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LETTERS TO THE EDITOR

Selective Ablation of Slow Atrioventricular Node Conduction Pathways: How Safe Is the Anatomic Posterior Approach?

We read with interest the recent article by Engelstein et al. (1) concerning the implications of radiofrequency ablation of atrioventricular (AV) node reentrant tachycardia in patients with posteriorly displaced fast AV node conduction pathways. Although their findings indicate that posteriorly located fast AV node pathways are generally rare (5% in their study), their data—combined with those of previous studies suggesting a higher incidence (2–4)—highlight the existence of a subgroup of patients with AV node reentrant tachycardia who may be at higher risk of developing AV block during radiofrequency ablation using the anatomic posterior approach.

Previous observations made by our group (2–4) provide important clues to explain these findings. In 28 consecutive patients with AV node reentrant tachycardia, we found that the fast conduction pathway was located anteriorly (A region) in 21 (75%) of 28 patients, the middle (or M region) in 4 (14%) of 28 and the posterior (or P region) in 3 (11%) of 28 (2,3). Of critical importance concerning the risk of AV block was our finding that in 12 of 28 patients exhibiting both retrograde slow and fast pathway conduction during ventriculoatrial Wenckebach cycles, 4 of 4 patients with retrograde fast pathway conduction mapping to the M region also had retrograde slow pathway conduction localized to this same region. Overall, 33% of all retrograde slow conduction pathways that could be mapped were localized to the M region, with 66% found in the P region. Given our findings, it is not surprising that radiofrequency ablation near the atrial exit of the retrograde fast conduction pathway in patients with AV junctional anatomy similar to that noted by Engelstein et al. (1) is high risk for producing AV conduction block.

We also previously reported (4) the results of complete mapping of retrograde atrial activation along the anteroposterior axis within the triangle of Koch in 67 patients. In 12 (18%) of 67 patients, the retrograde fast conduction pathway was mapped to the posterior region near the coronary sinus ostium. Importantly, in these 12 patients, a proximal His bundle electrogram was recorded either in the M region (10 patients) or within the ostium itself (2 patients). Atrial extrastimulus pacing and intravenous adenosine administration confirmed that the electrogram recorded posteriorly did indeed originate from the His bundle. In all 12 patients, successful selective ablation of AV node slow pathway conduction was achieved by applying radiofrequency energy to a site posterior to the coronary sinus ostium. Therefore, the findings of Engelstein et al. (1) confirmed our observation that a variant of normal AV junctional anatomy appears to include patients exhibiting posterior displacement of structures normally located anteriorly, including the His bundle. Consequently, it seems prudent to 1) identify high risk patients by means of atrial activation mapping of the retrograde fast conduction pathway; and 2) deliver radiofrequency energy posterior to the retrograde atrial insertion of the fast conduction pathway. The latter suggestion appears to be generally valid because, regardless of the location of the fast conduction pathway, most (but not all) available evidence indicates that the slow conduction pathway always appears to reside posterior to the fast pathway.

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Reply

We appreciate the interest of Lauer and Sung in our report on posterior fast atrioventricular (AV) node pathways and are pleased that their preliminary data confirm our findings. Although we agree with the general theme of their letter, we believe that several issues require clarification.

1. Lauer and Sung indicate that 25% of fast pathways are located in midseptal or posterior locations in patients with slow/fast AV node reentry. Their conclusions were drawn from a small number of patients (1) and are not consistent with our data, which reported a 5% prevalence in 130 patients (2), or with other reports (6% of 85 patients) (3).

2. The patient in our study with posterior displacement of the entire AV node–His bundle apparatus differs from the patients reported by Lauer and Sung. As indicated in their abstract (4), although the His bundle potential was recorded posteriorly, it was in continuity with a His bundle potential recorded from the conventional location. They also elected to ablate the slow pathway so that the exact location of the fast pathway was not determined. In contrast, the His bundle potential could only be recorded posterior to the ostium of the coronary sinus in our study. Ablation at a site immediately posterior to the His bundle potential abolished the anterograde fast pathway (the AH interval increased from 78 to 220 ms). A slow pathway potential was recorded ~2 cm posterior to the site of fast pathway ablation.

3. We are not comfortable with the argument that delivering radiofrequency energy posterior to the retrograde atrial insertion of the fast pathway is prudent because the slow pathway is nearly always located posterior to the fast pathway. There are important exceptions to this rule. We have previously provided evidence that the slow pathway can be displaced anteriorly (5). Therefore, whenever possible, ablation should be guided by careful activation mapping of both fast and slow pathways.

Aside from these clarifications, we are in agreement that posteriorly displaced fast pathways are an underrecognized entity and that

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careful retrograde mapping of the atrial exit site of the fast pathway before radiofrequency ablation can help avoid inadvertent heart block in these patients.

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Supplemental Oxygen Administration and Congestive Heart Failure

We read with concern the interesting work of Haque et al. (1), and we wish to raise two important points.

1. Should we be surprised by a diminution of cardiac output (CO) secondary to supplemental oxygen administration? It seems sensible that the increase in arterial oxygen content (Cao_2) obtained in this way could consequently permit a decrease in CO to maintain constant oxygen delivery (Do_2) at a given work load. Moore et al. (2) described such a variation in CO secondary to an increased inspired oxygen concentration in patients with chronic congestive heart failure undergoing exercice tests. Interestingly, the decrease in CO observed in their case was associated with reduced subjective scores of fatigue and breathlessness. It would have been appropriate for Haque et al. (1) to provide more information about the evolution of the clinical status of their patients during oxygen administration. In other words, we have to be sure that the reported observations truly reflect the detrimental effects of oxygen therapy. The formula of Do_2 permits prediction of the theoretic variation in CO that should be expected in response to a given increase in Cao_2. For example, in the case of the patients of Haque et al., and assuming a normal hemoglobin concentration of 15 g/dl, it can be calculated that the expected decrease in CO would be 8.92% in experiment 1 and 8.16% in experiment 2. Undoubtedly, the reported decrease in CO remains greater (16.22% and 23.68%, respectively), but the significance of the results should be discussed in light of this fundamental notion.

2. It can be calculated from the data of Haque et al. that oxygen consumption (Vo_2) was not constant during the investigation. In fact, Vo_2 values varied from 370.7 ml/min at 21% fraction of inspired oxygen (FIo_2) to 317.8 ml/min at 100% FIo_2 in experiment 1 (Vo_2 (21%) − Vo_2 (100%)/Vo_2 (100%) = 14.27%), and from 354.2 ml/min at 21% FIo_2 to 281.3 ml/min at 100% FIo_2 in experiment 2 (Vo_2 (21%) − Vo_2 (100%)/Vo_2 (100%) = 20.58%). Even if these calculated Vo_2 values are the result of a formula that includes CO, which implies a certain amount of mathematical coupling between Vo_2 and oxygen delivery (Do_2) calculated values, there is no guarantee that an external factor did not modify Vo_2 and, secondly, Do_2 and CO. It has been previously hypothesized (3) that hyperoxia-related vasoconstriction could decrease Vo_2 by increasing peripheral shunting, which could explain the increased mixed venous oxygen saturation (Svo_2) and decreased oxygen extraction ratios (ERo_2) reported in the study of Haque et al. (1). Another explanation could be that their patients were already on the oxygen supply/Vo_2 dependency slope of their Vo_2/Do_2 relations. This could explain the fact that Vo_2 decreased in response to any reduction of CO and Do_2. Some factors support this hypothesis, such as the extremely poor ejection fraction of the study patients or the relatively low Svo_2 measured. In contrast, the values of Do_2 calculated in both experiments (Do_2 (21%) = 386.86 ml/min per m^2 and Do_2 (100%) = 355.64 ml/min per m^2 in experiment 1 and Do_2 (21%) = 401.56 ml/min per m^2 and Do_2 (100%) = 333.5 ml/min per m^2 in experiment 2, assuming a mean surface body area of 1.8 m^2) remain greater than the previously described Do_2 critical limit of 330 ml/min per m^2 (4) below which oxygen supply/Vo_2 dependency appears. The same can be said for ERo_2 (ERo_2 (21%) = 53.23% and ERo_2 (100%) = 49.65% in experiment 1 and ERo_2 (21%) = 49.00% and ERo_2 (100%) = 46.86% in experiment 2), which remains lower than the reported critical ERo_2 limit of 70% (5). The latter two argue for a oxygen supply/Vo_2-independent situation. If Haque et al. (1) had reported the lactate serum levels of their patients, the question of oxygen supply/Vo_2 dependency or peripherical shunting would have been avoided. Actually, lactate serum level usually increases in situations of oxygen supply/Vo_2 dependency, as well as in any form of Do_2 insufficiency (6), and should have been used as the final tracer of oxygen imbalance. Despite these remarks, Haque et al. (1) have opened an important and exciting debate that merits further development even if the clinical implications of such a study are not obvious. Indeed, in a patient with severe heart failure steady state, why should oxygen therapy be initiated? In contrast, if the same patient develops any form of Do_2 insufficiency, he or she will undoubtly take advantage of supplemental oxygen administration.

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