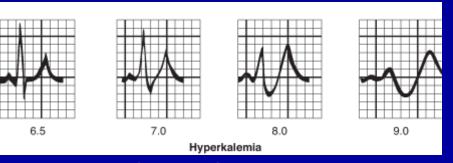
ECG Metabolic Abnormalities

Joe M. Moody, Jr, MD
UTHSCSA and STVAHCS

Electrolyte Disturbances with Significant ECG Effects

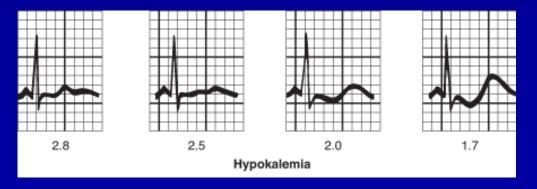
- Hyperkalemia, hypokalemia
- Hypercalcemia, hypocalcemia
- Hypothermia



- T waves become tall and peaked (>5.5)
- QRS widens uniformly (>6.5), QRS axis may shift either left or right
- Advanced hyperkalemia is same as dying heart
- Advanced hyperkalemia may give ST elevation
- P wave amplitude decreases, PR interval prolongs
- Sinoventricular conduction
- Concomitant hypercalcemia mitigates changes
- Concomitant hyponatremia worsens changes and hypernatremia mitigates

Example: Merck Manual Online

Hypokalemia



- Progressive ST segment depression > 0.5 mm
- Decrease in T wave amplitude
- Increase in U wave amplitude
 - ->1 mm
 - >T wave height in same lead
- No change in QT interval if measured before U wave
- Advanced hypokalemia T and U are fused
- Concomitant hypocalcemia: aggravates findings

Example: Merck Manual Online

Calcium

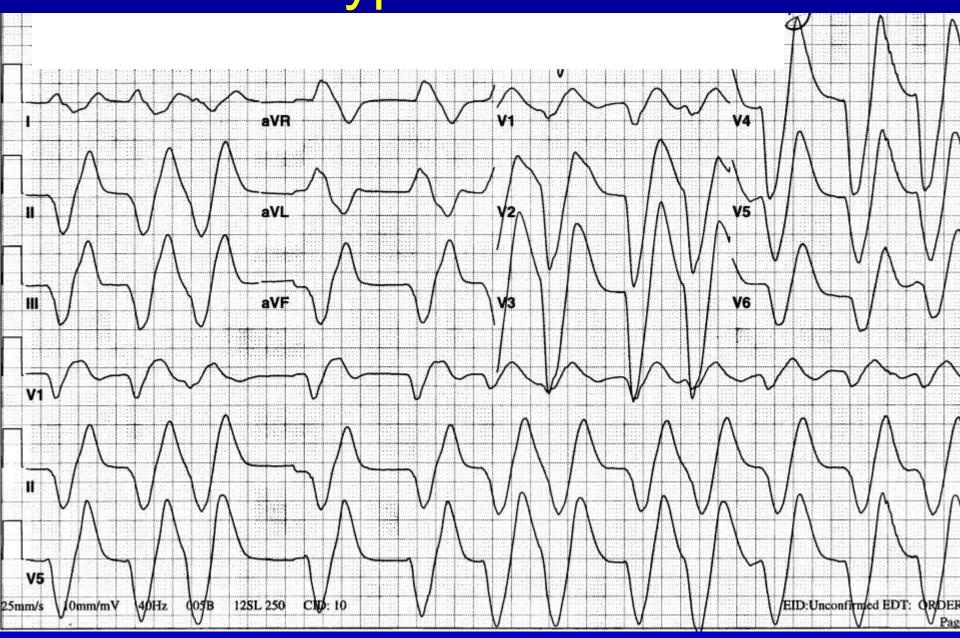
- Ionized calcium, so correct for albumin level
- Mainly change in ST segment duration, little change in T wave morphology
- Hypercalcemia shortens ST segment, so shortens the QaT (onset of QRS to apex of T)
- Hypocalcemia lengthens ST segment

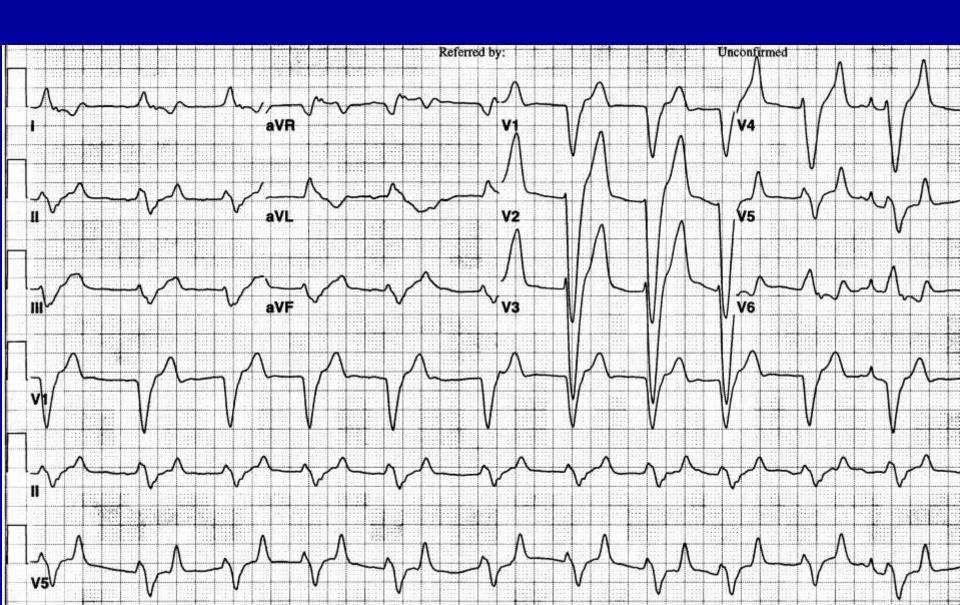
Situations that Don't Affect the ECG

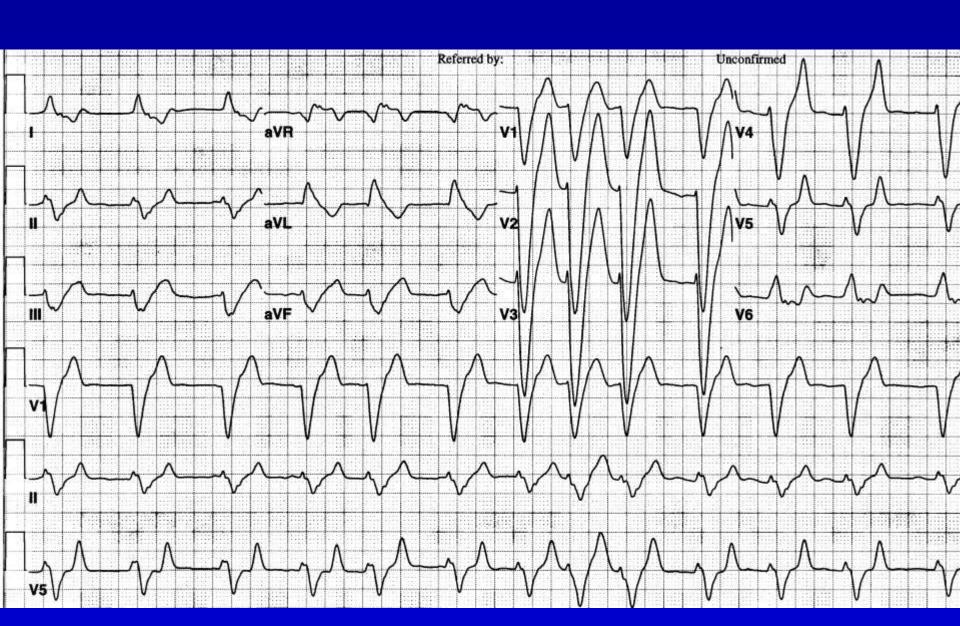
- Hyponatremia, hypernatremia
- Hypomagnesemia, hypermagnesemia
- Hyperthermia
- Alkalosis, acidosis
- Alcohol, coffee, tobacco

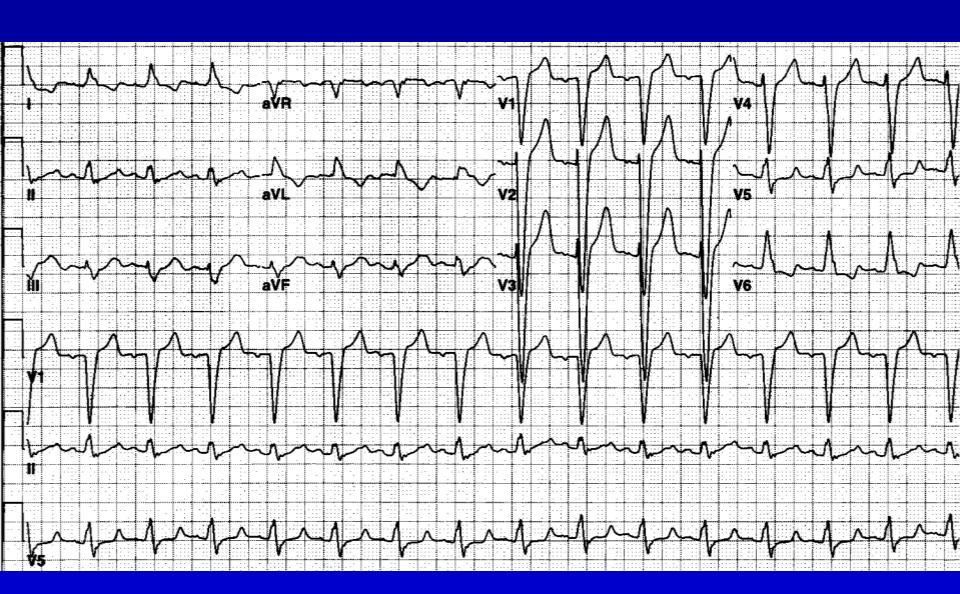
CNS Disorders

- Diffuse T inversion
- Particularly giant T inversion in precordial leads
- Prolongation of QT interval
- Can also have ST segment elevation or depression
- LV wall motion abnormalities have been described

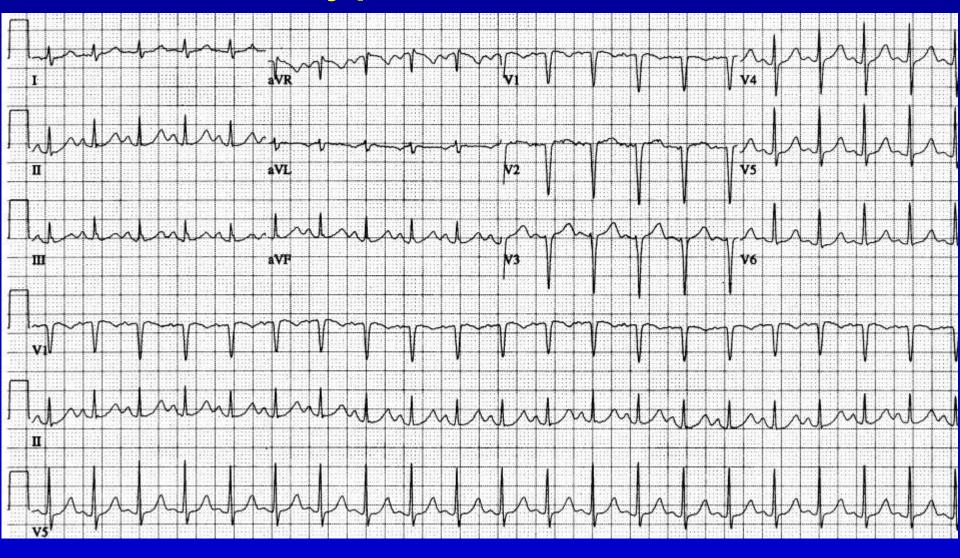




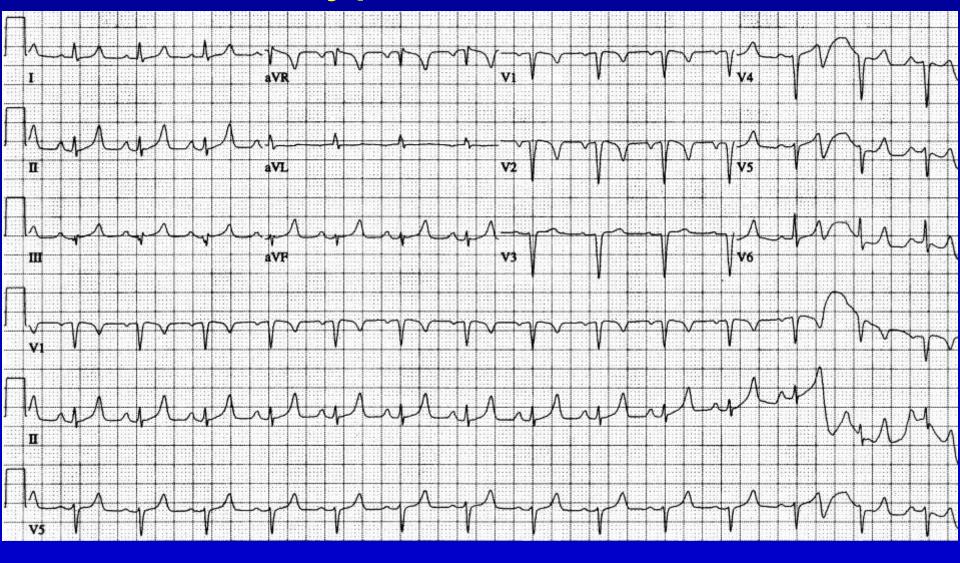




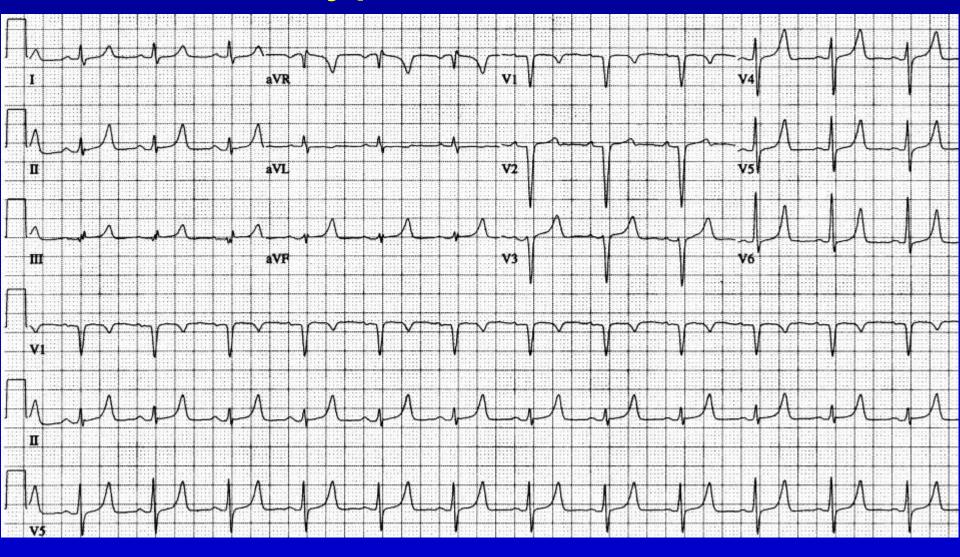
1996 Baseline Hyperkalemia case 2



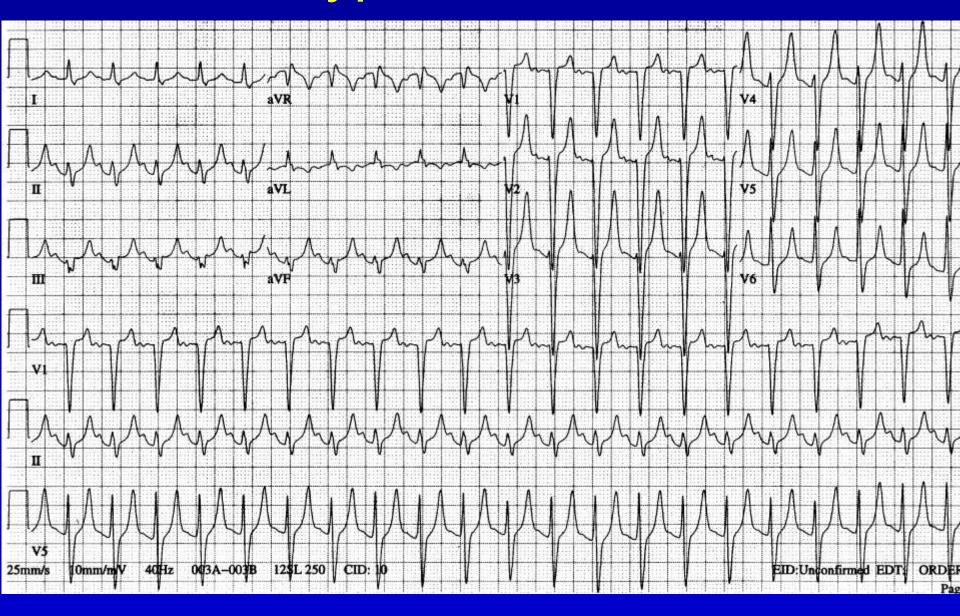
21 June 1998 Hyperkalemia case 2



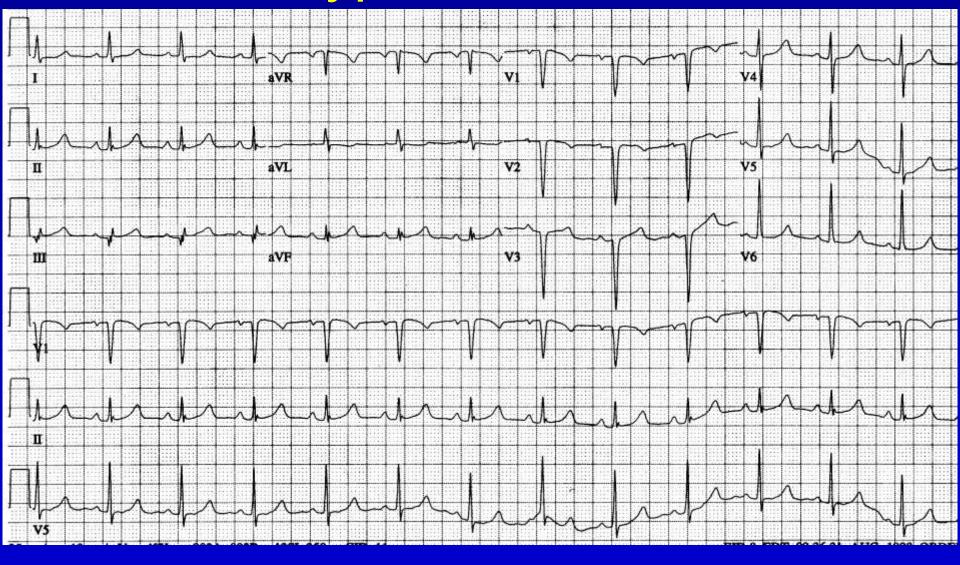
23 June 1998 Hyperkalemia case 2



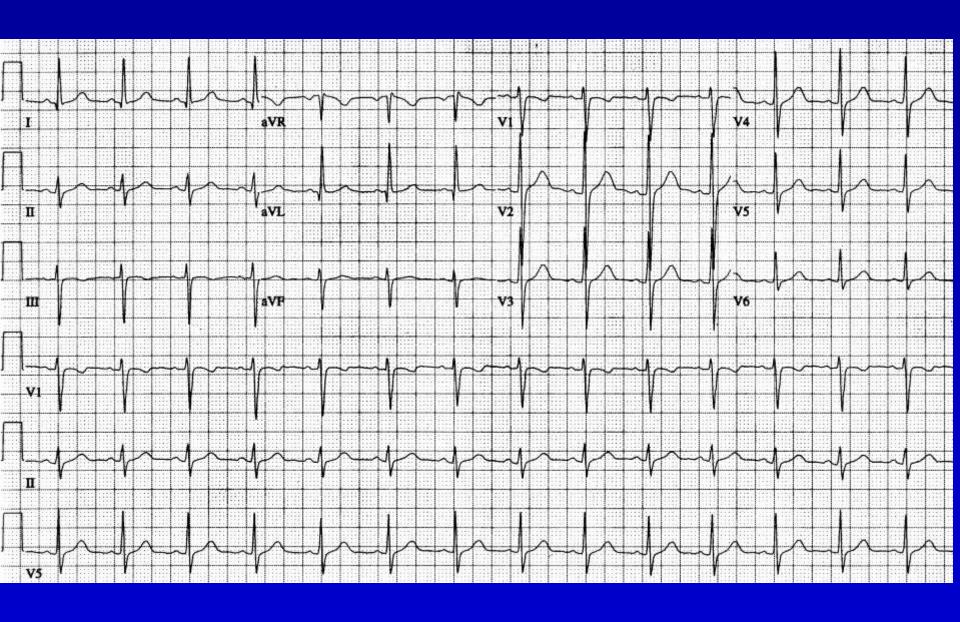
27 June 1998 Hyperkalemia case 2

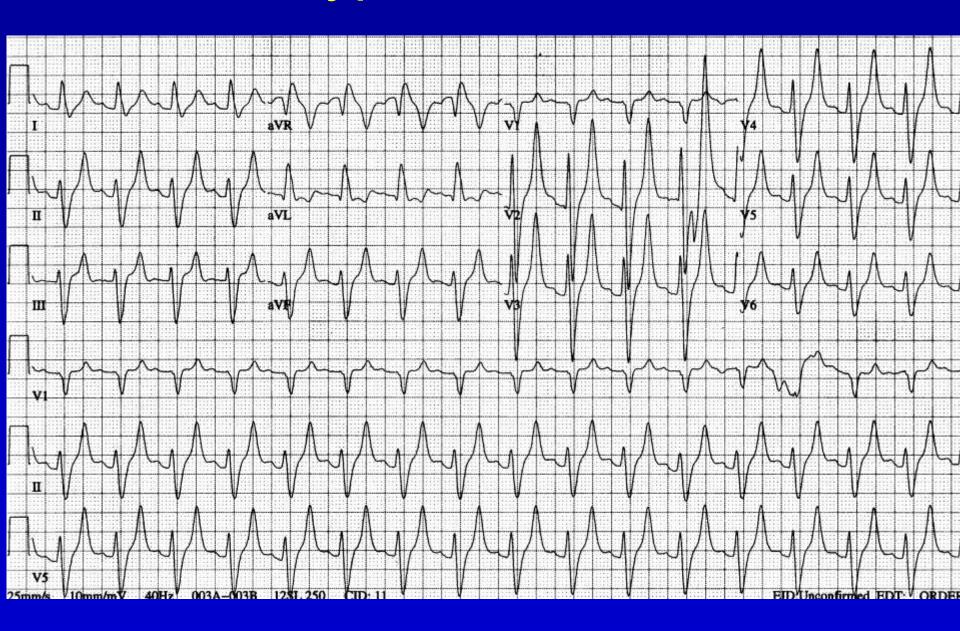


24 July 1998 Hyperkalemia case 2

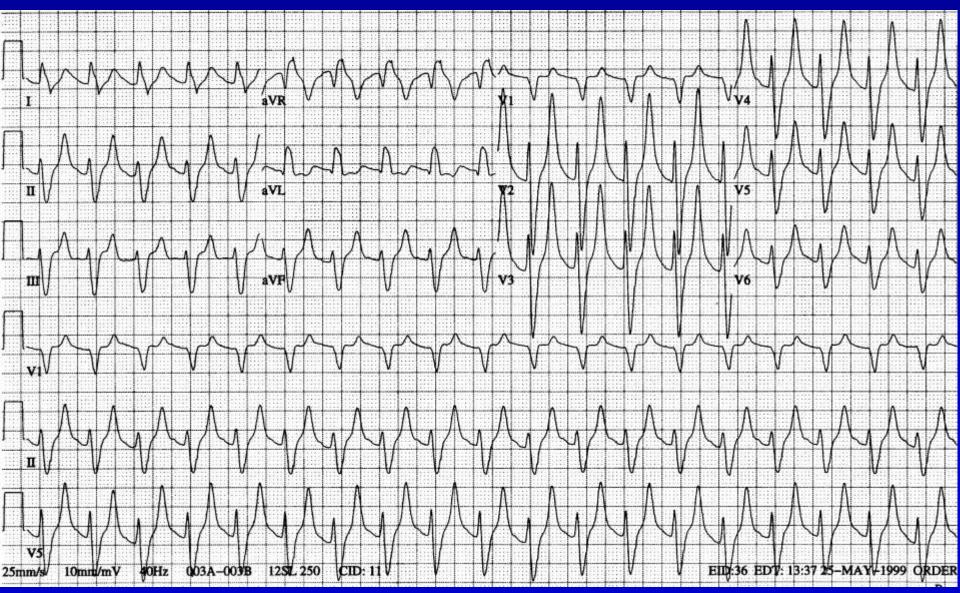


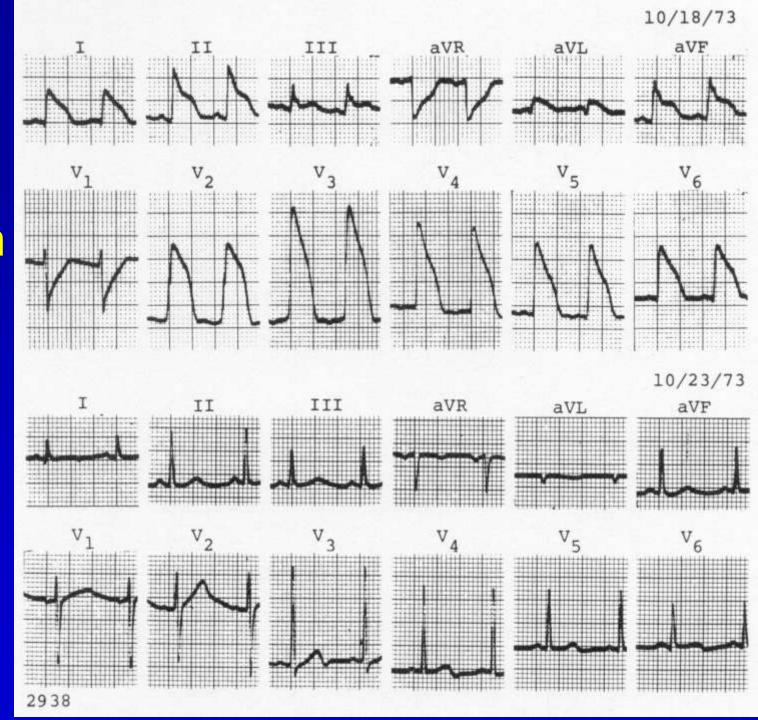
Baseline 1997 Hyperkalemia case 3





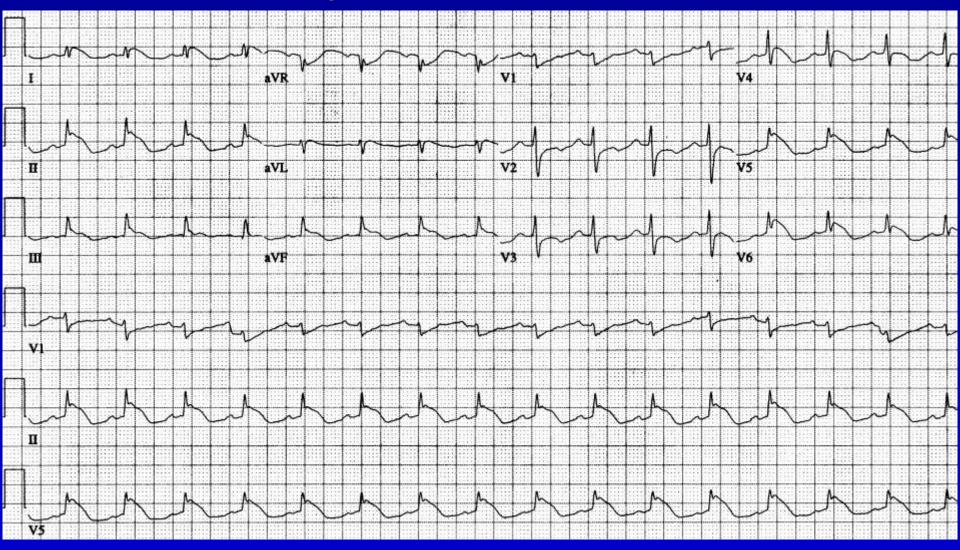
1999 plus 2 hours



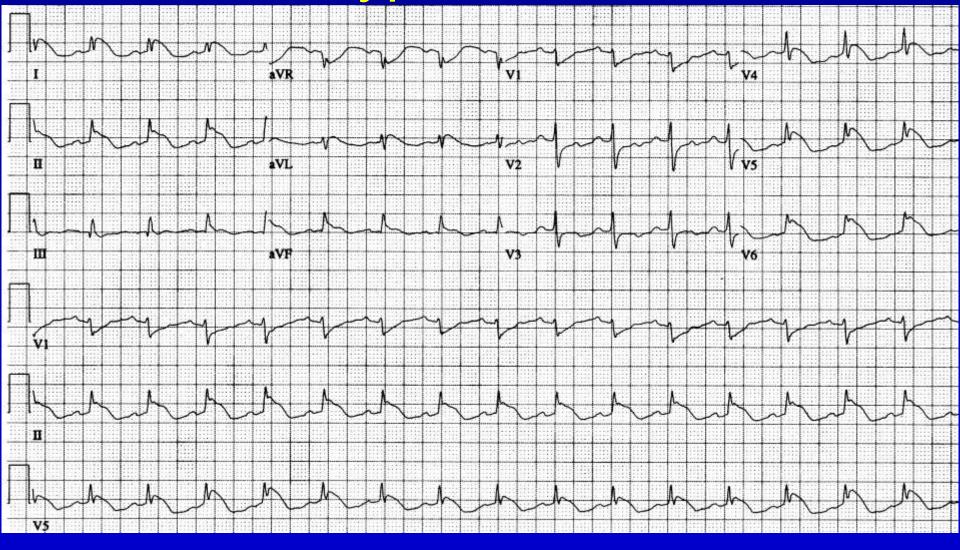


Surawicz, p. 520

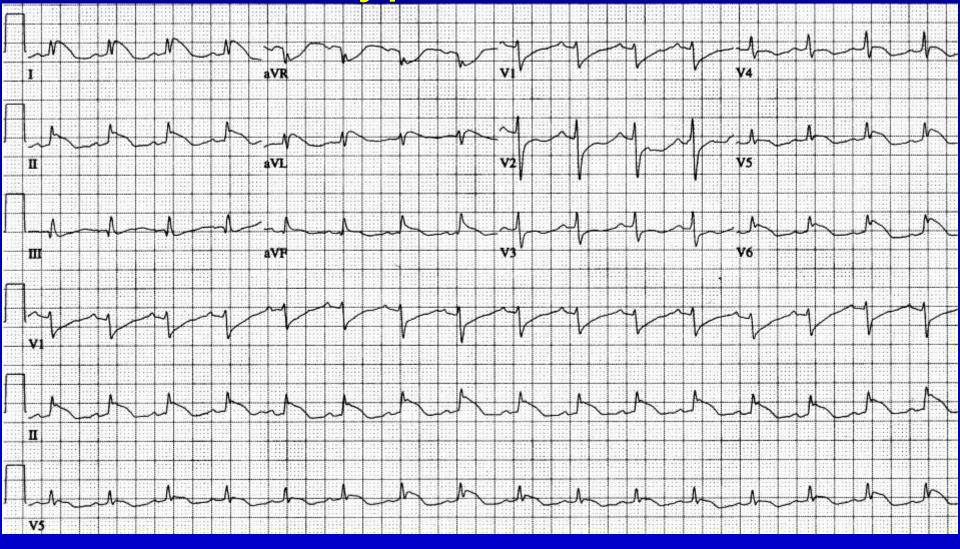
Initial

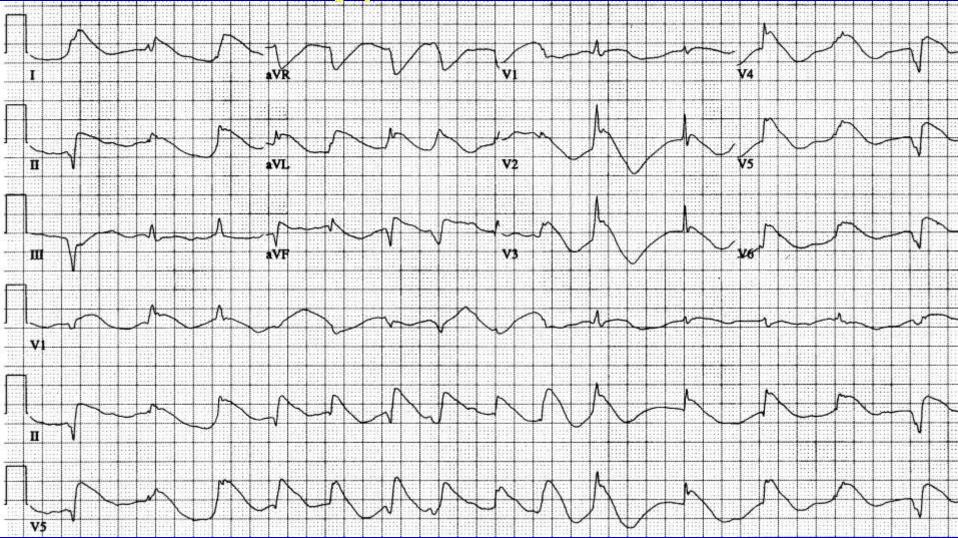


Plus 18 minutes Hyperkalemia case 4



Plus 36 minutes Hyperkalemia case 4

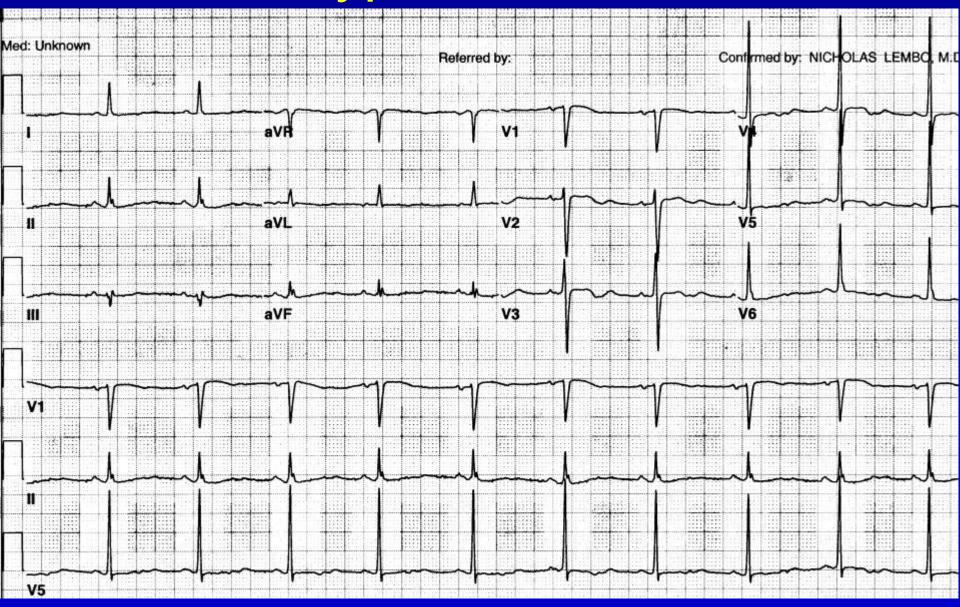


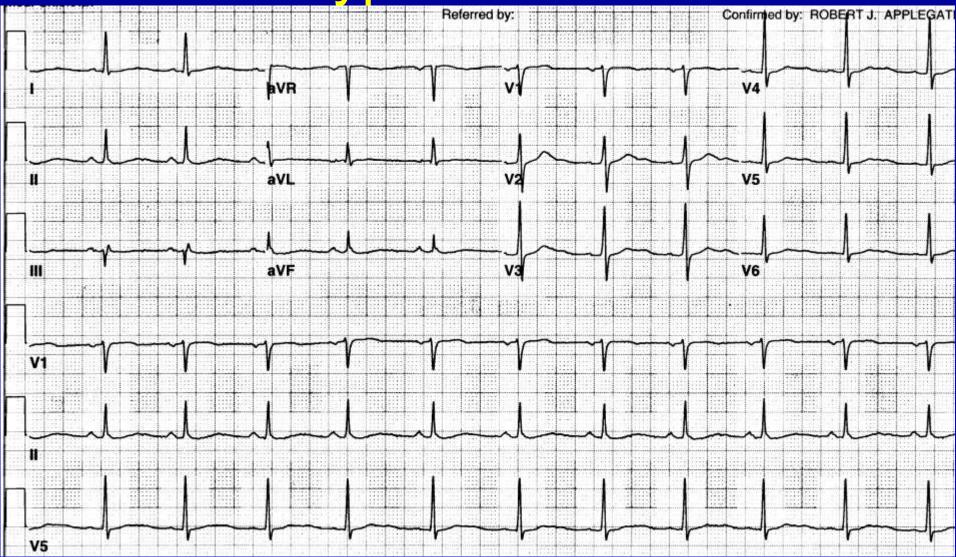


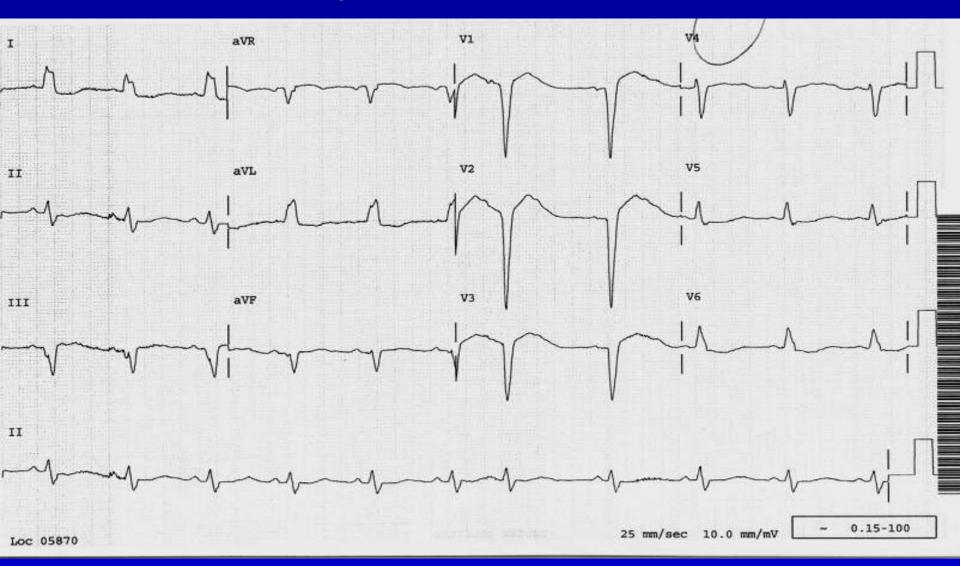




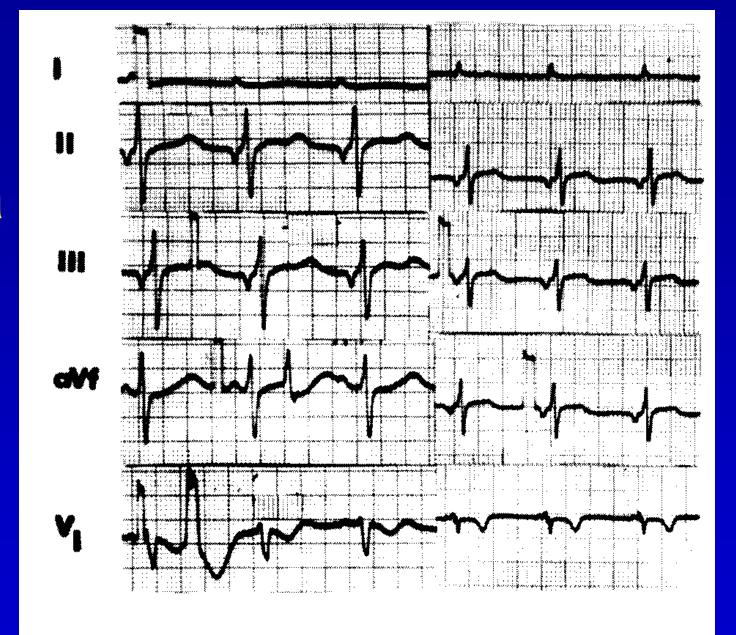
K unknown







Hypokalemia

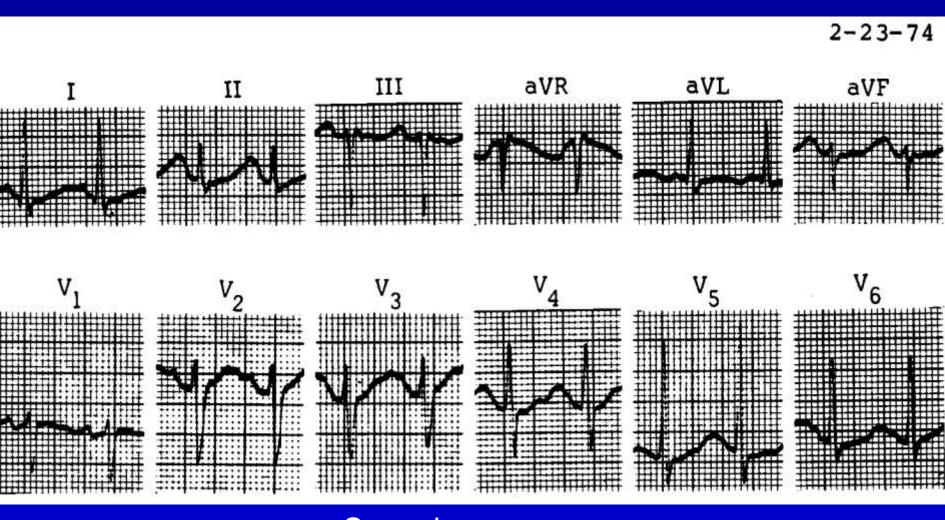


Surawicz, p. 526

Hypokalemia

Surawicz, p. 526

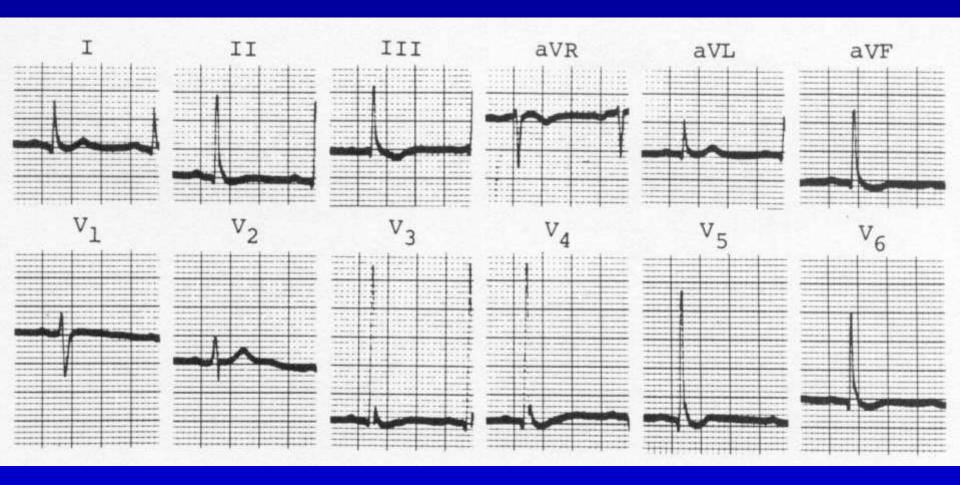
Hypokalemia



K = 2.4

Surawicz, p. 525

Hypercalcemia



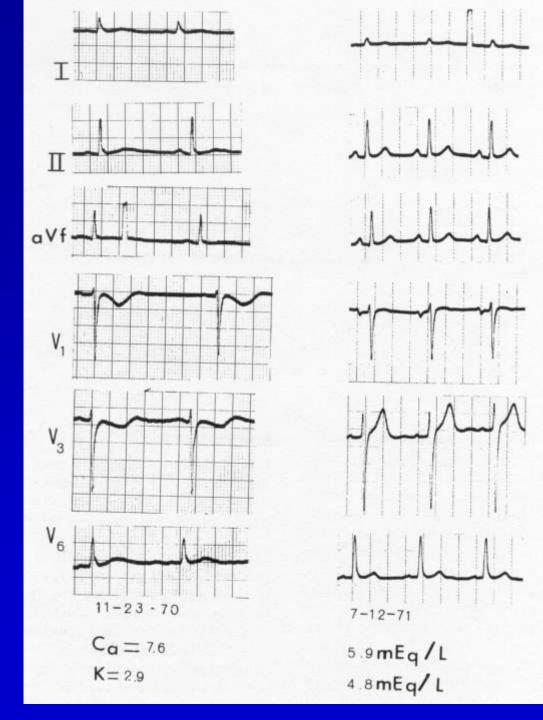
29-year old woman with lymphoma and bone involvement with Calcium 17.4

Surawicz, p. 529

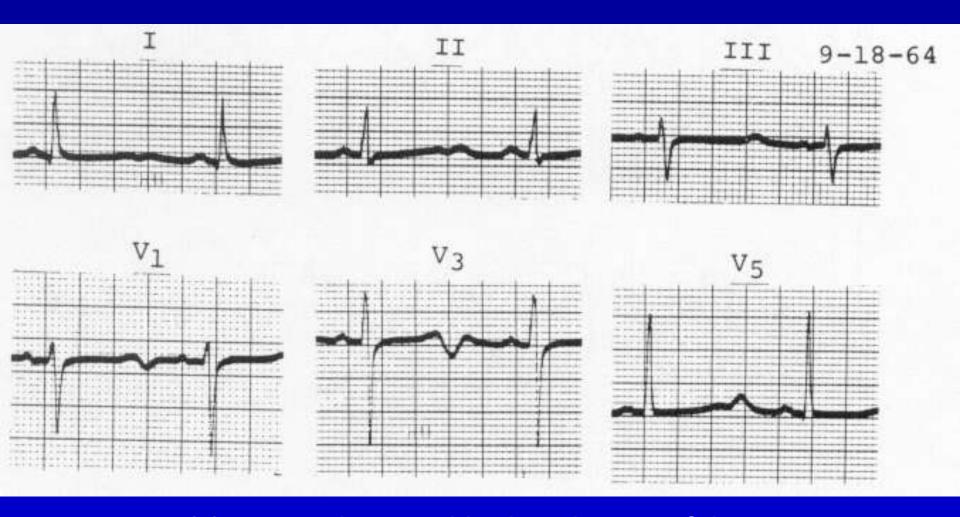
Hypercalcemia and Hypokalemia

41-year old man with multiple myeloma, later normalized

Surawicz, p. 530

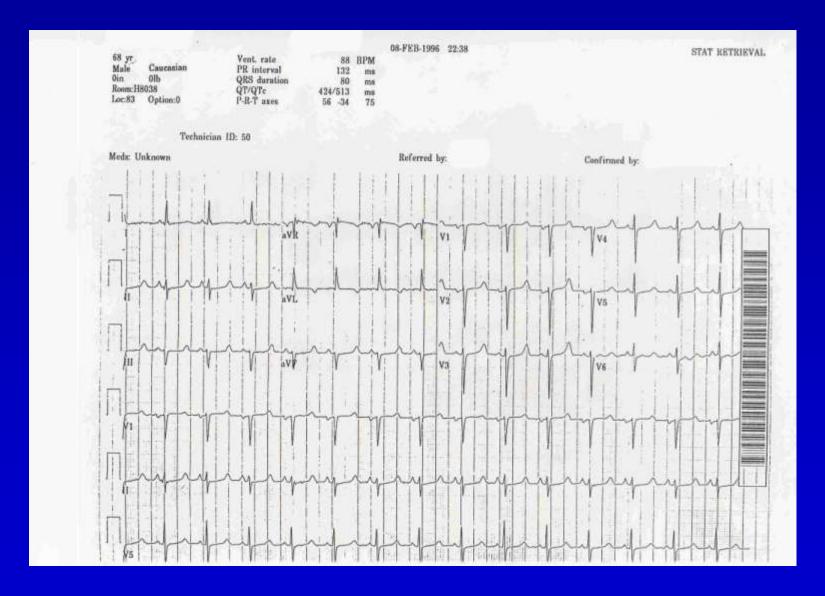


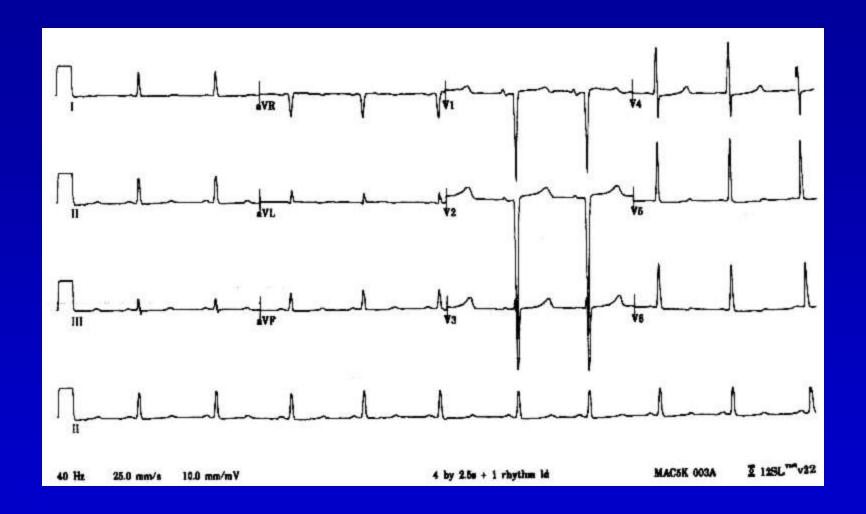
Hypocalcemia

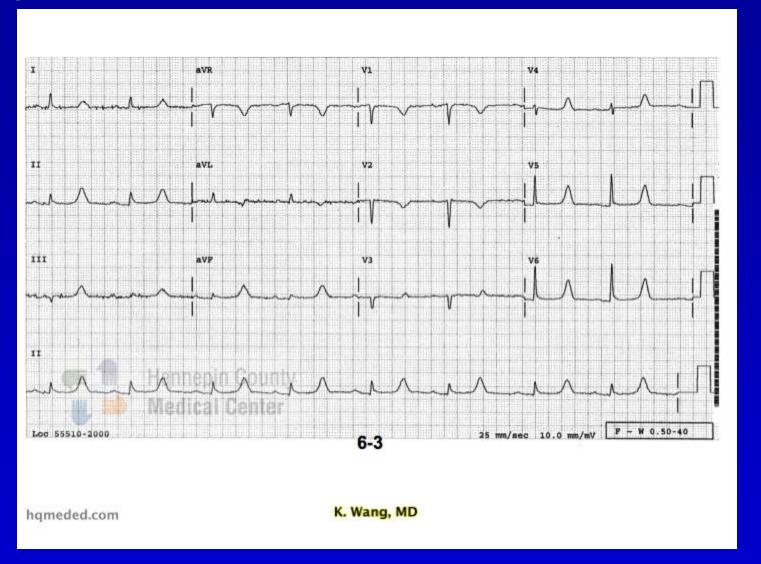


31-year old man with chronic renal failure Surawicz, Calcium 5.8 and K 3.3 p. 528

Hypocalcemia



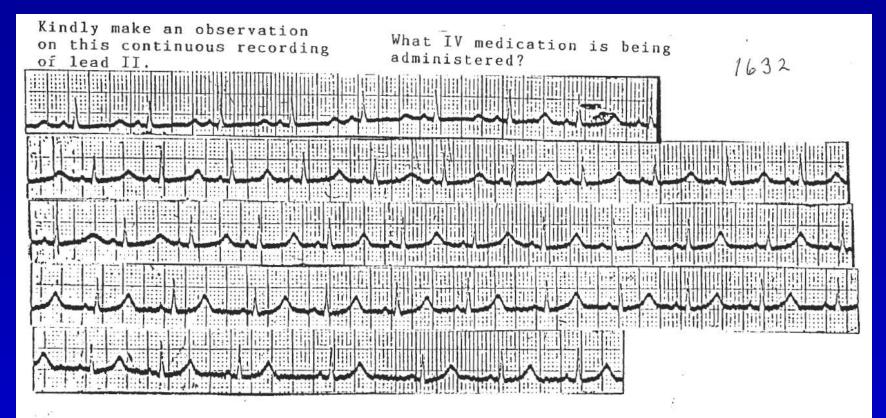


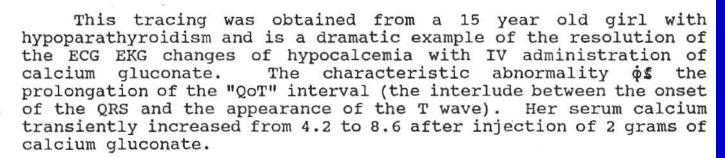


From hqmeded.com Dec 2010

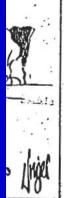
William Nelson, MD

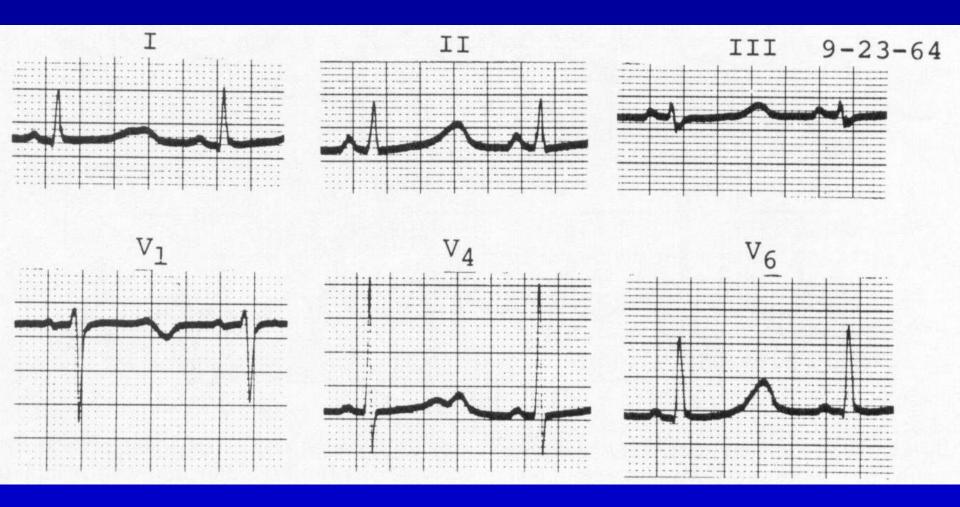
Hypocalcemia



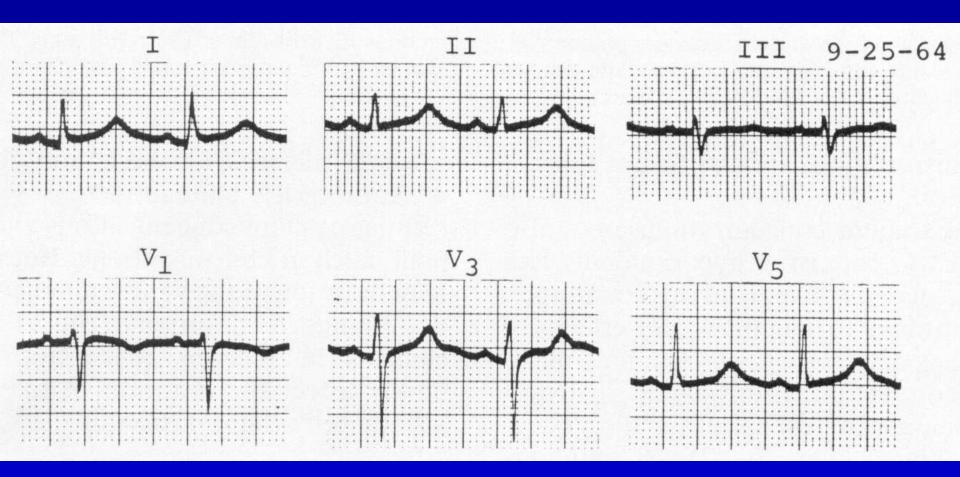


Kind of nifty - huh?



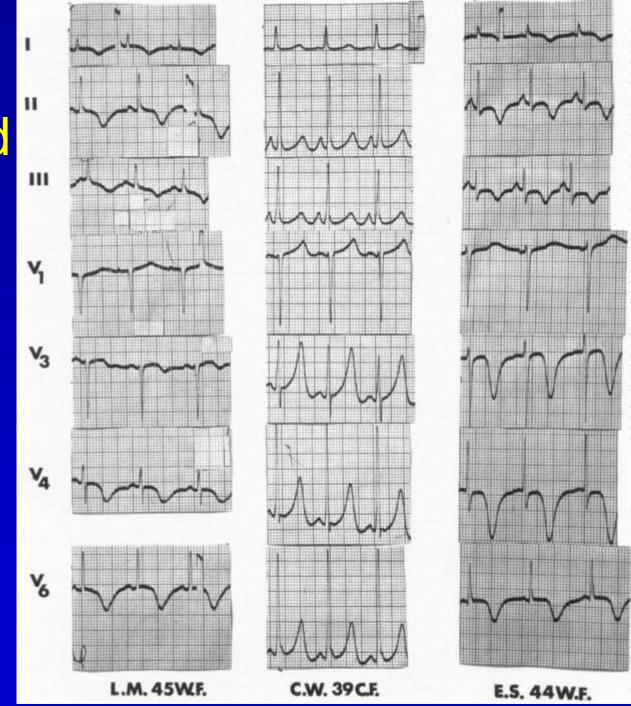


31-year old man with chronic renal failure Surawicz, K now down to 2.8 p. 528



31-year old man with chronic renal failure Surawicz, K now up to 3.5 and Calcium up to 6.5 p. 528

CNS: Subarachnoid Bleed



Surawicz p. 534

Dig Intoxication

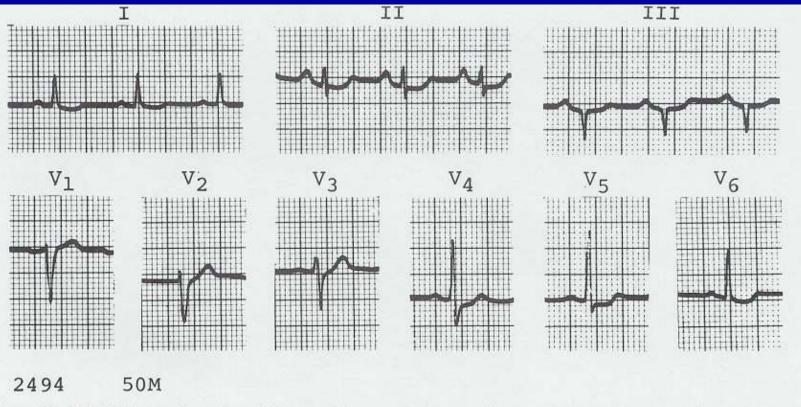
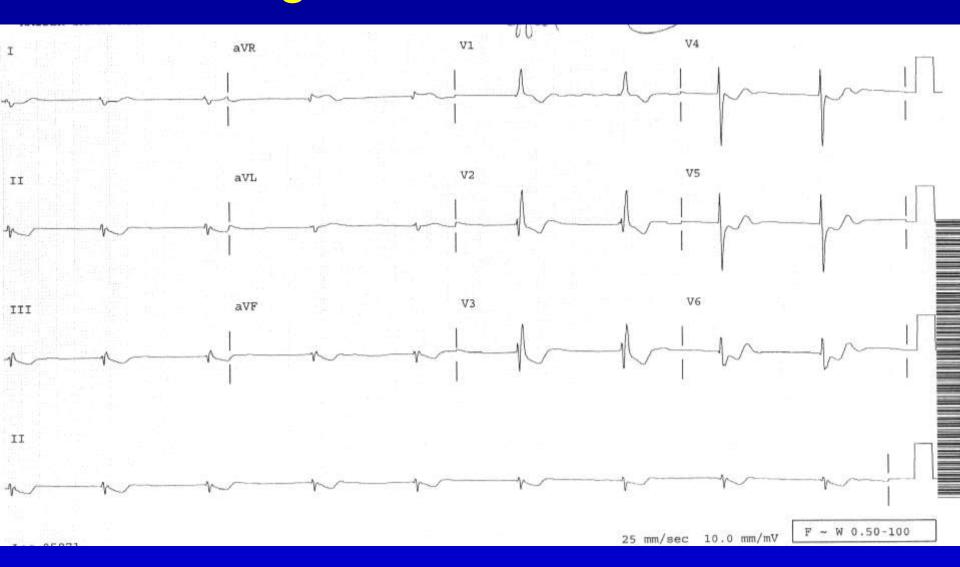


Figure 21-16. Digitalis effect in a 50-year-old man with no evidence of heart disease who took about 5 mg of digoxin in a suicide attempt 1 day before the tracing was recorded. The plasma digoxin level at the time of the recording was 4.1 ng/ml. Note the sagging of the ST segment, especially in leads I, II, and V_4 through V_6 . The T waves in leads II and V_5 are biphasic. No arrhythmia was observed.

Digitalis Intoxication



Short QT; Increase in automaticity; decrease in AV conduction

Hypothermia

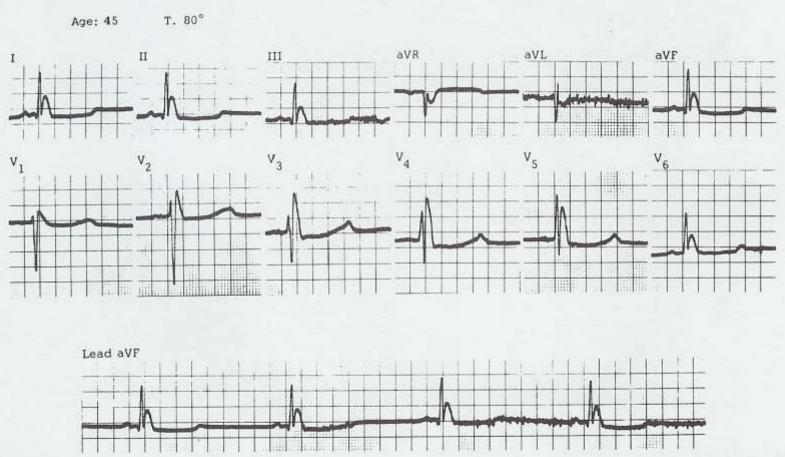


Figure 22–17. Hypothermia. The body temperature was 80°F. Note the J waves in all leads, with prolonged QT interval and low T waves. Intermittent baseline oscillation is present as a result of somatic muscle tremors. The heart rate is 32 beats/min.

Procainamide intoxication

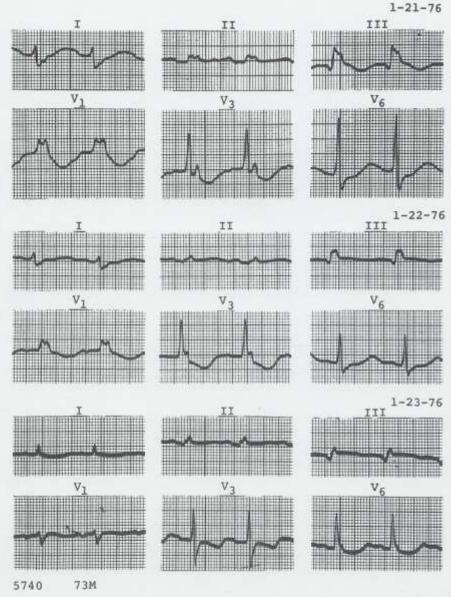


Figure 21–5. Procainamide intoxication in a 73-year-old man who was given procainamide (6 mg/m on 1/21/76. The tracing recorded on that date reveals first-degree AV block and marked prolongation QRS complex, with a duration of 0.20 second. The morphology of the QRS complexes resembles tright bundle branch block (RBBB). Additional primary ST and T wave changes are present. On 1/22/76 procainamide was discontinued, the PR interval and QRS duration have shortened. There are fewer ST wave changes. The QT interval, which now can be measured more accurately, is prolonged. On the day (1/23/76) the PR interval is within normal limits. The RBBB pattern is no longer present, and the duration has decreased to 0.12 second. ST and T wave abnormalities are still present, especially precordial leads.

Disopyramide Effect

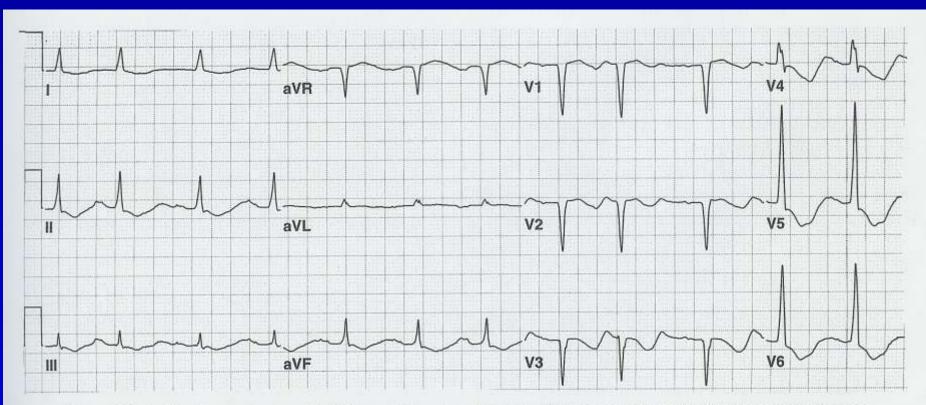


Figure 21–6. Electrocardiogram of a 68-year-old woman treated with disopyramide for atrial tachyarrhythmias shows sinus rhythm with premature atrial complexes, notched P wave, QRS duration of 96 ms, and QTc of 526 ms. Torsade de pointes occurred on the same day this ECG was recorded.

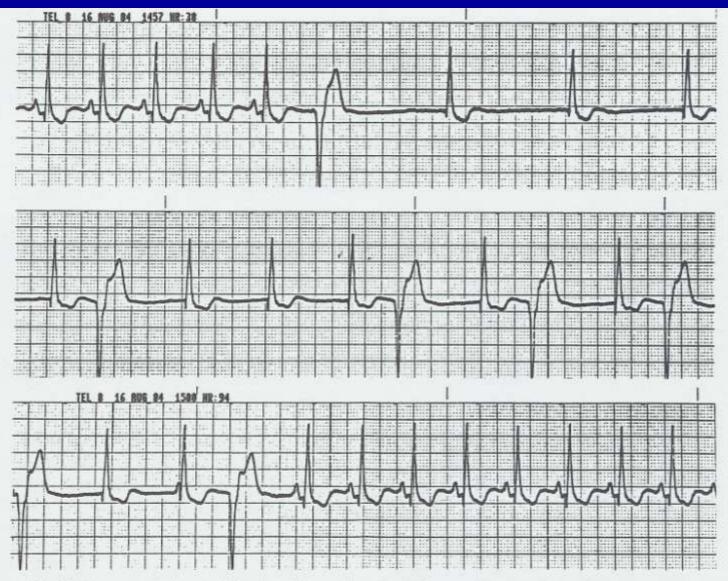


Figure 21–7. Sinus arrest due to lidocaine. The patient is a 56-year-old man who underwent aortocoronary artery bypass surgery. Lidocaine (100 mg) was administered intravenously as a bolus postoperatively for treatment of ventricular arrhythmias. The tracing was recorded 2 minutes after the injection. Note the normal sinus rhythm followed by the appearance of sinus arrest with junctional escape rhythm and premature ventricular complexes. Normal sinus rhythm resumed after about 3 minutes.

Propafenone Effect

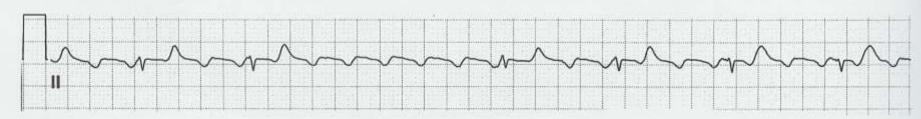


Figure 21–8. Electrocardiographic rhythm strip of lead II shows atrial flutter at a rate of 180 beats/min with 4:1 ventricular response in a patient treated with propafenone. Before propafenone the rate of atrial flutter was 244 beats/min.

Amiodarone Effect

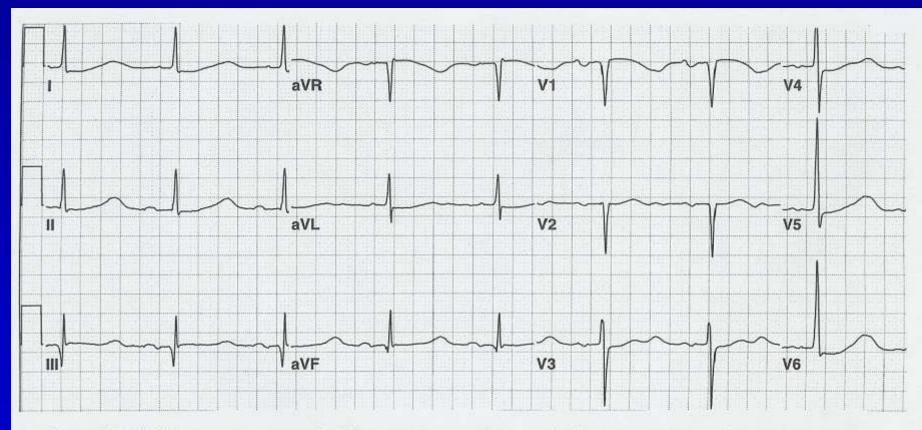


Figure 21–10. Electrocardiogram of a 72-year-old woman treated with oral amiodarone following electrocardioversion of atrial fibrillation to sinus rhythm. The wide P wave suggests left atrial enlargement. The PR interval is 190 ms and the QRS duration 104 ms. The QT interval appears prolonged but is difficult to measure because of fusion of the T wave with the U wave. In lead aVL the U wave amplitude is low, and there is a notch between the end of the T wave and the onset of the U wave. The QT interval in lead aVL is 520 ms. The duration of the QT+U interval is 716 ms.

Tricyclic Overdose

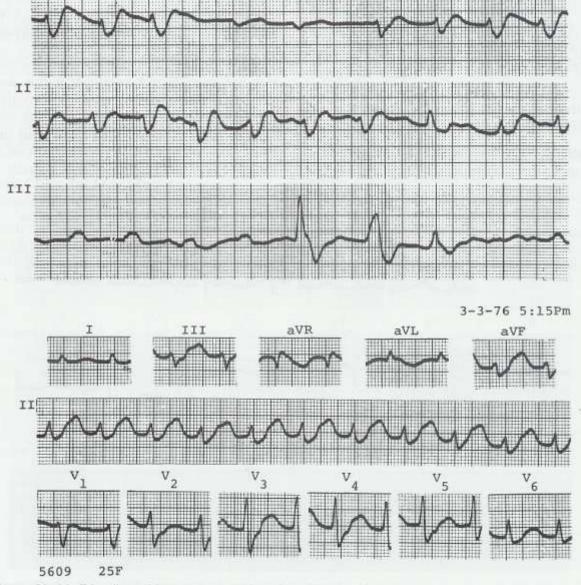


Figure 21–14. Tricyclic antidepressant overdose in a 25-year-old woman who took 500 mg of Tofranil (imipramine) 4 hours before the 5 P.M. tracing was recorded. The three standard limb leads show wide QRS complexes of varying morphology. The exact rhythm cannot be determined. She was severely hypotensive at this time. Fifteen minutes later the tracing shows probable supraventricular rhythm with intraventricular conduction defect.

Digitalis Effect/Toxicity

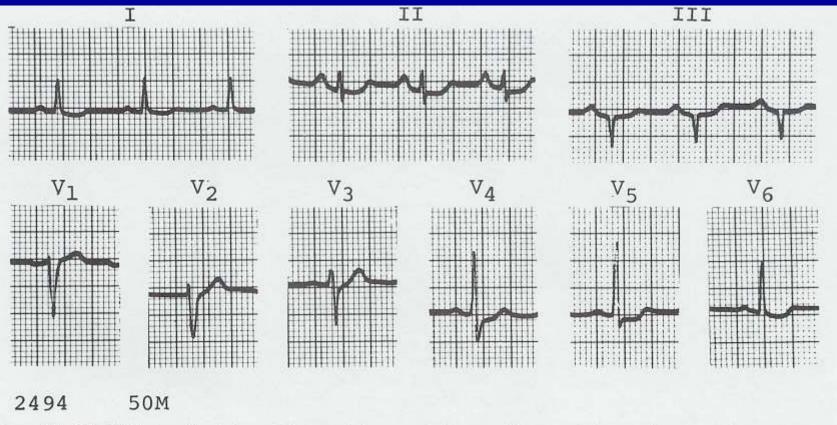


Figure 21–16. Digitalis effect in a 50-year-old man with no evidence of heart disease who took about 5 mg of digoxin in a suicide attempt 1 day before the tracing was recorded. The plasma digoxin level at the time of the recording was 4.1 ng/ml. Note the sagging of the ST segment, especially in leads I, II, and V_4 through V_6 . The T waves in leads II and V_5 are biphasic. No arrhythmia was observed.

Hypocalcemia and hyperkalemia

