ELECTROCARDIOGRAPHIC ANALYSIS OF INDUCED VENTRICULAR FIBRILLATION

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Abstract

Introduction: Ventricular fibrillation ECG is characterized by the presence of irregular QRS complexes, with variable morphology, amplitude and frequency. Aims: Analyze the electrocardiographic characteristic of induced sustained VF (ISVF) . Methods: The 12 lead ECG of ISVF was analyzed in 8 patients with coronary artery disease (CAD) and 7 with Brugada Syndrome (BS). The ECG was divided into triggers and two tachysystolic and sinusoidal phases, based on Wiggers' stages. Results: Triggers: Four QRS morphologies: similar to stimulated (IR); LBBB with superior axis (SA) (suggesting origin in moderate band of the right ventricle (MB); RBBB with superior left axis (SLA) (suggesting origin in posteromedial papillary muscle (PPM) predominant in CAD; and LBBB with inferior axis (IA) (suggesting origin in RVOT) predominant in BS. Tachysystolic stage: there is a predominant morphology with RBBB and SA in CAD; and morphologies with LBBB and IA in BS. Sinusoidal stage: Complexes of small amplitude with notches appeared in SVF, but not in non-sustained VF. Conclusions: The ECG at the onset and tachysystolic stage of ISVF shows morphologies which may be related to the activation of MB, PPM and RVOT. The notches could be a predictor of SVF .

TEXT

Introduction: The ventricular fibrillation ECG is characterized by the presence of irregular QRS complexes, with variable morphology, amplitude and frequency. Therefore, it is quite difficult to interpret. However, numerous researchers have tried to recognize discrete elements. Provided that VF primarily consists of a few large wave fronts on the endocardium of humans, the interpretation of ECG of VF should be less difficult. Based on experimental studies and published cases of ablation of VF triggers[1-10], we are aware of essential structures that participate in the fibrillation process. The activation of each can be seen in the electrocardiogram. It is evident that their simultaneous activation will lead to complicated electrocardiographic complexes, which is why there is talk of a disorganized signal in the VF.

Aims: Analyze the electrocardiographic characteristic of induced sustained and non-sustained VF in laboratory, and establish a method to manage ECG of VF.

Methods: The twelve lead ECG of induced VF has been analyzed . VF was induced through programmed electrical stimulation (PES) in 8 patients with coronary artery disease (4 anterior and 3 myocardial infarction) and 7 with Brugada Syndrome ; 14 males, aged 53 ±15 years. A programmed electric stimulation consisted of 3 drive cycle lengths (500, 430, and 330 ms) with 1 to 3 ventricular extrastimuli ([?]200 ms) from 2 right ventricle sites and burst pacing (cycle length [?]200 ms). Indications for the electrophysiological study were ventricular arrhythmias , unexplained syncope or risk evaluation of sudden death in patients with ischemic cardiomyopathy and Brugada Syndrome. All patients provided written informed consent. For analysis. the ECG has been divided into triggers (first premature complex PVC 1 and the second PVC 2) and two tachysistolic and sinusoidal phases, based on Wiggers' stages[11]. The tachysistolic phase comprises

the trace after 1-2 first complex. The sinusoidal phase would correspond to the trace from which there are no defined QRS complexes and only sinusoidal waves can be distinguished.

Results:

Triggers

In patients with CAD the mean coupling interval (CI) between the extrastimuli and PVC1 (S-PVC1) was 237+-75 ms and between PVC1- PVC2: 212+-70 ms. The duration of QRS: PVC 1 187+- 35 ms and PVC2 189+-32 ms. In patients with BS, SPVC1: 277+- 50 ms; PVC1-PVC2: 217+- 28 ms; QRS duration PVC 1: 207+- 32 ms; PVC2: 186+- 33 ms. (Fig 1). Four QRS morphologies were observed : similar to stimulated QRS (IR); LBBB with late transition and superior axis; RBBB with superior left axis and LBBB with inferior axis. These morphologies appear both in PVC1 and PVC2. In Brugada Syndrome cases, the predominant morphologies were those with LBBB morphology, and in CAD cases, those with RBBB morphology predominated (Fig 1)

Tachysistolic stage

In this first phase, QRS morphologies are still observed, unlike the following phase where only sinusoidal waves can be seen. In the group with coronary arterial disease there is a predominant morphology with right bundle branch and superior axis. (Fig 2,3). In the Brugada Syndrome group, RBBB and superior axis morphologies are not distinguished; on the contrary, LBBB and inferior axis morphologies are predominant, together with others which are less common with LBBB and superior axis morphologies (Fig 4).

Sinusoidal stage

The phase after what we call tachysistolic, is characterized by sinusoidal complexes, where we can no longer distinguish QRS, which following Wigger's description could correspond to Wigger's convulsive incoordination phase. If we analyze this portion of the ECG we can distinguish complexes of great amplitude and others of smaller amplitude with notches; established VF is characterized by sinusoidal complexes with large amplitude and traces with an appearance of notches. These should not be confused with the final phase of VF where there are only low amplitude waves corresponding to a phase of disorganization. Notches in the sinusoidal complexes appeared in sustained VF, but not in non-sustained VF (Fig 5).

Transitions

The transitions would correspond to the complexes that separate the programmed stimulation from the above mentioned triggers and between defined phases in established VF (tachysistolic, sinusoidal). Two morphologies of QRS complexes appear at the beginning of VF induction and in the transitions between stages, mainly between the tachysistolic and sinusoidal phases.

1.Narrow complexes: These morphologies may suggest the participation of Purkinje fibers as principal nexus between different activation zones in VF (Fig 6).

2. Complexes QRS characterized by a very high J point. The J point of extrastimulus which trigger VF has greater amplitude than those with longer coupled interval which do not triggering VF. There is actually a progressive increase in the amplitude of J point (Fig 7.1,7.2, 8). This phenomenon (elevated J point) can also be observed in the transition between the VF stage with differentiated QRS (tachysystolic) to the sinusoidal stage (when there is no clear QRS complex, only sinusoidal waves).

Discussion:

Triggers:

Spontaneous Ventricular fibrillation (VF) is initiated by premature ventricular contraction (PVC) originating in the right ventricle outflow tract (RVOT) , Purkinje from left or right ventricle[1,3,5]. It is not known whether these structures also participate in the onset of induced VF. In our study, the morphologies found suggest activation of specific structures: LBBB with late transition and superior axis (suggesting origin in moderate

band of the right ventricle); RBBB with superior left axis (suggesting origin in posteromedial papillary muscle PPM) and LBBB with inferior axis (suggesting origin in RVOT). Similar complexes of stimulated QRS (IR) would be due to local myocardial reentry[12]. In Brugada VF the most frequent triggers correspond to morphologies suggestive of RVOT, often showing moderator band morphologies in cases of induced VF and, exceptionally, in spontaneous VF analyzed in articles; this could be interpreted as the moderating band behaving as a bridge to the RVOT of the extrastimuli applied from the apex of the RV .

Tachysystolic stage

Wiggers described the onset of ventricular fibrillation (VF) as initiated by a short run of premature contractions which he described as the tachysystolic stage (TCHS). In all CAD patients, there was a predominant morphology with right bundle branch and superior axis, which remind us of the morphology of VTs with origin in posterior papillary muscle. It is known that the papillary muscle plays a vital role in the generation and maintenance of reentry during VF[13]. It could be hypothesized that in patients with coronary arterial disease that Wiggers' tachysystolic stage of VF is related to the activation of the posterior papillary muscle. It might also be considered that the activation proceeds from the scar in cases of inferior posterior infarction; in fact, the rotor has been located at the edges of the scar[14]. Favoring the hypothesis of the origin from the posterior papillary muscle, we have also seen this morphology in a case with anterior / lateral infarction , as well as in spontaneous cases of VF in ischemic patients regardless of the location of the scar location.

In VF cases in patients with Brugada, morphologies compatible with activation of RVOT predominate and less frequently in the moderating band. This RVOT morphology has been described previously[15]

Notches in sinusoidal phase

Established VF was characterized by large amplitude sinusoidal complexes and traces with an appearance of notches.

We should not confuse the presence of notches with ECG VF that generally have low amplitude waves in advanced stages, not analyzed in this study, which corresponds to what is known in the literature as "fine course" VF compared to " large course" VF[16].

Notches in the sinusoidal complexes appeared in sustained but not in non sustained VF, therefore, notches in the sinusoidal traces of ventricular fibrillation could be a predictor of sustained VF and may also be the electrocardiographic pattern of activation of essential structures in the VF maintenance. This hypothesis is reinforced with the observation of a case with delayed end after shock (Fig 5).

Krummen determined that sustained but not self-limiting VF was characterized by greater rotor stability . In addition, rotor exhibited greater surface ECG variation during VF than focal sourced due to wavebreak, secondary rotors and meander[17].

This variability in the amplitude of ECG waves could be due to the presence of higher amplitude waves over which others of lower amplitude are superimposed, producing the appearance of notches described in our study.

At an experimental level, Li et al [18]proved the following: long-duration ventricular fibrillation exhibits 2 distinct organized states, one potentially arising focally in the Purkinje system and the other potentially arising from a stable re-entrant circuit near the apical left ventricular endocardium. Could notches be the electrocardiographic manifestation of these components? . In this way, in figures presented by Masuda[19], sustained and non-sustained VF is shown ; the Purkinje firing may be responsable for the morphologies of notches in sustained VF , which is not present in non sustained VF.

Conclusions:

1.The ECG of the onset of induced VF in patients with CAD and BS shows QRS morphologies that could have their origin in the moderate band of the right ventricle, the posteromedial papillary muscle and right ventricle outflow tract.

2. In patients with coronary arterial disease, Wiggers' tachysystolic stage of VF could be related to the activation of posterior papillary muscle and in patients with Brugada, related to activation of RVOT

3. There are no specific fibrillatory structures for every VF etiology. However there is a predominance of activation in RVOT in Brugada Syndrome and papillary muscles in ischemic cardiomyopathy.

4. The notches in the sinusoidal traces of ventricular fibrillation could be a predictor of sustained VF. and in addition notches could also be the electrocardiographic pattern of activation of essential structures in the VF maintenance.

Appendice : Method for approach of VF ECG

1. Evaluate the first complexes that trigger VF: Triggers
2. Delimit a first stage with defined QRS complexes: Tachysystolic stage.
3. Define the moment when QRS complexes are not distinguished and sinusoidal waves appear: Sinusoidal stage.
4. Determine the ECG leads in which the sinusoidal waves are low amplitude with notches.
5. Look for QRS complexes between both Tachysystolic and Sinusoidal stages: Transition complexes.

Limitations:

VF is more complex than is apparent ECG and there remain other more accurate ways to define VF stages.

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Figure legends:

Fig1: Coupling Intervals and morphologies of triggers.

Fig 2: Morphology in coronary arterial disease in tachysystolic stage

Fig 3: Morphology in coronary arterial disease in tachysystolic stage

Fig 4 : Morphology in Brugada Syndrome in tachysystolic stage

Fig 5: Notches in sinusoidal stage.

Fig 6: Narrow complex in transition

Fig 7.1: Elevated J point in transition

Fig 7.2 : Elevated J point in transition

Fig 8 : Elevated J point in transition

Fig1: Coupling Intervals and morphologies of triggers. CD: coronary arterial disease. BS: Brugada Syndrome.

Case	S-PVC1 ms	PVC1-PVC 2 ms	QRS PVC1 ms	Probable Origin	QRS PVC2 ms	Probable Origin
CAD1	148	106	148	PPM	160	PPM
CAD 2	267	263	229	IR	222	MB
CAD 3	266	234	155	PPM	155	PPM
CAD 4	97	104	197	MB	193	MB
CAD 5	244	293	194	MB	198	PPM
CAD 6	271	207	187	MB	196	MB
CAD 7	297	244	148	MB	150	PPM
CAD 8	310	248	240	IR	271	MB
CAD Mean	237	212	187		189	
CAD SD	75	70	35		32	
BG 1	229	158	160	RVOT	159	MB
BG 2	256	206	217	IR	199	MB
BG 3	296	226	172	MB	149	MB
BG 4	274	235	243	RVOT	183	RVOT
BG 5	240	225	196	RVOT	181	RVOT
BG 6	264	246	239	IR	252	IR
BG 7	381	228	228	IR	181	MB
BG Mean	277	217	207		186	
BG SD	50	28	32		33	

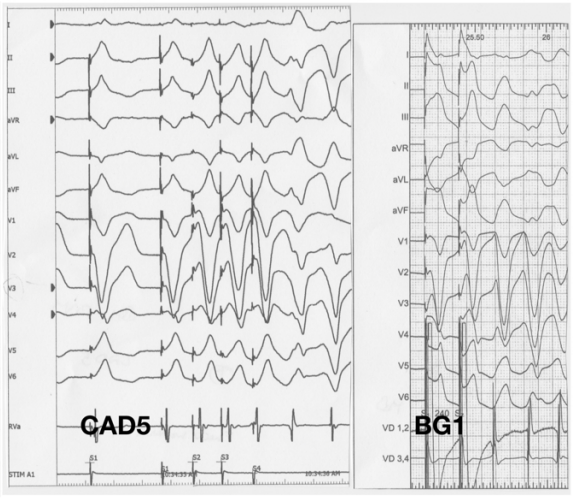

Fig 2: Morphology in coronary arterial disease in tachysistolic stage

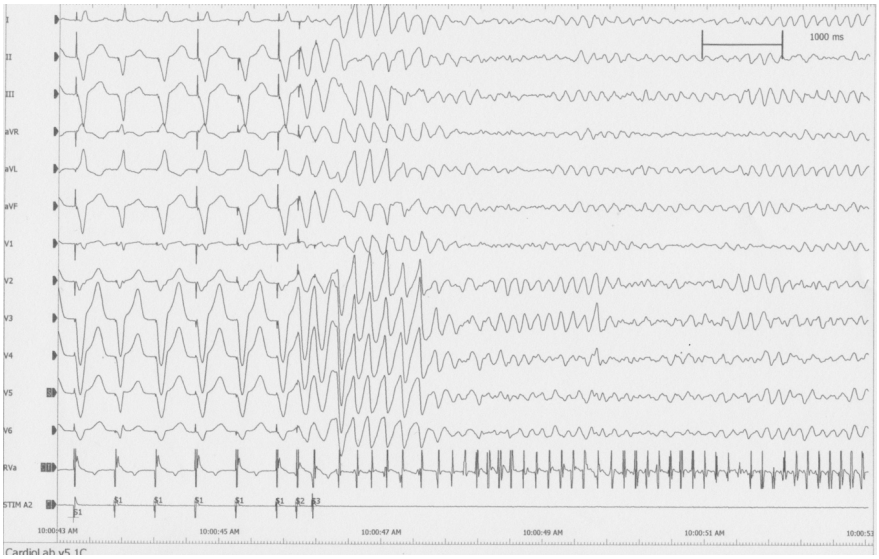

Fig 3: Morphology in coronary arterial disease in tachysistolic stage

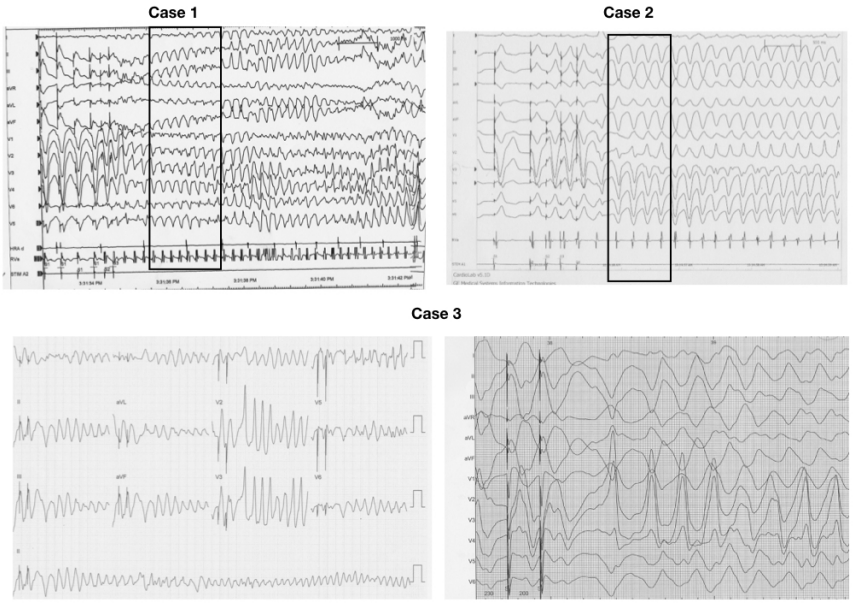

Fig 4 : Morphology in Brugada Syndrome in tachysistolic stage

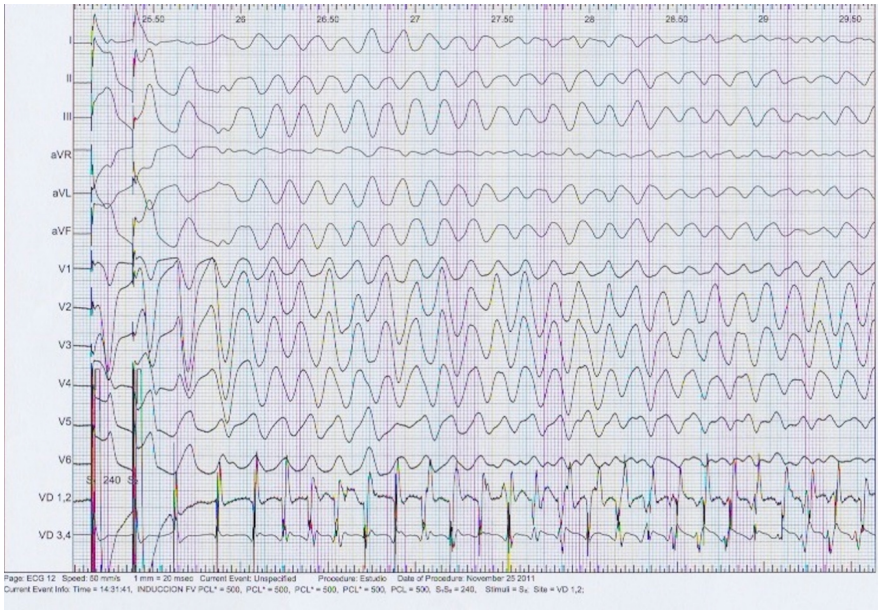

Fig 5: Notches in sinusoidal stage.

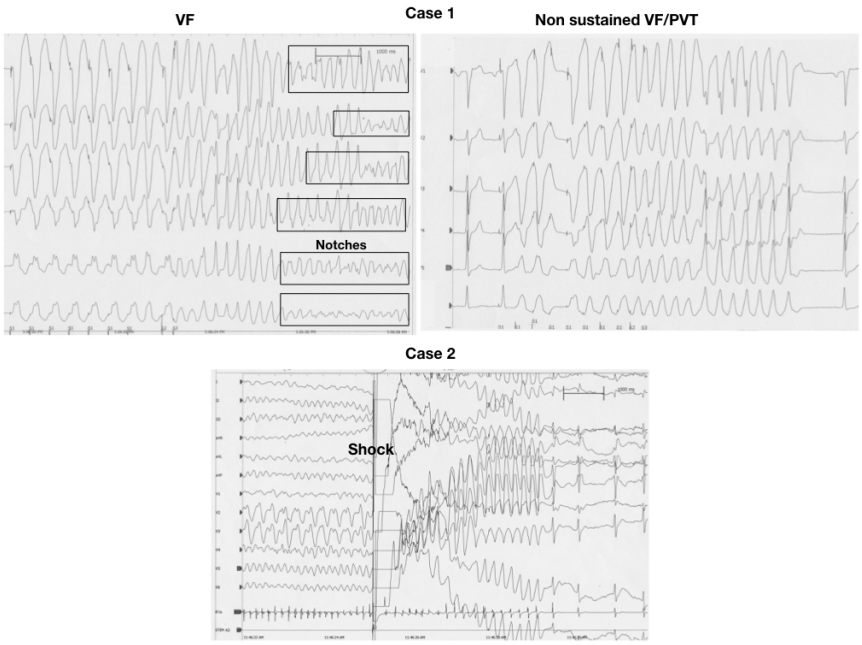

Fig 6: Narrow complex in transition

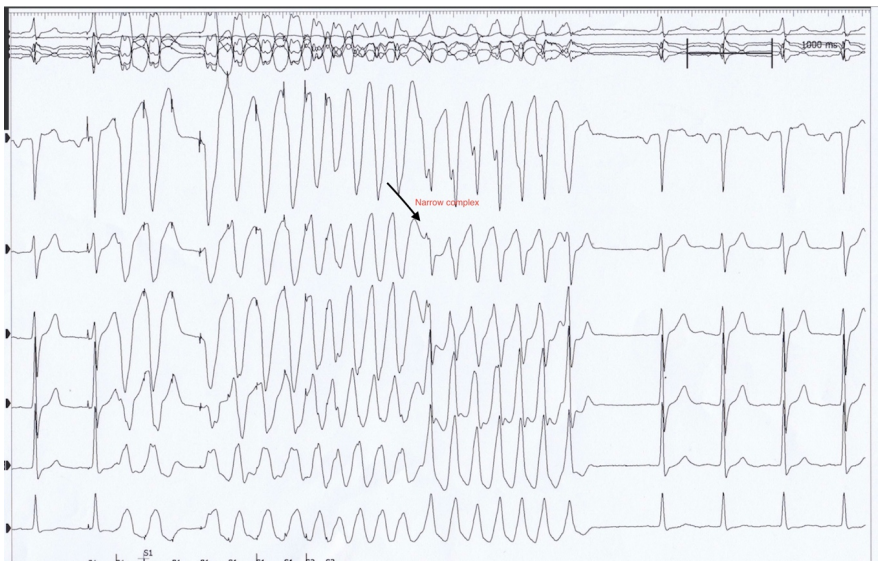

Fig 7.1 : Elevated J point in transition

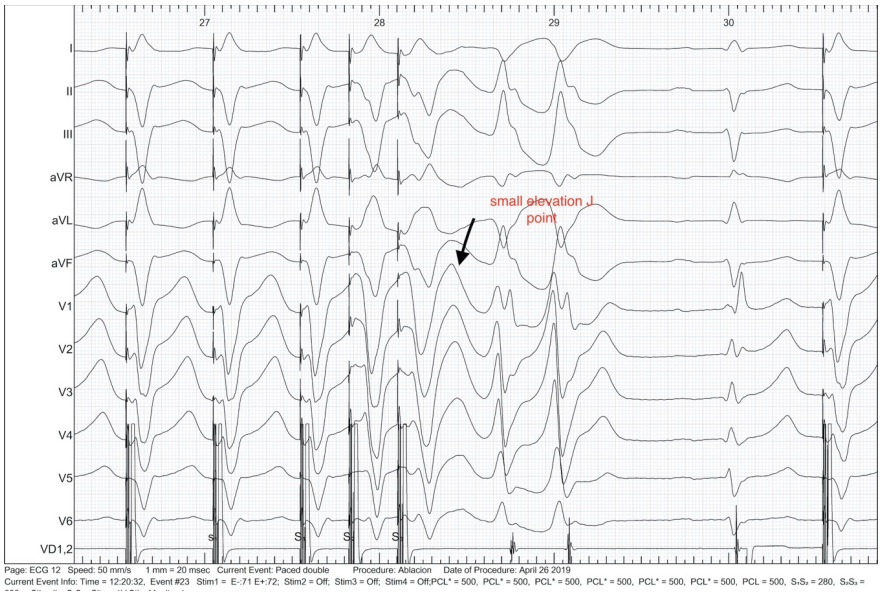

Fig 7.2 Elevated J point in transition

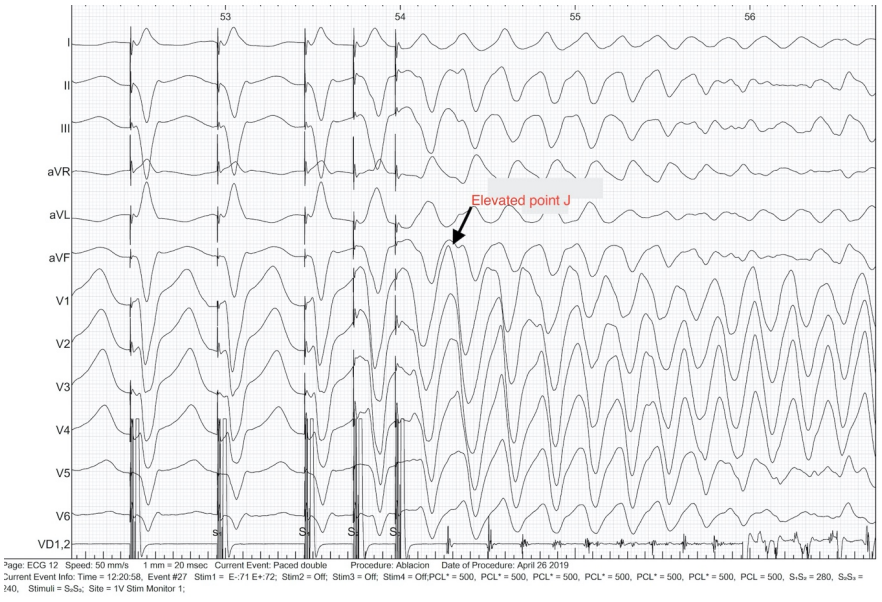

Fig 8 : Elevated J point in transition