Heart Failure

History of Presenting Illness

The heart failure patient, upon presentation, will complain of

- **EXTERTIONAL DYSPNOEA**
- **ORTHOPNOEA**
- **PAROXYSMAL NOCTURNAL DYSPNOEA**
- **ANKLE SWELLING**
- **ABDOMINAL SWELLING**
- **ANOREXIA**
- **NAUSEA**

...or, possibly, the bouquet of **INFARCT SYMPTOMS + ANGINA**

Differential Diagnoses

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Pertinent Findings on History

- **Orthopnea** = early symptom; 
  ... how many pillows?... 
  ... occurs rapidly, often within a minute or two of recumbency
- **Exertional dyspnea**
- **Non-productive cough**
- **Paroxysmal nocturnal dyspnea**
  = sudden awakening of the patient, after a couple hours of sleep, with a feeling of severe anxiety, breathlessness, and suffocation. The patient may bolt upright in bed and gasp for breath. 
  ... may require 30 minutes or longer in this position for relief.
- **Dyspnea at rest**
- **Nocturia**
- **Fatigue and weakness**
- **Cerebral symptoms:**
  - Confusion,
  - memory impairment,
  - anxiety,
  - headaches,
  - insomnia,
  - bad dreams or nightmares,
  - rarely, psychosis with disorientation, delirium, or hallucinations may occur in elderly patients with advanced heart failure, esp. those with cerebrovascular atherosclerosis.

**Predominant right heart failure:**

- Ascites,
- congestive hepatomegaly,
- increased abdominal girth
- epigastric and right upper quadrant (RUQ) abdominal pain.
- anorexia,
- bloating,
- nausea,
- constipation.
- In preterminal heart failure, inadequate bowel perfusion can cause abdominal pain, distention, and bloody stools. Distinguishing right-sided CHF from hepatic failure is often clinically difficult.
- **NO Dyspnea!!** unlike LHF
- **NOT UNTIL LATER** does dyspnea occur as a consequence of the reduced cardiac output, poor perfusion of respiratory muscles, hypoxemia, and metabolic acidosis.
Findings on Examination

**LVF**

**OBSERVATION:**
- Tachypnoea
- Central cyanosis
- Cheyne-Stokes breathing
- Peripheral cyanosis
- Hypotension
- Cardiac cachexia

**PULSE:**
- Sinus tachycardia
- low pulse pressure
- pulsus alternans
  (alternating strong and weak beats)]

**PALPATION:**
- displaced apex beat

**AUSCULTATION**
- left ventricular S3
- mitral regurg pansystolic murmur
- lung fields will crackle coarsely @ bases

**Tests and Investigations:** it's all about the UNDERLYING CAUSE

**FBC:** Looking for anaemia, which would in turn cause tachycardia in an underperfused heart

**Electrolytes** Looking for derangements of calcium and potassium (cause arrhythmia)

**Liver Function Tests** Looking for liver failure, to exclude a non-cardiac reason for hepatomegaly

**ABGs**
- Looking for O2; to see if more is needed.
- If there is hypercapnea, hypoxemia, and the pt. is acidotic
  - consider doing something RIGHT AWAY

**CHEST X-RAY**

**KEY WORDS FOR RADIOLOGICAL ABNORMALITIES OF HEART FAILURE:**
- Enlarged cardiac silhouette
- Spread fan of “batwings” around heart: opaque distended vessels
- cuffing of the bronchial walls (OEDEMA)
- pleural effusions at the costodiaphragmatic recesses
- Kerley Lines

**CARDIO-RADIOLOGY:** ALWAYS MEASURE PA FILM
to get the right magnification, else the heart seems too big
1st thing: HEART SHOULD OCCUPY NO MORE THAN HALF OF THE THORACIC DIAMETER
2nd thing: look for effusion (blunt angles)
3rd thing: look for pulmonary congestion (batwings)

Remember the Kerley lines!!
= signs of oedema; = fluid in the interlobular septum
ECG

**ECG**

- Normal
- Hypersensitive Infarction
- Acute Transmural Myocardial Infarction or Aneurysm
- Acute Transmural Myocardial Infarction Hours old
- Acute Transmural Myocardial Infarction Days old
- Ischemia
- Pericarditis
- Sub-endocardial Myocardial Infarction

**ECHOCARDIOGRAM**

*Shows everything! ➔ EASIEST + LEAST EXPENSIVE*
- Function of valves
- Whether anything regurgitates through the valves
- Thickness of LV wall
- Presence of pericardial disease
- Regional wall motion abnormalities

**Disease Definition**

*Heart failure: when cardiac output is less than what the tissues demand; i.e. the HEART IS NOT DOING ITS JOB*  

**Management: DRUGS to either live longer or feel better (and rarely both)**

*!! Most effective drugs are those that modify harmful neurohormonal adaptation!!*

**THERE IS AN ESTABLISHED PATHWAY OF TREATMENT according to patients condition:**

1. **NITRO VASODILATORS:** reperfuse the myocardium, reduce **TPR**
2. **Beta Blockers.** Start right away
3. **ACE Inhibitors** while LV dysfunction is asymptomatic
4. **Mild Diuretics + Digoxin** when it becomes symptomatic
5. **Loop diuretics**
6. **Spironolactone (aldosterone inhibitor)** when there’s dyspnoea at rest
7. **Specialised therapies, angioplasty transplant, multi-agent diuresis**

**AND ALL THE WHILE:**

- **Education:** QUIT SMOKING!! STOP DRINKING!!
- **Exercise**
- **Salt and Fat reduced diet**

**In general, patients with an ejection fraction below 25% have severe heart failure.**

**THERE IS NO DIAGNOSTIC “HEART FAILURE” ECG!**

Instead, you’re LOOKING FOR:

- **ARRHYTHMIA**
  - atrial fibrillation is present in 25 percent of patients with cardiomyopathy, especially elderly patients with advanced heart failure. The prognosis is worse for patients with atrial fibrillation, atrial or ventricular tachycardia, or left bundle branch block
  - *IF THEY ARE IN ATRIAL FIBRILLATION, GIVE THEM ANTICOAGULANTS RIGHT AWAY!! Don’t wait for the thrombus to break off and sail to the brain*

- **LV ENLARGEMENT:** left axis deviation
  - Extreme right axis deviation
  - Right axis deviation
  - ST SEGMENT CHANGES to indicate infarct; see left
Prognosis

HEART FAILURE ALONE: you have a 5-20% chance of dying in the hospital
WITH MYOCARDIAL INFARCTION, 20-40% mortality

Epidemiology
- Nearly 1 million hospital admissions for acute decompensated CHF occur in the United States yearly,
- Affects 2% of the USA population
- Nearly 2% of all hospital admissions in the United States are for decompensated CHF,
- An estimated $23 billion are spent on inpatient management of CHF every year
- Another $40 billion are spent in the outpatient setting on patients with compensated or mildly decompensated heart failure every year
- incidence and prevalence of CHF are higher in the underprivileged proletariat
- Men and women have equivalent incidence and prevalence of CHF
- most common in individuals older than 65 years

Behavioural science:  HOW TO DETECT AN ALCOHOLIC

Historical assessment:
Ask about
- amount, (men = 28 /wk, women = 14)
- regularity (need alcohol-free days)
- favourite drink (spirits more dangerous)
- “eye-openers” (= sign of addiction)
- withdrawal effects
- depression, anxiety
- social problems, psychological problems
- relationship, family, work-related or legal problems

Physical assessment:
Look for:
- liver edge:
  - swollen (steatosis, early)
  - or shrunken (cirrhosis, late)
- abdomen: distended with ascites?
- Jaundice?
- Cardiomegaly?
- Neuro exam: encephalopathy?

Laboratory assessment:
- LIVER FUNCTION TESTS are all-powerful; look to GGT and ALT
- BLOOD FILM AND COUNT: looking for megaloblastic anaemia of alcoholism
Aetiology / Pathophysiology: mechanism of HEART FAILURE

**CHRONIC ALCOHOL CONSUMPTION > 90 g/day**

Genetic defect of the SARCOMERE

Mitrail or Aortic REGURGITATION

INFACTION

ISCHAEMIA

**DILATION CARDIOMYOPATHY**

myocardium remodels in series in response to distending stimulus

**CHRONIC VOLUME OVERLOAD** = INCREASED PRELOAD

Myocyte death And FIBROSIS

**IMPAIRED CONTRACTILITY**

- Too few myocytes
- Reduced force per unit area (inefficient dilated anatomy)
- Too much preload (heart muscle stretched by influx of blood- too much blood filling it to contract effectively)

**SYSTOLIC DYSFUNCTION**

i.e diminished ejection fraction

LV filling obstruction: mitral stenosis, cardiac tamponade etc.

**DIASTOLIC DYSFUNCTION**

Myocardocytes remodel in parallel i.e wall thickness increases; = CONCENTRIC CARDIOMYOPATHY

Left Ventricular HYPERTROPHY

FIBROSIS

Genetic defect of the SARCOMERE

**CHRONICALLY STIFFENED LEFT VENTRICLE**

Transiently impaired relaxation – due to ion pump starvation

**IMPAIRED RELAXATION**

Thus DIMINISHED FILLING

**REDUCED CARDIAC OUTPUT**

**INCREASED HEART RATE**

Thus INCREASED METABOLIC DEMAND @ MYOCARDIUM

@ MEDULLA: decreased parasympathetic outflow increased sympathetic outflow

VIA CRANIAL NERVES 9 and 10

INCREASED BLOOD VOLUME !!!

**INCREASED AFTERLOAD**

REDUCED STROKE VOLUME

!!! REDUCED CARDIAC OUTPUT !!!

**INCREASED RENAL ARTERY PERFUSION**

Reduced delivery of salt to the macula densa of the kidney

RENIN IS SECRETED → converts circulating angiotensinogen into ANGIOTENSIN 1

→ converted by endothelial ACE into ANGIOTENSIN-2

= arterioconstrictor

= thirst stimulator via hypothalamus

= ADH secretion stimulator @ant. pituitary (thus reducing Na+ excretion)

= ALDOSTERONE secretion stimulator (thus further reducing Na+ excretion)

NO Na+ EXCRETION = WATER RETENTION

**INCREASED BLOOD PRESSURE**

**INCREASED VENOUS RETURN**

**VASOCONSTRICTION**

**REDUCED CARDIAC NUTRITION**

Some return to NORMAL CARDIAC OUTPUT

Increased perfusion pressure sensed by carotid sinus and aortic arch baroreceptors

Decreased perfusion pressure

> ATRIA DISTENDED

> Atrial Natriuretic Peptide released

> INHIBITS RENIN SECRETION

> REDUCES EFFECT OF Angiotensin 2 on ALDOSTERONE secretion

> INCREASED EXCRETION OF SODIUM;

> thus REDUCED BLOOD VOLUME
The Heart and Exercise

Cardiac Output increases in response to increased oxygen demand.

**MAXIMAL EXERCISE:**
Maximal heart rate does not increase after training. It stays the same (or might even decrease just slightly). However, maximal stroke volume increases.

**MAXIMUM HEART RATE:**
The rate beyond which the heart will not have time to fill = 220 minus age in years
- useful max = ~180 bpm

**EXERCISE:**
- As you begin to exercise, the oxygen demand increases.
- **THUS** cardiac output increases:
  - **BY INCREASING BOTH HEART RATE AND STROKE VOLUME**
  - **HOWEVER** one would expect mean arterial pressure to increase from all this extra blood being pumped in –
  - MAP does not increase – because the blood vessels also dilate, redirecting blood flow TO THE STARVING MUSCLES
  - The blood flow can increase 35-fold!!
  - **THUS** the blood pressure doesn’t increase nearly as much as the heart rate

Physiology of heart function:

**DIASTOLE:**
- Relaxed ventricles fill with atrial blood
- The ventricle wall distends

**SYSTOLE:**
- Ventricular wall contracts in response to pacemaker signal
- Rising ventricular pressure forces the “in” valve shut
- NO VALVES ARE OPEN AT THIS STAGE!!
- = ISOVOLEMIC CONTRACTION (volume does not change)
- Ventricles continue to contract
- Pressure rises
  - Eventually the “out” valve (e.g. aortic) is forced open
  - **THUS:** A jet of blood is squirted into the systemic circulation
  - THIS JET IS THE STROKE VOLUME
  - Now, the ventricle begins to relax
  - Pressure inside it falls
  - The back-pressure from the systemic circulation (e.g. pressure inside the aorta) forces the “out” valve closed.
  - Ventricle continues to relax until the pressure falls so far that the “in” valve is open again
  - **THUS,** FILLING FROM THE ATRIA COMMENCES AGAIN.

The cardiac output = (Stroke volume) times (Heart Rate)

**Summary of Training Effect on Heart Rate-Workload Relationship**

**PRELOAD:**
end-diastolic pressure just before contraction

**AFTERLOAD**
= pressure required to open the aortic valve

**End-diastolic LV volume:**
(= 70 ± 20 ml/m^2 s)

**End-systolic LV volume:**
(=25 ± 10 ml/m^2 s)

**Ejection Fraction:**
50% - 70%

**Normal atrial pressure:**
RA = 3-5 mmHg
LA = 5-10 mmHg

**Ventricles in diastole:**
1-3 mmHg
Up to 8-10 mmHg at the end of filling up

**NORMAL SYSTOLIC VENTRICLES:**
RV = 20-25 mmHg
LV = 110-130 mmHg

**NORMAL pulmonary artery pressure:**
= 25/12 mmHg
Normal Aortic Pressure:
= 120/80 mmHg,
all because the resistance is higher in the systemic circuit
**Molecular Biochemistry and Physiology of Myocardiocytes**

**The steps:**

The fast sodium channel waits for the arrival of an action potential at rest the voltage is –90 mV.

DEPOLARISES THE CELL (by allowing lots of Na+ into the cell)

The fast Na+ channel is ONLY ACTIVE FOR MILLISECONDS!

will not open again until the cell has reached –90 mV again

THIS DEPOLARISATION is the FAST UPSTROKE PHASE

Voltage activates the POTASSIUM CHANNEL

→ potassium rushes out of the cell

→ THUS: transient repolarisation

BUT: voltage opens L-type VOLTAGE GATED CALCIUM CHANNELS

→ CALCIUM RUSHES IN along concentration gradient

this (K+ out, Ca++ in) current maintains the flat PLATEAU

!! calcium influx opens Calcium-gated Calcium Channels !!

@ sarcoplasmic reticulum

THUS → MASSIVE INFLUX OF CALCIUM from the reticulum

THUS → CONTRACTION OCCURS

The calcium is then pumped out:

→ to the reticulum (SERCA ATPase)

→ to the outside (Na+/Ca++ exchanger)

(which means the Na+ ends up in the cell)

(thus → Na+ pumped out by Na+/K+ ATPase)

thus, the positive charge is removed from the inside of the cell, and it is ready to depolarise again when the fast NA channels re-activate and sit ready, waiting for an action potential

!! PACEMAKER CELLS DEPOLARISE SPONTANEOUSLY !!

= are much less negative (only –60mV) and thus THE FAST Na+ CHANNELS ARE PERMANENTLY CLOSED

THUS: no rapid upstroke! Relatively gentle upstroke instead, via Ca+ channels

**PACEMAKER CHANNEL** slowly depolarises the cell by slowly sucking Na+ back into it

**FRANK-STERLING PRINCIPLE:**

The more you stretch the sarcomeres, the more they will contract.

i.e. the more the heart fills, the more it will contract

If the sarcomere is overstretched, contractile force declines.

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Figure 1.15. Action potential of a pacemaker cell. Phase 4 is characterized by gradual, spontaneous depolarization owing to the pacemaker current (I). When the threshold potential is reached, at about –40 mV, the upstroke of the action potential follows. The upstroke of phase 0 is less rapid than in non-pacemaker cells, because the current represents Ca++ influx through the relatively slow calcium channels.
Relevant cardiac anatomy

The Pericardium: "A double-walled fibrous sack"

External Layer: tough and fibrous

Internal Layer: slippery, "serous" pericardium

Aortic Valve

Bicuspid Valve

Chorda tendinea

 Coronary Trigone

Cusp

Descending Aorta
About 25-30% of the human heart cell volume consists of mitochondria. THAT’S 30% of the crosssection! In contrast, mitochondrial make up less than 5% of the untrained skeletal muscle cell volume.

Thus: whereas skeletal muscle can survive for hours in hypoxia, the heart muscle RELIES ON OXYGEN TOO MUCH and thus will die rather quickly.

METABOLISM OF MUSCLE

4 chief sources of energy in order of importance:
- ATP: = 3-4 seconds of max contraction
- Phosphocreatine (PCr): PCr + ADP $\rightarrow$ ATP + Creatine = 10-15 seconds of max. contraction
- Glycogen (AEROBIC): Glycogen $\rightarrow$ lactic acid producing 3 ATP per glucose unit = 1-2 minutes

Cell biology: contractile properties of the MYOCARDIUM

Myocardium myocytes are quite small cells. They have to be, to let the oxygen diffuse more easily.

MAJOR DIFFERENCE from muscle cells: these myocytes can transfer their action potential to one another (and skeletal muscle cannot)- this is done via intercalated disks.

Intercalated Discs:
- contain anchoring desmosomes and gap junctions
- Desmosome: sites of attached between adjacent cardiac cells
- Gap Junction: essentially tiny holes in the disk

PHYSIOLOGY OF MUSCLE:

Skeletal muscle: composed from fibres of similar length.

Muscle Fibres are composed of multinucleated muscle cells.

Nuclei are on the outside while the inside is filled with myofibrils.

Myofibrils are composed of 2 proteins:
- Actin (thin)
- Myosin (fat)

Myosin heads bind to actin (ATP-powered)

This is ONE CYCLE; repeated cycles result in MUSCULAR CONTRACTION;
from rest it is a 50-fold increase in ATP consumption.

RELAXATION: Ca++ reabsorbed into sarcoplasmic reticulum (SERCA calcium pump is ATP-powered)
Control of contractility is achieved via the Calcium concentration

CAUSES OF HEART FAILURE

The underlying cause is the pathological process affecting the heart and leading to impaired myocardial pump function. A precipitating cause is a factor or event which results in decompensation of the heart and symptoms.

Typical precipitating causes are factors placing an additional load upon the heart such as
- fever,
- anaemia
- systemic infection.
- arrhythmias such as atrial fibrillation

potential underlying causes of heart failure:
- coronary artery disease, thus impaired blood supply
- myocardial infarction,
- valve disease, (thus increased haemodynamic load on the heart)
- cardiomyopathy.

Causes of dilated cardiomyopathy include
- alcohol abuse,
- previous myocarditis,
- hereditary defects in myocardial metabolism
- metabolic abnormalities such as hyper/hypo-thyroidism, or haemochromatosis.
- Occasionally drugs or heavy metal poisoning can cause cardiomyopathy.
- An important drug cause is the anti-cancer drug, adriamycin.

restrictive cardiomyopathy. These patients typically have thickened and stiff ventricular myocardium,
- due to fibrous infiltration or deposition of abnormal glycoproteins.
The most common cause in Australia is amyloidosis which is manifest mostly in older women.
Pharmacology, from the glorious mouth of the DEAN OF MEDICINE

Diuretics:

**Thiazides**: Inhibit Na-Cl cotransport in the early distal tubule

**K-sparing**: Inhibit reabsorption of Na in the late distal and cortical collecting tubule

**Loop diuretics**: Inhibit Na-K-2Cl cotransport in the thick segment of the ascending loop of Henle

**SIDE EFFECTS of THIAZIDE and LOOP DIURETICS**: Reduce volume, reduce NA+, K+, Ca++, Mg++ \( \rightarrow \) THUS: Confusion!!

INCREASED SERUM LDL, UREMIA, GOUT, DELIRIUM, alcalosis!!

Must titrate dose: no standard; observe patients condition and judge: try to hover between prune and blob

K-sparing diuretics will instead cause Acidosis and rash/pruritis

**BIOCHEMISTRY**

Increases intracellular calcium and allows more calcium to enter the myocardial cell during depolarization via a sodium-potassium pump mechanism; this increases force of contraction (positive inotropic effect), increases renal perfusion (seen as diuretic effect in patients with CHF), decreases heart rate (negative chronotropic effect), and decreases AV node conduction velocity.

**DIURETICS**

- **Cortex**
- **Medulla**
- **Loop of Henle**
- **Collecting tubule**

**THIAZIDES**: mild but powerful in combination cause increased excretion of Na, K, Ca, uric acid, HCO3

**K-sparing**: Weak, but spare potassium which is good if you want to avoid arrhythmia

**LOOP diuretics**

POWERFUL alone. beware: May excrete 15-20% of filtered Na+

**!! OBSESS !!**

**!! POTASSIUM !!**


**BEST EVER** for atrial fibrillation; **!! NARROW THERAPEUTIC RANGE !!**

may cause visual disturbance + arrhythmia (??)

**Effects:**

- Reduced serum noradrenaline (thus less sympathetic vasoconstriction)
- Reduced RAAS activity
- Reduced peripheral nervous activity
- INCREASED vagal tone

**LONG TERM: SURVIVAL SIMILAR TO PLACEBO**

FEWER hospital admissions, but...

MORE serious arrhythmias

MORE myocardial infarctions

**SIDE EFFECTS of DIGOXIN:**

Heart block, nausea, vomiting, diarrhoea, depression, disorientation, paraesthesia, blurred vision, scotomae, “yellow-green vision”, gynaecomastia.

**BIOCHEMISTRY**

= good in combination!

Thus, mix and match at will. BUT: 1 + 1 equals 50. So be careful.

DO NOT OVERDO IT

**GOOD COMBINATION:**

a loop diuretic + K sparing diuretic

**EFFECTS:** none on cardiac output, but certainly improves preload

**THIAZIDES**: mild but powerful in combination cause increased excretion of Na, K, Ca, uric acid, HCO3

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**LOOP diuretics**

POWERFUL alone. beware: May excrete 15-20% of filtered Na+

**!! OBSESS !!**

over **!! POTASSIUM !!**

Non-Glycoside Positive Inotropic Agents

\( \rightarrow \) **Adrenaline** and beta-adrenoceptor agonists

\( \rightarrow \) phosphodiesterase inhibitors (sympathomimicry)

INCREASE FORCE + RATE !!

great for resurrecting a massive acute MI: BUT NEVER FOR LONGER THAN 3 DAYS!!

**Only for SHORT TERM**

**!! CIRCULATORY SUPPORT !!**

you can kill the patient with these

**Aldosterone Inhibitors**, namely the great **SPIROLACTONE**

**SIDE EFFECTS**: gynaecomastia, renal failure

= a competitive antagonist of the aldosterone receptor

= DO NOT USE if the pt. has bad kidneys, hyperkalemia or metabolic acidosis
VASODILATORS:

These aren’t bad as the heart will require less effort to pump through a circulatory system which has less RESISTANCE:
BUT: You build up a resistance to them

NITRATES:
Not very important; only for acute MI with congestive failure
TOLERANCE DEVELOPS!
→ must abstain for 24hrs
DO NOT GIVE in hypotension

Angiotensin Converting Enzyme Inhibitors (These also vasodilate)

Angiotensin Converting Enzyme Inhibitors (ACEI)

MECHANISM OF ACTION

ACEI

ANGIOTENSIN II

A.L.D.O.

BRADYKININ

R N E R

K IN ase II

Angiotensin converting enzyme inhibitors improve survival
- they inhibit post-MI remodelling of the myocardium,
- they modify the progression of congestive heart failure
- they reduce the number of hospitalisations

ACEI UNDESIRABLE EFFECTS
- Inherent in their mechanism of action
  - Hypotension
  - Hyperkalaemia
  - Angioneurotic oedema
- Due to their chemical structure
  - Cutaneous eruptions
  - Neutropaenia, thrombocytopenia
  - Digestive upset
  - Dry cough
  - Renal Insuff.
  - Dysgeusia
  - Proteinuria

DO NOT GIVE ACE-Inhibitors in:
- Renal artery stenosis,
- renal insufficiency,
- hyperkalaemia,
- severe hypotension

β-ADRENERGIC BLOCKERS

CONTRAINDICATIONS
- Hypotension: BP < 90 mmHg
- Bradycardia: HR < 50 bpm
- Clinical instability
- Chronic bronchitis, ASTHMA
- ? Severe chronic renal insufficiency

START CAREFULLY, ON LOW DOSES; WITHDRAW SLOWLY, TITRATE CAREFULLY

Calcium Channel Blockers

Counter-ischaemic, and vasodilatory; reduce inotropy

Anticoagulants (for Atrial fibrillation, or previous Cerebrovascular accidents)
Antiarrhythmics eg. amiodarone if about to die from ventricular fibrillation

ALL ELSE FAILS: Implanted pacemaker