Longer and Longer

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An elderly woman is admitted to the intensive care unit following resuscitation from an out-of-hospital cardiac arrest. Her ECG is shown in Figure 1.

Figure 1. ECG at presentation.

What abnormality is shown and what causes should be considered?
There is a marked sinus bradycardia (40 beats per minute) and the QT interval is strikingly prolonged, even allowing for the bradycardia. It measures approximately 800 milliseconds (ms) and, when corrected for heart rate, still exceeds 600 ms (the upper limit of normal is 440 ms).

The initial question is whether the QT prolongation precipitated a life-threatening arrhythmia, whether it might be secondary to an acute intracranial event (e.g., cerebral hemorrhage) or whether it is an incidental finding due to some other cause. The appearance of the prolonged QT interval is relevant—neither the QRS complex nor the T wave appear especially abnormal in configuration, and it seems as though the QT interval has simply been “stretched out.” This is unusual in the arrhythmogenic form of acquired long QT syndrome due to drugs, electrolyte imbalance, etc. However, this type of QT prolongation is not unusual in certain metabolic disturbances, such as hypothermia, hypocalcemia and hypothyroidism.

Careful review of the clinical circumstances provides a satisfactory explanation for the ECG findings in this case. Following hospital admission, the patient had been subjected to a therapeutic hypothermia protocol, with a body temperature slightly below 32°C at the time the ECG was recorded. Note that a tiny Osborn or “J” wave can just be discerned in V4. The patient had both hypocalcemia (1.5 mmol/L) and hypokalemia (3.0 mmol/L), which presumably were additional contributors to the lengthening of the QT interval.

Finally, it transpired that the patient also received two boluses of intravenous amiodarone on admission as a prophylaxis against recurrent ventricular arrhythmia. The class III effects of this agent would have promoted further QT interval prolongation but, fortunately, the drug was discontinued before any iatrogenic arrhythmias occurred. Correction of the metabolic abnormalities and eventual re-warming resulted in a gradual normalization of the patient’s ECG.

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