Regarding the functional meaning, we have hypothesized that this lateralization may result in a change in anisotropy, which was supported by our rat model. In that model a higher degree of lateralization was seen than observed in human chronic AF. However, it is difficult to extrapolate from the rat experiments to the human immunostaining results, for we cannot know how much of the protein positively staining for connexins resembles functional channels. In the rat model did we observe both lateralization and changes in anisotropy. However, we completely agree with van der Velden and colleagues that inhomogeneities in connexin distribution as described in their study in a goat model (3) should have a high impact on the biophysics of the tissue. In contrast to our findings in humans, they describe a decrease in Cx40, which from our point of view might either be due to species difference or to the fact that they investigated goat atria during the first 16 weeks of sustained pacing, while we investigated chronic AF of one-year duration in patients.

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A Paradigm Shift in Myocardial Stunning

The study by Burnes et al. (1) in a recent issue of JACC was presented to show the close correlation of reconstructed and measured electrograms in and out of ventricular tachycardia, in a chronic (four-day-old) infarction model. The investigators make the additional observation that a large amount of living myocardium may underlie an epicardial region that manifests a pure Q-wave, that is, a pure QS complex.

The investigators suggest that the existence of “regions of noninfarcted tissue below recording sites that show no local electrophysiologic activity” (p. 2073) may imply “the presence of stunned or hibernating myocardium” (p. 2073). In characterizing the electrograms observed in these areas further, Burnes et al. (1) describe “electrograms . . . which showed pure Q-wave morphologies during RA pacing” and which “have a subthreshold –dV/ dtmax indicating no local activation” (p. 2074, emphasis added). Later, adding further description, they state that “the presence of pure Q-wave epicardial electrograms over a large portion of the infarcted myocardium . . . provides evidence for transmural myocardial damage and the absence of an excitable epicardial border zone. . . .” [Pure Q-wave electrograms, indicating lack of local activation, are reconstructed (and directly measured with both epicardial sock electrodes and rod-tip electrodes) over regions of tissue that appear viable in the TTC-stained slices. A possible explanation for these observations is that the apparently viable stained myocardium was not activated because of stunning caused by . . . [and there follows a list of causes of stunning]” (p. 2074, emphasis added).

This correspondent desires to make two points. First, the recording technique, relying as it does on ordinary metal electrodes placed epicardially and on the reconstruction of local electrograms from similarly recorded distant electrograms, may be insensitive to signal of lower frequency content, as these recording techniques record through a metal-electrolyte interface, which is a high-pass filter (2,3) and therefore fails to reproduce signal components of low-frequency content faithfully. Whereas the coordinated activation front of a hundred thousand heart cells depolarized simultaneously via the action potential Na channel is expected to have a high-frequency content, the uncoordinated depolarization perhaps also characterized cellularly by less sharp up(down)strokes may escape detection by this technique. Perhaps this latter scenario is familiar from the case of concealed conduction into atrioventricular (AV) node tissues, which cannot be recorded by these techniques either. The former, less familiar scenario has been presented by various researchers, including Delmar et al. (4). This first comment, even if accurate, does not detract from the accuracy of the interpretation offered by the investigators (1) for their data. This comment would merely support the possibility of local activation not detected by the presented techniques. That such may occur is in any case implicit in their discussion of the path of the ventricular tachycardia impulse.

The second point has to do with the usage of the term “stunning” in the text that is quoted above. When the term stunning is used, most investigators do not conceptualize viable myocardium with no local activation subjacent to a QS complex four days after the occurrence of acute myocardial infarction. Without questioning the interpretation or the propriety of use of the term stunning by the investigators (1), this correspondent merely wishes to point to the novelty of this use of the term. (Furthermore, there can be no question about the accuracy of localization of the QS complexes in relation to the viable myocardium in this carefully performed work, where quadruply redundant verification existed—electrogram by sock, electrogram by rod, distant electrogram and reconstructed local electrogram.)

If the usage is accurate, then a paradigm shift has been introduced surreptitiously—the idea that what is termed stunning may include myocardium that is viable, and perhaps even healthy, yet which is not activated electrically. If this were a tiny speck of muscle in a scar-encircled island, the paradigm shift would be, at most, of trivial significance. But being a large piece of viable muscle situated at a border zone of infarction (where it might have corresponded to clinical stunning or to clinical infarction) this third possibility—that is, stunned myocardium which is neither infarcted nor stunned in the ordinary fashion—is a significant extension of the meaning of the term stunning.

Concepts exist in the literature to account for this behavior as a new category of stunning, although the category has existed only theoretically prior to these observations of Burnes et al. (1). While in the relatively recent review of Bolli and Marban (5) of molecular and cellular mechanisms of stunning, no mechanistic concept was presented that could encompass this category of stunning, the more recent channels biophysics-based review (6) suggested that disruption of intercellular communication (i.e., gap junction uncoupling) could emulate clinical stunning, with precisely these characteristics of viable but not locally activated myocardium. Not emphasized in that review was the obvious implication that such viable tissue would underlie a region of QS complex, identical to what has been shown to exist by Burnes and colleagues.
Also significant is the fact that, of all the categories of stunning, this one is likely to be the most arrhythmogenic.

Finally, irrespective of whether these comments find their way into print, this correspondent appreciates the opportunity to comment to this great team of investigators who have taught electrocardiographic–electrophysiologic correlation to an entire generation of researchers.

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