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An interesting ECG in a patient with a dual chamber pacemaker☆,☆☆

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Case

A 71-year-old male with a bilateral lung transplant in 2006, coronary artery disease and history of percutaneous coronary intervention (PCI) in 2010, and chronic kidney disease presented with chest pain and volume overload. He was diagnosed with a non-ST elevation myocardial infarction and acute exacerbation of heart failure with preserved ejection fraction. He underwent coronary angiography with PCI of the left circumflex artery and was started on peritoneal dialysis.

Following the procedure, he was noted to have paroxysmal atrial fibrillation with slow ventricular response as well as sinus bradycardia with varying degrees of AV block (Fig. 1). All beta blockers and calcium channel blockers were discontinued, but he continued to have slow atrial fibrillation and sinus rhythm with occasional high-grade AV block with ventricular rate in the range of 40–45 bpm (in the absence of all negative chronotropic agents). A dual chamber pacemaker (Medtronic®, Table 1) was implanted. A follow-up ECG shown in Fig. 2. The ECG demonstrates which of the following:

A) Sinus rhythm with PVCs in bigeminy
B) Ventricular pacing with a bundle branch re-entry circuit
C) Atrial lead dislodgement with dual ventricular pacing
D) Bidirectional PVCs

Explanation/discussion

In the case presented, the post-pacemaker implantation ECG shows underlying atrial fibrillation with ventricular pacing with a bundle branch re-entry circuit. The proposed mechanism of such a rhythm is that an impulse originates from the right ventricular pacemaker lead and travels retrograde through the right bundle. Then, at the level of His, the signal turns around and travels antegrade down the left posterior fascicle to generate a ventricular contraction with a right bundle branch block and left anterior hemiblock configuration.

In 1974, Akhtar et al. first proved the existence of a re-entry circuit within the His-Purkinje system using human subjects [1]. He measured A, H, and V signals while providing ventricular extra-stimuli and noted that within a narrow range of ventricular coupling intervals, there was a critical V—H delay that consistently resulted in another beat of ventricular origin (V_3). He postulated that during the narrow range of ventricular coupling, the right bundle was selectively blocked and the signal travelled retrograde through the left bundle to activate the bundle of His, after which an antegrade signal would then travel through the right bundle to generate V_3. To support his hypothesis, he was able to demonstrate five characteristics of a bundle branch re-entry circuit:

1. V_3 occurred during a narrow interval of ventricular coupling and did not occur if the ventricular muscle was refractory.
2. The occurrence of V_3 required retrograde and delayed conduction though the His-Purkinje system.
3. V_3 did not occur if the delivered impulse was blocked retrogradely below the His bundle recording site.
4. The occurrence of V_3 was not related to retrograde AV node delay.
5. V_3 would even occur in the absence of V-A conduction when the AV node was blocked

In fact, one of the 24 patients in which bundle branch re-entry was demonstrated was in atrial fibrillation.

Without an electrophysiology study and His electrograms, which are not available, the diagnosis of bundle branch re-entry cannot be confirmed. However, there is a surface ECG (Fig. 2) and the pacemaker interrogation shown in Fig. 3 that contains A and V signals. All of the information available supports the presence of a bundle branch re-entry circuit via the mechanism described by Akhtar et al. First, the re-entry beat has a right bundle-like morphology with a left anterior fascicular block pattern due to selective activation of the right bundle during right ventricular pacing, which means that the right bundle is still refractory when the re-entry beat is moving antegrade. This pattern is the opposite of that described by Akhtar, but the underlying mechanism is still the same. Second, there is no relationship between the A signals on the pacemaker interrogation and the paced beat or re-entry beat, which indicates that the re-entry circuit is below the AV node. The PVCs demonstrate a stable coupling interval at 570 ms, which is not pathognomonic of bundle branch re-entry PVCs [2], and it's possible the PVCs could originate from infranodal scar tissue or the posteromedial papillary muscle. However, the initial selective activation of the right bundle, followed by subsequent activation of the left bundle supports

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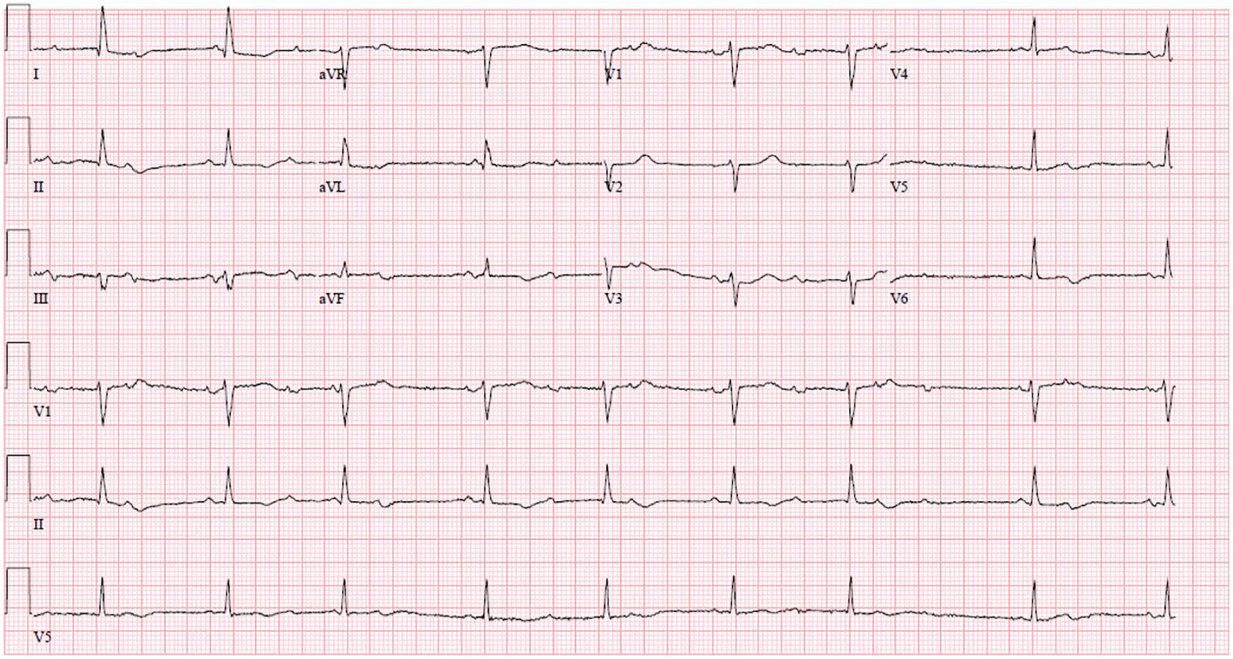

Fig. 1. 12-lead ECG following PCI.

Table 1
Pacemaker settings.

Manufacturer	Medtronic®
Model	Azure XT DR MRI W1DR01
Configuration	Dual chamber
Mode	DDDR
Lower rate limit	80 bpm
RA lead output	3.5 V
RV lead output	3.5 V
RA lead sensitivity	0.3 mV
RV lead sensitivity	0.9 mV
RA lead impedance	456 Ω
RV lead impedance	532 Ω

that the re-entry circuit is likely contained within the His-Purkinje system.

It can also be observed that the pacemaker senses the re-entry beat as ventricular activity and waits for the preset length of time before delivering a ventricular impulse in the absence of a spontaneous impulse. In this case, the pacemaker is set at a lower rate limit of 80 bpm. The pacemaker interrogation shows a cycle length of 740 ms (or 80 bpm) between every re-entry beat and the subsequent ventricular paced beat.

Conclusion

The His-Purkinje re-entry mechanism in humans was first described by Akhtar et al. in 1974 [1]. Since then, there have been many case

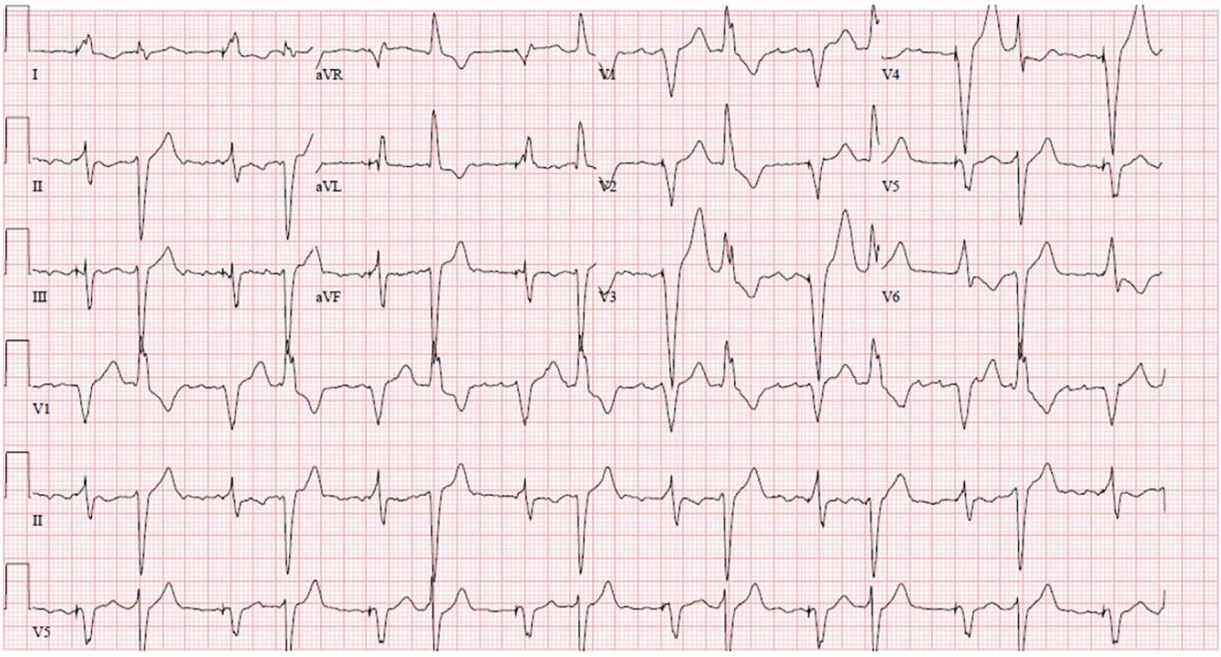

Fig. 2. 12-lead ECG following implantation of a dual chamber pacemaker.

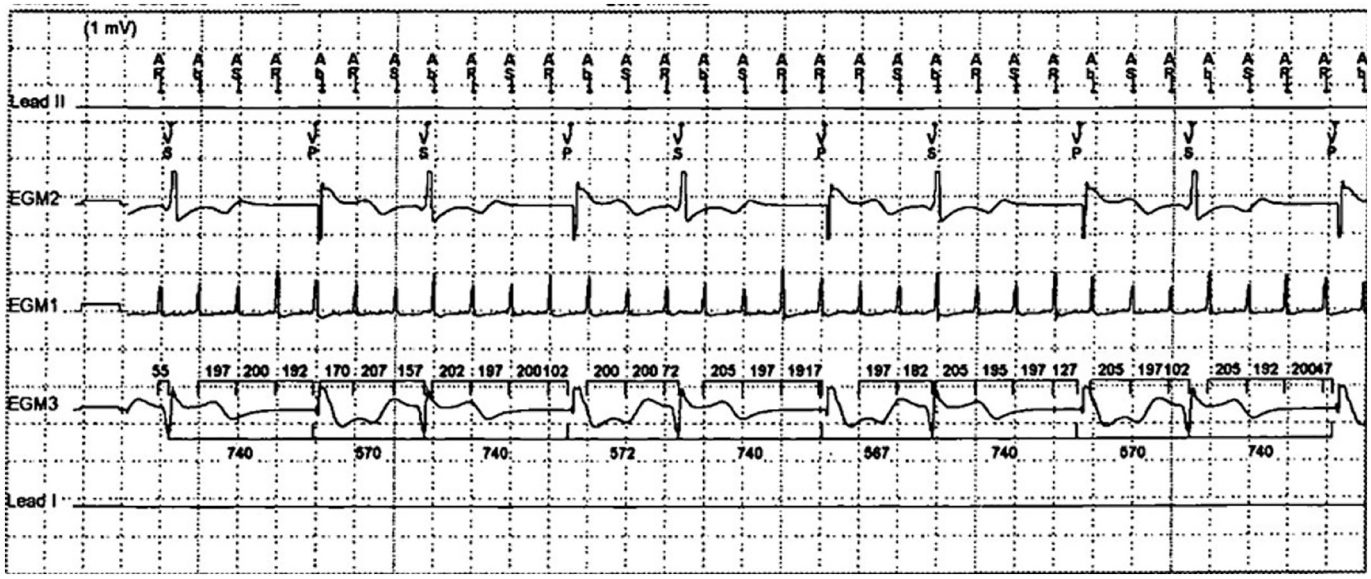

Fig. 3. Pacemaker interrogation. The cycle length between the ventricular paced (VP) beat and re-entry (VS) beat is constant at 570 ms, suggests that the re-entry beat is coupled to the VP beat. The cycle length between the reciprocal beat and VP beat is also constant at 740 ms, which corresponds to the programmed lower rate limit of 80 bpm.

reports and review articles describing a bundle branch re-entry circuit in the setting of ventricular tachycardia. This case, however, is interesting and unique because there is no ventricular tachycardia, and the re-entry circuit is due to the presence of a pacemaker. The mechanism cannot be proven without an EP study and His electrograms, and other possible mechanisms such as PVCs originating from infranodal scar tissue or the posteromedial papillary muscle are possible. However, the available information, that includes the bundle branch morphology of the re-entry beat seen on the surface ECG, the stable coupling interval between the paced beat and the re-entry beat seen on the interrogation, and the dissociation of A and V signals (also seen on the interrogation) all supports the presence of a bundle branch re-entry circuit.

References

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