LETTERS TO THE EDITOR

Letters to the Editor will be published when suitable and as space permits. They should not exceed 1,000 words (typed double spaced) in length, and may be subject to editing or abridgment.

Sinus Region Electrograms

To the Editor:

We read with great interest the article by Gomes et al.1 Our experience2 with a technique comparable to theirs (i.e., direct contact) has now been extended to 15 consecutive symptomatic sick sinus syndrome (SSS) patients prospectively selected on the criterion of a corrected sinus node recovery time (CSNRT) exceeding 1000 msec. This supports their view that direct recording of sinus region electrogram (SRE) is useful in “analyzing the relative contributions of changes in sinus node automaticity and sinoatrial conduction during . . . overdrive pacing for estimation of sinus node recovery time.”

Figure 1 illustrates our findings in 11 patients. During a long postpacing atrial pause, the sinus lead displays five local, low-frequency (0.5–25 Hz) SREs. Repetitivel with equal spacing (620–640 msec) validates these SREs against any known artifact (including repolarization). The SRE cycle length approximates the basic cycle length (630–680 msec) during sinus rhythm, suggesting absent overdrive suppression. A slow diastolic slope bridges SRE intervals, suggesting local automaticity.

Obviously, these features do not fit easily into the classic overdrive suppression theory. Indeed, they are precisely those reported experimentally after tetrodotoxin.3,4 Thus, sinoatrial block as the usual mechanism of long postpacing atrial pause is strongly suggested, although it remains to be directly proved that SREs recorded during postpacing atrial pause actually drive the atrium during sinus rhythm. This conclusion was reached by Castillo-Fenoy et al.3 in a pioneer study.

Although mapping of local conduction would be necessary to directly prove conduction, some reasonable information may be gained from temporal relationships, as exemplified by Gomes et al. Figure 2 from the same patient (on no medication) illustrates our findings with programmed atrial stimulation in four of the preceding patients. As the postextrasystolic (A2) atrial cycle (A2A3) progressively increases, an SRE progressively emerges before each return ventriculogram (A3) up to the point of complete exit block (fig. 2), accounting for a more than compensatory pause. The morphology of this SRE is exactly the same as during postpacing atrial pause as shown in the inset (note the different paper speeds); the morphology is different from that in figure 1 because the position of the sinus lead was slightly modified by this time.

Likewise, during prolonged spontaneous alternation of atrial cycles, an identical SRE emerges before A at the end of each long atrial cycle, suggesting that alternating conduction times is the mechanism for the short-long cycle alternation. From these observations and again assuming conduction, we suggest the working hypothesis that the unique ability of extrasystoles to markedly prolong sinoatrial conduction time (SACT) up to the point of complete block characterizes human SSS. It would account for the more than compensatory pauses during programmed atrial stimulation in some patients and for the long postpacing atrial pause (abnormal CSNRT) by a cumulative effect in most patients. These findings do not contradict the observations of Gomes et al. that indirect SACT correlates with its direct measure because the CSNRT (which measures the maximum depressive effect in our hypothesis) of their patients was much shorter than in ours (by design, because a long CSNRT is necessary to demonstrate multiple SREs). The mechanism of this postspacing block is unknown to us, but might be investigated in a dog model we have described.

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Figure 1. Postspacing atrial pause. From top: sinus lead (SL), high right atrium (HRA), right atrium (RA), D1, D2, V1. Time marks = 1000 msec.

Figure 2. Programmed atrial stimulation. From top: SL1, SL2 (proximal electrode same as SL1, distal electrode 1 cm from SL1), HRA, D1, D2, V1. Time marks = 1000 msec. A1 is the atrial electrogram during normal sinus rhythm; A2 = extra beat; St = stimulus; A3 = return ventriculogram; S = sinus region electrogram. The polarity of SL1 is such that the ventriculogram is inscribed downward. Inset: Postspacing atrial pause with the same positioning of the electrode (SL1).
References


The author replies:
To the Editor:
I am indeed pleased that the observations of Asseman et al. using a technique for sinus node recordings comparable to the one described in our paper confirm the viewpoint expressed by us that recording of sinus node electrograms (SNEs) is useful in analyzing the relative contributions of changes in sinus node automaticity and sinoatrial (SA) conduction during overdrive pacing for estimation of sinus node recovery time (SNRT). Their finding of the absence of overdrive suppression as a result of complete SA block (fig. 1) in 11 patients with sick sinus syndrome and corrected SNRT of over 1000 msec is most interesting. However, the possibility exists that in these patients there was no sinus penetration during pacing as a result of SA block. It would be interesting to know the basal SA conduction time on the SNE in these patients. Although I have not seen complete SA block on overdrive pacing as demonstrated in their figure 1, nonetheless our recent studies have shown that SNRT (as currently defined) when assessed on the SNE reflects both automaticity and SA conduction. This is due to significant depression of SA conduction for the first postponing beat as a result of overdrive pacing.

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Exercise Hemodynamic Effects of Captopril

To the Editor:
The article by Kugler et al. on the exercise hemodynamic effects of captopril (Circulation 66: 1256, 1982) falls short in answering the questions, "Where does the flow go when vasodilators increase cardiac output?" and "What does the flow do when it arrives at metabolically active tissue?" There are two reasons for these shortcomings. First, captopril hardly increased exercise cardiac output, and therefore could not be expected to change exercise metabolism. In fact, even though the authors report a small increase in thermoludation cardiac output during exercise, I have calculated from their data that Fick output during exercise did not increase (137 ml/min/kg at control and 136 ml/mm/kg during captopril, p > 0.20). Since the authors now have the availability of both methods, they should carefully examine their laboratory procedures. Second, evaluation of their data on oxygen difference across the leg requires knowing limb blood flow as well. For example, exercising leg oxygen consumption might have increased at the same oxygen difference if flow increased. Perhaps the most interesting aspect of captopril hemodynamics is that despite the negligible improvement in short-term exercise hemodynamics, there is frequently improvement in long-term exercise performance.

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The author replies:
To the Editor:

We appreciate the concern of Dr. Rubin regarding our methodology; however, in the Methods section of a previous paper,1 we reported an excellent correlation (r = 0.96, n = 48) between measurement of cardiac output by thermodiulation technique and by Fick principle during exercise. We congratulate Dr. Rubin for expanding our findings into a full paper.2

The cardiac outputs calculated by Dr. Rubin from our data are incorrect. Indeed, according to the Fick principle, cardiac output is obtained by dividing O2 consumption by arteriovenous O2 difference and not by multiplying them as Dr. Rubin has done.

Concerning his second problem, Dr. Rubin fails to recognize that if oxygen consumption in the exercising limbs had been significantly increased by captopril therapy, exercise capacity would have been improved. Inversely, since exercise capacity was not increased, and femoral arteriovenous oxygen difference was not changed, we can assume that limb blood flow was not increased.

Finally, concerning the effects of long-term therapy with captopril, we refer Dr. Rubin to our previous reports.3,4

We hope these clarifications will help Dr. Rubin in better comprehending our paper. We also encourage him to thoroughly review the literature concerning therapy with captopril in patients with heart failure.

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References

2. Rubin SA, Siemienczuk D, Nathan MD, Brause J, Swan HJC: Accuracy of cardiac output, oxygen uptake, and arteriovenous oxygen difference at rest during exercise after vasodilator therapy in patients with severe, chronic heart failure. Am J Cardiol 50: 973, 1982

2-D Echo for Detecting Blood Stasis

To the Editor:
The work of Mikell et al.1 brilliantly shows the two-dimensional
Sinus region electrograms.
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