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## **Arrhythmia Graphics**

# Digitalis toxicity: ECG vignette



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### ABSTRACT

"Digitalis toxicity, often candidly indexed as poisoning, has plagued the medical profession for over 200 years. The situation qualifies as a professional disgrace on the basis of three items: the situation persists, physicians are often slow to recognize it and, over the decades, writers have been harsh in their denunciation of fellow physicians when toxicity has occurred..." These are the opening remarks of an essay published in 1983 on the 2nd centenary of William Withering's 'magic potion from foxglove's extract for dropsy.' Even today, after many decades, these words appear relevant! We present and discuss an interesting ECG of digitalis toxicity.

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A 30-year-old woman with dilated cardiomyopathy (LVEF 0.25) was admitted for recurrent vomiting for 3 days. She had been on digoxin and frusemide. The ECG at presentation (Fig. 1) is as shown. At atrial level no sinus P waves can be identified, suggesting sinus node suppression or sinoatrial block. The P waves are regular, narrow, and inverted in inferior leads. At ventricular level, it shows a bigeminal rhythm. One P wave is just before the first QRS of the bigeminal rhythm while the other is just after the second QRS complex. The first of the bigeminal QRS complexes is narrow, suggesting atrioventricular (AV) junctional origin. The second (coupled) QRS complexes of bigeminal rhythm are wider and show a RBBB-like morphology with left-axis deviation (with subtle axis changes). It could have its origin in the AV junction and

simultaneous aberration in right bundle and left anterior fascicle due to premature His activation, but more likely they arise in the left posterior fascicle. A ladder diagram depicting the electrophysiological phenomenon for the rhythm is as shown (Fig. 2).

The serum digoxin level was 2.5 ng/ml (normal: 0.8–2 ng/ml); the creatinine and potassium levels were normal. The ECG shows a few important features of digitalis intoxication: sinus node depression, AV junctional rhythm, and ventricular bigeminal rhythm arising in the fascicles of left bundle branch system. Narrow negative P waves suggest atrial activation starting centrally near the low interatrial septum. The short PR interval is due to a junctional rhythm, the retrograde conduction being more rapid than the

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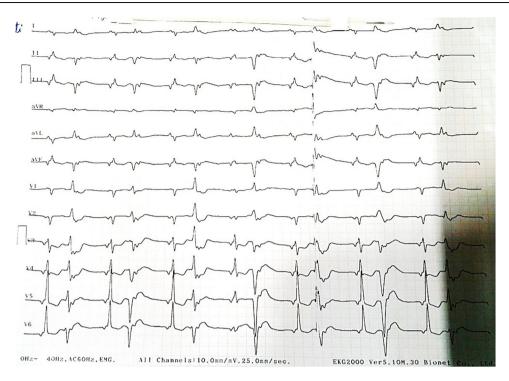


Fig. 1 - ECG at presentation.

conduction down to the ventricles. This suggests the junctional rhythm to be arising high in the AV node; it therefore has to encounter AV nodal delay, allowing the retrograde P to just precede the QRS. The coupled premature beats are relatively narrow, with a QRS configuration suggesting an origin in or close to the left posterior fascicle; the changing axis is due to different sites of impulse generation in this fascicle. The coupled premature beats make the His–Purkinje system and ventricles refractory to the next activation from the junctional rhythm. However,

the junctional rhythm does conduct retrogradely, explaining the regular P-P interval.

In such instances of changing site (competitive) of impulse formation in the bundle branch system during digitalis intoxication, the coupling interval stays about the same and the QRS is not very wide. The ectopics usually originate in the left bundle branch–Purkinje system. <sup>1,2</sup> The rhythm normalized over the next week after stopping digoxin (Fig. 3). Apart from sinus tachycardia, lead V1 shows left atrial abnormality and the limb leads show low voltage complexes, suggesting marked cardiac dilatation.

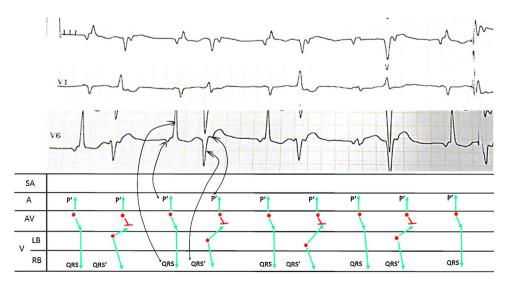


Fig. 2 - Ladder diagram for ECG mechanism (see text).

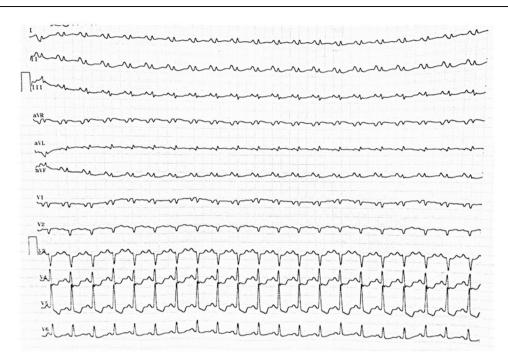


Fig. 3 - ECG after withholding digoxin for 1 week.

### **Conflicts of interest**

The authors have none to declare.

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