pharmacodynamics and pharmacokinetics, in particular significant cross-reactivity of certain drugs across different targets as well as variable effects on drug targets across drugs within the same category. There are regional differences in drug effect within the myocardium, and interpatient variations in drug metabolism play important roles. This has in part led to many instances of harm that have come from the adverse effects of many agents used over the years. In fact, many antiarrhythmic agents currently in use carry significant risks of side effects, some of which may be significant and even lethal. Therefore, judicious use of antiarrhythmic medications by those with appropriate knowledge base and experience is warranted. The practical result of the narrow therapeutic index of this class of medications has rendered their use more and more as ancillary options **(Table 243-2)**.

TABLE 243-2 Antiarrhythmic Drug Actions

DRUG	CLASS ACTIONS				OTHER ACTIONS/COMMON SIDE EFFECTS
	I	II	III	IV	
Quinidine	++		++		Anticholinergic
Flecainide	+++		+		Can promote reentrant arrhythmias (AFL, VT)
Propafenone	++	+			Mild beta-blocker effect
Amiodarone	++	++	+++	+	Multiorgan toxicity with long-term use
Sotalol		++	+++		Prominent beta-blocker effect
Dofetilide			+++		Prolongation of QT at slower heart rates
Dronedarone	+	+	+	+	Mild effect
Ibutilide			+++		Used only for acute cardioversion
Ranolazine	++		++		Late sodium channel blockade
Lidocaine	++				Used for reperfusion arrhythmias

The traditional nomenclature of antiarrhythmic drugs (AADs) is known as the Vaughan Williams classification schema. In this schema, there are four classes (I–IV; Table 243-2). Class I AADs primarily target the Na channel, Class II agents target the beta-adrenergic receptor, Class III agents target potassium channels,