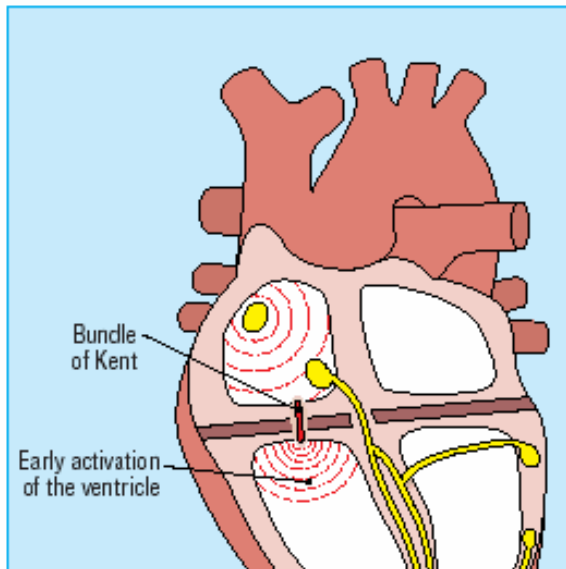
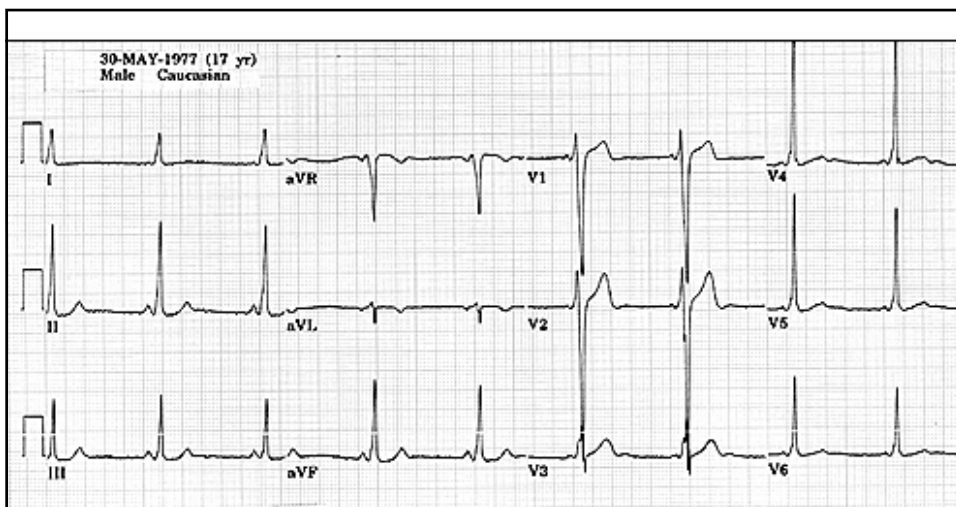


Wolff-Parkinson-White Syndrome



- The bundle of Kent provides a separate electrical conduit between the atria and the ventricles



Look at the QRS complexes in this ECG and see how these differ from a normal QRS ; also measure the PR interval



Wolff-Parkinson-White Preexcitation:

QRS complex represents a **fusion** between **two** ventricular activation fronts:

1. Early ventricular activation in region of the accessory AV pathway (delta wave, **Bundle of Kent**)
2. Ventricular activation through the normal AV junction, Hiss and bundle branch system

ECG criteria include all of the following:

1. Short PR interval ($<0.12s$)
2. Initial slurring of QRS complex (**delta wave**) representing early ventricular activation through normal ventricular muscle in region of the accessory pathway
3. Prolonged QRS duration (usually $>0.10s$)
4. Secondary ST-T changes due to the altered ventricular activation sequence

QRS morphology, including polarity of delta wave depends on the particular location of the accessory pathway as well as on the relative proportion of the QRS complex that is due to early ventricular activation (i.e., degree of fusion).

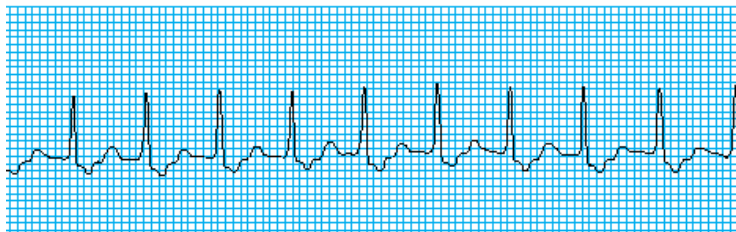
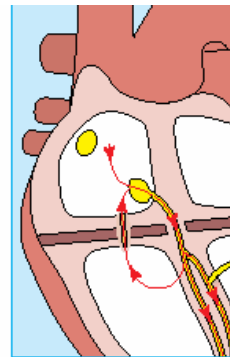
Delta waves, if negative in polarity, may mimic infarct Q waves and result in false positive diagnosis of myocardial infarction.

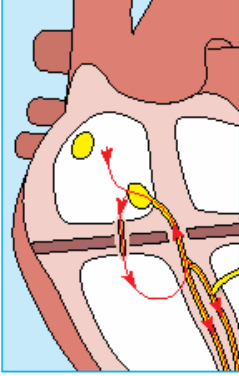
Orthodromic atrioventricular re-entrant tachycardias

Account for most tachycardias in the WPW syndrome.

A premature atrial impulse is conducted down the AV node to the ventricles and then in a retrograde fashion via the accessory pathway back to the atria.

The impulse then circles repeatedly between the atria and ventricles, producing a narrow complex tachycardia. Since atrial depolarisation lags behind ventricular depolarisation, P waves follow the QRS complexes. The delta wave is not observed during the tachycardia, and the QRS complex is of normal duration. The rate is usually 140-250 beats/min.

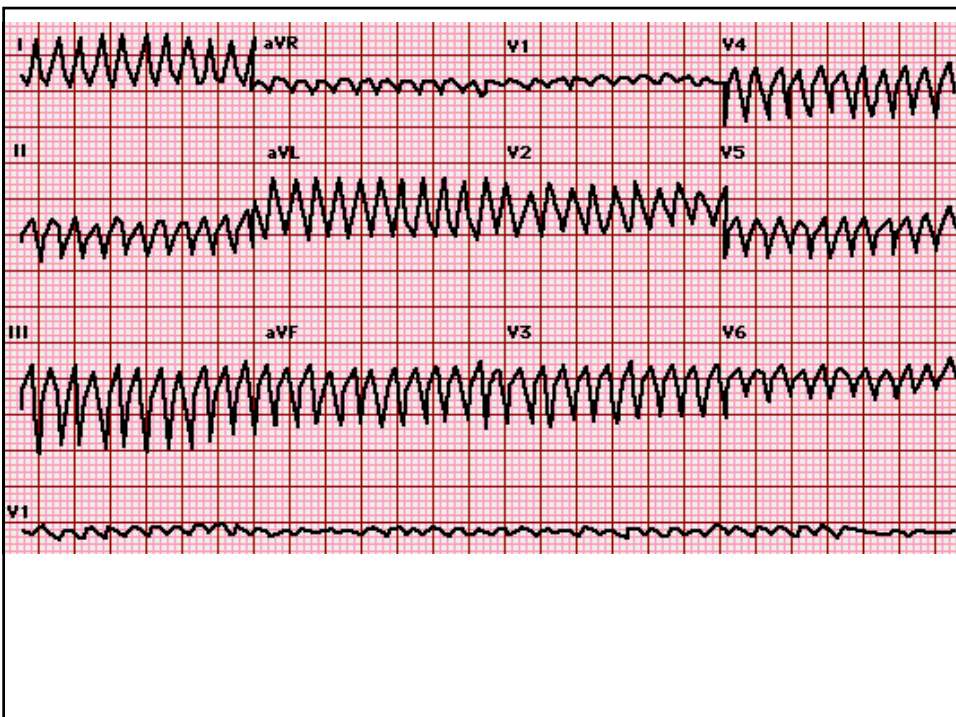
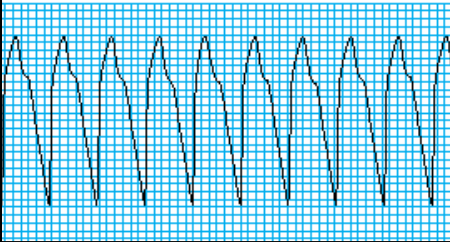


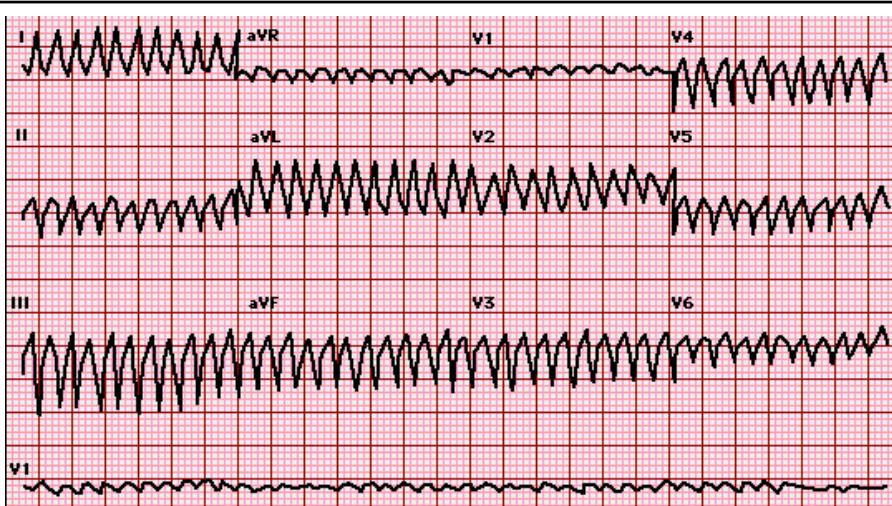


Antidromic atrioventricular re-entrant tachycardia

Is relatively uncommon, occurring in about 10% of Patients With the WPW syndrome. The accessory pathway allows antegrade conduction, and thus the impulse is conducted from the atria to the ventricles via the accessory pathway.

Depolarisation is propagated through non-specialised myocardium, and the resulting QRS complex is broad and bizarre. The impulse then travels in a retrograde fashion via the AV node back to the atria.

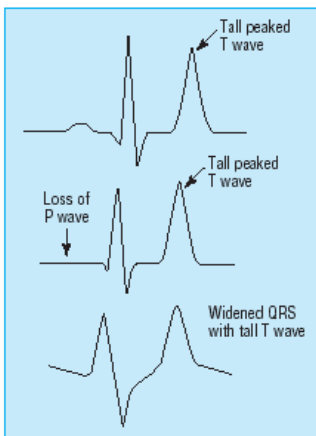




In patients without an accessory pathway the AV node protects the ventricles from the rapid atrial activity that occurs during atrial fibrillation. In the WPW syndrome the atrial impulses can be conducted via the accessory pathway, causing very rapid ventricular with broad QRS complexes with delta waves.

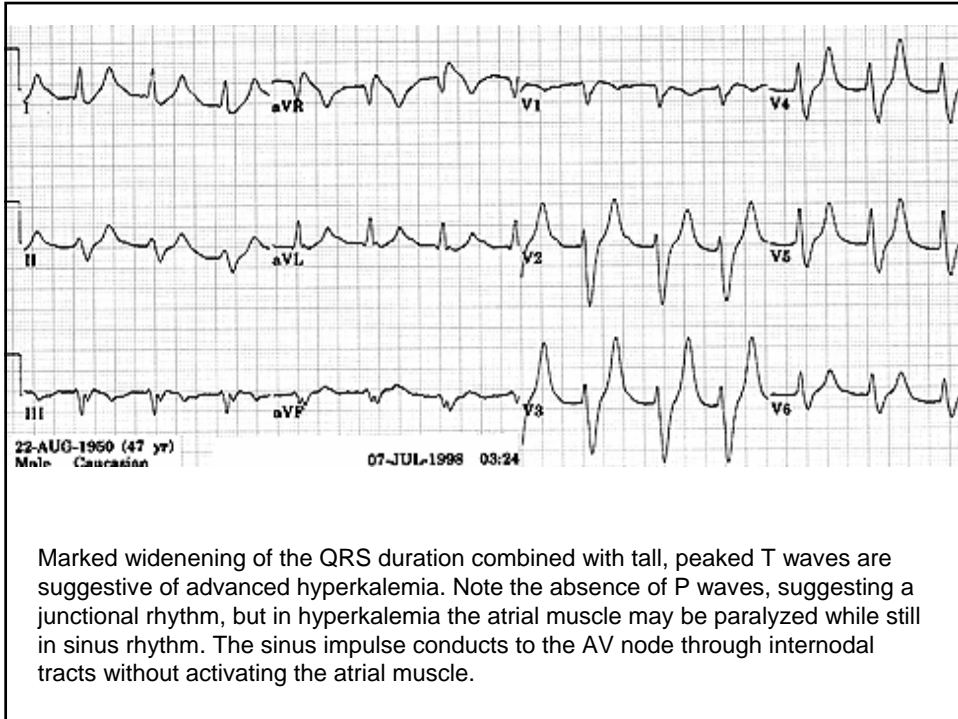
!!! DO NOT UDE MEDS THAT SLOW AV NODE CONDUCTION IN WPW TACHYCARDIAS (i.e.: Digoxin, BB, or CCB)!!!

ECG in Hyperkalaemia



Electrocardiographic features of hyperkalaemia

Serum K mmol/l)	Major change
5.5-6.5	Tall peaked T waves
6.5-7.5	Loss of P waves
7.0-8.0	Widening of QRS complex
8.0-10	Sine wave, ventricular arrhythmias, asystole



Treatment of Acute Hyperkalemia

- 1) Assess urgency
- 2) Stabilize myocardium: CaGluconate
- 3) Redistribute K⁺ from ECF to ICF
Ins/D50, Albuterol (high dose), NaHCO₃, ?diur
- 4) Remove K⁺ from body
Kayexalate, dialysis

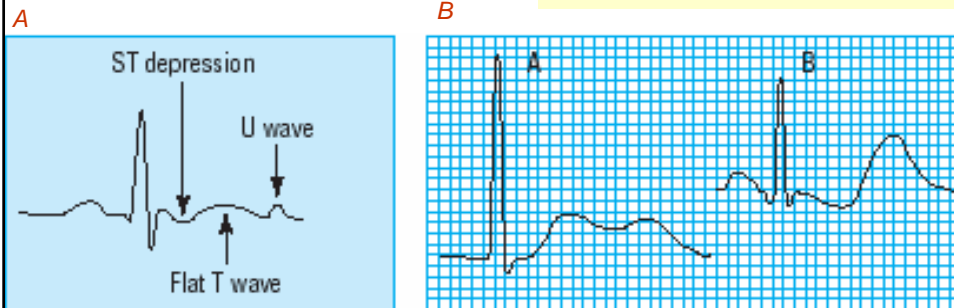
ECG in Hypokalaemia

A: Diagram of electrocardiographic changes associated with hypokalaemia.

B: Electrocardiogram showing prominent U wave, K concentration 2.5 mmol/l (A) and massive U waves with ST depression and flat T waves, potassium concentration 1.6 mmol/l (B)

Electrocardiographic features of hypokalaemia

- Broad, flat T waves
- ST depression
- QT interval prolongation
- Ventricular arrhythmias (PVC, torsades de pointes, VT, VF)

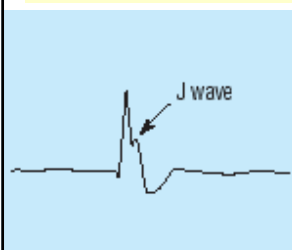


Hypothermia

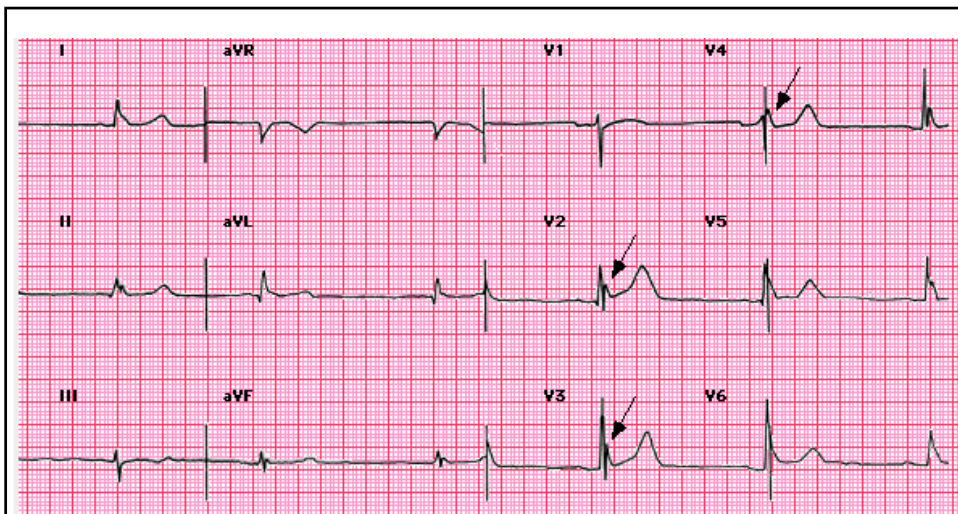
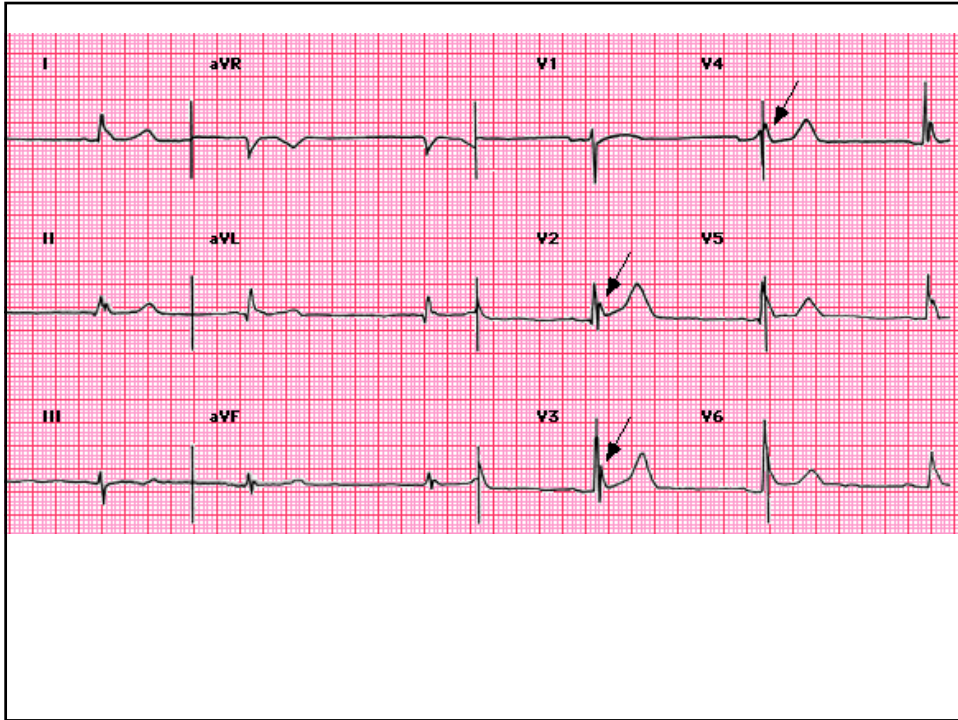
(core temperature less than 35°C)

Electrocardiographic features of hypothermia

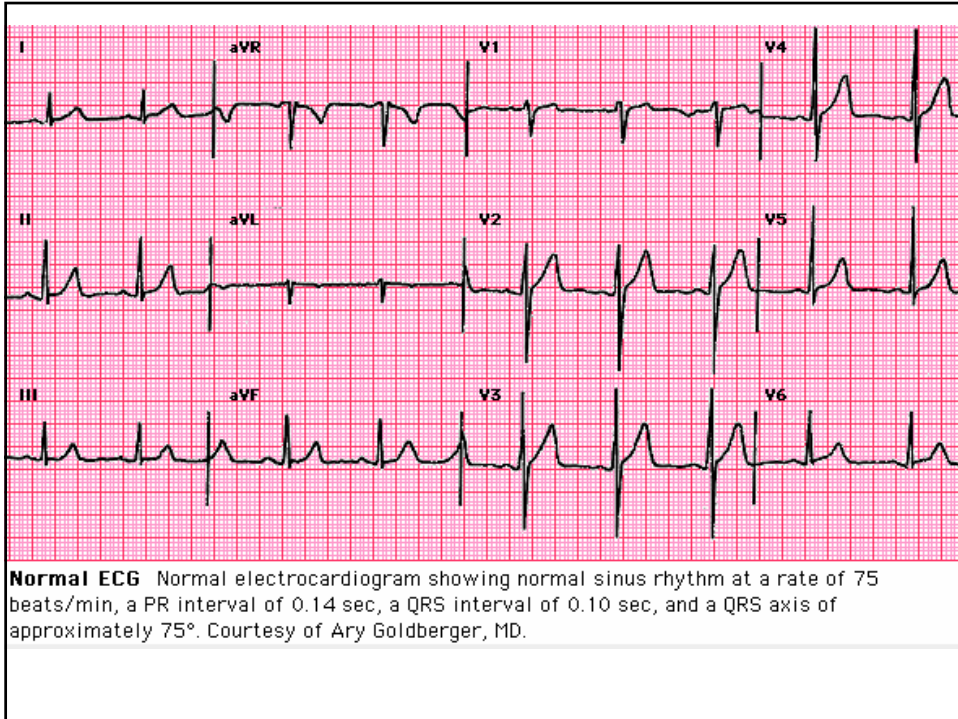
- Tremor artefact from shivering
- Atrial fibrillation with slow ventricular rate
- J waves (Osborn waves)**
- Bradycardias, especially junctional
- Prolongation of PR, QRS, and QT intervals
- Premature ventricular beats, ventricular tachycardia, or ventricular fibrillation
- Asystole



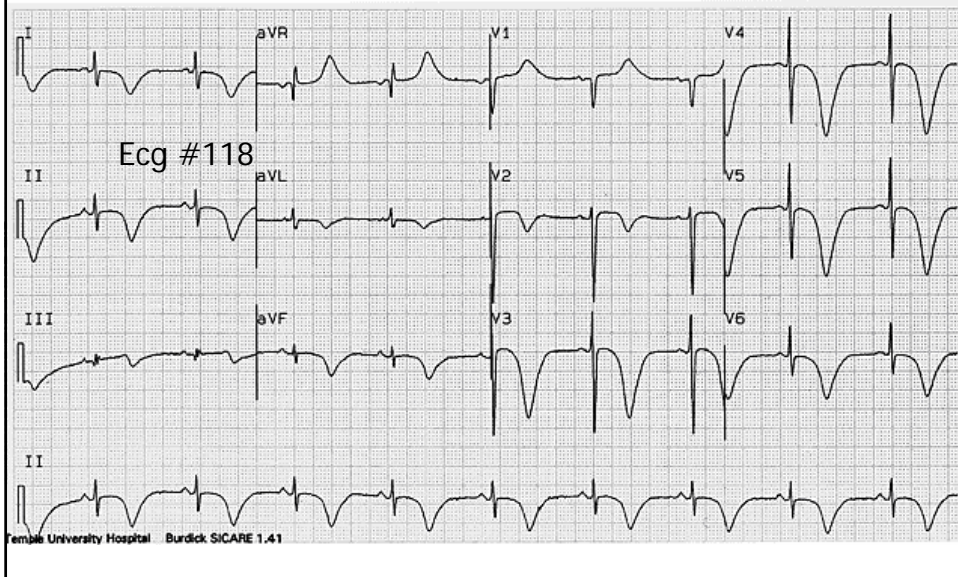
The J wave (Osborn wave) is the most specific electrocardiographic finding in hypothermia. It is considered by many to be pathognomonic for hypothermia, but it may also occasionally be seen in hypercalcaemia and in CNS disorders, including massive head injury and subarachnoid haemorrhage.



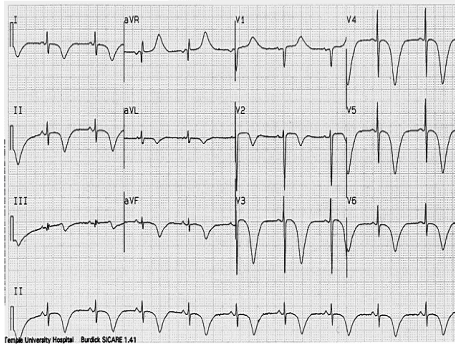
Electrocardiogram in hypothermia The ECG reveals marked sinus bradycardia (about 40 beats/min) with first degree atrioventricular block (PR interval = 0.23 sec). The slow heart rate in this patient is due to hypothermia (90°F, 32.2°C), which also produces prominent convex deflections at the J point (junction of QRS and ST segments) that are best seen in the precordial leads. The J waves or Osborn waves (arrows) are characteristic of severe hypothermia and resolve with rewarming; how they occur is not known. Courtesy of Arv Goldberger, MD.



40 yo with HA followed by coma



40 yo with HA followed by coma



- Striking finding of diffuse “giant” T waves
- Dx: SAH
 - CNS disease, classically SAH or herniation- look for co-existing QTc prolongation