Interference dissociation in the presence of dual atrioventricular nodal physiology

Emilio L. Garcia, MD, Robert Kim, MD, Steve S. Hsu, MD, FHRS, John N. Catanzaro, MD, FACC

From the University of Florida Health Science Center, Jacksonville, Florida.

Introduction

Dual atrioventricular nodal physiology (DAVNP) is present in 10% to 35% of the general population and is known to regress with aging.[1] It usually manifests as a critical AH jump on electrophysiologic testing or on surface electrocardiography as a change in PR interval with subsequent tachycardia. Preferential choice of pathway engagement (slow vs fast) is dependent upon refractory periods of the pathways and conduction velocity. We present a rare manifestation of DAVNP on surface electrocardiography with alternating engagement and disengagement of the 2 pathways masquerading as AV dissociation.

Case report

A 60-year-old woman with no significant past medical history, in her usual state of health, presented to her ophthalmologist's office for a scheduled procedure of cataract surgery. Prior to the procedure an electrocardiogram (ECG) (Figure 1) was performed, owing to an irregular pulse. The ECG was interpreted as abnormal by her physician and subsequently led to deferral of her procedure. She was referred to our hospital, where she was admitted for further assessment of her abnormal ECG. The patient denied any previous symptoms of palpitations, syncopal episodes, chest pain, or lightheadedness. She does not take any medications and her physical examination including her vital signs were all within normal limits.

Figure 1 demonstrates her presenting ECG at her ophthalmologist's office upon the ophthalmologist's noticing her pulse to be "irregular." Upon admission, alternating or "grouped beating" and intervals of progressive PR shortening were noted on telemetry. There was absence of symptom rhythm correlation. Transthoracic echocardiography demonstrated absence of structural heart disease and normal left ventricular ejection fraction. Exercise treadmill testing demonstrated shortening of her PR interval, chronotropic competence, and achievement of her maximum predicted heart rate.

Upon return to her room a continuous rhythm strip was performed (Figure 2), which again illustrated progressive PR interval shortening, as previously seen on telemetry. Close inspection of a repeat ECG (Figure 3) demonstrates abrupt shortening and lengthening of the PR interval ("PR alternans") with engagement and disengagement of the slow and/or fast pathway. Her QRS complex was narrow, suggesting brisk infranodal conduction. Her ECG normalized prior to discharge with 1:1 conduction down the fast pathway. Given that she was asymptomatic, she was followed as an outpatient.

Discussion

Dual AV nodal physiology indicates the presence of 2 distinct electrophysiologic pathways with different conduction velocities and refractory periods. The shorter PR interval represents conduction over the fast pathway and the longer PR interval represents conduction over the slow pathway. The shift in conduction from fast to slow pathway can occur spontaneously or can be provoked or terminated by an atrial premature complex, atrial tachycardia, interpolated junctional premature complexes, or a ventricular premature complex. Findings compatible with simultaneous conduction along 2 pathways in response to a ventricular premature complex were noted in our patient's ECG (Figure 3), indicating presence of underlying dual AV nodal physiology.

Dual AV nodal physiology can manifest itself as normal sinus rhythm, spontaneous shortening or lengthening of the PR interval persisting for varying periods of time, PR interval alternans,[2–7] PR interval alternans with Wenckebach sequence of the slowly and rapidly conducting pathways, and conduction along both pathways in response to a single sinus impulse.[8–12] In the presence of sinus rhythm the presenting ECG (Figure 1) and continuous rhythm strip (Figure 2) illustrate an unusual presentation masquerading as dual AV nodal physiology. These 2 ECGs show interference dissociation with progressive PR interval shortening in parallel with the influence of autonomic activity accelerating the heart rate. This causes the P waves to approach the QRS and allows a ventricular capture, which establishes restoration of normal sinus rhythm.

KEYWORDS Interference dissociation; Dual AV nodal physiology; PR alternans (Heart Rhythm Case Reports 2017;3:49–52)

Address reprint requests and correspondence: Dr John N. Catanzaro, University of Florida Health Science Center, 655 W. 8th St, Jacksonville, FL 32202. E-mail address: john.catanzaro@jax.ufl.edu.

http://dx.doi.org/10.1016/j.hrcr.2016.08.017

KEY TEACHING POINTS

- Contradirectional interference results when 2 stimuli arising in different foci in any part of the heart spread in opposite directions toward each other. Interference dissociation is defined as that type of dissociation which is due to repetitive contradirectional interference.
- Despite its appearance, there is no indication for invasive intervention in an asymptomatic patient.

Post–premature ventricular contraction PR interval prolongation: Concealed conduction

Concealed conduction is a phenomenon that describes partial penetration of an impulse into a given tissue (eg, the AV node) but can only be inferred by the behavior of the subsequent impulse that conducts through the same tissue.[13]

Concealed conduction can block the dual pathway physiology from being set into motion by prolonging the refractory period of the slow pathway. In Figure 3, premature ventricular contraction 1 conducts retrograde into the fast pathway of the AV node and renders it refractory. The next P wave conducts antegrade via the slow pathway of the AV node, resulting in significant PR prolongation. Anterograde slow pathway conduction persists owing to concealed retrograde conduction into the fast pathway (so-called concealed "linking") and continues until the middle of the bottom tracing, where, after a fully compensatory pause, anterograde conduction through the normal fast pathway resumes.[14]

Conclusion

This case illustrates a rare manifestation of DAVNP on surface electrocardiography. Perhaps increased familiarity with the 12-lead ECG manifestations of this physiologic

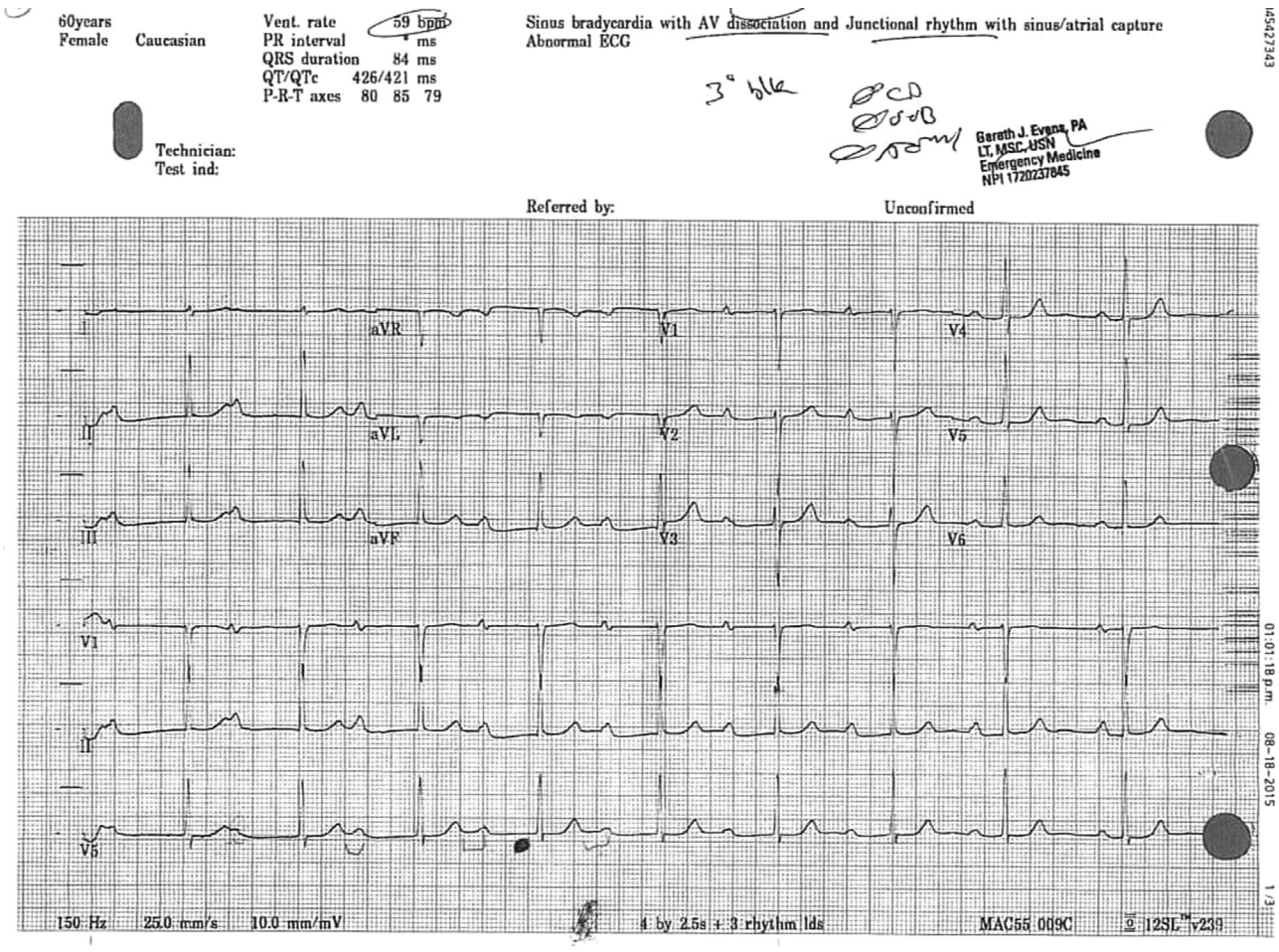

Figure 1 Presenting 12-lead electrocardiogram.

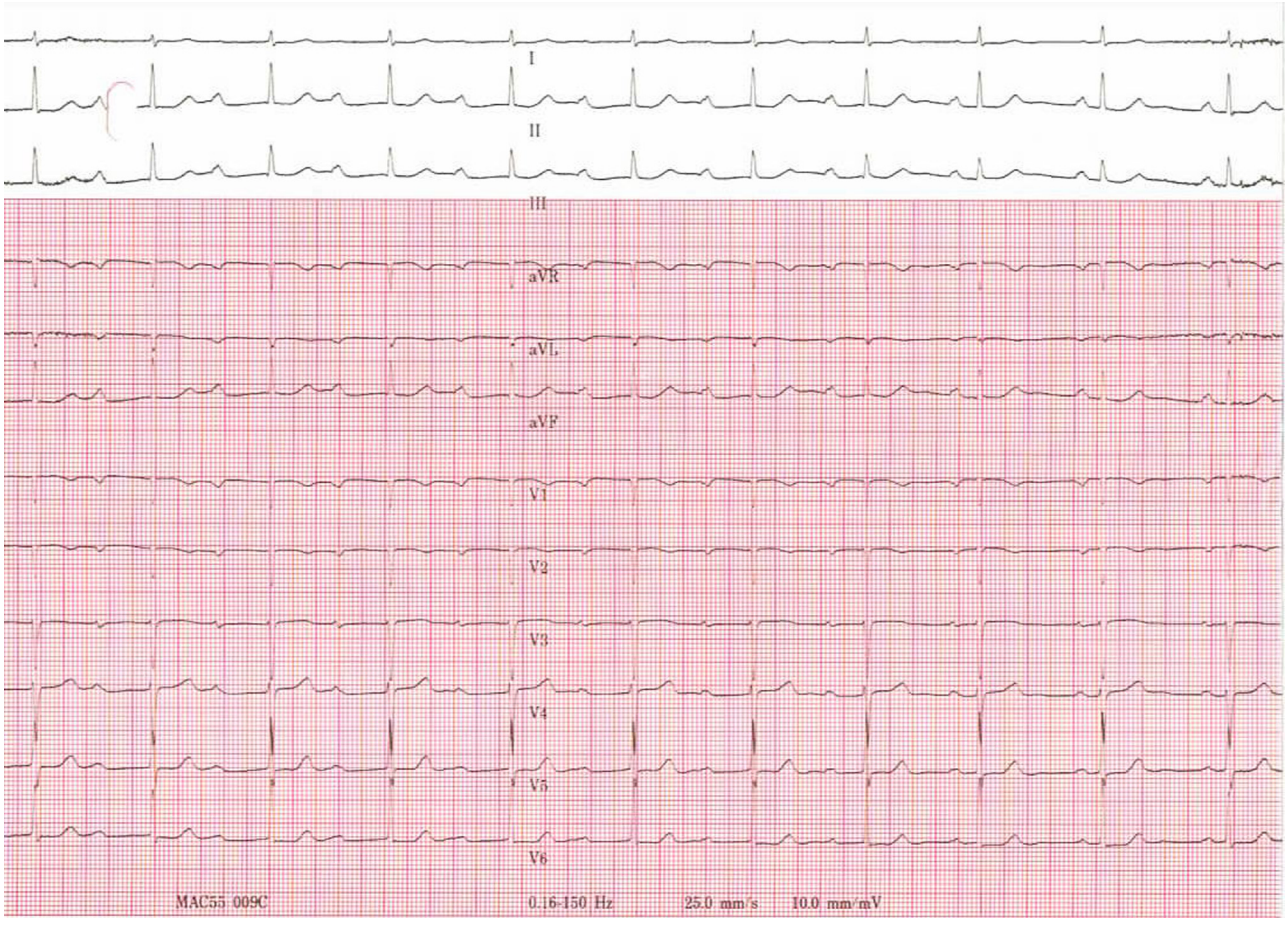

Figure 2 Continuous rhythm strip.

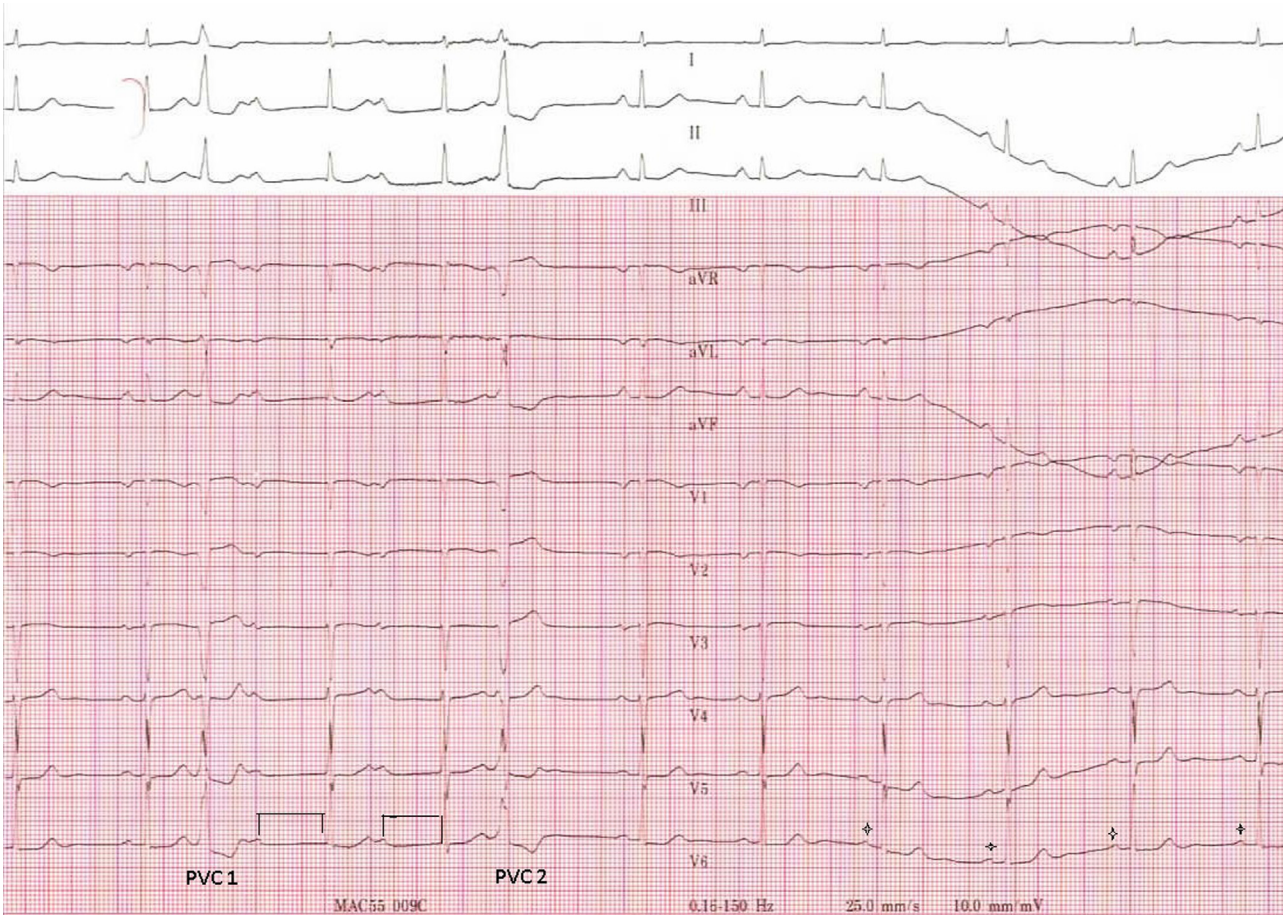

Figure 3 Fast pathway initially disengaged by a premature ventricular contraction (PVC1), subsequently engaging the slow pathway with the same premature ventricular contraction (PVC1), and reengaging the fast pathway once again (PVC2). Slow pathway conduction demarcated by long PR interval marked by brackets [ ] with transition to fast pathway conduction indicated by asterisks *.

phenomenon may alter a differential diagnosis to prevent unnecessary admissions or pacemaker implantation.

References

1. Mani BC, Pavri BB. Dual atrioventricular nodal pathways physiology: a review of relevant anatomy, electrophysiology, and electrocardiographic manifestations for the physician-in-training. Indian Pacing Electrophysiol J 2014;14(1):12–25.
2. Schamroth L, Perlman MM. Periodic variation in AV conduction: a study in differential dual AV pathway conduction and refractoriness. J Electrocardiol 1973;6:81–84.
3. Katz L, Pick A. Clinical electrocardiography: the arrhythmias. Philadelphia: Lea & Febiger; 1956:567–568.
4. Pick A, Langendorf R. Interpretation of complex arrhythmias. Philadelphia: Lea & Febiger; 1979:227, 231
5. Mamlin JJ, Fisch C, Sustained AV. conduction delay due to interpolated ventricular premature systole. Am J Cardiol 1965;16:765–766.
6. Kinoshita S, Kawasaki T, Fujiwara S, Okimori K. Periodic variation in AV conduction time: mechanism of initiation, maintenance and termination of periods of long PR intervals. Am J Cardiol 1984;53:1288–1291.
7. Surawicz B, Fisch C. Cardiac alternans: diverse mechanisms and clinical manifestations. J Am Coll Cardiol 1992;20:483–499.9.
8. Fisch C. Electrocardiography of Arrhythmias. Philadelphia: Lea & Febiger; 1990: 393.
9. Zipes DP. Specific arrhythmias: diagnosis and treatment. In: Braunwald E, ed. Heart Disease. Philadelphia: Saunders; 1992:689.
10. Lee KW, Badhwar N, Scheinman MM. Supraventricular tachycardia – part I. Curr Probl Cardiol 2008;33:467–546.
11. Lin LJ, Lin JL, Lai LP, Chen JH, Tseng YZ, Lien WP. Effects of pharmacological autonomic blockade on dual atrioventricular nodal pathways physiology in patients with slow-fast atrioventricular nodal reentrant tachycardia. Pacing Clin Electrophysiol 1998;21:1375–1379.
12. Belhassen B, Fish R, Glikson M, Glick A, Eldar M, Laniado S, Viskin S. Noninvasive diagnosis of dual AV node physiology in patients with AV nodal reentrant tachycardia by administration of adenosine-5'-triphosphate during sinus rhythm. Circulation 1998;98:47–53.
13. Denes P, Wu D, Dhingra R, et al. Dual atrioventricular nodal pathways. A common electrophysiological response. Br Heart J 1975;37:1069–1076.
14. Fisch C J Am Coll Cardiolol. 1989;Nov 1;14(5):1127-38.