Challenges in Clinical Electrocardiography

Bradycardia-Induced Syncope With a Twist

Thomas Lindow, MD; Olle Pahlm, MD, PhD; Adrian Baranchuk, MD, PhD

A man in his 60s without medications or history of cardiovascular disease noted a low pulse when checking his blood pressure at home. He had no symptoms of bradycardia, such as dizziness, syncope, or fatigue. A 12-lead electrocardiogram (ECG) showed a type 1 second-degree atrioventricular (AV) block and he underwent Holter monitoring. An appointment was scheduled with a cardiologist the following week. Later the same night, he had a syncopal episode and was referred to the emergency department. The Holter recording was analyzed immediately. Electrocardiogram strips from the recording can be found in the **Figure**.

Questions: What is the rhythm in the ECG strips described? In the second strip (Figure, B), what mechanisms of ventricular tachycardia (VT) can be identified?

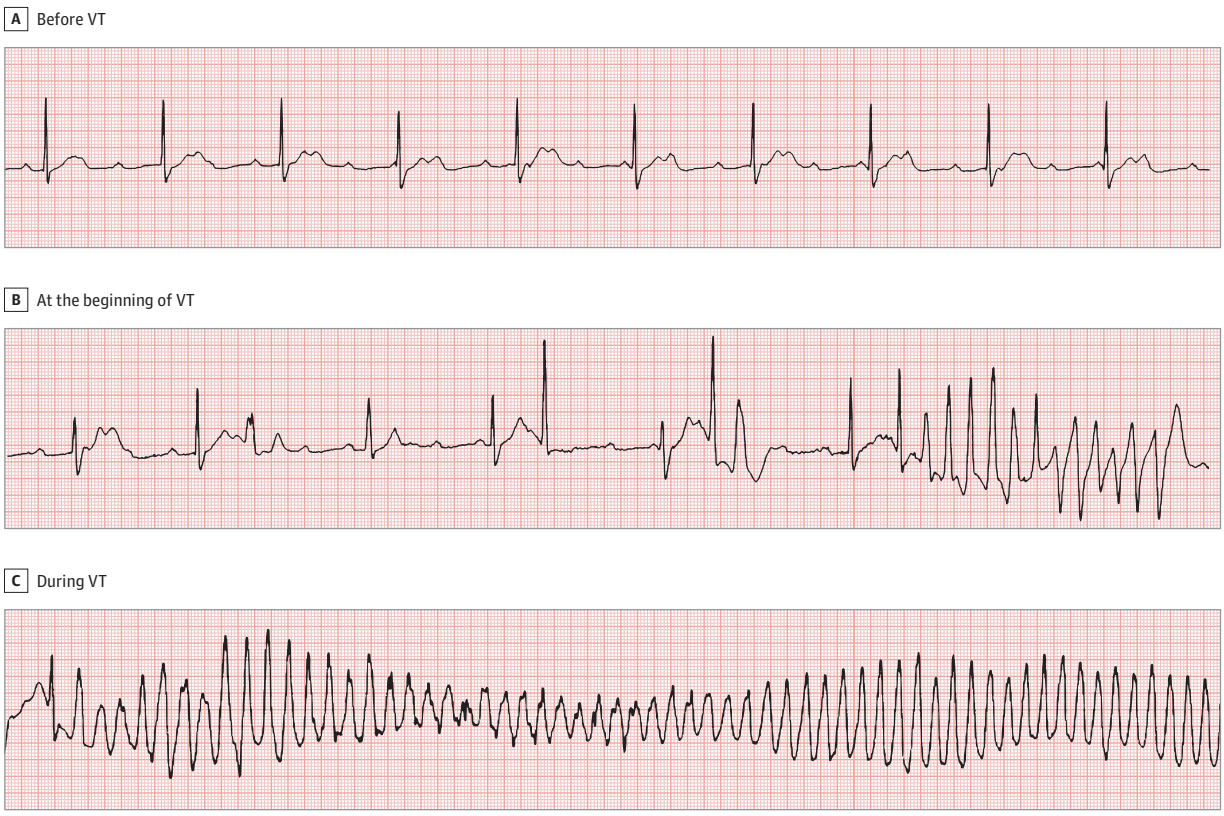
Interpretation

The ECG strips (Figure, A and B) show completely dissociated P waves and narrow QRS complexes, with some of the P waves superimposed on the T waves (ie, a complete AV block with a narrow complex escape rhythm at 42 beats per minute, suggesting an AV-nodal block). In the first 2 strips (Figure, A and B), QT intervals are prolonged (>600 milliseconds) and in the second strip, frequent premature ventricular contractions (PVCs) are present. At the end of the second strip, a PVC couplet is followed by a narrow beat and a PVC, which induces a polymorphic ventricular tachycardia with changing QRS complex amplitudes (Figure, C), twisting around the isoelectric line, which is known as torsade de pointes (TdP; English translation, twisting of the points).

Clinical Course

In AV block, atrial impulse propagation to the ventricles is either delayed (first-degree AV block) or interrupted (second/third-degree AV block) in the AV node or the His-Purkinje system. Most often, the treatment decision of pacemaker implant is based on bradycardia-associated symptoms and electrocardiographic evidence of an advanced block with (or without) episodes of asystole. However, AV block is also associated with malignant ventricular arrythmia.[1]

Figure. Electrocardiogram Strips From the Holter Recording



The duration of ventricular tachycardia (VT) was approximately 2 minutes.

Torsade de pointes may be caused by many conditions that affect repolarization that leads to prolonged QT intervals, such as drug therapy (eg, antiarrhythmic agents, some antibiotics, antidepressants, and antiemetics), electrolyte abnormalities (eg, hypokalemia, hypomagnesemia, and hypocalcemia) and congenital long QT syndrome.[2-4] The risk of drug-induced TdP is increased in elderly patients (>65 years), in patients with concomitant bradycardia, electrolyte abnormalities, or female sex.[3] In this patient, no electrolyte abnormalities were found and he was not taking medications that were known to prolong the QT interval.

Discussion

Prolonged repolarization, often present in a complete AV block, is a prerequisite for TdP.[5] Not all patients with AV block are at risk for developing TdP. Prolonged QT intervals and the presence of PVCs are known indicators of the risk of TdP. Patients with AV block and TdP have longer QT intervals than patients with AV block without TdP.[6] Interestingly, women seem to be at risk of developing TdP at QT intervals that are not arrhythmogenic for men.[6] Recently, it was suggested that AV block–induced TdP may be a marker of an underlying genetic predisposition for repolarization abnormalities, as a form of "latent" long QT syndrome. In 4 out 11 patients with AV-block induced TdP, a genetic variant associated with congenital long QT syndrome was found.[7] Another marker of increased risk of TdP is the prolongation of the T_{peak}-T_{end} interval.[1,8] In this patient, the time from T_{peak} to T_{end} was increased (250 milliseconds [cutoff, 117 milliseconds[1]]).

During short-term TdP treatment, it is important to avoid potential causes of TdP recurrence, such as medications associated with QT prolongation (they should be discontinued immediately), electrolyte disturbances, and ischemia.[9] Also, TdP may occur even after implanting a pacemaker, especially at low heart rates.[9,10] Although to our knowledge no specific programmed lower rate have been defined, rates less than 70 beats per minute cannot provide reliable protection against TdP recurrence.[10] A permanent pacemaker was implanted and no further symptoms were reported in the initial follow-up of 2 months.

Take Home Points

- Syncope in AV block is not always caused by asystole but may instead be caused by TdP.
- Several medications and conditions are associated with TdP, with the common denominator of a prolonged repolarization.
- Increased QT intervals, especially with an increased time from the peak to the end of the T wave, are a marker of increased risk of TdP in patients with bradycardia and AV block.

ARTICLE INFORMATION

Author Affiliations: Department of Clinical Physiology, Växjö Central Hospital, Department of Research and Development, Region of Kronoberg, Växjö, Sweden (Lindow); Clinical Sciences, Department of Clinical Physiology, Lund University, Lund, Sweden (Lindow, Pahlm); Division of Cardiology, Queen's University, Kingston, Ontario, Canada (Baranchuk).

Corresponding Author: Thomas Lindow, MD, Department of Clinical Physiology, Växjö Central Hospital, 351 88 Växjö, Sweden (thomas.a.lindow@gmail.com).

Published Online: March 25, 2019. doi:10.1001/jamainternmed.2019.0104

Conflict of Interest Disclosures: None reported.

REFERENCES

1. Topilski I, Rogowski O, Rosso R, et al. The morphology of the QT interval predicts torsade de pointes during acquired bradyarrhythmias. *J Am Coll Cardiol*. 2007;49(3):320-328. doi:10.1016/j.jacc.2006.08.058

2. Gupta A, Lawrence AT, Krishnan K, Kavinsky CJ, Trohman RG. Current concepts in the mechanisms and management of drug-induced QT prolongation and torsade de pointes. *Am Heart J*. 2007;153(6):891-899. doi:10.1016/j.ahj.2007.01.040

3. Zeltser D, Justo D, Halkin A, Prokhorov V, Heller K, Viskin S. Torsade de pointes due to noncardiac drugs: most patients have easily identifiable risk factors. *Medicine (Baltimore)*. 2003;82(4):282-290. doi:10.1097/01.md.0000085057.63483.9b

4. Yan GX, Lankipalli RS, Burke JF, Musco S, Kowey PR. Ventricular repolarization components on the electrocardiogram: cellular basis and clinical significance. *J Am Coll Cardiol*. 2003;42(3):401-409. doi:10.1016/S0735-1097(03)00713-7

5. Volders PG, Sipido KR, Vos MA, et al. Downregulation of delayed rectifier K(+) currents in dogs with chronic complete atrioventricular block and acquired torsades de pointes. *Circulation*. 1999;100(24):2455-2461. doi:10.1161/01.CIR.100.24.2455

6. Chorin E, Hochstadt A, Viskin S, et al. Female gender as independent risk factor of torsades de pointes during acquired atrioventricular block. *Heart Rhythm*. 2017;14(1):90-95. doi:10.1016/j.hrthm.2016.09.013

7. Subbiah RN, Gollob MH, Gula LJ, et al. Torsades de pointes during complete atrioventricular block: Genetic factors and electrocardiogram correlates. *Can J Cardiol*. 2010;26(4):208-212. doi:10.1016/S0828-282X(10)70369-X

8. Tse G, Gong M, Meng L, et al. Predictive Value of T_{peak}-T_{end} indices for adverse outcomes in acquired QT prolongation: a meta-analysis. *Front Physiol*. 2018;9:1226. doi:10.3389/fphys.2018.01226

9. Yazdan-Ashoori P, Digby G, Baranchuk A. Failure to treat torsades de pointes. *Cardiol Res*. 2012;3(1):34-36.

10. Pinski SL, Eguía LE, Trohman RG. What is the minimal pacing rate that prevents torsades de pointes? insights from patients with permanent pacemakers. *Pacing Clin Electrophysiol*. 2002;25(11):1612-1615. doi:10.1046/j.1460-9592.2002.01612.x