Transient Large Upright T-Wave on the Electrocardiogram during Multiple Monitored Electroconvulsive Therapy

GWENDOLYN GRAYBAR, M.D.*, JOHN GOETHE, M.D.,† THOMAS LEVY, M.D.,‡ JOHN PHILLIPS, M.D.,§ JOHN YOUNGBERG, M.D.*, DONALD SMITH, M.D.¶

Large T-waves on the electrocardiogram (ECG) frequently are associated with hyperkalemia and can be a manifestation of intracranial injury. Although many ECG alterations accompany electroconvulsive therapy (ECT), large upright T-waves have not been reported.

REPORT OF A CASE

A 42-year-old woman was admitted with the diagnosis of unipolar affective disorder (major depressive illness). Her weight had decreased from 73 kg to 51 kg. An extensive medical and neuropsychiatric workup revealed no organic basis for her depression or weight loss. She had been hospitalized for a similar episode 4 months prior to the present admission. Initial 12-lead ECGs were interpreted as within normal limits. An ECG taken prior to the fifth ECT session revealed a new finding of mild T-dominating, suggesting hyperventilation and/or drug effect.

Multiple Monitored Electroconvulsive Therapy (MMECT) is a procedure for administering electroconvulsive therapy, which allows multiple treatments to be given in a single therapy session. The voltage is varied automatically to provide a constant current of 800 milliamperes through a bipolar square wave pulse. Typically, the maximum energy administered during MMECT is 69 joules; the average energy was 17.5 joules, compared with an average of 50–100 joules in standard ECT.

During each session, anesthesia was induced with thiopental 150–350 mg and d-tubocurarine 3 mg, followed by succinylcholine 100–120 mg iv to facilitate endotracheal intubation. Nitrous oxide 60% and succinylcholine 0.2% as a continuous iv infusion were given for maintenance of anesthesia. A peripheral nerve stimulator was utilized to insure maximal relaxation prior to each stimulus. Hyperventilation was instituted a minute prior to each stimulus. Large, upright T-waves were noted first during the fifth session. Simultaneous ECG and EEG tracings returned rapidly to normal after the seizure and were not associated with apparent hemodynamic compromise (fig. 1). Therefore we elected to follow her next treatment session with a twelve lead ECG, as well as serum potassium and CPK levels (fig. 2).

DISCUSSION

ECG findings in patients with neurologic problems are similar to those described in this report. Burch and Phillips1 reported a series of 55 patients with a variety of central nervous system conditions including tumors, subarachnoid hemorrhages, and intracerebral hemorrhages, with ECG changes believed to be secondary to their CNS lesions. Seventeen of the 55 patients showed the more classic ECG findings of prolonged QT and large inverted T-waves. Thirty-eight had prominent upright T-waves, prolonged QT, prominent U-waves and TU fusion. The QRS complex was unchanged. Greenspahn’s group5 reported a patient with deep T-wave inversions simulating acute nontransmural myocardial infarction following cerebral concussion alone. The EEG, CT scan, enzymes, echocardiogram, and technetium pyrophosphate scan in their patient revealed neither CNS nor cardiac pathology. An imbalance in sympathetic tone to the heart is believed to be the mediating mechanism in ECG findings with CNS lesions.6 Yanowitz et al.7 found in dogs that right stellate ganglionectomy or left stellate stimulation would produce prolonged QT intervals and increased amplitude of T-waves. Left stellate ganglionectomy or right stellate stimulation produced increased T-wave negativity without measurable change in the QT interval. In contrast, Rogers et al.8 showed in cats right or left stellate ganglion block resulted in diphasic T-waves of lower amplitude. Stimulation of the right stellate ganglion in the cat increased the amplitude of the T-waves, while stimulation of the left stellate produced deeply inverted
T waves. In the human, Rogers et al. found that right stellate ganglion block decreased the heart rate, whereas left stellate ganglion block did not change the heart rate. Hugenholtz described a patient with deeply inverted T-waves following right stellate ganglionectomy after radical neck dissection. The changes and clinical course were more consistent with the CNS pattern.

Electroconvulsive therapy is accompanied by both ECG changes and sympathetic stimulation. Troup et al. followed their patients with Holter monitoring 24 h prior to and 24 h after ECT. Following single ECT, the mean heart rate increased from 106 to 142 beats/min. The PR interval decreased, while the QT interval increased. Tachycardia generally is associated with a decrease in the QT interval. Troup et al. attributed the prolonged QT interval during ECT to an imbalance of sympathetic discharge. They did not mention any changes in the T-waves or an increase in either ventricular or atrial ectopic beats. McKenna et al. correlated the incidence of ventricular ectopic beats during ECT with respiratory acidosis and recommended hyperventilation prior to ECT.

Methohexitol with ECT apparently is associated with fewer ECG abnormalities than thiopental. In the Woodruff et al. series, methohexitol for induction of anesthesia resulted in one-fourth the incidence of PVCs than did thiopental, as well as one-fourth the incidence of ST depression.

Valentin et al. found that thiopental and succinylcholine followed by ECT resulted in an average rise in plasma potassium of about 0.25 mEq/l. In their control

![Graphical representation of ECG and EEG tracings](http://anesthesiology.pubs.asahq.org/pdfaccess.ashx?url=/data/journals/jasa/931428/)

**Fig. 1:** Simultaneous ECG and EEG tracings taken during seizure 15 of 5th from MECTA apparatus.

![Graphical representation of ECG and EEG tracings](http://anesthesiology.pubs.asahq.org/pdfaccess.ashx?url=/data/journals/jasa/931428/)

**Fig. 2:** Lead I of 12-lead ECGs taken during the fifth session. Large, upright T-waves occurred during each of the four seizures and returned rapidly to baseline.
patients scheduled for orthopedic and plastic surgery, the rise was 0.1 mEq/l. Pretreatment with d-tubocurarine did not alter the increase in potassium. Our patient's potassium increased from 4.5 to 5.3 mEq/l, nearly three times that reported by Valentin et al.15 However, during the first induced seizure, the T-wave changes were just as prominent during the latter three seizures as during the first, although the potassium had fallen to 4.7, 4.5, and 4.6 mEq/l, respectively.

When succinylcholine is administered to burned patients or those with muscle trauma, denervation, paraplegia, or immobilization, a marked hyperkalemia severe enough to cause cardiac arrest may result. Gronert and Theye13 reported that starvation potentiated the hyperkalemic response to succinylcholine. Our patient's state of inanition may have made her more susceptible to hyperkalemia secondary to the administration of succinylcholine. Her normal CPK did not change during treatment; no MB bands were done because the initial values were so low.

An increase in the amplitude of the T-wave occurs as the potassium approaches 7 mEq/l14 and T-waves are described typically as narrow and tent shaped. Our patient's ECG more closely approximated that of the intracranial pattern described by Burch and Phillips1 than hyperkalemia. The T-waves were wide rather than narrow, and there was no change in the QRS, despite marked increased height of the T-waves; also U-waves were noted on the ECG at the time of the therapy. Additionally, the QT interval was prolonged, consistent with the intracranial pattern.

The transiency of the T-wave changes in our patient is interesting. However, Burch and Phillips1 did note regression in the ECG changes in their patients following surgical removal of their intracranial lesions. Perhaps electrically induced seizures cause transient intracranial damage.

In summary, acute and transient T-wave changes similar to those described by Burch and Phillips1 in patients with CNS lesions occurred during ECT. They subsided immediately with the cessation of the seizure. They were not associated with hyperkalemia and cannot be explained readily on the basis of respiratory acidosis, other drugs, or the anesthetic technique.

REFERENCES