Mechanisms of Antiarrhythmic Drugs

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The major mechanism of antiarrhythmic drugs is blocking of ion channels. These drugs bind to their receptor sites of ion channels. The affinity of a drug depends on the state of an ion channel: resting, open, and inactivated states. The kinetics of binding and recovery are different from each other, and these characteristics are associated with use dependence of drugs.

Selection of antiarrhythmic drugs for arrhythmia control in individual patients requires understanding of the mechanism of the arrhythmia that we are about to manage: abnormal automaticity, triggered activity, and reentry. Targets of arrhythmia related to abnormal automaticity are diastolic depolarization, threshold potential, and maximum diastolic potential. Those for triggered activity are action potential duration (early afterdepolarization) and intracellular Ca^{2+} overload (delayed afterdepolarization). Those for reentry mechanism are wavelength of the reentry, which is defined by the product of refractoriness and conduction velocity.

One of the major limitations of these drugs is proarrhythmic effect. Therefore safe medication and monitoring is as important as drug selection. Useful tools for toxicity

monitoring are treadmill test for drugs with use dependence, esp. class IC drugs, and QT interval for class Ⅲ drugs.