

Good Intentions

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About the Author

George Veenhuizen is an adult cardiac electrophysiologist at the Libin Cardiovascular Institute of Alberta in Calgary. He is interested in the diagnosis and management of all arrhythmias, particularly using catheter ablation.

A previously healthy 40-year-old man presented to his local emergency department complaining of 45 minutes of weakness and recurrent presyncope, particularly with any type of exertion. The nursing staff experienced difficulties in determining his pulse and blood pressure. A 12-lead EKG was recorded (Figure 1). *What is going on?*

Pre-excited atrial fibrillation, particularly with such a rapid ventricular response (at times over 300 bpm!) can precipitate ventricular fibrillation and sudden death in otherwise-healthy patients. This is a medical emergency, and therapies should be directed at the two components that contribute to the extremely rapid ventricular rate:

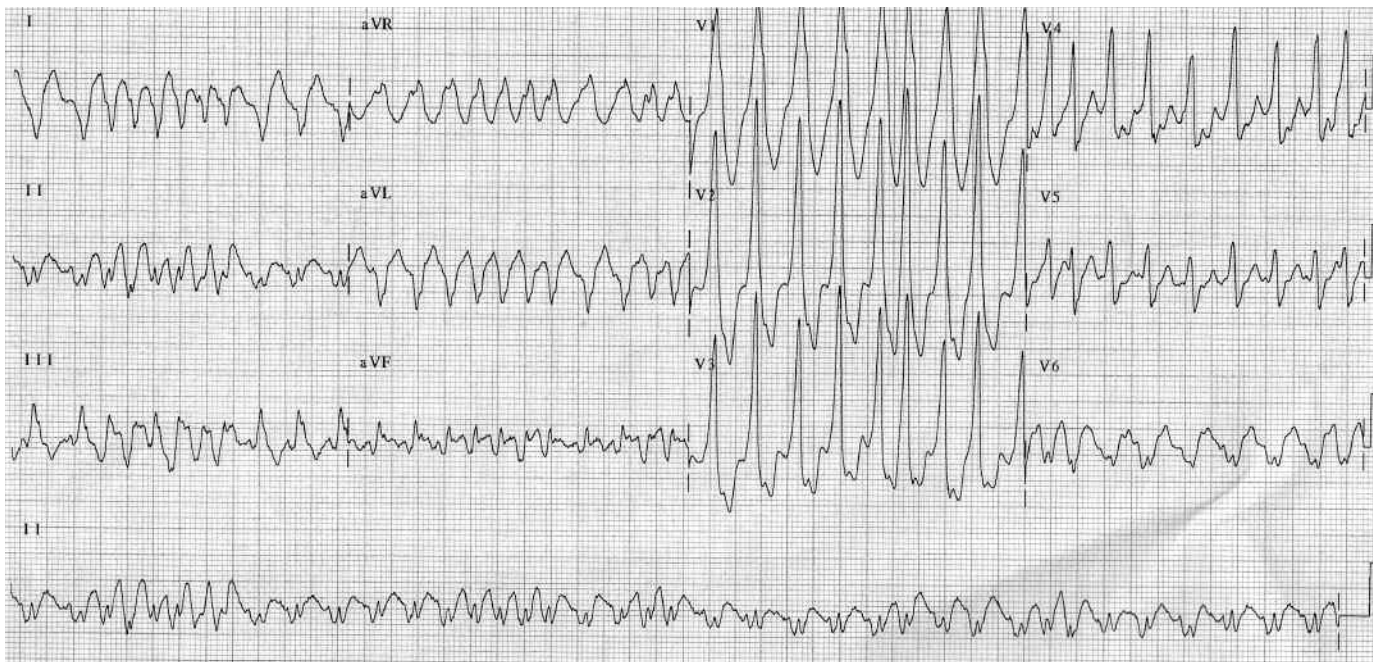


Figure 1

There is an irregularly irregular wide complex tachycardia with an average ventricular rate of 240 bpm (range 170–375 bpm). The former should prompt consideration of atrial fibrillation (AF) as the underlying rhythm. The extremely rapid ventricular response should prompt consideration of conduction over an accessory atrioventricular pathway (AP). The QRS complexes are slurred and bizarre with subtle beat-to-beat variation in their morphology and duration. This is consistent with ventricular activation predominantly over an AP, but variably fusing with conduction over the normal AV conduction system. Importantly, this patient's ventricular response to AF is determined by the electrophysiological properties of his AP, not those of his normal conduction system.

(1) the atrial fibrillation itself and (2) the very short refractory period of the AP. While the patient is being sedated and prepared for electrical cardioversion, it is logical to concomitantly (and quickly) administer an IV antiarrhythmic drug that will prolong the refractory period of the AP and slow the ventricular response (ibutilide, procainamide). This may improve hemodynamics, which may, in turn, facilitate the administration of adequate doses of sedation. Furthermore, the drug could result in pharmacological cardioversion and/or help to prevent early recurrences of AF.

Electrical cardioversion was performed (Figure 2A), paradoxically resulting in the development of ventricular fibrillation (VF). *Why did the swift application of the therapy of choice result in the very outcome that was meant to be prevented?*

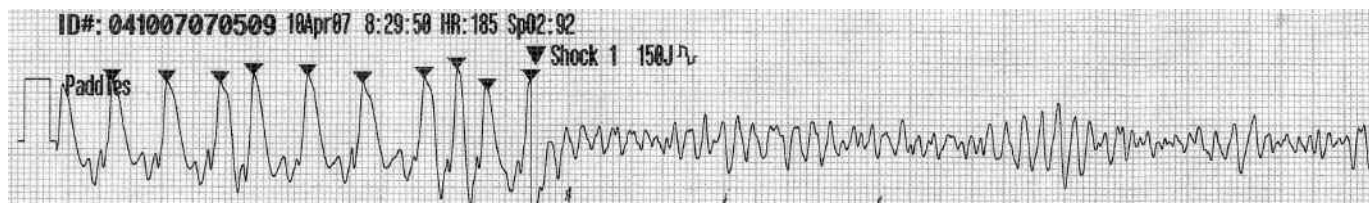


Figure 2A

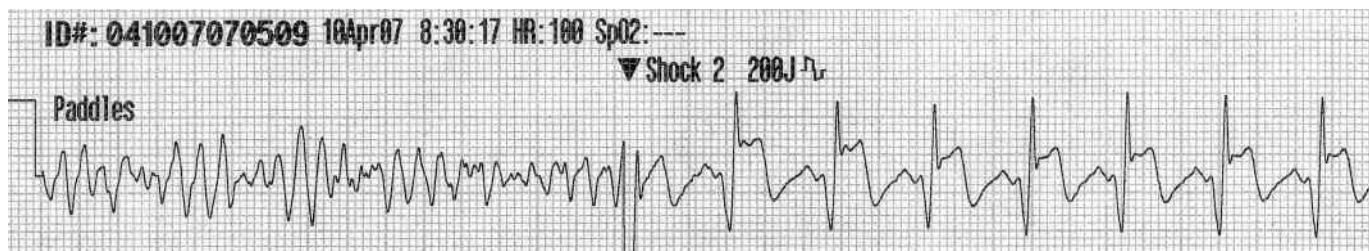


Figure 2B

Figure 2A shows the electrocardiographic recording made from the paddles positioned at the base and apex. This configuration corresponds to a lead-II-like recording; indeed, the tracing prior to the shock resembles the one recorded by lead II on the 12-lead EKG. Note the small black triangles, which represent the synchronization of the defibrillator in the cardiac cycle. A defibrillator should synchronize on the QRS complex. On this particular recording, the T wave happens to be larger in amplitude and more positive than the pre-excited QRS complex; the defibrillator is actually synchronizing on the T wave. (If you are unconvinced, compare the timing of the

QRS in lead I of the 12-lead EKG to the waveforms in lead II of the EKG.) It should be no surprise, then, that the shock, delivered directly on the T wave, resulted in VF. At this point, the patient was having a cardiac arrest, and the appropriate treatment was unsynchronized defibrillation, which promptly restored sinus rhythm (Figure 2B). A 12-lead EKG recorded after sinus rhythm was restored is shown in Figure 3, where clear evidence of ventricular pre-excitation was present (PR interval 110 ms, delta wave). This patient underwent successful catheter ablation of a left free wall AP, and his EKG returned to normal.

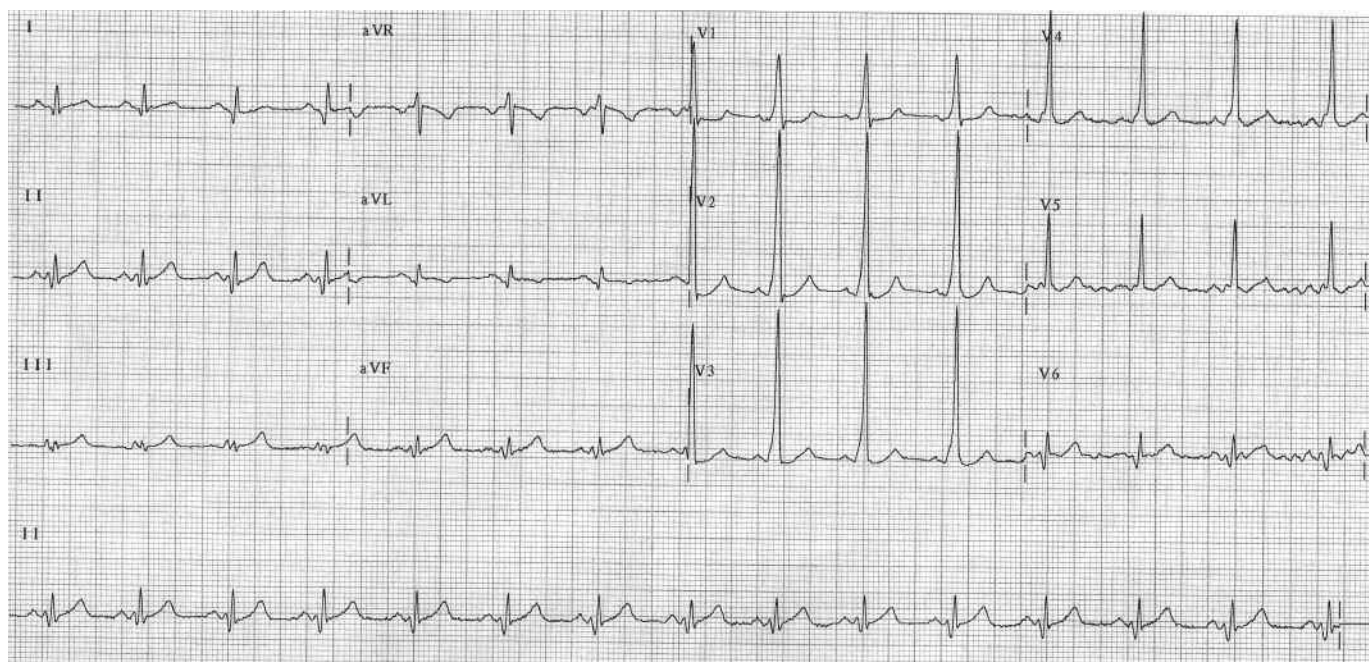


Figure 3