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ORIGINAL RESEARCH

Clinical Presentation of Sustained Monomorphic Ventricular Tachycardia Without Cardiac Arrest

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BACKGROUND: "Palpitations" are one of the most common complaints prompting medical attention. Textbooks of medicine and cardiology as well as guideline documents and position papers describe palpitations as a common symptom of ventricular tachycardia (VT). However, data to support this description are lacking. The aim of our study was to evaluate the symptomatology of sustained monomorphic VT with emphasis on the prevalence of palpitations.

METHODS AND RESULTS: Consecutive patients presenting to our center with a first event of a regular sustained monomorphic VT (n=59) or a regular supraventricular tachycardia (SVT; n=109) between January 2012 and September 2019 were interviewed regarding their symptoms during the arrhythmic event. We included only patients with a first arrhythmic event to avoid the influence of previous medical encounters on our patients' terminology. As expected, patients with VT were older (age 68.8±13.6 versus 52.6±16.8 years; $P<0.001$), more often of male sex (94.9% versus 37.6%; $P<0.001$), had lower left ventricular ejection fraction (37±11% versus 59±2%, $P<0.001$) and more comorbidities (87.6% versus 40.5%; $P<0.001$) compared with patients with SVT. Importantly, even though the heart rate upon presentation did not differ between the 2 groups (165±26 beats/min during VT versus 171±32 beats/min during SVT; $P=0.16$), symptomatology differed significantly; specifically, palpitations were reported in only 8.8% of VT patients, compared with 90.7% of SVT patients ($P<0.001$). Common symptoms in the VT group included chest pain (64%), dyspnea (21%), and dizziness (26%).

CONCLUSIONS: Despite similar heart rate, patients with VT rarely report having palpitations, whereas patients with SVT do so commonly. This finding may assist with decision making in patients reporting palpitations in whom an ECG tracing is not available.

Key Words: palpitations ■ symptoms ■ ventricular tachycardia

> *"Listen to your patient, he is telling you the diagnosis."*
> *William Osler, 1849-1919.*

The symptom of "palpitations" is one of the most common complaints prompting medical attention. Yet the exact mechanism leading to palpitations during a cardiac rhythm disturbance is uncertain and can be related to diverse sensory input and brain interpretation of changes in cardiac contractility and rhythm.[1–4]

Sustained monomorphic ventricular tachycardia (VT) is the most common arrhythmic cause of out-of-hospital cardiac arrest but may also be hemodynamically tolerated, presenting as a fast and regular tachycardia. Yet, surprisingly little is known about the symptoms caused by sustained monomorphic

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CLINICAL PERSPECTIVE

What Is New?

- This is the first study investigating the symptomatology of patients presenting with a sustained monomorphic ventricular tachycardia without cardiac arrest.
- Contrary to what is repeatedly quoted in important cardiology textbooks and position papers, the vast majority of these patients do not report having palpitations.

What Are the Clinical Implications?

- Patients reporting rapid palpitations without ECG-documented arrhythmias should not be assumed to have ventricular tachycardia.

Nonstandard Acronyms and Abbreviations

SVT	supraventricular tachycardia

VT *not* causing cardiac arrest. Despite the paucity of data, classic documents of medicine and cardiology generally claim that "rapid palpitations" are the characteristic symptom of hemodynamically stable sustained monomorphic VT.[5–8] Here, we present the first prospective study, to our knowledge, evaluating the symptomatology of patients presenting with sustained monomorphic VT not leading to cardiac arrest, clearly demonstrating that palpitations are reported only by a minority of patients.

METHODS

The data that support the findings of this study are available from the corresponding author upon reasonable request. This prospective study was conducted between January 2012 and September 2019 at the Tel Aviv Medical Center, Tel Aviv, Israel. Patients presenting with a first event of sustained tachycardia of regular rhythm were studied. The study group consisted of consecutive patients presenting with a first event of sustained monomorphic VT without cardiac arrest. They were compared with consecutive patients presenting with a sustained, regular, supraventricular tachycardia (SVT), including atrial tachycardia, atrioventricular nodal reentry, and atrioventricular reentry tachycardia. We studied only "naïve" patients (ie, patients with a first event of sustained regular tachyarrhythmia) to avoid potential "indoctrination" of specific terminology for our patients' description of their symptoms during previous encounters with physicians at our center or elsewhere.

To be included, the index tachycardia had to last at least 5 minutes. Patients with implanted defibrillators were included only if the arrhythmia was not treated by their device (because it was slower than the programmed VT detection rate). Patients in whom a defibrillator was implanted as a "secondary prevention" (ie, following cardiac arrest) were excluded from the study.

Participating patients were asked to fill in a short anonymous questionnaire describing their symptoms at the time of the event (Table S1). Data collection included 12-lead ECG recordings from the time of the event, baseline clinical, laboratory, and echocardiographic findings as well as the applied therapies. All data were collected while maintaining full confidentiality, and the study was approved by our local institutional review board.

Designation to the VT and SVT groups was done according to the interpretation of the ECG recordings by senior electrophysiologists (S.V., R.R.), who were blinded to the questionnaire results and following accepted definitions.[6,9] When the diagnosis was in doubt, patients underwent clinically indicated electrophysiologic studies.

Statistical Analysis

Categorical variables were reported as numbers and percentages, and continuous variables were reported as means and standard deviations. Continuous variables were tested for normal distribution using histograms and Q-Q plots and compared between groups using independent samples t-test or Mann-Whitney test, as appropriate. Categorical variables were compared using the chi-square test or Fisher's exact test. Unadjusted logistic regressions were used to evaluate the association between baseline characteristics and palpitations at presentation. Odds ratios and 95% CIs were reported. Adjusted logistic regressions were used to further evaluate the association between VT and palpitations at presentation after adjusting for (1) age and sex and (2) all baseline characteristics excluding VT (fully adjusted model). Missing data were handled with multiple imputations for the fully adjusted model. A 2-tailed $P<0.05$ was considered as statistically significant. All statistical analyses were performed with SPSS (SPSS Statistics for Windows, Version 22.0. IBM Corp., Armonk, NY).

RESULTS

The study cohort included 168 patients: 59 with sustained monomorphic VT and 109 with SVT (Table 1).

Table 1. Baseline Characteristics and Presentation

Baseline Characteristics	All	VT Group	SVT Group	*P* Value
n	165	58	107	
Age, y	57 (±18)	68 (±13)	52 (±17)	<0.001
Male sex	96 (58.2)	55 (94.8)	41 (38.3)	<0.001
Hypertension	67 (45.6)	35 (79.5)	32 (31.1)	<0.001
Diabetes mellitus	24 (16.6)	16 (37.2)	7 (7.8)	<0.001
Ischemic heart disease	54(35.1)	46 (83.6)	8 (8.1)	<0.001
Atrial fibrillation	17 (11.6)	12 (26.7)	5 (4.9)	<0.001
Heart failure	31 (20.9)	29 (63)	2 (2)	<0.001
%LVEF	48 (±14)	37 (±11)	58 (±8)	<0.001
VHD*	26 (27.7)	13 (33.3)	13 (24.6)	0.30
Cardiomyopathy#	5 (3.4)	4 (8.7)	1 (1)	0.03
Anticoagulation therapy	13 (10.1)	10 (27.8)	3 (3.2)	<0.001
Aspirin	46 (34.6)	27 (75)	19 (19.8)	<0.001
Beta blockers	86 (60.1)	36 (87.8)	50 (49)	<0.001
ACEI/ARB/ARNI	48 (36.6)	28 (75.7)	20 (21.3)	<0.001
Antiarrhythmics	16 (12.1)	4 (10.3)	12 (12.9)	0.77
Heart rate, beats/min	168 (±30)	163 (±25)	171 (±32)	0.94
SBP, mm Hg	109 (±30)	104 (±19)	111 (±36)	0.23
Symptoms				
Palpitations	103 (62.4)	5 (8.6)	98 (91.6)	<0.001
Chest pain	36 (21.8)	25 (43.1)	11 (10.3)	<0.001
Chest discomfort	14 (8.5)	12 (20.7)	2 (1.9)	<0.001
Dyspnea	15 (9.1)	12 (20.7)	3 (2.8)	<0.001
Dizziness	25 (15.2)	15 (25.9)	10 (9.3)	0.005
Syncope	10 (6.1)	9 (15.5)	1 (0.9)	<0.001

Data presented as mean (±standard deviation) or n (%). ACEI indicates angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; ARNI, angiotensin receptor neprilysin inhibitor; LVEF, left ventricular ejection fraction; SBP, systolic blood pressure; SVT, supraventricular tachycardia; and VT, ventricular tachycardia.

*More than mild valvular heart disease.

#Hypertrophic, arrhythmogenic, etc.

As expected, patients with VT were older (68.8±13.6 versus 52.6±16.8 years; *P*<0.001), more often of male sex (94.9% versus 37.6%; *P*<0.001) compared with patients with SVT. Additionally, the VT group had impaired left ventricular function (37±11% versus 59±2%; *P*<0.001) and more commonly suffered from comorbidities. Overall (VT and SVT combined), a statistically significant difference was shown in symptomatology between men and women; 90% of women but only 42.1% of men reported having palpitations as their presenting symptom (Tables 2–4). In a logistic regression analysis, there was no interaction between the presence of VT and sex (*P*=0.471).

Of note, heart rate upon presentation did not differ significantly between the 2 groups (165±26 beats/min during VT versus 171±32 beats/min during SVT; *P*=0.16). Symptomatology, however, significantly differed: While 90.7% of patients presenting with SVT reported having palpitations, only 8.8% of the patients with VT reported this symptom (*P*<0.001). Chest pain was the chief complaint among patients with VT (reported by 64%, of which 71% was angina-type chest pain). In contrast, only 11% of the SVT group had chest pain. Other common symptoms in the VT group included presyncope (27%) and shortness of breath (21%) (Figures 1 and 2,). Three disagreements between the electrophysiologists examining the ECG tracings required an electrophysiological study in 3 patients. All 3 were diagnosed with a VT (none had palpitations). There was no correlation between the type of symptoms and the heart rate. Similarly, there was no correlation between the symptomatology and the underlying heart disease (eg, nonischemic cardiomyopathy, idiopathic VT). Atrioventricular dissociation was found in the ECG tracings of 4 patients with VT, palpitations were reported in 3 of them.

DISCUSSION

Just as the first words used for the description of symptoms in acute myocardial infarction are *chest*

Table 2. Symptom Prevalence According to Sex Stratified by Arrhythmia

Symptoms	Male n=96	Female n=69	*P* Value
Chest pain	31 (32.3)	5 (7.2)	<0.001
VT	25 (45.5)	0 (0)	0.25
SVT	6 (14.6)	5 (7.6)	0.33
Chest discomfort	11 (11.5)	3 (4.3)	0.11
VT	11 (20)	33.3 (1)	0.51
SVT	0 (0)	4.5 (3)	0.28
Dyspnea	11 (11.5)	4 (5.8)	0.21
VT	11 (20)	1 (33.3)	0.51
SVT	0 (0)	3 (4.5)	0.29
Palpitations	40 (41.7)	63 (91.3)	<0.001
VT	4 (7.3)	1 (33.3)	0.24
SVT	36 (87.8)	62 (93.9)	0.3
Dizziness	21 (21.9)	4 (5.8)	0.004
VT	15 (27.3)	0 (0)	0.56
SVT	6 (14.6)	4 (6.1)	0.18
Syncope	9 (9.4)	1 (1.4)	0.04
VT	9 (16.4)	0 (0)	>0.999
SVT	0 (0)	1 (1.5)	>0.999

Data presented as n(%); patients may present with >1 symptom. SVT indicates supraventricular tachycardia; and VT, ventricular tachycardia.

pain in a classic textbook of cardiology,[5] the first word used for the description of a VT event is *palpitations*.[5] Similarly, palpitations are the first symptom

Table 4. Association of Palpitations With VT and SVT According to Age

	Age Below Median (≤58 years)	Age Above Median (>58 years)	*P* Value
Palpitations, % (n)	88.2 (75)	35 (28)	<0.001
VT	25 (3)	4.3 (2)	0.05
SVT	98.6 (72)	76.5 (26)	<0.001

SVT indicates supraventricular tachycardia; and VT, ventricular tachycardia.

associated with VT, according to the 2017 American Heart Association/American College of Cardiology/Heart Rhythm Society Guideline for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death.[10] Finally, the term *VT* is repeatedly mentioned as *a likely cause of palpitations* in the 2011 European Society of Cardiology position paper on palpitations.[7] However, little to no evidence is presented to support these statements. In fact, our study suggests that all these assumptions are incorrect. Specifically, despite similar heart rate of VT and SVT events, palpitations are *less* likely to be reported when the cause of the arrhythmia is VT.

The mechanism by which arrhythmias cause palpitations is not entirely clear. Different symptoms can be attributed to different pathophysiologic pathways, including hypercontractile states, slow or fast heart

Table 3. Association of Palpitations at Presentation With Baseline Characteristics in General and With VT in Particular

Baseline Characteristics	Unadjusted ORs		OR Adjusted for Age and Sex		OR Adjusted for All Other Baseline Characteristics	
	OR (95% CI)	*P* Value	OR (95% CI)	*P* Value	OR (95% CI)	*P* Value
VT	0.01 (0.01–0.03)	<0.001	0.02 (0.01–0.07)	<0.001	0.06 (0.04–0.11)	<0.001
Age	0.92 (0.89–0.95)	<0.001	0.92 (0.88–0.97)	0.001	0.91 (0.84–0.98)	0.018
Male sex	13.4 (5.10–30.0)	<0.001	3.14 (0.79–12.4)	0.100	2.22 (0.36–13.69)	0.391
Heart rate	1.01 (0.99–1.02)	0.170			0.98 (0.93–1.03)	0.409
Blood pressure	1.01 (0.99–1.03)	0.190			0.99 (0.94–1.05)	0.898
Hypertension	0.16 (0.08–0.34)	<0.001			0.71 (0.01–36.0)	0.851
Diabetes mellitus	0.20 (0.10–0.51)	0.001			1.99 (0.07–61.17)	0.672
Ischemic heart disease	0.04 (0.02–0.09)	<0.001			1.67 (0.11–25.97)	0.705
Atrial fibrillation	0.18 (0.06–0.54)	0.002			0.13 (0.01–55.65)	0.456
Heart failure	0.04 (0.01–0.14)	<0.001			7.68 (0.16–381)	0.296
LVEF	1.15 (1.10–1.20)	<0.001			1.06 (0.94–1.21)	0.325
Valvular heart disease	0.14 (0.05–0.41)	<0.001			0.77 (0.13–4.77)	0.782
Cardiomyopathy	0.10 (0.01–0.91)	0.040			0.41 (0.02–9.55)	0.580
Anticoagulation therapy	0.17 (0.05–0.59)	0.005			2.56 (0.01–137)	0.733
Aspirin	0.07 (0.03–0.17)	<0.001			0.16 (0.01–4.10)	0.252
Beta blockers	0.23 (0.10–0.52)	<0.001			1.85 (0.10–33.97)	0.658
ACEI/ARB/ARNI	0.19 (0.09–0.42)	<0.001			3.83 (0.09–167)	0.460
Antiarrhythmics	0.41 (0.14–1.18)	0.100			1.02 (0.04–30.0)	0.990

ACEI indicates angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; ARNI, angiotensin receptor neprilysin inhibitor; LVEF, left ventricular ejection fraction; OR, odds ratio; and VT, ventricular tachycardia.

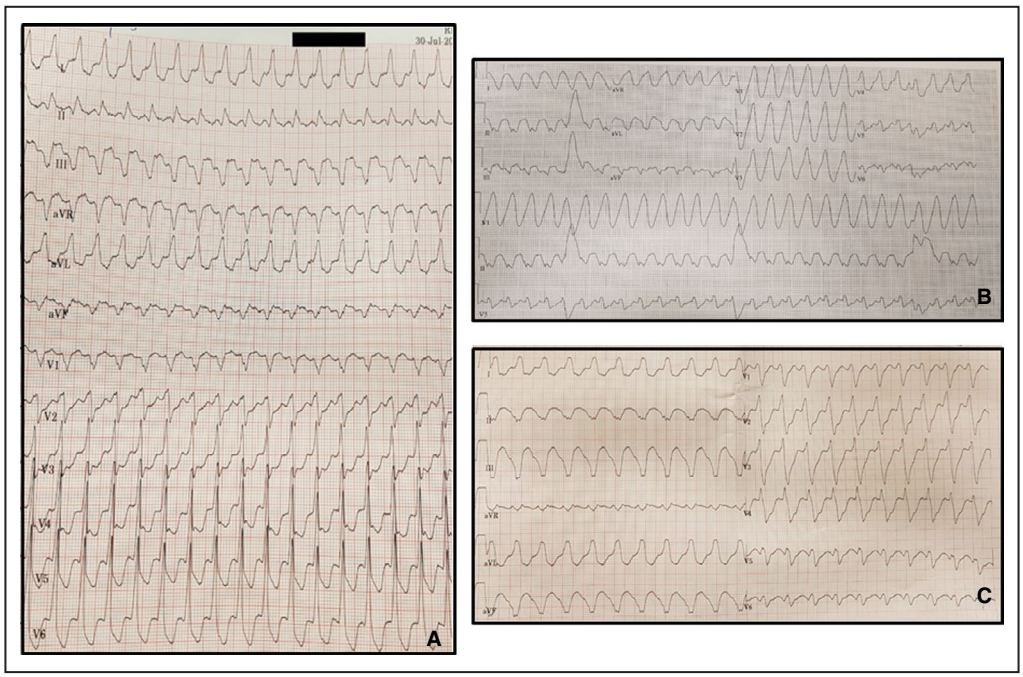

Figure 1. Examples of monomorphic VTs in our study.
Examples of 3 patients presenting with VT of different heart rate and origin, none of whom presented with palpitations. **A**, Cycle length 360 milliseconds (166 bpm), right bundle branch block (RBBB), superior axis, septal left ventricle (LV). **B,** Cycle length 360 milliseconds (166 bpm), left BBB, northwest axis, lateral base LV. **C,** Cycle length 440 milliseconds (136 bpm), RBBB superior axis, inferoseptal LV. VT indicates ventricular tachycardia.

rate and structural heart diseases.[7] Systemic illnesses and psychosomatic disorders may also affect the ways arrhythmias are perceived or reported.[7] The sensory input from the heart is complex—nociceptors were never explicitly proven to reside within the myocardium[2]—and different sensory receptors found

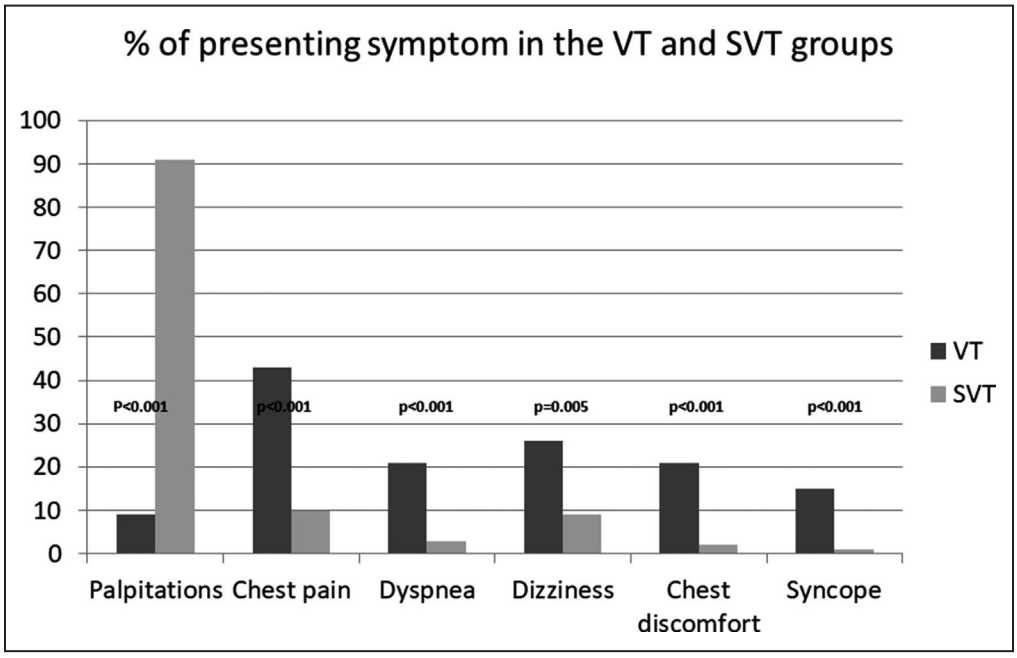

Figure 2. Presenting symptoms. Percentage of patients presenting with palpitations, chest pain, dyspnea, and the like in the VT vs the SVT group.
SVT indicates supraventricular tachycardia; and VT, ventricular tachycardia.

in extracardiac structures or within the pericardium or the myocardium have been suggested.[1,4] Additionally, multiple brain centers are involved in the interpretation of these sensory stimuli.[11] To further complicate this, it has been shown that cardiac maladies may produce unexpected symptomatology. For example, the degree or the duration of anginal pain is a poor indicator for the severity of coronary artery disease,[12] and clinically significant arrhythmias (such as atrial fibrillation and nonsustained VT) may not produce symptoms in many patients.[13]

A possible explanation for the divert symptomatology of patients with VT and SVT could be related to the baseline cardiac dysfunction of patients with VT and their high prevalence of comorbidities consequently making their myocardium more susceptible to ischemia, causing them to experience chest pain during rapid heart rate. A fundamental difference in myocardial oxygen consumption between these 2 patient groups could also contribute to different perceptions. In a well-conducted experiment, Badeer and Feisal[14] demonstrated that in a canine lung and heart model maintained at similar cardiac output and arterial pressure, atrial tachycardia and VT of similar rates caused an 11.5% versus 50% increase in myocardial oxygen consumption, respectively. The authors explained their findings by demonstrating a prolonged systolic period during VT attributable to an abnormal ventricular depolarization, causing an increase in the time-tension index (known to be an important determinant of myocardial oxygen consumption).[15] This observed significant difference in O_2 consumption might induce an ischemic pain, which may then mask the otherwise expected palpitations.

Another possible explanation for the occurrence of palpitations in some patients with VT can be attributed to atrioventricular dissociation. This finding is probably the result of the atria contracting against a closed atrioventricular valve, a phenomenon known to occur also during atrioventricular nodal reentrant tachycardia and ventricular pacing. Indeed, 3 of 4 patients with VT in our study in whom atrioventricular dissociation was found did experience palpitations.

The *Bates' Guide to Physical Examination and History Taking* encourages us to "ask open-ended questions and listen carefully and patiently to the patient's story." By doing so, we may "avoid mistakes in data collection or interpretation."[16] Arrhythmias are generally perceived as an "out-of-order heart rhythm," and as such are *expected* to cause palpitations. Consequently, it is plausible that physicians tend to designate the patient's complaints as "palpitation" when they hold in their hand an ECG recording of a clear-cut arrhythmia.

Our study is limited by the significant differences in sex, age, and comorbidities between our 2 study groups. Specifically, the overwhelming predominance of men in our VT group might trigger the question that our findings could be the result of sex-induced rather than arrhythmia-induced difference in symptomatology. Similarly, the age difference between the VT and the SVT group may have influenced patients' symptomatology. Nevertheless, in our entire study population (VT and SVT combined), palpitations occurred in 42.1% of male patients, indicating that men can suffer from this complaint (Table 2). Regardless of the sex effect, we show here that, as a group, patients who suffer from a first event of sustained monomorphic VT only rarely suffer from palpitations. This observation contradicts prevalent views expressed in leading textbook and guideline documents. The implications of these findings are 2-fold: First, patients reporting rapid palpitations without ECG-documented arrhythmias should *not* be assumed to have VT. Second, it shows us that even in this era of ever-growing technology, we should still find the time and patience to listen.

ARTICLE INFORMATION

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Disclosures

None.

Supplementary Material

Table S1

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SUPPLEMENTAL MATERIAL

Table S1. Arrhythmia Questionnaire.

1. What was the reason for your referral? (open question)
2. Did you suffer from: palpitations / chest pain / chest discomfort / shortness of breath / weakness / dizziness
3. In case you lost consciousness, what were your symptoms prior to losing consciousness? palpitations / chest pain / chest discomfort / shortness of breath / weakness / dizziness
4. Prior medical history.
5. Regular medications.
6. Investigations.
7. ECG recording.