LETTERS TO THE EDITOR

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The ajmaline challenge and a strange ECG

In the early years of invasive electrophysiology, the ajmaline test was introduced to induce or enhance intra-ventricular conduction delays in patients with fascicular block, because of its intrinsic pharmacological properties on the HV interval and QRS duration. Nowadays, a new ajmaline challenge has become a very popular test (both in tertiary and primary centres), because of the induction of a coved ST-segment elevation in the precordial leads, erroneously ascribed to a repolarization abnormality. Much evidence (simply recording late potentials during the infusion) has documented that a depolarization abnormality is what is obtained after the infusion. The induced ECG is similar to the one found in patients with the syndrome of right bundle branch block and sudden death. Unfortunately, the suspected diagnosis is so often told to the healthy subject, forgetting that for evidence-based data and ethical reasons nobody has the authority to drive the conclusions that an induced strange ECG is indicative of a lethal diagnosis.

The large series of Veltmann et al.1 is exhaustive but confusing. One hundred and ten asymptomatic subjects of a mean age of 49.3 ± 14 years have been submitted to the test only because of a basal non-diagnostic saddle back ECG, with 85% positive test, but without any detail regarding the predictive value. The authors report that this saddle back ST-segment elevation is rare in the European population. This might be true in the old population, as the one investigated, but is not the real world in the young. Type 2 and 3 morphologies and first-degree right bundle branch block are not so rare in the young population that we usually see either for sport eligibility or in sedentary students, accounting for a 2–4% prevalence of this ECG pattern in healthy population (personal observations). If we perform ajmaline challenge in this population, how many positive results will be obtained and how many families will be frightened?

I saw yesterday the trace of a 23-year-old asymptomatic man with an ECG showing a first-degree right bundle branch block with minor ST abnormality, in which a previous written diagnosis of ‘suspected Brugada Syndrome’ was done. After this assumption, not based on other evidence, his family, who firstly consulted ‘Google’, became struck with terror.

Five cardiologists have later been consulted giving five different diagnoses and prognoses. When I was also asked by e-mail to give an opinion about the opportunity of the ajmaline challenge, I gave my suggestion after thinking that the young man might be the same age as my son, never submitted to ECG and who might also have a similar trace.

Some years ago, Professor Hurst2 wrote ‘it is not possible to justify the study of a patient’s genes, perform an endomyocardial biopsy, perform an MRI, perform an electro-physiological study, and perform a coronary arteriography in every patient in whom the condition is suspected’. This wise assumption must be kept in mind when we decide to translate laboratory research into clinical work, relying on limited evidence-based data.

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References
3. Bortolo Martini Cardiovascular Unit Boldrini Hospital Via Boldrini 1 36016 Thiene (VI) Italy Email: bortolo.martini@gmail.com

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The ajmaline challenge and a strange ECG: reply

We have read with interest the letter by Dr Martini and we are grateful for his comments. We would like to reply to some of the issues raised by Dr Martini in his letter.

The authors are aware that ajmaline was used as a diagnostic tool in the past to unmask intra-ventricular conduction delays. However, nowadays sodium channel blocker challenge is an accepted and established tool to unmask the ECG pattern diagnostic of the Brugada syndrome.1 This test is especially important in the circumstance when the basal ECG in patients with the Brugada syndrome is fluctuating between the diagnostic and non-diagnostic patterns.2 The underlying cellular and pathophysiological mechanisms and the rationale of this test have been described in detail.3–7

The available data by now have shown that patients with a drug-induced Brugada ECG are at risk for sudden cardiac death. However, the risk in the asymptomatic patient without a spontaneous diagnostic ECG is assumed to be low.3–5

The question is whether to perform the ajmaline test in the asymptomatic patient presenting with a saddle-back type ECG. From the authors’ point of view, the test may offer the chance to exclude an underlying Brugada ECG. In the case of a positive test, the patient has to be risk stratified as mentioned in the manuscript guided by the recommendations of the Brugada consensus conference.6,7 At least repetitive ECG recordings to test for a spontaneous diagnostic Brugada pattern are indicated. Furthermore, general recommendations like avoidance of certain drugs (www.brugadadrugs.org) or lowering of fever can be given.

According to the literature, the prevalence of a saddle-back Brugada type II or type III ECG in European populations is rare ranging from 0.2 to 0.6.6,8 These data might diverge from Dr Martini’s personal observation.

We presented in the paper the results on consecutive ajmaline challenges performed in tertiary referral centres specialized in primary electrical diseases. The results described were based on a retrospective analysis of all tests conducted between 2001 and 2006. During 6 years, only 110 patients were referred for ajmaline challenge in the setting of solely a basal saddle-back type ECG. This indicates that the saddle-back type ECG is a rarity and that we are dealing with a highly selected population.

We agree with Dr Martini that we are still lacking data on the long-term follow-up on