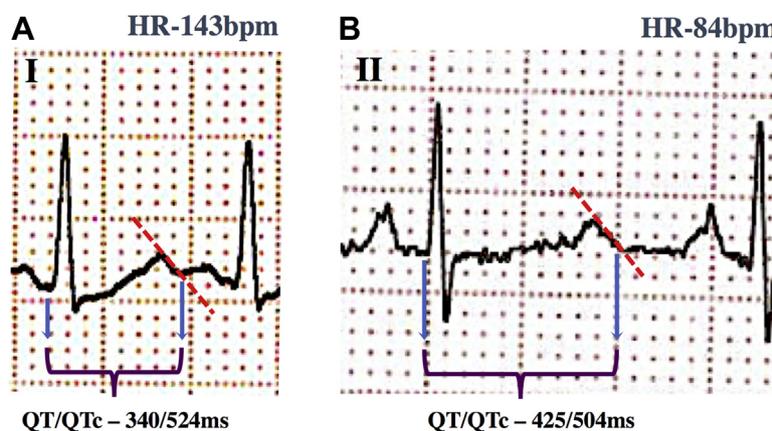


**Figure 1.** Initial ECG at admission.



**Figure 2.** A, Lead I from the initial ECG demonstrates a wide variation in uncorrected and corrected QT intervals. B, Lead II from the postresuscitation ECG demonstrates a prolonged QT and QTc interval.

[Ann Emerg Med. 2019;73:413-414.]

A 62-year-old woman with toxic multinodular goiter presented with 5 months' history of palpitations. She had had a thyroid swelling for 10 years that had gradually begun increasing in size 6 months earlier. She received radioactive iodine therapy at another hospital. Physical examination revealed sinus tachycardia and fine tremor of the hands. A provisional diagnosis of hyperthyroidism was made and tablet propranolol, hydrocortisone, and carbimazole was started.

Her admission ECG is shown in [Figure 1](#). No previous ECGs were available. Echocardiography revealed poor left ventricular function (left ventricular ejection fraction of 30%) and severe pulmonary arterial hypertension (pulmonary arterial systolic pressure of 64 mm Hg). Laboratory tests showed normal serum electrolyte levels and a thyroid function test revealed very low thyroid stimulating hormone ( $<0.008$   $\mu\text{IU/mL}$ ) and high free transcription factor T3 ( $>20$   $\mu\text{g/mL}$ ) and free transcription factor T4 ( $>12$   $\text{ng/mL}$ ), and anti-thyroid peroxidase antibodies was  $<20$   $\text{U/mL}$ .

The patient was shifted to a medical unit, where only oxygen saturation was monitored. After 4 hours, she complained of severe chest discomfort and collapsed. Initial rhythm was pulseless ventricular tachycardia and progressed to asystole (ECG not available). After 8 minutes of cardiopulmonary cerebral resuscitation, she was moved to the ICU. Serial troponin I testing and repeated echocardiography were inconclusive.

*For the diagnosis and teaching points, see page 414.*

*To view the entire collection of ECG of the Month, visit [www.annemergmed.com](http://www.annemergmed.com)*

## ECG OF THE MONTH

*(continued from p. 413)***DIAGNOSIS:****ECG Interpretation**

The patient's initial ECG demonstrated sinus tachycardia. Although the apparent QT interval was normal (340 ms), Bazett's corrected QT interval was prolonged (524 ms) (Figures 1 and 2A). This had been overlooked at the initial assessment because the corrected QT interval was not calculated. ECG after the return of spontaneous circulation showed a distinct prolongation of both the QT and the QTc intervals (425 and 504 ms, respectively) (Figure 2B).

**DISCUSSION**

Although atrial fibrillation is the most common cardiac arrhythmia described in a hyperthyroid patient, infrequently, excess thyroid hormone can also induce a long QT interval and ventricular arrhythmias.<sup>1</sup> The mechanism for this was not well elucidated, but one proposed hypothesis was the overexpression of the myocardial sodium/potassium ATPase gene by thyroid hormone. This leads to an increase in intracellular potassium and ultimately hyperpolarization of the cell membrane and prolongation of ventricular repolarization (Figure E1, available online at <http://www.annemergmed.com>).<sup>2</sup> However, not all patients with hyperthyroidism will develop a prolonged QT interval; this may have been due to the existence of different isoforms of ligand-modulated transcription factor T3 receptors and their variations in genetic expression.<sup>3</sup>

Physicians should have a high index of suspicion whenever they encounter a patient with hyperthyroidism because fatal consequences can be prevented if an abnormal QT interval is identified early.<sup>4</sup> Hence, a thorough inspection of the ECG is suggested for all hyperthyroid patients, with special attention paid to the QT interval. This also facilitates the judicious use of many antibiotics, antiarrhythmics, and anesthetics that have a potential for QT-interval prolongation.<sup>5</sup> Furthermore, early administration of antithyroid medications may normalize the QT interval and prevent the progression of rhythm to ventricular arrhythmias.<sup>1</sup> A general rule of thumb is that a normal QT interval is less than half of the preceding RR interval.<sup>6</sup> In sinus tachycardia, the uncorrected QT interval may be deceptively normal, but when corrected with the RR interval a prolonged QT interval may be unmasked. For instance, in Figure 1 the patient's pulse rate was 143 beats/min and the uncorrected QT interval was 340 ms, but when Bazett's correction ( $QT_c = QT / \sqrt{RR}$ ) was applied, it was realized that the QTc interval was extremely prolonged (524 ms).

In summary, this case demonstrates the need for vigilant ECG evaluation and the urgency required to treat a hyperthyroid state. Unfortunately, in our case, the treating team had not had enough time to optimize the thyroid status before the cardiac event despite the early initiation of antithyroid medications. After 5 days of ICU stay, the patient was withdrawn from mechanical ventilation because of hypoxic brain injury.

**PEARLS**

Use the rule of thumb that a normal QT interval is less than half the RR interval, especially when the QTc interval is not provided in the ECG.

Hyperthyroidism can be associated with prolonged QT interval and may predispose to ventricular arrhythmias.

High-risk patients can be identified with simple ECG screening for long QT syndrome. Vigilance is required for patients with sinus tachycardia because of the great chance of underestimating the QT interval.

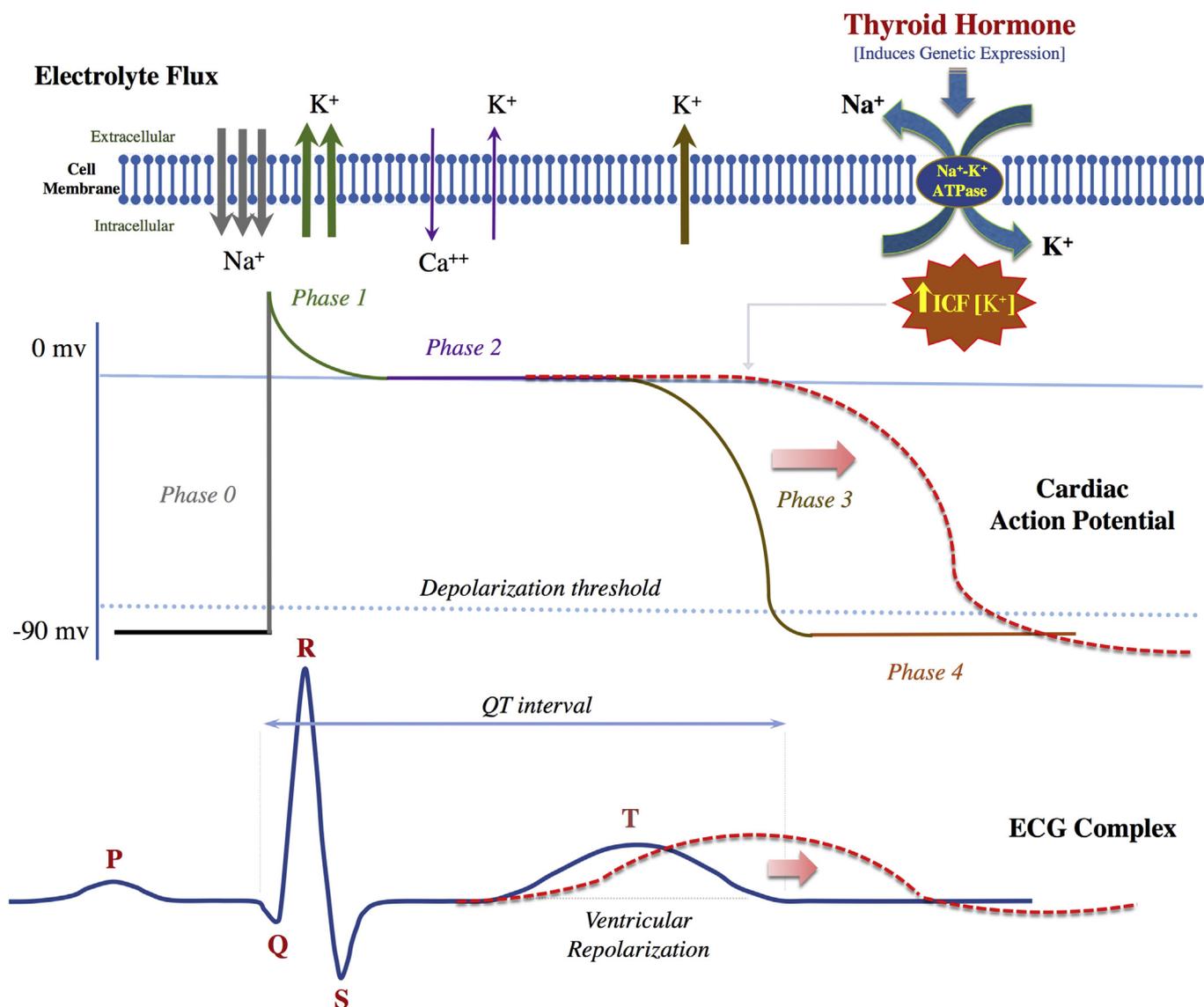
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**Figure E1.** Mechanism of QT-interval prolongation in hyperthyroidism. Thyroid hormones act at nuclear level to increase the expression of sodium/potassium ATPase on the myocardial cell membrane. Increase in intracellular potassium concentration leads to membrane hyperpolarization and QT-interval prolongation.