Current Role of Electrocardiography in Acute Ischemic Syndromes: Is It an Outdated Technique?

To the Editor:

We present the case of a 59-year-old woman with intense precordial pain 1 h in duration, arterial pressure at 136/84 mm Hg, and cardiac frequency at 72 BPM. The ECG (Figure 1A) showed a preinfarction stage with level 3 ischaemia (pointed T, elevated ST, and disappearance of the S wave) due to occlusion of the right coronary artery (TII>TI) distal to the first right acute marginal one (depressed ST in V_1-V_3). The second level block, Mobitz type II, indicated that the inferior part of the atrioventricular node (AVN) was involved in the ischaemic process (Figure 1A). Treatment with aspirin and nitroglycerin began. During the trip to the referred hospital, various arrhythmias were registered (Figure 2) in a short time: also a sinus rhythm of 75 BPM with a PR of 0.34 s; shortly after, typical Wenckebach tracing with alternate beats was observed with an atrial frequency of 110 BPM and varying ventricular frequency of 40-55 BPM. The first P wave conducted at a PR interval

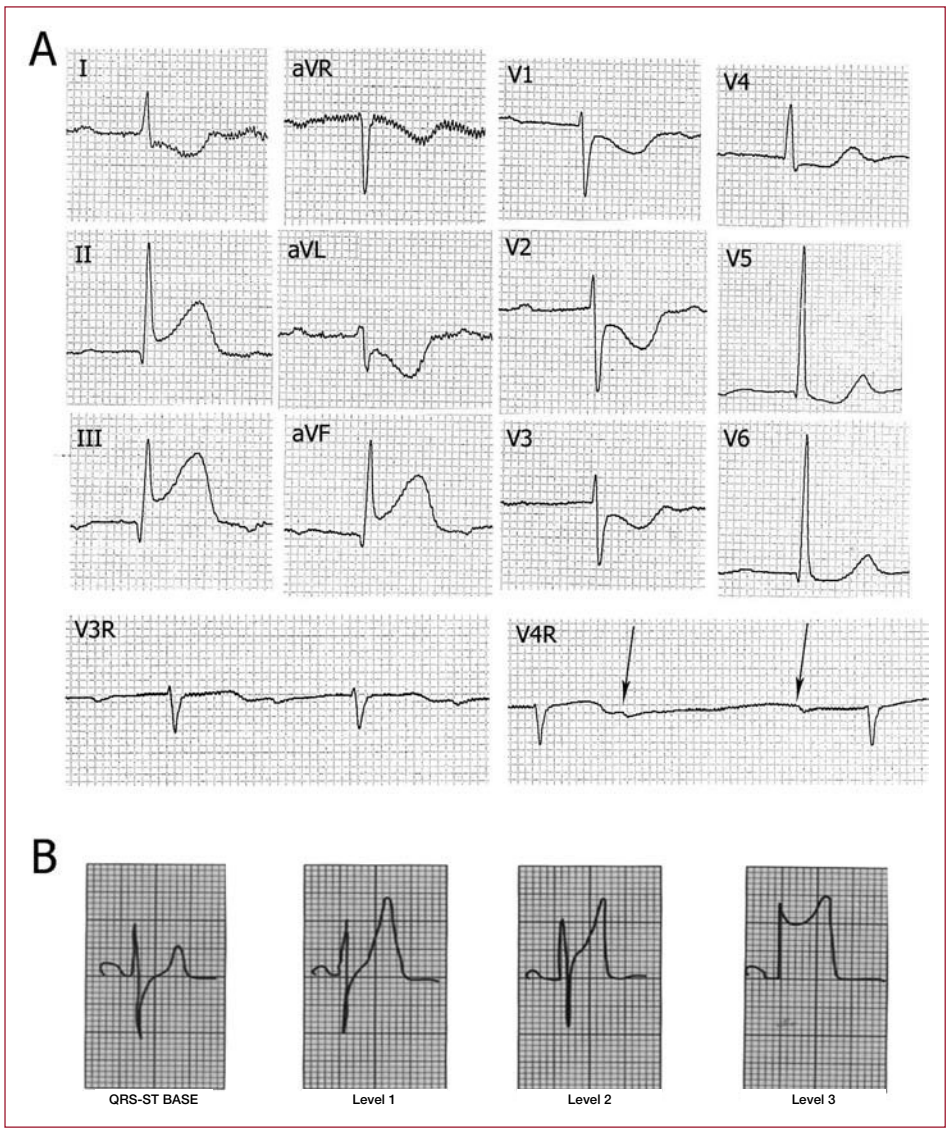

Figure 1. A: electrocardiogram in acute phase with level 3 ischaemia and Mobitz type 2 block, observable in V4R. B: levels of ischaemia.

of 440 ms. The second P wave remained blocked, and the third conducted at a PR of 240 ms. The fourth and fifth remained blocked, and the sixth conducted at a PR of 240 ms. The seventh P wave remained blocked, and the eighth conducted at a PR of 440 ms.

The following 2 did not conduct (ninth and tenth), and the eleventh conducted at a PR of 240 ms. The sequence continued as PR being shorter after 2 blocked P waves. The phenomenon was described in the acute inferior infarction as a transversal block[1] of the AVN. In the superior part of the AVN, conduction was like a typical Wenckebach block, but in the inferior part, it conducted with an atrioventricular block 2:1. This block appeared with a frequency of 110/min. Acceleration of cardiac frequency was probably due to the endovenous administration of nitroglycerin and the beginning of the clearance of adenosine, which up to that time worked to depress sinus automatism and atrioventricular conduction.[2]

In the following tracing, a surprising phenomenon was observed. The ST segment became isoelectric, and an atrial frequency of 150 sinus beats/min which conducted at 1:1 with a normal PR was observed. This paradoxical functioning of atrioventricular conduction is quite normal in the reperfusion stage. In this tracing, the ST descended, which indicated that it entered into advanced reperfusion stage, with the disappearance of ischaemia which affected the AVN, and consequently the clearance of adenosine.

A few minutes later, the patient presented with cardiorespiratory arrest, due to two consecutive episodes of polymorphic ventricular tachycardia, which required cardioversion. This phenomenon occurs frequently during the reperfusion stage of patients with level 3 ischaemia.[3]

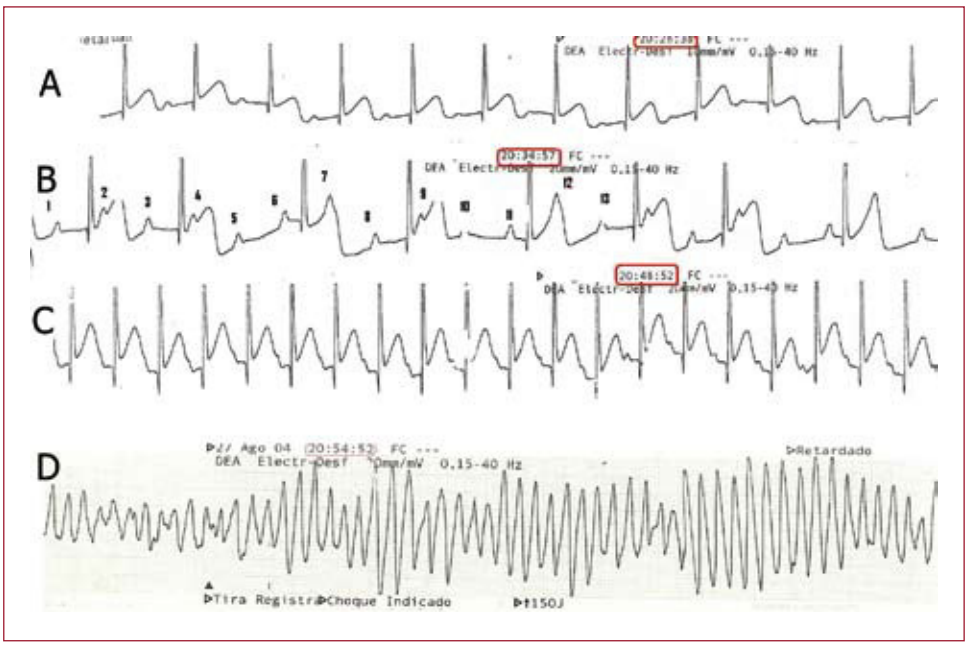

Figure 2. In a short time, sinus rhythm changed from a sinus rhythm of 75 BPM (A) to polymorphic ventricular tachycardia which required immediate defibrillation (D). In B, the phenomenon of Wenckebach with alternate beats can be observed.

This case is a clear example of the valuable information the ECG provides on various aspects such as the pathophysiological mechanism of acute ischaemia and its development phase, anatomical alterations in the ischaemic myocardium, intensity of the ischaemia, and coronary anatomy of acute ischaemia.

From the physiopathological point of view, we can obtain the following information from the ECG during an acute ischaemic episode:

– Increase in demand for energy in a case of chronic coronary disease. Manifestation is from the electrocardiographic point of view with sinus tachycardia and maximum depression of the ST-T in V_4-V_5[4]

– Sudden total or subtotal occlusion of an epicardial artery, with subsequent reduction of myocardial flow. Changes are produced in the ST-T segment, but usually with ventricular frequencies <90 BPM.[5] In this case, reduction of cardiac frequency was due to one of the most important biological phenomena which occurs in acute ischaemia, which is adenosine's function in the sinus and atrioventricular nodes.[2] In this instance, adenosine acts as a powerful selective antiadrenergic, depressing sinus automatism, and atrioventricular conduction[2]. It also decreases contraction of the ischaemic area by dilating the epicardial arterioles involved in that area, reducing blood flow from the healthy area (reduction of positive flow). In this way, cardiac damage is minimized

The second fact of critical importance is the developmental stage in an which ischaemic syndrome is found.[6] We find the following evolution patterns:

– The preinfarction stage, which is characterized by acute changes of the ST segment and T wave. This stage represents the last opportunity for possible reduction of myocardial damage caused by ischaemia

– If myocardial reperfusion has not been effective, a Q wave with elevated ST and positive T is observed

– Elevated ST with inverted or biphasic T indicates incomplete reperfusion

– Complete myocardial reperfusion is manifested by the presence of Q waves with an isoelectric ST segment and an inverted T wave[7]

A third aspect is information from the ECG on anatomy of the ischaemic myocardium. With sudden occlusion, total or subtotal, it provides valuable information. Regional Transmural ischaemia due to total occlusion of an epicardial artery becomes manifested due to ST segment elevation. Contrasting this, circumferential subendocardial ischaemia is manifested by a maximum depression of V_4-V_5 in sinus rhythm with frequencies <90 BPM. The electrocardiographic pattern may indicate a proximal occlusion of the left coronary or severe occlusion of 3 vessels.[5] Regional subendocardial ischaemia, with depression of ST from V_2 to V_4 with high and pointed T, contributes to the subtotal occlusion of the anterior descending artery.[8]

The fourth aspect illustrated by the ECG is determination of intensity of transmural ischaemia. The occlusion of an epicardial coronary artery, whether originating from atherothrombosis or by balloon inflation during percutaneous coronary intervention, produced in experimental animals,

causes three electrocardiographic patterns (Figure 1B) which correlate with three increasing levels of ischaemic intensity[7]:

– The high and pointed T wave indicates the first level of ischaemia (level 1 ischaemia). If ischaemia does not progress, it can be suspected that the myocardium is completely protected by coronary and extracoronary collateral circulation[9]

– ST segment elevation indicates a more advanced stage of ischaemia (level 2 ischaemia), probably due to a conditioning phenomenon[10]

– The electrocardiographic pattern of S wave disappearance corresponds to a completely unprotected myocardium (level 3 ischaemia). In this level of ischaemia, the most significant and compromising ventricular arrhythmias appear, such as disruption of conduction[10]

The fifth aspect is that the ECG in the preinfarction stage and with ST-T elevation gives invaluable information on coronary anatomy. The high sensitivity and specificity of the ECG with ST elevation are known for determining a suddenly blocked artery which is the originating artery. This information is highly important for haemodynamic laboratories.[11] It also offers useful information on the place of occlusion, considering that the occlusion most proximal or distal to determined collaterals affects the prognosis.[12] The size of the coronary has an effect, and consequently as well as the range of the ischaemic area at risk. Therefore, the ECG provides this valuable information about the patient with acute coronary syndrome.[13] Moreover, the electrocardiogram gives information on critical situations, such as occlusion of the main trunk artery of the left coronary artery,[5] and allows for recognition of intriguing syndromes, such as the one which produces subtotal occlusion of the anterior descending artery.[8]

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