S Mahmoud, LM Beauchesne, DR Davis, C Glover. Acute reversible left ventricular dysfunction secondary to alcohol. Can J Cardiol 2007;23(6):475-477.

To the Editor:

It is unfortunate that the electrocardiogram (ECG) revealing a 'nonspecific T wave abnormality' was not recorded in the paper by Mahmoud and colleagues. It is unfortunate because these observers' research has found the ECG invaluable in the differential causations of not only congestive heart failure per se, but also of dilated (congestive) cardiomyopathy. In chronic alcoholism, if a cirrhotic pathology coexists with heart muscle disease, the resultant hypomagnesemia, in itself encouraging hypokalemia, predisposes the patient to T wave alterations on ECG (1). When depleted, the metalloenzyme magnesium – the energy source for the sodium-potassium pump – produces T wave changes due to impairment of sodium-potassium-ATPase, resulting in a loss of intracellular potassium. The changes are accentuated by additive hypocalcemia if pancreatic exocrine dysfunction is concomitant.

Four specific repolarization mutations are worthy of description. First, Figure 1A illustrates the low-amplitude ('molehill') T wave of magnesium deficiency, in conjunction with the prolonged ST segment peculiar to hypocalcemia. Second, a diminutive low-voltage negative ('dimple') T wave is graphed when hypokalemia is predominantly operative (Figure 1B). Third, when the hypokalemic U wave exceeds the amplitude of the T wave (double-humped or axed) and the QRS complex widens diffusely, the resultant configuration is aptly termed 'cloven' (Figure 1C). Finally, what these authors have termed the somewhat bizarre 'antler' T wave (Figure 1D). Here, tall, narrow hypokalemic U waves coalesce with the peaked T waves of early magnesium deficiency to resemble the horn of a young deer.

In summary, given that magnesium and potassium deficits are fairly common in ethanol abuse, the specific ECG expressions of the same are valuable signposts that lead to the diagnosis, and ultimately therapeutics, of alcoholic heart muscle disease.

John Morphet MD
Department of Medicine
McMaster University
Hamilton, Ontario

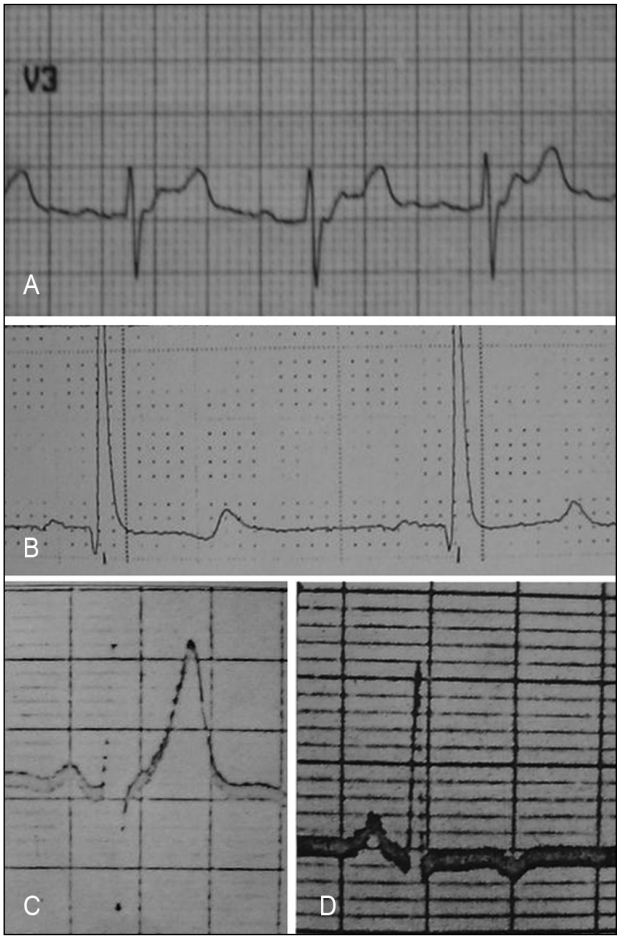

Figure 1) A *'Molehill' T wave*. **B** *'Dimple' T wave*. **C** *'Cloven' T wave*. **D** *'Antler' T wave*

REFERENCE

1. Seelig MS. Electrographic patterns of magnesium depletion appearing in alcoholic heart disease. Ann NY Acad Sci 1969;162:906-17.

From the Authors:

Thank you for your letter and your insights into ECG changes with chronic congestive cardiomyopathy complicated by electrolyte imbalances. In this particular case, the patient was not suffering from chronic dilated alcoholic cardiomyopathy, but had an acute nondilated cardiomyopathy that rapidly reversed with the cessation of alcohol intake. In addition, the patient presented with symptoms of alcohol withdrawal and only developed congestive heart failure symptoms when she was vigorously volume-resuscitated. The patient did indeed have hypokalemia (3.4 mmol/L), hypocalcemia (2.15 mmol/L) and hypomagnesemia (0.45 mmol/L) on presentation. We have included her presentation ECG (Figure 2), which does not illustrate classic features of electrolyte imbalances, but shows sinus tachycardia and artifact due to alcohol withdrawal.

Saad Mahmoud MD FRCPC
Luc M Beauchesne MD FRCPC
Darryl R Davis MD FRCPC
Christopher Glover MD FRCPC
University of Ottawa Heart Institute
Ottawa, Ontario

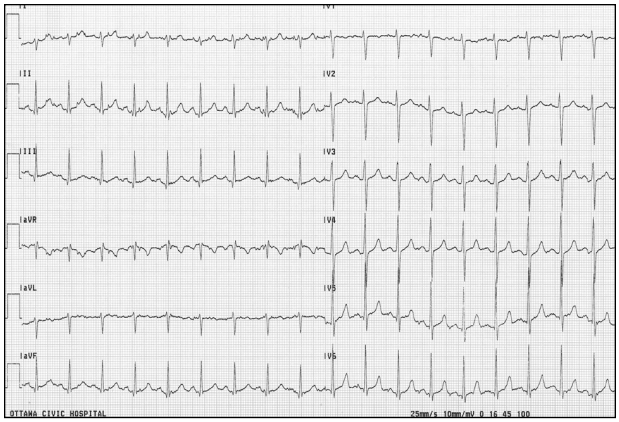

Figure 2) *Patient's electrocardiogram on presentation, showing sinus tachycardia and artifact due to alcohol withdrawal*