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Confounding factors leading to misdiagnosing ventricular tachycardia as supraventricular in the emergency room

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ABSTRACT

Studies conducted during the last 50 years have proposed electrocardiographic criteria and algorithms to determine if a wide QRS tachycardia is ventricular or supraventricular in origin. Sustained ventricular tachycardia is an uncommon reason for consultation in the emergency room. The latter and the complexity of available electrocardiographic diagnostic criteria and algorithms result in frequent misdiagnoses. Good hemodynamic tolerance of tachycardia in the supine position does not exclude its ventricular origin. Although rare, ventricular tachycardia in patients with and without structural heart disease may show a QRS duration <120 ms. Interruption of tachycardia by coughing, carotid sinus massage, Valsalva maneuver, or following the infusion of adenosine or verapamil should not discard the ventricular origin of the arrhythmia. In patients with regular, uniform, sustained broad QRS tachycardia, the presence of structural heart disease or A-V dissociation strongly suggest its ventricular origin. Occasionally, ventricular tachycardia can present with AV dissociation without this being evident on the 12-lead ECG. Cardiac auscultation, examination of the jugular venous pulse, and arterial pulse palpation provide additional clues for identifying A-V dissociation during tachycardia. This paper does not review the electrocardiographic criteria for categorizing tachycardia as ventricular but rather why emergency physicians misdiagnose these patients.

1. Introduction

Hein Wellens, in March 1977, submitted to the American Journal of Cardiology a paper on *the value of the electrocardiogram in the differential diagnosis of a tachycardia with a widened QRS complex*. This article was based on studies conducted at the Wilhelmina Gasthuis of Amsterdam. The journal published this paper in January 1978, when Wellens had already moved to the University Maastricht, where he stayed until his retirement in the year 2000 [1]. In the alluded paper, Wellens and his co-workers proposed electrocardiographic criteria to differentiate ventricular tachycardia (VT) from supraventricular tachycardia (SVT) with aberrant conduction. Wellens retired in 2000 but never stepped down. In 2001, Wellens published a review paper on diagnosing tachycardia with a broad QRS complex [2]. Moreover, a simple search in PubMed shows dozens of articles by Hein Wellens on electrocardiographic aspects of broad-QRS tachycardia or ventricular arrhythmias, published from 2000 to 2020, when he passed away. In addition, during the last two decades of his life, Wellens was an active teacher, often lecturing on the value of the 12-lead electrocardiogram (ECG) in the diagnosis, risk stratification, and therapeutic approach of patients with ventricular arrhythmias or at risk of sudden cardiac death. Our paper, written to honor the memory of Prof. Hein Wellens, intends to overcome the diagnostic difficulties faced by residents in the emergency room in patients with a broad QRS tachycardia.

2. Limitations of available criteria to decide the ventricular or supraventricular origin of a tachycardia

The correct diagnosis of the ventricular origin of tachycardia is not easy. Kashou et al. have recently reviewed the ECG-based criteria and algorithms proposed so far to differentiate ventricular from supraventricular tachycardia with a broad QRS complex [3].

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Abbreviations

ARVC	Arrhythmogenic right ventricular cardiomyopathy
AVID	Antiarrhythmics Versus Implantable Defibrillators
AVNRT	AV nodal reentry tachycardia
AVRT	Atrioventricular reciprocating tachycardia
bpm	beats per minute
CRT-D	Cardiac resynchronization therapy-defibrillator
ECG	Electrocardiogram
ICD	Implantable cardioverter defibrillator
IFLBB	Inferior fascicle of the left bundle branch
IV	Intravenous
LBBB	Left bundle branch block
LIFVT	Left inferior fascicular ventricular tachycardia
ms	milliseconds
NIVC	Normal intraventricular conduction
RBBB	Right bundle branch block
SFLBB	Superior fascicle of the left bundle branch
SVT	Supraventricular tachycardia
VT	Ventricular tachycardia

This comprehensive review discusses the presumed value of non-stepped electrocardiographic diagnostic criteria and structured algorithms as advocated by their proposers, as well as the limitations observed by subsequent evaluations by independent investigators when available [3].

Individual ECG criteria and algorithms have limitations in terms of sensitivity, specificity, and predictive values. Most importantly, they are difficult to remember unless used regularly or the emergency physician keeps in the pocket a note prompting the most relevant ECG signs (Table 1) [1–8]. Emergency room and general cardiology residents are not arrhythmia experts. However, they are the frontline physicians dealing with patients arriving at the emergency department with tachycardia.

Since arrhythmia specialists are not part of the medical staff of emergency rooms, errors in diagnosing the ventricular or supraventricular origin of tachycardia often occur. Thus, Dancy and co-workers showed that 99 physicians, 57 of whom were residents, repetitively misdiagnosed as supraventricular the broad QRS tachycardia of 24 patients with a sustained VT [9]. In this study, misdiagnoses often led to potentially dangerous therapies [9]. Stewart and co-workers found that 39% of episodes of VT were diagnosed as SVT at the emergency room and that this mistake resulted in the selection of risky therapeutic options [10] Errors occurred although an analysis of the 12-lead ECG during tachycardia would have strongly suggested a ventricular origin [10]. Herbert and his colleagues showed that three experienced emergency room physicians using the 4-step Brugada algorithm disagreed as to the ventricular or supraventricular origin of wide QRS tachycardias in 22% of ECG tracings [11]. The presentation of a regular broad QRS tachycardia in the emergency room is not a frequent event. Of 82,559 emergency department visits, only 27 were patients with a wide QRS tachycardia (0,03%) [11]. This justifies the difficulties of emergency physicians to remain competent in the identification of the ventricular origin of tachycardia.

Akhtar and his colleagues found that the first diagnosis was correct in only 39 of 122 patients with sustained VT (32%) [12]. However, considering a history of structural heart disease as indicative of VT in a patient with a broad QRS tachycardia would have resulted in a proper diagnosis in 95% of instances [7]. ECG diagnostic criteria available at that time would have led to the identification of VT in 92% of the patients, but emergency department doctors lacked the proper ability to use or remember these very specialized diagnostic tools [12].

Fig. 1 shows the 12-lead ECG of a hemodynamically stable broad QRS tachycardia misdiagnosed as SVT by 61% of a large group of cardiologists despite fulfilling ECG criteria suggesting a ventricular

Table 1
Most important non-stepped electrocardiographic criteria and structured algorithms to diagnose VT in broad QSR tachycardia [1–8].

non-staggered ECG criteria suggesting VT	•A-V dissociation •Capture & fusion beats •QRS concordance in chest leads •Positive QRS concordance in chest leads •QRS duration >140 ms in tachycardia with RBBB morphology •QRS duration >160 ms in tachycardia with LBBB morphology •Left axis deviation •Northwest QRS axis •Right axis deviation in tachycardia with LBBB morphology •Different QRS morphology during tachycardia compared to baseline pre-existing bundle branch block. •QRS morphologies in tachycardia with RBBB morphology: monophasic or biphasic QRS morphologies in V_1 with the initial peak taller than the subsequent crest • QRS morphologies in tachycardia with LBBB morphology: o initial *R*-wave >30 ms in V_1 or V_2 o QRS onset-to-nadir of the *S*-wave >60 ms in V_1 or V_2 o notching of the downstroke of *S*-wave in V_1 or V_2 o any Q-wave in V_6
4-step chest leads algorithm	• Step 1: absence of ***RS*** complexes in chest leads (VT) • Step 2: ***R***-to-nadir ***S*** ≥ 100 ms in chest leads (VT) • Step 3: A-V dissociation present (VT) • Step 4 morphology criteria for VT (VT) o ***R***, ***qR***, ***RS*** in V1 in RBBB tachycardia o ***R/S*** < 1 in V6 in RBBB tachycardia o ***QS***, ***qR*** in lead V6 in RBBB/LBBB tachycardia
4-step aVR algorithm	• Step 1: an initial ***R*** wave (VT) • Step 2: initial ***r*** or ***q*** waves >40 ms (VT) • Step 3: notching on downstroke of a predominantly negative QRS (VT) • Step 4: initial vs terminal 40 ms voltages [Vi/Vt] ≤1 (VT)
One-step lead II algorithm	• QRS onset-to-peak/nadir first wave (***R*** or ***S***) ≥50 ms (VT)

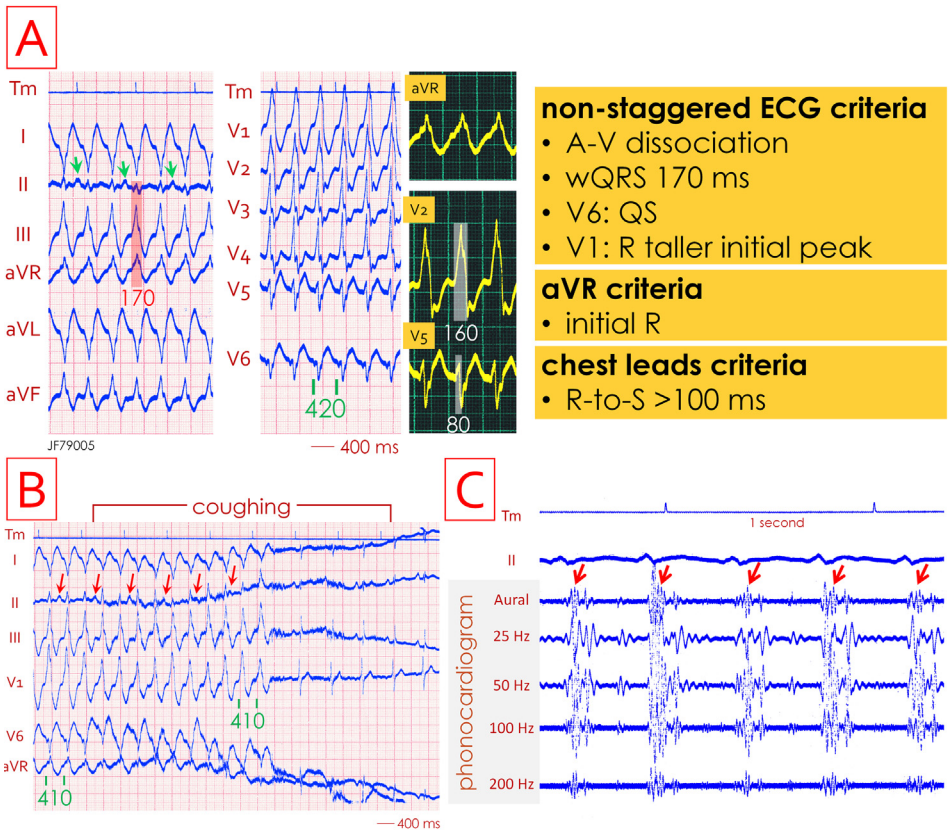

Fig. 1. Panel A: 12-lead ECG of an episode of well-tolerated sustained VT at a rate of 143 bpm. This ECG was presented to an audience of more than 200 cardiologists of diverse expertise during a European Society of Cardiology voting ECG session. Only 39% of attendees voted for VT. Other choices were: preexcited atrioventricular reciprocating tachycardia (AVRT) 21%, SVT in a patient with preexisting right bundle branch block (RBBB) 19%, SVT with RBBB aberrancy 17%, and not able to tell 4%. Theoretically, this was an easy quiz in which the classic criteria and the more recent algorithms indicated a ventricular origin for this wide QRS tachycardia. Green arrows point to ***P***-waves dissociated from the QRS complexes. The width of the ventricular complex is 170 ms and the frontal plane axis has a south-west direction ($\approx +150^{\circ}$). The QRS in lead V_1 exhibits a monophasic ***R***-wave configuration with the initial peak taller than the terminal tip. In lead V_6, the QRS has a ***QS*** morphology. In lead aVR, the ventricular complex shows a monophasic ***R***-wave, and in lead V_2 the interval from the onset of the ***R*** wave to the nadir of the ***S*** wave is 160 ms. This interval was only 80 ms in lead V_5. **Panel B**: vigorous coughing interrupted the tachycardia of this patient. This finding was an additional confounding factor to misdiagnose this VT as SVT in our emergency room. The red arrows point to the sinus ***P***-waves dissociated from the QRS complexes. During sinus rhythm, the patient exhibited an incomplete RBBB and abnormal ***Q***-waves in leads III and aVF secondary to a past episode of myocarditis. **Panel C**: phonocardiogram obtained during VT showing the changing intensity of the first heart sound (arrows).

Table 2
Confounding factors leading to a wrong diagnosis in patients with a sustained well tolerated regular and uniform VT.

clinical	• Hemodynamic stability • Lack of evidence of structural heart disease • Another previous misdiagnosis as SVT • Previous history of episodes of SVT
electrocardiographic	• Rate <100 bpm • QRS width during tachycardia <120 ms • Lack of electrocardiographic documentation of A-V dissociation during tachycardia even when the latter is present • QRS width during tachycardia narrower than that observed during sinus rhythm • Presence of pre-existing bundle branch block during sinus rhythm with a QRS configuration similar or identical to that observed during tachycardia • Presence of pre-existing bundle branch block during sinus rhythm that is ipsilateral to the bundle branch block configuration exhibited by the RS complexes during tachycardia • Slurring of the initial QRS forces mimicking a delta wave morphology • A triphasic QRS in V_1 with the terminal peak taller than the initial crest in tachycardia with a RBBB morphology • A relatively slow tachycardia rate
manoeuvres	• Interruption of tachycardia o by coughing o by carotid sinus massage o by Valsalva maneuver o by adenosine o by verapamil
undue influences	• opinion of other physicians • opinion of the patient

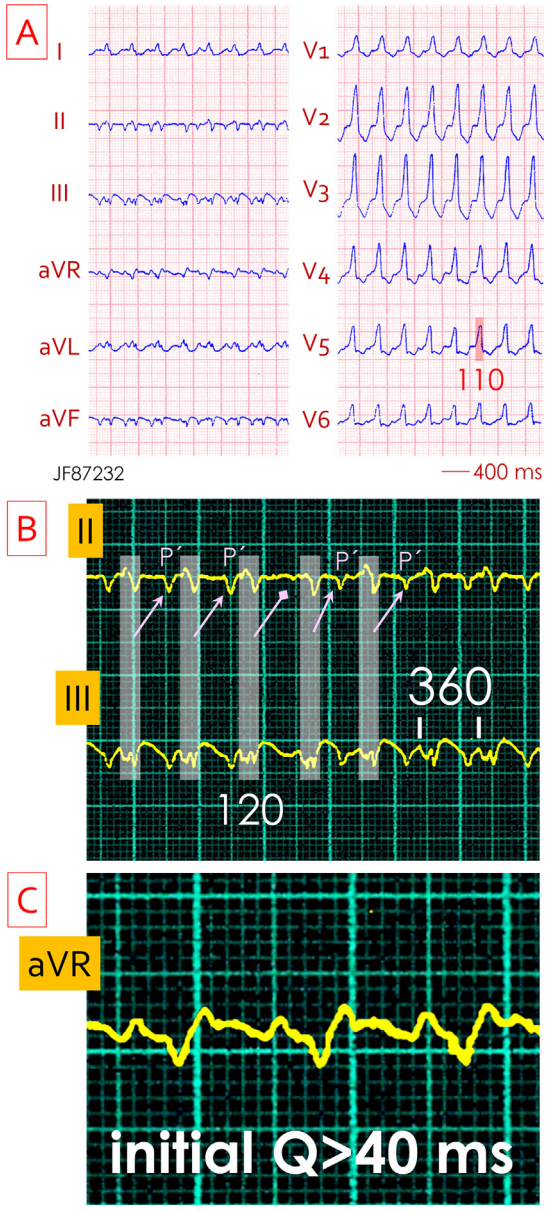

Fig. 2. Panel A: 12-lead ECG of an episode of well-tolerated sustained VT diagnosed as SVT in the emergency room despite the presence of ECG signs suggestive of a ventricular origin. Note that QRS complexes were all positive in the chest leads; however, we cannot consider this as an example of QRS-concordance since the ventricular complexes in leads V_4, V_5 and V_6 exhibit a tiny terminal ***s***-wave. The interval between the onset of the ***R***-wave and the nadir of the tiny ***s***-wave in lead V_5 is 110 ms. **Panel B:** detail of leads II and III during VT. Note that the width of the QRS is 120 ms; the lack of a very wide QRS complex during tachycardia and its hemodynamic stability were the reasons for its misdiagnosis. The emergency physicians did not notice the presence of a 2nd degree V-A block during tachycardia. Note that a retrograde ***P***-wave was lacking after the 3rd QRS complex. The 4th ventricular complex is again retrogradely conducted to the atria (with a shorter V-A conduction time). The presence of a 2nd degree V-A block during a wide QRS tachycardia eliminates the possibility of atrial tachycardia and AVRT using an accessory pathway. This finding does not exclude AV nodal reentry tachycardia but makes it very unlikely. The R-R interval during tachycardia is 360 ms corresponding to ≈ 165 bpm. **Panel C:** detail of lead aVR during VT. Note that there was an initial broad ***Q***-wave (width slightly above 40 ms). In addition, the initial versus the terminal voltages in aVR (Vi/Vt) is ≈ 1. Applying the latter two criteria is often difficult and subjected to wide inter- and intra-observer variation.

origin. Our emergency department doctors also considered this broad QRS tachycardia as supraventricular and even more so when vigorous coughing interrupted the arrhythmia (Fig. 1B).

3. Confounding factors

If a broad QRS tachycardia is hemodynamically unstable, physicians often consider it as ventricular in origin and subject the patient to DC shock cardioversion. If the poorly tolerated tachyarrhythmia is preexcited atrial fibrillation, it would be correct to perform a DC shock cardioversion. In such a patient, identification of ventricular preexcitation after restoring sinus rhythm allows the proper diagnosis and therapeutic planning in most instances. The problem in practice is the diagnosis in patients with well-tolerated tachycardias. Table 2 shows the factors that, in our experience, have led to misdiagnose hemodynamically stable regular and uniform VTs in the emergency department.

We like to indicate that other colleagues use the term monomorphic to describe a VT in which all the QRS complexes display the same morphology. We prefer using the term uniform to refer to such a VT. Monomorphic suggests a single morphology. However, patients with structural heart diseases often develop more than one morphologically distinct VT, each with a monomorphic configuration, a situation called pleomorphism. The word uniform means that all ventricular complexes of such a VT are of the same morphology, not excluding that the patient may develop another uniform VT with a different QRS configuration.

3.1. Clinical confounding factors for sustained regular uniform VT

1. Hemodynamic stability during tachycardia

A VT is hemodynamically stable when the patient in the supine position is conscious and either asymptomatic or with minor symptoms, such as slight chest discomfort or mild dyspnea, but without signs of tissue hypoperfusion. This clinical stability offers the possibility of obtaining a good 12-lead ECG of the tachycardia. We want to emphasize the need to obtain a decent quality 12-lead ECG in the emergency department during an episode of sustained tachycardia. Unfortunately, this tracing often is missing, incomplete, or of a quality that does not allow a subsequent detailed analysis by a specialist in cardiac arrhythmias. Misplacement of the extremity lead electrodes is not rare in the emergency room setting. We recommend checking the correct location and connection of electrodes to obtain a readable ECG. This is particularly crucial if one pretends to use the 4-step aVR algorithm (Table 1) [7]. If emergency physicians misdiagnose the tachycardia, an arrhythmia specialist can reinterpret this ECG, arriving at a correct diagnosis.

Hemodynamic stability during a broad QRS tachycardia is the most frequent confounding factor for misdiagnosing a sustained VT as SVT, although most well-tolerated wide QRS complex tachycardias arriving at the emergency department are of ventricular origin (Figs. 1, 2, and 3) [12,13]. Patients with an underlying structural heart disease developing an asymptomatic or minimally symptomatic VT have an overall poor prognosis, as shown by the AVID registry [14]. In patients with a previous myocardial infarction or cardiomyopathy, the hemodynamically stable VT can be a marker for an underlying arrhythmogenic substrate capable of producing a more malignant ventricular tachyarrhythmia [14]. Since an implantable cardioverter-defibrillator may be indicated in some of these patients, considering a VT as SVT can be catastrophic for the patient.

2. Lack of evidence of underlying heart disease

We apply the term “idiopathic” to VT developing in patients without structural heart disease. Idiopathic VT represents between 10% and 20% of sustained VTs in various series of patients studied in electrophysiology laboratories [15,16]. Therefore, the absence of

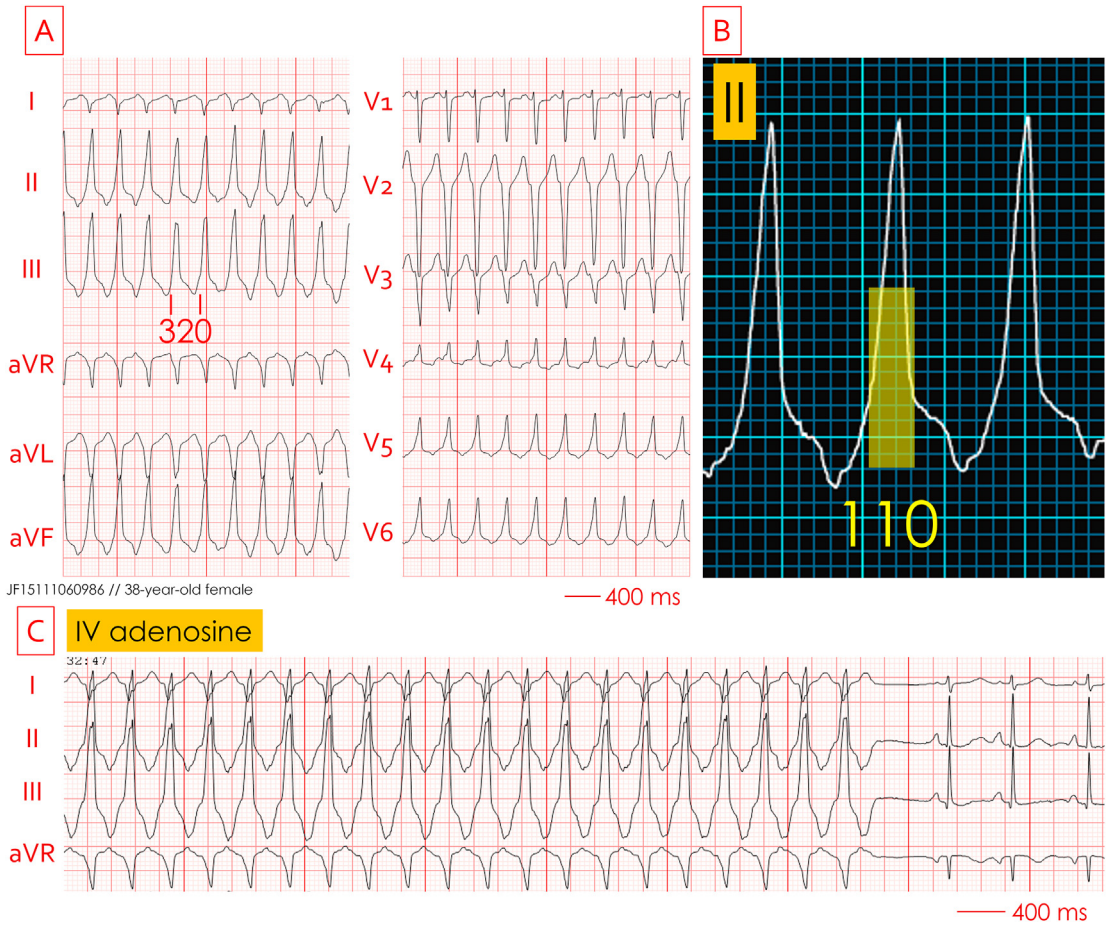

Fig. 3. Panel A: 12-lead ECG of an episode of well-tolerated idiopathic sustained uniform VT coming from the right ventricular outflow tract and diagnosed in the emergency room as SVT. Note the LBBB morphology of the QRS in lead V_1 and its southwest inferior axis in the frontal plane ($\approx +100°$). The R-R interval during VT was 320 ms corresponding to ≈188 bpm. **Panel B:** detail of lead II during VT showing that the duration of the QRS complex is 110 ms. In idiopathic VT, the width of the QRS complex can be < 120 ms, like in this case. **Panel C:** tachycardia interruption after 6 mg IV of adenosine. The emergency physicians considered this tachycardia as supraventricular due to the absence of a wide QRS and its interruption with adenosine.

evidence of underlying heart disease should not exclude VT. A situation in which an underlying organic heart disease may not be evident in the first evaluation at the emergency room is arrhythmogenic right ventricular cardiomyopathy (ARVC) (Fig. 4). In addition, idiopathic VTs and more rarely VTs in patients with ARVC or a myocardial infarction scar may display QRS complexes with a duration <120 ms. This is an added confounding factor for misdiagnosing VT as SVT (Figs. 3 and 4).

3. Another earlier misdiagnosis or SVT documentation before the current episode

We have noticed that patients arriving at the emergency room with VT may receive an SVT misdiagnosis if other emergency department doctors identified as SVT a previous paroxysmal tachycardia episode. These earlier diagnoses as SVT may have been right or wrong. Patients with VT may also present a paroxysmal SVT. Fig. 5 illustrates an idiopathic left inferior fascicular VT (LIFVT) erroneously diagnosed as SVT at the emergency room because the patient had suffered in the recent past two episodes of tachycardia correctly diagnosed as SVT elsewhere. Our stimulation study showed that, apart from the LIFVT, we could induce AV nodal reentry tachycardia (AVNRT) with normal intraventricular conduction (NIVC) and with right bundle branch block (RBBB) aberrancy. The association of LIFVT with AVNRT is well known [17].

3.2. Electrocardiographic confounding factors

1. Rate <100 bpm or QRS width <120 ms

Official definitions indicate that a VT must have a rate >100 bpm and a QRS width ≥120 ms [18]. Fig. 6 shows a post myocardial infarction VT with a rate <100 bpm resulting in hemodynamic deterioration. A rate <100 bpm should not be a limiting factor to diagnose VT no matter what the guidelines state.

A QRS duration <120 ms during VT is a particularly important diagnostic confounding factor. Figs. 3 and 4 have already illustrated VT examples with a QRS duration <120 ms. That idiopathic VT may have a narrow QRS complex is well known [15,16]. VT in ARVC may also show a QRS duration <120 ms (Fig. 4), a feature not well acknowledged in the literature [19]. It is beyond the scope of this paper to discuss this aspect in detail. The teaching point is that patients with ARVC can develop a sustained VT misdiagnosed as SVT because of a QRS duration <120 ms (Fig. 4). Also rare, but possible, is to observe a VT displaying narrow QRS complexes in patients with a post-myocardial infarction scar, as shown in Fig. 7 [20,21]. Because these patients usually have a depressed left ventricular systolic function, an erroneous diagnosis as SVT may have ominous clinical consequences (Fig. 7).

The QRS complex is wide during most VTs because ventricular activation spreads from a single point. In normal hearts,

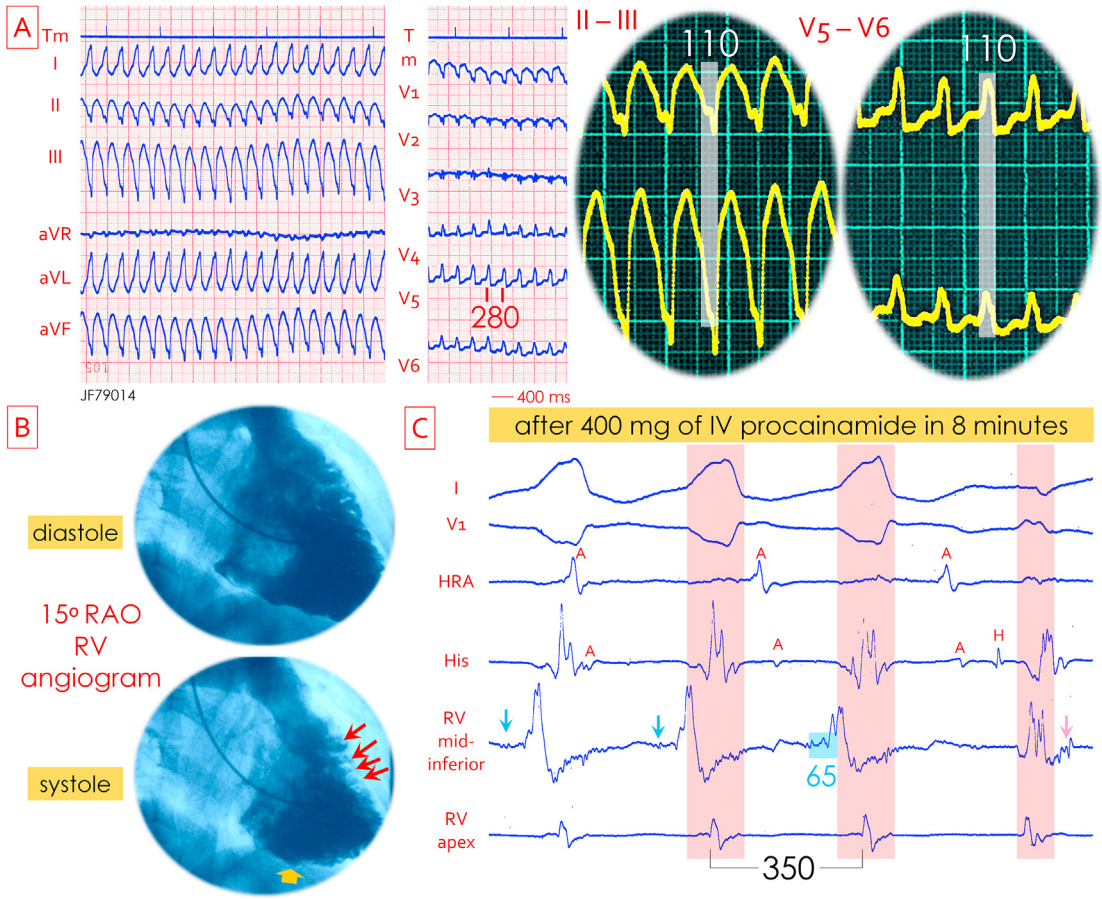

Fig. 4. Panel A: 12-lead ECG during a sustained uniform VT at ≈215 bpm in a patient without previous history of heart disease. An echocardiogram performed during tachycardia by an emergency room physician was felt not to show any cardiac abnormality. No mention was made of the presence or absence of A-V dissociation. Tachycardia had an LBBB morphology with a QRS axis at ≈ −60°. The systolic blood pressure during tachycardia was 90 mmHg. The tachycardia was interpreted as supraventricular because the QRS complexes had a duration <120 ms. The patient received IV verapamil resulting in hemodynamic deterioration. Tachycardia was interrupted by a DC shock. Note that A-V dissociation is not evident during tachycardia on the 12-lead ECG. However, the electrophysiological study demonstrated A-V dissociation during all forms of VT elicited in this patient including the clinically documented VT. **Panel B:** the patient underwent an angiographic and electrophysiologic study. The coronary arteries and the left ventricle were normal. The right ventricular (RV) angiogram showed a diastolic deformity with systolic bulging in the middle portion of the inferior right ventricle (yellow arrow), as well as a "*stacks of coins*" pattern on the septal slope of the right ventricle (red arrows). **Panel C**: endocardial mapping during tachycardia. The IV infusion of 400 mg of procainamide in 8 min resulted in the interruption of VT. There is A-V dissociation during tachycardia. The transparent red blocks represent the onset and offset of the QRS complexes. An electrode catheter positioned at the RV mid-inferior area registered a pre-systolic potential preceding the onset of the QRS by ≈ 65 ms (blue arrows). At this site, the sinus beat after the interruption of VT presents potentials following the offset of the QRS (pink arrow). Also, note that procainamide slowed the rate of VT to ≈170 bpm. HRA = high right atrium. RV = right ventricle.

supraventricular rhythms with normal intraventricular conduction display a narrow QRS complex because ventricular activation begins almost simultaneously, with only 5–10 ms of difference, at three endocardial sites [22]. To produce QRS complexes with a duration <120 ms requires a nearly synchronized exit of the right and left ventricular earliest activation fronts. Two mechanisms can generate a narrow QRS VT in patients with an intra-scar macro-reentry: an early invasion of the bundle branches by the re-entrant pathway, or the existence of two almost simultaneous exits at the right and left border of the scar (Figs. 7 and 8). More rarely, a comparable situation occurs in VT originating by focal, non-reentrant, mechanisms in the fascicular system, such as in unusual cases of bi-directional VTs due to digitalis intoxication [23]. An early invasion of the Purkinje system also occurs in patients with idiopathic fascicular VT.

2. Lack of electrocardiographic documentation of A-V dissociation during tachycardia even when the latter is present

The identification of A-V dissociation during a wide QRS tachycardia is 100% specific for the diagnosis of VT, but it is electrocardiographically clearly detectable in only ≈20% of instances (Fig. 1) [6]. A-V dissociation may be present but not evident in the 12-lead ECG of a VT (Fig. 4). The presence of capture or fusion beats during a tachycardia, independently of its QRS complex duration, requires the existence of A-V dissociation and, therefore, it has the same diagnostic value as the latter, even if the ECG does not clearly show ***P***-waves dissociated from the QRS complexes (Fig. 7). The important message here is that the lack of identification of A-V dissociation in the 12-lead ECG of a wide QRS tachycardia does not support its supraventricular origin. The presence of 2nd degree V-A block during a broad QRS tachycardia is highly suggestive of VT, although this sign is not 100% diagnostic (Fig. 2).

In patients with clinically well tolerated tachycardias in whom an A-V dissociation is not recognizable on the 12-lead ECG, cardiac auscultation may show a change in the intensity of the first heart sound which is a demonstration of the existence of A-V dissociation during tachycardia (Fig. 1). Irregular cannon "***a***"-waves in the jugular venous pulse also mark the presence of A-V dissociation during the tachycardia. Canon "***a***"-waves become visible when

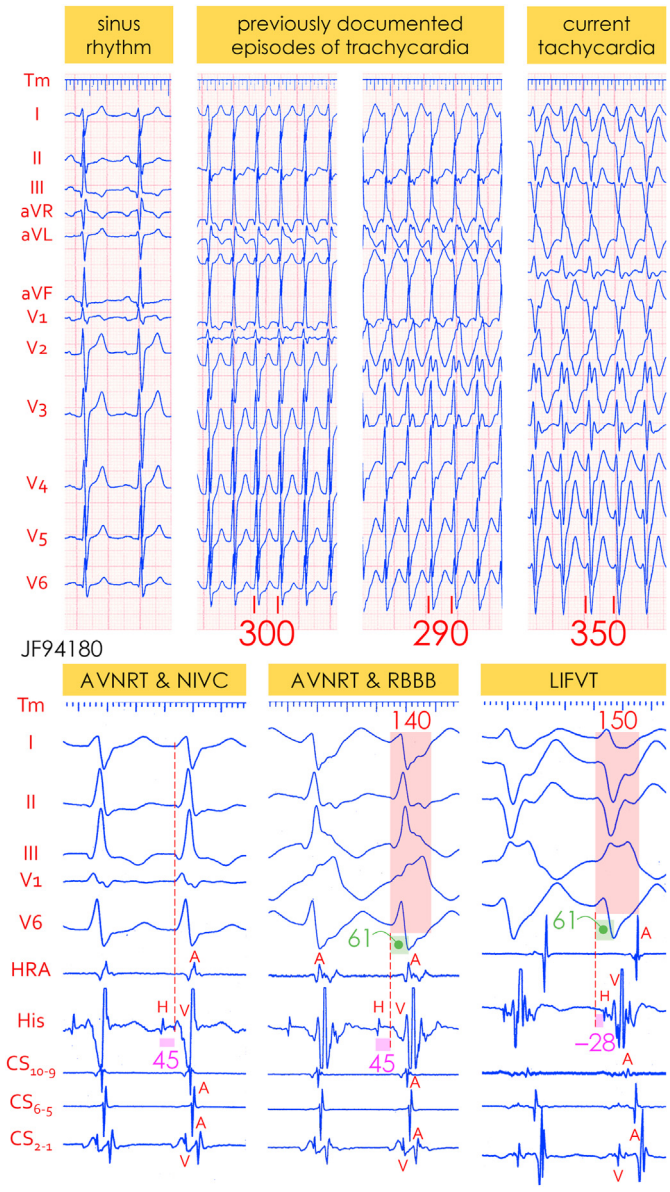

Fig. 5. Tracings from a 22-year-old male with a history of recurrent paroxysmal episodes of palpitations during the last year. He arrived at the emergency department with a well-tolerated tachycardia with a rate of ≈170 bpm (current tachycardia, superior right panel). The tachycardia had a broad QRS complex (150 ms wide), an RBBB morphology with an ***rS***-waveform in V_6, a superior north-west axis in the frontal plane, and a ***qR*** configuration in aVR with a narrow initial ***q***-wave. Emergency doctors considered this tachycardia as supraventricular mainly because the patient provided reports from emergency departments of two other hospitals describing a tachycardia with narrow QRS, and another tachycardia with wide ventricular complexes, RBBB configuration, and right axis deviation. Both tachycardias were interpreted as SVT, one interrupted with carotid sinus massage and the second with IV verapamil. IV verapamil also interrupted the current tachycardia. The surprise of our residents on duty was to hear the following day that we diagnosed the current tachycardia as an idiopathic left inferior fascicular VT (LIFVT). The patient underwent an electrophysiologic study during which we induced an AV nodal re-entry tachycardia (AVNRT) with normal intraventricular conduction (NIVC), an AVNRT with RBBB aberrancy, and the idiopathic LIFVT. All the recordings in this figure were obtained during the electrophysiological study. Note that AVNRT with RBBB aberrancy has a right axis deviation in the frontal plane, whereas the LIFVT has a left axis deviation. Also, note that a His bundle potential preceded the onset of the ventricular complex during AVNRT with NIVC and with RBBB aberrancy. Conversely, during LIFVT a His bundle potential is displayed 28 ms after the onset of the QRS. The QRS duration of LIFVT was 150 ms and the ***R***-to-***S*** interval in V_6 was 61 ms.

atrial contraction occurs against a closed tricuspid valve, namely when the P wave coincides with the ventricular systole. The QT interval, not the QRS complex, marks the duration of the ventricular mechanical systole, so cannon "***a***"-waves will be present whenever the ***P***-wave coincides with the QT interval. Cannon "***a***"-waves can be regular or irregular. Regular cannon "***a***"-waves occur in the presence of V-A conduction as is usually the case in A-V junctional reentrant tachycardia, both intranodal or in those retrogradely incorporating an accessory pathway with short conduction times. They will also appear in VT with 1:1 V-A conduction. Irregular cannon "***a***"-waves occur when there is A-V dissociation as in VT with A-V dissociation or during a complete A-V block. Irregular cannon "***a***"-waves will also be visible in VT with 2nd degree V-A block. In addition, palpation of the arterial pulse may show a changing pulse pressure which also suggests the existence of A-V dissociation. We are aware that the young generation of emergency physicians will consider these semiological pearls as remnants of an ancestral medicine. Residents in the emergency room must remember that A-V dissociation may be clearer in the physical examination than in the 12-lead ECG [2].

M-mode echocardiography has been proposed as an alternative to identify the presence of A-V dissociation during VT when the latter is not evident in the 12-lead ECG [24]. Training of emergency physicians in echocardiographic examinations is limited, so we believe that this approach to reveal the presence of A-V dissociation during tachycardia is of little practical value.

3. QRS width during tachycardia narrower than that seen during sinus rhythm

In patients with a complete bundle branch block in sinus rhythm, the development of a tachycardia with a narrower QRS is indicative of a ventricular origin. Although this can be due to interfascicular re-entry, the most common mechanism is macro-reentry through a ventricular myocardial scar with two almost simultaneous exits at separate right and left borders of the scar tissue (Fig. 8).

4. Presence of pre-existing bundle branch block during sinus rhythm with a QRS configuration similar or identical to that seen during tachycardia

When a complete bundle branch block is present in sinus rhythm, the documentation of a tachycardia with a wide QRS morphology similar or identical to that seen during sinus rhythm can make you think of a supraventricular origin. However, in patients with sick intraventricular conduction systems, such tachycardia can be ventricular due to bundle branch re-entry (Fig. 8) [25,26]. In these patients, we recommend looking for the presence of A-V dissociation to support the diagnosis of VT (Fig. 8, panel E).

5. Presence of pre-existing bundle branch block during sinus rhythm that is ipsilateral to the bundle branch block configuration showed by the QRS complexes during tachycardia

Patients with a complete bundle branch block during sinus rhythm can develop a wide QRS tachycardia displaying a bundle branch block pattern ipsilateral to that observed during sinus rhythm but with a different configuration. We should consider this tachycardia as VT, more so if the axis in the frontal plane during tachycardia is opposite to the one displayed during sinus rhythm

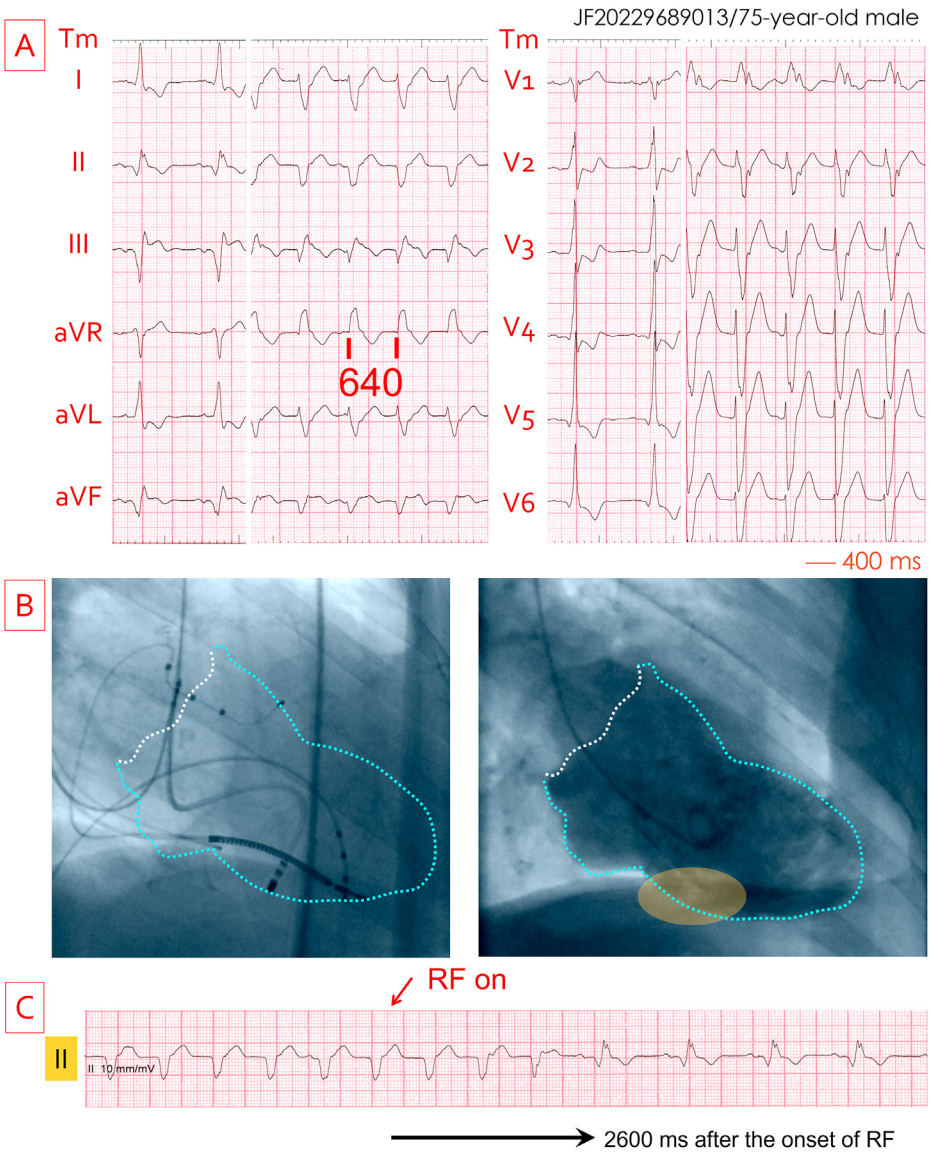

Fig. 6. Panel A: 75-year-old male with an old inferior wall myocardial infarction, severe left ventricular dysfunction and an atypical LBBB. Because of episodes of sustained VT, he had received a CRT-D device. Three months after the device implantation, he arrived at the emergency room with a "slow" tachycardia (rate ≈94 bpm), dyspnea, and signs of heart failure. Emergency room physicians initially thought that because of its rate, the arrhythmia was an A-V junctional supraventricular tachycardia. The automatic interpretation of the ECG was of no help because it diagnosed the rhythm as sinus tachycardia with RBBB. Here the key would have been to compare this recording with previously obtained ECGs. A ventricular rate slightly less than 100 bpm should not exclude VT. **Panel B**: The patient underwent a stimulation study to ablate the VT. The left fluorographic frame shows the site of ablation; the right frame shows a left ventricular angiogram obtained before the CRT-D implant outlining the ablation area (transparent yellow oval). **Panel C**: lead II during radiofrequency (RF) catheter ablation showing the interruption of VT 2600 ms after the onset of the RF pulse application.

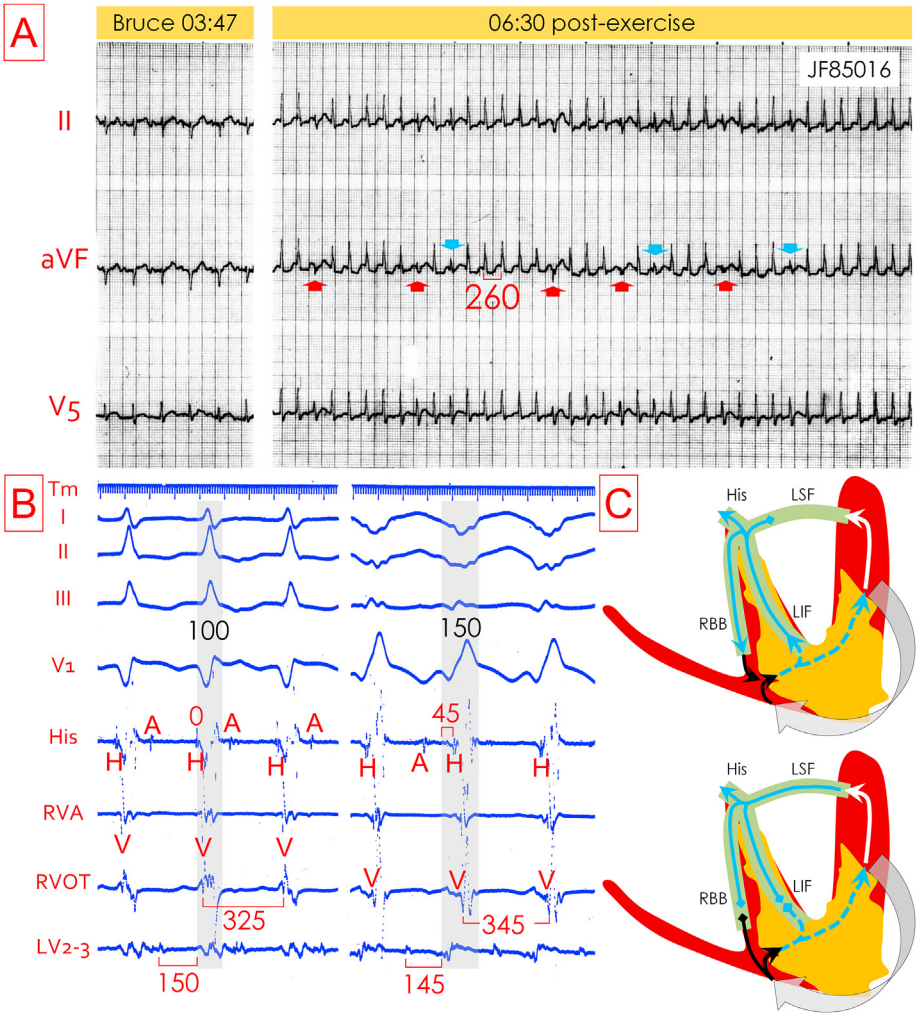

Fig. 7. Panel A: In 1985, a 67-year-old male underwent a treadmill exercise test 2 months after a transmural anterior wall myocardial infarction, completing 03:47 min of exercise of the Bruce protocol. In the post-exercise period, the patient developed a narrow QRS tachycardia at a rate of 230 bpm. The systolic blood pressure in supine position was 80 mmHg. The cardiologist supervising the stress test considered that the tachycardia was an SVT and proceeded to infuse 10 mg IV of verapamil. After that, the patient developed severe hemodynamic deterioration being cardioverted with a DC shock. Note that the QRS during tachycardia was positive in leads II and aVF, whereas it was negative in both leads during sinus rhythm due to a left superior fascicular block. Theoretically, with a superior fascicular block during sinus rhythm, it is not possible to develop an SVT exhibiting an inferior fascicular block. In addition, apart from the dominant positive QRS complexes during tachycardia, we observe negative (red arrows) and intermediate ventricular complexes (blue arrows) that are consistent with capture and fusion beats. The presence of capture or fusion beats during a tachycardia (independently of the QRS complex duration) requires the existence of A-V dissociation, even if the latter is not apparent in the ECG. **Panel B**: The patient underwent a stimulation study in which we induced a narrow and a wide QRS tachycardia. The narrow tachycardia (left inferior panel) had a QRS width of 100 ms. The recording of a His bundle potential coincidental with the onset of the QRS complex demonstrates its ventricular origin. Also, note that the QRS complex in leads I, II, and III is narrower than in lead V_1. This illustrates that we should consider the widest QRS to determine its duration. The broad tachycardia had a QRS width of 150 ms and we registered a His potential 45 ms after the onset of the QRS. Both VTs had similar rates (184 and 174 bpm, respectively). In both tachycardias we registered a septal pre-systolic potential 150 and 145 ms before the onset of the QRS, respectively. **Panel C**: mechanisms underlying the narrow (above) and wide (below) VT. Both tachycardias were based on the same macro-reentry circuit through the scar. During the narrow VT there was an early invasion of the left inferior fascicle (LIF) of the left bundle branch through which the activation wavefront reached the His bundle at the time when the reentry wavefront exited from the scar. The QRS during the narrow VT resulted from a fusion of two wavefronts: the exit of the reentry pathway at the left ventricular border of the scar, plus the anterograde wavefront through the RBB. During the wide QRS VT there was not an early invasion of the left inferior fascicle so that the ventricular activation proceeds exclusively from the left ventricular exit point of the scar-related macro-reentrant pathway.

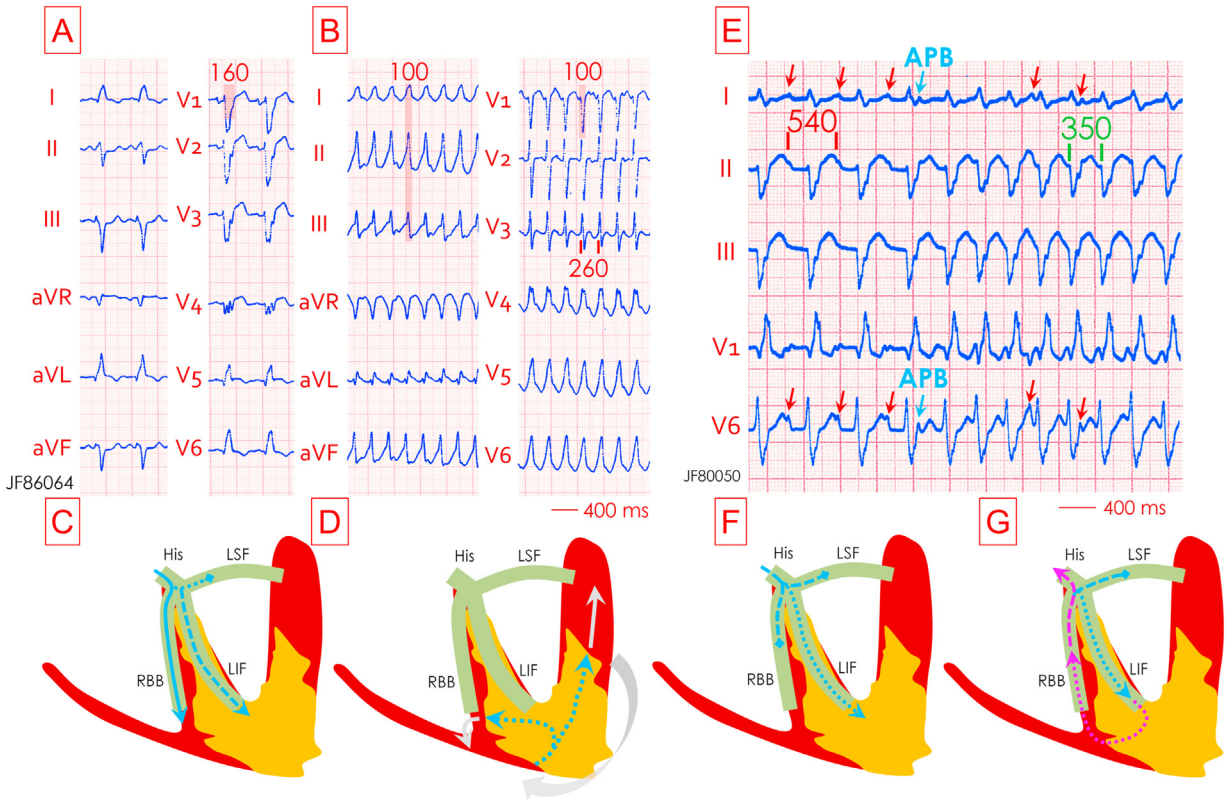

Fig. 8. Panel A: 12-lead ECG during sinus rhythm from a 72-year-old male with an old anterior wall myocardial infarction and LBBB. Note that the QRS duration was 160 ms. **Panel B**: The patient arrived in the hospital with a narrow QRS tachycardia (QRS width 100 ms) at a rate of ≈230 bpm. Emergency room doctors interpreted the tachycardia as SVT. The patient died after developing an irrecuperable cardiogenic shock following the IV administration of verapamil. **Panel C**: schematic interpretation of the ECG during sinus rhythm (main stem incomplete LBBB with complete left superior fascicular block and most likely slow conduction through the left inferior fascicle, thus resulting in LBBB with left axis deviation). SFLBB & IFLBB = superior & inferior fascicle of the left bundle branch, respectively. **Panel D:** in the presence of an LBBB, a narrow QRS tachycardia must be of ventricular origin. Interfascicular reentry can be excluded because of the intermediate QRS axis in the frontal plane during tachycardia. The most likely mechanism is macro-reentry through the post-infarction scar with two almost simultaneous exit points, one at the left ventricular border of the scar, the other its septal right ventricular margin. **Panel E:** 5-lead ECG from a 70-year-old male with post-infarction severe left ventricular dysfunction. The patient was in heart failure. During sinus rhythm he exhibited sinus tachycardia at 111 bpm with an RBBB and a superiorly directed frontal plane axis indicating a left superior fascicular block. The red arrows point to the P-waves. Note that after an atrial premature beat (APB, blue arrow) a tachycardia begins at a rate of 171 bpm. ***P***-waves during tachycardia are dissociated from the QRS complexes. The QRS morphology during tachycardia is almost identical to the configuration of the ventricular complexes during sinus rhythm. **Panel F**: schematic interpretation of the ECG during sinus rhythm in the above patient (RBBB and superior left fascicular block). Since the H–V interval was prolonged (not shown) conduction through the LIF is slow. **Panel G**: this patient underwent an electrophysiologic study during which we could demonstrate that the mechanism of the above tachycardia was bundle branch re-entry utilizing the RBB retrogradely, and the LIF anterogradely. The bundle of His was reached retrogradely and its activation preceded the onset of the QRS complex with an H–V interval much shorter than the H–V interval during sinus rhythm (not shown). V-A block was located at the level of the AV node.

(Fig. 9). If the tachycardia has a bundle branch block pattern contralateral to the one shown in sinus rhythm, the diagnosis of VT is clear (Figs. 6 and 9).

6. Slurring of the early QRS forces mimicking a delta wave morphology

Preexcited tachycardias represent the most challenging differential diagnosis with VT. Like for the vast majority of VTs, during preexcited tachycardia depolarizing the ventricles through a single atrioventricular accessory pathway, the front of ventricular activation spreads from a single point. In this regard, preexcited tachycardias display an ECG configuration identical to that of a VT having its exit close to the A-V groove. Antunes et al. have proposed electrocardiographic algorithms to differentiate VT from preexcited SVT. However, using such ECG criteria 25% of VTs can be qualified as SVT [27]. As shown in Fig. 1, 21% of people attending a voting ECG session at a European Society of Cardiology meeting, considered that the displayed VT could be a preexcited AVRT. If VT is an uncommon event in the emergency room, preexcited tachycardia is extremely rare. Most preexcited tachycardias develop in young patients without structural heart disease. Most idiopathic VTs do not have the very wide QRS complexes seen in preexcited SVT. Since ventricular preexcitation will, in most instances, be patent after the interruption of the broad-Wolffian tachycardia, the danger of inappropriately prescribing an ICD in such a patient is low.

7. A triphasic QRS in V_1 with the terminal peak taller than the first crest in tachycardias with a RBBB morphology

Marriott in the 1960's, introduced the so-called "*rabbit ear*" concept for the QRS morphology in lead V_1 to differentiate QRS complexes of a left ventricular origin from supraventricular impulses conducted to the ventricles with RBBB aberrancy. Thus, Sandler and Marriot proposed that triphasic (***rsR′***, ***rSR′*** or ***rsr′***) QRS complexes in V_1 favor RBBB aberrancy [28]. While this is correct, also is true that these triphasic RBBB-like morphologies are present in ≈10% of tachycardias of ventricular origin [6]. There are cases of idiopathic fascicular VT exhibiting a triphasic RBBB morphology in V_1. Rarely, post infarction VT may also display a triphasic configuration in V_1. Our recommendation is not to take a triphasic RBBB morphology as the sole evidence for SVT.

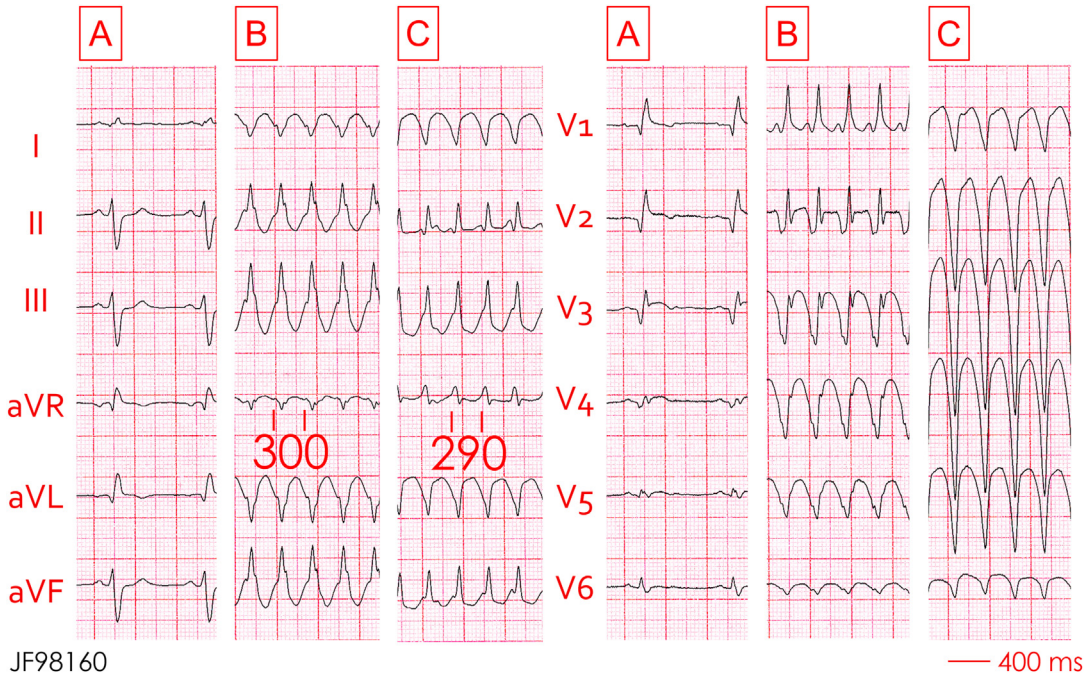

Fig. 9. 12-lead ECG during sinus rhythm and two morphologically distinct VTs from a 74-year-old male with an old anterior wall myocardial infarction. **Panel A**: During sinus rhythm the QRS displayed an RBBB with a left superior fascicular block. **Panel B**: the QRS complex during this VT presented an RBBB morphology. However, this tachycardia cannot be supraventricular because in the frontal plane the ventricular complexes have a right axis deviation, whereas in sinus rhythm there was a left axis deviation. This discordant fascicular block pattern in a patient with RBBB during sinus rhythm makes the diagnosis of SVT impossible for panel B tachycardia. **Panel C**: Tachycardia in panel C shows an LBBB morphology. Independently of other diagnostic criteria favoring VT, in the absence of ventricular preexcitation, an SVT cannot produce an LBBB morphology in a patient with RBBB and left superior fascicular block in sinus rhythm.

8. A tachycardia rate below 140 bpm

Both VTs and SVTs can course with extremely high ventricular rates as well as with slow ones. It is an error to consider that rates above 200 bpm suggest VT and that rates under 140 bpm suggest SVT. In this paper we have illustrated slow VTs (Figs. 6 and 10) and fast SVTs (Fig. 5). Tachycardias with rates below 140 bpm are more frequently hemodynamically stable, which is an added confounding factor.

3.3. Interruption of tachycardia with certain maneuvers as confounding factor (coughing, Valsalva maneuver, carotid sinus massage, adenosine, or verapamil)

Emergency room physicians often think that tachycardias interrupted by vagal maneuvers, IV adenosine, verapamil, or diltiazem, are supraventricular. This belief is not correct. Idiopathic VTs from the ventricular outflow tract or fascicular, can stop by these maneuvers or pharmacological agents [29,30]. Fig. 3 shows the interruption of an idiopathic sustained right ventricular outflow tract tachycardia by IV adenosine. Kim et al. found that IV adenosine interrupted 75% of sustained ventricular outflow tachycardias [30]. They also interrupted with IV Verapamil four instances of idiopathic ventricular outflow tachycardias. Carotid sinus massage or a Valsalva maneuver stopped 55% of sustained ventricular outflow idiopathic tachycardias [30]. Adenosine is less effective in idiopathic fascicular left ventricular tachycardias than in ventricular outflow tachycardias. However, IV verapamil always interrupts fascicular left ventricular tachycardias. Coughing or a Valsalva maneuver can also stop episodes of idiopathic fascicular VT [30,31].

The infusion of IV adenosine or calcium antagonists does not interrupt VT in patients with structural heart disease. On the contrary, in patients with left ventricular dysfunction, the infusion of IV verapamil usually produces a severe hemodynamic deterioration that may have fatal consequences (Figs. 4, 7 and 8). Uncommonly, coughing or a Valsalva maneuver may interrupt episodes of sustained VT in patients with post myocarditis (Fig. 1) or post-myocardial infarction macro-reentrant VT (Fig. 10) [32,33]. Mechanisms explaining the interruption of a macro-reentrant scar-related VT by a Valsalva maneuver are speculative [32,33].

3.4. Opinions of other physicians or the patient

We have seen VTs misdiagnosed as SVT because the opinion of a colleague, that had not analyzed carefully the 12-lead ECG of the tachycardia and the clinical circumstances of the patient, influenced the attending physician. Less often, the opinion of the patient on the origin of the tachycardia has prevented a thoughtful analysis by the emergency doctor in charge of the medical attention, leading to a wrong diagnosis. We warn emergency doctors against external influences.

4. Conclusions

In the emergency medicine setting, deciding the ventricular or supraventricular origin of tachycardias is difficult. The frequency with which emergency physicians approach a clinical problem decisively influences their diagnostic effectiveness. Paroxysmal tachycardias have a low rate of presentation in medical emergencies. In addition, the diagnostic criteria to figure out the ventricular or supraventricular origin of tachycardia are complex and easy to forget if not used regularly. Throughout the years of our professional activity, we have found that emergency doctors often held wrong clinical and electrocardiographic beliefs leading to misdiagnosing VT as SVT. This erroneous diagnosis results in incorrect therapeutic planning and inappropriate risk stratification of

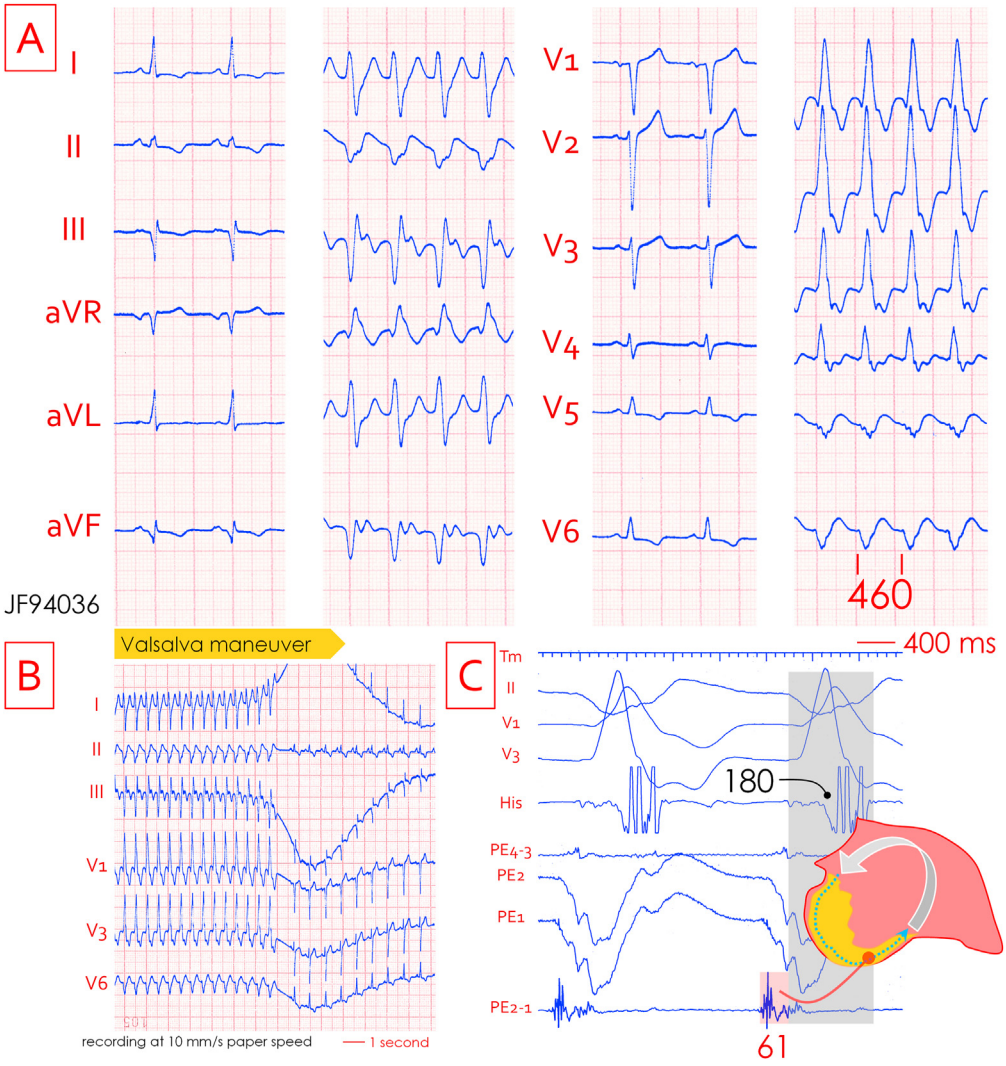

Fig. 10. Panel A: 12-lead ECG during sinus rhythm and a wide QRS regular tachycardia at a rate of 130 bpm. The slow rate of this tachycardia and its good hemodynamic tolerance led to a diagnosis of SVT. **Panel B:** emergency room physicians instructed the patient to perform a Valsalva maneuver that terminated the tachycardia (recording obtained at 10 mm/s paper speed). **Panel C:** on restoring sinus rhythm, the electrocardiogram evidenced the existence of an old inferior wall myocardial infarction (see panel A). The patient underwent an angiographic study, and the left ventricular angiogram depicted an inferior wall aneurism. We also performed a stimulation study that induced the clinically documented VT. We successfully ablated the latter at the mid-inferior border of the aneurysm. At this site, we registered a fragmented activation preceding by 61 ms the onset of the QRS during VT. The transparent grey box represents the duration of the ventricular complex during VT (180 ms).

patients arriving at the emergency room for VT. Based on our experience as clinicians and teachers, we have structured, discussed, and illustrated the confounding factors behind considering VT as SVT. This information should be part of the training programs for emergency or cardiology residents.

Dedication

We dedicate this article to the memory of Prof. Hein Wellens who made seminal contributions as a scientist and teacher in the field of the electrocardiographic diagnosis of paroxysmal tachycardia.

Author contribution

Jerónimo Farré: conceptualization; writing original draft & reviews & editing; design and creation of illustrations.

José Manuel Rubio: reviews & editing.

Eduardo Back Sternick: reviews & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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