

Pitfalls and Errors of the ECG and Monitoring Systems Recording

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17.1 Case 1

A 40-year-old female was admitted to our emergency department because of faintness and palpitations. A typical atrioventricular nodal reentrant tachycardia (AVNRT, see Chap. 5 for more details on supraventricular tachycardias) was diagnosed at the first ECG, and the patient was admitted to our clinic for radio-frequency (RF) ablation. Her previous medical history was negative, and she denied main cardiovascular risk factors. A standard evaluation was performed at admission (e.g., physical examination, echocardiography, etc.), and also a rest ECG was recorded while the patient was asymptomatic (Fig. 17.1a, b): “*bad news coming from a first glance?*”

17.1.1 ECG Analysis

The ECG shows regular rhythm at 83 beats/min. Every QRS complex is preceded by a P wave. P wave morphology is normal (positive in II and negative in VR) with normal axis and duration (see Chap. 1 for more information on P wave). Atrioventricular (AV) conduction is normal (PR segment of 160 ms); intraventricular conduction is also normal (QRS duration of 90 ms) with

normal QRS axis ($+75^\circ$). R wave progression is normal in the precordial leads. QT segment (measured in II, see Chap. 13) is 360 ms, and QTc is 426 ms. There are diffuse waves within PR, ST, and T waves. There is a PR segment depression in almost all leads, except in I and VL where it appears elevated. ST segment is diffusely elevated (also in VR), except in I and VL where it is depressed. T wave is biphasic in the most part of the leads with a positive predominant component in all leads. In leads I, VL, and III, T waves have an uncommon sinusoidal pattern. It is difficult to identify the TP segment since the isoelectric line is hard to find.

17.1.1.1 “Bad News Coming from a First Glance?”

We have a regular rhythm with normal P axis and a P wave preceding a normal QRS. It is a sinus rhythm.

A PR segment depression could be pathognomonic of acute pericarditis associated with diffuse ST segment elevation and a reciprocal PR elevation and ST depression in VR [1].

Another important possible diagnosis to evaluate is acute coronary syndrome. Also in that case, there is an ST elevation (see Chap. 9 for detailed information). An ST elevation in this ECG is clearly present in inferior leads ($\text{III} > \text{VF} > \text{II}$), thus potentially suggesting the right coronary artery involvement. Posterior wall is eventually not affected since V1 and V2 don't have the

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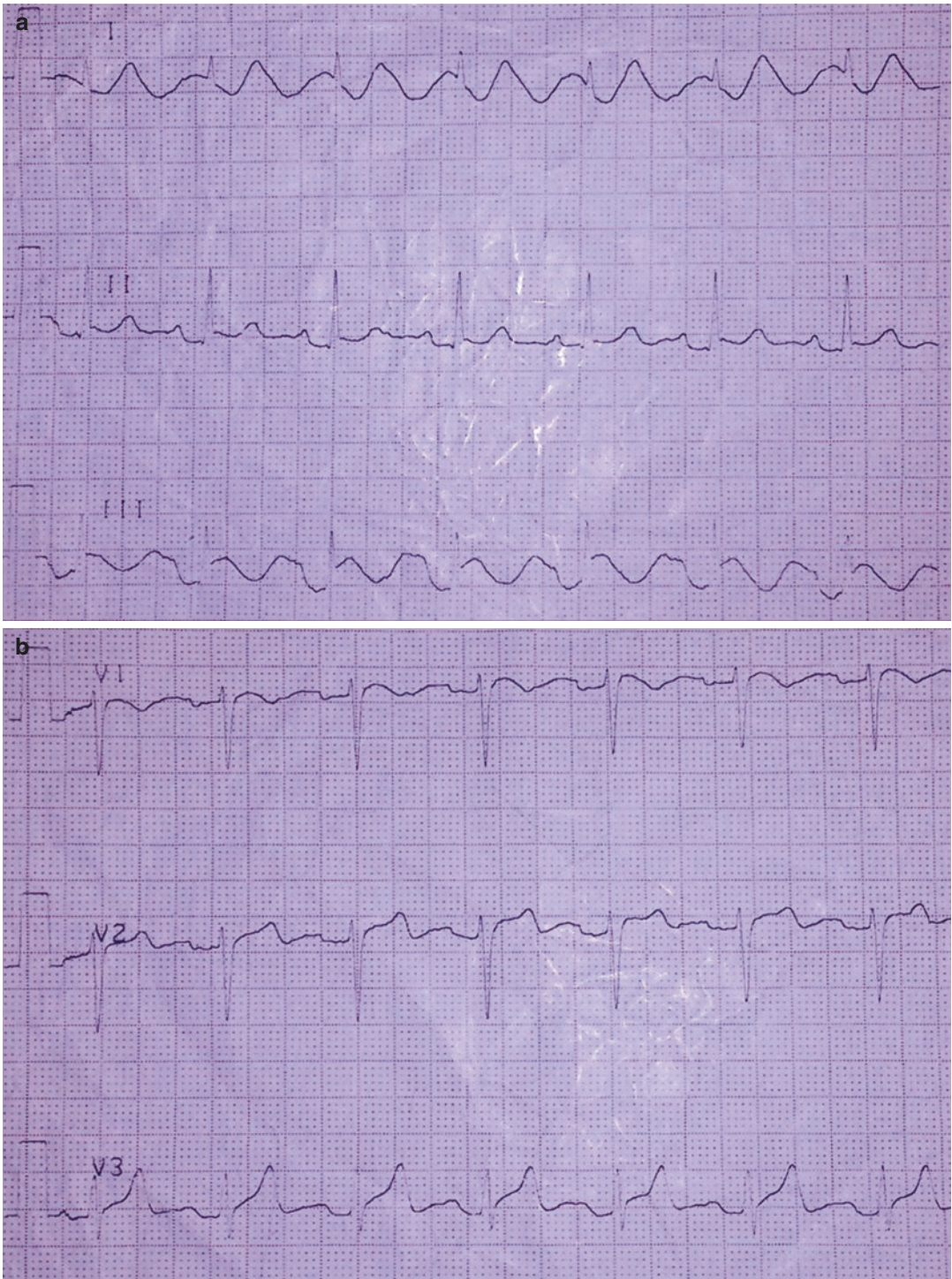


Fig. 17.1 (a) Peripheral leads. (b) Precordial leads

typical pattern. ST is also depressed in I and VL. Could we consider this as a possible sign of reciprocity?

The patients remained always asymptomatic. She didn't suffer from chest pain, swelling, fatigue, palpitations, or other cardiological symptoms. No pericardial rubs were present. Blood samples did not show any cardiac myocardial necrosis nor inflammation marker elevation. She didn't refer any flu-like syndrome before admission.

Finally a 2D echocardiogram was performed and resulted to be normal with normal ejection fraction and not any sign of pericardial fluid accumulation.

Other possible causes to rule out in this trace are ionic imbalances and specific drug effects. However, no ionic imbalances were detected, and not any drug was taken by the patient before admission.

After repeating the ECG a couple of minutes later, we simply realized that some of the electrodes were not correctly positioned on the skin with consequent poor contact.

17.1.1.2 This Was Thus Just an Artifact!

A similar problem did come 2 years after the above described case in another patient (Fig. 17.2a).

We have a regular rhythm. All the QRS are preceded by P waves with normal axis and duration (clearly positive in II–III and VF and negative in VR). It is a sinus rhythm.

Also in this case, PR segment is depressed in I and III; ST segment elevated in I–III and VL with a biphasic T waves. There is a similar sinusoidal pattern as seen in Fig. 17.1a, b. We must observe lead II and the precordial leads. In lead II the trace is clean and shows a normal sinus rhythm with a quite normal ECG. Even in this case, after a careful electrodes repositioning, everything returned to normal (Fig. 17.2b).

When you observe a sinusoidal isoelectric line strange pattern, check electrodes first!

17.1.2 Artifact Interpretation

Misplaced leads and electrodes malpositioning are surely the most common causes of artifacts in the 12-lead ECG. P and QRS axis and morphology in I and VR are essential in this analysis. “Patient motion” may also cause artifacts sometimes simulating even a ventricular tachycardia (VT, see below) or an atrial tachycardia (like atrial flutter). Sometimes (see Table 17.1) when the patches are overdue or the amount of gel is not sufficient, the “electrical skin contact” may not be optimal. This brings impedance instability and leads to sharp or slow waves with different morphologies and amplitudes. The isoelectric line is affected and could become sinusoidal. The skin must be correctly prepared before each ECG recording; that must be shaved and cleaned with isopropyl alcohol in order to remove lipids and impurities.

17.2 Case 2

This electrocardiogram of a 75-year-old patient (Fig. 17.3) comes from a bedside prolonged monitoring system. The patient was admitted for investigating a possible ischemic heart disease after a syncope.

17.2.1 ECG Analysis

In the chest leads (right part), the rhythm is regular at 75 bpm. P waves are visible, and they precede each QRS with a prolonged PR interval of 260 ms, consistent with a first-degree AV block. QRS duration is 100 ms and has an incomplete right bundle branch delay (R wave in V1). There are no evident repolarization abnormalities, and QT/QTc intervals are normal (380/420 ms).

In the limb leads (left part), there is a run of beats with wide QRS with very sharp initial upslope.

This run has a warm-up phase and very high rate of 300 bpm before ending after polymorphic

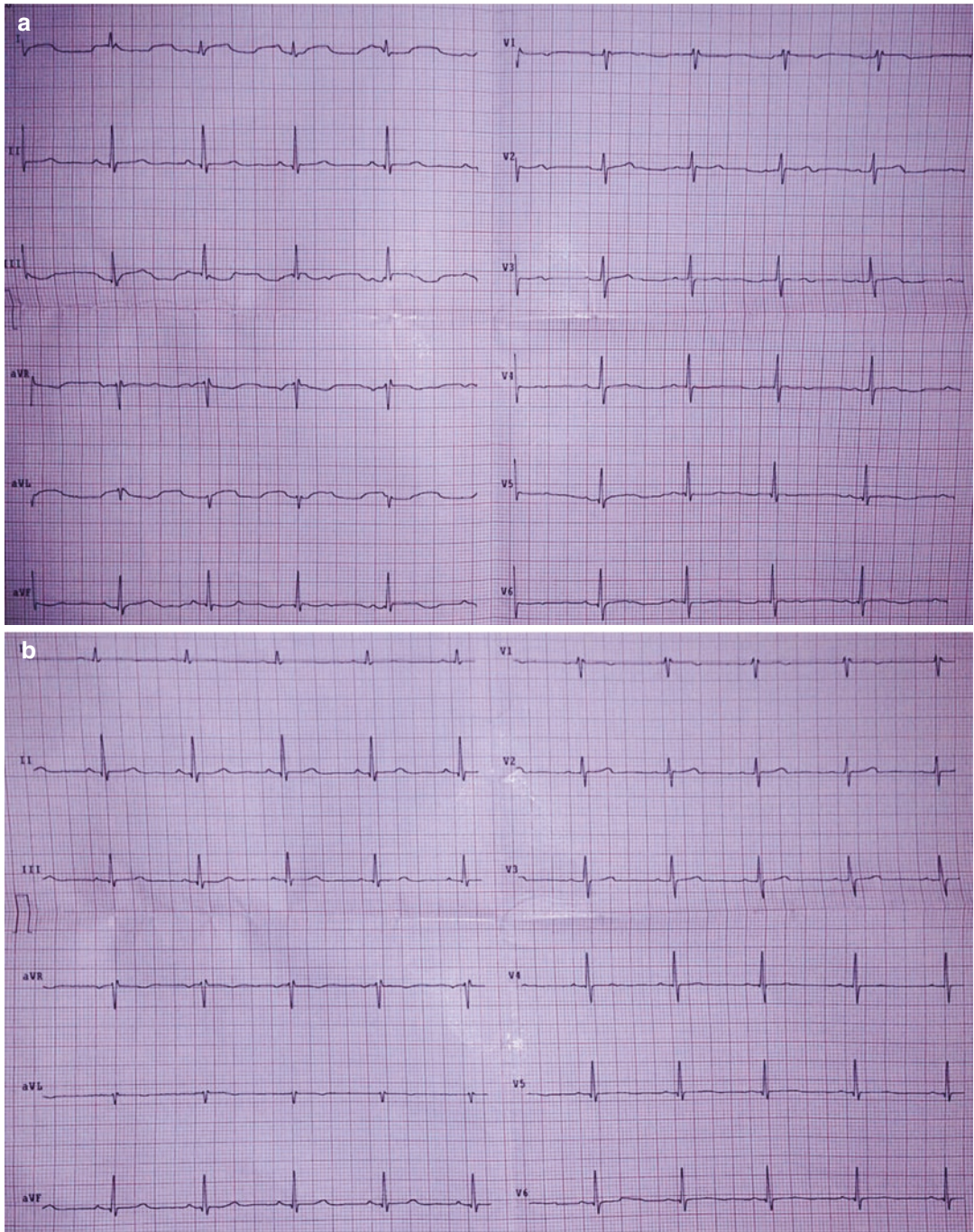


Fig. 17.2 (a) Typical ECG artifacts due to not properly stucked patches. (b) After patches repositioning

beats. The last two QRS are narrow and simulate the sinus origin; they have an indeterminate axis.

17.2.1.1 This Is a Pseudo Non-sustained VT

Diagnosis may be simple by comparing the peripheral and the chest leads (notably lead I). That ECG pattern could be particularly challenging when having a single lead in a bed monitoring system.

Table 17.1 Schematic subdivision of possible artifacts sources

Internal (physiological)	<ul style="list-style-type: none"> • <i>Patient motion</i>: does not allow electronic filtration (large swings, usually by stretching the epidermis) • <i>Muscular activity</i>: allows electronic filtration (small spikes)
External (not physiological)	<ul style="list-style-type: none"> • <i>Electromagnetic interference</i> (widely isoelectric line): light fixtures, electrocautery, electrical devices in the room • Cable and electrode malfunction, insufficient amount of electrode gel, fractured wires, inappropriate filter settings, loose connections, misplaced leads, accumulation of static energy

There are few features that may help in the differential diagnosis: the baseline trace movement before and after the “tachycardia episode,” the too sharp slopes of the terminal parts of the pseudo-ventricular tachycardia compared to the slower slopes of a true ventricular tachycardia, and, at last, the too fast rate (up to 300 bpm); that is unusual in a VT.

17.2.2 Artifact Interpretation

Huang et al. suggested a diagnostic algorithm for pseudo-VT caused by a tremor-induced artifact [2, 3]:

1. *Sinus sign*: one of the frontal leads (I, II, or III) shows normal P waves, QRS complexes, and T waves if one of the upper limbs is free of tremor or movement.
2. *Notch sign*: intersection of the native complex with the artifact creates visible notches and that the notch-to-notch intervals can be compared with the RR intervals of the underlying rhythm (*see asterisk on Fig. 17.3 for an example*).

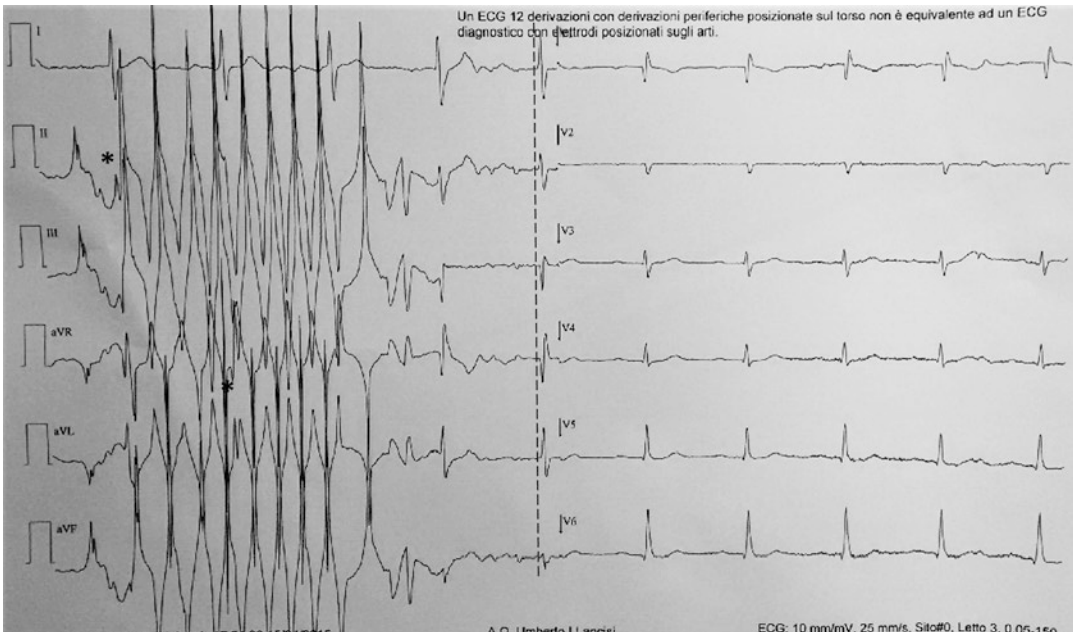


Fig. 17.3 Pseudo-VT



Fig. 17.4 Pseudo-VT with spike sign (reprinted with Springer permission from “Pseudo ventricular tachycardia: a case report” DOI: 10.1007/s11845-009-0387-4)

3. *Spike sign*: tiny “spikes” can be seen among the pseudo-ventricular tachycardia complexes, indicating the presence of real QRS complexes. In the ECG of Fig. 17.4, the limb leads, except lead III, show a rapid and irregular rhythm with a rate of up to 300 bpm. The QRS complexes show marked variability in amplitude, interval, morphology, and axis, resembling a torsades de pointes arrhythmia. On careful examination of the 12-lead ECG, it is obvious that this is a fake VT because lead III is spared and narrow QRS can be seen at regular intervals in the chest leads. Also, the diagnosis is aided by observing these tiny “spikes” (pointed by arrows on the figure), which are narrow QRS merged in the larger deflections falling exactly with the QRS of the other lead (dotted line).

17.3 Case 3

T. F., 51 years, previously implanted (Jan 2017) with a loop recorder for lightheadedness episodes and a family history of sudden cardiac death, is currently hospitalized in a near hospital because of ischemic left frontotemporal stroke. He came

to our attention, with the suspect of a cardioembolic origin of the clinical event, and the loop recorder was interrogated.

There were many episodes classified as atrial fibrillation, but just at first glance something was wrong. See the trace below (Fig. 17.5).

17.3.1 ECG Analysis

The rhythm is clearly irregular, but an evident P wave precedes each QRS. In this short strip, PP intervals are different in duration: the PP interval after the pause is longer than the preceding one, and the pause is shorter than twice of the PP interval before the pause. P waves, QRS complexes, and T waves are reconstructed by the device and may be affected by individual chest variation and implant position.

The evidence of a P wave excludes atrial fibrillation. A possible interpretation could be therefore a sinus arrhythmia in a young subject enhanced by night vagal tone, but the presence of a II degree sinoatrial block with a Wenckebach phenomenon should also be considered in the differential diagnosis. Supraventricular bigeminism caused by a perisinusoidal focus can be excluded

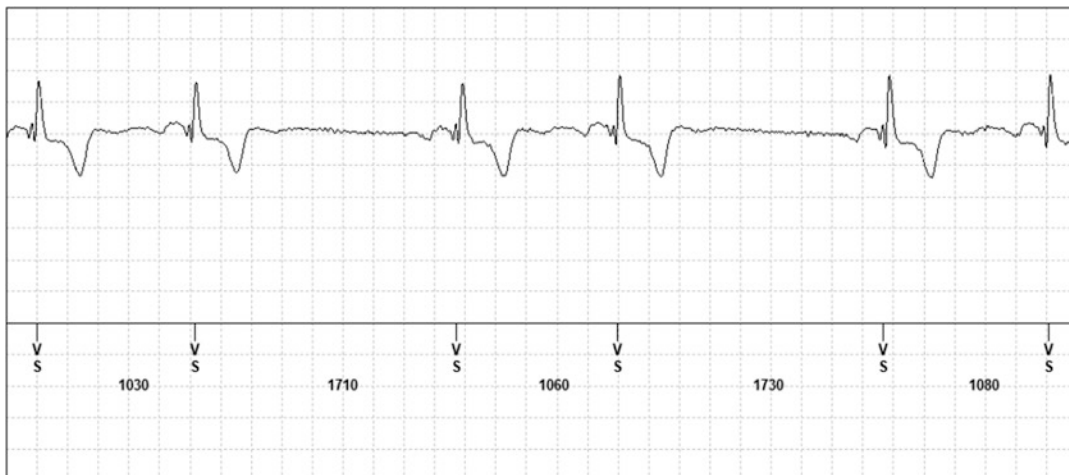


Fig. 17.5 Loop recorder trace

because of an extremely variable coupling interval during the all recording.

17.3.2 Artifacts Interpretation

The hallmark of atrial fibrillation interpretation is an irregularly irregular ventricular rhythm.

First-generation loop recorders discriminate atrial fibrillation occurrence on RR interval variation in a period of 2 min, and then the difference in consecutive RR intervals is reported in Lorenz diagram [4].

Other devices are endowed with exclusion algorithms that ignore ectopic beats and avoid atrial fibrillation misdiagnosis [5]. Another algorithm searches a P wave between two R waves combined with the previous mentioned methods [6]. The “smart atrial fibrillation detection” signs research evolution in this field, leading to about 50% false-positive reduction and preserving the 99.1% diagnostic sensibility [6]. Trademarks have obtained a high-quality signal in their devices which permits to distinguish artifacts (noise) from genuine ECG signals.

This is the result of multiple electrodes analysis and multiple vectors reconstruction leading to a reliable arrhythmia detection [7, 8]. Modern implantable devices record high-amplitude waves with a stable sensing, even in breathing tests or in

body position changes, that it is of main importance for a precise ECG interpretation [9]. A main challenge for clinicians is to find reliable algorithms in implantable cardiac monitors that diagnose atrial fibrillation and quantify its burden [10]. The correct interpretation of this ECG trace avoided the initiation of an unnecessary anticoagulation therapy.

17.4 From ECG to Diagnosis

Artifacts are common during ECG monitoring in several clinical settings (e.g., ambulatory 12-lead ECG, telemetry during hospitalization, loop recorder devices, etc.). Despite ECG monitoring systems are widely used in common clinical practice, little is written in literature about possible pitfalls and errors. Pitfalls and errors are also more often present in emergency department where it could be much more dangerous, leading to wrong diagnosis and treatment [11]. There have been suggested two large categories of artifact sources in order to easily solve possible pitfalls: *internal and external artifacts sources* (Table 17.1) [12, 13].

One interesting algorithm was also proposed, by Baranchuk and colleague, to avoid misleading interpretations and systematically and carefully examine ECG traces: *REVERSE* (Table 17.2) [12].

Table 17.2 Reverse algorithm (modified from Baranchuk A. et al. [12])

	Finding of interest	Meaning
R	R wave is positive in lead aVR (P wave also positive)	Possible reversal of left arm and right arm electrodes
E	Extreme axis deviation: QRS axis between +180° and -90° (negative R wave in lead I, positive R wave in AVF)	Possible reversal of left arm and right arm electrodes
V	Very low (<0.1 mV) amplitude in an isolated limb lead (isolated “flat” lead)	Possible reversal of right leg and left arm or right arm electrodes
E	Exchanged amplitude of the P waves (P wave in lead I greater than in lead II)	Possible reversal of left arm and left leg electrodes
R	R wave abnormal progression in the precordial leads (predominant R wave in V1, predominant S wave in V6)	Possible reversal of precordial electrodes (V1 through V6)
S	Suspect dextrocardia (negative P waves in lead I)	Possible left arm-right arm electrode reversal
E	Eliminate noise and interference (artifact mimicking tachycardias or ST-T changes)	

Stop patient’s tremors and match ECG with clinical findings. A very simple and effective technique to solve diagnostic dilemmas particularly on the suspicion of a wide QRS tachycardia is to mark the RR intervals preceding the false arrhythmia and extend the marks to the underlying spontaneous QRS hidden within the wide deflections.

The ECG monitoring systems changed the physicians’ way to operate. These devices allow a complete rhythm monitoring but sometimes need a specific interpretation skills. An incorrect analysis could be inherent to the monitoring system itself. That is only a tool in the physician hands, not the opposite. A mere acquiescence of the “machine diagnosis” could be harmful by directing medical efforts toward incorrect strategies. Clinical practice should guide more and more the engineer research in solving the unmet issues,

overcoming pitfalls of the traces interpretation. We must always treat patients and not their ECG.

Special thanks to Dr. Enrico Rita for the support on this chapter

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