P wave and PR segment

Normal sinus P wave (atrial depolarisation)

P wave is a composite deflection of right and left atrial depolarisation. As sinoatrial (SA) node is situated in right atrium, right atrium depolarises first and left atrium depolarises later (Fig.1a). However, in normal person the time difference in the depolarisation of two atria is not significant with the result that P wave is smooth and rounded in lead II. Upstroke is formed by right atrial depolarisation and down stroke by left atrial depolarisation. In frontal plane the activation of the two atria is directed inferiorly and to left towards positive pole of lead II. Therefore P wave is positive and most clear in lead II. In horizontal plane, the right atrial depolarisation is anterior (towards lead V1) and left atrial depolarisation moves posteriorly (away from lead V1) (Fig.1b). This results in biphasic P wave in lead V1. Initial deflection due to anteriorly directed RA depolarisation is positive and posteriorly directed LA depolarisation produces terminal negative deflection.

- Duration—less than 0.12 seconds.
- Amplitude—less than 0.2mV.
- Frontal plane P wave axis—+45° to +65° (Fig. 2). Sinus tachycardia deviates frontal plane P wave axis to right by about 10° to 15°.
- In lead V1 terminal negative deflection is less than 0.1 millivolt in depth and less than 0.03 seconds in width.

Atrial repolarisation (Ta wave)

It is a low amplitude wave with a polarity against P wave. It is usually not seen because of low amplitude and superimposition by QRS complex. It may be visible during A-V block (Fig. 3).

Atrial depolarisation abnormality

Depolarisation abnormality is a better term than enlargement because such abnormalities can also occur in atrial overload and intra-atrial conduction defects (Fig. 4) without any echocardiographic evidence of enlargement.

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‘A’ like peaked (pointed) P wave in leads II and aVF (Fig. 6c). There is no increase in duration. Amplitude may not be more than 0.2mV. Although amplitude criterion has high specificity, sensitivity is low. As such P waves are commonly seen in cases of cor-pulmonale, they are also called “P-pulmonale.”

In patients with gross right atrial enlargement, terminal negative deflection of P wave in lead V1 may also be prominent giving wrong impression of concomitant left atrial enlargement. This is more likely to occur in patients with vertical heart as in emphysema. In vertical heart conventional lead V1 remains higher in relation to right atrium, with the result that the right atrial activation moves away from lead V1 (Fig. 7a). However, unlike left atrial enlargement, the downstroke of P wave is rapid with an intrinsicoid deflection of less than 0.03sec. Also, there is no evidence of biatrial enlargement in lead II. Lead V3 frequently shows tall and pointed P wave (Fig. 8a and 8b).
Such abnormalities may also be transient during acute LVF or acute pulmonary embolism.

**Left atrial depolarisation abnormality**

It manifests as:

- Frontal plane P wave axis of less than +45°
- P wave duration of 0.12 sec or more
- Notched, bifid P wave (M like appearance) with a distance of more than 0.04 sec between two peaks (Fig. 6a) (sensitivity 15%, specificity 100%) in lead II, V5 and V6. As such P waves are frequently seen in mitral valve disease, they are also called “P-mitrale.”
- Negatively directed portion of P wave in lead V1 is of 0.04 sec or more in duration (Fig. 6a) (sensitivity 83%, specificity 80%) and 0.01 mV or more in amplitude (sensitivity 60%, specificity 93%). At times top P wave in lead II may not show clear notch but only flattening (Fig. 6b). Similarly the negative terminal component in lead V1 may not be deep but may be only broad (Fig. 6b)

**Right atrial depolarisation abnormality**

It is characterised by:

- Right-ward shift of P wave axis to around +75°. Sinus tachycardia also tends to deviate the frontal plane P wave axis to right by about 10° to 15°

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**Fig. 3:** Lead II in a case of inferior myocardial infarction and first degree AV block showing atrial repolarisation wave (Ta wave) (arrow)

**Fig. 4:** Continuous strip of lead II showing M shaped and tall peaked P waves in same patient over a short period due to changing intra-atrial conduction defect

**Fig. 5:** ECG features of atrial depolarisation abnormalities. LAE- Left atrial abnormality RAE- Right atrial abnormality BAE- Biatrial abnormality

**Fig. 6(A):** Lead II showing M shaped P wave. Lead V1 showing deep and broad terminal negative deflection of P wave

**Fig. 6(B):** Lead II showing only flattening of top of P wave in lead II and increased duration of terminal negative deflection of P wave in lead V1.

**Fig. 6(C):** Tall and peaked P wave
QR in lead V1: In patients with gross right atrial enlargement with right ventricular displacement lead, lead V1 may show a small Q wave before a prominent R wave. This occurs because of anatomical shift of RV by an enlarged and dilated RA with the result that V1 records potential of proximal basal region of RV (Fig. 7b, 8c).

Biaatrial depolarisation abnormality

It manifests as

- Lead II shows broad and bifid P wave with initial component taller than terminal component. Lead V1 shows P wave with tall positive initial component followed by deep, wide and delayed terminal component (Fig. 9).

Atrial ectopics (atrial premature beats-APB)

These are characterised by

- Premature and abnormally appearing P wave (Fig. 10a, b). Very premature P wave may fall in the T-wave of previous sinus beat and may be missed unless looked very carefully. A little tall, more pointed or slightly notched preceding T wave (Fig. 10c, d) as compared to T wave of sinus beat should raise suspicion of P wave merged in T wave. APB arising in close proximity of SA node may produce a P wave similar to sinus beats.

- Negative P wave in inferior leads II, III, aVF suggests origin in lower atrium (Fig. 11a). Left atrial origin of ectopic rhythm produces P wave inversion in leads V5 and V6 in addition to inverted p wave in lead II, III, aVF (Fig. 11b). Uncommonly, left atrial rhythm may produce “dome and dart” appearance in lead V1. There is initial slow upstroke (dome like) followed by a sharp upstroke (dart). Atrial ectopics could arise from more than two sites (multifocal) (Fig. 11c).
PR interval of atrial ectopic beats is usually slightly longer than the PR interval of normal sinus beat due to relative refractoriness of AV node (Fig. 12a). If atrial premature beat occurs so early that AV node is in its absolute refractory period, APB may not be conducted to ventricle with the result that there is no QRS complex following APB (blocked or non-conducted APB) (Fig. 12b). Non-conducted APBs may occur in a bigeminal pattern (Fig. 12c). ECG so produced may be confused with sinus bradycardia or SA exit block specially if the blocked APB is merged in the preceding T-wave and is not identified.

If ectopic beat arises from AV junction, PR interval can be short.

QRS complex following an ABP is usually similar to that of normal sinus beat because of normal conduction through ventricular conduction system (Fig. 10a, b). If the APB is significantly premature or refractory period of bundle branches or fascicles is prolonged, APB may find the ventricular conduction system in partly refractory state. This results in slow ventricular conduction through conducted APBs may occur in a bigeminal pattern (Fig. 12c). ECG so produced may be confused with sinus bradycardia or SA exit block specially if the blocked APB is merged in the preceding T-wave and is not identified. If ectopic beat arises from AV junction, PR interval can be short.

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one or other bundle branch or fascicle resulting in abnormal QRS- aberrant conduction.

- Differentiation of APB with aberrant ventricular conduction from premature ventricular beats:
  - Identification of a preceding P wave supports aberrant conduction. It may be difficult to identify if it falls in preceding T wave.
  - Initial deflection of QRS identical to sinus beat supports aberrancy (Fig. 10a, b).
  - If the broad QRS has RBBB configuration, rSR configuration in lead V1 and QRS configuration in lead V6 supports aberration (Fig. 13a).
  - Uncommonly aberrant conduction occurs over LBB specially in patients with cardiac disease.
  - If there is a cluster of ectopic beats, then broadening of QRS in second beat of cluster supports aberrancy (Fig. 13b).
  - Long QRS cycle followed by a short cardiac cycle with broad QRS (long-short sequence) is not helpful as it can occur in aberrant conduction as well as in ectopic. However, absence of a long preceding cycle supports ectopic activity.
  - If an even longer cycle, followed by an even shorter cycle is conducted with a normal QRS, broad QRS beats are suggestive of PVC.
  - Varying degrees of change in QRS depending on length of preceding normal RR cycle supports aberrancy.
  - Alternating patterns of bundle branch block separated by single normal beats supports possibility of bilateral aberrancy rather than alternate PVC arising alternately from the two ventricles (Fig. 13c).

Aberrant conduction is rate dependent. Usually justified it with a short cycle length (Fig. 13a). Uncommonly it can be bradycardia dependent.

Post ectopic pause:
Pause following an APB is longer than normal PP cycle (Fig. 10a, b, Fig. 14a) because it includes:
• Time for atrial impulse to enter SA node and depolarise it
• Normal SA node cycle length
• Exit time for impulse to come out of SA node to atrium

However, this pause is less than double of normal PP cycle length (incomplete compensatory pause). This pause, however, may be variable due to several factors:
• Underlying SA node dysfunction. If the post ectopic pause is longer than 25% of the original basal cycle, there is probability of structural SA nodal disease (Fig. 14b)
• Temporary suppression of SA node automatically by atrial ectopic beat.
• Sinus arrhythmia.
• Blocking of sinus P-wave (Fig. 14c)

Atrial ectopics can occur after each sinus beat (bigeminy) (Fig. 15a) or after two sinus beats (trigeminy). Frequent atrial ectopics can precede atrial tachyarrhythmias. When the interval between sinus P-wave and atrial ectopic beat is less than 50% of the preceding PP cycle, APB is more likely to initiate a reentrant atrial tachyarrhythmia. In such instance APB forms the first beat of atrial tachycardia / flutter or fibrillation. QRS following an atrial ectopic is mostly normal in configuration. Rarely the beat following compensatory pause may show broadening of QRS (bradycardia dependant aberration) (Fig. 15b).

Atrial ectopics occur in conditions with atrial overload e.g., mitral valve disease, tricuspid valve disease, left and right ventricular dysfunction. They can also occur during infection, inflammation, thyrotoxicosis, tension states or consumption of tobacco, alcohol or caffeine. They can also occur without any apparent cause. Management is usually directed towards underlying cause. In absence of underlying cardiac disease, β-blockers and diltiazem may be helpful.

Junctional premature beats
Premature beats arising from AV junction also produce inverted P waves in lead II, III and aVF. Relation of P wave to QRS depends on relative retrograde velocity to atrial and antegrade velocity to ventricles. Faster retrograde velocity (common with premature beat arising in proximal AV node) produces inverted P wave before QRS (Fig. 16a). In premature beats with equal retrograde and antegrade velocity, P wave is hidden in QRS (Fig. 16b). If retrograde conduction time is longer than antegrade conduction to ventricles, inverted P wave follows QRS (Fig. 16c). This is common when ectopic focus is in distal AV node or proximal HIS bundle.

Junctional ectopic may or may not be able to penetrate SA node. Sinus rhythm, therefore, may or may not be affected. Post ectopic compensatory pause, therefore, may be incomplete or complete.

PR segment
The segment between end of P wave and beginning of QRS is isoelectric. Acute pericarditis can be associated with depression of PR segment in all leads (Fig. 17a,b) other than aVR and V1. Absence of this finding, however does not exclude the diagnosis. Rarely atrial infarction may be associated with elevation or depression of PR segment (Fig. 17c) like ST-segment changes in ventricular infarction. Other findings like intraatrial conduction defects or atrial tachyarrhythmia are more common in patients with atrial infarction. PR segment deviation in a case of ventricular infarction are more likely to be due to concomitant pericarditis rather than atrial infarction. Sinus tachycardia during exercise testing is associated with down sloping PR segment which smoothly merges with rapid up-sloping ST segment (Fig. 17d).

Reference