# Acute Digoxin Toxicity

Digoxin's mechanism of action is the inhibition of the sodium/potassium ATPase pump. In the heart, this results in increased calcium concentrations in myocytes, as well as increased vagal tone. Adverse drug reactions to digoxin are relatively common due to a narrow therapeutic index. Hyperkalemia is seen in acute toxicity. Patients with pre-existing hypokalemia, however, are particularly vulnerable to side effects since digoxin normally competes with K+ ions for the same binding site on the Na/K ATPase pump. The increased vagal tone causes cholinergic symptoms like nausea, vomiting, and diarrhea due to increased gastrointestinal motility. Eye involvement can include blurry yellow and green vision, with halos around each point of light. Cardiac effects can involve a range of arrhythmias secondary to increased intracellular calcium, such as bradycardia, bigeminy and ventricular tachycardia or fibrillation. The combination of increased atrial arrhythmias with inhibited AV conduction (like paroxysmal atrial tachycardia with AV block) is highly suggestive of acute digoxin toxicity. Electrocardiogram changes include a prolonged PR interval, QT interval shortening, ST segment scooping and T wave inversion.




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## Clinical Symptoms

**Hyperkalemia**
Hiker-banana
In digoxin toxicity, hyperkalemia is common due to paralysis of the Na/K ATPase pump. This can be a life threatening issue that must be quickly addressed when managing digoxin toxicity.

**Cholinergic (Nausea, Vomiting, Diarrhea)**
Cola-can Vomiting on Toilet
Common adverse effects include cholinergic symptoms like nausea, vomiting, and diarrhea due to increased gastrointestinal motility.

**Blurry Yellow Green Vision with Halo of Light**
Yellow and Green Halo
Common adverse effects include disturbance of color vision involving mostly yellow and green. Additionally, digoxin can cause generalized blurry vision as well as seeing a halo around each point of light.

**Arrhythmia**
Broken-arrhythmia-drum
Increased intracellular calcium can cause arrhythmias including bigeminy and ventricular tachycardia or fibrillation. The combination of increased atrial arrhythmias and inhibited AV conduction like paroxysmal atrial tachycardia with AV block is commonly said to be pathognomonic of digoxin toxicity.

**Bradycardia**
Snail-heart
Digoxin use can lead to bradycardia, which is a condition of heart rate characterized by an abnormally slow rate.

## EKG Changes

**Prolonged PR interval**
Elongated PRada Purse
While normal digoxin use can cause certain EKG changes, an elongated PR interval is specifically associated with digoxin toxicity.

**Decreased QT**
Down-arrow QT-heart
Digoxin use can cause shortening of the QT interval on EKG, which is thought to be related to increased calcium and increased ionotropy of the heart.

**Scooping on EKG**
Ice Cream Scooper
Digoxin can cause a characteristic scooping of the ST-T complex in which the ST segments and T waves are fused together making it impossible to tell where one ends and the other begins. Scooping can occur even when digoxin is in the therapeutic range.

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**T Wave Inversion**

Upside Down Mr. T

T wave inversion is a common EKG finding in digoxin use, even when the levels are within a therapeutic range.

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