Cardiac repolarization abnormalities and potential evidence for loss of cardiac sodium currents on ECGs of patients with Chagas' heart disease

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Background: Some individuals with Chagas' disease develop right precordial lead ST segment elevation in response to an ajmaline challenge test, and the prevalence of right bundle branch block (RBBB) is also high in Chagas' disease. Because these same electrocardiographic abnormalities occur in the Brugada syndrome, which involves genetically defective cardiac sodium channels, *acquired* damage to cardiac sodium channels may also occur in Chagas' disease.

Methods: We studied several conventional and advanced resting 12-lead/derived Frank-lead ECG parameters in 34 patients with Chagas'-related heart disease (mean age 39±14 years) and in 34 age-/gender-matched healthy controls. All ECG recordings were of 5-10 min duration, obtained in the supine position using high fidelity hardware/software (CardioSoft, Houston, TX).

Results: Even after excluding those Chagas' patients who had resting BBBs, tachycardia and/or pathologic arrhythmia (n=8), significant differences remained in multiple conventional and advanced ECG parameters between the Chagas' and control groups (n=26/group), especially in their respective QT interval variability indices, maximal spatial QRS-T angles and low frequency HRV powers (p=0.0006, p=0.0015 and p=0.0314 respectively). In relation to the issue of potential damage to cardiac sodium channels, the Chagas' patients had: *1*) ≥twice the incidence of resting ST segment elevation in leads V1-V3 (n=10/26 vs. n=5/26) and of both leftward (n=5/26 versus n=0/26) and rightward (n=7/26 versus n=3/26) QRS axis deviation than controls; *2*) significantly increased filtered (40-250 Hz) QRS interval durations (92.1±8.5 versus 85.3±9.0 ms, p=0.022) versus controls; and *3*) significantly *decreased* QT and especially JT interval durations versus controls (QT interval: 387.5±26.4 versus 408.9±34.6 ms, p=0.013; JT interval: 290.5±26.3 versus 314.8±31.3 ms; p=0.0029). Heart rates and Bazett-corrected QTc/JTc intervals were not significantly different between groups.

Discussion: Patients with Chagas' heart disease have increased cardiac repolarization abnormalities, especially by advanced ECG. Moreover, as a group, they have *decreased* uncorrected JT and QT interval durations and *increased* filtered QRS interval durations (versus age/gender-matched controls), all suggesting a potential loss of cardiac sodium channel function that might be mediated, in part, by cardiac autonomic damage. Overall findings support Brugada et al's recent hypothesis that the pathway leading to sudden death may often be similar in Chagas' disease and Brugada syndrome – i.e., damage to the sodium channel (infectious/immunologic/autonomic in Chagas', genetic in Brugada) with consequent loss of sodium currents may facilitate a phase II-reentry based arrhythmic substrate for ventricular fibrillation in both conditions. In general, JT interval-related results have been underreported in the Chagas' literature.