World Journal of Cardiology

Online Submissions: http://www.wjgnet.com/1949-8462office
wjc@wjgnet.com
doi:10.4330/wjc.v3.i5.127

World J Cardiol 2011 May 26; 3(5): 127-134
ISSN 1949-8462 (online)

EDITORIAL

Differential diagnosis of tachycardia with a typical left bundle branch block morphology

Jeffrey S Neiger, Richard G Trohman

Jeffrey S Neiger, Richard G Trohman, Department of Medicine, Section of Cardiology, Clinical Cardiac Electrophysiology Service, Rush University Medical Center, Chicago, IL 60612, United States
Author contributions: Neiger JS and Trohman RG contributed solely to this paper.
Correspondence to: Richard G Trohman, MD, Department of Medicine, Section of Cardiology, Clinical Cardiac Electrophysiology Service, Rush University Medical Center, 1750 W. Harrison St., Suite 983 Jelke, Chicago, IL 60612, United States. rtrohman@rush.edu
Telephone: +1-312-9426858 Fax: +1-312-9425862
Received: March 12, 2011 Revised: April 13, 2011
Accepted: April 20, 2011
Published online: May 26, 2011

Abstract

The evaluation of wide QRS complex tachycardias (WCT) remains a common dilemma for clinicians. Numerous algorithms exist to aid in arriving at the correct diagnosis. Unfortunately, these algorithms are difficult to remember, and overreliance on them may prevent cardiologists from understanding the mechanisms underlying these arrhythmias. One distinct subcategory of WCTs are those that present with a "typical" or "classic" left bundle branch block pattern. These tachycardias may be supraventricular or ventricular in origin and arise from functional or fixed aberrancy, bystander or participating atriofascicular pre-excitation, and bundle branch reentry. This review will describe these arrhythmias, illustrate their mechanisms, and discuss their clinical features and treatment strategies.

Key words: Typical left bundle branch block; Wide complex tachycardia; Bundle branch reentrant ventricular tachycardia

Peer reviewers: Jacob Tfelt-Hansen, MD, PhD, Laboratory of Molecular Cardiology, Copenhagen University Hospital, section 9312, Juliane Mariesvej, 20 2100 Copenhagen Ø, Denmark; Brian Olshansky, MD, Professor of Medicine, Cardiac Electrophysiology, University of Iowa Hospitals, 200 Hawkins Drive, Room 4426a JCP, Iowa City, IA 52242, United States

Neiger JS, Trohman RG. Differential diagnosis of tachycardia with a typical left bundle branch block morphology. *World J Cardiol* 2011; 3(5): 127-134 Available from: URL: http://www.wjgnet.com/1949-8462/full/v3/i5/127.htm DOI: http://dx.doi.org/10.4330/wjc.v3.i5.127

INTRODUCTION

The evaluation of wide QRS complex tachycardia (WCT) remains a common clinical dilemma[1]. The process of determining the correct diagnosis can be confusing and cumbersome. Even the presence of hemodynamic stability is not helpful in determining the tachycardia mechanism.

Most physicians understand that the pivotal diagnostic challenge focuses on determining whether the tachyarrhythmia is of supraventricular (with aberrant ventricular conduction) or ventricular origin. While the presence of atrioventricular (AV) dissociation during wide complex tachycardia is highly specific for a ventricular origin, this finding is often not present or is difficult to discern on the surface electrocardiogram (ECG). Complex algorithms have evolved (from 1978 to 2008) to assist the treating physician in making this distinction (Table 1)[2-6]. Many of these algorithms use specific features of the QRS complex, and subcategorize tachycardias into "right bundle branch-like" and "left bundle branch like" morphologies. However, committing these algorithms to memory can be challenging and overreliance on them may impede cardiologists from understanding the underlying tachycardia mechanism.

This review will focus on the subcategory of WCTs that present with a typical (or "classic") left bundle branch block (LBBB) pattern. These tachycardias may be

supraventricular or ventricular in origin and arise from functional or fixed aberrancy, bystander or participating atriofascicular pre-excitation, and bundle branch reentry. We describe the types of tachycardia associated with a typical LBBB morphology, discuss their clinical features and provide diagrams to elucidate their mechanisms.

DEFINITIONS

It is important to understand the distinction between "LBBB-*like*" patterns and a typical LBBB morphology. LBBB is defined as a prolonged QRS duration (≥ 120 ms) with broad monophasic R waves in leads I, V5 and V6 that are usually notched or slurred. There is delayed onset of the intrinsicoid deflection (the beginning of the QRS to the peak of the R wave is > 50 ms) in leads I, V5 and V6. There are secondary ST and T-wave changes in the opposite direction of the major QRS deflection[7]. Most algorithms define LBBB-like morphology as a QRS complex that is predominantly negative in the right precordial leads (specifically, V1) and predominantly positive in the lateral leads (I, aVL, V5, V6)[3]. In typical, or "classic" LBBB, lead V1 will demonstrate either an rS or QS complex, and, more importantly, Q waves will be absent from the left lateral leads. Kindwall and colleagues used the following criteria to distinguish ventricular tachycardia (VT) from supraventricular tachycardia (SVT) with aberrant conduction in patients with LBBB-like morphology: (1) R wave in lead V1 or V2 > 30 ms; (2) any Q wave in V6; (3) a duration of ≥ 60 ms from the onset of the QRS to the nadir of the S wave in V1 or V2; and (4) notching of the downstroke of the S wave in V1 or V2[3].

The ECG appearance of typical LBBB depends on antegrade ventricular activation occurring *via* the right bundle branch. In most instances, activation of the right bundle branch occurs *via* the AV node and His bundle. Atriofascicular accessory pathways bypass the AV node and His bundle and insert directly into (or extremely close to) the right bundle branch. The interventricular septum is depolarized *via* the His-Purkinje system with *subsequent* activation of the left ventricle. Because there is no antegrade conduction *via* the left bundle branch, the electrical impulse must propagate in a cell-to-cell fashion, resulting in delayed conduction to the left ventricle. Q waves will not be seen in the lateral leads on a surface ECG (Figure 1). In contrast, LBBB-like morphologic tachycardias are not confined to the specialized conduction system. They arise from the ventricular myocardium. Electrical impulses travel in a delayed fashion, and may propagate rightward (away from the lateral leads) before exciting the bulk of the left ventricle (Figure 2)[8]. This frequently results in a QRS complex that is wider than a typical LBBB aberrant complex, and explains why Q waves may be recorded in the left-sided ECG leads[9].

The differential diagnosis of a WCT with a typical LBBB morphology is limited to five entities: SVT with fixed LBBB, SVT with functional aberrancy, pre-excited reentrant tachycardias using an atriofascicular accessory pathway as the antegrade limb of the circuit (the retrograde limb is usually the normal ventriculo-atrial conduction system, but may be a second accessory pathway), SVT with a "bystander" atriofascicular pathway and bundle branch reentrant VT (Table 2).

Most of the above-mentioned tachycardias have a reentrant mechanism. Exceptions are limited to physiologic (non-reentrant) sinus tachycardia and automatic atrial tachycardias. Reentrant tachycardias involve continuous propagation of an activation wavefront. Three requirements must be met in order for reentry to occur. First, there must be two anatomically adjacent pathways of myocardial tissue to form a circuit. Differences in the pathways' electrophysiologic properties (refractoriness and conduction velocity) are also a requisite for reentry. Unidirectional conduction block must be present, otherwise the excitation wavefronts travelling down both limbs of the reentrant circuit will collide and extinguish each other. Finally, there must be an area of slow conduction within the circuit in order to allow enough time for previously refractory tissue to regain its excitability before the reentrant wavefront arrives to depolarize it again.

SVT WITH FIXED OR FUNCTIONAL LBBB

Any form of SVT, including sinus tachycardia, that propagates *via* the normal conduction system will demonstrate a typical LBBB morphology in patients with a preexisting (baseline) LBBB. In these patients, a resting ECG in sinus rhythm that demonstrates LBBB will quickly aid in the diagnosis. Such patients often have underlying cardiac disease.

Alternatively, a patient may have a narrow QRS complex during normal sinus rhythm but may develop tachycardia-dependent physiologic (functional) LBBB aberration (intermittent or transient LBBB) (Figure 3)[10]. In this case, the first aberrant complex results from encroachment on the refractory period of the left bundle branch of the previous complex (Figure 4)[11]. Aberration may also result from concealed retrograde penetration (e.g. from a premature ventricular contraction (PVC) originating in the left ventricle) into the left bundle branch rendering it refractory to subsequent beats. Repetitive transseptal retrograde concealed penetration from impulses conducting antegrade *via* the contralateral (right) bundle perpetuates local refractoriness or results in repetitive impulse collision (Figure 5). Other causes of transient BBB such as acceleration-dependent or bradycardia-dependent block result from disease in the His-Purkinje system and should be regarded as abnormal[12].

Abrupt recovery (facilitation) of normal conduction may result when a PVC excites the left bundle early and allows more time for it to recover (peeling back its refractoriness). The refractory period may also shorten because the PVC shortens the cycle length before (and thus the refractory period of) the next spontaneous impulse. In either case, the PVC ends the "linking" sequence created by repetitive concealed transseptal impulses[13].

SVT with fixed or functional aberrancy accounts for approximately 15%-20% of WCTs, and is a significantly

Table 1 Electrocardiographic QRS morphology criteria favoring ventricular tachycardia over supraventricular tachycardia

Authors	Date	Morphology	Criteria favoring ventricular tachycardia
Wellens *et al*[2]	1978	RBBB-like	Monophasic R in V1
			qR, QS, RS in V1
			rS, QS, qR in V6
			R/S < 1 in V6 (S > R or QS in V6)
			Left axis deviation
			QRS width > 140 ms
Kindwall *et al*[3]	1988	LBBB-like	R in V1 or V2 > 30 ms
			Any Q wave in V6
			Onset of QRS to nadir of S ⩾ 60 ms in V1 or V2
			Notching of downstroke of S in V1 or V2
Akhtar *et al*[4]	1988		Positive QRS concordance across the precordium
			Extreme left axis deviation (-90° to ± 180°)
		LBBB-like	Right axis deviation
			QRS > 160 ms
		RBBB-like	QRS > 140 ms
Brugada *et al*[5]	1991		Absence of RS complex in all precordial leads
			R to S interval > 100 ms in ⩾ one precordial lead
			Wellens' morphologic criteria in leads V1 or V6
Vereckei *et al*[6]	2008		Initial R wave in lead aVR
			Initial r or q wave > 40 ms in lead aVR
			Notch on descending limb of negative onset, predominantly negative QRS in lead aVR
			vi/vt ⩽ 1

vi/vt: Ratio of voltage amplitude during initial 40 ms of QRS complex relative to terminal 40 ms in any lead with a bi- or multiphasic QRS complex; LBBB: Left bundle branch block; RBBB: Right bundle branch block.

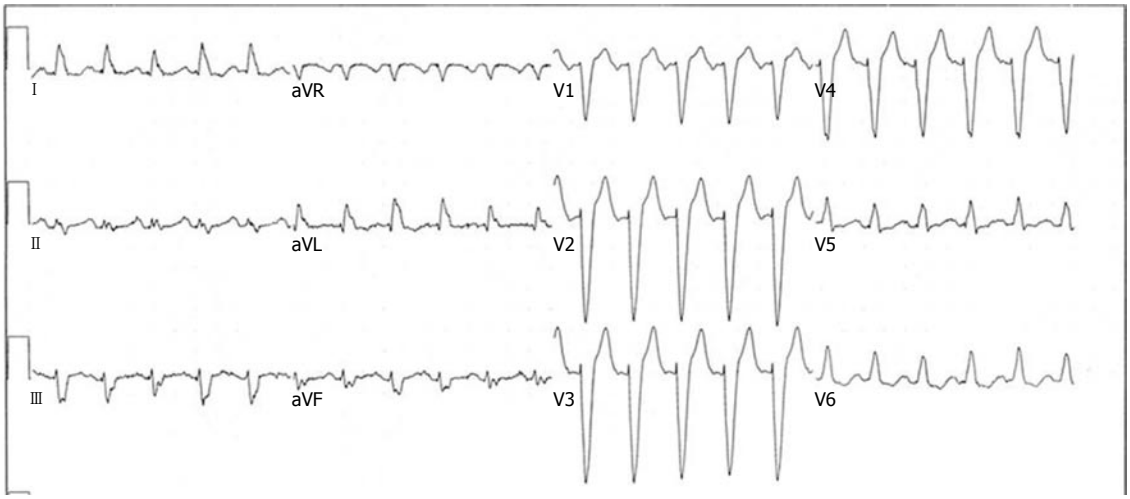

Figure 1 Electrocardiogram example of typical left bundle branch block pattern in a patient with sinus tachycardia. Lead V1 demonstrates an rS complex, while there are monophasic, notched R waves in leads I, aVL, V5 and V6. Q waves are absent in these leads.

more common cause of WCT in patients under the age of 35[14]. Although any SVT may exhibit functional LBBB, it is most commonly seen during orthodromic AV reentry tachycardia with antegrade conduction *via* the AV node and right bundle branch and retrograde conduction *via* a left free wall accessory pathway (Figure 6)[15,16].

ATRIOFASCICULAR ACCESSORY PATHWAY AND SVT WITH A BYSTANDER ATRIOFASCICULAR PATHWAY

Atriofascicular fibers originate in the right atrial free wall and insert into the distal part of the RBB or the adjacent ventricular myocardium. The tissue of these fibers is functionally similar to that of the AV node, and they demonstrate decremental conduction and a Wenckebach-type response to rapid atrial pacing. In addition, they are sensitive to adenosine[17].

Approximately 6% of patients presenting with SVT with a typical LBBB morphology have been found to have an atriofascicular bypass tract[12]. In such cases, antidromic AV reentrant tachycardia (AVRT) results from antegrade conduction down the bypass tract and retrograde propagation *via* the normal conduction system (Figure 7A)[18]. Orthodromic AVRT almost never occurs, because these

Table 2 Differential diagnosis of tachycardia with a typical left bundle branch block QRS morphology

Arrhythmia	ECG and clinical features
SVT with fixed left bundle branch block	LBBB present on baseline ECG QRS during tachycardia usually an identical match
SVT with functional LBBB aberrancy	Most often due to orthodromic AVRT At rapid rates, QRS alternans may be present
Atriofascicular antidromic tachycardia	Preexcitation may be minimal or absent during sinus rhythm Late QRS transition, leftward axis common Frequently coexists with other accessory pathways or AV nodal reentry
SVT with bystander atriofascicular accessory pathway	Accessory pathway does not participate in reentrant circuit of orthodromic AVRT, AVNRT, or atrial tachycardias (including atrial fibrillation and flutter)
Bundle branch reentrant ventricular tachycardia	Associated with acquired structural heart disease (cardiomyopathy, valvular disease) Prolonged PR interval and nonspecific IVCD often present during sinus rhythm

SVT: Supraventricular tachycardia; AVRT: Atrioventricular reentrant tachycardia; IVCD: Intraventricular conduction defect; AVNRT: Atrioventricular nodal reentrant tachycardia.

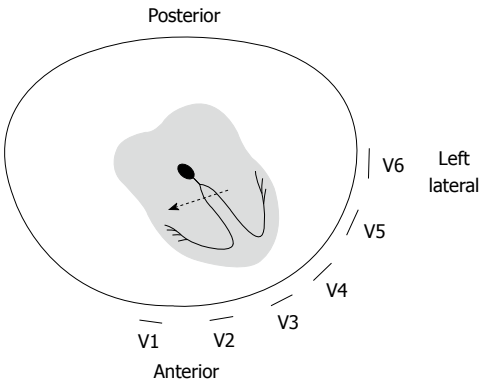

Figure 2 Mechanism of left bundle branch block-*like* electrocardiogram morphology. Electrical activation arises from outside the specialized conduction system and travels rightward before activating the left ventricle. This results in a Q wave in the left lateral electrocardiogram leads. Adapted from[8], with permission.

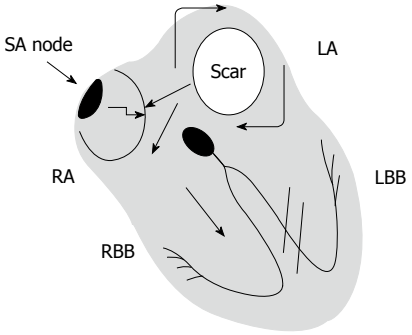

Figure 3 Atrial tachycardia can result in functional left bundle branch block. A reentrant circuit in the left atrium results in tachycardia that rapidly conducts to the ventricle *via* the atrioventricular node. Rate-dependent conduction block occurs in the left bundle branch. Adapted from[10], by permission of Oxford University Press, Inc. LBB: Left bundle branch; RBB: Right bundle branch.

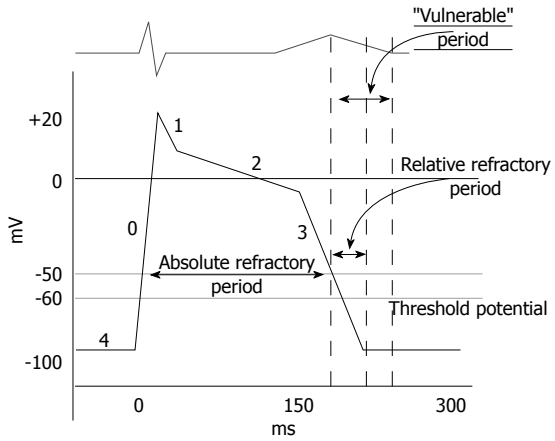

Figure 4 Typical action potential from within the His-Purkinje system. If this potential was from the left bundle branch, impulses that occur (encroach upon) the absolute refractory period will not excite the tissue. A new action potential will not occur and (assuming it is not also refractory) conduction will occur solely through the right bundle branch. Adapted from[11], with permission.

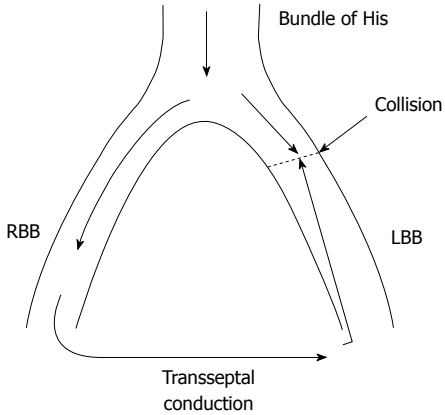

Figure 5 Mechanism of linking in functional left bundle branch block aberrance. Repetitive transseptal retrograde concealed penetration from impulses conducting antegrade *via* the right bundle perpetuates local refractoriness or results in repetitive impulse collision. LBB: Left bundle branch; RBB: Right bundle branch.

bypass tracts generally do not conduct in a retrograde direction. It is not uncommon for atriofascicular fibers to co-exist with other accessory pathways which may serve as the retrograde limb of a pre-excited (typical) LBBB tachycardia or become apparent after ablation of the atriofascicular pathway[19]. Antegrade conduction *via* most right-sided accessory pathways will produce a LBBB-*like* morphology on the ECG. However, since the vast major-

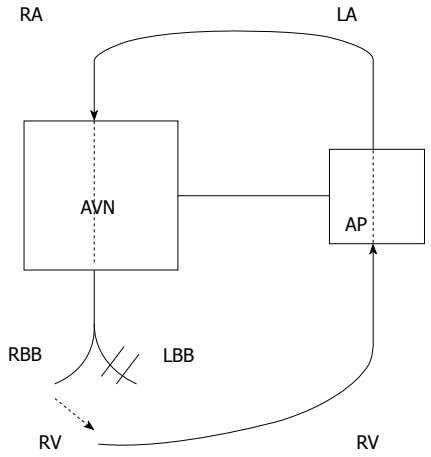

Figure 6 Illustration of orthodromic AV reentry tachycardia resulting in a wide QRS tachycardia with left bundle branch block morphology. A left sided accessory pathway is present. During tachycardia, antegrade conduction is *via* the atrioventricular node and right bundle branch (RBB), and retrograde conduction is *via* the accessory pathway. Adapted from[15], with permission. LBB: Left bundle branch.

ity of these pathways do not insert into the right bundle branch, antidromic conduction during AVRT will not demonstrate the features of typical LBBB.

Atriofascicular bypass tracts may coexist with other SVTs that do not require an AV bypass tract for initiation and maintenance. Therefore, during AV nodal reentrant tachycardia, atrial tachycardia, atrial flutter or atrial fibrillation, atriofascicular pathways may be present and function as a bystander; a conduit to ventricular activation that results in a typical LBBB morphology, but not participating as a requisite component of the tachycardia' s reentrant circuit (Figure 7B)[18].

The site where atriofascicular bypass tracts cross the tricuspid annulus is the preferred target for radiofrequency ablation. Acute success rates have been reported in the range of 90% to 100%[20-22]. Targeting the distal, ventricular insertion site is complicated by its typically broad insertion, with distal arborization. This requires ablation of a large area of the ventricular myocardium in order to be effective, and is commonly associated with development of right BBB.

BUNDLE BRANCH REENTRANT VT

Bundle branch reentrant tachycardia (BBRVT) is a form of VT resulting from macroreentry within the bundle branches. Macroreentry involving the His-Purkinje system was originally described by Akhtar in 1974, in which premature right ventricular stimulation produced ventricular echo beats with a LBBB morphology[23]. Sustained BBRVT as a clinical entity was identified by Caceres *et al*[24] in 1989 and is most commonly seen in patients with acquired structural heart disease. BBRVT has been estimated to be the mechanism of VT in up to 6% of patients with sustained monomorphic VT. BBRVT has also been reported to account for 41%-45% of monomorphic VT in patients with nonischemic dilated cardiomyopathy[25]. However, most electrophysiology laboratories would report percentages that are considerably less than 6% and 41%. BBRVT may also be found in patients with ischemic and valvular heart disease. Patients typically present with presyncope, syncope, or sudden cardiac arrest.

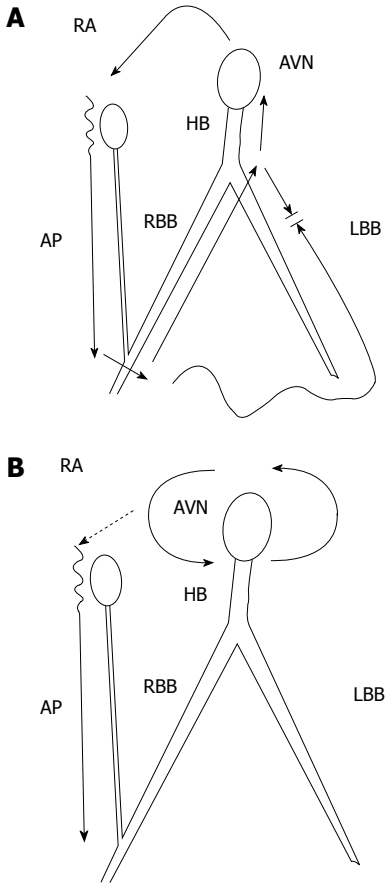

Figure 7 Pre-excitation *via* atriofascicular ("Mahaim") bypass tracts. A: Antidromic AV reentry tachycardia. The tachycardia circuit conducts antegrade down the Mahaim fiber and inserts into the distal right bundle branch (RBB). It propagates retrograde back to the atrium *via* the more proximal portion of the RBB; B: Atrioventricular nodal reentrant tachycardia with a "bystander" Mahaim fiber present. The bypass tract is not part of the tachycardia circuit, but contributes to ventricular activation. A wide QRS complex will be present with a left bundle branch block configuration, but ablation of the bypass tract will not eliminate the tachycardia. Adapted from[18], with permission. LBB: Left bundle branch.

The right bundle branch is responsible for the antegrade limb of the circuit in a majority of cases, with retrograde activation *via* one of the fascicles of the left bundle branch. This results in a typical LBBB pattern on the surface ECG. Tachycardia induction occurs when a paced ventricular beat finds the retrograde right bundle branch refractory. If slow (delayed) retrograde conduction through the left bundle branch occurs, the right bundle branch will recover and be capable of antegrade reactivation resulting in macroreentry (Figure 8). Subsequent retrograde left bundle conduction may perpetuate the sequence resulting in sustained tachycardia. ECGs recorded during sinus rhythm often show evidence of distal conduction system disease, with a prolonged PR interval and a nonspecific "LBBB-like" intraventricular conduction

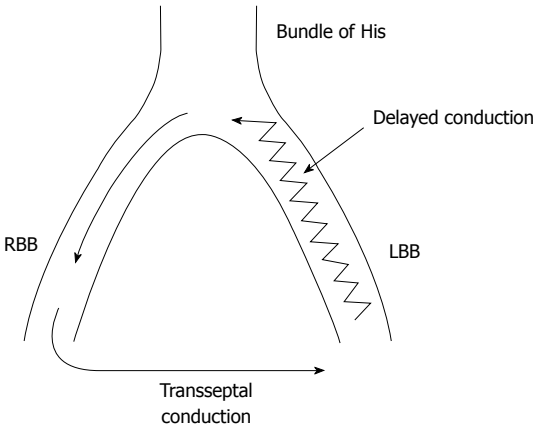

Figure 8 Mechanism of bundle branch reentrant ventricular tachycardia. The right bundle branch (RBB) is the antegrade limb of the circuit, with retrograde conduction *via* the slowly conducting left bundle branch. This allows the RBB to recover and be capable of reactivation, thereby perpetuating the reentrant circuit. LBB: Left bundle branch.

delay (wide QRS complex). Typical LBBB is an uncommon finding on the resting ECG although some patients may have apparent complete antegrade LBBB with intact retrograde left bundle branch conduction[26]. On electrophysiologic testing, conduction through the His-Purkinje system (baseline HV interval) is typically prolonged, averaging 80 ms (normal 35-55 ms) compatible with trifascicular conduction disease. Antiarrhythmic therapy is usually ineffective. Fortunately, radiofrequency ablation of the right bundle branch easily cures this arrhythmia. However, the patient may require permanent pacing if there is baseline antegrade LBBB or the HV interval prolongs to more than 90-100 ms. About 25% of patients have other inducible VTs[27]. These patients are best managed with an adjunctive implantable cardioverter-defibrillator (ICD). Many patients with BBRVT have advanced left ventricular dysfunction, wide QRS complexes and symptoms of heart failure which makes them appropriate candidates for cardiac resynchronization-ICD therapy.

DIAGNOSTIC AND ACUTE THERAPEUTIC MANEUVERS

The patient presenting with a WCT is often a source of anxiety and a diagnostic dilemma for the treating physician. However, when the patient presents with a "typical" or "classic" LBBB, without the presence of Q waves in the lateral precordial leads, the differential diagnosis is limited to the five entities discussed above.

With a more limited differential, the cardiologist can take a number of steps to clarify the underlying mechanism and determine the most appropriate treatment strategy. If a prior ECG is available, it should be reviewed for the possible clues it may provide. LBBB on the resting ECG is highly suggestive of SVT with fixed aberrancy. When LBBB is present on a resting ECG during sinus rhythm the QRS morphology during SVT with fixed aberrancy typically matches precisely[28].

As noted above, the 12-lead ECG during sinus rhythm may help differentiate between VT and SVT as well as provide clues about the tachycardia mechanism. However, a number of circumstances may limit its utility. SVT with fixed LBBB may exhibit electrical alternans (alteration of 0.1 mV or greater of the QRS or T wave) that is absent during sinus rhythm, suggesting the diagnosis of orthodromic AVRT. The presence of QRS alternans is a rate-related phenomenon and does not distinguish SVT from VT[29]. Patients with BBRVT (uncommonly) exhibit LBBB in sinus rhythm (as noted above, a prolonged PR interval and nonspecific intraventricular conduction delay are more common findings) and may have a matching QRS or QRS alternans during tachycardia. Another situation in which the ECG pattern of LBBB during sinus rhythm may not match that seen during SVT may occur with atriofascicular preexcitation. In sinus rhythm, antegrade fusion between AV nodal and accessory pathway conduction contributes to the QRS morphology. During antidromic atriofascicular tachycardia antegrade conduction occurs exclusively *via* the accessory pathway. This may result in a shift in QRS axis and/or duration[30]. Among the features suggestive of atriofascicular preexcitation include a short PR interval (during sinus rhythm) and late (after lead V4) transition of the QRS complex from a negative to a positive deflection[31]. If preexcitation is not visible during sinus rhythm and the LBBB pattern is the result of block in the His-Purkinje system (true anatomically fixed LBBB) the precordial QRS transition may be altered during antidromic atriofascicular tachycardia.

If an LBBB pattern is due to His-Purkinje conduction delay, rather than complete conduction block, it may be subject to rate-related changes[32]. LBBB with right axis deviation (RAD) is uncommon and suggestive of cardiomyopathy[33]. Intermittent RAD has been reported with induced aberration[34]. Hence, the QRS amplitude, duration, precordial transition and axis may all be altered during tachycardia, relative to sinus rhythm with fixed LBBB. It is, however, pivotal to remember that these are all exceptions to a very reliable rule.

If the cardiologist is confident that the WCT has a typical LBBB configuration, and a resting ECG is unavailable, it is reasonable to attempt a vagal maneuver such as carotid sinus massage. In 2009, Marill and associates demonstrated the safety of administering the intravenous AV nodal blocking agent adenosine in hemodynamically stable patients with wide QRS tachycardias[1]. A defibrillator should be present in case of the (unlikely) precipitation of rapid pre-excited atrial fibrillation.

In patients with SVT due to AV nodal reentrant tachycardia or AVRT, blocking AV nodal conduction will effectively terminate the tachycardia and restore sinus rhythm. SVT due to sinus tachycardia, atrial fibrillation or atrial flutter may exhibit transient AV conduction block, allowing for easier diagnosis of the unmasked supraventricular rhythm. This will be followed by subsequent return to an increased ventricular rate. The impact of adenosine on atrial tachycardia is variable (ranging from

no response to transient AV block to tachycardia termination)[35]. Antidromic atriofascicular fiber-mediated tachycardia may also terminate with adenosine if either antegrade accessory pathway or retrograde AV nodal blockade occurs.

BBRVT should be suspected in patients with typical LBBB tachycardia and significant structural heart disease. BBRVT is the only one of these clinical entities that usually exhibits no response to adenosine[36]. Although administration of adenosine may occasionally result in VA dissociation during hemodynamically stable VT, it should be noted that BBRVT is usually rapid and hemodynamically unstable, making DC cardioversion the initial option of choice.

LONG-TERM MANAGEMENT

Long-term management of sinus tachycardia usually requires reversal of its underlying cause. Sinus node reentry tachycardia is uncommon as an isolated entity. When present, it is usually quite amenable to catheter ablation[37]. Catheter ablation is first line therapy for atrial tachycardia and flutter, as well as tachycardias which require participation of an accessory pathway. We believe that catheter ablation is appropriate first-line therapy for AV nodal reentrant tachycardia because of its high cure rate and low complication rate[38,39]. As noted, BBRVT is exquisitely amenable to cure *via* ablation of the right bundle branch. Adjunctive therapy with an ICD is frequently (if not always) indicated. While antiarrhythmic drugs are still first-line treatment for atrial fibrillation, this arrhythmia may be amenable to catheter ablation after a failed trial of drug therapy[40].

CONCLUSION

In 2011**,** it is important for the clinician to be aware of "cutting edge" algorithms and diagnostic/therapeutic maneuvers[1,6]. In addition, respect for traditional electrocardiography is requisite to help physicians distinguish between "typical" LBBB and "LBBB-like" tachycardia morphologies. The clinical features and electrophysiologic characteristics of the five types of tachycardia with a typical LBBB pattern have been outlined above. A clear understanding of their mechanisms should facilitate tachyarrhythmia management. Most are quite amenable to treatment. Administration of adenosine is usually safe in the presence of hemodynamic stability and may aid in making the correct diagnosis. Long-term management strategies usually require referral to an electrophysiologist for catheter ablation.

REFERENCES

1 **Marill KA**, Wolfram S, Desouza IS, Nishijima DK, Kay D, Setnik GS, Stair TO, Ellinor PT. Adenosine for wide-complex tachycardia: efficacy and safety. *Crit Care Med* 2009; **37**: 2512-2518

2 **Wellens HJ**, Bär FW, Lie KI. The value of the electrocardiogram in the differential diagnosis of a tachycardia with a widened QRS complex. *Am J Med* 1978; **64**: 27-33

3 **Kindwall KE**, Brown J, Josephson ME. Electrocardiographic criteria for ventricular tachycardia in wide complex left bundle branch block morphology tachycardias. *Am J Cardiol* 1988; **61**: 1279-1283

4 **Akhtar M**, Shenasa M, Jazayeri M, Caceres J, Tchou PJ. Wide QRS complex tachycardia. Reappraisal of a common clinical problem. *Ann Intern Med* 1988; **109**: 905-912

5 **Brugada P**, Brugada J, Mont L, Smeets J, Andries EW. A new approach to the differential diagnosis of a regular tachycardia with a wide QRS complex. *Circulation* 1991; **83**: 1649-1659

6 **Vereckei A**, Duray G, Szénási G, Altemose GT, Miller JM. New algorithm using only lead aVR for differential diagnosis of wide QRS complex tachycardia. *Heart Rhythm* 2008; **5**: 89-98

7 **O'Keefe JH Jr**, Hammill SC, Freed MS, Pogwizd SM. The ECG Criteria Book. 2nd ed. Sudbury, MA: Physicians' Press, 2010: 118-119

8 **Klabunde RE**. Electrocardiogram Chest Leads (Unipolar). 1998-2010. Last accessed November 4, 2010. Available from: URL: http://www.cvphysiology.com/Arrhythmias/A013c.htm

9 **Trohman RG**, Kessler KM, Williams D, Maloney JD. Atrial fibrillation and flutter with left bundle branch block aberration referred as ventricular tachycardia. *Cleve Clin J Med* 1991; **58**: 325-330

10 **Malmivuo J**, Plonsey R. The Basis of ECG Diagnosis. In: Malmivuo J, Plonsey R, editors. Bioelectromagnetism: Principles and Applications of Bioelectric and Biomagnetic Fields. New York: Oxford University Press, 1995: 320-335

11 **Potger KC**. Vulnerable period of cardiac cycle. 2011. Last accessed Mar 9, 2011. Available from: URL: http://www.anzcp.org/CCP/Biomedical electronics/biomed/Vulnerable period.htm

12 **Josephson ME**. Clinical cardiac electrophysiology: Techniques and interpretations. 4th ed. Philadelphia: Lippincott Williams & Wilkins, 2008

13 **Lehmann MH**, Denker S, Mahmud R, Addas A, Akhtar M. Linking: a dynamic electrophysiologic phenomenon in macroreentry circuits. *Circulation* 1985; **71**: 254-265

14 **Issa ZF**, Miller JM, Zipes DP. Approach to wide QRS complex tachycardias. In: Issa ZF, Miller JM, Zipes DP, editors. Clinical Arrhythmology and Electrophysiology: a Companion to Braunwald's Heart Disease. 1st ed. Philadelphia: Saunders, 2009: 393-403

15 Arrhythmia. In: Ashley EA, Niebauer J, editors. Cardiology Explained. London: Remedica, 2004

16 **Knight BP**, Ebinger M, Oral H, Kim MH, Sticherling C, Pelosi F, Michaud GF, Strickberger SA, Morady F. Diagnostic value of tachycardia features and pacing maneuvers during paroxysmal supraventricular tachycardia. *J Am Coll Cardiol* 2000; **36**: 574-582

17 **Ellenbogen KA**, Rogers R, Old W. Pharmacological characterization of conduction over a Mahaim fiber: evidence for adenosine sensitive conduction. *Pacing Clin Electrophysiol* 1989; **12**: 1396-1404

18 **Tchou PJ**, Trohman R, Kidwell G, Mehdirad AA. Retrograde migration of the site of functional block: a mechanism underlying resolution of functional retrograde bundle branch block during AV reentrant tachycardia. *J Cardiovasc Electrophysiol* 1996; **7**: 335-340

19 **Prystowsky E**, Yee R, Klein GJ. Wolff-Parkinson-White Syndrome. In: Zipes DP, Jalife J, editors. Cardiac Electrophysiology: From Cell to Bedside. 4th ed. Philadelphia: Saunders, 2004: 869-878

20 **Haïssaguerre M**, Cauchemez B, Marcus F, Le Métayer P, Lauribe P, Poquet F, Gencel L, Clémenty J. Characteristics of the ventricular insertion sites of accessory pathways with anterograde decremental conduction properties. *Circulation*

1995; **91**: 1077-1085
21 **Klein LS**, Hackett FK, Zipes DP, Miles WM. Radiofrequency catheter ablation of Mahaim fibers at the tricuspid annulus. *Circulation* 1993; **87**: 738-747
22 **McClelland JH**, Wang X, Beckman KJ, Hazlitt HA, Prior MI, Nakagawa H, Lazzara R, Jackman WM. Radiofrequency catheter ablation of right atriofascicular (Mahaim) accessory pathways guided by accessory pathway activation potentials. *Circulation* 1994; **89**: 2655-2666
23 **Akhtar M**, Damato AN, Batsford WP, Ruskin JN, Ogunkelu JB, Vargas G. Demonstration of re-entry within the His-Purkinje system in man. *Circulation* 1974; **50**: 1150-1162
24 **Caceres J**, Jazayeri M, McKinnie J, Avitall B, Denker ST, Tchou P, Akhtar M. Sustained bundle branch reentry as a mechanism of clinical tachycardia. *Circulation* 1989; **79**: 256-270
25 **Galvin JM**, Ruskin JN. Ventricular tachycardia in patients with dilated cardiomyopathy. In: Zipes DP, Jalife J, editors. Cardiac Electrophysiology: From Cell to Beside. 4th ed. Philadelphia: Saunders, 2004: 575-587
26 **Blanck Z**, Deshpande S, Jazayeri MR, Akhtar M. Catheter ablation of the left bundle branch for the treatment of sustained bundle branch reentrant ventricular tachycardia. *J Cardiovasc Electrophysiol* 1995; **6**: 40-43
27 **Blanck Z**, Dhala A, Deshpande S, Sra J, Jazayeri M, Akhtar M. Bundle branch reentrant ventricular tachycardia: cumulative experience in 48 patients. *J Cardiovasc Electrophysiol* 1993; **4**: 253-262
28 **Fox DJ**, Tischenko A, Krahn AD, Skanes AC, Gula LJ, Yee RK, Klein GJ. Supraventricular tachycardia: diagnosis and management. *Mayo Clin Proc* 2008; **83**: 1400-1411
29 **Kremers MS**, Miller JM, Josephson ME. Electrical alternans in wide complex tachycardias. *Am J Cardiol* 1985; **56**: 305-308
30 **Mata MB**, Kloss BT, Campoli JA, Teelin K. Unusual source of tachycardia in an adolescent. *Int J Emerg Med* 2011; **4**: 9
31 **Bardy GH**, Fedor JM, German LD, Packer DL, Gallagher JJ. Surface electrocardiographic clues suggesting presence of a nodofascicular Mahaim fiber. *J Am Coll Cardiol* 1984; **3**: 1161-1168
32 **Issa ZF**, Miller JM, Zipes DP. Intraventricular conduction abnormalities. In: Issa ZF, Miller JM, Zipes DP, editors. Clinical Arrhythmology and Electrophysiology: a Companion to Braunwald's Heart Disease. 1st ed. Philadelphia: Saunders, 2009: 143-156
33 **Nikolic G**, Marriott HJ. Left bundle branch block with right axis deviation: a marker of congestive cardiomyopathy. *J Electrocardiol* 1985; **18**: 395-404
34 **Childers R**, Lupovich S, Sochanski M, Konarzewska H. Left bundle branch block and right axis deviation: a report of 36 cases. *J Electrocardiol* 2000; **33 Suppl**: 93-102
35 **Trohman RG**. Adenosine for diagnosis of wide QRS tachycardia: rapid infusion for an easier conclusion. *Crit Care Med* 2009; **37**: 2651-2652
36 **Rubenstein DS**, Burke MC, Kall JG, Kinder CA, Kopp DE, Wilber DJ. Adenosine-sensitive bundle branch reentry. *J Cardiovasc Electrophysiol* 1997; **8**: 80-88
37 **Sanders WE**, Sorrentino RA, Greenfield RA, Shenasa H, Hamer ME, Wharton JM. Catheter ablation of sinoatrial node reentrant tachycardia. *J Am Coll Cardiol* 1994; **23**: 926-934
38 **McElderry HT**, Kay GN. Ablation of Atrioventricular Nodal Reentry by the Anatomic Approach. In: Huang SKS, Wood MA, editors. Catheter Ablation of Cardiac Arrhythmias. 1st ed. Philadelphia: Elsevier, 2006: 325-346
39 **Trohman RG**, Pinski SL, Sterba R, Schutzman JJ, Kleman JM, Kidwell GA. Evolving concepts in radiofrequency catheter ablation of atrioventricular nodal reentry tachycardia. *Am Heart J* 1994; **128**: 586-595
40 **Calkins H**, Brugada J, Packer DL, Cappato R, Chen SA, Crijns HJ, Damiano RJ, Davies DW, Haines DE, Haissaguerre M, Iesaka Y, Jackman W, Jais P, Kottkamp H, Kuck KH, Lindsay BD, Marchlinski FE, McCarthy PM, Mont JL, Morady F, Nademanee K, Natale A, Pappone C, Prystowsky E, Raviele A, Ruskin JN, Shemin RJ. HRS/EHRA/ECAS expert Consensus Statement on catheter and surgical ablation of atrial fibrillation: recommendations for personnel, policy, procedures and follow-up. A report of the Heart Rhythm Society (HRS) Task Force on catheter and surgical ablation of atrial fibrillation. *Heart Rhythm* 2007; **4**: 816-861

S- Editor Cheng JX **L- Editor** Cant MR **E- Editor** Zheng XM