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ScienceDirect
Journal of Electrocardiology 41 (2008) 575–580
JOURNAL OF Electrocardiology
www.jecgonline.com

The meaning of the Tp-Te interval and its diagnostic value

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Received 18 April 2008; revised 5 July 2008; accepted 5 July 2008

Abstract

Background: The interval between T peak (Tp) and T end (Te) has been proposed as a measure of transmural dispersion of repolarization, but experimental and clinical studies to validate Tp-Te have given conflicting results. We have investigated the meaning of Tp-Te and its diagnostic potential.
Methods: We used a digital model of the left ventricular wall to simulate the effect of varying action potential durations on the timing of Tp and Te. Furthermore, we used the vectorcardiogram to explain the relationships between Tp locations in the precordial electrocardiogram leads.
Results: Prolongation or ischemic shortening of action potentials in our model did not result in substantial Tp shifts. The phase relationships revealed by the vectorcardiogram showed that Tp-Te in the precordial leads is a derivative of T loop morphology.
Conclusion: Tp-Te is the resultant of the global distribution of the repolarization process and is a surrogate diagnostic parameter.

Keywords: Dispersion of repolarization; T_{peak}-T_{end} interval; Vectorcardiography

Introduction

Increased dispersion of repolarization, the disturbance of the normal orderly pattern of ventricular recovery, is generally thought to predispose to ventricular arrhythmias. It is, therefore, most desirable to be able to read the subtle signs in the ST-T that might foretell trouble. QT dispersion was hailed as such a sign but finally had to be dismissed as an erroneous concept.[1,2] Now the interval between the peak of the T wave (Tp) and the end of the T wave (Te) is being proposed with the same intent.

The idea was born in the realm of left ventricular cellular electrophysiology. Haws and Lux[3] measured that the end of repolarization (EOR) as estimated from pericardial unipolar electrograms approximately coincided with the end of a single given intracellular action potential (AP). The relation between intracellular APs and the electrocardiographic T wave was investigated in greater detail by Yan and Antzelevitch[4] with the use of left ventricular wedge preparations. They recorded APs separately from the subendocardial, midmyocardial, and subepicardial layers synchronously with a bipolar "pseudo" electrocardiogram (ECG) straddling the preparation. The APs from the midmyocardium had the longest duration, presumably produced by the M cells earlier described by Sicouri and Antzelevitch.[5] The peak of T in the pseudo ECG aligned with the end of the epicardial AP, the earliest completion of repolarization in the ventricular wall, and the end of T aligned with the end of the midmyocardial AP, the total conclusion of repolarization. The Tp-Te interval was, therefore, taken to be a reflection of the transmural dispersion of repolarization (TDR). When an ECG was constructed from the potential differences between the APs, it matched the actually recorded pseudo ECG.

It is quite a step from the wedge preparation to the in vivo experiment, where TDR is measured in the whole heart and its imprint on the ECG is examined in a regular surface ECG. In some studies (eg, Bai et al[6]), the findings of Antzelevitch were confirmed, but contrary results emerged from other studies. Xia et al[7] report that, in pigs, the earliest EOR as well as the latest EOR are both found in the endocardium. In the precordial leads, Te more or less coincides with the latest endocardial EOR and Tp with the earliest endocardial EOR. Tp-Te, therefore, is not a measure of transmural but of global (apico-basal) dispersion of repolarization. In their experiments, there is no role for M cells, which might even be absent from the porcine myocardium altogether. Similar conclusions are reached by Opthof et al.[8] In open chest dogs, they performed endocardial, epicardial, and intramural mapping of repolarization times, defined as the sum of activation time and

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0022-0736/$ – see front matter © 2008 Elsevier Inc. All rights reserved.
doi:10.1016/j.jelectrocard.2008.07.030

activation-recovery interval. The mean difference in local repolarization times between endocardium and epicardium was only 2.7 milliseconds in normal dogs, increasing to 14.5 milliseconds in so-called memory dogs, pretreated with 3 weeks of ventricular pacing. Tp-Te in the surface ECG (lead II) measured 42 milliseconds in both groups and therewith clearly exceeded any TDR but appeared tightly correlated with global dispersion of repolarization, defined as the difference between earliest and latest moment of repolarization at any myocardial recording site. No midmyocardial zone of latest repolarization was found. In all dogs, epicardial repolarization lagged behind endocardial repolarization, and Tp in lead II coincided with the average EOR of the right ventricle.

Matters are compounded by the fact that measurement of Tp-Te is far from straightforward and coherent instructions for its measurement are lacking. The problems arise as soon as the T wave deviates from the standard form. Must a flat ST-T be simply ignored? What to do with a biphasic T? What if the T wave gradually changes sign across the leads? Is the maximum ST-T negativity equivalent to a Tp?

However unsettled the issues still are, clinical studies have already appeared that say that increased Tp-Te is associated with a heightened tendency to, or an enhanced inducibility of, ventricular tachycardia and, consequently, may be seen as a sign of a harmful dispersion of repolarization.[9,10] Oddly enough, a shorter Tp-Te (averaged over V_4-V_6) went along with increased cardiovascular mortality in a study by Smetana et al.[11]

The present study is an attempt to explain and perhaps reconcile these conflicting findings. We investigated the meaning of Tp-Te and its diagnostic potential by 2 different methods. Firstly, a digital model of the left ventricular wall served to simulate the effect of varying AP durations on the timing of Tp. Secondly, the vectorcardiogram (VCG) was called to assistance for understanding the behavior of Tp-Te in the precordial ECG leads.

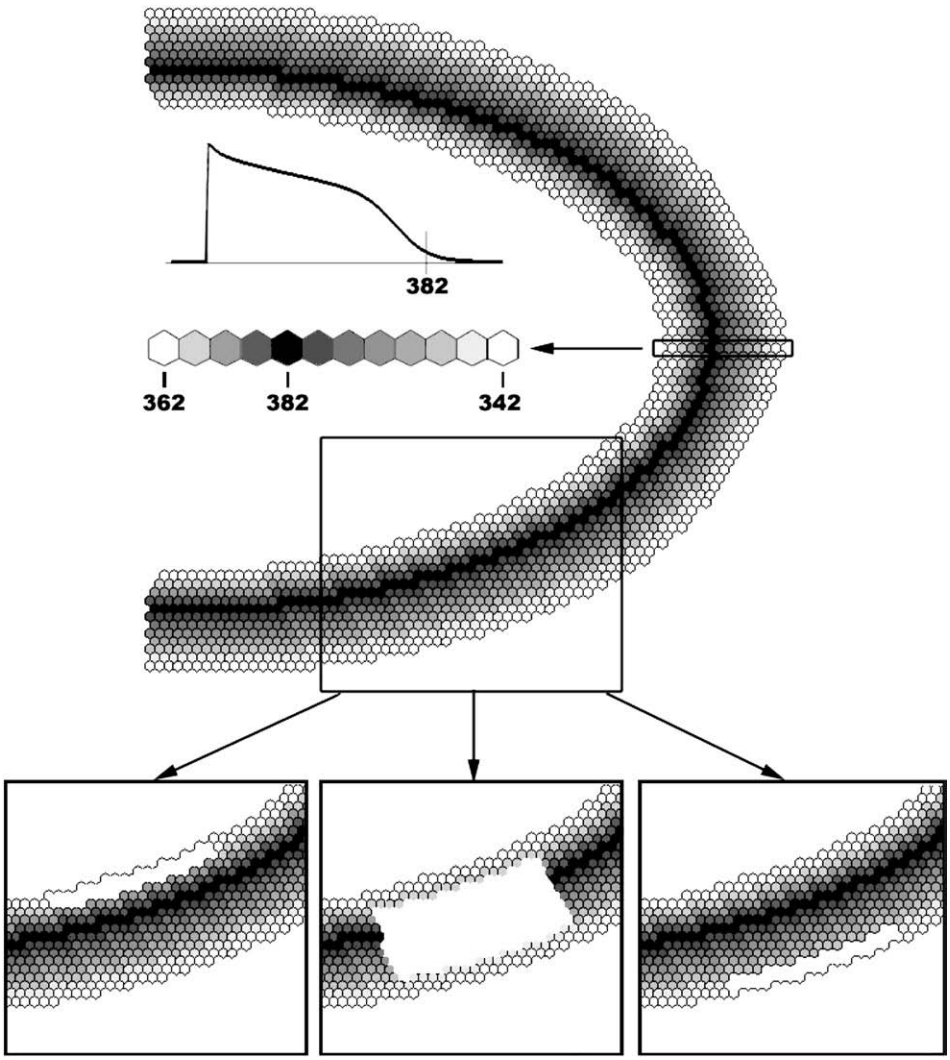

Fig. 1. A slice of the left ventricular myocardium, containing 1961 hexagonal cells organized in 12 cell layers from endocardium to epicardium. The AP90 durations of the cell layers are rendered by shades of gray, with gradually increasing and decreasing AP duration between the epicardial and endocardial layers (figures in milliseconds). As a typical example of AP shape, the AP with AP90 at 382 milliseconds is shown. The inserts at the bottom show 3 areas in the anterior wall in which APs were shortened to simulate ischemia.