabsorption (rarely can use acetazolamide)
- Increased distal sodium reabsorption
  - Distal convoluted tubular hypertrophy and hyperplasia (can use higher dose of loop diuretic or use combination with thiazide such as metolazone)
- Increased collecting duct reabsorption (can use aldosterone antagonists)
- Decreased gastrointestinal diuretic absorption (can use IV furosemide or can use torsemide)
- Hypotension (can use inotropes or alpha agonists as pressors)

NSAIDs

Kazory A et al. Circulation. 2008;117:975.

ACE-Inhibitor in Heart Failure

Trial	Eligibility	Timing of first dose after AMI	Agent and regimen	Average follow-up (months)
SAVE	LVEF <40%	3–16 days	Captopril or placebo 12·5 mg initial dose, up to 25–50 mg three times daily	42
AIRE	Clinical heart failure	3–10 days	Ramipril or placebo 2·5 mg twice daily initial dose, up to 5 mg twice daily for at least 6 months	15
TRACE	Wall motion index <1·2 (LVEF <35%)	3–7 days	Trandolapril or placebo 1 mg daily initial dose, up to 4 mg daily	36
SOLVD treatment	CHF; LVEF ≤35%	>1 month	Enalapril or placebo initial dose 2·5 or 5 mg twice daily up to 10 mg twice daily	41
SOLVD prevention	No treatment for CHF; LVEF ≤35%	>1 month	Enalapril or placebo initial dose 2·5 or 5 mg twice daily up to 10 mg twice daily	37

AMI=acute myocardial infarction; LVEF=left-ventricular ejection fraction; CHF=congestive heart failure. All trials were double blind.

Table 1: **Key design features of trials**

- ACE – inhibitor in heart failure reduces mortality and morbidity and increases LVEF

Flather MD et al. Lancet 2000;355:1575.

ACE Inhibitors in HF Management

- Function: inhibit renin-angiotensin, potentiate kinin and prostaglandin (from kinin)
- Effects: improve symptoms and reduce hospitalization and death
- Populations that benefit: CHD and IDCM, mild mod or severe sx, BP> 90, Cr<2.5
- Use in most, usually with beta blocker, and sometimes with diuretic

ACE Inhibitors in HF Management - 2

- Avoid in prior angioedema or anuric RF, pregnancy
- Caution in SBP<80, Cr>3.0, bilateral renal art stenosis, K>5.5, or near cardiogenic shock
- Prefer agents with published outcome studies: captopril, enalapril, lisinopril, perindopril, trandolapril, ramapril
- Start therapy at low dose, check electrolytes in 1-2 weeks
- Response: maybe in 2 days, but usually weeks to months; withdrawal may result in deterioration

ACE Inhibitors in HF Management - 3

- Unstable patients – ACE-I may be adverse, antagonizing natriuretic response to diuretics from hypotension, and antagonizing pressor effects of inotropes, may require temporary interruption of ACE-I until patient stabilizes
- A reported adverse interaction with aspirin is small: some ignore it, some use no aspirin, some use clopidogrel, I ignore it and use aspirin
- Hypotension is a problem only if postural symptoms, syncope, blurred vision, or worsening renal function

ACE Inhibitors in HF Management - 4

- Worsening renal function is more likely in patients with severe HF (15-30% will increase Cr by >0.3) or in bilateral renal artery stenosis and with concomitant NSAID (avoid NSAID)
- Hyperkalemia in deteriorating renal function or concomitant K replacement or K-sparing diuretics, esp in DM patients
- Cough in 5-10% (50% if Chinese), stops in 1-2 weeks after cessation of ACE-I and returns in 1-2 days on rechallenge – exclude HF exacerbation as cause, encourage patients to tolerate cough
- Angioedema in 1% (more if black) and life-threatening, don't rechallenge

Effects of Beta Blockade in HF

- Improve HF symptoms (should also use in asymptomatic LV systolic dysfunction)
  - Improve clinical status
  - Enhance sense of well-being
- Reduce death or hospitalization
- Patients:
  - With or without CAD
  - With or without DM
- Cause reverse remodeling after 1 month, continuing improvement for 12 months
  - Increase in LVEF by 5-10% or more
  - Decrease in LV diastolic volume

ACC/AHA Heart Failure Guideline 2009, p. e420

Selection of Patients for Beta Blockade in HF

- SBP>85 or 80, HR>65
- Stable heart failure with systolic dysfunction (EF<45%), likely also beneficial in diastolic HF
  - Not in ICU, no recent IV inotropes, but OK predischarge
  - Euvolemic when therapy initiated (not wet OR dry)
  - Not in reactive airways disease or bradycardia or AV block
- Not without diuretics, unless no prior congestion

ACC/AHA Heart Failure Guideline 2009, p. e420

Which Beta Blocker for HF

- All beta blockers are NOT the same
- Proved and recommended in HF (not head-to head)
  - Carvedilol 25 mg po bid (Coreg); COMET, 2003 (Indicated for NYHA class II-IV), superior to metoprolol tartrate
  - Metoprolol succinate 200 mg po qd (Toprol XL) Merit HF, 1999 (Indicated for NYHA class II-III)
  - Bisoprolol CIBIS-II, 1999 (FDA approved for htn, but not HF)
- Not proved in HF – all others, including bucindolol

ACC/AHA Heart Failure Guideline 2009, p. e420

Initiating Beta Blockade in HF

- Proper patient selection
- Start low: 3.125 bid carvedilol or 12.5 qd for metoprolol succinate (25 mg for NYHA II) or 1.25 qd bisoprolol
- Go slow: double every 2-4 weeks and when stable; patient should weigh daily
- Reduce if transient worsening does not respond to increase in diuretics or if symptomatic bradycardia (<55)
- Target: 25 bid carvedilol (50 bid if >187 lb and mild-to-moderate HF) or 200 qd for metoprolol succinate or 10 qd bisoprolol
- Achieved: carvedilol 42 mg/da (COMET), metoprolol succinate 159 mg/da (MERIT-HF)

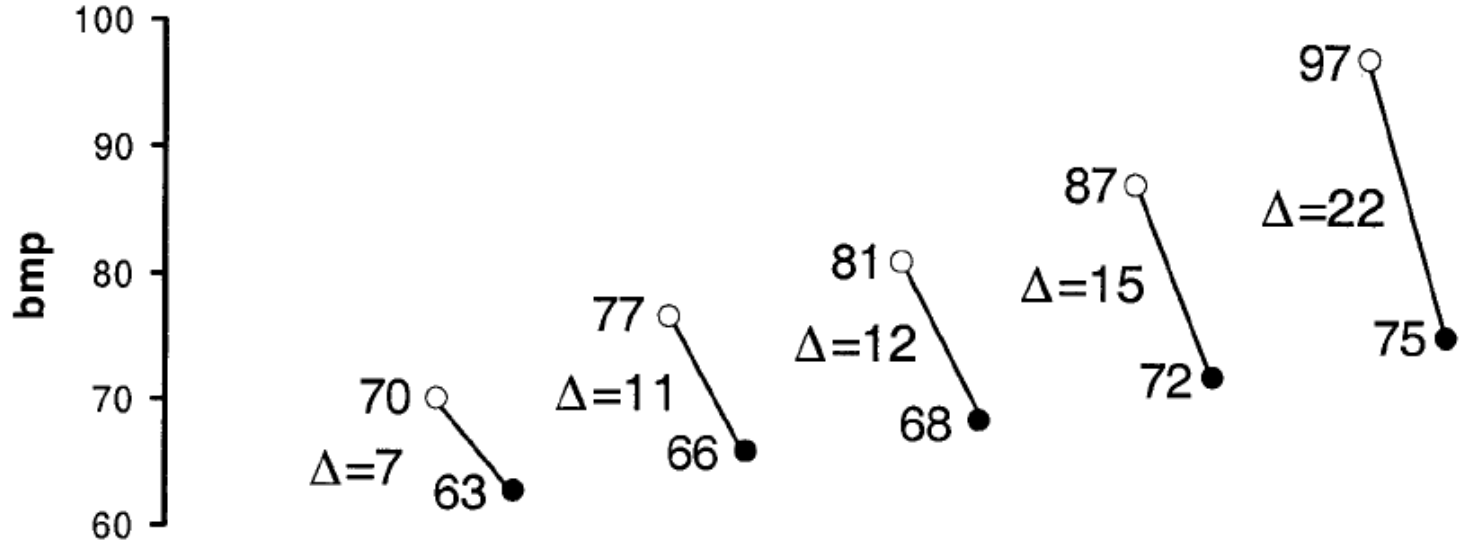
ACC/AHA Heart Failure Guideline 2009, p. e420

Questions about Beta Blockers

- If the baseline HR is low, will they still work? -- yes*
- If the baseline BP is low, will they still work? -- yes**
- Do they work in diabetes? -- yes
- Do they work in Class IV? -- yes
- Do they work in both ischemic and nonischemic HF? -- yes
- Can I use in COPD? -- yes if not reactive; Elderly? -- yes
- Are they hard to use in HF? -- no, but take your time and be persistent***
- Which agents have outcomes data? -- carvedilol, metoprolol succinate, bisoprolol

*Gullestad L et al. J Am Coll Cardiol. 2005;45:252 (HR 58-73, 146 mg/da metop)
**Rouleaux JL et al. J Am Coll Cardiol. 2004;43:1423
***Krum H et al. JAMA. 2003;289:712

Beta blocker and baseline HR in MERIT-HF



Quintile (Q)	Q1	Q2	Q3	Q4	Q5
Meto CR/XL dose (mg)	146	149	163	167	166
Net reduction (bpm)	8	10	11	13	14
% reduction (bpm)	10	14	16	18	23

Gullestad L et al. J Am Coll Cardiol. 2005;45:252 (HR 58-73, 146 mg/da metop.)

Loose Ends: Beta Blockade in HF

- Clinical response takes 2-3 months; avoid abrupt withdrawal
- Clinical deterioration in patients on chronic beta-blocker: if mild, adjust other medications, but if severe with hypoperfusion or need for IV inotropes (milrinone), prudent to suspend and reintroduce when restabilized
- Adverse reactions: worsening HF and fluid retention, fatigue which is usually self-limiting after several weeks but if with hypoperfusion must discontinue, bradycardia and heart block (pacemaker?), hypotension (may administer ACE-I and beta-blocker at different times of the day or relax diuretics if too dry)

ACC/AHA Heart Failure Guideline 2009, p. e420

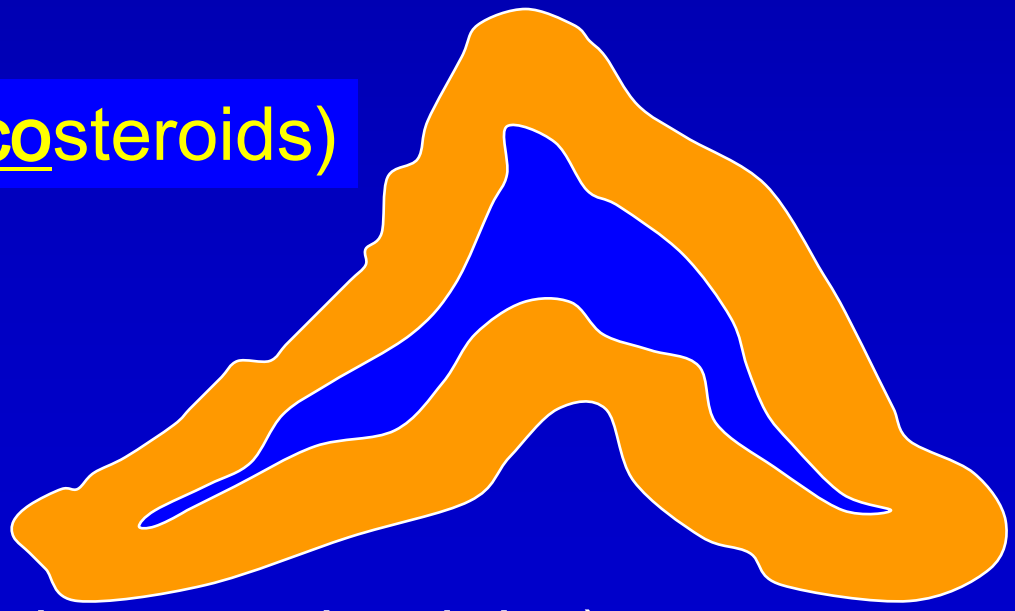
The Adrenal Gland

Aldosterone

Salt	**mineralocorticoid**
Sugar	glucocorticoid
Sex	androgens

"GFR"
Zona glomerulosa
Zona fasciculata
Zona reticularis

Cortex (adrenal **cortico**steroids)



Medulla

(Catecholamines: Epinephrine, norepinephrine)

Aldosterone is Increased in HF

- Plasma level is higher
  - Normal: 5-15 nanograms/dl
  - CHF: up to 300 nanograms/dl
  - Severe Na restriction: similar to CHF
- Secretion rate is increased:
  - Normal: 100-175 micrograms/da
  - CHF: 400-500 micrograms/da
- Hepatic clearance of aldosterone is reduced:
  - Worsened in upright posture and ambulation

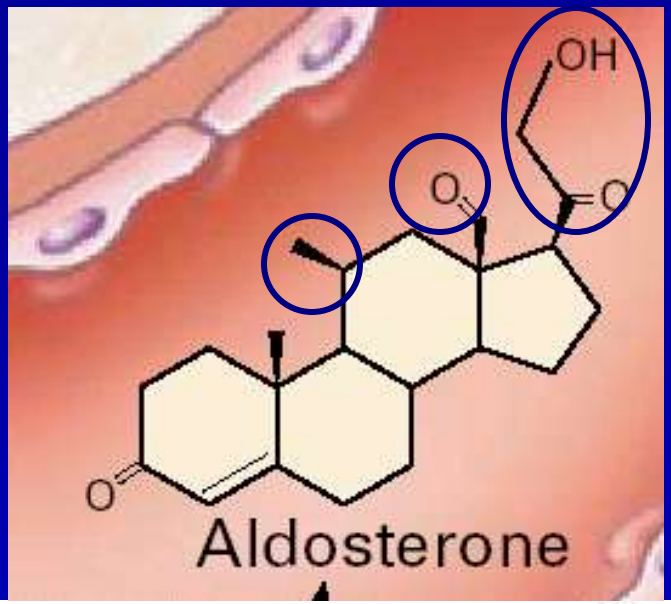
Weber KT. N Engl J Med. 2001;345:1689.

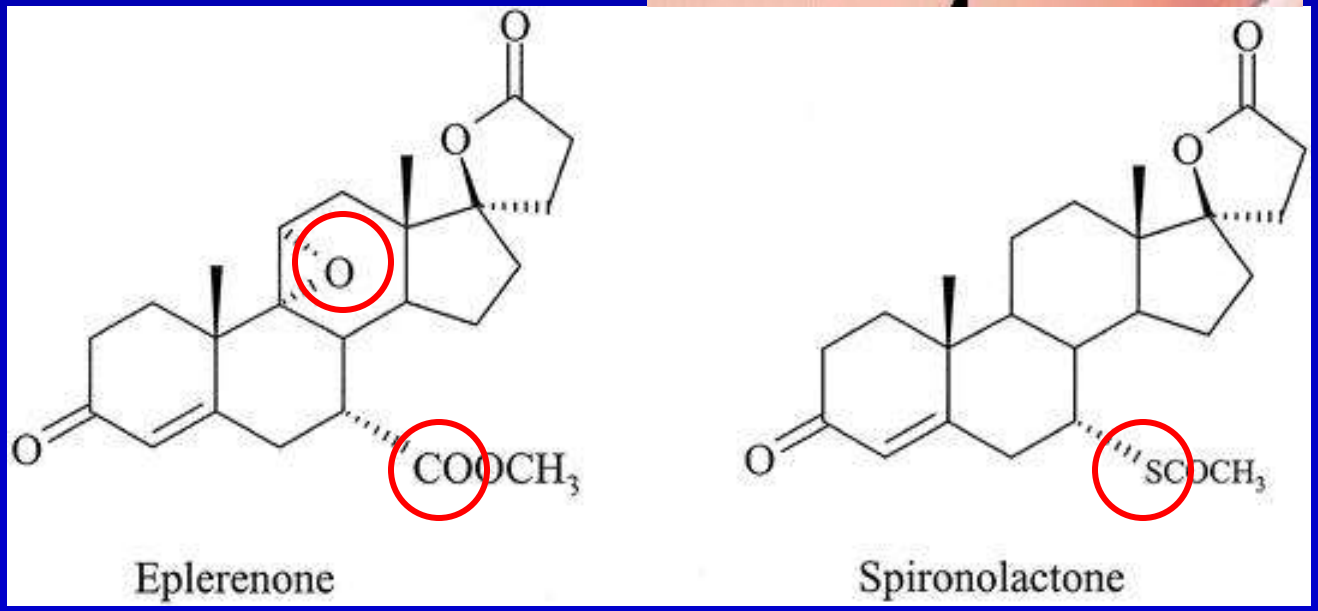
Actions of Aldosterone

- Sodium retention (distal tubule – angiotensin II stimulates sodium retention in the proximal tubule)
- Magnesium and potassium wasting
- Sympathetic activation
- Parasympathetic inhibition
- Myocardial and vascular fibrosis
- Baroreceptor dysfunction
- Vascular damage
- Impairment of arterial compliance

Pitt B, et al. N Engl J Med. 1999;341:709

Both spironolactone and epleronone are FDA approved for heart failure





Moore TD et al. Heart Disease 2003;5:354

Aldosterone Antagonists

- Spironolactone – RALES trial in class IV used only 25 mg/da, and patients generally were on high dose furosemide 80 mg/da and 10% had breast pain or gynecomastia, showed reduction in mortality; indicated in NYHA class IV patients or patients with K <3.8 on diuretics despite K supplementation
- Epleronone –more selective, less gynecomastia, approved for hypertension, the EPHESUS trial showed a reduction in all-cause mortality in CHF
- Indication: Symptoms despite other agents, low dose, not if Cr>2.5 or K>5.0. If K rises above 5.4, reduce the dose

ACC/AHA Heart Failure Guideline 2009, p. e418.

Criteria for Minimizing Hyperkalemia

- Avoid if Creatinine > 1.6 or Ccl < 30
- Don’t use if Baseline K > 5.0
- Initial dose: spironolactone 12.5mg or eplerenone 25mg
  - Double dose if appropriate
- Increased risk with high dose ACEI’s
  - Captopril > 75 mg / day
  - Enalapril or lisinopril > 10mg / day

Criteria for Minimizing Hyperkalemia - 2

- Avoid non-steroidal antiinflammatories and cyclo-oxygenase-2 inhibitors
- Discontinue or reduce K supplements
- Close monitoring of potassium and renal function is REQUIRED
  - In 3 days
  - In 1 week
  - Monthly for first 3 months
  - Diarrhea/dehydration must be addressed emergently

Aldosterone Antagonist Indications

- I - LVEF <35% and on loop diuretics and prior or current NYHA class IV
- IIa - MI and LVEF <40% and HF on ACE-I and beta blocker
- Not:
  - Cr >2.5 or baseline K>5.0 (absolute)
  - Cr >1.6 or Ccr <30 (relative)

ACC/AHA Heart Failure Guideline 2009, p. e418.

Nitrate-Hydralazine in HF

- Hypothesis: Black patients may have lower renin-angiotensin activity and lower availability of nitric oxide, and post-hoc clinical response to hydralazine and isosorbide dinitrate

- 1050 black patients, (age 57, 60% men, 94 kg, 95% III, 40% DM, 17% ICD, EF 24%, LVID 6.5, NYHA III-IV >3 mo
- Baseline ACE (70%), ARB (17%), β-blocker x 3 mo (74%), dig (60%), diuretic (90%), spironolactone (40%), 2 week stable wt and meds
- EF<35 or cardiomegaly and EF<45
- 37.5 mg hydralazine plus 20 mg isosorbide dinitrate tid then 2 tid (225 hydralazine plus 120 isosorbide) .. Achieved 3.8 tabs/da, 68% took 6 tab/da

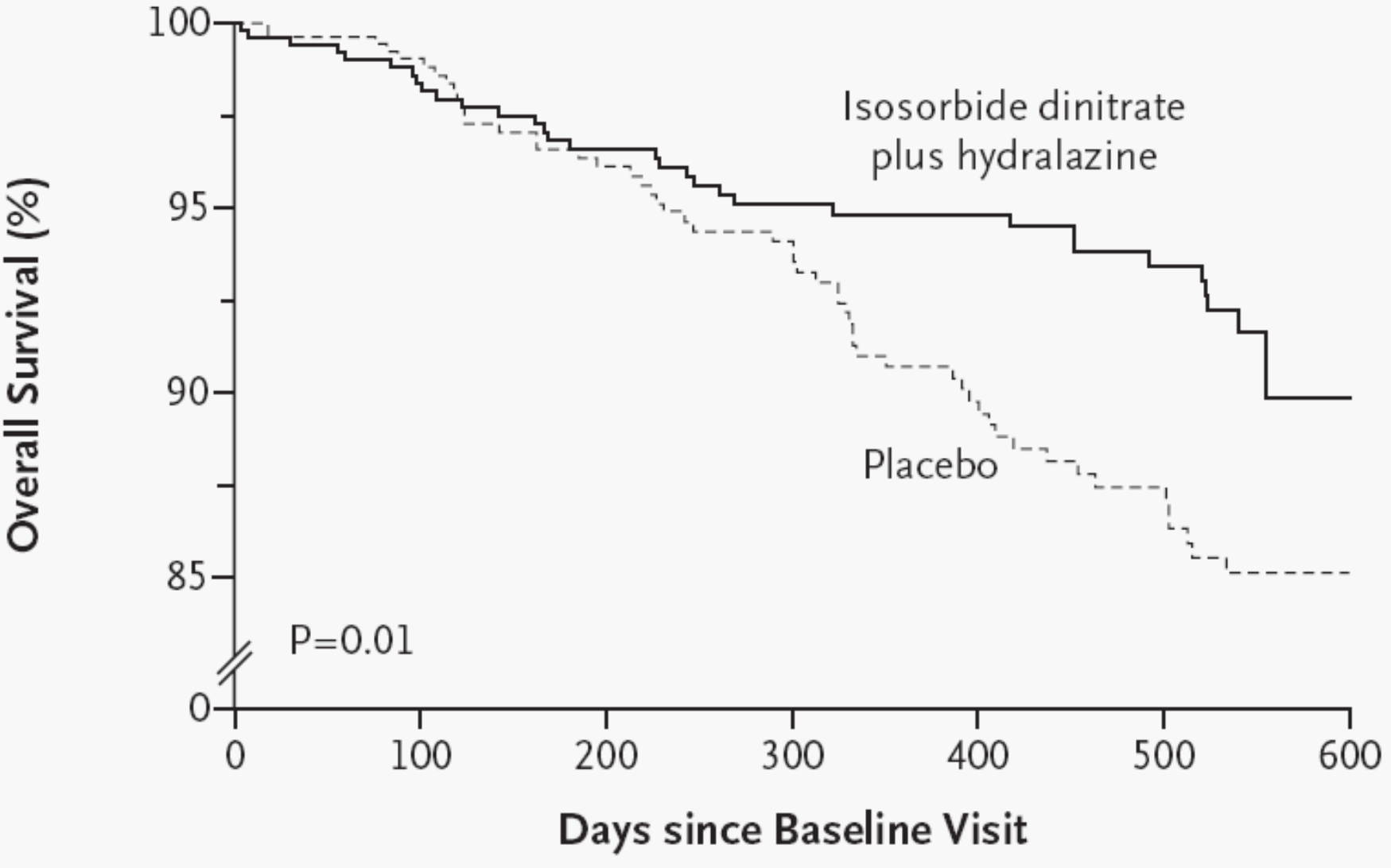
Taylor AL et al. A-HEFT investigators. N Engl J Med 2004;351:2049

Nitrate-Hydralazine in HF - Results

- SBP and DBP 3 mmHg lower than placebo, more headache and dizziness
- 18 months, f/u LVEF, LVED, wall thickness, BNP, qual of life
- Primary outcome: composite score – all cause death, first hospitalization for HF in 18 mo, quality of life at 6 mo
- Secondary outcomes: Each - CV death, total num hosp HF, total num hosp, tot day in hosp, qual of life overall, num unscheduled office or ER visits, 6 mo change in BNP, new need for transplant, change in LVEF, LVED and wall thickness at 6 mo
- Study stopped for higher mortality in placebo than hydralazine-nitrate (43% lower with hydralazine-nitrate, $P<0.01$), mean f/u 10 mo.
- 33% lower first hospitalization for HF

Taylor AL et al. A-HEFT investigators. N Engl J Med 2004;351:2049

Nitrate-Hydralazine in HF



Taylor AL et al. A-HEFT investigators. N Engl J Med 2004;351:2049

Thoughts about Hydralazine-Isosorbide

- The benefit is in the presence of active medical therapy with neurohormonal blockade
- Maybe the benefit is due to nitrate acting as nitric oxide donor and hydralazine acting to protect against degradation of nitric oxide by oxidative stress (endothelial dysfunction and impaired nitric oxide bioavailability occur in HF)
- It is not known if the benefit might occur in other ethnic groups

Taylor AL et al. A-HEFT investigators. N Engl J Med 2004;351:2049

Recommendation for Isosorbide Dinitrate and Hydralazine Use

- Whereas diuretics, ACE-inhibitors, aldosterone antagonists, beta-blockers, digitalis, and ICDs are “recommended for routine use”, the combination isosorbide dinitrate and hydralazine is “to be considered for use in selected patients”
- “This combination is recommended for African Americans who remain symptomatic despite optimal medical therapy”

ACC/AHA Heart Failure Guideline 2009, p. e426.

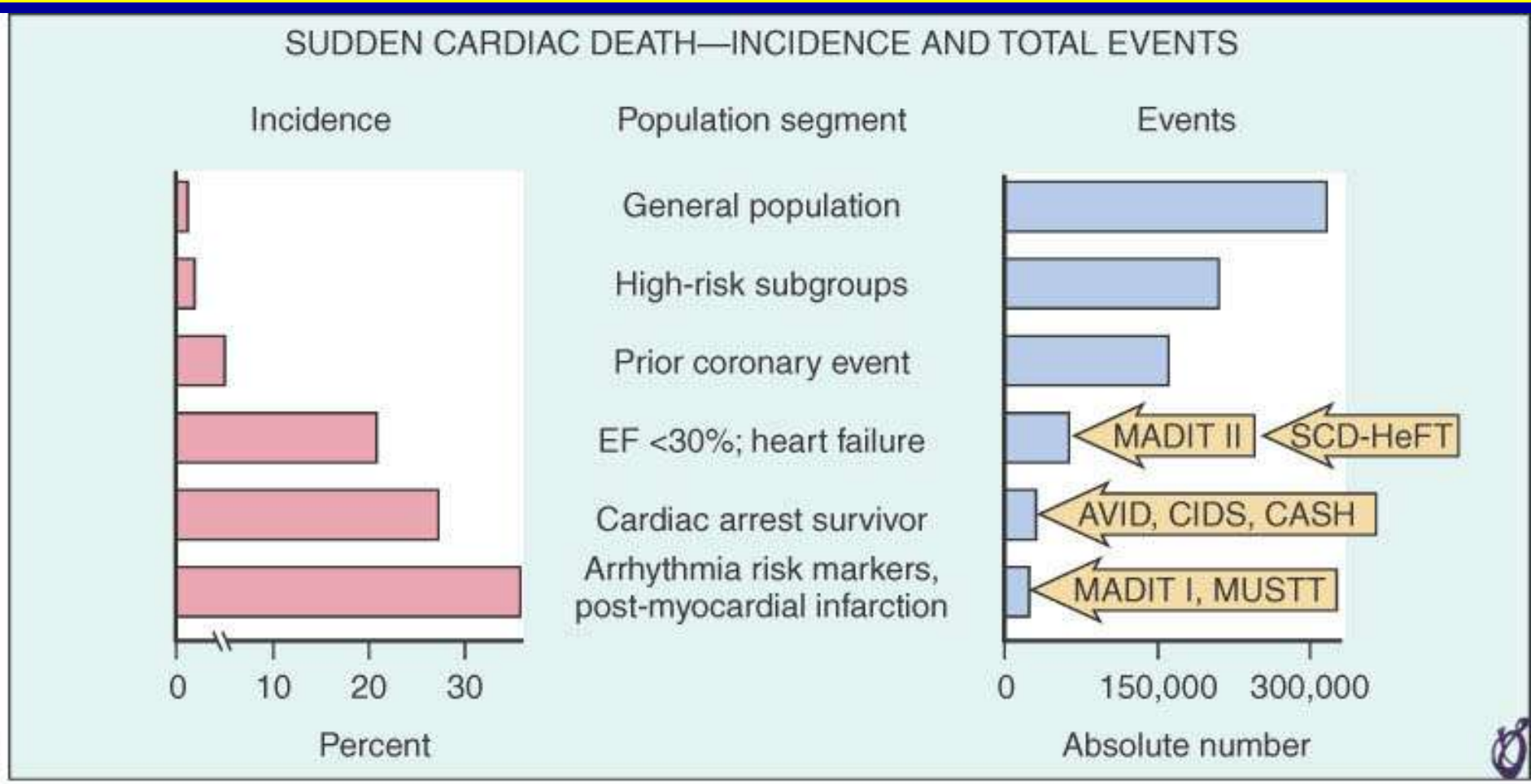
Devices for Heart Failure

- Implantable cardioverter-defibrillator = ICD
- Biventricular pacemaker (cardiac resynchronization therapy), generally with ICD = Bi-V-ICD
- Mechanical circulatory support with Left ventricular assist device (LVAD)
  - Bridge to transplant
  - Bridge to recovery (disappointing)
  - Destination therapy

Implantable Cardioverter Defibrillator

- Sudden cardiac death is common and hard to predict and nearly impossible to treat
- So, prevention and selection are key responses
- In heart failure, many of the deaths are sudden

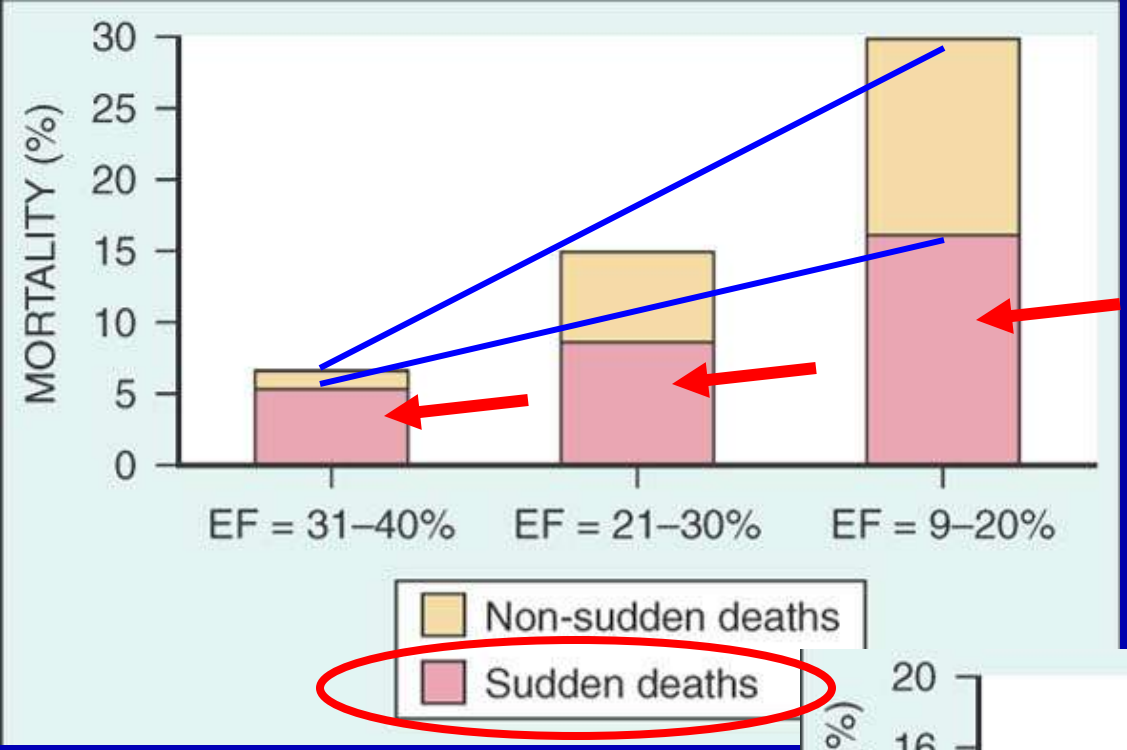
Most Victims are Low Risk

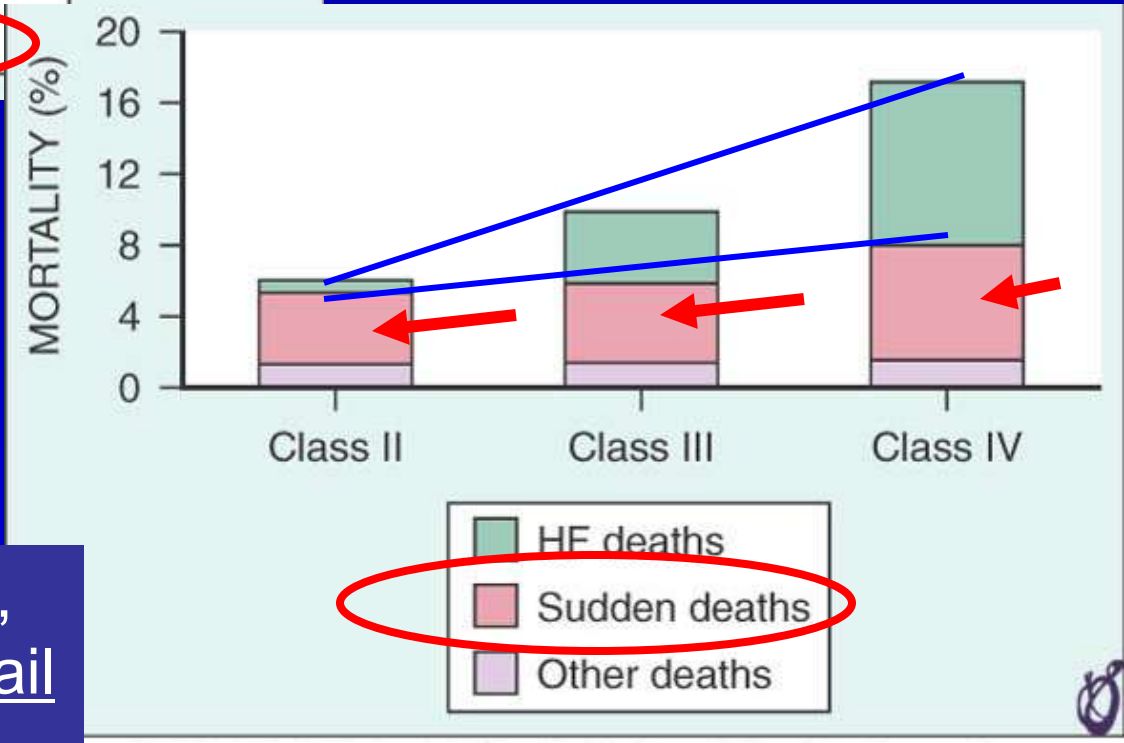


(Modified from Myerburg RJ, Kessler KM, Castellanos A: Sudden cardiac death: Structure, function, and time-dependence of risk. Circulation 85(Suppl I):I2, 1992. With permission of the American Heart Association. © 1992 American Heart Association. B, Modified from Myerburg RJ: Sudden cardiac death: Exploring the limits of our knowledge. J Cardiovasc Electrophysiol 12:369, 2001.)

Braunwald's Heart Disease, 8th ed, p. 935,
from J Cardiovasc Electrophysiol, 2001.

Proportional SCD Risk vs EF and NYHA Class





Braunwald's Heart Disease, 8th ed, p. 943, from Heart Fail Rev, 2002.

(Modified from Cleland JG, Chattopadhyay S, Khand A, et al: Prevalence and incidence of arrhythmias and sudden death in heart failure. Heart Fail Rev 7:229, 2002, with kind permission from Springer Science and Business Media.)

Mortality in Primary Prevention

Population	Control	ICD	ARR
MI, EF<35, NSVT, induc VT	32/2y	13	19
MI, EF<40, NSVT, induc VT	55/5y	24	31
CABG, EF<36, +SAECG	18/2y	18	0
MI>1mo, EF<30	22/2y	16	6
DCM, EF<35	14/2y	8	6
NYHA 2-3, EF<35	36/5y	29	7

MADIT, MUSTT, CABG-Patch, MADIT-II, DEFINITE, SCD-HeFT

Patient Selection for ICD: Basics

- Patient selection is often determined by LV EF and there is no gold standard – the clinician should use whatever method he judges best in his practice location
- ICD implantation is a purely elective procedure
- ICD implantation is always in the context of optimal medical therapy for an extended period of time, 4-6 months with persistent low LVEF (up-titration of beta-blocker can take 2 months, and then progressive improvement in EF may occur for 12 months)

ACC/AHA/HRS Guidelines for Device-Based Therapy. 2008.

Patient Selection for ICD - 1

- Secondary prevention – VT or VF arrest, sustained VT, syncope + inducible VT
- Primary prevention
  - EF<35, NYHA 2-3 (CAD >40d post MI or DCM) … (NYHA 1 is IIb level)
  - EF<30, NYHA 1 (CAD >40d post MI)
  - EF<40, prior MI, NSVT, and inducible VT/F

ACC/AHA/HRS Guidelines for Device-Based Therapy. 2008.

Patient Selection for ICD - 2

- DCM, significant ↓EF, unexplained syncope
- Sustained VT and normal EF (or mild)
- Risk factors or poor medical response in HCM, ARVD, long QT, Brugada syndrome, catecholaminergic VT, outpatient awaiting transplant, sarcoidosis, Chaga's, giant cell myocarditis

ACC/AHA/HRS Guidelines for Device-Based Therapy. 2008.

Patient Exclusions for ICD

- No reasonable expectation of 1 year survival with acceptable functional status (Class 4, recurrent admissions despite meds and not candidate for transplant or CRT)
- Incessant VT or VF
- VT or VF amenable to therapy (ablation or surgery) or due to reversible cause
- Psychiatric illnesses that may be aggravated or impair follow-up

ACC/AHA/HRS Guidelines for Device-Based Therapy. 2008.

Outcomes in ICD Therapy

- Complications – arterial puncture, pneumothorax, air embolism, AV fistula, hematoma, inadvertent LV placement, lead perforation
- Inappropriate action – fail to deliver therapy; delivering inappropriate shock; generally addressed by reprogramming device
- Recurrent shocks can result in PTSD
- Live longer (careful in counselling) – not immortality
- Don’t feel better

Cardiac Resynchronization Therapy

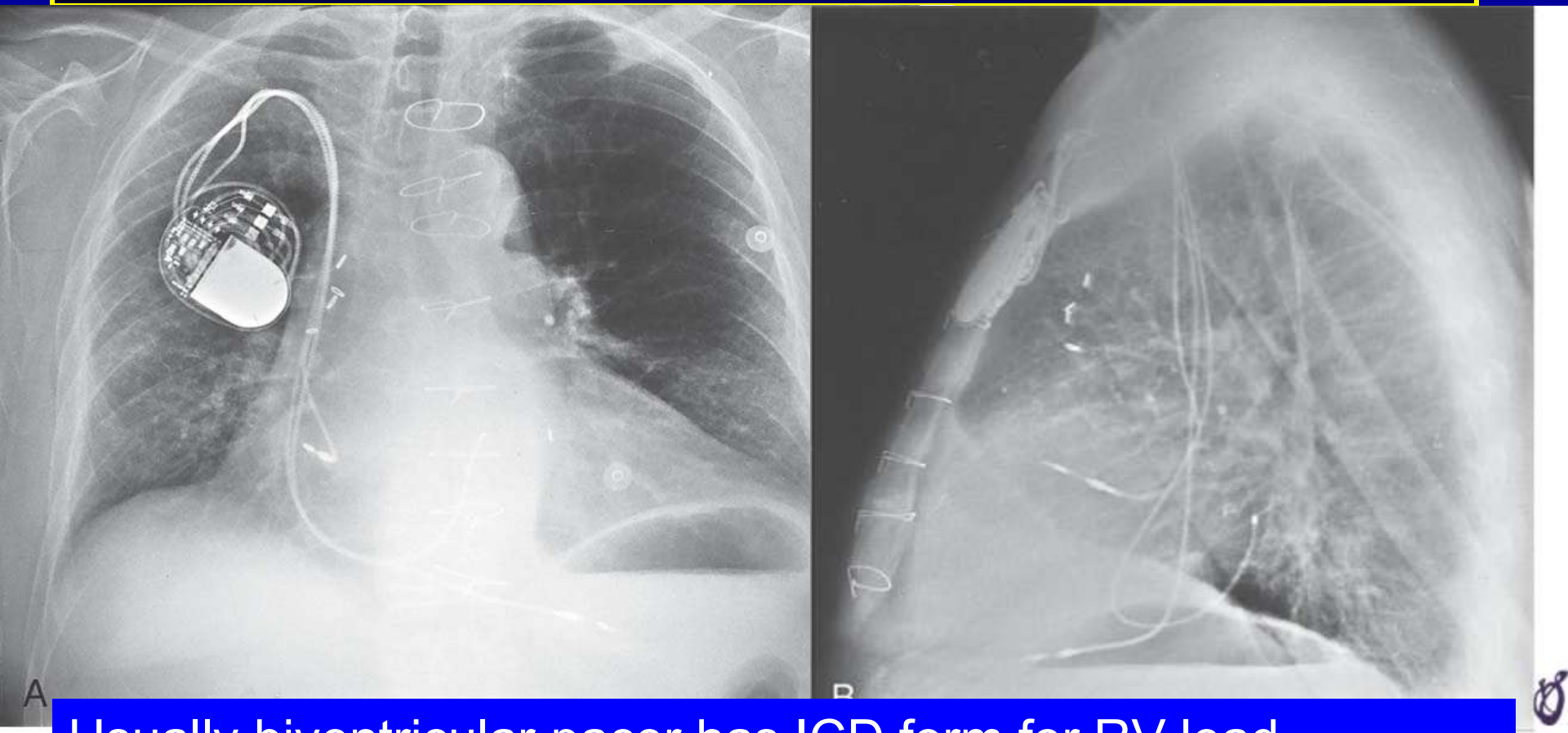
- Biventricular pacing
- Generally with ICD included
- Three pacing leads
  - RA appendage
  - RV apex
  - LV high lateral wall via coronary sinus

Biventricular Pacing in Heart Failure: Background and Rationale

- About 1/3 of patients with NYHA 3-4 systolic HF have wide QRS
- Wide QRS (esp LBBB or ventricular paced beat) causes poor synchrony of LV contraction and impairs LV systolic and diastolic function and mitral valve function (MR)
- Simultaneous pacing of RV apex and LV lateral wall (via lateral cardiac vein from coronary sinus) improves synchrony of contraction and may relieve symptoms and improve MR and has shown decreased mortality

ACC/AHA Guideline update: heart failure in the adult. 2009

Chest X-Ray in Biventricular Pacer

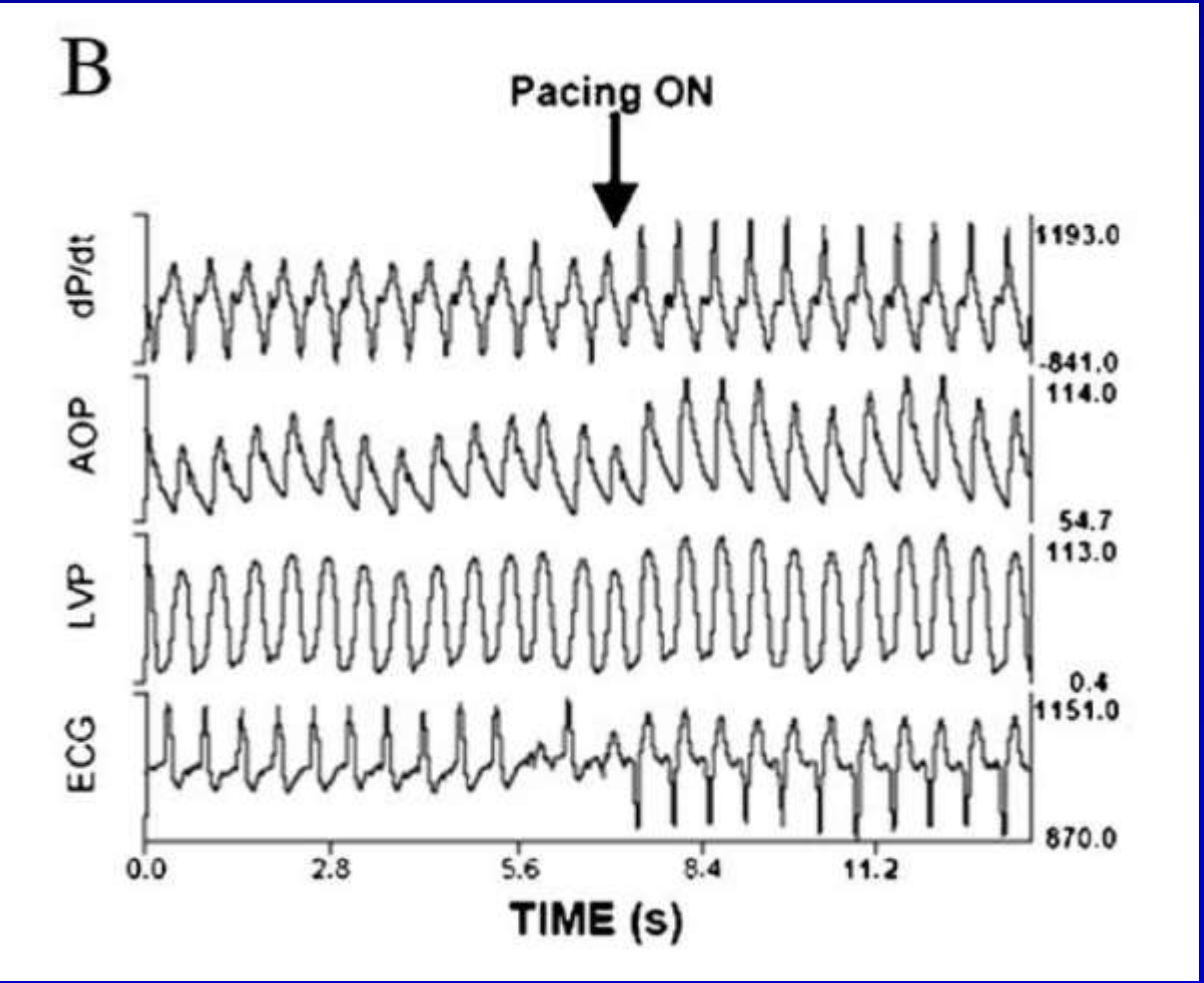


Usually biventricular pacer has ICD form for RV lead
3 leads from the generator: RA appendage, RV apex, LV lead in high lateral wall of LV via the coronary sinus

Braunwald's Heart Disease, 8th ed, p. 835.

Biventricular Pacing in Heart Failure: Background and Rationale

- Biventricular pacing shows improved LV developed pressure and dP/dt, and aortic pulse pressure



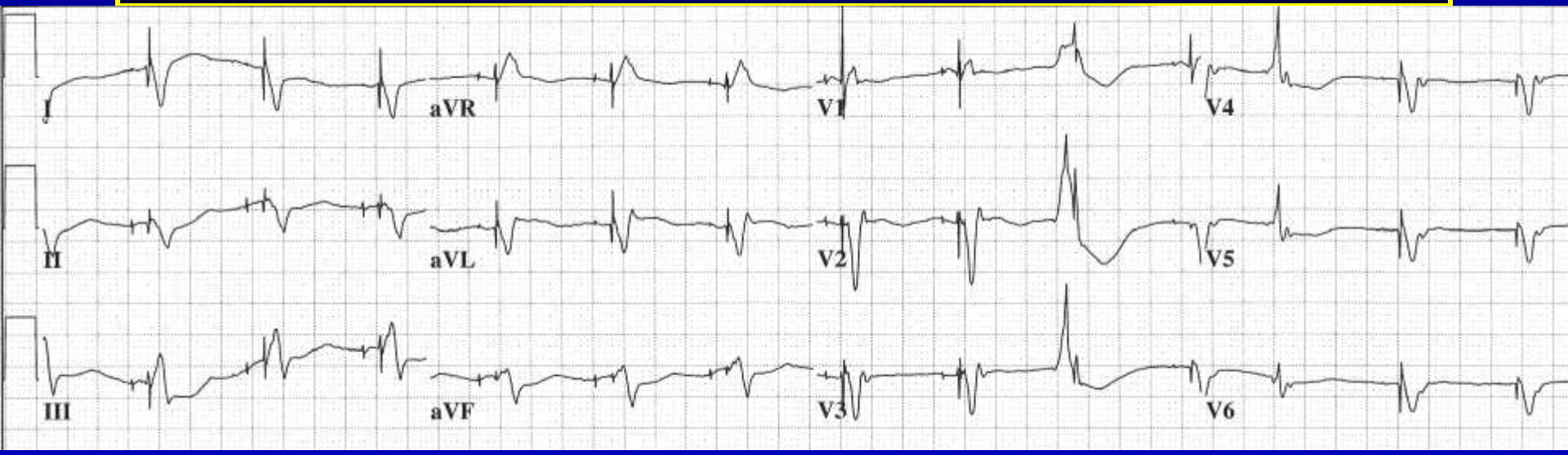
ACC/AHA Guideline update: heart failure in the adult. 2009

Biventricular Pacing in Heart Failure

- Context: optimal medical therapy for 4-6 months with persistent low EF, and a reasonable expectation of survival with good functional status for >1 year
- NYHA class III-IV (if class IV should be ambulatory) with sinus rhythm and EF<35% and QRS widening >0.12 sec (often much wider, average in some studies 0.15 sec)
- Class IIa: if AFib, or if likely to be frequently ventricular paced
- Bi-V without ICD: controversial

ACC/AHA Guideline update: heart failure in the adult. 2009

ECG in Biventricular Pacemaker



Short PR interval allows pacer to completely capture the ventricle

QRS morphology is contrasted with usual RV apex paced beat

- QRS is negative in lead I
- QRS is positive in lead V1

Outcomes in Biventricular Pacing

- Improvement in exercise capacity, symptoms, MR severity, HF hospitalizations, LV EF, and overall mortality (RBBB pattern probably not)
- Procedural mortality 0.4%, device success 90%, complications, maybe 10%
- Up to 1/3 don't improve

ACC/AHA Guideline update: heart failure in the adult. 2009.
Van Bommel et al. Eur Heart J. 2009.

Thanks!

- Questions?
- Comments?