technically simple. It does not require special tools or leads and yields good results at 6 months’ follow-up. Longer-term follow-up and dedicated clinical trials are needed to evaluate the efficacy and safety of this approach.

Conflict of interest: none declared.

References

A heartless brain

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A 81-year-old woman was admitted for right-sided hemiplegia. She had a one-month history of palpitations related to atrial tachycardia treated with oral anticoagulants and sotalol (80 mg twice daily). Brain magnetic resonance imaging demonstrated left middle cerebral artery territory infarct affecting the insular region (Figure 1A, arrow). On day 2 she had syncope associated with torsade de pointe (TdP) ventricular (Figure 1B). The corrected QT interval was 460 ms on admission and increased to 640 ms after the arrhythmic event (Figure 1C). Plasma sotalol concentration was normal. There was no mutation in LQT2, KCNE1, KCNE2, KCNQ1, and SCN5A. It is well established that a combination of different parameters, acquired and congenital, are required to facilitate the occurrence of long QT syndrome. In this case, TdP occurred 10 days after initiation of low-dose sotalol and 2 days after stroke. Since the proarrhythmic potential of sotalol is dose-dependent, the insular stroke may have facilitated the occurrence of TdP. This hypothesis is further strengthened by the time course of QT interval changes. This observation suggests that all potentially QT prolonging drugs may need to be withdrawn in patients presenting with acute stroke.

The full-length version of this report can be viewed at: http://www.escardio.org/communities/EHRA/publications/ep-case-reports/Documents/A-heartless-brain.pdf

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