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Digoxin Medical Uses and Its Side Effects

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INTRODUCTION

Digoxin (better known as Digitalis), also known by the brand name Lanoxin, is a medication used to treat various heart conditions. It is most commonly used for atrial fibrillation, atrial flutter, and heart failure. Digoxin is one of the oldest medications used in cardiology. It works by increasing myocardial contractility, increasing stroke volume and blood pressure, decreasing heart rate, and slightly extending the time frame of the contraction. Digoxin can be taken orally or by injection into a vein. Digoxin has a half life of approximately 36 hours when administered at average doses to patients with normal renal function. It is mostly excreted in the urine undamaged. Breast enlargement is a common side effect, with other side effects generally caused by an excessive dose. these side effects may include nausea, vomiting, difficulty seeing, confusion, and an irregular heartbeat. Elderly people and those with impaired kidney function require extra attention. It's unclear whether using it while pregnant is safe.

The primary mechanism of action of digoxin is the inhibition of sodium potassium adenosine triphosphatase (Na^+/K^+ ATPase), primarily in the myocardium. This inhibition raises intracellular sodium levels, which reduces the activity of the sodium-calcium exchanger, which normally imports three extracellular sodium ions and transports one intracellular calcium ion out of the cell. The reversal of this exchanger caused by an increase in intracellular sodium, results in an increase in intracellular calcium concentration available to contractile proteins. Increased calcium concentrations cause calcium to bind to troponin, resulting in the formation of the Ca^{+2} troponin complex and increased inotrophy. Increased intracellular calcium lengthens phases 4 and 0 of the cardiac action potential, resulting in a slower heart rate. Increased Ca^{+2} levels because increased calcium storage in the sarcoplasmic reticulum, resulting in a corresponding increase in calcium release during each action potential. This causes the heart's contractility (the force with which it contracts) to increase without increasing the heart's energy expenditure.

DESCRIPTION

Inhibiting the sodium pump may improve baroreceptor sensitivity in heart failure and may explain some of digoxin's neurohormonal effects. Digoxin has significant parasympathetic effects, especially on the atrioventricular node. While it increases the magnitude of myocardial contractility, it only slightly increases the duration of the contraction. Its use as an antiarrhythmic drug stems from its parasympathetic stimulating properties, both direct and indirect. By increasing the refractory period of cardiac myocytes, vagus nerve stimulation slows conduction at the AV node. Because the AV node

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is slower, the ventricles have more time to fill before contracting. This anti-chronotropic effect works in tandem with the direct effect on cardiac pacemaker cells. The arrhythmia is not affected, but the heart's pumping function improves as a result of improved filling.

Other electrical effects include a brief increase in action potential, followed by a decrease as K^+ conductance increases due to increased intracellular Ca^{2+} ion concentrations. The refractory period of the atria and ventricles is reduced, whereas it increases in the sinoatrial and AV nodes. The resting membrane potential becomes less negative, resulting in increased irritability.

Conduction velocity rises in the atria but falls in the AV node. The impact on Purkinje fibres and ventricles is insignificant. The atria, AV node, Purkinje fibres, and ventricles all have increased automaticity. Digoxin is typically administered orally, but it can also be administered intravenously in emergency situations (the IV injection should be slow, and heart rhythm should be monitored). While IV therapy may be more tolerable (less nausea), digoxin has a very long distribution half-life into cardiac tissue, delaying the onset of action by several hours. Digoxin is given once daily, usually in 125 g or 250 g doses, and has a half-life of about 36 hours in patients with normal renal function.

Digoxin is primarily eliminated through renal excretion and involves P-glycoprotein, resulting in significant clinical interactions with P-glycoprotein inhibitor drugs. Examples include spironolactone, verapamil, and amiodarone, which are commonly used in pa-tients with heart problems. Depending on the medical indication, effective plasma levels differ. Levels between 0.5 and 1.0 ng/ml are recommended for congestive heart failure. This recommendation is based on a post-hoc analysis of prospective trials, which indicates that higher levels may be associated with higher mortality rates. Plasma levels are less defined for heart rate control (atrial fibrilla-tion) and are generally titrated to a target heart rate. Digoxin levels between 0.5 and 2.0 ng/ml (or 0.6 and 2.6 nmol/l) are typically considered therapeutic for heart rate control. Digoxin levels should be monitored if toxicity or ineffectiveness is suspected. Plasma potassium levels must also be closely monitored (see side effects, below). Quinidine, verapamil, and amiodarone raise plasma levels of digoxin (by displacing tissue binding sites and decreasing renal digoxin clearance), so digoxin levels must be carefully monitored when Coad ministered.

CONCLUSION

A study that looked to see if digoxin affected men and women differently discovered that digoxin did not reduce overall deaths, but it did result in less hospitalization. Women taking digoxin died "more frequently" (33%) than women taking a placebo (29%). Digoxin raised the risk of death in women by 23%. In the study, there was no difference in the death rate between men and women.