Chapter 20

Polymorphic Ventricular Tachycardia I

Torsade de Pointes

Let's begin by seeing how much you may (or may not) already know about ***polymorphic ventricular tachycardias*** in general…

Are you familiar with this pattern of ventricular tachycardia (Figure 20-1)?

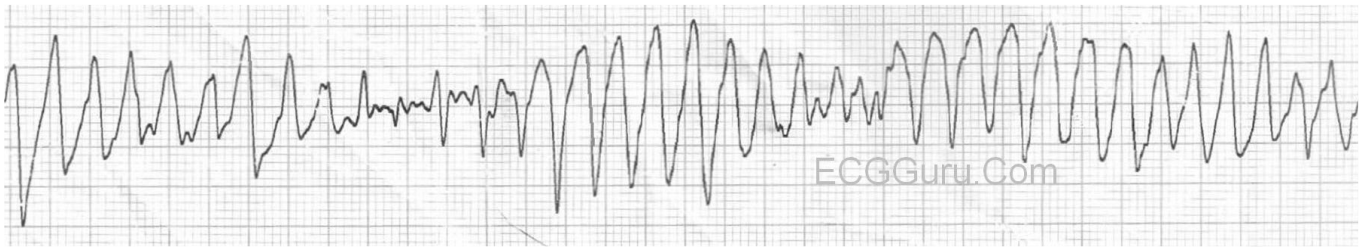


Figure 20-1

Can you identify it?

If you said *torsade de pointes* (TdP) – then you *aren't* familiar with this type of tachydysrhythmia! All that can be said is that it is a *polymorphic ventricular tachycardia* – a much more *general* term. What makes you think this is torsade de pointes? The spindle-shaped episodes in which the polarity changes from negative to positive and appears to "twist" around the baseline? The other polymorphic VTs that have *no connection* to torsade de pointes can look *the same*. Is it because TdP always occurs with a prolonged QT interval? You're *right*, but… can you show me a prolonged QT interval on this rhythm strip? You can't, can you?

The truth is that you don't know WHAT this tachycardia represents for certain *without more information* – and by more information, I mean *significant personal knowledge of this patient* and/or a *previous ECG recorded during sinus rhythm, preferably at the onset of the polymorphic VT*. In most monomorphic VTs, we study the QRS complexes during the tachycardia to learn more about them. With polymorphic ventricular tachycardias, we need to see the ECG during sinus rhythm to properly diagnose them. There is no need to show you a "documented episode" of torsades de pointes because it would look exactly like what you see in Figure 20-1.

What IS polymorphic ventricular tachycardia?

Ventricular tachycardias can also be divided into *monomorphic* and *polymorphic* based on the morphology of the QRS complexes during the tachycardia. ***Monomorphic*** ("one shape") means that all the QRS complexes *within a given lead* will look the same, i.e., all the QRS complexes in Lead II will look the same – but they may not look like the QRS complexes in Leads aVR or V1, for instance. Polymorphic ("multiple shapes") means that there are different QRS morphologies within the *same* lead.

Polymorphic VT itself can be expressed in different forms: as...

1. a simple variations in the QRS complexes:

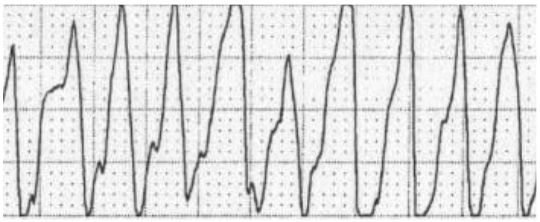


Figure 20-2

2. the iconic spindle-shaped polymorphic VT:

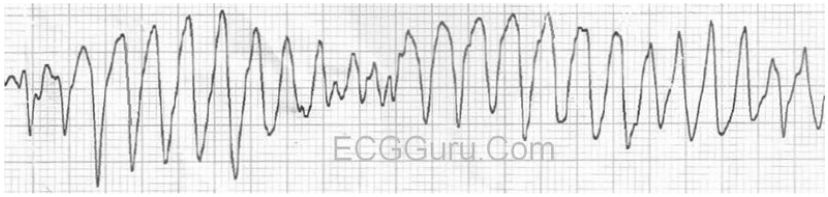


Figure 20-3

3. and bidirectional VT:

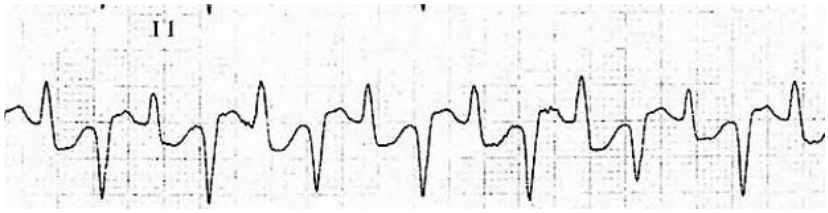


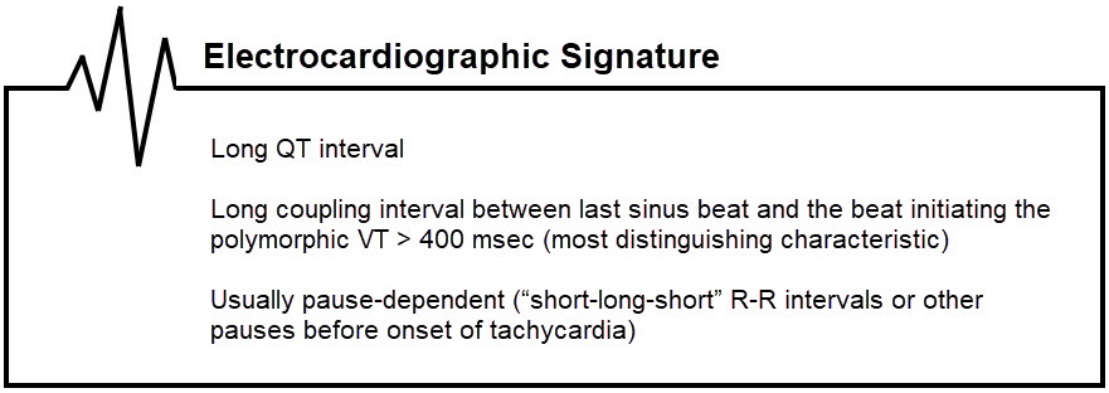
Figure 20-4

Torsade de Pointes

Torsade de pointes is the *only* polymorphic ventricular tachycardia associated with the long QT syndrome. Consequently – as demonstrated in the example that opened this chapter – if you cannot show that the spindle-shaped tachycardia is occurring in the presence

of a baseline QTc prolongation, you cannot call it torsade de pointes. It should remain a polymorphic VT until the association with the long QT interval is demonstrated.

What is the **Electronic Signature** for Torsade de Points?



The Origins of Torsade de Pointes

A long QT interval promotes and potentiates torsade de pointes by two methods:

1. prolonged repolarization allows more Ca^{++} to enter the cell during bradycardia and pauses and
2. an increased heart rate that promotes the entry of extra Ca^{++} into the cell.

Let's address each one of these methods...

Prolonging Repolarization

By prolonging repolarization, more Ca^{++} enters the cell during Phase 2 and the myocyte becomes overloaded with Ca^{++}. To remove the extra Ca^{++}, the **sodium-calcium exchanger (NCX)** is activated. This will exchange ONE intracellular Ca^{++} ion for THREE extracellular Na^{+} ions. This transport of Ca^{++} out of the cell results in an inward positive Na^{+} current. Since *all inward positive currents are depolarizing currents*, this Na^{+} current counteracts the outward K^{+} currents trying to repolarize the cell. If strong enough, the inward Na^{+} current will overwhelm the outward current and an early afterdepolarization occurs. If that early afterdepolarization reaches the threshold potential it will produce a PVC – usually during Phase 3, the T wave. Thus, an "R-on-T" phenomenon occurs. Because the QT prolongation is potentiated by bradycardia and pauses, these LQTS are referred to as *pause-dependent*.

Increased Heart Rate Introducing More Ca^{++} Into the Cell

With some long QT syndromes, a faster heart rate allows for more entry of Ca^{++} into the cell with every heartbeat. These long QT syndromes are potentiated by catecholamines which promote Ca^{++} entry. The extra Ca^{++} entering from the outside may cause a release of even greater Ca^{++} stores within the cell and the resulting Na^{+}/Ca^{++} exchanger goes into action. You know the rest. These LQTS are referred to as *tachycardia-dependent*. Although torsade de pointes is not really considered tachycardia-dependent, it can occur during episodes of acquired LQTS which are tachycardia-dependent due to the "short-long-short" sequence of intervals. The long pause is interjected and the torsade de pointes may then develop.

> **TIP |** Most people think that the Ca^{++} entering the cell is what precipitates excitation-contraction coupling – but it isn't. The Ca^{++} *entering* the cell during Phase 2 is just the "trigger" for the release of truly massive Ca^{++} stores from within the sarcoplasmic reticulum.

Differentiating Torsade de Pointes from Non-torsade Polymorphic VT

It is important to know if you are dealing with a torsade de pointes or a non-torsade polymorphic VT because they have very different causes, very different treatments, and somewhat different prognoses.

To distinguish between torsade de pointes and non-torsade VT we use the fact that there is a QTc prolongation during torsade de pointes but no significant QTc prolongation in non-torsade polymorphic VT.

First – let's understand a very important piece of information:

Both forms of polymorphic ventricular tachycardia can look the same *during* the tachycardia. You may not be able to distinguish one from the other!

Of course, this is regarding types 1 and 2 as shown at the beginning of the chapter. Torsade de pointes *never* presents with bidirectional VT.

To properly distinguish torsade de pointes from non-torsade polymorphic VT, ***you will need to see some of the sinus rhythm and the point at which the polymorphic VT was initiated***.

Why sinus rhythm? Is that to see if a prolonged QTc is present?

If the prolonged QTc were significant – over 500 msec, then "Yes!" – that would be enough to make a distinction. However, non-torsade polymorphic VT patients can occasionally have slightly prolonged QT intervals, also. This can lead to an overlap of QT intervals between the two forms of polymorphic VTs. There is a better way to distinguish them…

We use the *coupling interval* of the last sinus-conducted beat and the beat that initiates the polymorphic VT – whether it's torsade or non-torsade.

> **Important Definition! |** A *coupling interval* is the distance from the *onset* of a sinus-conducted QRS to the *onset* of an ectopic QRS that immediately follows it. It *suggests* – but *does not necessarily establish* – a relationship between the two beats.

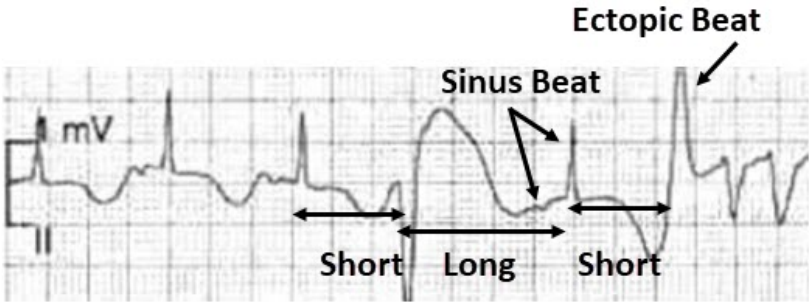


Figure 20-5

The coupling interval that we are interested in is the one that begins with the P-QRS labeled "Sinus Beat" (Figure 20-5). The sinus beat is followed by an ectopic beat at – or near – the end of the inverted T wave, which initiates the tachycardia. Note that the coupling interval is greater than two large squares (400 msec). Note the abnormally enlarged T waves indicated by the dotted arrows. This is typical of the onset of torsade de pointes.

Because of the prolonged QT interval, the coupling intervals for torsade de pointes will be long – at least 400 msec and often a lot longer.

Non-torsade polymorphic VT is not associated with a prolonged QT interval, so a coupling interval at the onset of the tachycardia will be shorter – 400 msec or less.

> **PEARL |** Torsade de pointes will have a longer coupling interval because of the prolongation of the QTc. Since non-torsade polymorphic VT is not associated with a significantly prolonged QTc, its coupling interval will be shorter.

So, in a "nutshell"…

Coupling Interval > 400 msec: **Torsade de Pointes**