Indian Pacing and Electrophysiology Journal 18 (2018) 193–194

Contents lists available at ScienceDirect

Indian Pacing and Electrophysiology Journal

journal homepage: www.elsevier.com/locate/IPEJ

Mapping ventricular fibrillation ... another piece from the jigsaw

In this issue of the Indian Pacing and Electrophysiology Journal, Jason Tri and coll. report their interesting experience in mapping ventricular fibrillation (VF) in dogs [1]. In this model, they demonstrate some intramural differences in activation rate and organisation, as well as between insulated (proximal) and non-insulated (distal) regions of the His-Purkinje network. In addition, some areas have been shown to exhibit periods of regular electrical activity despite continuing fibrillatory pattern on surface ECG [1].

VF is the end-stage of the electro-physiological adventure. After deciphering all kind of supraventricular arrhythmias, after the huge workload dedicated to atrial fibrillation, VF remains the ultimate challenge. Knowledge about the intime mechanisms underlying VF in humans may ultimately lead to a definitive solution for most sudden cardiac deaths, thus representing a major public health issue.

Unfortunately, to date, studies about VF mostly involved animal models or human hearts far from usual clinical conditions. Thus, their conclusions may not apply to clinical VF. The study by Jason Tri does not make exception to the rule, and moreover, even if interesting, many findings had been in fact already noted in the past.

Historically, VF had been classified in type 1 VF (predominantly caused by multiple wandering wavelets) and type 2 VF (when myocardial activation is mainly driven by a mother rotor), which were associated with differences in conduction and repolarisation restitution [2,3]. The fact that VF was sometimes not a random disorganized rhythm came from optical mapping in explanted perfused hearts [2,3]. Then various endocardial and epicardial VF mapping studies have been conducted in humans [4–9], often revealing large sustained and repeated activation wavefronts, together with the existence of a limited number of rotors or localized reentry.

However, in most of these studies, the experimental conditions made these unsuitable to human clinical VF in the real life. Only a few studies involved short-living « natural » human VF as occurring in clinical practice [10,11]. According to some of these works, endocardial activation during the initial steps of VF was already not found to be random, more consisting of a few large wavefronts [10].

Non invasive « panoramical » view of the clinical spontaneous human VF process was more recently reported [12], showing rather consistent and fixed drivers and rotor waves. However, these results were achieved after complex mathematical data processing of surface recordings, and it is not clear whether this is a true translation of what is really going on in cardiac activation during VF.

In human clinical VF, using simple multi-electrode endocardial mapping, we also previously found fast and regular discrete activation covering the whole duration of every intracardiac recording, whereas surface ECG consistently displayed chaotic and fibrillatory pattern [13]. We also found that most of the VF episodes were slightly faster and more regular at recording sites including the right ventricular apex compared to RVOT or basal LV [13].

Previous studies focusing on frequency or time-domain analysis of human VF already showed some differences between left and right ventricles [9,11]. Endo-epicardial gradient had been already noted, probably due to more resisting Purkinje tissues to ischemia [14]. Although rapid, a slower activation rate was also found at the epicardium in intraoperative mapping of myopathic human hearts [8]. However epicardial activation rate was found rapid in humans during short-duration VF or during cardio-pulmonary bypass [4,5], probably reflecting the lack of ischemia in these works. In clinical VF, left epicardium showed higher dominant frequency compared to right endocardium [11]. In some experiments, fastest sites were also found to be the most regular ones [[13,15]].

Finally, involvement of the Purkinje network in induction and maintenance of VF has been evoked and documented for many years [16–18].

Since any new insight into VF mechanisms is wellcome, the authors should be felicited again in mapping VF. They demonstrated regular fast endocardial activation and especially at sites involving the distal His-Purkinje conduction tissue (possibly at interface with myocardium) and the likely non involvement of epicardial sites in this model. Although very interesting, these datas can be discussed keeping in mind that these were not recorded at the same time (unlike “paroramical” mapping with EGM imaging), so that change may have occur between each recording site due to the ischemia and ongoing fibrillation process. Thus, conclusions should be prudent, even if order of mapping sites was randomly allocated limiting this bias. Furthermore, duration of VF was long and mechanisms involved during earlier parts of VF are probably different.

Nevertheless, the paper by Jason Tri and coll. from the current issue of the Indian Pacing and Electrophysiology Journal is another important piece to add to the VF jigsaw.

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Available online 5 November 2018