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Letters to the editor

OVERVIEW

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Brugada phenocopies are the leading differential diagnosis of Brugada syndrome

Editor – We read the literature review on Brugada syndrome (BrS) by Sheikh and Ranjan with great interest (*Clin Med* 2014;5:482–9). Their manuscript provides a concise review of BrS and identifies associated differential diagnoses. We would like to direct the authors to our work on Brugada phenocopies (BrP).[1] BrP are the leading differential diagnosis of true congenital BrS. Many of the underlying BrS differential diagnoses that the authors mention are actually various underlying etiologies of BrP. We would like to provide the authors with a brief explanation of BrP and its clinical distinction from BrS.

BrP are clinical entities that present with identical ECG patterns to those of true congenital BrS but are elicited by various other clinical circumstances.[1] Conditions that induce BrP are characterised into six etiological categories: metabolic conditions, mechanical compression, myocardial ischemia and pulmonary embolism, myocardial and pericardial disease, ECG modulations and miscellaneous.[1] Our international registry and online educational portal provides an updated registry of BrP cases along with the diagnostic criteria.[2]

Many of the conditions that the authors mention as part of their differential diagnosis for a Brugada ECG pattern are confirmed causes of BrP, including acute pericarditis, myocardial ischemia, pulmonary embolism, hyperkalemia, hypercalcemia, hypothermia and pectus excavatum. BrP form a group of heterogeneous conditions that are perhaps the most difficult to differentiate from true congenital BrS due to identical ECG patterns. Distinguishing between BrP and BrS relies on a series of clinical and ECG characteristics (Table 1). A systematic diagnostic approach to differentiate between BrP and BrS is imperative because the same clinical condition can elicit BrP or unmask true congenital BrS. For example, hyperkalemia has been associated with BrS[3] and BrP.[2] Similarly, myocardial ischemia has been found to unmask true BrS patients[4] and also present as BrP.[2] Important criteria that differentiate BrP and BrS are that patients with BrP have a negative provocative challenge with a sodium channel blocker, along with an absence of clinical history suggestive of a sudden cardiac death syndrome.

We encourage authors to use the term Brugada phenocopy for consistency in the literature and to facilitate future research. ■

Table 1. Clinical features indicative of BrP and differentiation from BrS.

BrP	> ECG pattern of either type-1 or type-2 Brugada morphology
	> Patient has an underlying condition that is identifiable
	> Resolution of the ECG pattern upon resolution of the underlying condition
	> Low clinical pretest probability for BrS determined by a lack of symptoms, medical history and family history
	> Negative provocative test with sodium channel blocker such as ajmaline, flecainide or procainamide
	> Negative genetic testing results
BrS	> ECG pattern of either type-1 or type-2 Brugada morphology; pattern may be 'masked' under normal conditions
	> High clinical pretest probability for BrS determined by a lack of symptoms, medical history and family history
	> Positive provocative test with sodium channel blocker such as ajmaline, flecainide or procainamide indicating sodium channel dysfunction consistent with BrS
	> Positive genetic testing results

BrP = Brugada phenocopies; BrS = Brugada syndrome.

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3 Postema PG, Vlaar AP, DeVries JH, Tan HL. Familial Brugada syndrome uncovered by hyperkalaemic diabetic ketoacidosis. *Europace* 2011;13:1509–10.
4 Gottschalk BH, Anselm DD, Baranchuk A. Brugada phenocopy induced by ischemia or Brugada syndrome unmasked by ischemia? *Int J Cardiol* 2014;177:619–20.

Response

Editor – We read with great interest the letter from Gottschalk *et al*, and we would like to thank them for their interest in our review article. We are grateful to the authors for drawing our attention to the terminology 'Brugada phenocopy' (BrP) and their work on BrP.

Riera *et al*[1] introduced the term 'Brugada phenocopy' to describe the Brugada pattern that can be linked to a pre-existing and well-known condition. They chose this term based on a previous definition of phenocopy: 'an environmental condition that imitates (copies) one produced by a gene'. The authors described a classic Brugada-type 1 ECG pattern in a patient intoxicated with propofol. In this particular case, the environmental condition was the infusion of propofol that triggered this particular ECG manifestation.

BrP are clinical entities that are etiologically distinct from true congenital BrS. BrP are defined by ECG patterns that are identical to BrS but are elicited by various clinical circumstances.

There are few key features that helps in distinguishing between BrP and the true congenital BrS.[2] First, patients with BrP have a reversible underlying condition, such as adrenal insufficiency, hypokalemia or myocardial ischemia, that elicits or induces the Brugada ECG pattern. There is prompt normalisation of the ECG once this underlying condition is resolved. This is contrary to true congenital BrS where the ECG manifestations are unmasked by sodium channel blockers, vagotonic agents, febrile states and various metabolic conditions. Second, patients with BrP have a low clinical pretest probability of true congenital BrS, as opposed to a high clinical pretest probability in patients with true congenital BrS who have a documented personal history of cardiac arrest, non-vagal syncope or a family history of sudden cardiac death.[3] Third, patients with BrP have a negative provocative challenge with a sodium channel blocker, while those with true congenital BrS have a positive provocative challenge.[2]

Gottschalk *et al* recently developed a morphological classification system which divides BrP into type-1 and type-2 BrP according to the manifested ECG pattern. The type-1 BrP is identical to a coved or type-1 Brugada ECG pattern and the type-2 BrP is identical to a saddleback or type-2 Brugada ECG pattern.[4–6] These two categories include A, B, and C qualifiers. Class A includes BrP that have met all mandatory diagnostic criteria, including negative provocative challenge with a sodium channel blocker. Class B includes highly suspected BrP; however, not all mandatory diagnostic criteria are complete. Class C includes highly suspected BrP; however, provocative testing is not justified, such as in cases with recent surgical right ventricular outflow tract manipulation[7] or BrP secondary to inappropriate ECG high pass filters.[8] The systematic diagnostic criteria discussed needs to be applied for suspected cases of BrP.

We agree with the authors that the term 'Brugada phenocopy' should be used to replace 'Brugada-like ECG pattern' in the absence of true Brugada syndrome, in order to achieve consistency in the literature. ■

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A new kid on the block: the role of physician associates

Editor – You have previously highlighted the potential benefits of the deployment of physician associates (PAs) to emergency departments in the UK (*Clin Med* 2014;3:219–20). Tamara Ritsema subsequently elaborated on the scope of practice of PAs working in UK emergency departments from a national perspective. (*Clin Med* 2014;6:691–4). To expand on this further, I offer a brief case study of PAs working in a UK emergency department.

In July 2011, a district general hospital in the West Midlands recruited three UK-trained PAs to join a US-trained PA already working in emergency medicine. The Trust has two acute hospital sites, each with an emergency department and two PAs were placed in each of these. On one site the PAs were part of the junior doctor rota, taking gaps that would have been filled by locum doctors, while at the second site they had a separate rota. Each had a designated consultant acting as an educational supervisor and on a day-to-day basis worked under the supervision of the consultant and registrars in the department.

All four PAs could work across the departments, seeing undifferentiated patients presenting to 'minors', 'majors', 'paediatrics' and 'resuscitation', taking patient histories, undertaking physical examination, ordering and interpreting diagnostic tests and procedures, formulating a diagnosis and initiating management, or referring to speciality as