T-WAVE ALTERNANS IN A PATIENT WITH LEFT VENTRICULAR DYSFUNCTION

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Case Presentation
A 78 year-old man with a history of type 2 diabetes mellitus, hypertension, hyperlipidemia, seizure disorder, dementia, and alcohol abuse was admitted to the Wilmington VA Medical Center for treatment of deep vein thrombosis of the lower extremity. During the hospital course, the patient had a brief episode of supraventricular tachycardia. An echocardiogram revealed dilated cardiomyopathy, an ejection fraction of 20%, and apical and inferolateral akinesis-hypokinesis. On the following day, the patient developed cardiac arrest with ventricular fibrillation (VF) that was terminated by electric defibrillation. An electrocardiogram (Figure 1) obtained immediately after defibrillation demonstrated sinus bradycardia, a prolonged QT interval, and T-wave alternans. After the cardiac arrest, the patient was ventilator-dependent, and his family decided to withdraw life-supporting care.

Since the early 1900s, T-wave alternans (TWA), or repolarization alternans, has been linked to ventricular arrhythmias. TWA refers to the alternation of T-wave shape or timing from beat-to-beat on the electrocardiogram (ECG). TWA may also involve the ST segment or U wave but ultimately reflects T-wave changes. Note that this is still in stark contrast to electrical alternans totalis, which refers to an alternating axis or voltage of all ECG components.

Although the exact pathophysiology of TWA is unknown, it is most likely caused by a temporal heterogeneity in ventricular repolarization. This dispersion in ventricular repolarization is an important mechanism underlying ventricular arrhythmias. In essence, TWA represents an electrical state of heightened arrhythmogenicity and thus possibly a gateway to VF.

References

Figure 1. The post-cardiac arrest electrocardiogram demonstrates sinus bradycardia at a heart rate of ~40 beats per minute and a prolonged QT interval. Note the alternating T-wave morphologies most easily appreciated in leads V2-V5.