

## Digoxin Mechanism and Indication

Digoxin is a cardiac glycoside extracted from the plant *Digitalis*. The mechanism of action is thought to be related to direct inhibition of the Na/K ATPase pump in the membranes of myocytes. Direct inhibition via binding to a site on the extracellular aspect leads to an increase in the level of sodium ions in the myocytes which decreases the sodium concentration gradient. A decrease of this gradient leads to indirect inhibition of the Na/Ca exchanger, which depends on a constant inward sodium gradient to pump calcium out of myocytes. Therefore, digoxin decreases the sodium concentration gradient and indirectly decreases subsequent calcium outflow. This results in increased calcium concentration in the heart cells which leads to increased contractility of the heart also referred to as increased positive inotropy. Because it can increase the contractility of the heart, it is sometimes used in patients who have congestive heart failure who remain symptomatic despite adequate diuretic and ACE inhibitor treatment. There is also evidence that digoxin increases vagal activity, thereby decreasing heart rate by slowing depolarization of pacemaker cells in the AV node. Slowed conduction through the AV node makes this drug commonly used in the treatment of atrial fibrillation with rapid ventricular response. In atrial fibrillation, rapid ventricular rate leads to insufficient diastolic filling time. By slowing down the conduction in the AV node, digoxin can reduce ventricular rate therefore improving ventricular filling and the pumping function of the heart.



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### Direct inhibition of Na K ATPase

#### [Direct-route Inhibiting-chains on Salt-shaker with Bananas](#)

The mechanism of action is thought to be related to direct inhibition of the Na/K ATPase pump in the membranes of myocytes. Direct inhibition via binding to a site on the extracellular aspect leads to an increase in the level of sodium ions in the myocytes which decreases the sodium concentration gradient.

### Indirect inhibition of Na Ca exchanger

#### [Indirect-route Inhibiting-chains on Salt-shaker and Calcified-cows](#)

A decrease of this gradient leads to indirect inhibition of the Na/Ca exchanger, which depends on a constant inward sodium gradient to pump calcium out of myocytes. Therefore, digoxin decreases the sodium concentration gradient and indirectly decreases subsequent calcium outflow.

### Increase Ca in cell

#### [Build up of Calcified-cows](#)

Digoxin decreases the sodium concentration gradient and indirectly decreases subsequent calcium outflow. This results in increased calcium concentration in the heart cells, which leads to increased contractility of the heart. This is also referred to as increased positive inotropy.

## Positive Inotropy

[Cows wearing I heart shirts and flexing and heart and muscles are getting progressively larger](#)

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## CHF

[CHF Heart-balloon](#)

Because digoxin can increase the inotropy/contractility of the heart, it is sometimes used in patients who have congestive heart failure who remain symptomatic despite adequate diuretic and ACE inhibitor treatment. It is important to note it is not a first line treatment of CHF and has not shown to improve mortality in patients with CHF.

## Stimulates Vagus Nerve

[Vegas-sign Nerve](#)

There is also evidence that digoxin increases vagal activity, thereby decreasing heart rate by slowing depolarization of pacemaker cells in the AV node.

## Decreased conduction at AV node

[Down-arrow Snail Conductor with AViator-Nose](#)

There is also evidence that digoxin increases vagal activity, thereby decreasing heart rate by slowing depolarization of pacemaker cells in the AV node.

## Atrial Fibrillation

[Atria-heart Alarm-clock](#)

Slowed conduction through the AV node makes this drug commonly used in the treatment of atrial fibrillation with rapid ventricular response. In atrial fibrillation, rapid ventricular rate leads to insufficient diastolic filling time. By slowing down the conduction in the AV node, digoxin can reduce ventricular rate therefore improving ventricular filling and the pumping function of the heart.