

Blocks classification by their constancy or steadiness

By their constance the blocks can be:

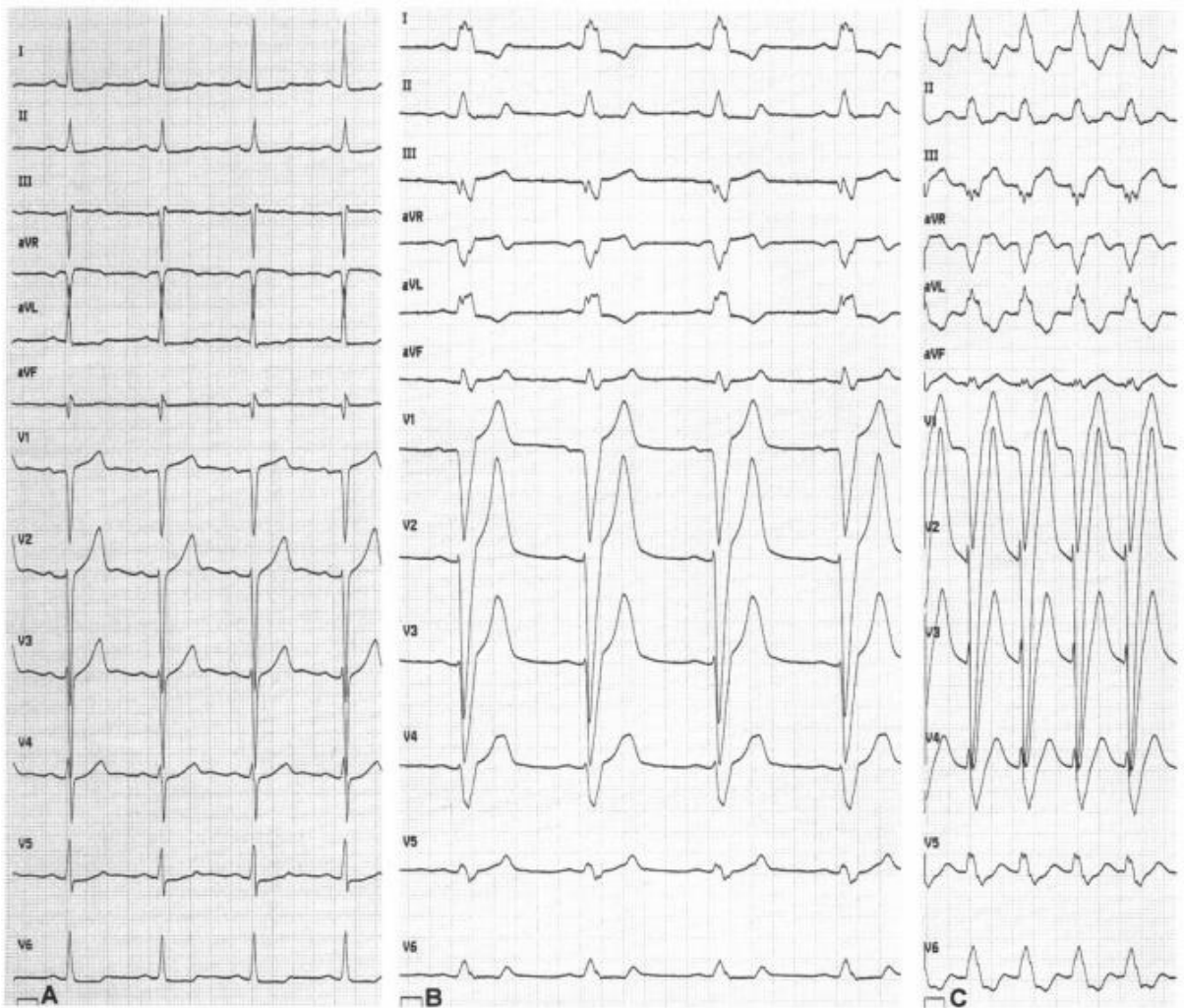
1. Permanent
2. Temporary, transient or transitory
3. Intermittent (**Okajima 1980**):

3a) Dependent on heart rate:

- **Tachycardia-dependent or Phase 3 block (Izumi 1996)** related with increased heart rate (tachycardia-dependent or phase-3 bundle branch block). Phase-3 block is the most common type of paroxysmal intraventricular conduction disturbances (IVCD) documented.
- **Deceleration-dependent bundle branch block, Bradycardia-dependent or Phase 4 block (Kinoshita 2003)** by heart rate reduction: by mild o moderate hypopolarization (enhanced phase IV). Rare. Singer, Lazzara and Hoffman attributed bradycardia-dependent IVCD is attributed, to the inherent capacity of one or another branch to spontaneous diastolic depolarization (“spontaneous diastolic depolarization” or “diastolic hypopolarization”) during phase 4 of the action potential. (**Singer 1967**) The next impulse from the atria or the atrioventricular node would arrive at the branch during the refractory period, in which the cell would be hypopolarized, not reaching the triggering threshold. El-Sherif confirmed the relationship between the increased slope of diastolic depolarization and decreased responsiveness of the cell membrane associated with a variation of the threshold potential towards 0.

- **With both tachycardia- and bradycardia-dependent paroxysmal intraventricular conduction disturbances. coexistence of both mechanisms in the same patient has rarely been described. Very unusual. (Al Hashimi 2004) figure 1**

Figure 1



- Panel A: Basic ECG with a normal interventricular conduction.**
Panel B: bradycardia-dependent or phase 4 LBBB.
Panel C: Tachycardia-dependent or Phase 3 block LBBB.

Aberrant ventricular conduction is the abnormal, asynchronous propagation of an impulse through the His-Purkinje system resulting in a widened QRS complex due either to a delay or block in one of the bundle branches or within the intramyocardial conduction system itself. The occurrence and extent of aberrancy are determined by prematurity of the supraventricular impulse (excitation prior to completion of repolarization), basic RR cycle and RR cycle preceding the premature beat, velocity of the bundle branch, premature enough to reach the cell when the membrane has not been fully repolarized, will be either delayed or blocked. (Sarachek 1970) This type of conduction disturbance may be caused by one or a combination of the following factors:

- *Decreased membrane action potential (AP) due to incomplete repolarization or a low resting potential;*
- *Depressed responsiveness;*
- *Decreased dV/dt independent of the level of resting membrane potential at excitation;*
- *Impedance mismatch (Parameswaren 1970).*

Retrograde-concealed conduction is responsible for its sustained nature. Retrograde invasion into one of the bundle branches leads to what is called **linking phenomenon**. This term indicates the concealed trans-septal retrograde activation of the antegradely blocked bundle branch by the impulse traversing the opposite bundle branch (Hiromitsu 1976) Another form of aberrant conduction is a bradycardia-dependent, deceleration-dependent bundle branch block or Phase 4 block, which might occur following lengthening of the cardiac cycle. Under normal conditions, the membrane potential of atrial and ventricular muscles remains steady throughout diastole. However, in fibres found in certain parts of the sinus node, atria, distal part of the AV-node, muscles of the mitral and tricuspid valve and

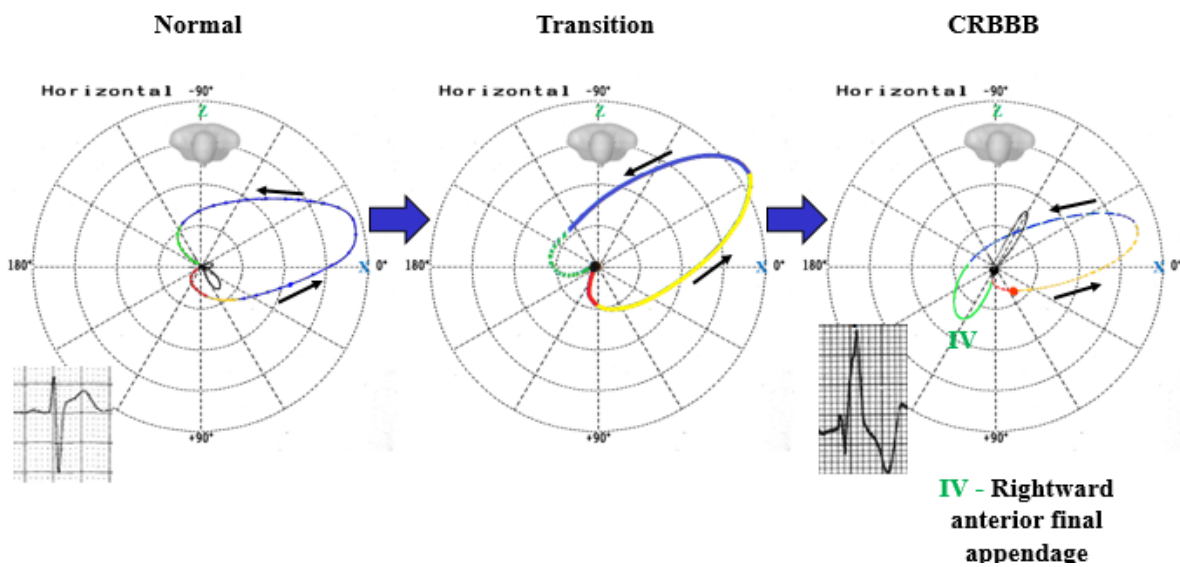
His-Purkinje fibres, the resting membrane action potential does not remain constant in diastole but gradually depolarises. If a triggering impulse does not depolarise the cell, it may reach threshold by itself and produce a spontaneous action potential, a phase IV or spontaneous diastolic action potential. Normally, the sinus node discharge rate exceeds the discharge rate of other potentially automatic pacemaker sites; it dominates the cardiac rhythm. In patients with structural heart disease normal or abnormal automaticity from different sites may discharge at rate faster than the sinus node which may lead to overtaking the cardiac rhythm for one cycle or more. The most common explanation for the deceleration-dependent bundle branch block is enhanced or spontaneous diastolic depolarization (SDD) of automatic cells. Conduction velocity is optimal in fibres with a transmembrane potential of -90 mV, and becomes slower when the membrane potential becomes less negative (-70, -50 mV). During a long pause or long cycle, the fibres of the His-Purkinje system spontaneously depolarize, becoming less and less negative, making it possible for the block to occur with the impulse that ends the pause. It is possible that partial depolarization and failure to reach normal maximal diastolic potential can induce automatic discharge in most if not all cardiac fibres. (Carbone 2002) Less common explanations for the deceleration dependent aberrant conduction are:

- Stretching of the conduction system secondary to transient enlargement of a cardiac chamber during long diastolic intervals resulting from bradycardia, transient hypoxia or ischaemia in the conduction system due to bradycardia;
- Enhanced vagal tone will result in slowing the heart rate and impaired or slowed conduction through the bundle

3b) Independent from hear rate: By severe hypopolarization.

The figure 2 shows the effect on the configuration of the horizontal plane QRS loop produced by development of progressive right bundle branch block during cardiac catheterization. With onset of the block, a rightward anterior final appendage was produced, which gradually went away has the transient BBB cleared. A slight increase in the leftward forces, which were partially uncanceled because of the delay in activation of the RV, occurred with the block and gradually cleared.

Figure 2



For the purpose of obtaining fundamental data to make the diagnostic criteria for coexisting electrocardiographic abnormalities in RBBB, the Frank-VCG/ECG parameters were studied in both normal and RBBB conductions in 25 patients with intermittent or transient RBBB. There were no statistically significant differences in the corresponding instantaneous QRS vectors between normal and RBBB conductions during the initial first 10.4 ± 3.2 ms vector of the QRS-loop. No statistically significant differences existed in the mean Q amplitudes of the

Frank leads between normal and RBBB conductions. After the development of RBBB, voltages of R_X and R_Y, and the planar maximum QRS vectors decreased approximately by 10% on the average, and R_Z decreased significantly by 63.9% on the average, and S amplitudes of the 3 Frank leads increased significantly. The authors observed 3 patients showing a complete reversal of the sense of inscription of the horizontal QRS loop from counterclockwise to clockwise after the development of RBBB. Two of these patients showed no clinical evidence of cardiac or pulmonary diseases except for RBBB. After the appearance of RBBB, maximum T vectors deviated significantly posteriorly in the HP and left sagittal planes(LSP). 23 patients showed counterclockwise inscription of the T loop both in the HP and LSP, and 9 patients in the FP. After the development of RBBB, they observed a complete reversal in the sense of inscription of the T loop from counterclockwise to clockwise in 13 cases (56.5%) in the horizontal plane, in 15 cases (65.2%) in the LSP, and in 8 cases (88.9%) in the FP. (**Okajima 1980**):

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