QUIZ

The ECG Quiz: "Culprit Pauses"

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A 78-year-old lady presented with a several-day history of "not feeling well." On the day of admission she had several spells of syncope at home. She was admitted for observation and further evaluation.

Her past medical history included arterial hypertension and mild dementia; otherwise she was healthy. Her physical examination was unremarkable and her echocardiography was within the normal limits for her age.

In the hospital she developed the rhythm shown below in Fig.1.

Questions:

1. What is the interpretation of the rhythm strips?
2. How do you treat this arrhythmia?
3. Will this patient need an implantable cardioverter defibrillator (ICD)?

Answers:

1. High-grade AV block associated with bradycardia-dependent Polymorphic Ventricular Tachycardia (PMVT).
2. Give intravenous magnesium sulfate and/or Lidocaine, then work on accelerating her heart rate using drugs or transvenous temporary pacemaker.
3. The patient will need a permanent pacemaker. An ICD is not indicated.

Discussion

The most impressive finding on the tracings is the fast wide QRS tachycardia which is consistent with PMVT (note a slight, but definite, change in QRS morphology at initiation and termination of VT). This occurred on a background of a slow heart rate (i.e., bradycardia). So, how to explain this combination?

As illustrated in Fig.2, the underlying rhythm is sinus with the P waves most visible on top and bottom strips (black arrows). Some of these P waves were conducted to the

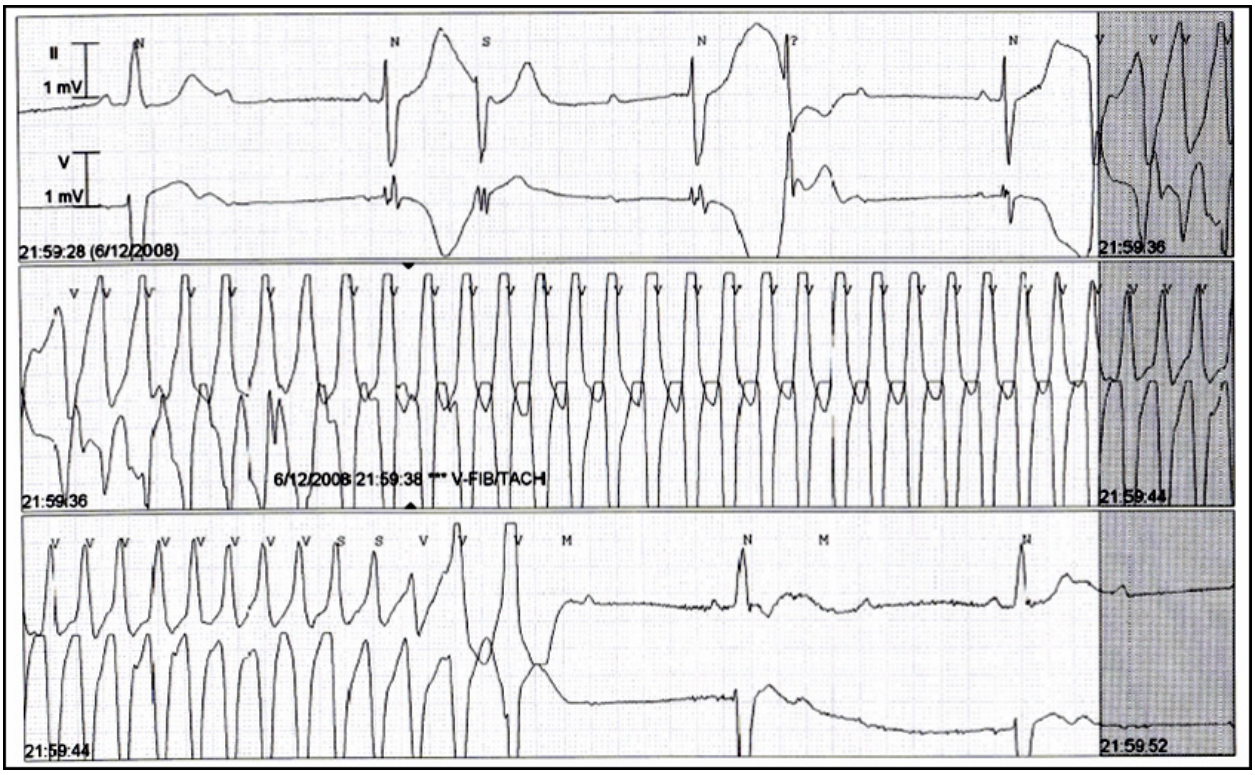

Fig 1: Telemetry Tracing

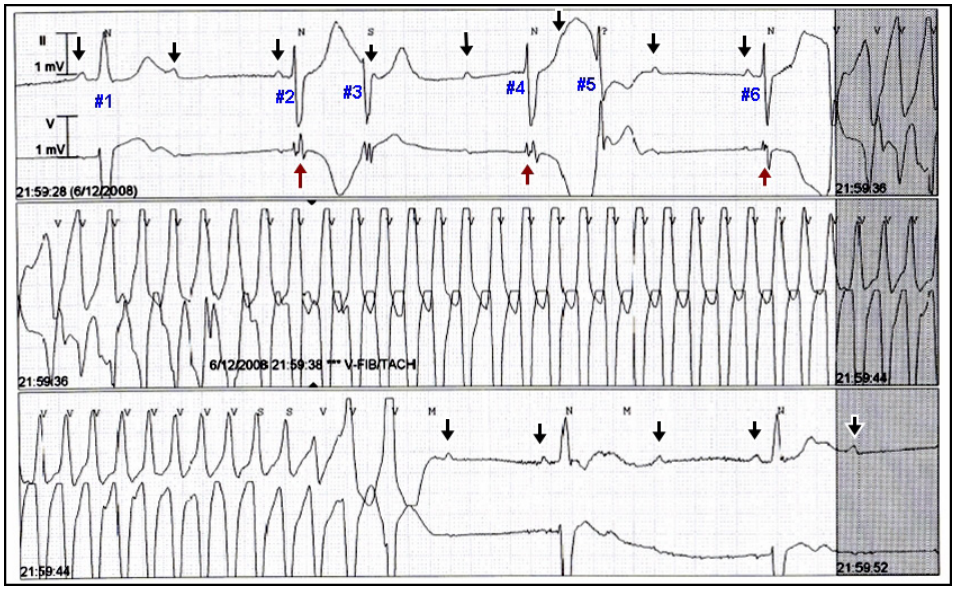

Fig 2: Mechanisms of bradycardia

ventricle (beat # 1 on top strip and last two ventricular beats on bottom strip); however, several of them failed to conduct. On the top strip, the first P wave was conducted, however, after that a high-grade AV conduction disturbance ensued. There was probably some degree of AV dissociation with the P waves being faster than the slower, and regular, ventricular escape rhythm (beats #2, #4, and #6 - brown arrows). In addition, there were two premature ventricular contractions (PVCs) (beats #3, and #5) following the ventricular escape beats, and; finally a wide QRS complex tachycardia started after beat # 6. One may wonder if beats #2 and # 6 were conducted sinus beats as they were preceded by P waves with a relatively close PR interval. Based on this strip alone it is hard to know for sure whether these P waves were conducted or this was just a coincidence. However, with a closer look at the possible ventricular escape beats on the top strip, one may notice that beat # 6 is slightly different in morphology and is slightly thinner than beats #2, and #4. This is most apparent in lead V. Also, the PR interval of beat #6 is probably marginally longer than that of beat #2. Therefore, it is possible that beat #6 is a fusion between a ventricular escape beat and a conducted P wave, while the P wave before beat #2 was not conducted as it did not affect the timing or the morphology of the otherwise regular escape rhythm. Regardless, it is obvious that the top rhythm strip displays a high-grade AV conduction disturbance. Besides, even the conducted P waves (first complex of the top tracing and last two on the bottom tracing) generated wide QRS complexes which is another manifestation of conduction disease. The severe conduction abnormality explains the underlying bradycardia, but how to explain the wide QRS tachycardia? And what is the relationship between the two?

Fig. 3 illustrates the pathogenesis of the wide QRS tachycardia, and again, the clues are most visible on the top strip. On that strip, the combination of blocked P waves and/or PVCs created several "pauses" (black double headed arrows). The beats immediately following these pauses (beats #2, #4 and #6) display significant changes in ventricular repolarization characterized by bizarre-looking T waves that are unusually giant and wide and are inverted in certain leads (brown arrows). Also the QT intervals of these beats were prolonged (>600 ms). These beats following the pauses are very destabilizing and usually end up with PVCs (beats #3 and #5), short runs of VT, or the initiation of a polymorphic ventricular tachycardia (PMVT) as it shown in this tracing. This "pause-dependent" repolarization abnormality is the hallmark of the famous arrhythmia: "Torsades de pointes." In fact, the pause-dependent initiation (also known as: "Long-short" initiation) is

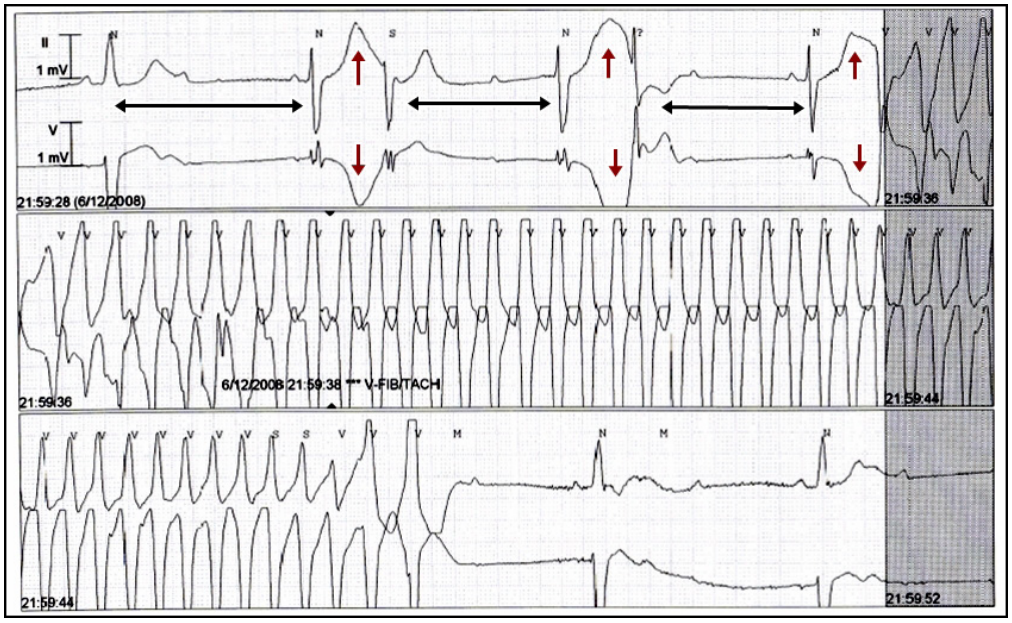

Figure 3: Pathogenesis of PMVT

probably a more important feature in characterizing this arrhythmia than the typical "flipping of the points" which can be seen in PMVTs of different mechanisms (typically ischemia). Nevertheless, another rhythm strip taken from the same patient during monitoring (Fig.4) showed that typical appearance (brown double headed arrows).

In this patient the pauses created by the slow heart rate due to AV block led to the repolarization abnormalities needed to trigger this PMVT. The susceptibility to develop these "bradycardia-dependent" repolarization abnormalities varies from one patient to another (probably on a genetic basis) and is related to some defect involving the repolarizing K+ channels. The typical patient is an elderly woman with a slow, and irregular, heart rate (like in this case). Other acquired predisposing factors include electrolyte abnormalities (hypokalemia or hypomagnesaemia), medications that prolong QT, etc.

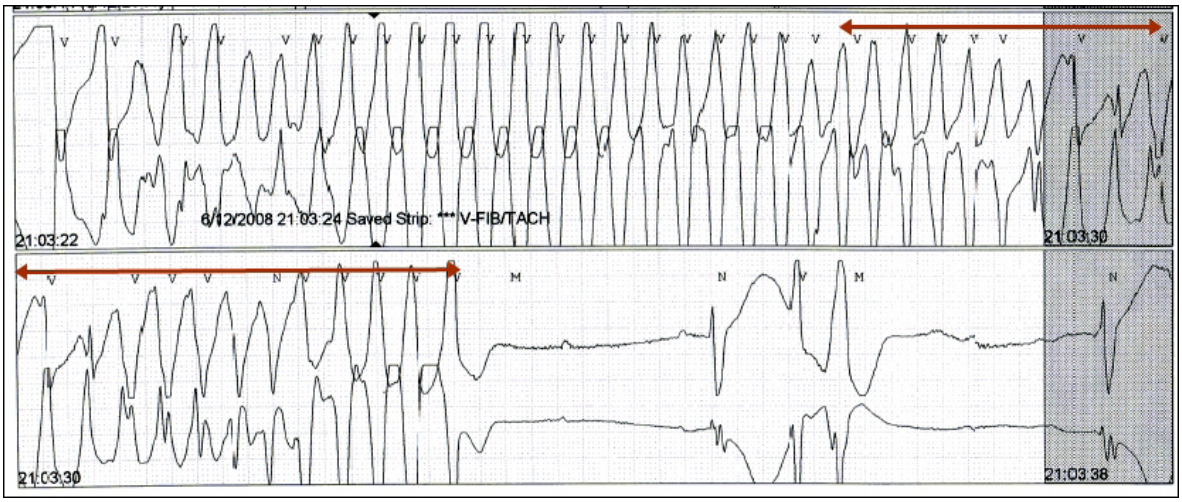

Figure 4: Typical "flipping of the points"

These VT spells are typically fulminant and clustered, but are usually self-terminating. Most patients present with syncope and/or palpitations. When witnessed, most of these PMVT runs terminate spontaneously before there is a chance for cardioversion. Nevertheless, these runs typically recur shortly after if the underlying problem is not corrected.

Acutely, the patient should be given intravenous magnesium sulfate even if the blood magnesium level is normal. Intravenous Lidocaine can also be tried acutely. However, the main urgent intervention should be directed at increasing the heart rate and hence minimizing these "culprit pauses." The most effective way to achieve that is by pacing the heart fast enough to eliminate these pauses. Intravenous positive chronotropic drugs (e.g., Isoproterenol) can also be tried. In addition, other potential causes for QT prolongation (e.g., hypokalemia, drugs, etc) should be sought and treated. In general, once the insulting event is corrected, the prognosis of this arrhythmia is good.

This patient got a temporary transvenous pacemaker inserted emergently which eliminated these runs of PMVT. On the second day she received a dual chamber pacemaker then she was sent home in good condition the day after. Since this patient had a normal LV function and her VT was secondary to a correctible cause (heart block), an ICD implantation was not indicated. This problem could be treated simply by implanting a permanent pacemaker to eliminate pauses, and hence the trigger for these PMVT. The detail of the ideal pacemaker setting is beyond the scope of this discussion.

The take home message is to recognize this unique form of arrhythmia based on the mode of initiation as its treatment and prognosis are very different from other forms of PMVT.

Suggested Readings

1. Tan HL, Hou CJY, Lauer MR, Sung RJ. Electrophysiologic mechanisms of the long QT interval syndromes and torsade de pointes. Ann Intern Med. 1995;122:701-14.
2. Tzivoni D, Banai S, Schuger C, Benhorin J, Keren A, Gottlieb S, Stern S. Treatment of torsade de pointes with magnesium sulfate. Circulation. 1988;77:392–97.