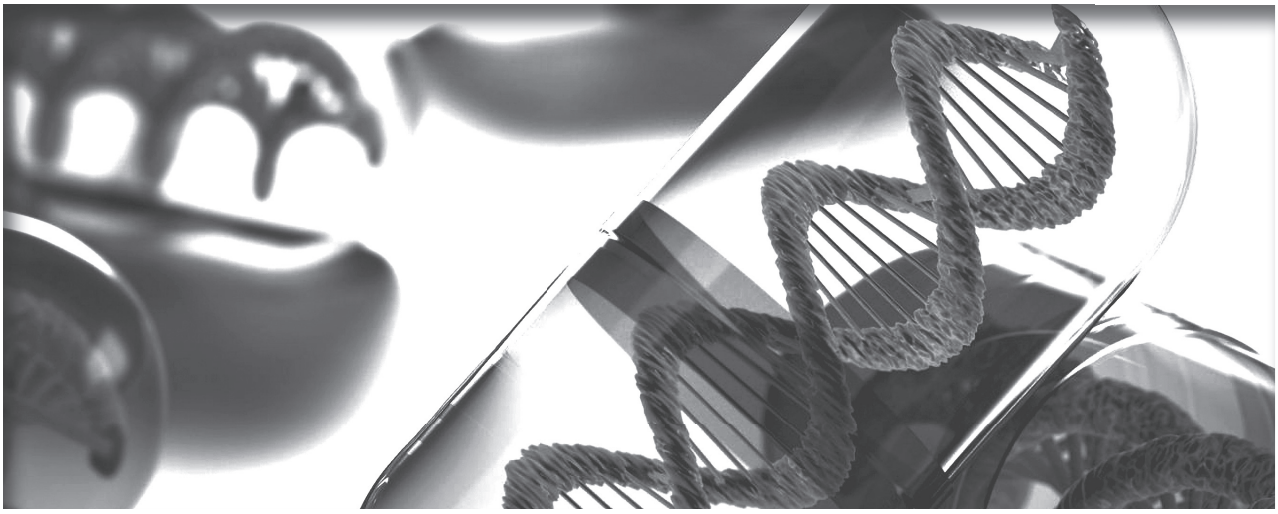

CHAPTER 25

Ventricular Arrhythmias

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Drugs may induce potentially life-threatening ventricular arrhythmias. Drug-induced ventricular arrhythmias can be divided into three categories: monomorphic ventricular tachycardia, the polymorphic ventricular tachycardia known as torsades de pointes (TdP), and drug-induced Brugada syndrome.

MONOMORPHIC VENTRICULAR TACHYCARDIA

CAUSATIVE AGENTS

Drugs that have been reported to cause or exacerbate monomorphic ventricular tachycardia are listed in **Table 25-1**.[1-177] Any of the drugs listed may cause new monomorphic ventricular tachycardia. Incessant, sinusoidal-appearing ventricular tachycardia is most often associated with the potent sodium-channel blocking agents belonging to the Vaughan Williams antiarrhythmic class IC (e.g., flecainide, propafenone), although it has also been reported in association with amiodarone. Nonsustained and sustained ventricular tachycardia have been widely reported in association with dobutamine during stress echocardiography. Although ventricular arrhythmias are a known risk associated with stress echocardiography, the incidence of ventricular tachycardia associated with dobutamine stress echocardiography is significantly higher than that associated with exercise stress echocardiography.[77,79]

EPIDEMIOLOGY

Although the overall incidence of drug-induced ventricular tachycardia is not known, the incidences associated with some specific drugs have been reported (Table 25-1). Ventricular tachycardia is recognized as a consequence of digoxin toxicity.[34-38] The incidence of ventricular tachycardia in patients taking digoxin is unknown. However, in an analysis of patients discharged from a major urban medical center with the diagnosis of digoxin intoxication, 7% had "definite" ventricular tachycardia.[35]

Table 25-1 Agents Implicated in Drug-Induced Monomorphic Ventricular Tachycardia

Drug	Incidence	Level of Evidence[a]
Aconite alkaloids[1-8]	NK	C
Acetylsalicylic acid[9,b]	NK	C
Adenosine[10-16]	Up to 5%	B
Amiodarone[17,18]	NK	C
Arsenic trioxide[19]	NK	C
Bupropion[20]	NK	C
Bupivacaine[21-25]	NK	C
Chlorpromazine[26]	NK	C
Citalopram[27,b]	NK	C
Cocaine[28-30]	NK	B
Desipramine[31-33,b]	NK	C
Digoxin[34-38]	NK	A
Dipyridamole[39-41]	0.03–0.8%	B
Disopyramide[42]	NK	C
Dobutamine[41,43-85]	0–15.7%[c]	A
Ephedrine[86,87]	NK	C
Flecainide[88-122]	0–13%	A
Gingko biloba[123]	NK	C
Ibutilide[124-131]	0–9.8%	A
Imipramine[132]	NK	C
Lacosamide[133]	NK	C
Lamotrigine[134]	NK	C
Levosimendan[135,136]	NK	A
Lithium[137,d]	NK	C
Methamphetamine[138]	NK	C
Milrinone[44,139-142]	0–9.5%	B
Niferidil[143]	3.7%	B
Procainamide[144]	NK	C
Propafenone[145-158]	0–10%	B
Ropivacaine[159,160]	NK	C
Sotalol[161]	NK	C
Terbutaline[162-164]	0–15%[e]	A
Theophylline[164-174]	NK	B
Thioridazine[32]	NK	C
Trazodone[175,176]	NK	C
Venlafaxine[134,177,b]	NK	C

NK = not known.

[a]Definitions for Levels of Evidence: Level A—evidence from one or more randomized, controlled clinical trials; Level B—evidence from nonrandomized clinical trials, prospective observational studies, cohort studies, retrospective studies, case-control studies, meta-analyses and/or postmarketing surveillance studies; and Level C—evidence from one or more published case reports or case series.

[b]Primarily in overdose.

[c]Reported as high as 29% in patients with severe heart failure.

[d]Chronic toxicity.

[e]15% incidence reported in patients with a history of ventricular arrhythmias.