## A dangerous "alternans"

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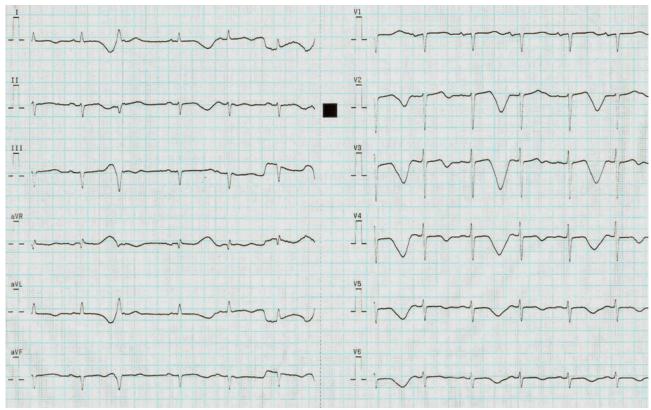
A 68-year-old man was hospitalised because of three syncopes in the same day. He had a history of inferior myocardial infarction, was hypertensive and was on haemodialysis for four years. His medical treatment consisted of aspirin, torasemide, amlodipine and calcium carbonate. For two weeks he has been taking haloperidol because of psychomotor agitation. The 12-leads

ECG recorded on admission is illustrated in figure 1. What is typically shown? What is the imminent danger?

## Discussion

The 12-leads ECG in figure 1 shows a beat-to-beat alternans of the T-wave morphology, amplitude, polarity and length of the QT interval reaching an extreme

Figure 1
12-lead ECG showing beat-to-beat alternating of the T-wave morphology, amplitude, polarity and length of the QT interval reaching a QTc value of 744 msec.



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Figure 2
ECG strip (above) and arterial pressure (below). The ECG strip shows T-wave alternans, premature ventricular beats and the onset of a polymorphic ventricular tachycardia rapidly degeneration into a ventricular fibrillation with loss of pulse (tracing of the arterial pressure).

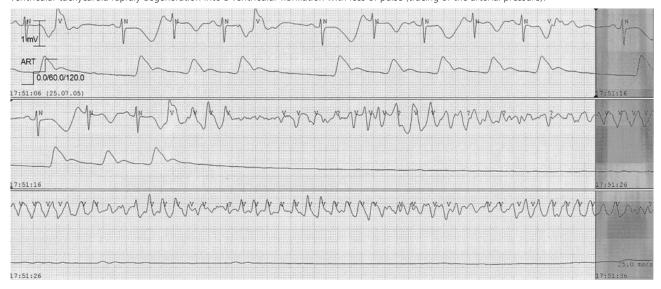
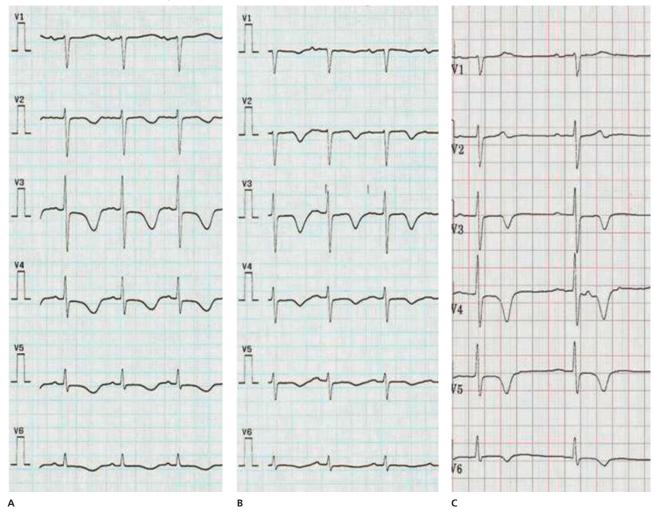


Figure 3
Precordial ECG leads V1-V6 showing the evolution of the T-wave morphology and the QT interval. A: 2 hours after the ECG represented in figure 1.
After an infusion of magnesium sulphate, T-wave alternans disappeared, the QTc was 692 msec. B: 3 hours after the ECG represented in A the QTc shortened to 571 msec. C: after 3 days the QTc normalised (QTc 440 msec).



value (QTc of 744 msec). This phenomenon is known as macroscopic T-wave alternans. Soon after admission he experienced sudden loss of consciousness. The ECG (fig. 2) documented a polymorphic ventricular tachycardia rapidly degenerating into a ventricular fibrillation terminated with an external electrical shock. The serum potassium concentration was 2.4 mmol/l (3.5-5.0 mmol/l), the magnesium 0.42 mmol/l (0.65-1.05 mmol/l) and the ionised calcium 1.11 mmol/l (1.14-1.29 mmol/l). An infusion of magnesium sulphate was immediately started, potassium and calcium were replaced, haloperidol was withdrawn. Some other episodes of non-sustained polymorphic ventricular tachycardia were observed. The macroscopic T-wave alternans disappeared, the QTc progressively shortened and normalised after three days (fig. 3). An echocardiogram showed a mild left ventricular hypertrophy and an inferior akynesia with a mild left ventricular dysfunction (EF 45%). A coronary angiography revealed a chronic occlusion of the right coronary artery without other critical stenosis.

Macroscopic T-wave alternans is a rare phenomenon that can arise in the context of both congenital or acquired long QT interval [1, 2]. It results from large beat-to-beat alternations in action potential duration especially in the ventricular endocardium to midmyocardium causing alternating directions of the transmural gradient of repolarisation [3]. In our case the QT in-

terval prolongation was acquired, induced by a combination of electrolyte abnormalities (severe hypokaliemia and hypomagnesemia, mild hypocalcemia) and drug (haloperidol) with a structural heart disease as a predisposing factor (previous myocardial infarction, mild ventricular hypertrophy). Macroscopic T-wave alternans is considered a marker of an extreme electrical instability and an ominous premonitory ECG sign of torsades de pointes, the polymorphic ventricular arrhythmia typically associated with a prolonged QT interval [1–3]. Torsades de pointes are often self-terminated leading to syncope but may degenerate into ventricular fibrillation, as illustrated in our case. Prompt detection of macroscopic T-wave alternans is critical because of impending danger that is associated with it.

## References

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