Readers' Comments: Was the Interatrial Block in Patients With Takotsubo Syndrome in the Spanish National RETAKO Registry Partially or Totally Reversible?

I very much enjoyed reading the contribution by Martín-Demiguel et al[1] in the American Journal of Cardiology, reporting for first time on the prevalence of interatrial block (IAB) in patients with takotsubo syndrome (TTS) from the Spanish National RETAKO Registry. The authors found in their 246 patients that 61% had a normal P wave, 24% (58 patients) had a partial IAB, defined as P-wave duration of ≥120 ms and positive morphology in inferior leads, 5% (13 patients) had advanced IAB, defined as P-wave duration of ≥120 ms and biphasic (+/−) P-wave morphology in inferior leads, 7% had atrial fibrillation (AF), and 2% were pacemaker dependent in the admission electrocardiogram (ECG).[1] IAB is known to increase the risk of AF and stroke. At a mean 12 months follow-up, the authors observed a higher composite all-cause mortality/hospital readmission in patients with advanced IAB and AF than in the rest of the cohort, and advanced IAB was an independent predictor of the composite outcome.[1]

Patients with TTS suffer both ventricular (monomorphic and Torsades de pointes ventricular tachycardia), and atrial arrhythmias (e.g., AF), atrioventricular (second and third degree AV blocks), and intraventricular blocks (e.g. left bundle branch block), and sinoatrial block, some of which are transient or persistent, occasionally requiring the implantation of temporary, or even permanent electronic devices. Atrial arrhythmias and IAB may be related solely to the underlying aging process and associated atrial fibrosis, or hemodymnamic decompensation due to TTS, or myocardial and even atrial edema consequent to TTS,[2] or a combination of the previously mentioned.

I know that the authors do not have data on left atrial size or function, or cardiac magnetic resonance imaging, or ECGs beyond the ECG recorded on admission[1]; however the RETAKO Registry Investigators will provide an additional service to both clinicians and researchers if they obtain and analyze ECGs, corresponding to the mean 12-months follow-up, of their 13 patients with advanced IAB, to ascertain whether the IAB was transient (due to TTS), or permanent. Were the ECGs of these 13 patients with TTS show advanced IAB, partial IAB, or normal interatrial conduction?

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1. Martín-Demiguel I, Núñez-Gil IJ, Pérez-Castellanos A, Vedia O, Uribarri A, Durán-Cambra A, Martín-García A, Corbí-Pascual M, Guillén Marzo M, Martínez-Sellés M. Prevalence and significance of interatrial block in takotsubo syndrome (from the RETAKO Registry). *Am J Cardiol* 2019. https://doi.org/10.1016/j.amjcard.2019.03.028. pii: S0002-9149(19)30328-5. [Epub ahead of print].
2. Madias JE. To the editor- implantation of permanent devices in patients with Takotsubo syndrome. *Heart Rhythm* 2016;13:e328.

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Type 2 Myocardial Infarction: Trying to Fit a Square Peg Into a Round Hole?

The Humors...will be compacted into...most obstinate Infarctions
G. Harvey 1689

Any cardiologist who performs consultative services acknowledges the numerous requests to clarify the significance of an elevated troponin, frequently involving patients who did not present with chest pain. Typical symptoms and ECG changes are often lacking, arguing against an acute coronary syndrome. We are then pressed to provide a diagnosis, and more importantly, to recommend therapy. Is an elevated troponin in a dialysis patient a "Type 2 myocardial infarction" or the newly coined "myocardial injury?"[1] Are aspirin, B-blockers, statins, and stress testing or cardiac catheterization the appropriate diagnostic and therapeutic approaches? We believe the current classification schema categorizing this heterogenous group of patients is misleading mechanistically, may cause epidemiologic confusion, and implies a set of therapies which may not be useful or may even be harmful.

There are numerous settings where troponin elevations are commonly found without associated chest pain and ECG changes: arrhythmias, myocarditis, sepsis, heart failure, pulmonary emboli, intracranial bleeding and stroke, surgical disorders (trauma, massive gastrointestinal bleeding, bowel obstruction), renal failure, ablation, cardioversion, cardiotoxicity due to chemotherapy, inflammatory (myocarditis pericarditis) or infiltrative processes of myocardium (amyloidosis), infective endocarditis, significant left ventricular hypertrophy in aortic stenosis or hypertrophic obstructive cardiomyopathy, severe hypertension, hypertensive crisis, aortic dissection, or even strenuous exercise.[2] Multiple mechanisms have been proposed to explain the presence of an elevated troponin in these settings, including localized ischemia due to insufficient oxygen delivery or microvascular dysfunction, direct myocyte injury from endogenous or exogenous toxins, cellular apoptosis, direct injury from stretch or local inflammation, leakage of cytosolic troponin[2] . . . the list of proposed mechanisms is daunting, but what they all have in common is that none of them involve occlusion of a coronary artery. And despite the assortment of proposed mechanisms involved, we would expect that the standard approach for managing ischemic myocardial injury (antiplatelet agents, β-blockers, statins) may not be useful in these settings, and that diagnostic studies and therapies aimed at identifying and opening coronary obstructive lesions will be very low yield and likely offer little benefit.

We commend the World Health Organization for their efforts to construct a classification system which would unify the various causes of myocardial damage,[3] and we also acknowledge the efforts of the writing group for the recent Fourth Universal Definition of Myocardial Infarction where they further attempt to define the subset of patients with troponin release without actual vascular occlusion, and introduce the useful concept of "myocardial injury."[1] Still, we fear there remains confusion as we try to include these nonvascular causes of cardiac damage in a classification scheme