Erratum: This Letter to the Editor was published with an incorrect figure in the January 2008 issue of The Canadian Journal of Cardiology. The corrected figure is below. Pulsus Group extends sincere apologies to the author.

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

Ethanol and the ECG

It is unfortunate that the electrocardiogram (ECG) revealing a 'non-specific T wave abnormality' was not recorded in the paper by Mahmoud and colleagues. It is unfortunate because this observer's 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 metallocoenzyme 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, 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 1A). Second, Figure 1B illustrates the low-amplitude ('molehill') T wave of magnesium deficiency, in conjunction with the prolonged ST segment peculiar to hypocalcemia. Third, what this author has termed the somewhat bizarre 'antler' T wave (Figure 1C). Here, tall, narrow hypokalemic U waves coalesce with the peaked T waves of early magnesium deficiency to resemble the horn of a young deer. Finally, a diminutive low-voltage negative ('dimple') T wave is graphed when hypokalemia is predominantly operative (Figure 1D).

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.

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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.

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

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