strips of tape running below the nose, around the tube, and around the neck below the ear, will prevent dislodgement and allow visualization of all areas of contact with the nose. The etiology of the nasal necrosis in Case 2 is unclear, in that the patient underwent an uncomplicated, elective surgical procedure, following which no unusual angulation or pressure on the nose was observed. In both cases, disposable polyvinylchloride tubes were used eliminating the possibility of ethylene oxide or other sterilization byproducts as possible toxins. Nonetheless, careful inspection of the tube’s contact with the nose at frequent intervals might have allowed earlier detection and the prevention of frank necrosis.

REFERENCES
1 Markham WG, Blackwood MJA, Conn AW: Prolonged nasotracheal intubation in infants and children, Canad Anesth Soc J 14:11, 1967

U Wave Alternans and Increased Ventricular Irritability*

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Electrical alternation of a single electrocardiographic wave is rare and has not been adequately explained. We describe a case of U wave alternans associated with extreme ventricular irritability. Relationship to hypomagnesemia is indicated by rapid response to magnesium therapy. Mechanisms that may be responsible for this phenomenon are discussed.

Electrical alternation is rare and consists usually of total alternation of the various waves that compose the electrocardiographic cycle. Alternation of the amplitude, polarity or vectorial direction of a single wave is a far less common finding for which no well-defined mechanism has been established. The finding of U wave alternans is an exceptionally rare phenomenon. Its clinical implications and electrophysiologic determinants remain obscure. Our patient constitutes the second reported case in the English literature of U wave alternans. Its etiologic relationship to hypomagnesemia and hypocalcemia and its association with increased ventricular irritability are discussed.

CASE REPORT

A 36-year-old woman presented to the emergency room complaining of numbness around the mouth and demonstrated carpal spasm. A few minutes after her admission, ventricular fibrillation appeared and was successfully halted by DC countershock and IV administration of lidocaine. During the ensuing three to four hours, frequent premature ventricular contractions and episodes of ventricular tachycardia were observed and treated with continuous infusion of lidocaine 2-3 mg/min. History disclosed an average daily alcoholic intake of a half pint for more than 12 years. Medications prior to this admission included folie acid 5 mg bid, one multivitamin capsule daily, vitamin C 500 mg tid, and diazepam (Valium) 5 mg bid. Physical examination after termination of ventricular fibrillation disclosed a somnolent woman. Her blood pressure was 120/90 mm Hg, respiration 16/min and pulse 86/min. Lungs were clear. The apical impulse was felt in the fifth left intercostal space at the midclavicular line. Occasional premature beats were present. No murrmor or gallop was heard.

Pertinent Laboratory Data: Hematocrit 34 percent, white blood cell count 6900 per cu mm, BUN 5 mg percent, sodium 141 mEq/L, blood sugar 102 mg percent, magnesium 1.3 mEq/L (normal 1.8 to 2.5), calcium 7.5 mg percent, phosphorus 4.3 mg percent, uric acid 8.9 mg percent, albumin 2.4 gm percent.

Chest x-ray showed clear lung fields and normal heart size. ECG on admission July 21, 1972 showed normal sinus rhythm and prominent U waves. Three hours after admission U wave alternans was recorded (Fig 1) and the frequency of ventricular tachycardia increased despite administration of lidocaine IV (Fig 2). Disappearance of the U wave alternans with gradual decrease of the magnitude of the U wave followed the institution of magnesium therapy (Fig 3), (4 gm of MgSO4 in 1000 ml of dextrose 5 percent in water over 24 hours, followed by oral maintenance dose of magnesium oxide, 200 mg bid).

DISCUSSION

Electrical alternans is an uncommon electrocardiographic finding, and when present, is caused by changes in the depolarization process and accompanied by secondary alterations in repolarization. This form of electrical alternans usually seen in cases of pericardial effusion may reflect the pendular and rhythmic motion of the heart contained in a limiting pericardial sac distended by pericardial fluid. Primary alternation of repolarization waves is rarely encountered and its mechanism is less understood. Studies by Kleinfeld et al have suggested that alternation in the rate and extent of calcium and potassium transport across the myocardial cell membrane was responsible for T wave alternans. Ricketts et al have reported T wave alternans in a patient with alcoholism, hypomagnesemia, and cardiomyopathy. Kimura and Yoshida and Dolara and Pozzi reported T wave alternans in individuals with docu-
FIGURE 1. Recording of leads 1, 2, and 3 of the electrocardiogram revealing alternans of the U wave, a T wave of low amplitude proceeds the U wave lead 3. Paper speed—25 mm/sec.

FIGURE 2. Representative ECG strip of lead 2. Ventricular premature beats and runs of ventricular tachycardia commence after the peak of the giant U waves following a well defined T wave of small amplitude as seen in the complex marked (X). Note that repetitive ventricular complexes follow an ectopic beat occurring at a short coupling interval suggesting a relationship between the "U" wave and "T" wave as to the vulnerable period.

FIGURE 3. Two complete electrocardiograms, one taken shortly after admission and the second on the day of discharge. The first displays giant U waves clearly distinct from the T waves, the ECG is otherwise normal. The second ECG reveals the amplitude of the U wave to have returned to normal following treatment.
mented hypocalcemia and hypopotassemia. These reports suggest, in a clinical setting, the electrophysiologic mechanisms described by Kleinfeld. It is possible that the large repolarization wave appearing in an alternating manner in some of these cases has resulted from fusion of U and T waves. These two waves represent the repolarization process and the latter is currently considered a reflection of Purkinje system repolarization or of a repolarization after potential. The increased ventricular irritability observed in our patient is similar to that reported by Mullican and Fisch in a subject with post extrasystolic alternation of the U wave and hypokalemia. These findings are in agreement with the concepts presented by Lepeschkin et al suggesting that alternation may lead to a circus movement that may provoke ventricular fibrillation.

Another factor which could account for ventricular irritability is the temporal dispersion of repolarization, better recognized in the syndromes of prolonged QT, leading to instability during repolarization.

The role played by hypocalcemia in our patient remains speculative, but since the principal effect of hypocalcemia is manifested by ST segment prolongation rather than T or U wave changes, there is reason to believe that hypomagnesemia is the principal cause of large U waves, as well as U wave alternans in our patient.

In conclusion, increased amplitude of the U wave and U wave alternans may be manifestations of hypomagnesemia with possible aggravating effect of concomitant hypocalcemia. Since hypomagnesemia is commonly encountered in alcoholic subjects, it may be responsible for instances of sudden death in these patients by inducing ventricular arrhythmias. Large U waves may be a clue to increased ventricular excitability and impending serious dysrhythmia.

REFERENCES
2 Kimura E, Yoshida K: A case showing electrical alternans of the U wave without change in the QRS complex. Am Heart J 65:301, 1963
9 Lepeschkin E: Genesis of the U wave. Circulation 15:77, 1957
10 Lepeschkin E: Electrocardiographic observation on the mechanism of the electrical alternans of the heart. Cardiologia 16:278, 1950