Letter to the Editor

What is a monophasic action potential recorded by the Franz contact electrode?

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After over 20 years of successful use of the Franz contact electrode technique by many others and myself (over 1000 publications to date), the recent paper by Kondo et al. [1] claims that it is not the tip electrode, but rather the distant-from-tip or “indifferent” electrode that records the monophasic action potential (MAP). The introduction starts out by staging a contradiction between the classical MAP theory and the one stated by Franz [2] when in fact there is none really stated. The wording is confusing throughout their text and, in my view, of little help to foster the understanding of the complex issue of MAP genesis and recording. More important, the study by Kondo et al. [1] contains a plethora of methodological flaws of which only some can be addressed here.

The isolated canine wedge preparation used by the authors is small compared to the BARD catheter used in this study. Despite the authors’ assertion, the BARD catheter is not “Franz-like”. It also is not drawn to proper scale in their figures. In reality, the BARD electrode shaft would look more like a “tower” sitting on the small isolated wedge preparation. The Franz catheter was designed for human and large animal hearts and in those has provided high-resolution recordings of spatial heterogeneities in myocardial repolarisation. For smaller hearts or preparations we have developed greatly miniaturized MAP electrodes [3].

The study by Kondo et al. reports on a multitude of interventions (KCl application, local cooling, ATX-II application, multiple micro-electrode impalements which are time-consuming and often unstable, and intramural plunge electrodes). All data sets are n=1 with little if any statistics provided on spatial confinement of the effects of KCl, ATX-II, or cooling. Clearly, diffusion must occur and blur the distinction between sites only 2–5 mm apart.

It has long been recognized that KCl depolarizes myocardium, and when an electrode is placed onto the KCl site an MAP is obtained [4]. We thus maintain that the KCl site in the study by Kondo et al. [1] is the MAP-generating site. The KCl electrode and the intramural needle electrode were fed into the same amplifier. As the authors stated, the intramural needle electrode (after initial injury and subsequent healing-over) recorded an electrogram. An electrogram recorded at one input and an MAP recorded at another (the KCl site), when superimposed and of similar amplitude, still looks like an MAP. However, this is an MAP generated at the KCl site—with an intramural electrogram superimposed. The fact that the MAP is upright when the KCl electrode is connected to the positive amplifier input (as was the case in their study) further supports this contention [2].

To pick one of the illustrations that are used to reinterpret the role of the MAP electrodes, Fig. 2 in Kondo et al. [1] at first glance produces convincing evidence of their alternative hypothesis because both the MAP signal and the transmembrane action potential prolong in concert in response to a decrease in temperature. However, it should be realized that the KCl electrode was coupled against the needle electrode at the cooled site. Naturally, this would produce a prolonged electrogram (QT) that by superimposition onto the KCL-MAP template makes the MAP appear longer. Another

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example is the ATX-II injection experiments (Figs. 6 and 7). Here, the BARD tip electrode was not even in direct contact with the injection site, which was either lateral from it or in deeper layers. Even the large BARD electrode didn’t record those effects, which supports rather than refutes the notion that the tip electrode is the exploring electrode with a very discriminative field-of-view. The interested reader might want to apply similar scrutiny to the remainder of the figures.

The widely accepted and validated interpretation of the MAP signal is based on the development of extracellular potentials caused by current sources in a volume-conductor [2]. The field-of-view potential recorded by a differential amplifier with 2 electrodes in a volume conductor depends on the solid angle of each electrode with respect to the current source or voltage subtended by it [5]). In a large heart (human, canine, and similar) the tip electrode “sees” mainly the local electro-motive force that is created at the boundary between normal tissue and tissue locally depolarized by the contact electrode via shear stress or compaction [6,7].

The proximal electrode (5 mm distant from tip) does not, or very weakly, detects these local current sources. In a large heart, a 5-mm inter-electrode distance and differential amplification effectively cancels out far-field potentials (QRS and T wave) and leaves only potentials generated by local current sources near the tip electrode, i.e. the local MAP. This is the reason for the term “close-bipolar” MAP electrode, first introduced by Olsson for the suction MAP electrode [8].

Antzelevitch and co-workers went to great length to support their alternate hypothesis, and I have addressed a possible motivation previously [9]. Yet, the role of each electrode of the MAP recording circuit can be easily and unequivocally established, as was already done [10]. The contact tip electrode (T) is placed against heart tissue with slight pressure. Another electrode is placed proximally at a small distance from the tip and heart tissue and serves as the “indifferent” electrode (I). A third “common” or “ground” electrode (G) is placed away from electrodes T and I in the tissue bath. These 3 electrodes are fed into 3 different high-impedance differential amplifiers that do not allow crosstalk. Thus, each output represents the individual input. This simple experiment [10] shows that electrodes T-I (“close-bipolar” MAP) record a “clean” MAP. T-G (“unipolar MAP”) record an MAP with electrogram or ECG contaminations. I-G (“unipolar electrogram”) record just that: a unipolar electrogram but not an MAP. Neither the I nor the G electrode detects the MAP information. This leaves only one electrode—the tip electrode. Kondo et al. [1] opted not to perform this very simple and definitive experiment.

The genesis of the MAP and which electrode records it has been a matter of spirited debate for more than 50 years. Apparently, this debate still goes on. However, a reinterpretation of the role of the MAP-exploring tip electrode, based on the data by Kondo et al. [1], is not warranted.

References: