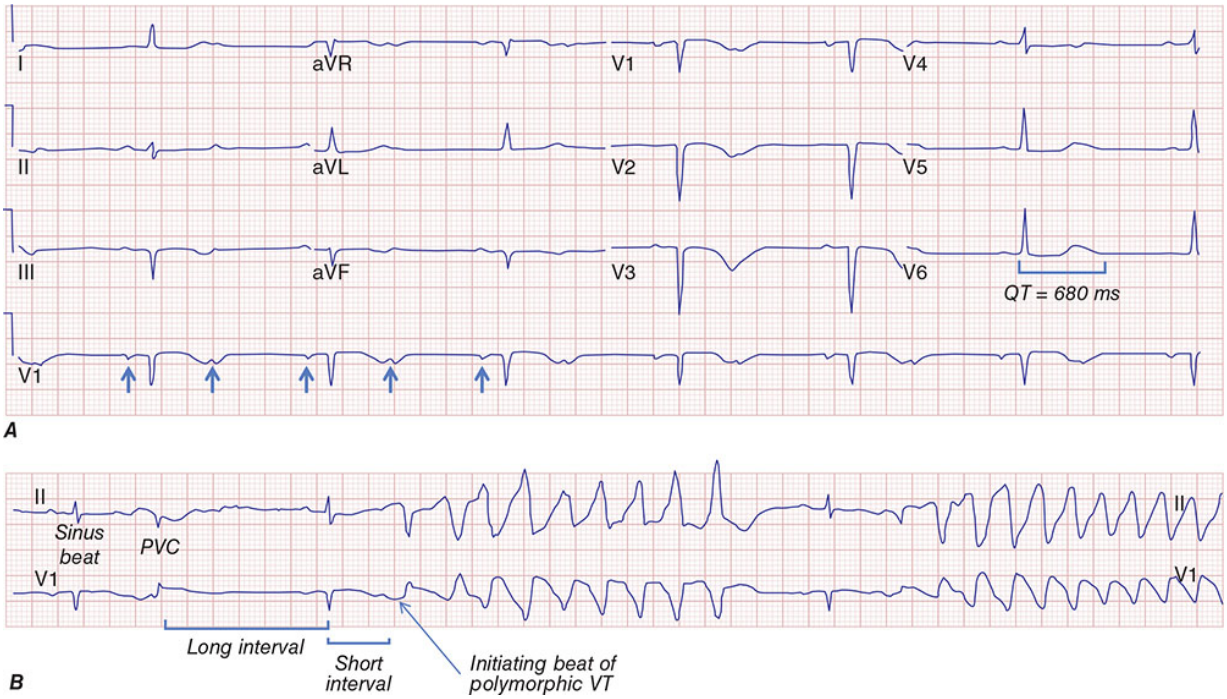

FIGURE 255-1 Torsades des pointes ventricular tachycardia (VT) in a patient with bradycardia and marked QT prolongation. ***A.*** Twelve-lead electrocardiogram showing 2:1 atrioventricular block (P waves marked by *blue arrows*) with a heart rate of 40 beats/min and QT interval of 680 ms and corrected QT of 550 ms. ***B.*** The bottom panel shows a telemetry rhythm strip with periods of self-limiting torsades des pointes polymorphic VT. Following a normally conducted sinus beat, a premature ventricular contraction (PVC) causes a compensatory pause, leading to a long RR interval. A PVC after the next sinus beat initiates VT. This is the classic pause-dependent mode of initiation of torsades des pointes VT with long–short intervals.

Patients typically present with near-syncope, syncope, or cardiac arrest. Sustained episodes degenerate to VF requiring defibrillation. PVCs and nonsustained VT often precede episodes of sustained VT. Intravenous administration of 1–2 g of magnesium sulphate usually suppresses recurrent episodes. If magnesium alone is ineffective, increasing heart rate with isoproterenol infusion or pacing, to a rate of 100–120 depolarizations/min as required to suppress PVCs, usually suppresses VT recurrences. These maneuvers allow time for correction of associated electrolyte disturbance (hypokalemia and hypocalcemia) and bradycardia and removal of any causative drugs **(Table 255-1)**. Drug interactions that elevate levels of the offending agent are often a precipitating factor. Patients who experience a