

Pharmacology

Pharmacy Department

4th Grade

Antiarrhythmics Drugs

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## Overview:

- In contrast to skeletal muscle, which contracts only when it receives a stimulus, the heart contains specialized cells that exhibit **automaticity**. That is, they intrinsically generate rhythmic action potentials in the absence of external stimuli.
- These “**pacemaker**” cells differ from other myocardial cells in showing a slow, **spontaneous depolarization during diastole (phase 4)**, caused by an **inward positive current carried by sodium and calcium ions**. This depolarization is fastest in the sinoatrial **(SA) node** (the normal initiation site of the action potential), and it decreases throughout the normal conduction pathway through the atrioventricular **(AV) node to the bundle of His and the Purkinje system**.
- **Dysfunction of impulse generation or conduction** at any of a number of sites in the heart can cause an **abnormality in cardiac rhythm**.

## B. Adenosine

- *Adenosine* is a naturally occurring nucleoside, but **at high doses, the drug decreases conduction velocity, prolongs the refractory period, and decreases automaticity in the AV node.**
- Intravenous *adenosine* is the drug of choice for abolishing **acute supraventricular tachycardia.**
- It has low toxicity but causes **flushing, chest pain, and hypotension.**
- ***Adenosine* has an extremely short duration of action (approximately 10 to 15 seconds) due to rapid uptake by erythrocytes and endothelial cells.**

## C. Magnesium sulfate

- *Magnesium* is necessary for the transport of sodium, calcium, and potassium across cell membranes.
- It slows the rate of SA node impulse formation and prolongs conduction time along the myocardial tissue.
- Intravenous *magnesium sulfate* is the salt used to treat arrhythmias, as oral *magnesium* is not effective in the setting of arrhythmia.
- Most notably, *magnesium* is the drug of choice for treating the potentially fatal arrhythmia **torsades de pointes** and **digoxin-induced arrhythmias**.

*Thank  
you!*