Current electrocardiographic criteria for diagnosis of Brugada pattern: a consensus report

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Abstract

Brugada syndrome is an inherited heart disease without structural abnormalities that is thought to arise as a result of accelerated inactivation of Na channels and predominance of transient outward K current (Ito) to generate a voltage gradient in the right ventricular layers. This gradient triggers ventricular tachycardia/ventricular fibrillation possibly through a phase 2 reentrant mechanism.

The Brugada electrocardiographic (ECG) pattern, which can be dynamic and is sometimes concealed, being only recorded in upper precordial leads, is the hallmark of Brugada syndrome. Because of limitations of previous consensus documents describing the Brugada ECG pattern, especially in relation to the differences between types 2 and 3, a new consensus report to establish a set of new ECG criteria with higher accuracy has been considered necessary. In the new ECG criteria, only 2 ECG patterns are considered: pattern 1 identical to classic type 1 of other consensus (coved pattern) and pattern 2 that joins patterns 2 and 3 of previous consensus (saddle-back pattern). This consensus document describes the most important characteristics of 2 patterns and also the key points of differential diagnosis with different conditions that lead to Brugada-like pattern in the right precordial leads, especially right bundle-branch block, athletes, pectus excavatum, and arrhythmogenic right ventricular dysplasia/cardiomyopathy.

Also discussed is the concept of Brugada phenocopies that are ECG patterns characteristic of Brugada pattern that may appear and disappear in relation with multiple causes but are not related with Brugada syndrome.

Keywords: r’ in V1; ST elevation; Brugada syndrome

Concept

Brugada syndrome (BrS) is a familial, genetically determined syndrome characterized by autosomal dominant inheritance in about 50% of cases with variable penetrance.
More than 70 mutations, most commonly in the cardiac Na\(^+\) channel, have been described. In around 20% of cases, there are SCN5A mutations that explain the accelerated inactivation of Na channels.\(^2\)

The diagnosis of BrS is suggested by the clinical history in a patient with specific electrocardiographic (ECG) pattern (Brugada pattern [BrP]) (Table 2). Sometimes, it is necessary to use other electrocardiographical findings and methods (Table 1B and C).

The number of idiopathic ventricular fibrillation (VF) cases diagnosed as having BrS depends on the ECG diagnostic criteria used. In fact, the ECG manifestation of BrS cases diagnosed as having BrS depends on the ECG criteria used. The ECG criteria are a key point not only for diagnosis but also for prognosis and risk stratification. However, we do not want to discuss all these aspects in this article as we have stated. We plan only to comment on the 12-lead ECG clues for the diagnosis of BrS and how to perform the differential diagnosis with other ECG patterns that present with ST elevation in V1-V2 and/or r\(^{'}\) in these leads. In the presence of the ECG criteria of BrP, BrS is diagnosed if one/more of the following clinical factors are present: (a) survivors of cardiac arrest, (b) presence of polymorphic ventricular tachycardia (VT), (c) history of nonvagal syncope, (d) familial antecedents of sudden death in patients younger than 45 years without acute coronary syndrome, and (e) type 1 ST pattern in relatives.

The prevalence of the symptoms is estimated to be 5 per 10 000 inhabitants in Southeast Asia; and apart from accidents, it is the leading cause of death in men younger than 40 years in this part of the world.\(^3\)

The incidence is higher in young men without apparent structural heart disease, but this concept of the structurally normal heart in BrS has been challenged.\(^4\) Syncope or sudden death usually occurs during rest or sleep, sometimes without any isolated, preceding premature ventricular contractions. Most often, polymorphic ventricular tachycardia triggers ventricular fibrillation possibly because of a phase 2 reentrant mechanism due to a voltage gradient between the different action potential duration of layers of the right ventricle (Fig. 1).

There are currently 2 pathophysiologic hypotheses used to explain the ECG changes in BrS: (a) the repolarization hypothesis,\(^6\) involving the presence of a voltage gradient due to transmural or transregional dispersion of the action potential of different layers of the right ventricle at the beginning of repolarization, as a consequence of a loss of Na current combined with a dominant transient outward K current (\(I_{\text{to}}\)) (Fig. 1) and (ii) the depolarization hypothesis, involving right ventricular conduction delay at the end of repolarization\(^7\) combined probably with subtle structural right ventricular anomalies.\(^8\) This latter mechanism is indirectly supported by the presence of late potentials,\(^9\) fragmented potentials on the anterior aspect of right ventricular outflow tract (RVOT),\(^10\) and delayed right ventricle free wall contraction after ajmaline infusion.\(^11\)

Borggrefe and Schimpf\(^12\) commented that both mechanisms may play a role in the genesis of the ECG pattern and that both may coexist. Elizari et al\(^13\) published the speculative but fascinating theory encompassing the 2

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**Table 1**

Electrocardiogram alterations in Brugada syndrome

<table>
<thead>
<tr>
<th>Type</th>
<th>Characteristics</th>
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<tbody>
<tr>
<td>A. ECG diagnostic criteria</td>
<td>Changes in precordial leads.</td>
</tr>
<tr>
<td>1. Morphology of QRS-T in V1-V3. ST elevation (sometimes only in V1 and exceptionally also in V3) (BrP)</td>
<td>Type 1. Coved pattern: initial ST elevation ≥ 2 mm, slowly descending and concave or rectilinear with respect to the isoelectric baseline, with negative symmetric T wave (see other characteristics in Table 2 and text). Type 2. Saddle back pattern: The high take-off (r(^{'})) is ≥ 2 mm with respect to the isoelectric line and is followed by ST elevation; convex with respect to the isoelectric baseline with elevation ≥ 0.05 mV with positive/flat T wave in V2 and T wave variable in V1. If there is some doubt (ie, r(^{'}) &lt; 2 mm), it is necessary to record the ECG in 2nd, 3rd ICS. Other characteristics may be seen in Table 2 and text.</td>
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<tr>
<td>2. New ECG criteria:</td>
<td>a. Corrado index (2010): Ratio high take-off of QRS-ST/height of ST at 80 ms later is in V1-V2 &gt; 1 because the ST is downsloping. In athletes, the ST especially in V2 is upsloping; and the index is &lt; 1. The end of QRS (J point) often does not coincide with the high take-off of QRS-ST as was suggested by Corrado.(^36) However, using the high take-off of QRS-ST for the application of the Corrado index is valid for discriminating BrP and other conditions mimicking BrP.</td>
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<tr>
<td>b. The (\beta) angle formed by ascending S and descending r(^{'}) is &gt; 58(^\circ) in type 2 BrP (in athletes, it is much lower) (Chevalier et al. 2011) (SE 79% SP 84%).</td>
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<tr>
<td>c. Duration of the base triangle of r(^{'}) at 5 mm from the high take-off is more than 3.5 mm in BrP 2 (SE, 81%; SP, 82%).(^31)</td>
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<tr>
<td>B. Other ECG findings</td>
<td>1. QT generally is normal. May be prolonged in right precordial leads.(^45)</td>
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<tr>
<td>2. Conduction disorders: Sometimes, prolonged PR interval (long HV interval). The conduction delay located in RV explains the r(^{'}) and longer QRS duration in right precordial leads compared with mid/left precordial leads.</td>
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<tr>
<td>4. Some other ECG findings may be seen: the presence of r(^{'}) wave in aVR &gt; 3 mm,(^44) early repolarization pattern in inferior leads,(^46) alternans of T wave after ajmaline injection,(^47) etc.</td>
<td></td>
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<tr>
<td>C. Other electrocardiological techniques: In some occasions, it is necessary or convenient to find some new diagnostic clues with exercise testing,(^48) late potentials,(^49) and impaired QT dynamics studied by Holter. Electrophysiological studies remains controversial for diagnosis and for risk stratification(^50)–(^52).</td>
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**Fig. 1.** In BrS, the heterogeneous dispersion of repolarization takes places between the endocardium and the epicardium of the RV at the beginning of phase 2 (voltage gradient [VG]) because of the transient predominance of outward \(I_{\text{to}}\) current. Epi indicates epicardium; Endo, endocardium.
previous ones proposing that abnormal neural crest cells in the RVOT cause conduction delay, resulting in the typical ST changes of ECG BrP. The repolarization gradients may occur not only between epicardium and endocardium but also between RVOT and normal surrounding myocardium. In fact, the evidence of right ventricular conduction delay and abnormal repolarization, which are already seen in the ECG (at least part of the QRS end and symmetric T wave), are also demonstrated by VCG (delay of final part of QRS loop and rounded T loop). Also Hoogendijk et al., based on the theory that explains ST-segment elevation due to current-to-load mismatch, proposed a unifying mechanism for the Brugada pattern. Although both these unifying theories are scientifically attractive, we need further studies to definitively establish the underlying mechanism of BrS. It is likely that both mechanisms, the RVOT conduction delay and the voltage gradient during phase 2 of repolarization, play a role in the explanation of ECG pattern and in the triggering of VT/VF.

**ECG abnormalities of the BrP**

Firstly, we have to say that the abnormal ECG constitutes the hallmark of BrS; but it is necessary to emphasize that the ECG changes can be dynamic and sometimes are concealed. Indeed, the ECG BrP is sometimes intermittent; and it may be observed only in certain situations, such as fever, intoxication, vagal stimulation, and electrolyte imbalance (Table 1). Furthermore, there are some drugs (sodium channels blockers) that may unmask a BrP. The ECG pattern characteristic of BrS that may arise from multiple causes (metabolic disorders, electrocution, ischemia, etc) and disappear upon resolution of the injury has been named Brugada phenocopy. Although this concept has to be confirmed, it is considered that the Brugada phenocopies have a negative pharmacological challenge test result (to Na channel blockade) and a negative genetic test result for recognized BrS mutations. The prognosis in these cases is unknown. For more details, consult www.brugadadrugs.org.

It is also important to be sure that the ECG is recorded correctly and that there are no artifacts due to the use of inappropriate filters (Fig. 2). However, currently, most ECG devices use linear-phase digital filters that allow cutoff points until 0.67 Hz without inducing ST alterations.

Currently, the most commonly used ECG criteria are the ones proposed by the 2 consensus documents that were published in 2002 and 2005 under the auspices of the European Society of Cardiology. However, the limitations of previous reports, especially in relation to the differences between types 2 and 3 ECG patterns, and the evidence that it is very important to define ECG criteria with a high degree of precision because the decision to implant an ICD depends on it mean that it is necessary to review the current criteria. More recently, the criteria proposed by previous consensus documents have also been used in other articles related to this topic, although, in a Japanese study, it was suggested to combine types 2 and 3 into only 1 type.

We will now describe the most useful current ECG criteria to make this diagnosis and also some other ECG abnormalities that may be found.

**New ECG criteria**

**Morphology in V1-V2**

Only 2 ECG patterns have to be considered (Tables 1 and 2). Type 1 is identical to the classic type 1 described in the...
previous consensus, although we will describe some new parameters to identify it. The new type 2 pattern combines patterns 2 and 3 of previous consensus. These 2 patterns may be unified in only one that encompasses both patterns. In fact, there are only small morphological differences between the 2 of them that do not impact on prognosis and risk stratification. Even with drug challenge, conversion of type 3 to type 2 is considered inconclusive.

Table 2
ECG patterns of BrS in V1-V2

<table>
<thead>
<tr>
<th>Type 1: coved pattern</th>
<th>Type 2: saddle-back pattern</th>
</tr>
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<tbody>
<tr>
<td>High take-off</td>
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</table>

This typical coved pattern present in V1-V2 the following:

a. At the end of QRS, an ascending and quick slope with a high take-off ≥ 2mm followed by concave or rectilinear downsloping ST.

b. There are few cases of coved pattern with a high take-off between 1 and 2 mm.

c. There is no clear r’ wave.

d. The high take-off often does not correspond with the J point (Fig. 4 B).

e. At 40 ms of high take-off, the decrease in amplitude of ST is ≤ 4mm.

f. ST at high take-off ≥ ST at 40 ms ≥ ST at 80 ms.

g. The duration of QRS is longer than in RBBB, and there is a mismatch between V1 and V6 (see text).

This typical saddle-back pattern present in V1-V2 the following:

a. High take-off of r’ (that often does not coincide with J point) ≥ 2 mm.

b. Descending arm of r’ coincides with beginning of ST (often is not well seen).

c. Minimum ST ascent ≥ 0.5 mm

d. ST is followed by positive T wave in V2 (T peak > ST minimum > 0) and of variable morphology in V1.

e. The characteristics of triangle formed by r’ allow to define different criteria useful for diagnosis (see above and text).

f. ST is followed by negative and symmetric T wave • β angle.

• Duration of the base of the triangle of r’ at 5 mm from the high take-off greater than 3.5 mm.

g. The duration of QRS is longer in BrP type 2 than in other cases with r’ in V1, and there is a mismatch between V1 and V6 (see text).

Fig. 3. A, Typical example of BrP type 1. Note the morphology in V1-V2, with a concave ST down-sloping elevation with respect to the isoelectric baseline, with no clear evidence of R’. B, Typical example of type 2 BrP (saddle-back pattern). Note the morphology in V1-V2 with final r’ of special characteristics (Table 2).
Type 1 (Fig. 3A): This pattern, known as a coved pattern, presents as ST-segment elevation followed by a symmetric negative T wave in the right precordial leads. Usually, the pattern is seen only in V1-V2. But in some cases, it is recorded only in V1 or V2; and, in others, it is observed from V1 to V3. Usually, a clear r' is not seen. This typical electrocardiographic pattern that may be considered diagnostic of BrP presents the following characteristics (Table 2):

1) The high take-off of QRS-ST that is at least 2 mm in V1 is followed by downsloping ST-segment elevation concave or rectilinear with respect to the isoelectric line. There are a few cases of coved pattern with high take-off less than 2 mm and at least 1 mm. This high take-off level is higher than ST level after 40 milliseconds, and this is higher than ST after 80 milliseconds (high take-off > ST after 40 milliseconds > ST after 80 milliseconds) (Table 2).

2) At 40 milliseconds from the high take-off of QRS-ST, the decrease in amplitude is less than 0.4 mV. This is much less than the decrease observed in right bundle-branch blocks (RBBBs) because the down-slope is slower.

3) The index QRS-ST elevation at high take-off/height of ST at 80 milliseconds later is greater than 1 in BrP and less than 1 in athletes (Fig. 6). According to Corrado (Fig. 4A), the high take-off QRS-ST coincides with J point (end of depolarization). However, although this phenomenon is not present in all cases (Fig. 4B), this index is still valid (Figs. 4 and 6).

4) The QRS duration is longer in V1-V2 than in mid/left precordial leads because of evidence of right ventricular conduction delay. However, in many cases, it is impossible to measure it in the ECG because we cannot identify exactly the duration of the terminal QRS. Although the VCG may measure with more accuracy the milliseconds of the QRS loop, it is also difficult to ensure the exact duration of the QRS loop because in the first and late part, around 30 to 40 milliseconds, it is slowly inscribed and difficult to measure exactly.

5) The ST segment is followed by an asymmetric T wave.

The ECG BrP type 1 with the above criteria is easily recognized; and when found in a patient without apparent structural heart disease, it strongly supports the diagnosis of BrS (Tables 1 and 2 and later differential diagnosis). The cases with coved-type pattern that present a high take-off of QRS-ST between 0.1 and 0.2 mV but with negative T wave in V1-V2 have been considered suggestive of type 1. These cases are very infrequent; and to confirm the diagnosis, some of the other ECG findings listed in Table 1 should be present.

Type 2 (Fig. 3B): It is characterized in V1 and V2 with the presence of terminal positive wave called r', although in fact, it is a mixture of final QRS and beginning of repolarization. This r' presents a high take-off of at least 0.2 mV of amplitude, followed by elevated ST segment (≥0.5 mm) convex with respect to the isoelectric line (saddle-back pattern) and by a T wave that is positive in V2 (amplitude of the peak of the T > minimum amplitude of ST) and of variable morphology in V1: mildly positive, flat, or mildly negative (Fig. 3B and Table 2). It has been considered that the descending limb of the r’ terminates when a sudden change in the slope occurs. However, sometimes, especially in V1, there is no clear change in the slope direction so that the T wave onset cannot be determined.

In this scenario, it is important to verify whether intravenous administration of a drug that blocks the sodium channels (ajmaline, flecainide, procainamide, pilsicainide) turns the type 2 pattern into type 1 (Fig. 5).

The characteristics of the “r’” in BrP are different than in RBBB and other conditions with r’ in V1-V2. In a BrP ECG,
the high take-off of r’ is not peaked; and the descending arm of r’, considering the “r’’” as a triangle, has a gradual slope. The r’ of BrP type 2 compared with other types of r’ has the following features:

1. The angles between both arms of r’ are wider (α and β angle). The sensitivity (SE) and specificity (SP) are higher in β angle (79% and 84%, respectively) with a threshold of 58º (Table 2).

2. The measurement of the duration of the base of triangle of r’ at 5 mm from the high take-off is more than 3.5 mm in BrP type 2, with an SE of 81% and an SP of 82%. This criterion is easier to measure than the α and β angles (Table 2).

Accuracy of these ECG criteria

The use of these ECG criteria is very helpful for the diagnosis of BrP that may be undertaken even by nonexperienced physicians, especially type 1 ECG pattern that has a high specificity.

Recently, in a Japanese population, a computerized diagnostic criteria for BrS that demonstrated a high level of accuracy have been published (type 1 >90%, type 2 >85%). This study was based on the comparison with RBBB in apparently healthy people but not with athletes and people with pectus excavatum.

Other ECG findings or ECG techniques may be used for correct diagnosis of BrS

Table 1B and C shows the most important other ECG findings that may be used for correct diagnosis of BrS.

Changes of morphology according the location of the electrodes

In some cases, the ECG pattern is only evident or is more evident in the upper precordial V1-V2 leads recorded at the third or second intercostal space. This occurs because the abnormal electrical activity leading to BrP arises from a limited zone located in the RVOT. Accordingly, only an electrode located exactly over the affected zone is able to record the BrP. Recently, it has been published that, in BrP type 1, lead positioning according to RVOT location by using CV magnetic resonance imaging correlations may improve the diagnosis of BrP. The ECG pseudonormalization observed when the electrodes are located in the correct position does not constitute any guarantee that the diagnosis is incorrect, especially if some ST elevation persists, albeit less markedly (Fig. 5). Therefore, it is very important to position the electrodes in the same location when serial recordings are performed to allow comparative assessments, and to perform ECG recordings in upper precordial leads if some suggestion of BrP exists in V1-2 in the ECG recorded at the fourth intercostal space, such as minimal ST elevation and/or positive terminal forces of the QRS.

Variability of BrP over time

In most patients, BrP is not evident at any time, changing from the most obvious type 1 to a barely recognizable type 2. In a follow-up study based on 90 ICD patients with BrS, one third of analyzed tracings showed type 1 BrP and one third showed perfectly normal ECG tracings. This is not surprising because the sodium channel dysfunction underlying BrP is variable, being influenced by different factors (drugs, fever, autonomic imbalance, etc).
3. Differential diagnosis

We have to distinguish (a) the cases of spontaneous typical BrP type 1 in case of BrS; (b) the typical BrP type 1 induced by some drugs (sodium channel blockers) or other circumstances (fever, etc) that unmask a BrS; (c) the cases of ECG BrP especially type 1 induced by many circumstances that disappear upon resolution of the injury and that do not present BrS (named phenocopies) (These include acute ischemia, pericarditis, myocarditis, pulmonary embolism, metabolic disorders, ionic disorders, administration of some drugs, electrocution, and a miscellaneous group [consult www.brugada.org and Baranchuk et al20]); and (d) the cases of similar BrP (Brugada-like) that are permanent patterns and may be confused with a BrP type 1 or 2. These include RBBB, septal hypertrophy, arrhythmogenic right ventricular dysplasia/cardiomiopathy, athletes, pectus excavatum, and a long number of processes (consult www.brugada.org) (Fig. 6).

Sometimes, the differential diagnosis of BrP from other conditions associated with ST elevation by ECG alone may be quite difficult, even for the more experienced cardiologist.34 The clinical setting is very important, bearing in mind that, in many cases, the patient with this problematic pattern presents with acute symptoms. Patients with acute coronary syndromes with ST elevation may present in some cases a similar pattern but obviously in a very different clinical setting. In some doubtful cases of type 2 pattern, to determine the correct diagnosis, we may check if there are changes after the intravenous administration of Na channel blockers (ajmaline) (already discussed). There are however frequent cases of Brugada-like pattern, especially type 2, in which the ECG is usually seen in asymptomatic patients that require a detailed ECG analysis to obtain the correct interpretation. We will comment on the most challenging cases (Fig. 6).

Isolated RBBBs

Type 1 BrP and advanced RBBB are both characterized by a terminal positive wave and a negative T wave in V1-V2.

The distinction between the 2 conditions is straightforward because, usually, in advanced RBBB, the ST segment is not elevated in the right precordial leads, the terminal wave (r' or R') is synchronous with the broad S wave observed in leads I and V6, and the QRS is wider (≥ 120 milliseconds). In type 1 BrP, however, usually no wide S wave is present in the left leads because the terminal forces of the ventricular complex in V1-V2 can be recorded only by electrodes placed in proximity of the site where the abnormal electrical activity occurs (the outflow tract of the right ventricle) and not from further away leads. Therefore the QRS in left leads is usually less than 120 milliseconds.

Type 2 BrP and incomplete RBBB raise more problems: whenever a small positive terminal QRS deflection (r’) is present in V1-V2, it is possible to distinguish the r’ wave of incomplete RBBB from the terminal positive r’ wave of type 2 BrP on the basis of the following: (1) the positive terminal r’ wave is peaked in RBBB, whereas, in BrP, type 2 is rounded, wide, and usually of relatively low voltage; (2) in incomplete RBBB, the QRS complex duration in leads V1-V2 is identical to that observed in lead V6. In type 2 BrP, in contrast, the QRS duration is longer in the right precordial leads than in V6 because the terminal deflection observed in V1 (the r’ wave) cannot be recorded by the V6 electrode. In other words, in BrP, the QRS complex end is earlier in V6 than in V1-V2.35

Athletes

Type 1 BrP compared with athletes (Fig. 6) depicts an ST segment clearly elevated and down-sloping, usually with no visible r’. This pattern is difficult to be mistaken with the ECG pattern seen in V1-V2 in athletes.

In type 2 BrP, the high take-off is an r’ usually rounded (see characteristics of r’ before), which is followed by a down-sloping ST elevation with a final T wave of variable morphologies in V1 and positive in V2 (Table 2).

Fig. 6. Comparison between type 1 and 2 BrP, arrhythmogenic right ventricular dysplasia, pectus excavatum, athletes, and partial RBBB to view the presence of mismatch in the QRS duration in V1-V2 and mid/left precordial leads. There is clear mismatch in type 1 and 2 of BrP, and there is none in the other cases. The second vertical line is located at 80 milliseconds of the J point (first line) to check the Corrado index. Color illustration online.
Compared with this pattern, the ECG of athletes presents the following:

1. The end of the QRS in V1 coincides with the end of the QRS in V5-6, whereas in type 2 BrP, this is not usually clearly seen because the QRS complex ends later in V1-V2 than in V6 (see above).

2. In athletes, lead V1 shows an r' usually peaked and sharp with no ST elevation or only mildly elevated (<1 mm); and the ST segment starts after the clear end of QRS and is often followed by a negative and sometimes deep T wave in V1. Occasionally, the T wave is positive, especially in V2; but in these cases, the ST segment is gradually ascending (Fig. 6). Therefore, the index ratio ST-segment elevation at high take-off of QRS-ST/ST gradient immediately following positive QRS complexes at the onset of the ST segment, recorded especially in the midleft precordial (not right precordial) and sometimes inferior leads. The transition from QRS to ST segment is considered “slurred” and not a clear J point (wave) when the R wave gradually becomes the ST segment with upright concavity.41 The characteristics of this ECG pattern are very different from BrP, but sometimes both patterns may coexist.42

**Pectus excavatum**

The ECG diagnosis of this normal variant of the ECG seen in pectus excavatum37 is supported by the following:

- A negative P wave in V1 taken in the correct location (fourth ICS)
- A peaked r' in V1 very well defined, which may be followed by an elevated ST usually mild. The T wave is usually negative or plus/minus in V1 and positive in V2 (Fig. 6).

**Arrhythmogenic right ventricular dysplasia/cardiomyopathy**

From an electrocardiographic point of view, it is usually characterized by an ECG pattern in V1-V238 different from BrP by the following:

- Atypical RBBB morphologies have been shown, with a “plateau” in the R wave in lead V1. The ST segment is at times somewhat elevated but does not usually mimic type 2 BrP, although, in some cases, it may be similar because the ε wave may be confused with the r’ wave of BrP.
- In arrhythmogenic right ventricular dysplasia/cardiomyopathy, moreover, T waves are more frequently negative in many precordial leads (V1 to V3-V5).

The ECG pattern is always fixed.

The signal averaging technique may help to differentiate both processes because although in both of them abnormal high-frequency components are present (positive late potentials), wavelet analysis demonstrates that the frequency level is higher in arrhythmogenic right ventricular dysplasia/cardiomyopathy.39

**Early repolarization pattern**

The early repolarization variant is a well-recognized common enigmatic idiopathic electrocardiographic phenomenon, considered to be present when at least 2 adjacent precordial leads show ST-segment elevation with values of at least 1 mm. This pattern, which has recently been associated to ventricular fibrillation (sudden death),40 may be defined as a positive, sharp, and well-defined “hump-like” deflection immediately following positive QRS complexes at the onset of the ST segment, recorded especially in the midleft precordial (not right precordial) and sometimes inferior leads. The transition from QRS to ST segment is considered “slurred” and not a clear J point (wave) when the R wave gradually becomes the ST segment with upright concavity.41 The characteristics of this ECG pattern are very different from BrP, but sometimes both patterns may coexist.42

**Conclusions**

We have exposed the current ECG criteria of BrP, including the ones described after the last consensus statement,24,25 which have very high accuracy and could be used by nonexperienced physicians for a reliable diagnosis of both types of BrP, especially type 1. The most important points are the following:

1. Only 2 ECG patterns should be considered: type 1 (coved type) and type 2 (saddle-back type) that encompass the patterns 2 and 3 of previous consensus (Table 2).

2. In both types 1 and 2, the end of the QRS in the mid/left precordial leads (J point) occurs before the end of the QRS in V1 due to localized RVOT conduction delay. However, it is impossible to confirm by surface ECG the exact difference because of the impossibility of determining exactly the end of QRS in V1. Furthermore, often, the J point in mid/left precordial leads does not coincide with the high take-off of the QRS-ST in V1,29 as was considered by Corrado et al.36

3. Type 1 pattern is very specific; and when it appears in a patient without apparent heart disease, it strongly suggests the diagnosis. The small number of cases with no typical pattern (ST elevation <2 mm) has to be evaluated with complementary ECG criteria test to reach a final diagnosis (Table 1B and C).

4. Electrocardiographic patterns identical to BrP can be seen in the absence of true BrS (phenocopies).

5. In BrP, the slope of ST in V1-V2 is descending; and in athletes and other cases with r’ in V1, it is usually ascending at least in V2 (Fig. 6).

6. Type 2 pattern may be confused with several other situations with r’ in V1-V2. The characteristics of r’ in incomplete RBBB, athletes, and pectus excavatum however are especially different from those observed in BrS. These differences include (Table 2) (a) the angle of ascending and descending arms of r’; (b) the duration of the base triangle of r’ at 5 mm from the high take-off; and (c) the configuration of r’ is peaked in RBBB, pectus excavatum, and athletes and rounded in BrP. The Corrado index can also be helpful in distinguishing BrP from athlete’s ECG (Table 2 and Fig. 6).