Tall T waves in the precordial leads may manifest in many and varied conditions. Hyperkalemia is one of the most common. This presentation reflects two cases of severe hypokalemia which manifested with unusually tall and peaked precordial U waves. The presentation in one of the cases closely simulated the tall T-waves of hyperkalemia.

**Case Reports**

**Case 1**

A 38-year old black man with a history of marked alcohol abuse presented with complaints of epigastric pain, lower substernal pain, and generalized weakness and vomiting for a few days before admission. The patient had been drinking heavily until two days before admission when he stopped because of vomiting. He had a history of hypertension and was being treated with hydrochlorothiazide. Physical examination revealed vague epigastric tenderness and a mild tremor of the extremities. There was no evidence of hypotension or muscle weakness. Laboratory tests on admission to the hospital revealed Na 110 mmol/L, K 1.6 mmol/L, Cl 63 mmol/L, CO₂ 38 mmol/L; BUN 9 mmol/L; creatinine 1.1 μmol/L; glucose 9.6 mmol/L; Ca 2.2 mmol/L; magnesium 0.9 mmol/L; albumin 37 g/L. Arterial blood gas analysis showed: pH 7.72; PaO₂ 63 mm Hg; O₂ Sat percent 96.4; Pco₂ 28 mm Hg; HCO₃ 35.1 mmol/L. Cardiac enzymes levels: creatinine phosphokinase 1.06 IU; CK MB fraction 0.1 percent; SGOT 243 IU; LDH 316 IU.

His admission electrocardiogram (Fig 1) revealed sinus rhythm with a frontal plane QRS axis of +90°. Tall U waves, which looked like tall and symmetrical T waves, are evident in the limb and precordial leads. The T waves are actually buried in the tall U waves and the two wave forms cannot be separated from each other. Unlike the usual form of U wave, these are sharply pointed and symmetrical with a relatively blunt apex. There are nonspecific ST-T segment changes. The apparent Q-T interval, in effect a Q-U interval, is markedly prolonged; the apparent Q-Tc being 0.64 sec. There is also muscle tremor artifact in some of the limb leads.

The patient was treated with intravenous fluids, initially with 3 percent saline and then normal saline solutions. He also received more than 240 mmol of potassium chloride over a period of 24 hours. His serum electrolytes were monitored frequently. The patient was also closely monitored for any arrhythmia.

Laboratory investigations on the following day revealed the following: Na 130 mmol/L; K 3.5 mmol/L; Cl 93 mmol/L; total CO₂ content 36 mmol/L; BUN 7 mmol/L; creatinine 15 umol/L; glucose 7.3 mmol/L; calcium 1.93 mmol/L; magnesium 0.99 mmol/L; albumin 29 g/L; pH 7.48; PaO₂ 64 mm Hg; Pco₂ 40 mm Hg; HCO₃ 29.5 mmol/L. The electrocardiogram recorded on day 2 (Fig 2) revealed normal sinus rhythm with a mean frontal plane QRS axis of +90°. The U waves have become markedly diminished in ampli-

**FIGURE 1.** Case 1, electrocardiogram recorded on admission.
tude, especially in leads V1 and V6. The U and T waves are now evident as two distinct waves and can be clearly separated (for example, standard lead 1, lead V1 and lead V6 of Fig 2). This contrasts with the electrocardiogram recorded on admission where the T waves are actually buried and hidden within the U waves. The electrocardiogram recorded on the third day was normal (not illustrated). The giant U waves had disappeared completely.

CASE 2

A 65-year-old man was admitted to the hospital with acute alcoholic intoxication. While inebriated he had taken an unspecified number of verapamil tablets. He had chronic obstructive airways disease and was receiving the verapamil as well as a thiazide diuretic for treatment of systemic hypertension. His blood pressure was 120/65 mm Hg and his pulse rate 60 per minute. The heart was clinically normal. The chest had diffuse rhonchi. There was a 4 cm tender hepatomegaly.

Laboratory investigations showed Na 145 mmol/L; K 2.3 mmol/L; Cl 105 mmol/L; BUN 2.9 mmol/L; creatinine 170 µmol/L; glucose 5.4 mmol/L; calcium 2.36 mmol/L. The electrocardiogram recorded on admission shows low T waves associated with tall, peaked and widened U waves (Fig 3). These are particularly well seen in leads V1 to V6. The U waves can be clearly separated from, and are taller than, the T waves.

He was treated with potassium salts. The electrocardiogram recorded on the second day when his serum potassium level was 2.9 mmol/L is shown in Figure 4. The U waves have diminished in amplitude but still tend to be rather peaked and wide. The electrocardiogram recorded on the fourth day when the serum potassium was 3.6 mmol/L (not illustrated), was normal. The T waves had regained their amplitude, and the U waves were normal and of low amplitude.

DISCUSSION

The first case had severe hypokalemia, severe hyponatremia with hypochloremic metabolic alkalosis. In addition, he had the early effects of alcohol withdrawal. Myocardial infarction was ruled out by the absence of the characteristic clinical and electrocardiographic presentation and a normal CK MB fraction level. Total CK elevation was due to rhabdomyolysis and possibly to alcohol withdrawal. Large and peaked U waves are also clearly evident in Case 2 where they can nevertheless still be separated from the T waves.

The factors common to both cases were hypokalemia and acute alcoholism. The mechanism which results in the peaking and marked increase in amplitude of the U waves is not known. This particular U wave manifestation is uncommon even with hypokalemia.

There appears to be only one case report in the literature similar to the cases presented here. In this report, we could not measure the blood pH nor the serum magnesium level due to a lack of facilities.

It is well known that hypomagnesemia may, at times,
cause tall T waves. In Case 1, however, the serum calcium and serum magnesium levels were within normal limits. Indeed, the admitting physician for Case 1 did not suspect hypokalemia from the electrocardiogram recorded on admission, and actually entertained a diagnosis of hyperkalemia. Nevertheless, hypokalemia should have been suspected in this case even from the electrocardiogram recorded on admission, by evaluation of the Q-T interval. The apparent Q-T interval—the actual Q-U interval—was markedly prolonged. This is not seen with hyperkalemia, where the Q-T interval is either normal or actually decreased. Thus, the manifestation of what appears to be tall T waves in association with an apparent markedly prolonged Q-T interval is a pointer to the presence of tall waves rather than tall T waves. Moreover, the T waves of hyperkalemia have a narrow base, whereas the giant U waves of hypokalemia have a broad base. These U waves are not unlike the appearance of a broad-based mountain. Himalayan U waves would therefore appear to be an appropriate description.

Note that the Q-T interval may rarely be prolonged in hyperkalemia, if it is associated with hypocalcemia, a situation which occurs with chronic renal failure. This is due to prolongation of the S-T segment from the hypocalcemia. The S-T segment in our patient was normal in duration.

It was Lepeschkin who first drew attention to such U waves when he stated that, "In pronounced hypopotassemia, the U waves may become tall and pointed so that they may be mistaken for T waves . . . " The paucity of reports since then clearly underline the necessity for its re-emphasis. Nevertheless, the manifestation of such tall and pointed U waves especially when the T waves are not clearly visible, may well mislead and discourage the clinician from the administration of potassium salts when he is unaware of the significance of an apparently prolonged Q-T interval.

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