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

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Abstract
The evaluation of wide QRS complex tachycardias (WCT) remains a common clinical 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.

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

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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 tachycardia 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[1]. 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)[6]. 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 > 50 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[7].

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)[8]. 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)[9]. 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[10].

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[11].

SVT with fixed or functional aberrancy accounts for approximately 15%-20% of WCTs, and is a significantly
more common cause of WCT in patients under the age of 35\cite{14}. Although any SVT may exhibit functional LBBB, it is most commonly seen during orthodromic AV reentry tachycardia with antegrade conduction \textit{via} the AV node and right bundle branch and retrograde conduction \textit{via} a left free wall accessory pathway (Figure 6)\cite{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\cite{17}.

Approximately 6\% of patients presenting with SVT with a typical LBBB morphology have been found to have an atriofascicular bypass tract\cite{18}. In such cases, antidromic AV reentrant tachycardia (AVRT) results from antegrade conduction down the bypass tract and retrograde propagation \textit{via} the normal conduction system (Figure 7A)\cite{19}. Orthodromic AVRT almost never occurs, because these

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

| Authors         | Date | Morphology | Criteria favoring ventricular tachycardia |
|-----------------|------|------------|------------------------------------------|
| Wellens et al\cite{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                      |
| Kindwall et al\cite{3} | 1988 | LBBB-like  | QRS width > 140 ms                       |
|                 |      |            | R in V1 or V2 > 30 ms                    |
| Akhtar et al\cite{4}   | 1988 | LBBB-like  | Onset of QRS to nadir of S ≥ 60 ms in V1 or V2 |
|                 |      |            | Notching of downstroke of S in V1 or V2  |
| Brugada et al\cite{5}   | 1991 | LBBB-like  | Positive QRS concordance across the precordium |
|                 |      |            | Extreme left axis deviation (-90° to ± 180°) |
| Vereckei et al\cite{6}   | 2008 | RBBB-like  | QRS > 160 ms                             |
|                 |      |            | QRS > 140 ms                             |
|                 |      |            | 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 |

\textit{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.
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-

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

**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                                                             |
| SVT with functional LBBB aberrancy            | QRS during tachycardia usually an identical match                                          |
| Atriofascicular antidromic tachycardia         | Most often due to orthodromic AVRT                                                       |
|                                              | At rapid rates, QRS alternans may be present                                              |
|                                              | Preexcitation may be minimal or absent during sinus rhythm                                 |
| 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              |

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.

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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 left bundle branch block (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.

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 occurs through the left bundle branch, 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
Neiger JS et al. Typical left bundle branch block tachycardia

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[35]. 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.

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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

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[29].

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.

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
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. 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. As noted, BBRVT is the only one of these clinical entities that usually exhibits no response to adenosine. 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.

CONCLUSION

In 2011, it is important for the clinician to be aware of “cutting edge” algorithms and diagnostic/therapeutic maneuvers. 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.

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