whether and which potentials really are involved in AV nodal reentry and which ones are only bystanders.

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Plasma Endothelin in Chronic Heart Failure

McMurray et al's1 recent article raises interesting questions concerning the role of endothelin in heart failure. Perhaps limitations of space restrained the authors from providing information that might have enhanced the ability to evaluate the significance of their results. The following questions seem germane.

1) Was there any difference in endothelin values in male versus female healthy volunteers?

2) Heart failure patients in group 4 seem different, as the authors point out, not only with respect to sex and age and less severe heart failure, but also with a higher frequency of valvular and congenital heart disease. Accordingly, it would have been particularly appropriate to know what their peripheral venous plasma endothelin values were and whether such differences might explain some of the overlap evident in the other heart failure patients as opposed to the normal subjects depicted in Figure 1.

3) The authors state that “endothelin concentration did not appear to relate to the level of left ventricular dysfunction.” Are specific data on this point available?

4) Although differences in aortic and renal vein endothelin concentrations may reflect renal extraction, they could be an indication of nonspecific extraction across arterial venous circulations generally, as has been previously demonstrated in both endogenous and exogenous vasoactive substances. Again, peripheral venous endothelin concentrations in group 4 would have been helpful in identifying the specificity of this contention.

5) Although the authors suggest that no correlation exists between ANP and endothelin (Figure 4), no r value or statistical significance is provided. Arguably, the data could contain a subset of patients in whom the correlation was excellent and others in whom it was not; in this regard, the duration of heart failure, with those of more recent onset being expected to potentially have such a correlation and those with chronic heart failure not, might provide further insight.

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Temperature Monitoring During Radiofrequency Catheter Ablation

The Editorial Comment by Wittkampf1 presents a balanced perspective of the role of temperature measurement during radiofrequency ablation. However, several points merit additional comment.

In our study2 of patients with the Wolff-Parkinson-White (WPW) syndrome undergoing catheter ablation,3 the temperature of the electrode/tissue interface rose rapidly and reached a steady state after only 2–5 seconds. Wittkampf and coworkers recently reported results of an animal study in which thermocouples embedded within the myocardium were used to measure temperature at various distances from the site of ablation.4 They noted a very slow rise in myocardial temperature, with continued increases even after 2 minutes of radiofrequency energy application. On the basis of these differences, Wittkampf concluded that “the rate of rise of interface and myocardial temperature differs with approximately a factor of 60.” However, if myocardial temperature rose very slowly during radiofrequency ablation, then progressive electrophysiological effects might be expected with long energy applications. In fact, it is very rare to see progressive electrophysiological effects after about 15 seconds of radiofrequency energy application. A previous study by the same authors also argues strongly against the notion of slowly progressive heating of the myocardium adjacent to the ablation site. In a careful analysis of the dynamics of lesion formation in vivo, Wittkampf et al.5 showed that there was no further increase in lesion size after approximately 20 seconds of radiofrequency energy application. These data are consistent with their more recent observations and suggest that tip temperature monitoring is a reasonable reflection of heating in the zone of the lesion.

Our study of temperature monitoring during WPW ablation was notable for marked variability in the efficiency of heating between target sites. As a result, there was no correlation between applied power and steady-state temperature. Wittkampf states that “this conclusion may not be justified” because some high-power applications were interrupted by coagulum formation. Even if these four applications are removed from the analysis, the results are the same. Regardless, the issue is not whether there is no correlation or a weak correlation between power and temperature. The important point is that there are a multitude of variables that can affect heating during ablation and that temperature is a much more accurate index of lesion formation than applied power.

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References


*J.J.L. serves on the scientific advisory board of EP Technologies, Inc., the company that provided the prototype catheters used in the study under discussion.
Plasma endothelin in chronic heart failure.

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