Rate Dependent Bundle Branch Block

By

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Bundle Branch Block may be transient, intermittent or permanent. Transient and intermittent left bundle branch block has beep the subject of many publications and reports.[1-9] Transient left bundle branch block has been defined as an intraventricular (I-V) conduction defect that changes, if only temporarily, to normal intraventricular conduction,[6] where as intermittent left bundle branch block is characterised by the presence in a single electrocardiographic (ECG) tracing of QRS complexes showing both left bundle branch block and normal intraventricular conduction.[6] However since some cases are rate dependent (not considering for the moment other intricate factors) it will be hard to make a clear cut distinction not only between transient and intermittent bundle branch block but also between transient and permanent block. Infact many cases of intermittent block are usually diagnosed from fortuitiously obtained cardiographic records and are rarely actively searched for. The situation is further confused by the fact that the records showing normal intraventricular conduction at a 'reasonable' heart rate may be masking a latent degree of bundle branch block which would only be uncovered by slight acceleration of the rate or other appropriate measures. The purpose of this presentation is to high-light the presence of aetiological factors such as ischaemic heart disease (manifest or occult) and hypertension etc. and to show that with the extention of the disease process cases of intermittent bundle branch block develop life threatening complications as complete heart block or they may be accompanied by acute myocardial infarction. Many reports dealing with the detection of myocardial infarction complicating left bundle branch block have appeared in the literature.[15-16-20-22-25-29] The patients with intermittent left bundle branch block provides a unique opportunity to compare in the same tracing the QRS and T Waves configuration of the normally conducted complexes with those showing left bundle branch block and thus be able to define the criteria for the diagnosis of myocardial infarction and ischaemia, in the presence of left bundle branch block.[10]

The following review presents a critical appraisal of the subject through a study of eight cases of intermittent bundle branch block observed in the past 3 years at the Cardiology Department of Central Government Poly Clinic, Islamabad.

Case Histories:

Eight cases with rate dependent bundle branch block were studied. These include 6 cases with tachycardia-dependent block, 1 with bradycardia-dependent block, and 1 case showing both at different heart rate.

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Tachycardia-Dependent Bundle Branch Block:

Case-I.

A 38 years old women with hypertension and anaemia was first seen on 19th Feb. 1980 with the complaints of paroxysms of palpitation, chest pain and dyspnoea. Her cardiographic record showed evidence of inferior and anterior ischaemia. Same evening the electrocardiogram (ECG) showed intermittent bundle branch block. She was admitted after a year on 7.3.1981 with severe chest pain and shock. Electrocardiographically she had permanent left bundle branch block with a heart rate of 88 pm. Previous changes of ischaemia were masked, Q Waves disappeared and the T Waves became upright. Serum enzymes were high and suggested acute myocardial infarction.

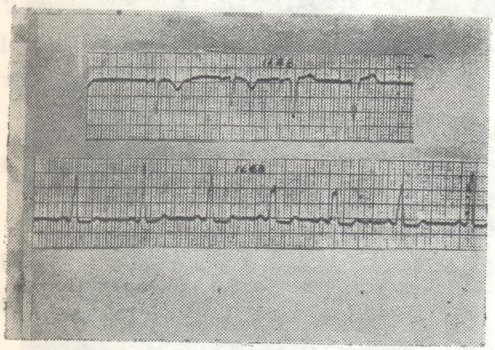

Fig. 1 (*CASE I*)

A: 1st & 2nd Complex with Normal Conduction at Heart Rate 60 P.M. 3rd & 4th Complex Showing LBBB at Heart Rate 75 P.M.

B: 2nd Complex Showing Normal Conduction-3rd, 4th-5th & 6th Complexes Showing LBBB with Heart Rate of 70 P.M. Last Complex Showing Normal Conduction at Heart Rate of 62 P.M.

Comments:

This is a classical case of tachycardia dependent left bundle branch block, where the critical rate is 70 pm. When the rate falls below this there is normal intraventricular conduction. Hypertension and Ischaemic Heart Disease could be the aetiological factors. The R-R interval becomes shorter just before the intermittent bundle branch block intervenes and longer R-R intervals restore normal intraventricular conduction. Ultimately this patient developed complete left bundle branch block which depicts the natural history of intermittent bundle branch block. This case also shows the practical diffi culty of diagnosing myocardial infarction in the electrocardiographic setting of left bundle branch block where serum enzymes become all important. In this regard a high diagnostic value of the disproportionately large ST-T elevation during the acute episode of a cardiac infarction is accepted.

Case-II.

A 48 years old lady was admitted to the hospital on 4.6.1981 with a history of acute chest pain and giddiness and transient black out. She had a normal ECG record 7 months ago. The ECG on admission showed sinus bradycardia with heart rate of 45 pm. She also had 1st degree heart block with right bundle branch block. When the heart rate increased to 52 pm., she showed left bundle branch block. Again on 25.6.1981 when her heart rate fell to 36 pm., electrocardiographically she showed right bundle branch block with complete A.V. dissociation. She had Stokes Adam's attack with marked fall in blood pressure. She had to be paced and a permanent internal pacemaker was put in.

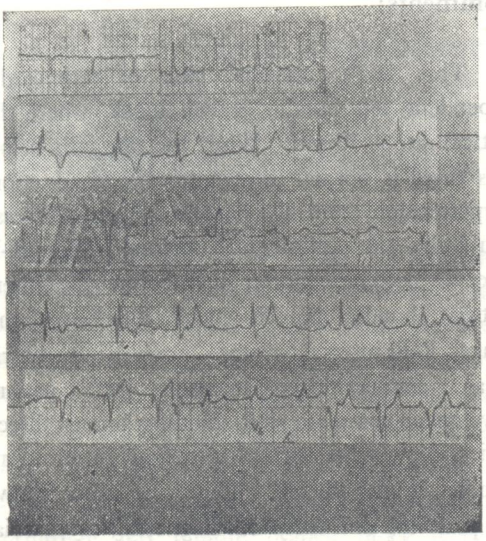

Fig. 2. (*Case II*)

A: Normal Sinus Rhythm Heart Rate 84 P.M.

B: RBBB at Heart Rate 44 P.M. with 2:1 Block.

C: LBBB at Heart Rate 54 P.M. with 2:1 Block.

C: LBBB at Heart Rate 54 P.M. with 2:1 Block.

D: RBBB at Heart Rate 46 P.M. with Complete A.V. Dissociation.

E: Temporary Transvenous Pacemaking Showing LBBB at Heart Rate 72 P.M.

Comments:

This patient had rate dependent bundle branch block, showing both types at different occasions. At heart rate of 45 pm., or below she had right bundle branch block but at the rate of 52 p.m., she developed left bundle branch block. The bundle branch block later progressed to complete heart block with A.V., dissociation and the patient became critically ill-necessitating the use of pacemaker.

Case-III.

A 45 years old patient presented in the O.P.D., with the main complaints of attacks of palpitation and pain chest. Physical examination and the resting ECG were normal. Exercise electrocardiography was undertaken on ergometer. When his heart rate reached 130 p.m. ECG showed left bundle branch block. It coincided with the feelings of fluttering in the chest along with some pain. As soon as the heart rate fell down, the ECG returned to normal.

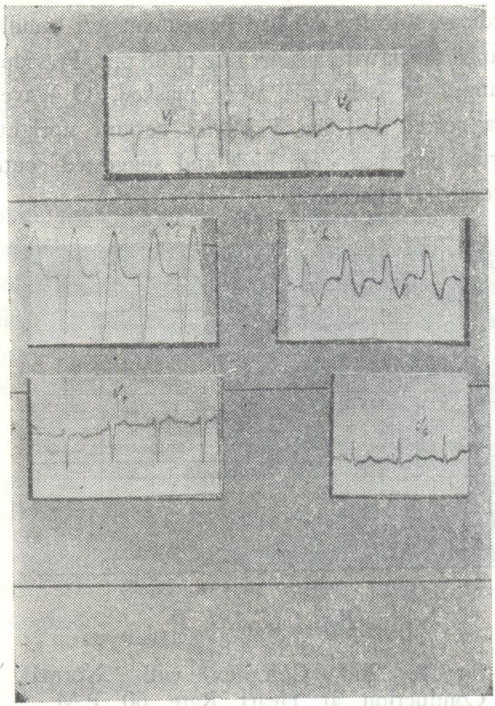

Fig. 3. (*Case III*)

A: Resting ECG. Normal Sinus Rhythm at Heart Rate 80 P.M.

B: Just after Exercise LBBB at Heart Rate 130 P.M.

C: Return to Normal Conduction, at Heart Rate 110 P.M.

Comments:

This is a case of tachycardia-dependent left bundle branch block. Interestingly his complaints are associated with the developmenl of left bundle branch block at the higher rate. He has been put on B—Blockers and his complaints are now alleviated. It would be an interesting case to follow and to see if he eventually develops coronary artery disease.

Case-IV.

A 46 years old patient of hypertensoin with history of angina on effort was admitted to the hospital for investigations and treatment. His resting heart rate was between 80-90 per minute. His antihypertensive therapy was changed from methyl-dopa to propranalol. When his heart rate fell to 64 pm., he developed left bundle branch block which disappeared when the heart rate improved.

Comments:

This is a case of bradycardia dependent left bundle branch block with a positive history of hypertension and ischaemic heart disease.

Case-V.

Mr. I, aged 25 years was referred to Cardiology Department with history of chest pain and palpitation. He was diagnosed as a case of sick sinus syndrome. He underwent an exercise tolerance test. When his heart rate reached 190 p.m., electrocardiogram showed right bundle branch block which returned to normal when heart rate dropped to 150 p.m.

Comments:

This was a case of sick sinus syndrome who while performing the exercise tolerance test developed tachyarrhythmia and showed right bundle branch block which returned to sinus rhythm once the rate dropped.

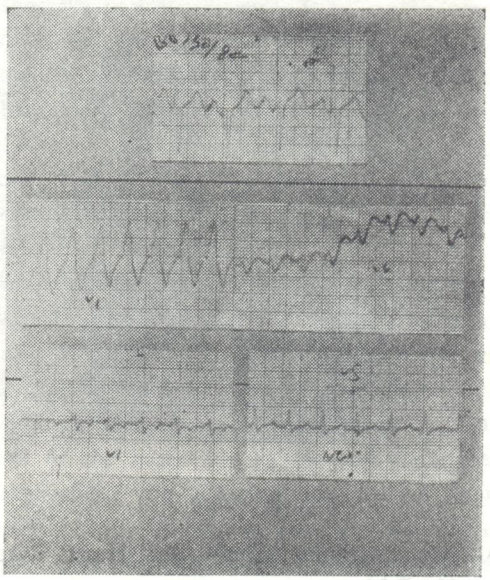

Fig. 4 (*Case V*)

A: Normal Conduction at Heart Rate 100 P.M.

B: RBBB at Heart Rate 187 P.M.

C: Normal Conduction at Heart Rate 158 P.M.

Case-VI.

A 57 years old patient with ischaemic heart disease developed congestive cardiac failure and was admitted with pain chest His ECG showed normal interaventricular conduction. But when his heart rate went up to 150 p.m.,

he developed right bundle branch block. The rhythm reverted to sinus when the rate dropped to 110 p.m.

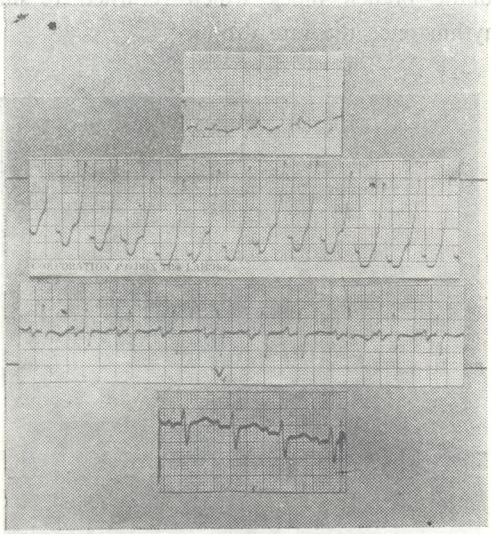

Fig. 5. (*Case VI*)

A: Normal Sinus Rhythm at Heart Rate 115 P.M.

B: RBBB at Heart Rate 150 P.M.

C: Back to Sinus Rhythm with the Dropping of Heart Rate to 115 P.M.

Case-VII.

Mr. M.S. aged 65 years was a case of Angina with normal resting ECG. During Exercise Tolerance Test when his heart rate went to 120 p.m., he developed left bundle branch block. The E.C.G., became normal when the heart rate dropped.

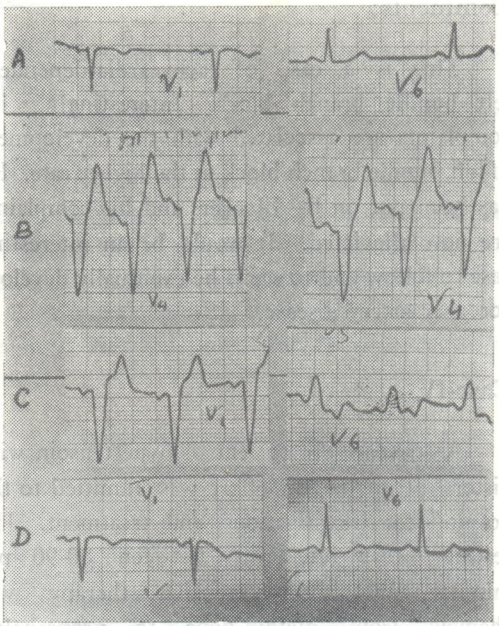

Fig. 6 (*Case VII*)

A: Normal Conduction at Heart Rate 55 P.M.

B: During Exercise LBBB at Heart Rate 125 P.M.

C: Just after Exercise LBBB at Heart Rate 90 P.M.

D: Normal Conduction at Heart Rate 68 P.M.

Case-VIII.

A case of acute coronary insufficiency showing rate dependent left bundle branch block with a heart rate of 150 p.m.

Comments:

Relative coronary insufficiency at higher heart rate appears to be the aetiological factor in the above three cases showing rate dependent bundle branch block.

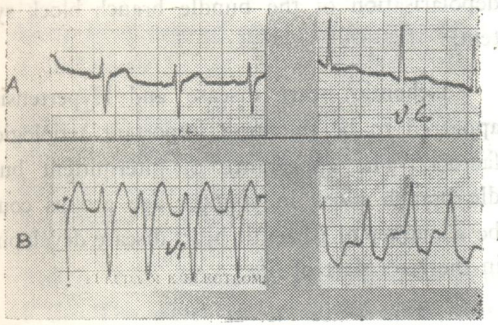

Fig 7. (Case VIII)

A: Normal Conduction at Heart Rate 88 P.M.

B: LBBB at Heart Rate 130 P.M.

Discussion:

The effect of heart rate, vagal maneuvers and pharmacologic agents on cases with intermittent left bundle branch block has been extensively studied. Wallace and Laszlo,[5] from their investigations, concluded that no single factor was responsible for initiating the left bundle branch block and that the basic mechanism still remained uncertain. Ischaemic and Hypertensive Heart Disease or a combination of both, have been found to be the underlying aetiological factors in most of the cases. Rarely, Syphlitic Heart Disease, Rheumatic Heart Disease, acute infection like diphtheria, thyrotoxicosis, anaemia, sepsis, and a number of drugs with depressent effect on cardiac conduction like quinidine, procanamide and potassium have been associated with this condition.[1-7-9] Carter and Dieuaide[23] put forth the idea that in patients with intermittent bundle branch block, a few intact fibres of the conducting bundle were carrying on the normal excitation process under favourable conditions[6] and a minor local circulatory insuficiency might be responsible for failure of the surviving fibres to conduct. This was experimentally proved by Baschmakoff by his work on dogs.[24] He showed that when a narrow strip of conduction tissue existed, conduction was normal at a slow rate, but was impaired at rapid rate.

Our observation are similar to those of other workers that ischaemic and/or hypertensive heart disease are by far the most important underlying problems in the pathogenesis of this electrocardiographic entity. The demonstration of a cycle length, recovery time, relation stimulated various authors to formulate the concept of critical heart rate in intermittent bundle branch block.[11-12-13] Some even suggested that the change from intermittent to permanent bundle branch block might be effected through a constantly decreasing critical heart rate until such a time as the block was no longer reversible.[3] Some people however, argue against incrimination of the heart rate as the sole determinant responsible for intermittent bundle branch block. Other factors, besides the critical heart rate, were searched for. The effect of vagal impulses and momentary changes in coronary perfusion received much attention.

The bradycardia dependent bundle branch block cannot be explained on the basis of impulses spreading through incompletely repolarised fibres as the case is with tachycardia dependent bundle branch block. It finds a more ready explanation through the concepts of enhanced phase-4 depolarisation.[30] It has been shown that a 'slower' ventricular rate may facilitate phase-4 depolarisation of automatic cells. Spontaneous depolarisation of one or the other bundle branches appears to be the most plausible explanation. Inspite of the previous

considerations bradycardia-dependent bundle branch block is still the subject of a good deal of misconception.[14]

In some cases intermittent bundle branch block changes to permanent bundle branch block as in our case. It was postulated that this may be due to constantly decreasing critical heart rate until such a time as the block was no longer reversible.[3]

What significance or importance should be given to the cases who have no evidence of ischaemic or hypertensive heart disease, or no other obvious cause of intermittent or transient bundle branch block? Should they be treated as potential candidates for ischaemic heart disease and be investigated further with coronary angiography?

Is Sick Sinus Syndrome also the basis in some cases, for the development of rate dependent bundle branch block?

It will be interesting to follow the natural history of these cases and to get more insight to this particular problem.

Summary:

Eight cases of intermittent bundle branch block showing rate dependence are presented. Six cases were tachycardia dependent, one bradycardia dependent and one case showed both Right Bundle Branch Block and Left Bundle Branch Block at different rates.

Tachycardia — dependent bundle branch block was explained on the basis of cycle-length recovery time relation and revealed a critical rate for normal intraventricular conduction. Bradycardia — dependent bundle branch block was best explained on the basis of enhanced phase—4 depolarisation of the bundle branch block system.

Ischemic heart disease and Hypertension appeared to be the most important aetiological factors. The appearance of intermittent bundle branch block in younger age groups could be a marker of ishaemic heart disease developing in the later part of life.

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