

Disclosures

There is no conflict of interest in this study.

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Interatrial Block: Thromboembolism Risk in the Absence of Atrial Fibrillation



Smietana et al presented a very good review on the possible mechanisms of cardiogenic thromboembolism in the absence of clinical atrial fibrillation (AF).¹ They succinctly highlighted the potential mechanisms including subclinical AF (incidentally detected by monitoring devices), atrial cardiomyopathy (collagen deposition and/or atrial myocardial fibrosis), left atrial appendage mechanical dysfunction (decreased appendage flow velocities, contractility dysfunction such as atrial standstill and/or variation in appendage morphology) and embolism from other sources (such as aortic atheroma or paradoxical embolism across interatrial septal defects).

The authors surprisingly did not even mention about interatrial block

(IAB) or Bayes' syndrome in their review which in fact is a well-recognized risk factor for thromboembolism. IAB refers to a P-wave duration of >120 ms on surface electrocardiogram, while advanced IAB denotes a prolonged P-duration in association with biphasic P-morphology in the inferior leads representing caudocranial activation.^{2–4} IAB has been shown to be a significant risk factor for stroke and other thromboembolic conditions including mesenteric ischemia.^{2,5–7} Although IAB is a known precursor for atrial arrhythmogenesis, IAB can predispose to thromboembolism in the absence of atrial arrhythmias.^{2–4} IAB, although may often accompany left atrial enlargement, is indeed a representative of left atrial electromechanical dysfunction, which may either precede or occur without the presence of left atrial enlargement and atrial arrhythmias.^{2,3} IAB-induced electromechanical dysfunction causes a sluggishly contractile left atrium which may serve as a nidus for thrombi or microthrombi, and may increase the risk for thromboembolic events.^{2,3,6,7} The propensity for thromboembolism, especially in advanced IAB appears independent of the associated atrial arrhythmias.³ In smaller observational and retrospective studies, IAB has previously been shown to be associated with embolic stroke and systemic thromboembolism in patients without a history of known AF.⁸ There has been a significant body of literature to also suggest that many patients develop systemic thromboembolism just by the virtue of an increased cardiac risk profile (such as the one estimated by the presence of a higher CHADS score, an acronym for congestive heart failure, hypertension, age >65 years, diabetes mellitus, and stroke), even without the presence of clinical AF.^{3,9,10} The prospective Asymptomatic Stroke and Atrial Fibrillation Evaluation in Pacemaker Patients study demonstrated a sequential increase in the risk of cerebrovascular attack (CVA) with increase in CHADS score in all patients without clinical AF.⁹ The results from another large prospective registry also demonstrated that the CHADS and CHADS-Vasc scores predicted CVA with fair accuracy in patients without AF and suggested a potential role for antithrombotic

therapy in such patients based on their CHADS risk score.¹⁰

In fact, IAB may be an electrocardiographic hallmark which may either precede or even represent an underlying microstructural atrial cardiomyopathy. Some studies have debated that patients with high CHA2DS2-VASc and advanced IAB, even without known AF may be at high risk for stroke and may potentially benefit from anticoagulation.^{3,4} Thus, IAB as a risk factor for thromboembolism should not be ignored by clinicians. IAB's specific risk contribution toward thromboembolism and its clinical application should be further investigated in the future in large randomized investigations.

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