SHORT COMMUNICATION

Grouped beating: to couple into trouble

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We discuss the 12-lead electrocardiogram and differential diagnosis of a regularly irregular narrow QRS-complex rhythm obtained from a patient with a history of atrial fibrillation who was admitted because of bradycardia-associated symptoms.

KEYWORDS
Grouped beating;
Atrial fibrillation;
Non-paroxysmal junctional tachycardia;
Wenckebach conduction;
Digitalis toxicity

Case presentation
A 73-year-old man with a history of non-ischaemic cardiomyopathy and persistent atrial fibrillation was referred for clinical work-up of new-onset symptomatic bradycardia. At the time of admission, the 12-lead electrocardiogram (ECG) showed a pattern of grouped beating with couplets and triplets (Figure 1). Which underlying condition should immediately be considered based on the electrocardiographic presentation? What is the differential diagnosis of this rhythm?

Commentary

Figure 1 shows a regularly irregular arrhythmia with grouped beating of narrow QRS complexes with an apparently isoelectric interval in-between. No P-waves can clearly be discerned. There is a concave depression or scooping of the ST-segment in leads V4–V6 and in the inferior leads. Unusually, flat T-waves and a low QRS-complex voltage are present ubiquitously. These ST-T-segment changes are typically seen during digitalis use, and the regularity of an irregular rhythm or grouped beating should immediately raise the suspicion of digitalis intoxication. Indeed, our patient was known to be on digoxin and he reported having accidentally taken his medication twice. Subsequent determination of the serum digoxin concentration revealed a level at the upper therapeutic range. Serum electrolyte concentrations were within normal limits.

The absence of discernable P-waves during the arrhythmia shown in Figure 1 could be caused by: (i) sinus arrest or severe sinus bradycardia, (ii) fine atrial fibrillation without manifest atrial fibrillatory activity, and (iii) retrograde atrial activation with P-waves buried within the QRS complex. In all instances, an atrioventricular (AV) nodal or junctional rhythm must be present, which is also true in the case of atrial fibrillation. The appearance of an irregular narrow QRS-complex rhythm that regularly repeats itself with exactly the same cycles (grouped beating) virtually excludes atrial fibrillatory activity being conducted to the ventricles. Grouped beating is an electrocardiographic hallmark of Wenckebach conduction. Assuming the presence of an AV junctional rhythm, 3:2 and 4:3 Wenckebach-type exit block out of the AV junction is the only explanation for the observed grouped beating with couplets and triplets. On the basis of the beating groups of two different, but fixed, RR intervals (880 and 520 ms), the cycle length of the AV junctional rhythm can be calculated to be around 470 ms. A digitalis-induced AV junctional tachycardia is called non-paroxysmal because of its gradual onset and is characterized by an entrance block into the AV junction and complete AV dissociation.1 Additional Wenckebach exit block from the AV junction to the ventricles is rare and usually occurs with atrial fibrillation.2,3 The proposed arrhythmia mechanism is further supported by the observation that, with discontinuation of digoxin, the rate of the AV junctional tachycardia decreased and allowed 1:1 conduction to the ventricles. This resulted in rhythm regularization and increased ventricular rate. These considerations make the possibility of retrograde atrial activation with P-waves completely hidden within the QRS complex highly improbable. However, in the absence of fibrillatory waves on the 12-lead ECG, a total sinoatrial block cannot be excluded.

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Another possible explanation for grouped beating of narrow QRS complexes without identifiable P-waves is based on the presence of dual AV nodal pathways and AV nodal re-entrant cycles with Wenckebach conduction to the ventricles. The two differential diagnoses include: (i) AV nodal re-entrant tachycardia and (ii) accelerated junctional rhythm with single or double AV nodal re-entrant cycles resetting focal activity. However, AV nodal re-entry is very unlikely to occur with digitalis toxicity, because digitalis early impairs AV nodal conduction by lengthening the AV nodal effective refractory period and leads to enhanced impulse formation in AV junctional pacemaker cells. Atrioventricular nodal conduction block accompanied by an accelerated AV junctional discharge is usually the consequence. Therefore, the most likely arrhythmia mechanism is a non-paroxysmal junctional tachycardia with complete entrance block into and Wenckebach exit block out of the AV junction and underlying atrial fibrillation (Figure 2).

Indeed, with this double tachycardia presenting as a coupled ventricular rhythm, the patient could really get into trouble. The present case illustrates the importance of a careful examination of the 12-lead ECG of an irregular supraventricular arrhythmia. Paradoxical regularization or grouped beating of the ventricular rhythm is a typical manifestation of digitalis toxicity in patients with atrial fibrillation.

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References