Focus on ECGs: Case #12

Varying Morphology of QRS Complexes: A Possible Explanation

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A 62-year-old man with nonischemic cardiomyopathy (left ventricular [LV] ejection fraction, 0.30–0.34) presented for evaluation of weakness. In 2011, he had undergone placement of a D224TRK Consulta® CRT-D (Medtronic, Inc.; Minneapolis, Minn) biventricular implantable cardioverter-defibrillator. The pacemaker, programmed in DDD mode, had a lower rate limit of 60 beats/min (cycle length, 1,000 ms) and an upper limit of 130 beats/min (cycle length, 430 ms), a paced atrioventricular (AV)-delay period of 130 ms, and a sensed AV-delay period of 100 ms. The LV lead threshold was 1.25 V at 0.6 ms and was programmed to deliver 1.75 V at 0.6 ms. The right atrial and right ventricular leads were in standard positions. The electrocardiogram appeared as follows (Fig. 1).

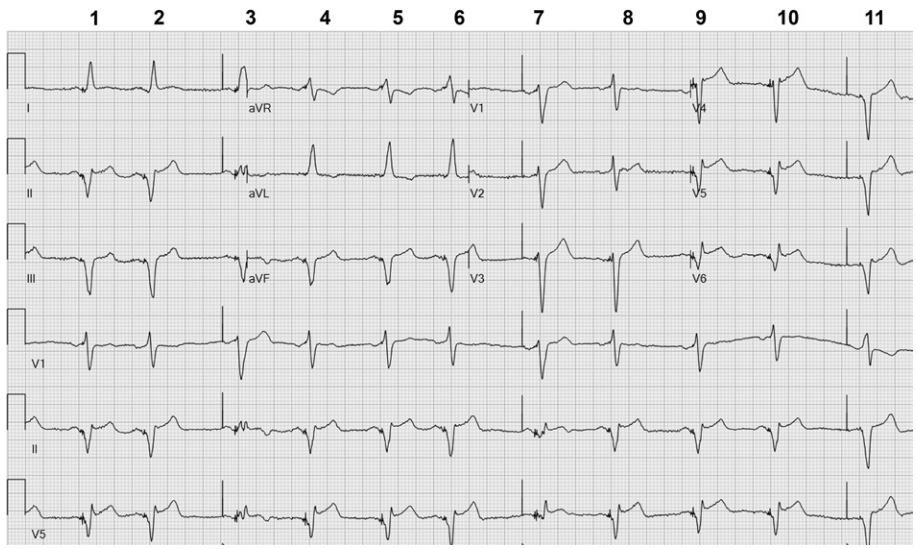

Fig. 1

What explains the different QRS morphology seen in complexes 3 and 7?

A) Intermittent programmed interventricular pacing delay
B) Decreased ventricular threshold for pacing because of intermittent lead malfunction
C) Unintended atrial lead mobility causing atrial capture at multiple sites and leading to variance in the AV interval
D) Atrial pacing causing a frequency-dependent alteration in the Purkinje fiber conduction velocity

See next page for the answer, as well as a link to the Focus on ECGs *blog, where you can participate in a moderated discussion.*

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Focus on ECGs: Answer #12

Answer

D) Atrial pacing causing a frequency-dependent alteration in the Purkinje fiber conduction velocity

The ECG shows ventricular pacing in complexes 1, 4, 5, 8, 9, and 10, which exhibit the same QRS morphology (Fig. 2).

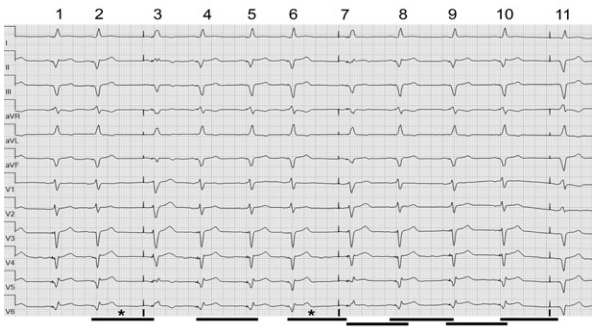

Fig. 2

Bolded bars = R-R invervals

However, complexes 3 and 7 have a different morphology. Complexes 2 and 6 result from ventricular pacing after a premature atrial complex (PAC). The R-R intervals after beats 2 and 6 are much longer than those in the other complexes. The sinus pause after each of these PACs (asterisks) triggers an atrial pacing complex in complexes 3 and 7. The additional time before the next ventricular pacing event enables full recovery of the intrinsic conduction system. The resulting fusion of intrinsic conduction and biventricular pacing produces QRS complexes 3 and 7, which are different from the baseline biventricular pacing alone. The decrease in the R-wave amplitude in lead V_1 in complexes 3 and 7 suggests less contribution from the LV pacing lead.

The dynamic refractoriness of the electrical conduction tissue of the heart is well described. Shorter cycle lengths cause decremental conduction in the AV node; in contrast, longer cycle lengths enable greater recovery of the intrinsic conduction system. Moreover, longer R-R intervals might prolong ventricular refractoriness downstream to the LV lead, reducing the amount of captured LV myocardium (for example, through latency or exit block).

Here we see an example of a rate effect on the conduction system: a short R-R interval followed by a long cycle length, more involvement of the intrinsic conduction system, fusion of conduction and pacing, and different QRS morphology. Longer programmed paced AV delay after paced atrial beats (vs sensed) may also facilitate intrinsic conduction and, thus, more fusion. The change in the R-R interval is due, in part, to the atrial pacing programming.[1] The atrial paced beat occurs after an interval of prolonged R-R, whereas baseline atrial tracking results in a faster rate (shorter R-R interval in complexes 1, 4, 5, 8, 9, and 10). On the other hand, complex 11 appears morphologically like the other atrial tracking beats, because it results from an atrial paced complex without a preceding PAC. The preceding R-R interval is similar to the baseline R-R intervals; therefore, the intrinsic conduction system has no additional time for recovery. The QRS-complex morphology thus varies, depending on the recovery time dictated by the preceding R-R interval.[1-3]

An alternative explanation is refractoriness of the conduction system caused by intermittent capturing of the right ventricular outflow tract by a local effect of the atrial pacing stimulus.[4] However, increasing the atrial pacing rate and amplitude did not mimic these changes. Of note, when this report was being prepared, the patient's original device had been replaced, and further testing opportunities were lost.

References

1. Francis J. ECG profile in atrial fibrillation. In: Chopra HK, Wander GS, Chandra P, Kumar V, editors. Atrial fibrillation update: a textbook of cardiology. London (UK): JP Medical Ltd.; 2017. p. 231-8.
2. Issa ZF, Miller JM, Zipes DP. Clinical arrhythmology and electrophysiology: a companion to Braunwald's heart disease. 2nd ed. Philadelphia: Elsevier Saunders; 2012. p. 194-211.
3. Lakusic N, Mahovic D, Slivnjak V. Ashman phenomenon: an often unrecognized entity in daily clinical practice. Acta Clin Croat 2010;49(1):99-100.
4. Pastori JD, Garro HA, Baranchuk A, Chiale PA. Intermittent capture of the right ventricular outflow tract by atrial pacing in a patient with a dual-chamber pacemaker. J Electrocardiol 2012;45(6):787-9.

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