3.3 Intervals and waveforms: A–Z of abnormalities

ABERRANT CONDUCTION
See Heart rate and rhythms section, p. 80.

ANEURYSM (LEFT VENTRICULAR)
The presence of persistent ST segment elevation of greater than 1 mm in the precordial leads several days or weeks following an acute myocardial infarction is suggestive of a ventricular aneurysm. This finding is most often associated with an acute anterior myocardial infarction when the ST segment elevation either reappears or persists beyond the subacute phase of MI. Elevation persisting for greater than one week (but more often greater than three months) post MI warrants further investigation because the aneurysmal ventricular wall can be a potential site of left ventricular thrombus and the initiation of ventricular arrhythmias. The test of choice if an aneurysm is suspected is an echocardiogram.

ASYSTOLE
See Cardiac arrest rhythms (pp. 83–84).

AXIS DEVIATION
See above (p. 104).

BIATRIAL ENLARGEMENT
As the term implies, biatrial enlargement is simply a combination of left and right atrial enlargement (see separate entries for each) and is commonly seen in advanced cardiomyopathy, multivalvular heart disease and in congenital heart disease. P waves are typically both broad and tall, often more than 3 mm tall in limb leads.

BIFASCICULAR BLOCK
When the right bundle branch becomes blocked in combination with a block in either fascicle of the left bundle branch the term bifascicular block is used. Bifascicular block manifests itself on the ECG as right bundle branch block (RBBB) with either left or right axis deviation. If the anterior fascicle is blocked (which is most commonly the case) then left axis deviation will be present. If the posterior fascicle is blocked (rarely) then right axis deviation is seen. (See also Right bundle branch block (p. 145) and section on Cardiac axis (p. 104)).

Fig. 3.70 Left ventricular aneurysm.
This ECG was taken from a 62-year-old gentleman during a visit to cardiology outpatients. He had sustained a large anterior MI six weeks before, and was now complaining of persistent chest pain and shortness of breath on moderate exertion.

Key features
- Persistent ST elevation in precordial leads following an acute MI
HOW TO READ AN ECG

3.3. A–Z of abnormalities

Fig. 3.71 Bifascicular block: right bundle branch block with left axis deviation. This ECG was recorded in the A&E department from a 60-year-old male who was later admitted due to loss of consciousness. On further questioning, he claimed to have sustained two further episodes of drop attacks over the last 12 months. He smoked 20 cigarettes a day for several years, and was on beta-blocker treatment for hypertension.

Key features
● RBBB with either a left or right axis deviation

Fig. 3.72 Dextrocardia (standard leads).

Key features
● Reversed R wave progression
● Inverted complexes in lead I
● Right axis deviation

CAUSES
● As for other fascicular blocks.

BRUGADA SYNDROME

See Sudden cardiac death syndromes (p. 147).

BUNDLE BRANCH BLOCK

The presence of block (either complete or incomplete) in the left or right bundle branch causes an abnormal spread of depolarisation through the ventricles and a greater amount of time is taken than usual for ventricular activation to occur. See Left bundle branch block and Right bundle branch block under separate entries (pp. 145 and 119 respectively).

Hints and tips
● Having established that a broad complex QRS is present the key to determining which type of bundle branch block is in evidence – at a glance – is to look for the M-shaped notching (sometimes called “bunny ears”) in the chest leads. If there is M-shaped notching of the left-sided chest leads (leads V5 and 6) then LBBB is present. Conversely, M-shaped notching in the right-sided chest leads (V1–V3) indicates RBBB.

DEXTROCARDIA

Dextrocardia is a congenital abnormality in which the heart lies off to the right side of the chest instead of the left. The pattern in both the chest leads and the limb leads are...
affected. Because of the change in orientation the chest leads show reversed R wave progression with the QRS complexes becoming increasingly negative from V1 through to V6. The limb leads will show an inverted complex in lead I and right axis deviation. In patients with dextrocardia right-sided chest leads (V1R–V6R) should be recorded. (See also Special considerations, p. 32, in Chapter 2.)

Dextrocardia may occur in isolation or in the presence of situs inversus where the abdominal organs are also transposed. Isolated dextrocardia is often associated with other congenital lesions such as transposition of the great vessels, pulmonary stenosis, tetralogy of Fallot, truncus arteriosus and ventricular or atrial septal defects.

**DRUG EFFECTS**

Changes in the appearance of the ECG and the incidence of particular arrhythmias can be caused by the actions of certain drugs. Unfortunately there are a number of antiarrhythmic drugs which have, as a side-effect, the ability to cause arrhythmias. These are suitably described as pro-arrhythmics.

All of the agents listed in the Vaughan-Williams classification of antiarrhythmic drugs in Table 3.7 below can potentially cause abnormalities on the ECG.

Beta-adrenergic receptor antagonists and some calcium channel blockers may produce sinus bradycardia and varying degrees of AV block.

▶ Fig. 3.73 Dextrocardia with right-sided chest positions results in normal precordial R wave progression and upright complexes in lead I.

▶ Fig. 3.74 Atrial fibrillation with digoxin effect demonstrated by downsloping ST segments in leads II, III, aVF and V5 and V6. The ECG was recorded from a 40-year-old man who had been admitted with AF with an initial ventricular rate of 140 bpm. He had been feeling light-headed, and was started on IV, followed by oral digoxin. You should note that these ECG changes are not related to ischaemia.
Table 3.7 Vaughan-Williams classification

**Class I:** Fast sodium channel blockers
- Ia: quinidine, procainamide, disopyramide
- Ib: lidocaine, phenytoin, mexilitene, tocainide
- Ic: encainide, flecaïnide, propafenone

**Class II:** ß adrenergic receptor antagonists
- (e.g. propranolol)

**Class III:** Potassium channel blockers
- (e.g. sotalol, amiodarone, dofetilide, ibutilide, bretylium)

**Class IV:** Calcium channel blockers
- (e.g. verapamil, diltiazem, nifedipine)

Digoxin and quinidine-like agents (class Ia) can potentially produce ventricular arrhythmias. Digoxin may cause a short QT interval and a prolonged PR interval on the ECG. T wave inversion and downsloping ST segment depression is produced by the so-called “digoxin effect.”

Quinidine has the ability to prolong the QT interval, widen the QRS and precipitate AV nodal blocks. Drugs such as flecaïnide may cause bundle branch blocks and, occasionally, ventricular tachycardia. Flecaïnide can also change pacemaker thresholds and cause loss of capture during pacing.

It is helpful, then, to have to hand details of a patient’s current medication when interpreting an ECG.

### HYPERCALCAEMIA

**Definition:** Elevated calcium levels in the body.

**Table 3.8 Causes and ECG findings in hypercalcaemia**

<table>
<thead>
<tr>
<th>Causes</th>
<th>ECG features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drugs such as thiazide diuretics</td>
<td>Short QT interval</td>
</tr>
<tr>
<td>Excessive vitamin D intake</td>
<td>Prolongation of PR interval and QRS complex (less commonly)</td>
</tr>
<tr>
<td>Hyperparathyroidism</td>
<td>Ventricular arrhythmias</td>
</tr>
<tr>
<td>Malignancy</td>
<td></td>
</tr>
<tr>
<td>Milk-alkali syndrome</td>
<td></td>
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<tr>
<td>Sarcoidosis</td>
<td></td>
</tr>
<tr>
<td>Thyrotoxicosis</td>
<td></td>
</tr>
</tbody>
</table>

### HYPERKALAEMIA

**Definition:** Elevated potassium levels in the body.

**Table 3.9 Causes and ECG findings in hyperkalaemia**

<table>
<thead>
<tr>
<th>Causes</th>
<th>ECG features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Renal failure</td>
<td>Tall T waves ≥ associated R waves in more than one lead (potassium levels between 3.5–6.5 mmol/l)</td>
</tr>
<tr>
<td>Acidosis</td>
<td>/-</td>
</tr>
<tr>
<td>Shock</td>
<td>Low amplitude “flattened” P waves</td>
</tr>
<tr>
<td>Overadministration of potassium</td>
<td>Prolongation of PR interval (potassium levels between 6.5–7.5 mmol/l)</td>
</tr>
<tr>
<td>Potassium-sparing diuretics</td>
<td>/-</td>
</tr>
<tr>
<td>Angiotensin-converting enzyme inhibitors</td>
<td>Broad QRS complex (potassium levels between 7.0–8.0 mmol/l)</td>
</tr>
<tr>
<td></td>
<td>/-</td>
</tr>
<tr>
<td></td>
<td>Ventricular arrhythmias, asystole (potassium levels between 8.0–10.0 mmol/l)</td>
</tr>
</tbody>
</table>

**Fig. 3.75** This is hyperkalaemia secondary to renal failure and potassium-retaining medication as demonstrated by peaked T waves in all leads and widespread broadening of the QRS complexes. The ECG was taken from a 58-year-old male with hypertension, peripheral vascular disease and heart failure. His drug history included ramipril 10 mg od and spironolactone 25 mg od. His serum creatinine was noted to be 350 µmol/l.