Reviewer’s report

Title: Surface ECG interatrial block-guided treatment for stroke prevention: Rationale for an attractive hypothesis

Version: 0 Date: 18 Jul 2017

Reviewer: Aamir Hameed

Reviewer’s report:

Dear Authors,

Thank you for this very interesting debate about interatrial block and AF and stroke. You have very lucidly presented the evidence and I agree with the recommendation that anticoagulation should be considered over and above the "documented atrial fibrillation" because it limits the population prone to having strokes being appropriately treated due to our lack of proper and cost-effective surveillance systems. The recent device-based data suggest that AF of duration exceeding a few minutes may put patients at risk. Although this brings more insight into the clinical condition of anticoagulation without the classical clinical documentation of AF. As stated we also know that AF is a risk marker and not a clear causal factor for the strokes seen. We are also grappling with the double-edged sword of anticoagulation with patients at higher risk of annual bleed. Whereas you have recommended anticoagulation for patients with a higher CHADS VASC score you have not mentioned the HASBLED risk. In patients in the 80's and 100's with polypharmacy and multiple risk factors confounding the clinical condition, a HASBLED score would be of paramount importance.

The point of variable fibrosis and expression of spectrum of AF, would make one think as to the role IAB both absolute and partial would play. We know that AF begets more AF. We also know that with more fibrosis the chances of AF go higher as seen in rheumatic heart disease. We also know that stroke even embolic and that in the presence of AF is multifactorial. Do we have enough evidence to show that there is a congruent relationship between IAB to fibrosis to AF to stroke. So is there evidence to suggest that patients with IAB may never go to AF or stroke or the contrary. If there are data to suggest that IAB is a milestone in the journey to stroke with a higher CHADS VASC score with a balanced HASBLED score then surely as you have suggested randomised clinical trials may be planned to answer this important question. However, if data does not support this causality then we should recommend that a study to show direct causality of IAB and AF/stroke be conducted.

Importantly, if IAB is just a transient effect without AF in a normal non-fibrotic atrium with a CHADS VASC risk of 1 would we again be considering oral anticoagulation. So the moot point is, have we placed IAB as an independent marker of stroke or do we need more data to substantiate that and once we have done that would we be positioned for more clinical data. I assume that all of this is for non-rheumatic AF. What about IAB and mild rheumatic disease (disease sparing the valve and causing more atrial fibrosis). Rheumatic disease, AF and stroke forms a large piece of the pie globally or more so in the developing world.
The thought of reversing or remodelling IAB with ACE inhibitors, ARBs and statin would be worthwhile. The role of beta blockers and catecholamine block and surveillance of the P wave. Would you recommend a standard ECG process paper speed and amplitude enhancement to study the P wave in detail?

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