

Preface

The ECG is a wonderful test – what other investigation can decide so quickly, easily and cheaply whether the heart is undergoing necrosis, where the pathology is located, indicate whether the lining of the heart is inflamed, whether the patient has a background of long-standing high blood pressure, an underactive thyroid, or an important genetic diseases, whether the patient is going to blackout, why they have blacked out, how fit they are, when they go to bed, when they get up?

The ECG is one of the most powerful of clinical investigations. However, there is a down-side – the ECG doesn't always tell the truth. It is liable to misinterpretation and can fool the unwary. It may suggest critical coronary disease when in fact the coronary arteries are normal; it can appear to indicate a myocardial infarct when the heart muscle is actually perfectly healthy. It can also suggest that the heart is completely normal, when in fact the coronary arteries are critically diseased. It can imply that the pumping function of the heart is normal, when it is substantially impaired. It can suggest that there is no serious cause for an arrhythmia, when in fact the heart is prone to stopping for prolonged periods of time.

So, used correctly the ECG allows great insights into the functioning of the heart, but used incorrectly it can lead the unwary clinician into all sorts of errors. The aim of this book is to provide the information that will turn the reader into a 'savvy' ECG reader, one who can handle the ECG calmly, confidently and accurately. He/she will not be led astray, and will understand when the ECG tells the truth and when it does not.

Understanding the basic mechanisms that underline the generation of the ECG signals is crucial to such an understanding – this book enables this, using a multitude of diagrams. However, mere mechanics are not sufficient to a full understanding – one needs to know how clinical factors interact with the ECG. The reader of this book will have the information needed to correctly interpret the clinical ECG in a plethora of clinical contexts. Indeed, the fundamental philosophy of this book is that the ECG will only be useful

when it is combined with data on the clinical situation –furthermore, that the ECG cannot be understood without this clinical data. This is the key message of the book.

I hope this book leaves you with a full understanding of the ECG, a confident sense of its uses and limitations, and a thirst to learn even more about this fascinating test.

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Vasculitis and autoimmune disease. Rheumatoid arthritis and SLE can infrequently cause isolated pericarditis. Following an MI or any cardiac operation that involves cutting through the pericardium ('pericardotomy'), autoantibodies against the pericardium may develop, provoking pericardial inflammation and pain. Dressler's syndrome is the name given to post MI autoimmune pericardial inflammation. This is usually a self-limiting illness causing retrosternal pleuritic chest pain, sometimes shoulder pains and occasionally systemic upset (malaise, anorexia and fever). It usually responds to nonsteroidal anti-inflammatory drugs, although very occasionally steroids are required.

Malignancy is an exceptionally rare cause of pericardial pain, although it can cause painless pericardial effusion and tamponade. Occasionally a very large pericardial effusion causes a dull, continuous retrosternal chest ache due to stretching of the pericardium. However in this situation features other than chest pain often dominate the clinical situation (preceding weight loss, malaise, and signs of tamponade including low blood pressure, pulsus paradoxus, raised venous pressure and progressive renal failure).

The ECG may be completely normal in those with pericardial pain or may show the classic changes of pericarditis (see p. XXX), with 'concave upwards' ST segment elevation often occurring in many leads (Figure 5.4). Over time (several days to weeks) the ST segments first flatten, then the T wave inverts, followed by return of the ECG to normal. The ECG changes may become 'arrested' at any stage in this process. In addition to these changes, the ECG may show changes relevant to any other ongoing disease process (eg Q waves in peri- or post-MI pericarditis).

Large pericardial effusions classically cause two abnormalities: first, a decrease in the size of the ECG complexes; and second, 'electrical alternans', wherein alternate beats vary dramatically in size or axis, as the heart swings around within the pericardial sac (Figure 5.5).

Oesophageal chest pain

Pathology of the oesophagus can cause two quite distinct pains:

Reflux pain occurs when the oesophageal mucosa is damaged and acid irritates the submucosal nerves. It is felt as a retrosternal burning ('heartburn') often worse lying flat, usually unrelated to effort and often lasting many hours. Abdominal obesity is a potent predisposing factor. Milk and other fluids may relieve symptoms. Belching may accompany the pains,

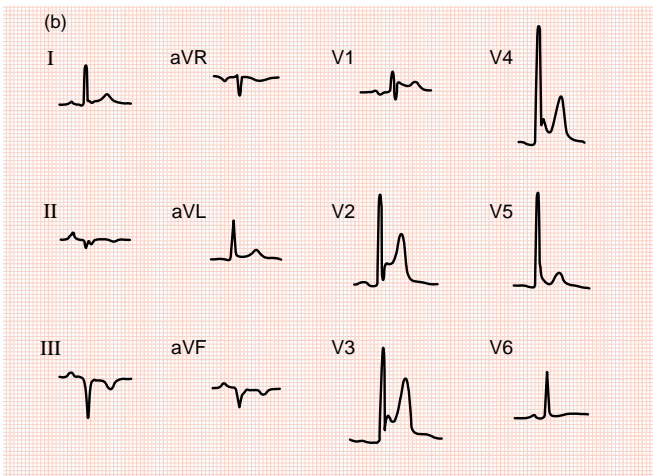
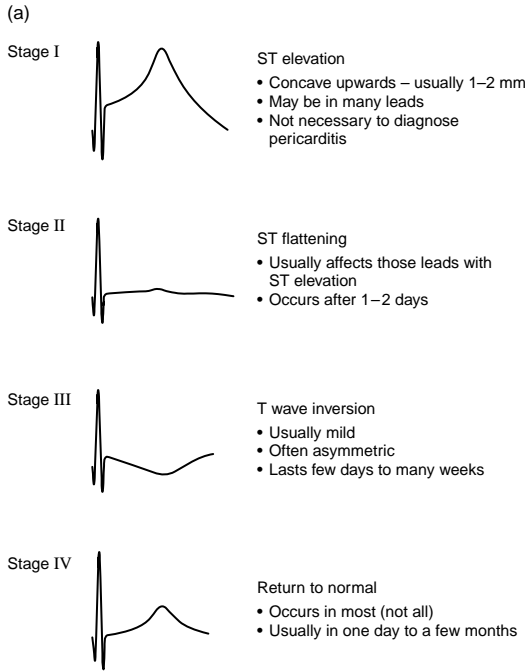


Figure 5.4

(a) Sequential ECG changes in pericardial disease. (b) Q wave inferior wall myocardial infarct with anterior lead ST elevation due to complicating pericarditis.

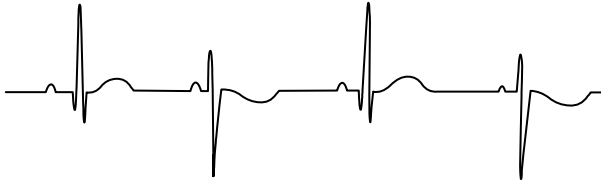


Figure 5.5

Electrical alternans (also termed 'cardiac nystagmus') in pericardial effusion. The QRS (\pm T wave) direction and amplitude may alternate with every heart beat, as here, (or may change more slowly over every three to four beats) due to dramatic shifts in heart position within the fluid-filled pericardial sac. However, note that less than 10% of patients with cardiac tamponade show electrical alternans.

but this may also occur in myocardial ischaemia. Likewise, the relationship to effort does not always discriminate between the two, as oesophageal reflux (as well as spasm) can sometimes be provoked by exercise, which relaxes the oesophagogastric sphincter; however pains often take a long time to resolve after effort. The ECG is normal.

Oesophageal spasm can very easily be confused with myocardial ischaemia as both pains are due to ischaemia in structures within the mediastinum (the smooth muscle of the oesophageal wall and the heart, respectively). Acid reflux induces the smooth muscle in the oesophageal wall to go into spasm, generating a pressure greater than that of the arterioles contained within it, so interfering with its blood supply and provoking oesophageal ischaemia. This results in a 'tight' pain which is often very uncomfortable [the patient tries numerous positions to seek relief, whereas in myocardial ischaemia the patient often (but not always) sits quietly]. The pain may be severe. As it is caused by acid refluxing from the stomach into the oesophagus, factors that promote acid reflux (obesity, especially abdominal, lying down) promote symptoms, which are often relieved by antacids. The ECG is usually normal but pain and anxiety can induce ST changes (see Chapter XXX).

If oesophageal pain is a serious possibility a trial of a proton pump inhibitor (PPI) may be diagnostic. Endoscopy has a role in a small number of PPI-resistant patients, and oesophageal pH monitoring has a role in an even smaller number of patients. Most patients with oesophageal pain have normal resting ECG and exercise tests. However, recent data shows that in a very small number of patients oesophageal pain can, through an oesophagocardiac reflex, cause coronary vasoconstriction and thus exercise-induced ST depression in the absence of atherosclerotic CAD.

Aortic pain

Aortic pathology (aortic dissection and aortic aneurysm) gives rise to two different sorts of pain.

Aortic aneurysm

Exceptionally rarely, a massive aortic aneurysm can give rise to chest pain; this is usually due to the aorta compressing adjacent structures (ribs, vertebra etc) that carry somatic innervation rather than arising from the aorta itself.

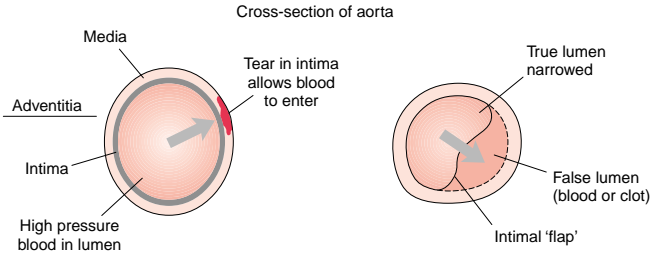
Aortic dissection

By far the most important cause of aortic pain is aortic dissection (Figure 5.6). This pain has a number of key features:

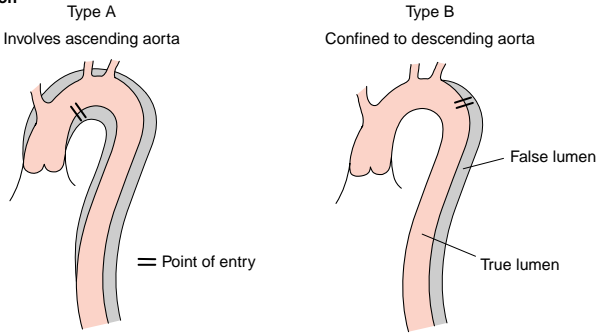
- It is often ‘tearing’ or ‘sharp’ in quality. Many patients find the quality of the pain difficult to articulate.
- It is severe, often exceptionally so and requires frequent opiates for relief.
- The pain usually (but not always) lasts for many hours. Thus transient severe chest pain (eg less than few minutes) is usually not due to dissection.
- The site where the pain is felt depends on which part of the aorta is dissected.
- Ascending aortic pain is felt in the anterior chest, aortic arch pain is felt in the neck, and descending aortic pain is felt in the back.
- The fact that a dissection propagates rapidly (within a few minutes) distally from its entry point is reflected in the symptoms. In a type A dissection (originating in the ascending aorta) pain classically starts in the anterior chest then moves rapidly into the neck and then into the back, whereas in type B dissection (originating below the origin of the left subclavian artery) pain starts in the middle of the back and may propagate more distally as the dissection travels down the abdominal aorta.

Examination shows a patient in obvious pain (which may itself cause tachycardia). Hypertension is common. Pulses may be lost (typically in the left arm and also the femoral arteries, although any pulse can be affected). Carotid or spinal artery damage may cause neurological damage (stroke or paraparesis). In proximal aortic dissection, the tear tracks back, rupturing into the pericardium and resulting in tamponade and death, usually within a number of hours. Occasionally proximal tracking of the dissection ‘tears’ a

Pathology



Classification



Complications

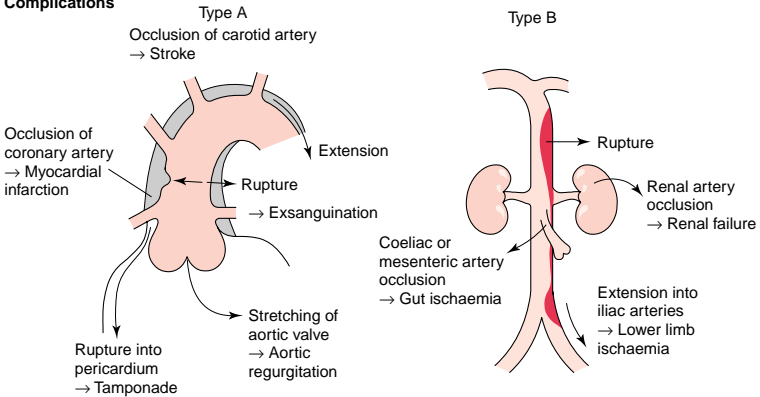


Figure 5.6

Consequences of aortic dissection. Reproduced with permission from Davey P. *Medicine at a Glance*. Blackwell Science Ltd, Oxford.