

Digoxin Toxicity

ANWCCS
Simulation Center

Overview

- » Cardiac glycoside toxicity potentially fatal with mortality ranging from 3-50%
- » Caused by numerous substances usually by digitalis (one form is Digoxin)

Conditions leading to Dig Toxicity

- » Renal insufficiency/ESRD
 - » ESRD prolongs half-life and reduces volume of distribution
- » Advanced age
- » Cardiac diseases
 - » Active ischemia, myocarditis, cardiomyopathy, amyloidosis, cor pulmonale
- » Metabolic factors
 - » Hypokalemia, hypomag, hypoxemia, hypernatremia, hypercalcemia, acid-base

Pharmacology

- » Dig inhibits Na-K-ATPase
 - » Increasing intracellular Na reducing gradient
 - » Na-Ca driving force reduced increasing intracellular Ca--increasing cardiac contractility; positive inotropic effect
- » Digoxin also increases the automaticity of Purkinje fibers but slows conduction through the atrioventricular (AV) node. Cardiac dysrhythmias associated with an increase in automaticity and a decrease in AV node conduction may result.

Kinetics

- » Digoxin bioavailability is 80%
- » Half-life 1.6 days
- » Major storage area in body skeletal muscle
- » Not removed by HD
- » 1/3 body stores/day excreted
 - » 30% Unchanged in urine
 - » 3% as metabolites in stool

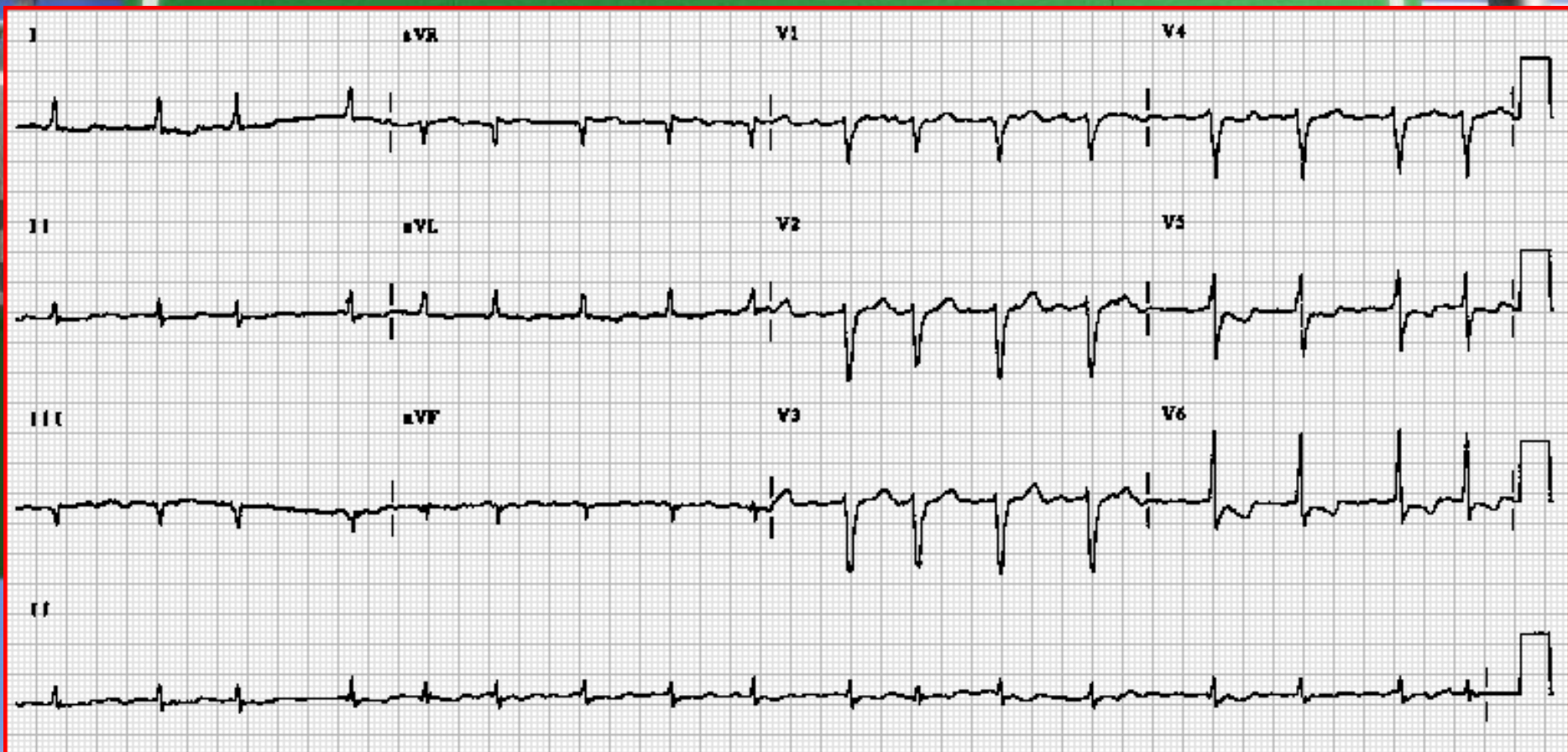
Signs/Syptoms of Dig Overdose

- » History suggesting change in Dig dosage
- » History of any other new drugs
- » Fatigue, blurred vision, disturbed color perception, N/V, anorexia, diarrhea, abdominal pain, HA, dizziness, confusion, delirium, hallucinations
- » Bradycardia
- » Occasional tachycardia
- » Hypotension in severe cases

K

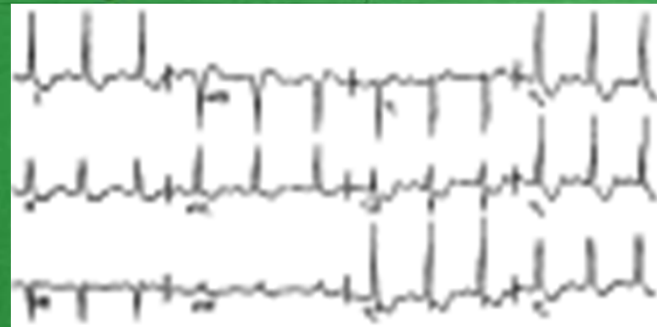
- » Hyperkalemia
 - » Hyperkalemia in acute settings shows degree of Na-K-ATPase poisoning
- » Hypokalemia
 - » Potentiates toxicity--correct immediately

Normal Dig ECG



ECG-normal

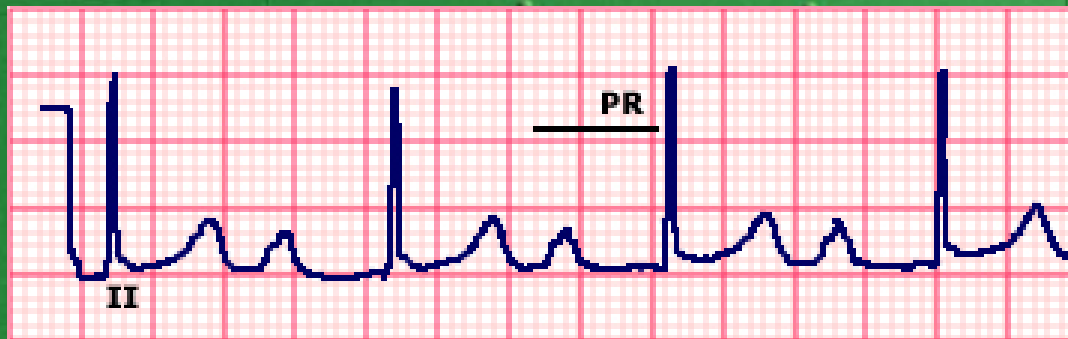
- » T wave changes
- » QT interval shortening
- » "Scooped" appearance of ST segment
- » Increase U wave amplitude



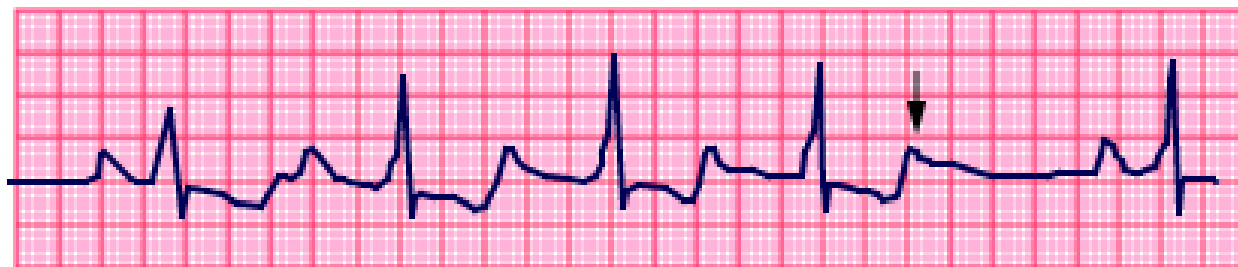
Atrial Tachycardia with AV block



First Degree AV block

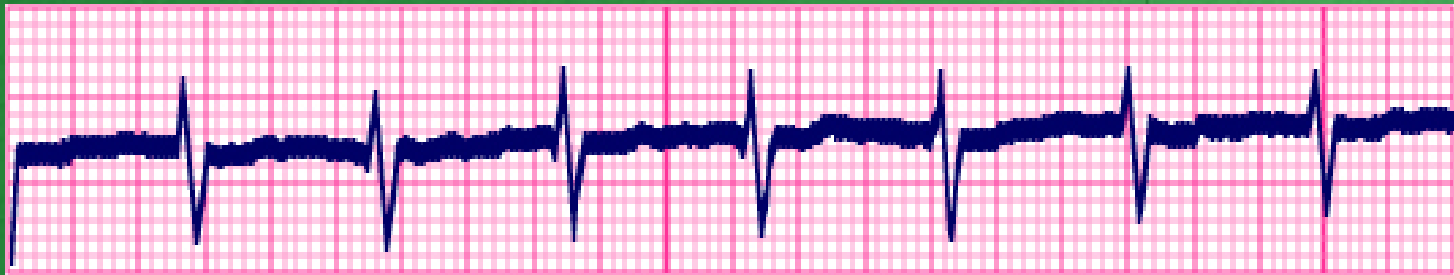


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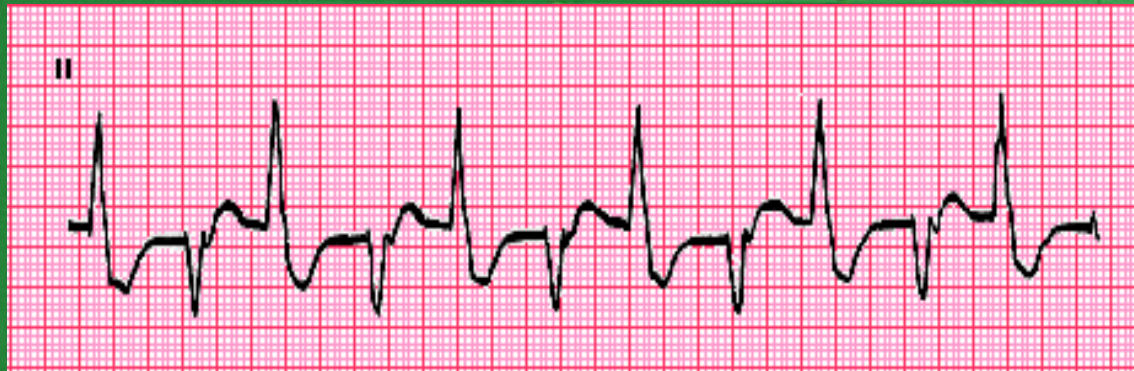


PR	.18	.23	.26	.27	.18
Increment		.05	.03	.01	
RR	.57	.54	.53	.96	

Afib with accelerated Junctional Rhythms



Bidirectional Ventricular Tachycardia



Ventricular Bigeminy



Treatment

- » Support treatment if needed-intubation, etc
- » Symptomatic bradycardia-atropine
- » Do not use transvenous pacing as first line -can lead to arrhythmias
- » Avoid Beta agonists (isoproterenol)
- » Gut decontamination with activated charcoal (w/in 6-8 hours of acute ingestion)
- » Manage K as usual except **do not use calcium salts**
- » Replace Mg

Treatment

Digibind

- » Digoxin-specific Fab fragments
- » Made in sheep
- » Bind rapidly to intravascular dig
- » Dig stored in other tissues then goes into intravascular space and digibind binds that also
- » Digibind/digoxin complex small and is rapidly removed by normal kidneys
- » ESRD on HD responds clinically the same to digibind except elimination of complex slow
 - » Theoretically can get rebound dig toxicity

When to Use Digibind

- » Hemodynamic instability
- » Life-threatening arrhythmias
- » Severe Bradycardia-even if atropine works
- » Plasma K above 5
- » Plasma Dig above 10
- » Presence of dig toxicity rhythm combined with dig toxic level

References

- » http://www.sciencedirect.com.libproxy.lib.unc.edu/science?_ob=ArticleURL&_udi=B6T8B-42C07N1-8&_user=130907&_coverDate=02%2F28%2F2001&_rdoc=1&_fmt=&_orig=search&_sort=d&view=c&_acct=C000004198&_version=1&_urlVersion=0&_userid=130907&md5=461c196c5db7aa1916fb4a1dec1035dd
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