Summary. It is suggested that torsades de pointes may be only one of a group of arrhythmias that are characterized by being pause induced or bradycardia induced. A distinction is made between the cause of the “twisting of the points” and the cause of the action potentials that initiate and sustain the tachycardia. It is pointed out that torsades de pointes and other pause-induced arrhythmias share many features with rhythmic activity arising from early afterdepolarizations. Both are seen after pauses or at low rates, both are seen in quinidine intoxication, and both are seen in hypokalemia. The short-long-short sequence that is seen in torsades de pointes and certain other pause- or bradycardia-induced arrhythmias can be fully explained by the behavior of rhythmic activity initiated and sustained by early afterdepolarizations, as can the abrupt onset and termination of pause-induced arrhythmias and their tendency to show initial warming up and terminal slowing down.

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Key Words. torsades de pointes, early afterdepolarizations, cardiac arrhythmias, triggered arrhythmias.

Torsades de pointes (twisting of the points) is the name given by Dessertenne [1] to a form of ventricular tachycardia “which displays alternation of the QRS complexes so they they successively point upwards and downwards.” Since the time of Dessertenne’s description, various other features have come to be associated with torsade de pointes. For example, it has come to be regarded as “pause-induced,” i.e., precipitated by a long R-R interval or by bradycardia. It has also come to be associated with the “long QT syndrome,” in which bursts of tachycardia are foreshadowed by prolongation of the QT interval or by the appearance of a U wave.

Surawicz recently included amenability to being suppressed by an increase in heart rate as part of his definition of the arrhythmia [2]: “Torsades de pointes is a polymorphic ventricular tachycardia associated with a prolonged QT interval or increased U-wave amplitude and amenable to suppression by an increase in heart rate.” Brachmann et al. and Cranefield and Aronson have, on the other hand, suggested that torsades de pointes may be only one member of a class of ventricular tachycardias that arise from rhythmic activity occurring during an early afterdepolarization [3-5]. That class of tachycardias would be more likely to arise after a pause or at a low heart rate; would be amenable to suppression by an increase in heart rate; would, unless they arise in a small focus, often be associated with a prolonged QT interval; and could, but need not, show twisting of the points. Jackman et al. made similar suggestions and also suggested that some arrhythmias that show oscillation in QRS morphology may not even be torsades de pointes [6]: “We advocate distinguishing only those ventricular tachycardias occurring in patients manifesting QT prolongation or prominent slow waves.” Cranefield and Aronson [4], agreeing with Jackman et al. [6], did point out that to retain the name torsades de pointes for a class of arrhythmias that need not show torsades may present difficulties.

As we will see in more detail below, Cranefield and Aronson [4,5] also pointed out that the characteristics of QT prolongation and pause induction might be “concealed” if they occur only in a focus that is so small that activity in it is not manifest in surface leads. Indeed, were the features of pause induction and bradycardia induction to result from entry block, they too might be “concealed,” since they need occur only in the focus. Thus, at one extreme we have Dessertenne’s definition of torsades de pointes as a ventricular tachycardia with a specific kind of polymorphism; at the other extreme, Cranefield and Aronson’s analysis of what kind of arrhythmias might arise from sustained rhythmic activity occurring during an early afterdepolarization, namely, ventricular tachycardias that might, but need not, show twisting of the points. True torsades de pointes and the tachycardias postulated by Cranefield and Aronson [4] have in common the fact that each may well be pause induced.

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Before analyzing pause-induced tachycardias in more detail, it is important to note that there are two quite distinct questions about torsades de pointes: a) What causes the twisting of the points? and b) What causes the generation of the action potentials that give rise to the tachycardia?

What Causes the Twisting of the Points?

The simple answer to this question is: we do not know. Dessertenne [1] postulated the presence of two competing foci of ventricular activity, “one initiating QRS complexes pointing upwards, the other initiating complexes pointing downward... the transition from one focus to the other generates a torsades de pointes.” As Bardy et al. [7] later put it, the arrhythmia “can be characterized as a ventricular arrhythmia with two or more epicardial breakthrough sites vying for control of epicardial activation.” Thinking in terms of the possibility of the arrhythmia originating in a single focus, Cranefield and Aronson [4] suggested that the impulses originating in such a focus might “activate the ventricle by different pathways, perhaps because different impulses leave the focus or subsequently invade the ventricle by different pathways. Indeed, even rather slight irregularities in the interval between successive impulses arising in a single focus might give rise to substantial variations in the activation of the ventricle if the responsiveness of the exit pathway is extremely sensitive to the prematurity with which it is excited. (In this connection, Brian F. Hoffman has pointed out to us that ‘with respect to the varying conduction path from a focus as a cause of torsade, the use-dependent properties of antiarrhythmic drugs may contribute to the variation in QRS complexes or at least enhance the likelihood of such variation. Because of the increase in rate due to the tachycardia, use-dependent block might develop in some parts of the exit path and then, as these fibers recover from use-dependent block, similar block might develop in another part of the exit path. One could thus have either a “flip-flop” or an oscillatory change in the exit path in the presence of antiarrhythmic drug and, at a comparable rate, no such change in the absence of drug. Since not all cases of acquired torsades de pointes result from antiarrhythmic drugs, one would have to postulate some rate-induced delay in channel reactivation for cases induced by hypokalemia and related causes.’)

“If there is a single focus of repetitive discharge arising from an early afterdepolarization and if the impulses arising in that focus always exit from the same point and invade the ventricle in the same way, one would expect to see a monomorphic tachycardia. Certainly a bradycardia-induced monomorphic tachycardia might well be attributed to rhythmic activity arising from an early afterdepolarization, a possibility we will discuss in detail below. In reviewing torsade de pointes, Bhandari and Scheinman [8], analyzing data from various sources, including Nguyen, Scheinman, and Seger [9], comment that ‘At times, the ventricular arrhythmia may not assume the classic “torsade” appearance but may be pleomorphic in nature and, therefore, some have favored the designation of polymorphic ventricular tachycardia. Monomorphic ventricular tachycardia may also alternate with the “torsade” pattern.’

The possibilities thus include two distinct foci vying for control, a single focus with more than one exit to the rest of the ventricle, or a “wandering focus,” slowly migrating across the ventricle and back again. A variant of the “wandering focus” would be a macroscopic circus movement slowly shifting its course back and forth between two limiting pathways. But, as we said, the answer to the question, “what causes the twisting of the points?” is “we do not know.”

What Generates the Action Potentials?

Alas, once again the simple answer is: we do not know. The usual suspects are reentry, activity generated by afterpotentials, activity generated by electrotonic interactions, etc. [see 4, pp. 427-428]. Surawicz [2] has recently given a list of 11 characteristics of torsades de pointes and has discussed them in relation to reentry and to rhythmic activity during an early afterdepolarization as possible causes of the arrhythmia. He concluded that “It may be that each of these substrates exists under appropriate circumstances... At this point neither of the two proposed substrates has been conclusively documented as the cause of torsades de pointes in humans.” We agree with Surawicz’s analysis, but will now turn to other characteristics of at least some torsades de pointes and certain other ventricular tachycardias, characteristics not included in Surawicz’s list, which lead us to favor rhythmic activity during an early afterdepolarization as the cause of torsades de pointes. We have in mind peculiarities of the timing of the events that often precede a burst of torsades de pointes and certain peculiarities of the timing of the action potentials within the burst.

The Short-Long-Short Sequence

As long ago as 1969, Dessertenne, Coumel, and Fabiato said that an essential feature of torsades de pointes is it always starts after a delay following the