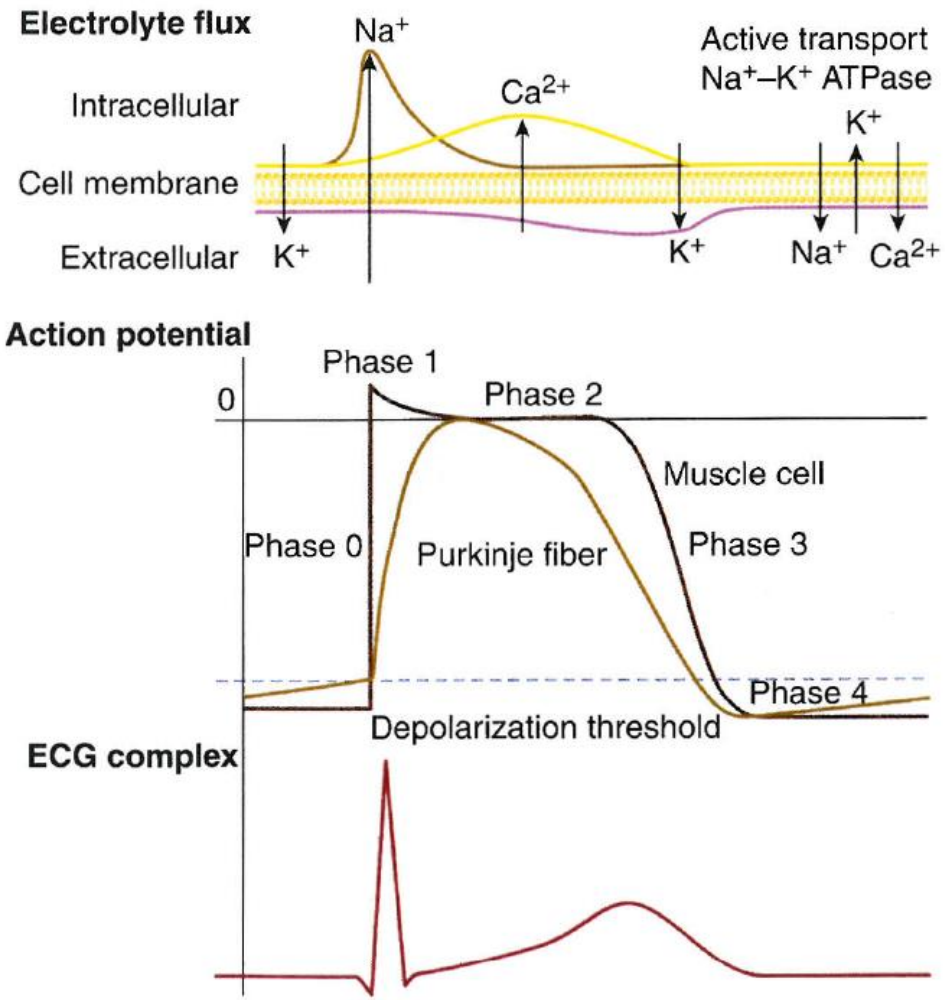
Common ECG Changes in Poisonings

Relationship of electrolyte movement across the cell membrane to the action potential and the surface ECG recording.



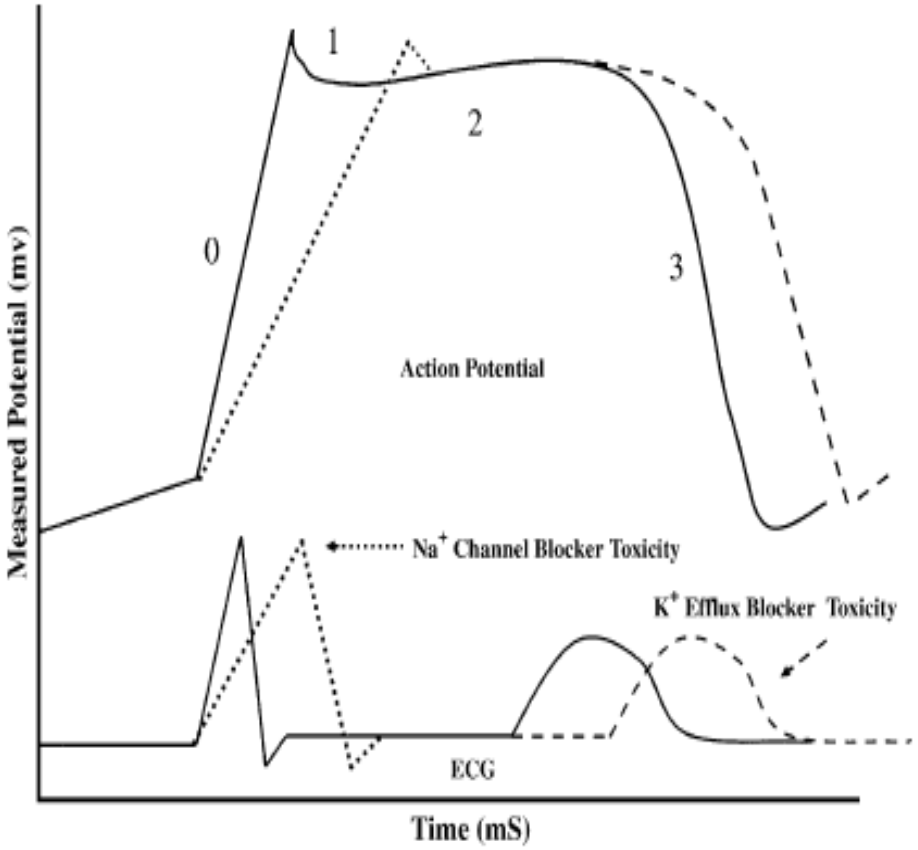
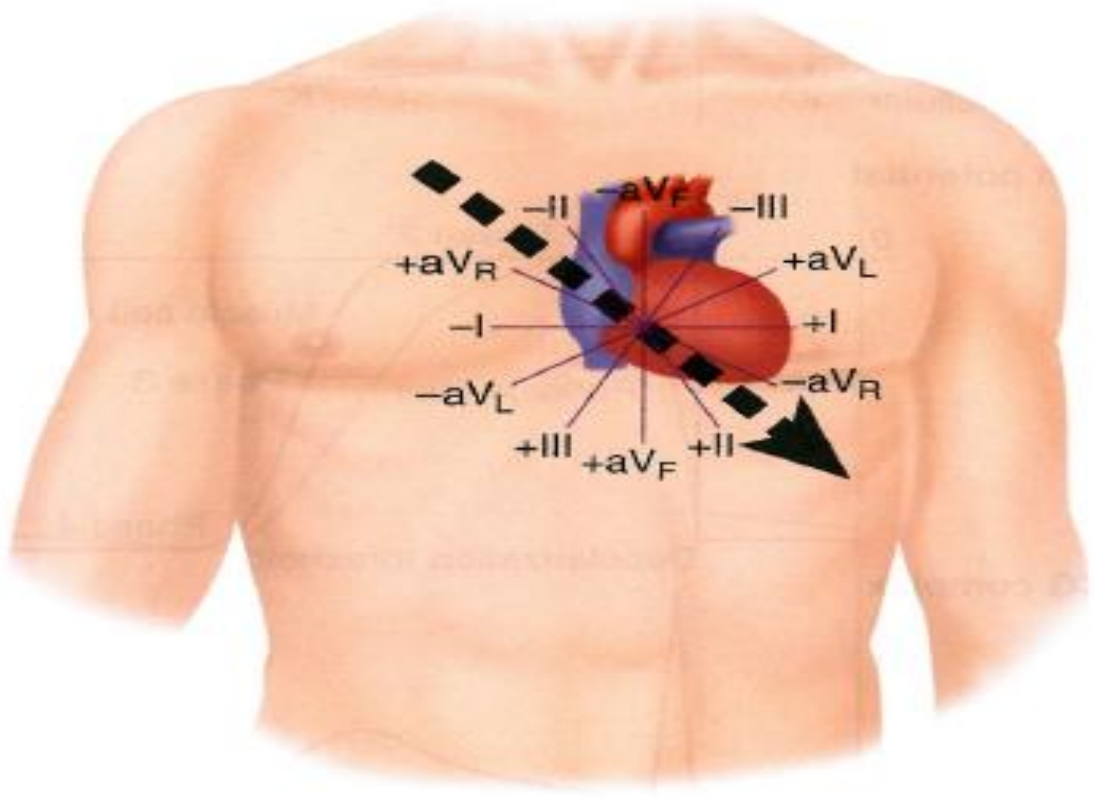
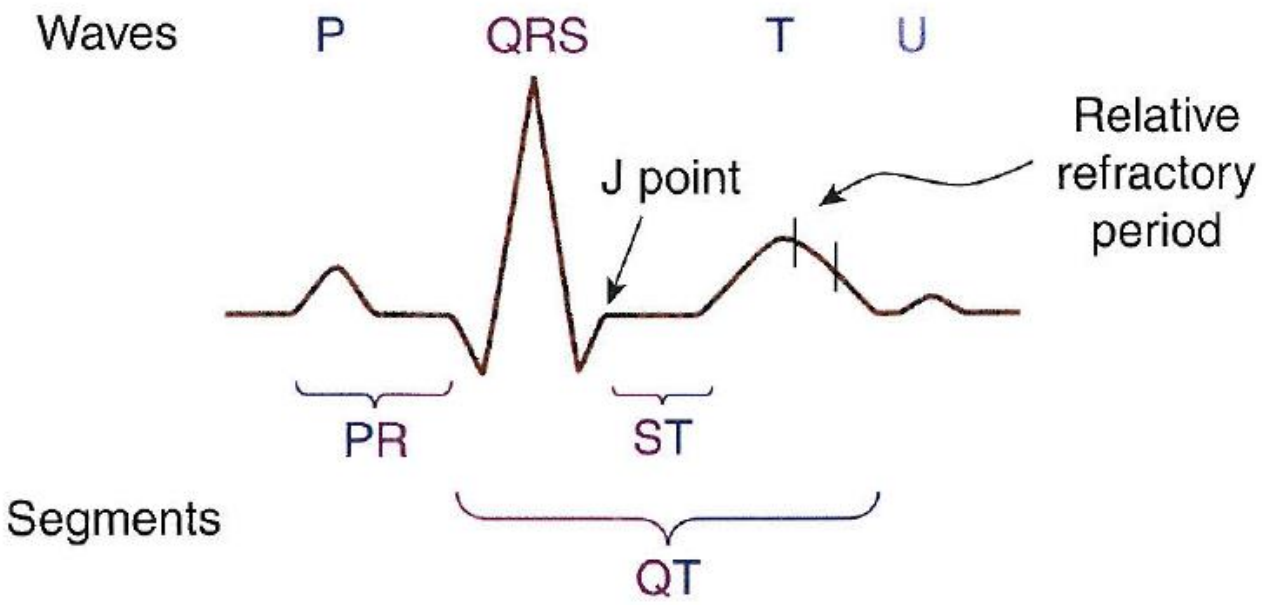
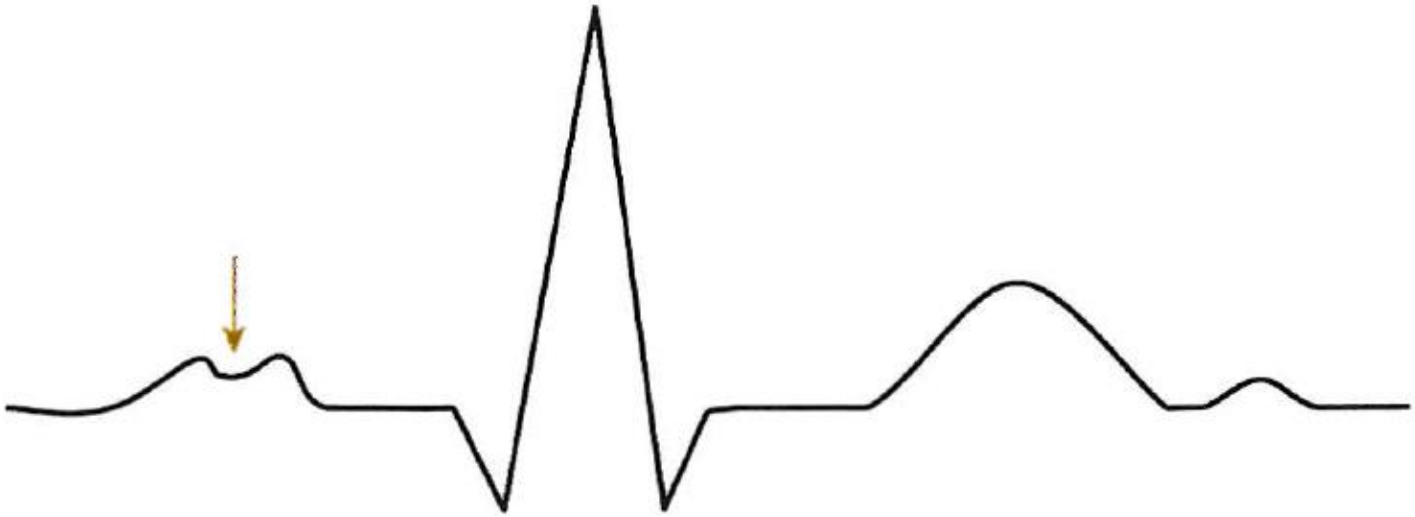


Fig. 1. Cardiac cycle action potential with corresponding electrocardiographic tracing. *Dotted line* indicates the changes associated with Na+ channel blocker toxicity. *Dashed line* indicates the changes associated with K+ efflux blocker toxicity.

The hexaxial reference system derived from the Einthoven equilateral triangle defining the electrical potential vectors of electrocardiography showing the relationship between cardiac anatomy and electrocardiographic leads

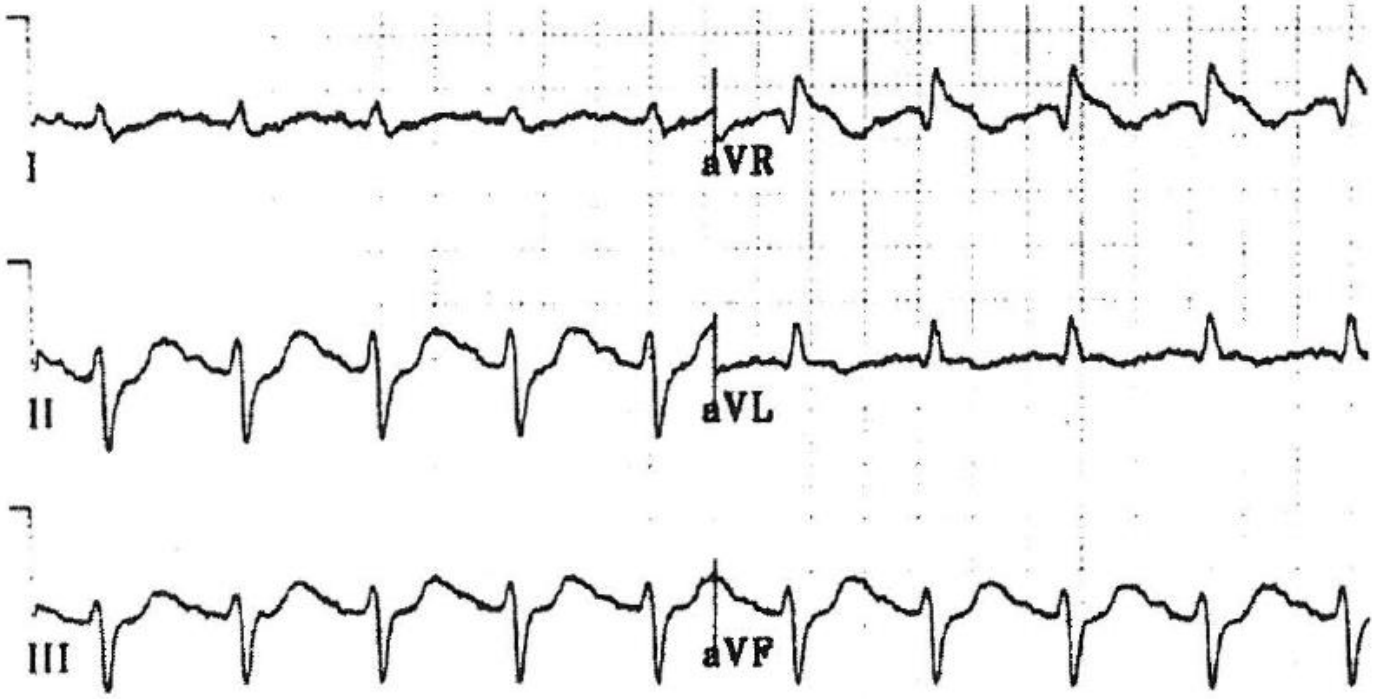



Waves
P
QRS
T
U
J point
Relative
refractory
period
PR
ST
Segments
QT

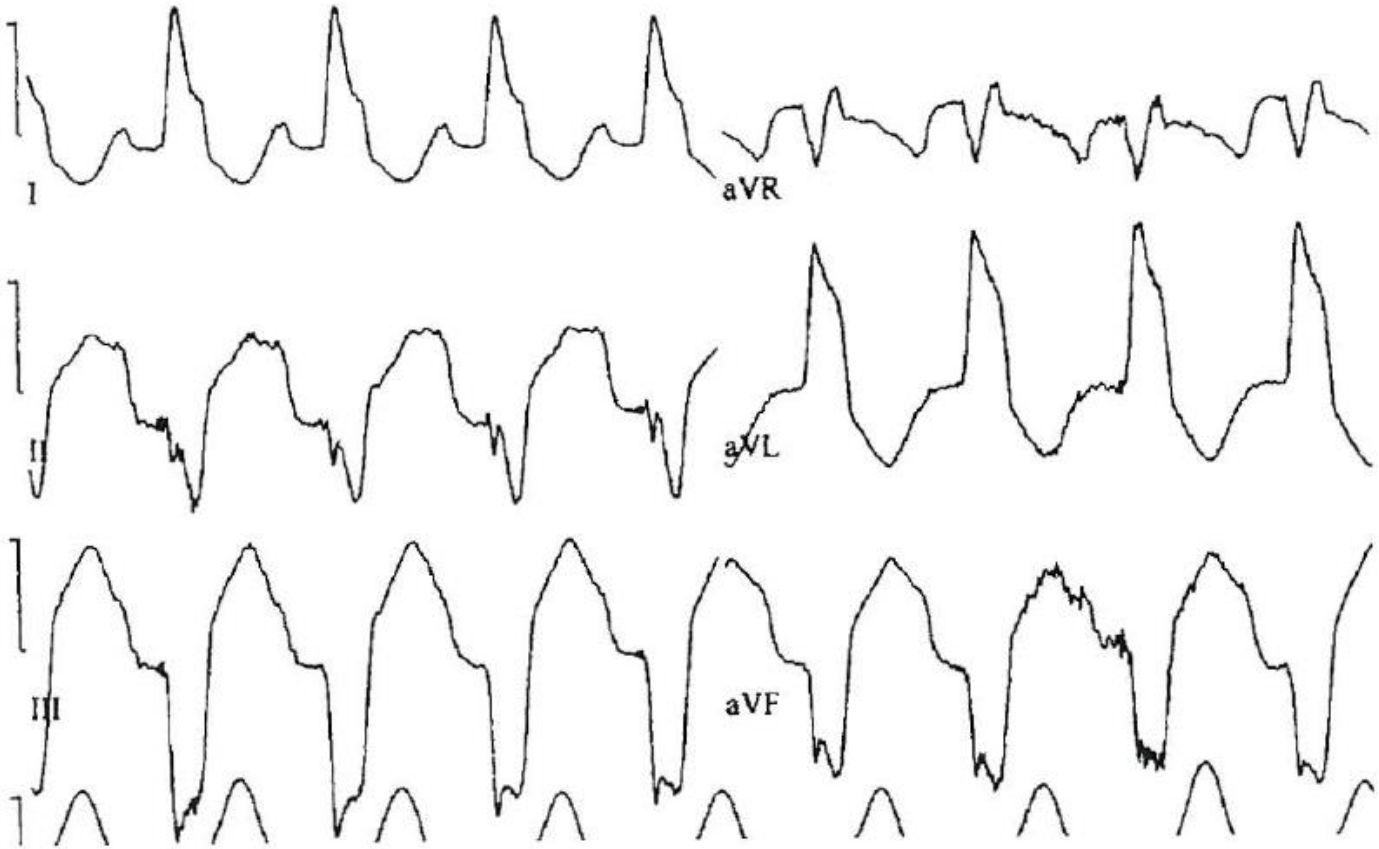


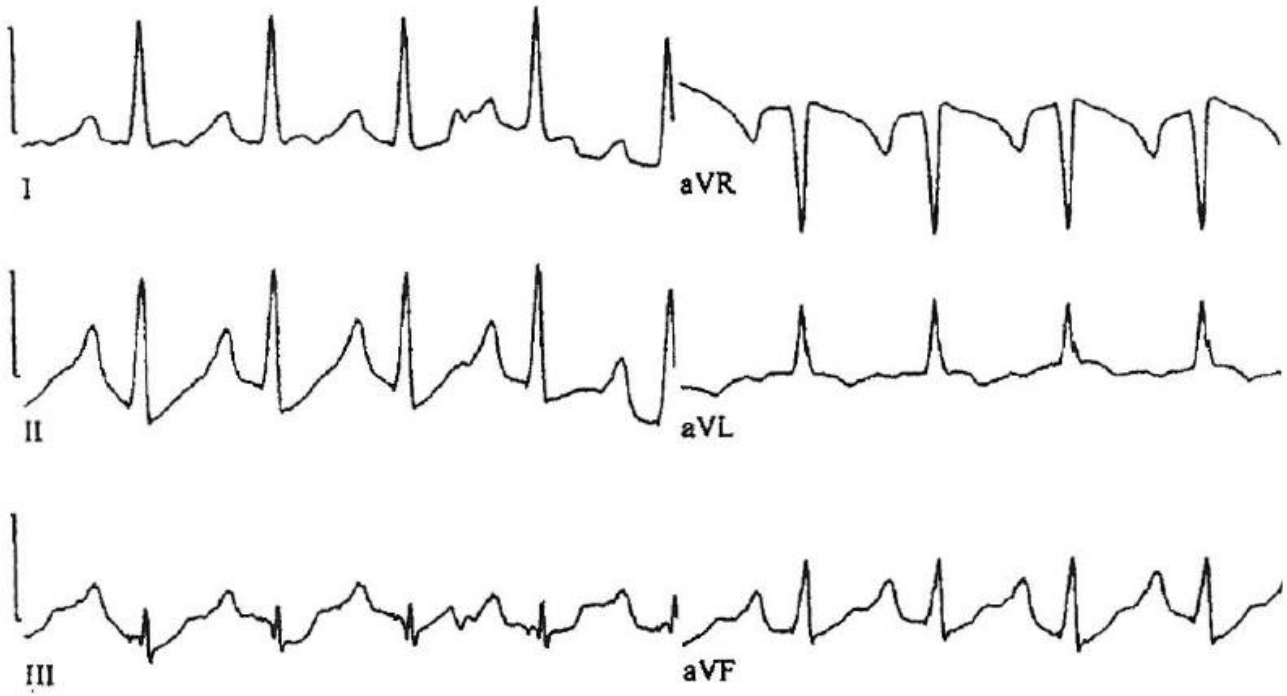
- A notched p wave (arrow) suggests ***delayed conduction across the atrial septum and is characteristic of***

Quinidine poisoning.

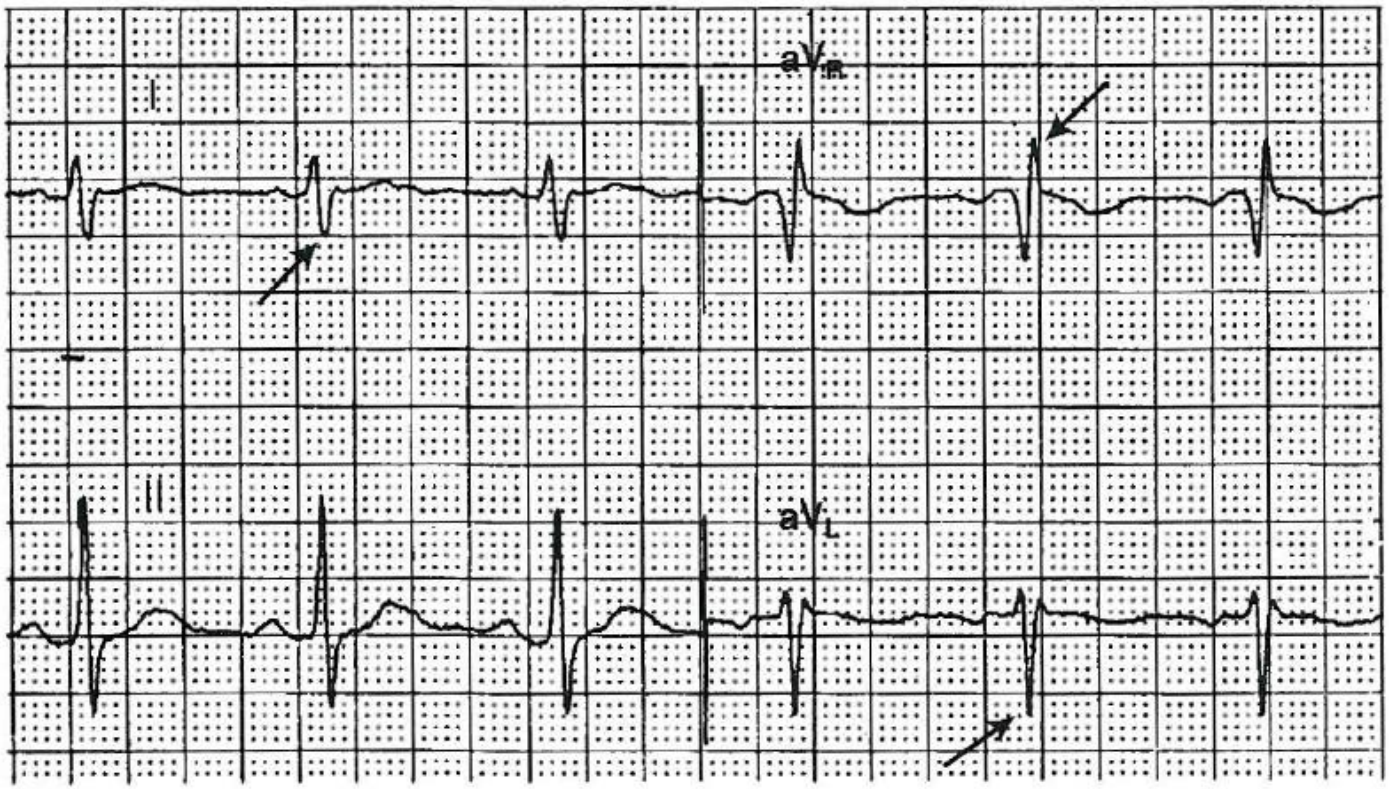

I
aVR
II
aVL
III
aVF

- A 35-year-old woman with Doxepin poisoning.


I
aVR
II
aVL
III
aVF

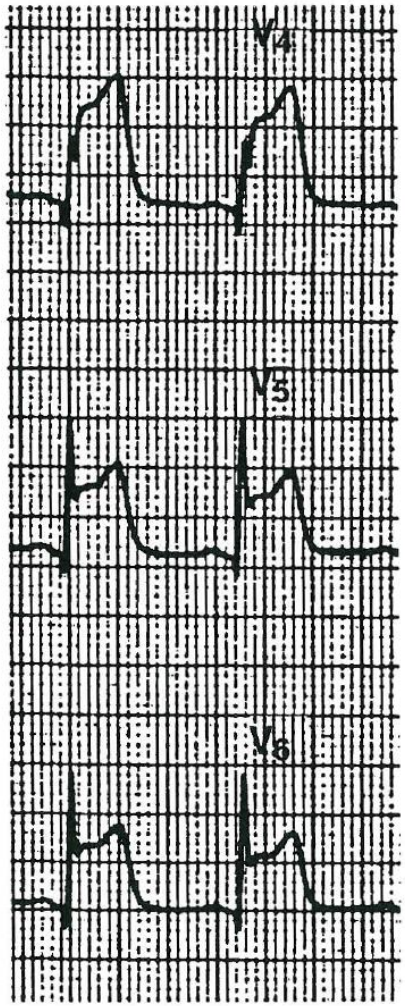

I
aVR
II
aVL
III
aVF

In the next hour: marked improvement after hypertonic sodium bicarbonate therapy.


I
aVR
II
aVL

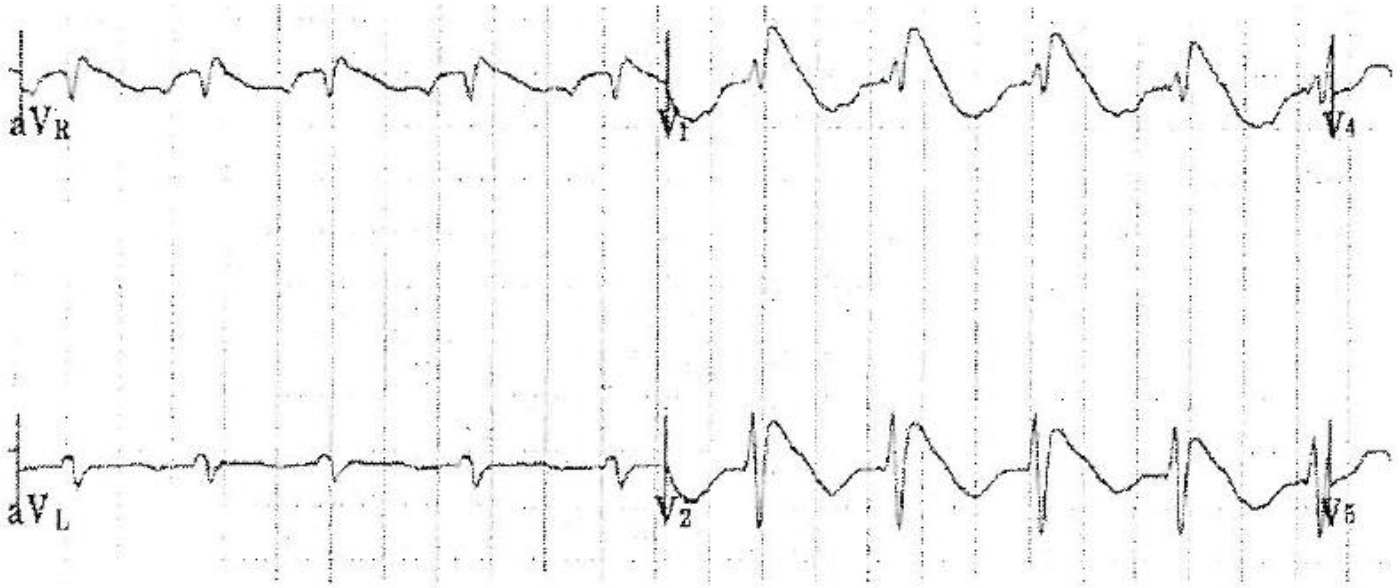
ECG of a patient with a tricyclic antidepressant overdose.

The arrows highlight prominent S wave in leads I and aVL and R wave in aVR.


V4
V5
V6

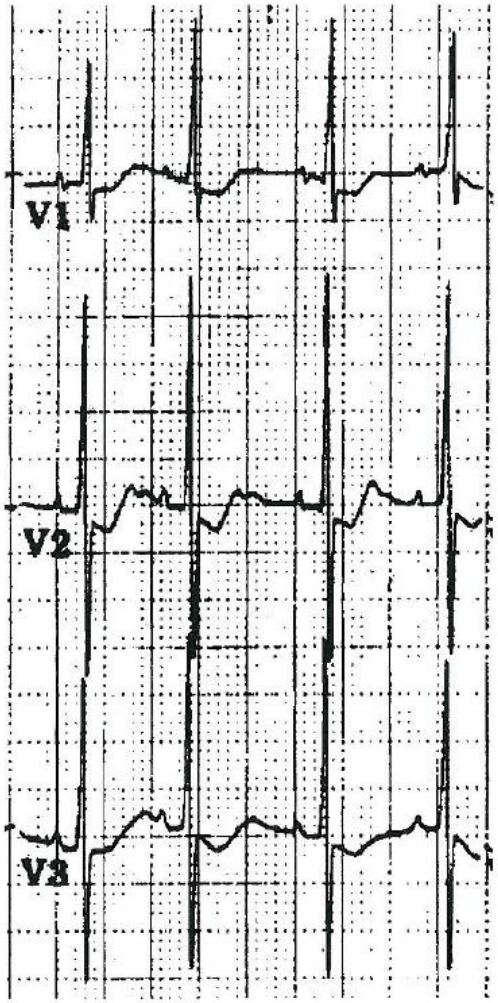
Leads V4-V6 suggestive of a lateral STEMI are shown from the ECGof a 27-year-old man with substernal chest pain after using

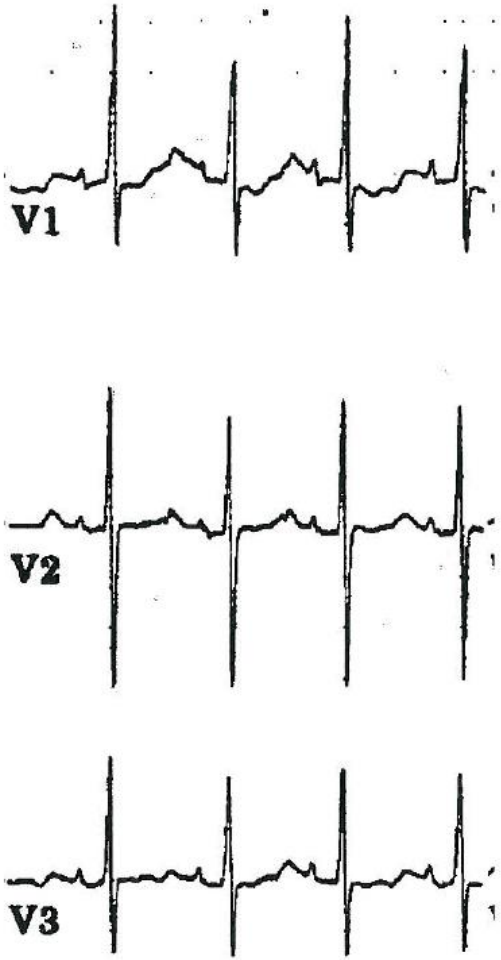
Crack Cocaine.


aVR
V1
V4
aVL
V2
V5

The Brugada pattern is characterized by terminal positivity of the QRS complex and Sf-segment elevation in **the right precordial leads** and is a similar ECG pattern to that noted in patients poisoned by **sodium channel blocking agents such as**

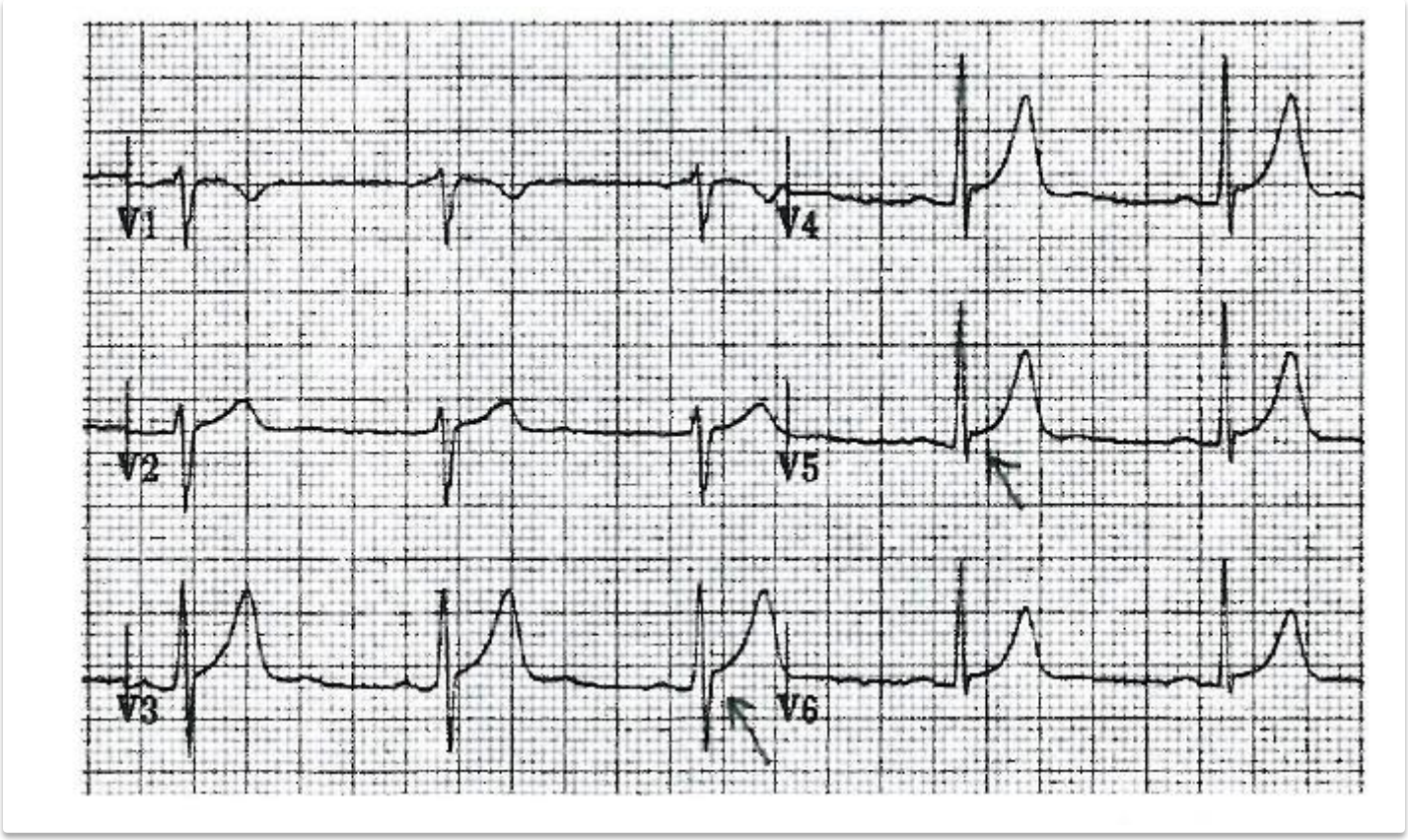
Cyclic Antidepressants


V1
V2
V3

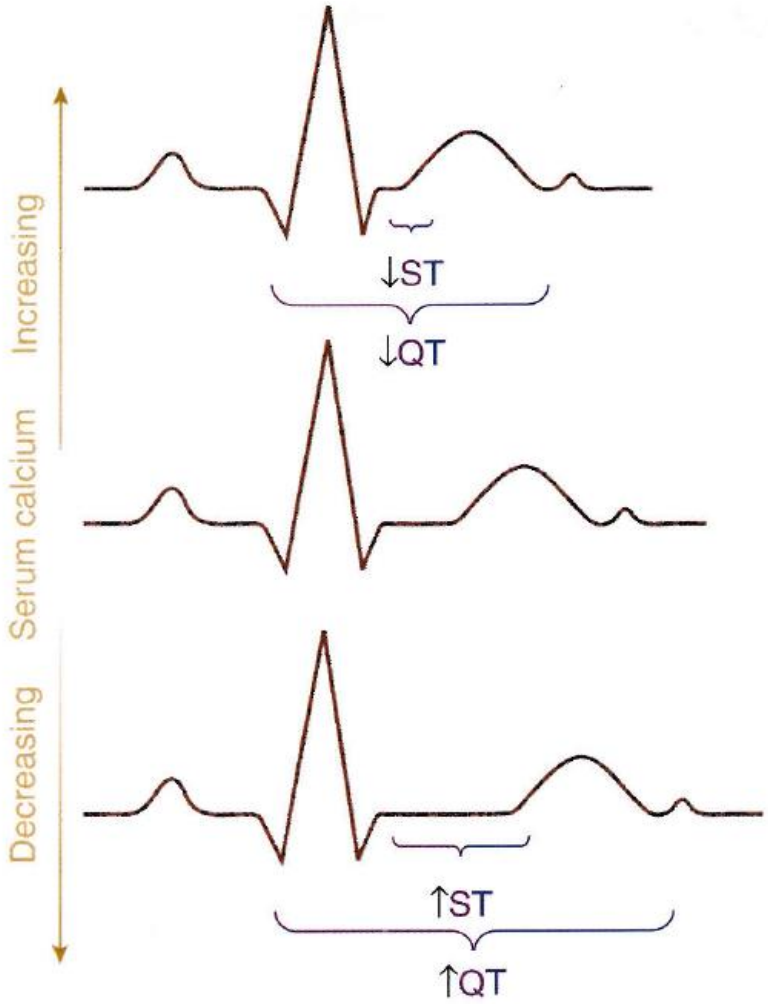

V1
V2
V3

Digitalis effect

Salvador Deli's mustache.

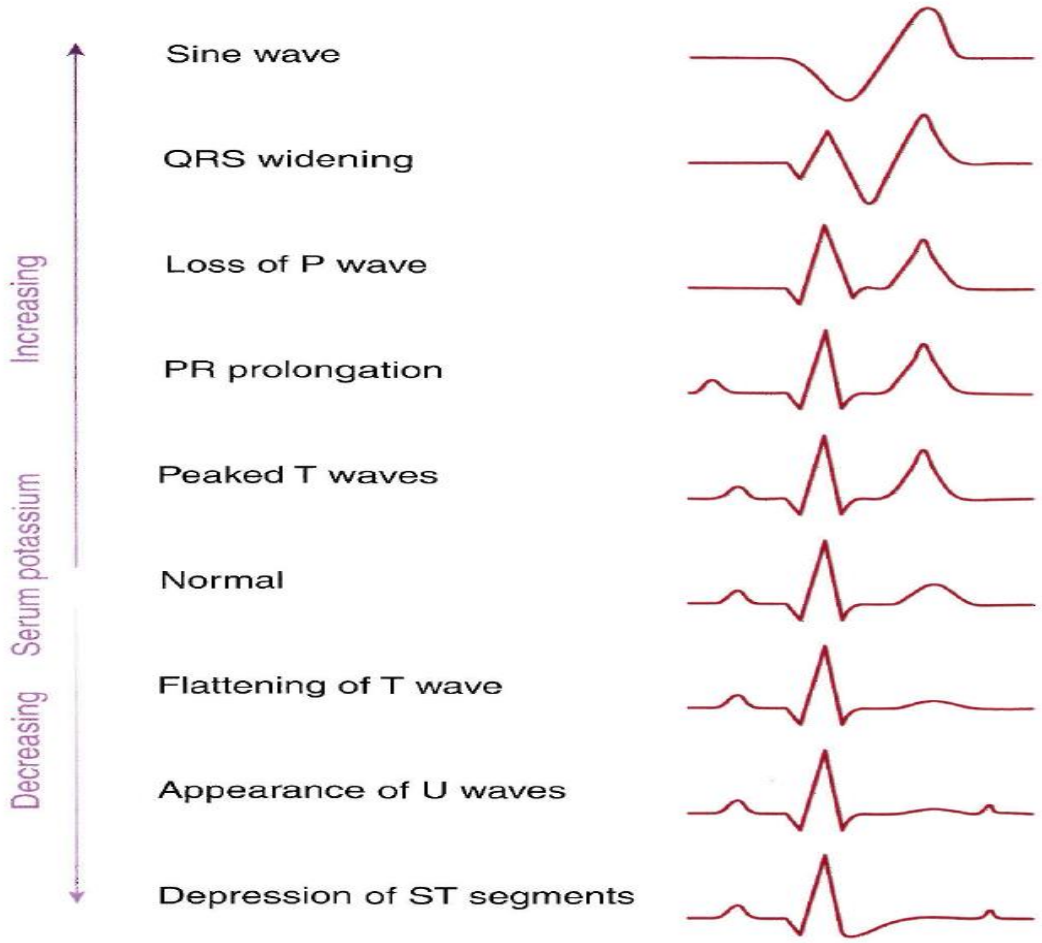

V1
V4
V2
V5
V3
V6

“**Early Repolarization**" abnormality


Increasing
Serum calcium
Decreasing
↓ST
↓QT
↑ST
↑QT

Electrocardiographic findings associated with changes in

Serum Calcium concentration.


Increasing
Serum potassium
Decreasing
Sine wave
QRS widening
Loss of P wave
PR prolongation
Peaked T waves
Normal
Flattening of T wave
Appearance of U waves
Depression of ST segments

Electrocardiographic manifestations associated with changes in

serum

potassium concentration.

TABLE 22–1. Xenobiotic Causes of an Acquired Long QT Syndrome*

Antidysrhythmics

Class IA, IC, and III antidysrhythmics

Antifungals: itraconazole, ketoconazole

Antihistamines: astemizole and terfenadine (no longer available)

Antihypertensives: angiotensin converting enzyme inhibitors

Antimicrobials: amantadine, chloroquine, erythromycin, halofantrin, fluoroquinolones, pentamidine, trimethoprim-sulfamethoxazole,

Electrolyte disturbances

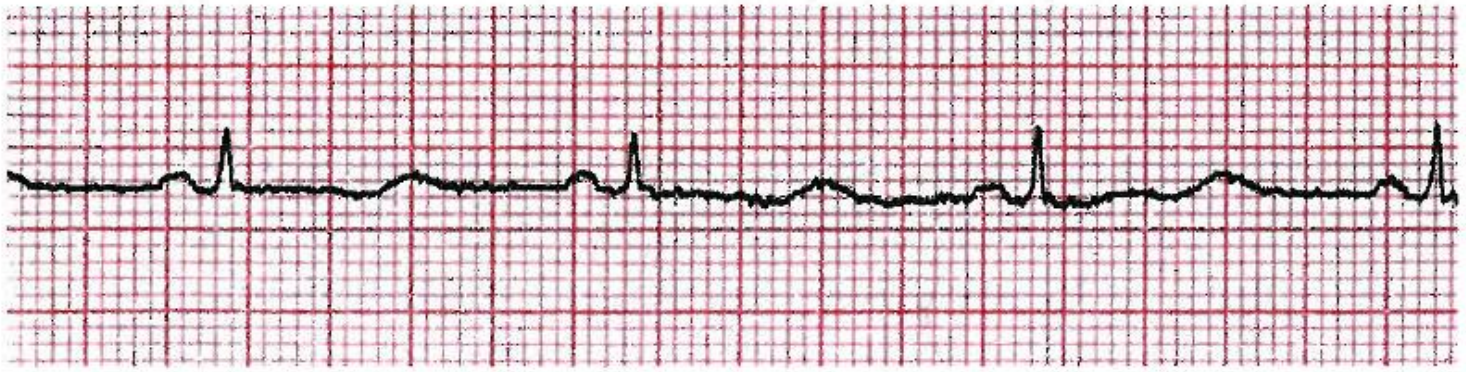
Hypocalcemia: oxalate (eg, ethylene glycol), fluoride

Hypokalemia: barium

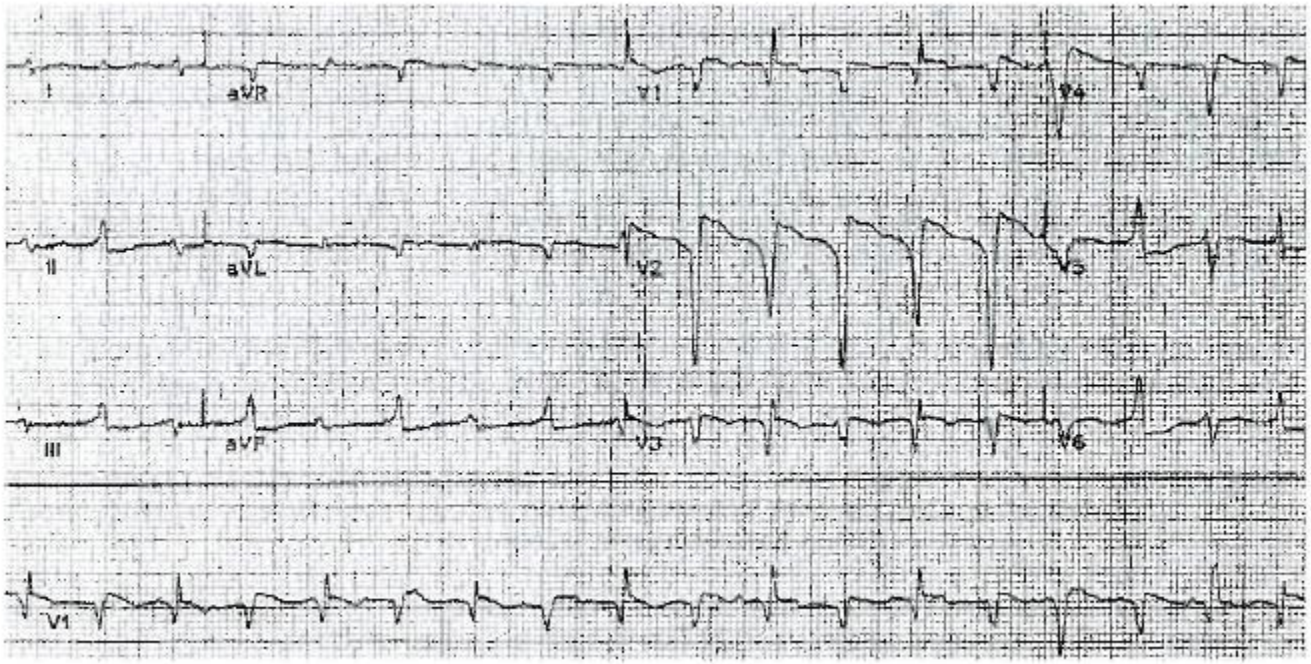
Hypomagnesemia: (eg, diuretics, digoxin, amphotericin, phosphates [IV], ethanol)

Other: arsenic trioxide, cisapride, cocaine, methadone, organic phosphorus insecticides, vasopressin

Psychotropics: atypical antipsychotics, cyclic antidepressants, droperidol, haloperidol, pimozide, phenothiazines, zisprasidone, citalopram



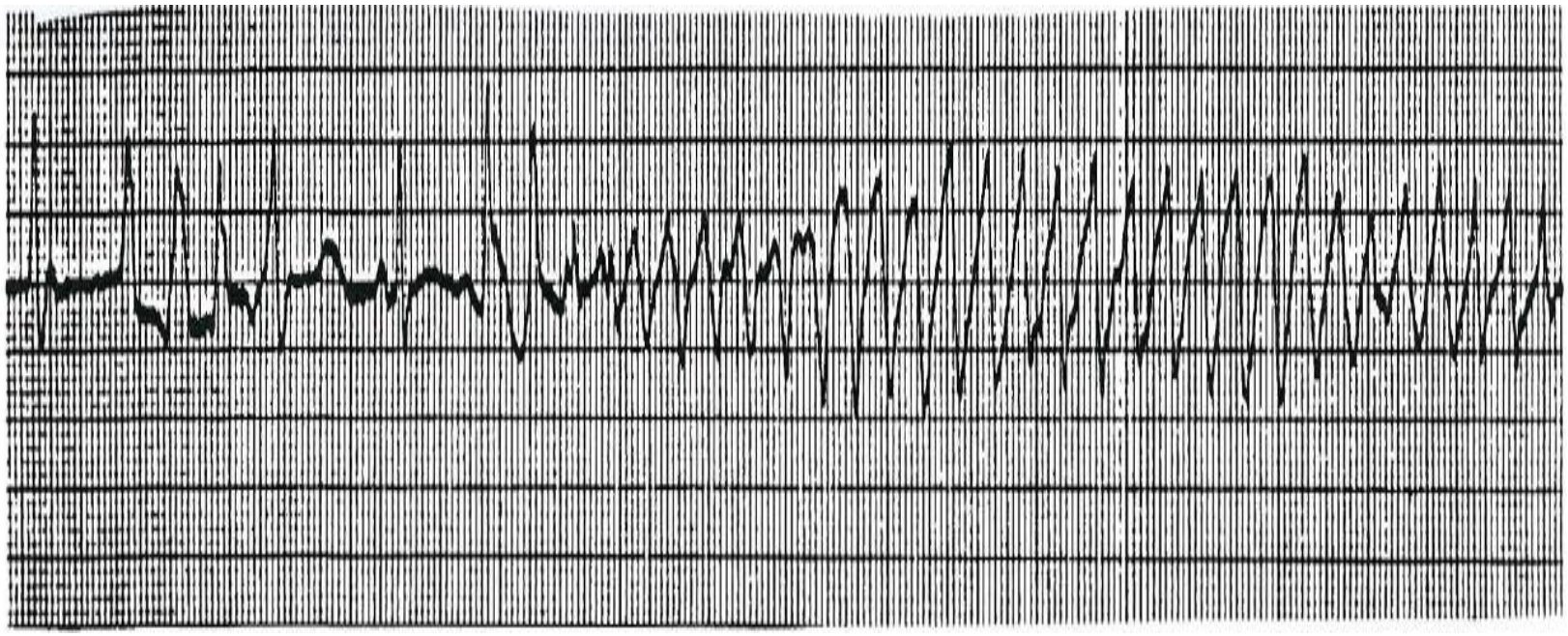
excessive methadone along **with ethanol** 3 hours before admission, sinus bradycardia and QT prolongation.


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1

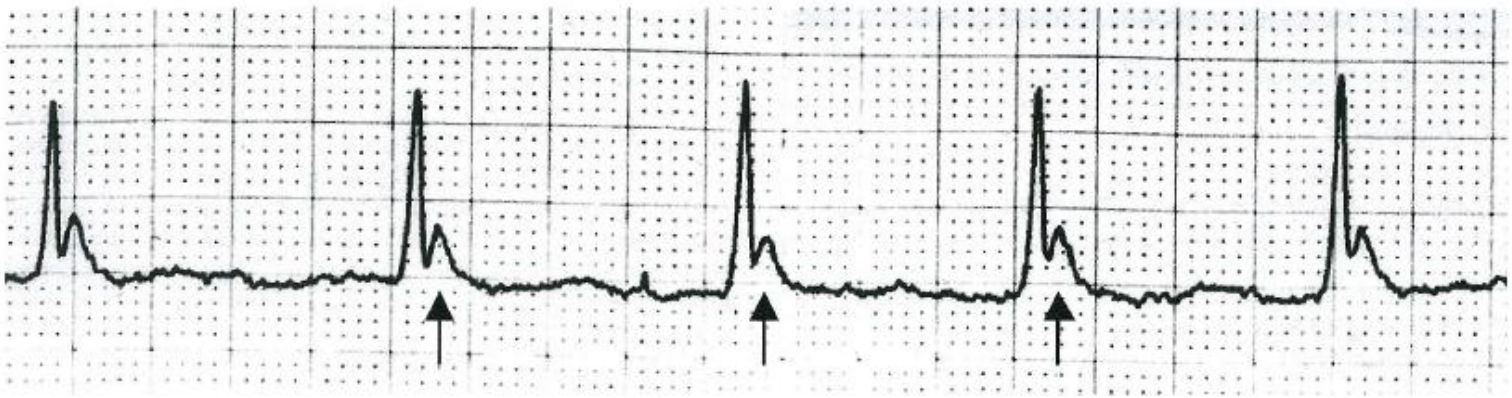
Digoxin-induced

bidirectional ventricular tachycardia.

is nearly pathognomonic for cardioactive steroid poisoning.



Torsades de pointes in a patient who ingested an unknown amount of **thioridazine**



profound hypothermia. The arrows (I) indicate the Osborn wave of the QRS complex.