Other Localized Intraventricular Conduction Defects

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There can be several forms of localized intraventricular conduction defects other than fascicular blocks. These include left septal fascicular block, rate dependent aberrancy, concealed conduction in bundle branches, Ashman phenomenon, peri-ischemic block, peri-infarction block and other focal blocks.

(A) LEFT SEPTAL FASCICULAR BLOCK (LEFT MIDDLE FIBERS BLOCK)

Normally interventricular septum is supplied by fibers from left bundle branch. These fibers are known as septal fibers or middle fibers. These fibers are responsible for normal depolarization of interventricular septum from left to right and anteriorly resulting in initial r wave in V1 and initial q wave in V5, V6 (Fig 1 A). Slow conduction in these fibers (incomplete block) increases the initial right ward vector. This may produce prominent R wave in V1 and RS morphology in V2 (Fig 1 B), q in V5V6 is preserved. Definite electrocardiographic diagnosis is difficult because of several differential diagnoses. Transient ECG changes associated with acute clinical event support diagnosis.

Total block of conduction in these fibers (complete block) may result in loss of initial left to right vector. Interventricular septum is depolarized from right bundle branch with the result that initial QRS vector moves from right to left. This results in loss of septal q wave in V5, V6 (Fig 1 C). Small r may be present in V1 due to activation of free wall of right ventricle through right bundle block.

![Diagram showing effect of septal depolarization on leads V1, V2 and V6. A- Normal depolarization. B- Incomplete left septal block. C- Complete left septal block.](image)

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increase in heart rate.

(b) Bradycardia dependant
Such aberration is seen less frequently. Aberrant conduction occurs after a longer pause (Fig 2 C). Usually it is seen only in presence of significant disease of the conduction system. It is frequently associated with myocardial dysfunction.

(2) Concealed conduction in the bundle branches
This is due to an ectopic beat not manifesting on surface ECG (concealed) but depolarizing part of the conduction tissue. This can be antegrade or retrograde.

(a) Antegrade
This occurs from an ectopic arising in His bundle or proximal bundle branches. Ectopic depolarizes part of the distal conduction tissue (Fig 3 A) but is not strong enough to conduct up to ventricles with the result that there is no ectopic beat on surface ECG. When the next sinus impulse arises through AV node, it finds part of the conduction tissue in partial or absolute refractory period. This results in slow or no conduction through that part of intraventricular conduction tissue with widening of QRS.

(b) Retrograde
Ectopic impulse arising in distal conduction tissue conducts retrogradely without antegrade conduction to ventricles (Fig 3 B). This produces depolarization of proximal part of conduction tissue. Next sinus impulse finds the conduction tissue in depolarized state resulting in slow or no conduction through that part of conduction tissue with broadening of QRS.
Figure 5. Diagram showing transient intraventricular conduction defect (broadening of QRS) during ischemia.

Figure 6. Electrocardiogram showing focal block (notching of QRS) – in leads II, III, aVF.

Figure 7. Electrocardiogram showing focal block (notching of QRS marked with arrow) in leads III and aVF.

Figure 8. Electrocardiogram showing focal block (notching of QRS marked with arrow) in leads III and aVF in the setting of LVH.

Figure 9. Electrocardiogram showing focal block (high frequency notching in QRS marked with arrow) in lead V2 in setting of LVH.

Figure 10. Electrocardiogram showing focal block (notching of QRS marked with arrow) in leads III, aVF and V2.

Figure 11. Diagram showing effect of peri-infarction block on QRS.
(3) Effect of changing cycle length on refractoriness of conduction tissue (Ashman phenomenon)

Refractoriness of conduction tissue is dependent on preceding cycle length. Longer the preceding cycle length, longer is the subsequent refractory period. Supraventricular impulse following such a long cycle finds the conduction tissue in partial refractory period and conducts slowly. On surface ECG it looks like a broad QRS in the short cycle that follows a long cycle-long short sequence. Right bundle branch is commonly involved. This phenomenon is frequent in supraventricular tachyarrhythmias with irregular AV conduction eg. atrial fibrillation (Figure 4).

(4) Peri-ischemic block

It manifests as reversible widening of QRS complex in leads with transient ST segment elevation or depression (Figure 5). It is due to transient focal delay in conduction in the area of ischemia.

(5) OTHER FOCAL BLOCKS

(1) Localized notching in QRS

Such blocks present as multiple deflections or high frequency notches within the QRS without prolongation of QRS duration (Figure 6, 7). Such high frequency notches are usually due to localized fibrosis due to old small infarcts. Such changes can also be seen in long standing systemic hypertension with left ventricular hypertrophy (Figure 8, 9, 10).

(2) Peri-infarction block

There is localized delay in conduction within the tissue surrounding the infarcted region (Figure 11 to 16). Initial QRS vector is directed away from infarction resulting in Q wave. Terminal forces are directed towards infarction producing delayed terminal and slurred r or R. Such blocks can be focal or may involve a fascicle.

REFERENCES
MCQs

LOCALIZED INTRAVENTRICULAR CONDUCTION DEFECTS

Q1. Normally which part of myocardium is depolarized last?
   (A) Apex of LV
   (B) Base of LV
   (C) Base of IVS
   (D) Base of RV

Q2. Normally interventricular septum is depolarized from.
   (A) Left to right.
   (B) Right of left
   (C) Base of apex
   (D) Anterior to posterior

Q3. Loss of septal q wave is not seen in
   (A) LBBB
   (B) Septal infarction
   (C) Left septal fascicular block
   (D) RBBB

Q4. Prominent R wave in lead V1 is not seen in
   (A) RVH
   (B) Dextrocardia
   (C) Incomplete left septal fascicular block
   (D) Emphysema

Q5. What is not correct for tachycardia dependant aberrant conduction?
   (A) It is usually benign
   (B) It can occur in absence of structural heart disease
   (C) It frequently involves left bundle branch
   (D) It is not a marker of significant coronary artery disease

Q6. What is not correct for bradycardia dependant LBBB?
   (A) It is uncommon
   (B) It suggests significant conduction tissue disease
   (C) It is usually associated with myocardial dysfunction
   (D) It is usually benign

Q7. Concealed retrograde conduction in left bundle branch presents as
   (A) Inverted P wave
   (B) Ventricular ectopic with RBBB configuration
   (C) Prolonged PR interval in next sinus beat
   (D) LBBB configuration in next sinus beat

Q8. Concealed antegrade conduction of his bundle ectopic in right bundle branch presents as
   (A) Atrial ectopic
   (B) Ventricular ectopic with LBBB configuration
   (C) Prolonged PR interval in next sinus beat
   (D) RBBB configuration in next sinus beat

Q9. Ashman phenomenon is
   (A) Abnormal phenomenon
   (B) It is seen in short pause following a long pause
   (C) It is seen in long pause following a short pause
   (D) It is seen in sinus tachycardia

Q10. Peri-ischemic block is seen
   (A) Before ischemia
   (B) After recovery from ischemia
   (C) Produces widening of QRS
   (D) Prolongs PR interval

Q11. Peri-infarction block produces
   (A) High frequency notching in QRS
   (B) QRS duration >0.12 second
   (C) Abnormal P wave configuration
   (D) Abnormal T waves