Challenge Diagnosis

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Case of Dr. Peter Kukla, MD, PhD
From Poland

Commentaries:
Prof. Dr. Melvin Scheinman – USA
Raimundo Barbosa Barros MD – Brazil
Dr Emilio Marigliano MD – Argentina

Theoretical Background:
Andrés Ricardo Pérez Riera, MD
Dear Prof. Andres Ricardo Perez Riera
First of all greetings from Poland
I would like to show you interesting case (maybe for such the world-wide famous ECG expert as You - it is not interesting ?)
Case - 71 yo man, with ischemic cardiomyopathy, EF % - 25%, LVEDD- 65mm, after inferior MI and anterior MI, after RCA-PCI - 15 years ago. Hospitalized because of congestive heart failure and ventricular arrhythmia, he was directed for CRT-ICD thx. I have a question to Professor: Is his ECG presenting RBBB - like or true- RBBB with LBBB ? or other explanation = LBBB + PAF as a picture of septal block ????? I wonder what is your opinion and Your Friends ?
in attachment I send ECG tracings
ECG nr 1. typical LBBB
ECG nr 2 - 6 months later
ECG nr 3 i 4 from Holter monitoring
Kinds regards,
Peter Kukla, MD, PhD.

Português
Prezados amigos do foro.
Acaba-me de enviar esta seqüência de traçados o brilhante colega da Polônia  Peter Kukla Piotr MD PhD para vossa apreciação.
Apresentação do caso: Masculino, 71 anos portador de cardiomiopatia isquêmica FE de 25%. Diâmetro diastólico de VE de 65mm, consequência de infartos prévios nas paredes inferior e anterior tendo sido abordado côn PCI sua CD há 15 anos. O paciente fora hospitalizado por ICC e arritmia tendo sido encaminhado de imediato par rencronização c/ CDI. Eu tenho uma pergunta: ele é portador de BCRE ou de BCRD. Ou outra explicação como ser BCRE + LSFB. Em adjunto mando os traçados
ECG1: típico BCRE
ECG2: seis meses mas tarde
ECG3 3 4 : do monitor do Holter.
Saudações
Dr Peter Kukla MD PhD.
Colleagues Opinions

First Opinion Dr. Melvin Scheinman Opinion

Biography: Dr. Melvin is Professor of Medicine, Walter H. Shorenstein Endowed Chair in Cardiology, and one of the founding fathers of the field of cardiac electrophysiology. Dr. Melvin Scheinman is one of the founding pioneers of clinical cardiac electrophysiology. He grew up in Brooklyn, New York and took his undergraduate degree at Johns Hopkins University where he graduated first in his class. Postgraduate medical education included Albert Einstein College of Medicine, residency training at the University of North Carolina (Chapel Hill) and cardiology training at the University of California, San Francisco Medical Center.

Dr. Scheinman is best known as the first person to have performed catheter ablation in humans. This was done after extensive animal studies. Dr. Scheinman and his team used high-energy direct current shocks and were the first to ablate accessory pathways and used this technique to ablate the fast AV nodal pathway. Dr. Scheinman and his colleagues were instrumental in the development of radiofrequency energy applications for a whole gamut of cardiac arrhythmias. More recently, Dr. Scheinman and his colleagues have developed techniques for modification of sinus node function in patients with inappropriate sinus tachycardia and for cure of patients with automatic junctional tachycardia. Dr. Scheinman is also well known for his work in defining the role of electrophysiologic studies in determining need for pacemakers in patients with AV conduction disturbances. He was the first to initiate combined pacemaker and beta-blocker therapy for patients with the long QT syndrome. He was among the first to prove the efficacy of intravenous amiodarone therapy for patients with malignant ventricular tachycardia. Dr. Scheinman's current interest revolve about use of new antiarrhythmic agents (i.e., Ibutilide, Azimalide) and in defining the basic mechanisms of unusual forms of atrial flutter. Dr. Scheinman has made many contributions in the area of genetic causes of sudden death and will initiate a Cardiac Arrhythmia Genomics Clinic at UCSF. Dr. Scheinman is a renowned teacher and was awarded the Henry Kaiser Award for Excellence in Teaching as well as the Teacher of the Year Award in 1973. He was also honored with the Paul Dudley White Award for Excellence in Teaching by the American Heart Association. Dr. Scheinman is director of an annual course on Advanced Clinical Electrophysiology and Ablation given at the American College of Cardiology, Heart House in Bethesda, Maryland. Dr. Scheinman is a superb clinician and among the busiest clinician on faculty. His opinion is sought for difficult clinical cases and his clinic is amongst the most active at our medical center.
Fig. 1 - shows AV block and a LBBB configuration. Note that $r > 2\text{mV}$ is recorded in $V_1$ and this is compatible with an incomplete LBBB (Prystowsky JCE July 10) Dear Melvin could you send to us the exact citation?
Fig. 2 - shows a prolonged PR and RBBB pattern with probable LAFB (note that the terminal r in aVR is later than that of aVL. In addition, left septa fascicular block is present. The PR interval is longer with RBBB. If the BBB pattern changes with change in PR, this is diagnostic of infranodal disease (Lepeschkin).

Fig. 3 - the RBBB pattern is interrupted by a PVC which is followed by switch to a LBBB pattern. This by itself would suggest that the RBBB pattern is due to retrograde concealed invasion of the RBB which is terminated by a PVC (and peeling back of refractoriness) This explanation is not tenable as the PVC is not ipsilateral to the site of block (PVC is of LV origin) and later in the bottom strip there is abrupt conversion of the LBBB and RBBB pattern.
Fig. 4 - shows a bigeminal pattern with alternate RBBB and LBBB.
Dr. Melvin Conclusion  
1. Bilateral Bundle Branch Block.  
2. Pacemaker is indicated.  

A evolução eletrocardiográfica sugere BLOQUEIO DE RAMO BILATERAL. O Holter confirma bloqueio de ramo bilateral alternante.  
The ECG evolution suggests Bilateral Bundle Branch Block. The Holter confirms alternant bilateral bundle branch block  

Raimundo Barbosa Barros MD Fortaleza Ceará Brazil  

En el segundo trazado me impresiona BDAS + BDAM. En el Holter creo que este fenómeno es intermitente. El QRS es ancho(BRI )y tiene BAV de Primer grado. Si consideramos, de acuerdo a lo aprendido en el curso de ECG, que el haz medio septal esta irrigado por la DA en 100% de los casos quizas se deberia hacer otra coronariografia.  
In the second ECG I think that the diagnosis is LSFB + LAFB. In the Holter trace these dromotropic disorders are intermittent. The QRS duration is broad with LBBB configuration and first-degree AV block. If we consider that the LSF is irrigated exclusively by LDA septal branches coronariography is mandatory.  
Dr Emilio Marigliano MD Argentine
Andrés Ricardo Pérez Riera commentaries

There are not adequate criteria for determining the precise location of the site of complete heart block for the surface ECG alone. However, it is often possible to diagnose bilateral bundle branch block electrocardiographically prior to the development of complete heart block or during periods when AV conduction returns.

1. A definitive diagnosis of bilateral bundle branch block can be made if the pattern of RBBB and LBBB accompanied by changes in the PR interval, occur alternately or intermittently in the same patient. (Figure 4 of our patient)

2. If the pattern of RBBB and LBBB appear at different times in the same patient but the PR interval remains constant, the diagnosis of bilateral bundle branch block is reasonably secure.

3. The combination of first and second-degree AV block with bundle branch block may represent bilateral branch block (i.e. complete block in one bundle branch with incomplete block in the contralateral bundle branch. However when bundle branch block is accompanied by incomplete AV block, the latter may be sited in the AV node, the bundle of His, or the other bundle branch. The exact location of the block can only be determined by the use of His bundle recordings. Thus, the finding of bundle branch block together with incomplete AV block in surface leads can only suggest the possibility or probability of bilateral bundle branch block.

4. A first-degree AV block associated with RBBB pattern, LAFB and LSFB observed in figure 2 from my point of view it is a tetra-fascicular block. The PR interval is longer with RBBB. If the BBB pattern changes with change in PR, this is diagnostic of infranodal disease (Lepeshkin). And indicate incomplete LPFB. Please see the next slide. Additionally we have inferior myocardial infarction associated with LAFB.
"TETRAFASCICULAR BLOCK"
Is this nomenclature appropriated?

Stimulus pathway inside AV and His system

1. Complete RBBB
2. Complete LAFB..... Extreme left QRS axis deviation on FP
3. Complete LSFB...Prominent Anterior Forces (PAF) on right and middle precordial leads
4. Incomplete LPFB..... First-degree AV block( PR interval prolongation)
5. Conclusion: TETRAFASCICULAR BLOCK (new proposal nomenclature.).
ALTERNATING BILATERAL BUNDLE BRANCH BLOCK.

Definition: It is defined as the dromotropic disorder that affects both proximal branches of the His system, the right branch and the left branch trunks of the His bundle. To prevent a total block from occurring, they should not be concomitant in a maximal degree. This diagnosis could be made by ECG when the CRBBB and CLBBB patterns, accompanied by changes in the PR interval duration, occur in an alternating and intermittent way in the same patient, in tracings made at different times.

We could register:

1) Alternating complete block of both the left and right branches;

2) CLBBB + IRBBB: in this case, the ECG shows CLBBB with prolonged PR (≥200 ms) and, and second degree IRBBB;

3) CRBBB associated to ILBBB. CRBBB is seen associated to PR interval ≥200 ms;

4) Incomplete block of both branches with a similar degree. In this case, the QRS is normal, because the activation of the biventricular chamber is simultaneous and the PR is prolonged, or of second degree.

5) If in the right branch there is first degree block, and in the left branch there is 2:1, the ECG will show a pattern of CLBBB with long PR, alternating with QRS of the IRBBB type and normal PR.

The following is a typical example of alternating bilateral BBB.
The patient was admitted on May 12th, 2009, after several syncope episodes. These 2 tracings confirm the diagnosis of alternating bilateral branch. It is first degree bundle branch block + second degree contra-lateral block. The ECG shows alternating patterns of RBBB and LBBB, each pattern with a different PR interval (See these next 2 ECGs that show it clearly). The Echo of this patient reveals concentrically LVH with normal ejection fraction. He already underwent coronary angiography, which did not show obstructive coronary lesions. In this case, there is no indication of invasive electrophysiologic study. The patient should undergo permanent PM implantation.
Patients with chronic bifascicular block can progress to advanced atrioventricular block (AVB). The presence of:
1. Syncope or presyncope
2. QRS duration >140 ms
3. Renal failure
4. HV interval > 64 ms
Are independent predictors of progression to AVB in patients with bifascicular block.

Guevara Valdivia et al presented the case of a 66 years old man with recurrent episodes of syncope, up to three times during the last two months without previous symptoms. An ECG after the syncope showed LAFB, RBBB and first grade AV block (bilateral bundle branch block).

The exercise test did not demonstrate either AV conduction disorders or tachyarrhythmia episodes. Holter monitoring showed premature ventricular complexes; tilt testing and carotid sinus massage were normal. The electrophysiologic study revealed no alteration in the conduction system. Throughout atrial and ventricular stimulation documented no tachyarrhythmias. However, intravenous administration of 12 mg of adenosine induced complete AV infra-His block with ventricular asystolia of 7.2 sec duration. The authors concluded that adenosine testing can identify patients with syncope due to paroxysmal AV block even when the electrophysiological findings and other conventional tests are not conclusive.

In patients with syncope and bundle branch block (BBB), syncope is suspected to be attributable to a paroxysmal atrioventricular (AV) block, but little is known of its mechanism when electrophysiological study is negative.

Brignole et al(1) applied an implantable loop recorder in 52 patients with BBB and negative conventional workup. During a follow-up of 3 to 15 months, syncope recurred in 22 patients (42%), the event being documented in 19 patients after a median of 48 days. The most frequent finding, recorded in 17 patients, was one or more prolonged asystolic pause mainly attributable to AV block; the remaining 2 patients had normal sinus rhythm or sinus tachycardia. The onset of the bradycardic episodes was always sudden but was sometimes preceded by ventricular premature beats. The median duration of the arrhythmic event was 47 seconds. An additional 3 patients developed nonsyncopal persistent III-degree AV block, and 2 patients had presyncope attributable to AV block with asystole. No patients suffered injury attributable to syncope relapse. The authors concluded that patients with BBB and negative electrophysiological study, most syncopal recurrences have a homogeneous mechanism that is characterized by prolonged asystolic pauses, mainly attributable to sudden-onset paroxysmal AV block.

21 of 496 (4%) patients with chronic bifascicular block, studied and followed prospectively, had block distal to the His bundle induced by atrial pacing during initial electrophysiologic studies. In 6, block distal to the His bundle was noted during pacing-induced atrioventricular (AV) nodal Wenckebach periods (at paced rates of 150--190 beats/min), with block distal to the His bundle in the short HH cycles after the AV nodal blocked P (long cycle).

The AH interval was normal in all 6 patients and HV was normal in 4. None of the 6 patients has developed AV block during a mean follow-up of 5.33 +/- 0.48 years. In 15 patients, pacing-induced block distal to the His bundle was noted during intact AV nodal conduction (paced rate of 80--200 beats/min).

The AH interval was prolonged in one, and HV was prolonged in 10 of the 15 patients. During a mean follow-up of 3.4 +/- 0.59 years, 7 of these patients developed AV block, 1 had treadmill-provoked AV block, and 2 died suddenly (major morbid event in 10 of 15 patients).

In conclusion, block distal to the His bundle induced by atrial pacing is an infrequent finding in patients with bifascicular block, and can be a functional as well as a pathologic response. The latter is associated with a high risk of major morbid events (AV block and sudden death).

STANDARD MASQUERADING
RIGHT BUNDLE BRANCH BLOCK

Leads I and aVL have a pure R wave pattern, suggestive of CLBBB.
There are extreme Left Axis Deviation with SIII>SII (QRS axis -65°: LAFB).
Right precordial leads with Rs pattern and V3 qRs = CRBBB + LSFB.

QRS Duration = 122ms
Conclusion: Standard masquerading RBBB + LAFB + LSFB.
Currently a novel form of trifascicular block,
The primary concern in patients with bifascicular block is the increased risk of progression to complete heart block. Further, an additional first-degree A-V block in patients with bifascicular block or LBBB might increase the risk of block progression. Anesthesia, monitoring and surgical techniques can induce conduction defects and bradyarrhythmias in patients with pre-existing bundle branch block. In the setting of an acute MI, several different types of conduction disturbance may become manifest and complete heart block occurs usually in patients with acute myocardial infarction more commonly if there is pre-existing or new bundle branch block. The question that arises is whether it is necessary to insert a temporary pacing catheter in patients with bifascicular block undergoing anesthesia. It is important that an anesthesiologist should be aware of the indications for temporary cardiac pacing as well as the current recommendations for permanent pacing in patients with chronic bifascicular and trifascicular block. It is necessary to know the recent guidelines for temporary transvenous pacing in the setting of acute MI and the different pacing modalities that are available for an anesthesiologist.

The Masquerading Bundle Branch Block Concept

**Definition:** Masquerading bundle branch block is not a specific entity but is the result of RBBB with varying combination of LAFB, intramural left ventricular block, left ventricular enlargement, and anterior myocardial infarction characterized on ECG by an atypical Complete RBBB with two possible patterns:

1. **Standard Masquerading BBB:** it is characterized by LBBB pattern in the limb leads and the precordial ECG remain RBBB
2. **Precordial Masquerading BBB:** RBBB pattern in right precordial leads and LBBB configuration in left leads. This pattern results from RBBB associated with severe LVH, localized block in the anterolateral wall of the LV or both, intramural LV block. These associations produce predominant leftward forces which tend to cancel out the late rightward forces of the RBBB in the left precordial leads and I.

**Observation:** Standard and precordial masquerading NNN may occur in the same tracing. When precordial masquerading BBB occurs in the absence of LAFB abnormal left axis deviation will not seen in the limb leads.

MAIN REFERENCES ABOUT BILATERAL BBB