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Ineffective ICD Therapy Due to Excessive Alcohol and Exercise

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PAPAIOANNOU, G.I., ET AL.: Ineffective ICD Therapy Due to Excessive Alcohol and Exercise. *A 49-year-old man with dilated cardiomyopathy and an ICD received eight unsuccessful shocks for a single episode of ventricular tachycardia after excessive alcohol intake and exercise. The patient had low defibrillation thresholds just 8 days prior to his event during a routine 6-week postimplant electrophysiological study. Defibrillation threshold testing after his incident confirmed the low energy requirement for ventricular tachycardia termination. A previous animal study reported that intravenous ethanol elevates the defibrillation energy in a dose dependent manner. However, a recent study in humans showed that modest alcohol intake had no effect on defibrillator thresholds. This case report supports the animal data and suggests that excessive alcohol consumption can cause a life-threatening situation in ICD patients. (PACE 2002; 25:1144–1145)*

ethanol, implantable cardioverter defibrillator, defibrillation threshold

Case report

A 49-year-old man was transferred to Hartford Hospital after he had received multiple implantable cardioverter defibrillator (ICD) shocks. Two months earlier he had been evaluated for symptomatic ventricular tachycardia (VT). The work-up at that time revealed a dilated cardiomyopathy with an ejection fraction of 0.25, possibly related to a viral infection, even though alcohol consumption may have been contributing to his left ventricular dysfunction. An electrophysiological study failed to induce sustained VT. The patient had a Guidant Prizm DR (St. Paul, MN, USA) ICD implanted and was discharged on Sotalol (160 mg twice a day). Predischarge defibrillation threshold (DFT) was 11 J. At 6-week postimplant, a routine noninvasive ICD electrophysiological study confirmed a DFT of 11 J.

Eight days after his electrophysiological study, the patient consumed eight glasses of whiskey (16 oz) over a period of 3 hours. He returned to his room and started to exercise. While exercising, he recalled receiving two shocks, but did not remember details of the event prior to calling the paramedics. His vital signs were stable on admission in a local emergency department. A monitor demonstrated nonsustained VT. The patient was compliant to his medical regimen including his sotalol. An electrocardiograph (ECG) was unchanged from a previous one and the QTc interval was 420 ms. Interrogation of his ICD revealed that he had received eight defibrillator shocks for a single episode of VT with therapy exhausted after 4 minutes and 23 seconds. The episode ended spontaneously after 7 minutes and 30 seconds (Fig. 1).

Chemistries, including magnesium level and cardiac enzymes, were normal. An ethanol level obtained 2.5 hours after his admission was 26 mg/dL. Ethanol undergoes first pass metabolism and follows a linear curve with a half-time of approximately 30 minutes. Based on that model, the patient's blood ethanol level at the time of the ICD shocks was at least 250 mg/dL. While the legal limit for intoxication varies by state (80–100 mg/dL), most people become intoxicated at levels between 100 and 200 mg/dL.[1]

Subsequently, the patient was started on metoprolol and mexiletine, and sotalol was discontinued. He completed 12 minutes of exercise stress testing (maximal heart rate of 150 beats/min) without chest pain or electrocardiographic changes. Only up to 5 beats of nonsustained VT was observed. Noninvasive ICD electrophysiological study revealed stable lead parameters and a DFT at $\leq$ 6 J. The patient was discharged and admonished to refrain from drinking alcohol.

Discussion

This report demonstrates a potential relationship between ethanol and defibrillation energy requirements in a patient with an ICD. Routine DFT testing just 8 days prior to hospital admission confirmed a DFT at 11 J. Although the patient had an elevated ethanol level, the authors can not ex-

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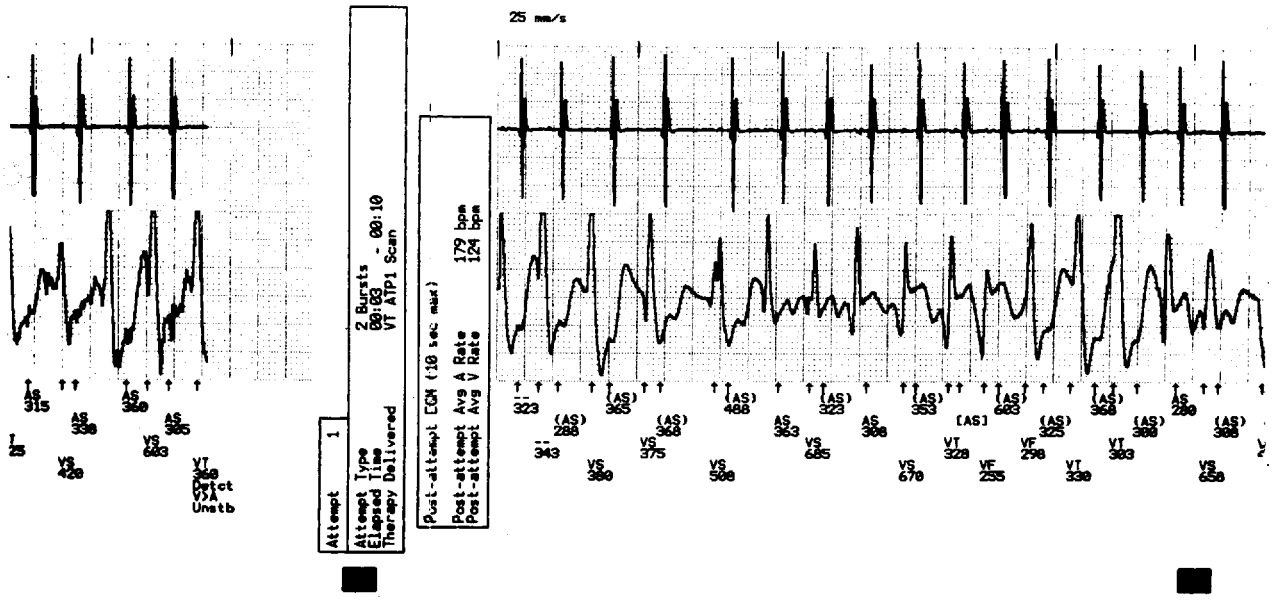

Figure 1. *Implantable cardioverter defibrillator interrogation with top tracing recording intraatrial activity and bottom tracing recording morphology of tachycardia. Initial tracing revealed polymorphic ventricular tachycardia. Antitachycardia pacing (ATP) and a first shock of 21 J fail to convert ventricular tachycardia to sinus rhythm. Polymorphic ventricular tachycardia persists despite a maximal shock of 31 J. Therapy exhausted after 4 minutes and 23 seconds. The whole episode ended after 7 minutes and 30 seconds with spontaneous recovery to sinus rhythm.*

clude the effect of exercise (through the release of catecholamines) as an explanation for failure of ICD therapy. In humans, physiological increases in the plasma epinephrine may increase the number and energy shocks needed to terminate ventricular fibrillation.[2]

Acute ethanol intoxication may indirectly stimulate the release of catecholamines and also causes an increase in duration of atrial and ventricular signal-averaged electrocardiograms.[3] It is possible that this prolongation and a direct effect of ethanol on the heart may contribute to cardiac dysrhythmias. Other mechanisms like ethanol related electrolyte changes might be additionally involved. In rats treated with ethanol there was a high rate of isoproterenol induced ventricular fibrillation.[4]

In animal studies intravenous ethanol elevates the defibrillation energy requirement in a dose related manner. Using a porcine model Kim et al.,[5] found that moderate ethanol concentrations (100 and 275 mg/dL) had no effect on DFT with the higher alcohol level group approaching statistical significance. An alcohol concentration of 400 mg/dL was associated with a significant increase in DFT by 32%. Strickberger et al.[6] evaluated the effect of intravenous ethanol infusion in humans with a recently implanted ICD. Since a blood ethanol level of 95 ± 44 mg/dL had no effect on DFTs, the authors concluded that modest consumption of alcohol would be safe in patients with an ICD. However, the investigators did not address the effect of higher levels of alcohol on DFTs. The current case supports the animal data and demonstrates that excessive alcohol intake in humans may render ICD therapy ineffective in patients with dilated cardiomyopathy and life-threatening ventricular arrhythmias.

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