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Understanding the ECG. Part 7: Chamber enlargement

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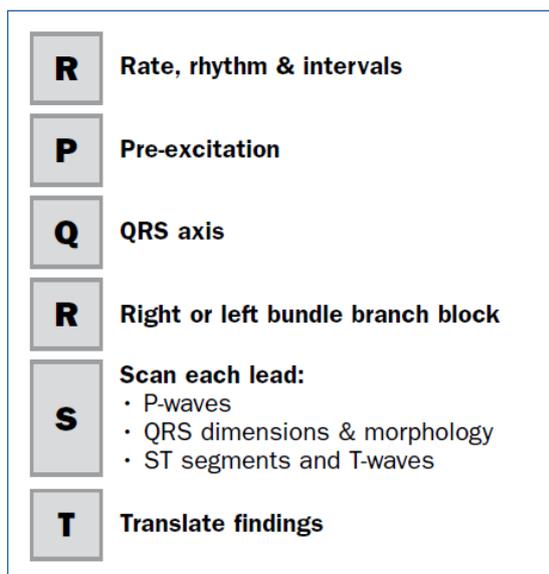
Introduction

Enlargement of the cardiac chambers is common, and results from diseases affecting the heart and lungs (Houghton & Gray, 2014). The growth in chamber size or wall thickness reflects disease progression, and is associated with increasing morbidity and mortality (Artham et al, 2009; Haddad et al, 2008). Although echocardiography or magnetic resonance imaging (MRI) is required to confirm chamber enlargement, the ECG often reflects these changes, and is a more widely performed test (Rautaharju & Soliman, 2014).   An abnormal ECG may therefore provide the first evidence of an underlying disease process, and a need for further investigation and treatment (Hampton, 2013a). Because of this, a search for the signs of chamber enlargement should be included in any structured assessment of the 12-lead ECG (Garcia, 2015).

Recognising chamber enlargement is also important from a broader diagnostic viewpoint (Houghton & Gray, 2014). On the ECG, chamber enlargement may alter the size and shape of the QRS complex, and result in ST-segment depression and T-wave inversion (Hancock et al, 2009). A failure to recognise the cause of these changes could lead to misdiagnosis, particularly in the setting of suspected myocardial ischaemia (Edhouse et al, 2009).

In this seventh part of our ECG series, we briefly revisit our structured ECG interpretation tool before turning our attention to chamber enlargement. We discuss why chamber enlargement occurs, and describe how it can be recognised on the ECG using a variety of criteria. We also consider the limitations of using the ECG to screen patients for chamber enlargement, and examine the sensitivity and specificity of published criteria.

Structured ECG interpretation

By now, regular readers of this series will appreciate that the 12-lead ECG contains a great deal of information (Garcia, 2015). In the second article in this series, we discussed the use of a structured approach to interpretation, and highlighted its value in ensuring that important information is not overlooked (Sampson & McGrath, 2015). We proposed the use of the RPQRST system, which is shown in *figure 1* (Gregory, 2006). Let's take a few moments now to revisit this tool, and to briefly review the ground that we have covered so far.

R	Rate, rhythm & intervals
P	Pre-excitation
Q	QRS axis
R	Right or left bundle branch block
S	Scan each lead: • P-waves • QRS dimensions & morphology • ST segments and T-waves
T	Translate findings

Figure 1. System of interpretation adapted from Gregory (2006) with permission

The first step in structured assessment of the ECG is to evaluate heart rate, rhythm, and conduction intervals. *Box 1* suggests the key questions that we need to ask when evaluating rate and rhythm, while *table 1* outlines normal conduction intervals (Sampson & McGrath, 2015). If heart rate, rhythm or intervals are abnormal, further analysis should be made to determine the cause. Common causes for abnormalities include sinus bradycardia or tachycardia, arrhythmias and conduction blocks (Sampson, 2016a; Sampson, 2016b). Remember that a prolonged corrected QT interval (QTc) suggests acquired or inherited long QT syndrome, and is associated with an increased risk of ventricular arrhythmias (Mizusawa et al, 2014).

1. Is the rhythm regular?

2. Is the heart rate between 60 and 100 beats per minute?

3. Are there upright P-waves, and are they all the same shape?

4. Is there one P-wave in front of each QRS complex?

5. Is the PR interval normal (3 to 5 small squares?)

6. Is the QRS complex narrow (less than 3 small squares wide?)

Box 1: Rhythm evaluation questions

| Interval	Normal value
PR interval	0.12 to 0.20 seconds (3 to 5 small squares)
QRS complex	Less than 0.12 seconds (< 3 small squares)
Corrected QT interval (QTc)	Not greater than 450ms in men
	Not greater than 460ms in women

Table 1. Normal conduction intervals.

The second step is to search for signs of pre-excitation on the ECG. These include a short PR interval, a wide QRS, and a delta wave (Mark et al, 2009). We need to remember that pre-excitation alters the electrical activation of the ventricles, and therefore the shape of the QRS complexes (Maden et al, 2015). Pre-excitation can alter normal R-wave progression, and cause axis deviation (Sampson, 2016c). Patients with pre-excitation need specialist evaluation because of an increased risk of arrhythmias and sudden cardiac death (Obeyesekere et al, 2012).

The third step is to evaluate QRS axis using the limb leads (*table 2*). The normal axis lies between -30 and +90 degrees, with any value outside this range referred to as axis deviation (Surawicz et al, 2009). Axis deviation results from numerous conditions including fascicular blocks, ventricular hypertrophy, pre-excitation, and myocardial infarction (Burns, 2014). It may also be a normal variant, especially in people who are especially tall and thin (right axis deviation), or short and broad (left axis deviation). A finding of axis deviation should trigger thorough evaluation of the 12-lead ECG to determine the cause, and further investigations if necessary (Sampson, 2016d).

	Lead I	Lead aVF	Lead II
Normal axis	**+**	**+ or -**	**+**
Left axis deviation	**+**	**-**	**-**
Right axis deviation	**-**	**+**	**+ or -**
Extreme axis deviation	**-**	**-**	**-**

Table 2. Polarity of limb leads in normal and abnormal axis (+ = positive, - = negative)

The final step that we have covered so far is consideration of bundle branch block. Failure of conduction through either the left or right bundle branch delays ventricular depolarisation, and therefore widens the QRS complex (Sampson, 2016b). Bundle branch block should be suspected if the QRS is wide despite a normal rhythm. Because the pattern of ventricular activation is altered, characteristic changes in the appearance of the QRS complexes are seen, according to whether the right or left bundle is blocked (Aehlert, 2011). These changes are compared in *figure 2.* Bundle branch blocks suggest underlying cardiovascular disease, and can result from acute myocardial infarction, so warrant clinical evaluation (Kumar et al 2013).
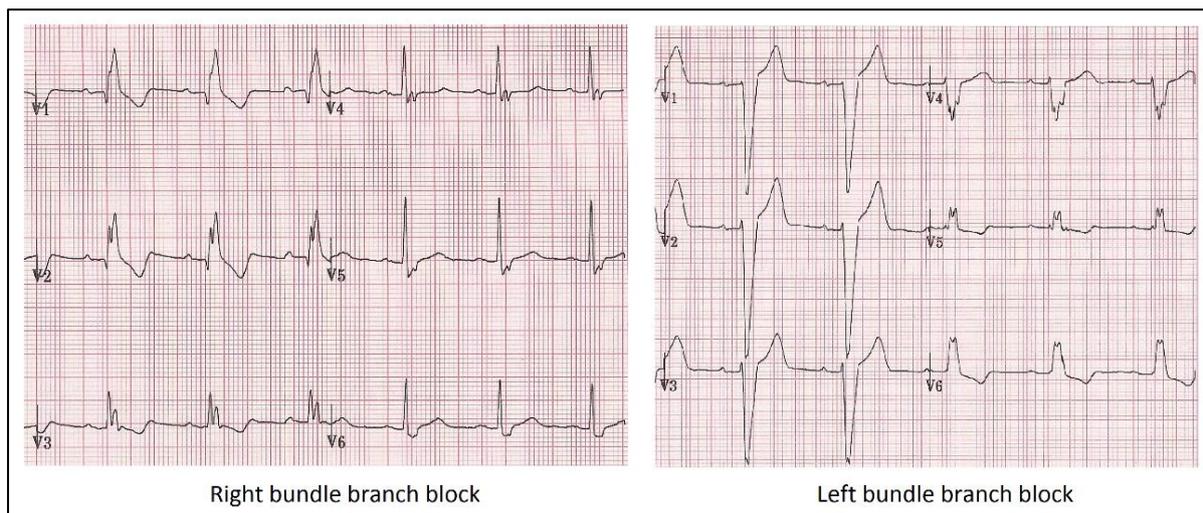
Figure 2. Characteristics changes seen in the chest leads when bundle branch block in present. In right bundle branch block, there is an RSR in V1/V2, and a wide S-wave in V6. In left bundle branch block, there are deep S-waves in V1-V3, and a stumpy, notched R-wave in V6.

Scanning each lead

These first four steps in our system will identify many of the most common, and important, abnormalities on the 12-lead ECG (Gregory, 2006). There are, however, many others that we have not discussed so far (Hampton, 2013b). So, how do we evaluate the ECG for these other abnormalities?

The answer is that we examine, or scan, each of the 12 leads of the ECG to evaluate whether the waveforms and segments appear normal (Gregory, 2006). This should be done in a methodical fashion, to ensure that every lead is looked at in turn (Aehlert, 2011).   Any abnormality detected should be evaluated in the light of the surrounding leads. In other words, we should ask whether this is an isolated finding (and therefore more likely to be a normal variant), or part of a larger pattern of abnormal findings that suggests an underlying disease process (Garcia, 2015). Houghton & Gray (2014) suggest that we should look for the following features

- P-waves that are too tall, or too wide
- Pathological Q-waves
- QRS complexes that are too small, or too large
- QRS complexes that have an abnormal shape
- ST-segments that are depressed, or elevated
- T-waves that have an abnormal shape, or are inverted

Although there are many conditions that can cause these types of change, among the most common are chamber enlargement and myocardial ischaemia or infarction (Hampton, 2013a). In this article we will discuss chamber enlargement, turning to ischaemia and infarction in the next article in the series. Let's start by discussing left ventricular hypertrophy, the most common type of chamber enlargement encountered in clinical practice (Hancock et al, 2009).

Left ventricular enlargement

The left ventricle (LV) is responsible for pumping blood into the systemic circulation (Klabunde, 2012). This requires considerable force. To move blood out of the heart, the left ventricle must push blood through the aortic valve, and overcome the diastolic pressure in the aorta, typically 80mmHg in a healthy individual (Marieb & Hoehn, 2015). In consequence, the LV has thick, muscular walls that can generate the necessary pressure (Tortora & Nielson, 2014).

If pressure within the aorta is increased, or there is an obstruction to blood flow, the LV must work harder, and its walls thicken to cope with the additional work load (Pappano & Wier, 2013). This process is referred to as hypertrophy (Kenney et al, 2015). LV hypertrophy (LVH) commonly occurs in response to systemic hypertension, but can also be due to aortic stenosis, or coarctation of the aorta (Houghton & Gray, 2014; Porthon et al, 2015). Hypertrophic cardiomyopathy (HCM) also causes LV hypertrophy, although the mechanism is proliferative disease rather than pressure overload (Elliot, 2010).

Enlargement of the LV without wall thickening is also possible if the chamber walls become stretched (Klabunde, 2012). This is referred to as dilatation, and may occur secondary to volume overload in conditions such as chronic kidney disease, or aortic regurgitation (Artham et al, 2009). Another common cause is dilated cardiomyopathy (DCM), in which case the primary problem is diseased heart muscle. DCM is often an inherited disorder, but can also be caused by ischaemic heart disease, alcohol abuse, and viral myocarditis (Jefferies & Towbin, 2010).

On the ECG, it is not possible to determine whether chamber enlargement is due to hypertrophy or dilatation (Garcia, 2015). Current clinical guidelines acknowledge this, but suggest using the term hypertrophy when describing ECG changes due to LV enlargement (Hancock et al, 2009). In this article, we will use the same convention.

Voltage criteria for LVH

The ECG changes that occur in LVH can be divided into two categories; voltage and non-voltage criteria (Edhouse et al, 2009). Voltage criteria refers to an increase in the size of the QRS complexes (Houghton & Gray, 2014). This occurs because the enlarged ventricle has more myocytes, and therefore produces more electricity (Garcia, 2015). So which QRS complexes are affected?

Broadly speaking, all of the QRS complexes may be affected, however the pattern seen reflects the orientation of the leads in relation to the LV (Hampton, 2013a). On the normal ECG, the LV dominates the shape of the QRS complex because of its larger mass (Sampson, 2016b). As a result, leads that face away from the LV (V1 and V2) have deep S-waves, while those facing the LV (I, aVL, V5 and V6) have tall R-waves (Garcia, 2015) (*figure 3*). In LVH, this pattern is exaggerated by the increased LV voltages (Hampton, 2013a). The S-waves in leads V1 and V2 are therefore deeper than normal, while the R-waves in leads I, aVL, V5 and V6 are unusually tall (*figure 4*).
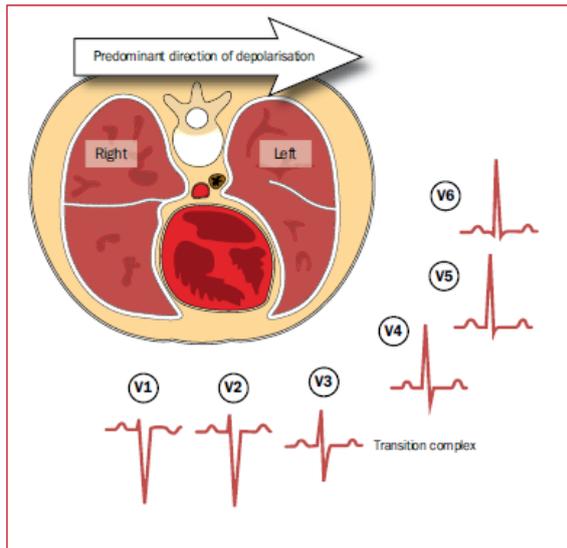
Figure 3. Leads facing the RV have deep S-waves, while those facing the LV have tall R-waves.
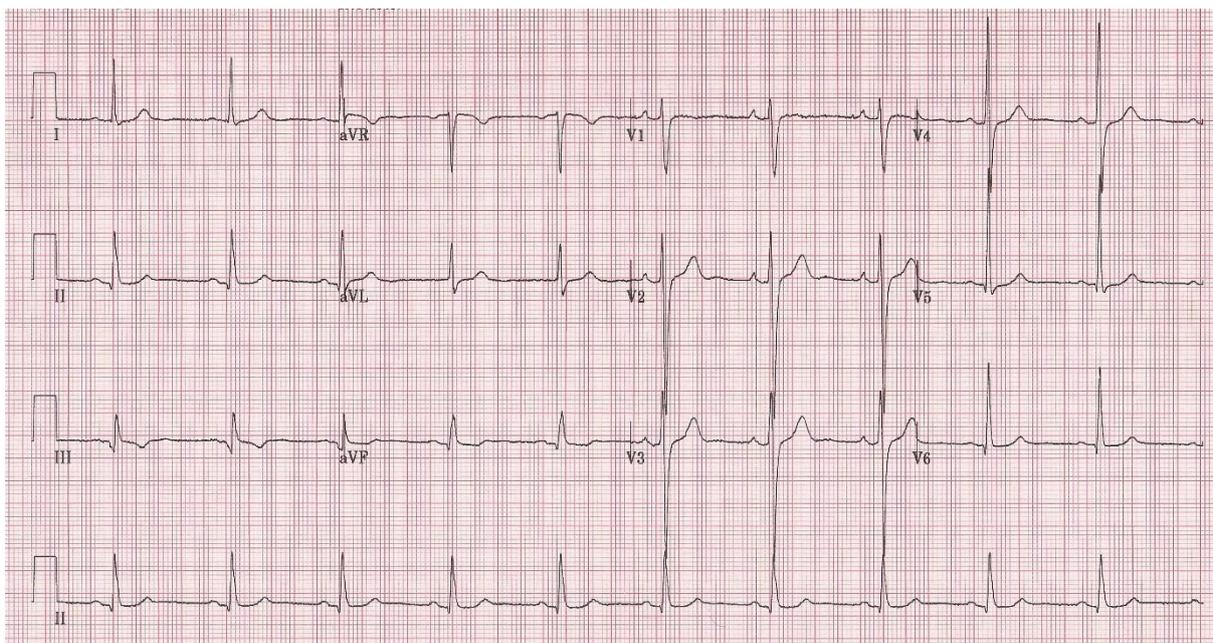
Figure 4. LVH. In this example, the R-waves are especially tall in leads I, V4 and V5. S-waves are abnormally deep in V2 and V3.

How much is too much?

You might be wondering how tall the R-waves need to be, or how deep the S-waves, in order to diagnose LVH. This is a very good question, that unfortunately does not have a simple answer. Various voltage criteria have been published, most of which are based on comparison of the ECG with LV mass measured at autopsy or by echocardiogram (Rautaharju & Soliman, 2014). Some of these criteria are quite simple, while others are more complex.

The simplest criterion is the evaluation of a single lead, for example an R-wave in aVL that is greater than 11mm (Garcia, 2015). In a study by Gosse et al (2012), this criterion was found to be accurate at predicting echocardiographic LVH, and has the advantage of being simple to assess on the ECG. The disadvantage of this single lead approach is demonstrated by *figure 4*. On this ECG, the R-wave in aVL is only 8mm tall, suggesting that LVH is not present. This seems unlikely, given the large QRS complexes in other leads.

If we use different criteria to evaluate this ECG, the outcome is very different. The Sokolow-Lyon and Cornell criteria are widely used in clinical practice, and use more than one lead in assessing the ECG for LVH (Sokolow & Lyon 1949; Casale et al 1985; Hancock et al, 2009) (*box 2*). According to both of these criteria, the ECG in *figure 4* is consistent with LVH.

Sokolow-Lyon	S-wave in V1 or V2 + R-wave in V5 or V6 ≥ 35mm (using the deepest and tallest leads)
Cornell	S-wave in V3 + R-wave in aVL > 28mm (men) S-wave in V3 + R-wave in aVL > 20mm (women)

Box 2. The Sokolow-Lyon and Cornell voltage criteria for LVH

This demonstrates a widely recognised limitation of the ECG diagnosis of LVH, namely that sensitivity of individual criteria is low, despite a high specificity (Hampton, 2013a; Hancock et al 2009; Rautaharju & Soliman 2014). In other words, in many cases the ECG will fail to detect LVH, although when it does the likelihood of a correct diagnosis is high. A study by Porthan et al (2015) found that using the Sokolow-Lyon and Cornell criteria together increased sensitivity, an approach that has also been suggested by clinical guidelines (Hancock et al, 2009). In short, the more criteria that you use, the more likely that LVH will be detected.

Further considerations when evaluating the ECG for LVH are age, athletic training, and lead placement. QRS voltages are often higher in young people, despite normal hearts. Voltage criteria for LVH are therefore inaccurate in individuals below the age of 36 years, and should not be used (Hancock et al, 2009).   Voltages are also increased in athletes, whose hearts are enlarged as a result of cardiovascular training (Corrado et al, 2010). Finally, lead placement must also be considered. In one study, voltages varied by as much as 25% due to daily variation in lead placement (Farb et al, 1990). Lead placement should therefore be considered if unexpected results are seen.
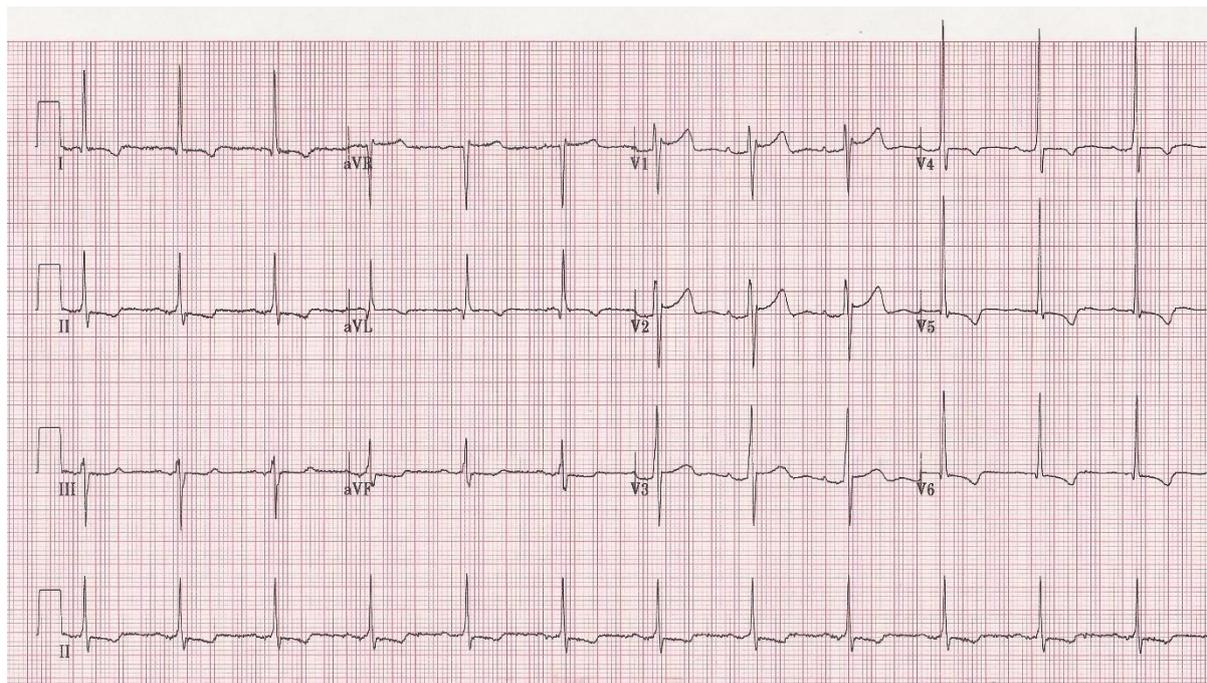
Non-voltage criteria for LVH

The increase in LV size that occurs in LVH does not merely increase the number of cells, and therefore the voltage of QRS complexes. A range of additional electrical effects is seen, including changes to depolarisation, repolarisation, and mean QRS axis (Edhouse et al, 2009). Left axis deviation is one non-voltage criterion for LVH (Garcia, 2015). The other important criteria are secondary ST-T abnormalities, and left atrial enlargement (Garcia, 2015; Hancock et al ,2009).

Secondary ST-T abnormalities (strain)

As the LV enlarges, progressive change may occur in the ST-segment and T-wave (Schocken, 2014). The ST-segment shifts downwards, and the T-wave flattens and ultimately inverts (Garcia, 2015). This pattern is commonly referred to as 'strain' from its early association with increased LV afterload (Hancock et al, 2009). We now know that a strain pattern is also seen in conditions that do not result from increased LV burden, such as HCM. Current guidelines therefore suggest that the term 'secondary ST-T abnormalities' is more appropriate (Hancock et al, 2009).

In LVH, secondary ST-T abnormalities are seen in those leads that face the LV, in particular leads I, aVL, V5 and V6 (Hampton, 2013a) (*figure 5*). Typically, the ST-segment is down-sloping and depressed, and the T-wave asymmetrically inverted (Hampton, 2013a). This appearance has been compared to a 'reverse tick', and is unlike the symmetrical T-wave inversion typically seen during myocardial ischaemia (Garcia, 2015) (*figure 6*). This is an important distinction to make in clinical practice. Changes to the ST-segments and T-waves are pivotal in the evaluation of patients with acute chest pain (Steg et al 2012). A failure to recognise LVH with strain can result in a misdiagnosis of myocardial ischaemia (Hampton, 2013a). In the case of patients with known LVH, secondary ST-T abnormalities can mask ischaemic changes, and make it more difficult to diagnose myocardial infarction (Edhouse et al, 2009). Evaluation of previous ECGs is essential in these cases.

Figure 5. LVH with strain. This ECG meets multiple voltage criteria for LVH (the patient was a 68-year-old woman). There are secondary ST-T abnormalities in all of the left sided leads.
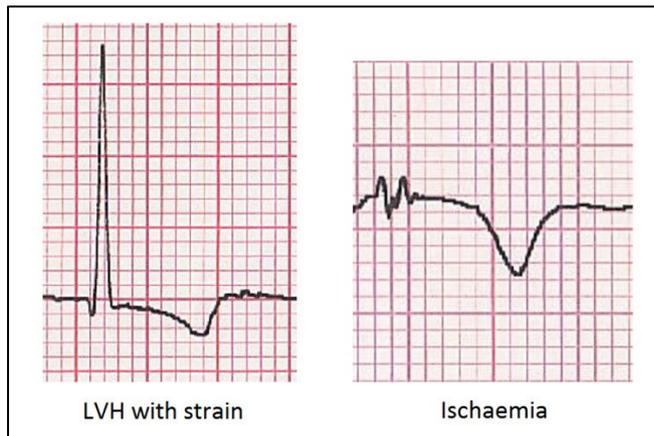
Figure 6. In typical LVH with strain there is down-sloping ST depression and asymmetrical T-wave inversion. T-wave inversion due to myocardial ischaemia is more often symmetrical.

From a prognostic perspective, ST-T abnormalities are associated with more advanced disease, and a greater risk of adverse cardiovascular events (Rautaharju and Soliman, 2014). In the PIUMA study, for example, patients whose ECG met the Sokolow-Lyon or Cornell criteria for LVH were found to have approximately twice the risk of adverse cardiovascular events compared to patients without LVH on the ECG (Verdecchia et al 1998). For those individuals with a strain pattern, the risk was nearer five-fold. In trials of anti-hypertensive drugs, a regression of the ECG markers of LVH has been demonstrated following control of blood pressure, with a corresponding fall in cardiovascular risk (Okin et al, 2004; Verdecchia et al 2003).

Left atrial enlargement

In the normal heart, the LV distends as blood fills it during diastole (Marieb and Hoehne, 2015). This stretching of the heart allows it to accommodate increased venous return during exercise, and stimulates contractility via the Frank-Starling mechanism (Klabunde, 2012). In LVH, the ventricle is less compliant, so stretches less readily (Artham et al, 2009). This stiffening of the chamber increases filling pressures, making it harder for the left atrium to eject blood into the ventricle. As a consequence, the left atrium enlarges (Douglas, 2003). A recent meta-analysis demonstrated a strong association between left atrial enlargement (LAE) and LVH (Cuspidi et al, 2013). LAE also occurs in mitral valve disease and LV dysfunction, and is associated with an increased risk of adverse cardiovascular outcomes including atrial fibrillation, stroke and death (Patel et al, 2009).

On the ECG, LAE causes widening of the P-wave and changes in waveform morphology (Bayes de Luna et al, 2012). The normal duration of the P-wave is less than 0.12s (Houghton and Gray, 2014). In LAE, this duration is extended to 0.12s or more (Garcia, 2015). The traditional explanation for this is that the wave of depolarisation has further to travel in an enlarged atrium (Edhouse et al, 2009). This view has been challenged by Bayes de Luna et al (2012) who argue that conduction delay or block within the atria may be responsible for the P-wave changes seen in LAE.

Changes in P-wave morphology are seen in the inferior leads, and lead V1 (Hampton, 2013b). The P-wave is produced by depolarisation of first the right, and then the left atrium. The two waveforms created are superimposed on each other to produce the single waveform that we call the P-wave

(Garcia, 2015). In LAE, delayed depolarisation of the left atrium causes a separation of these two waveforms, producing a bifid or 'M shaped' P-wave in the inferior leads (Hancock et al, 2009). A slightly notched P-wave may be a normal variant, so Edhouse et al (2009) suggest that a distance of one small square (40ms) between the two peaks of the P-wave is necessary to diagnose LAE with confidence (*figure 7*).
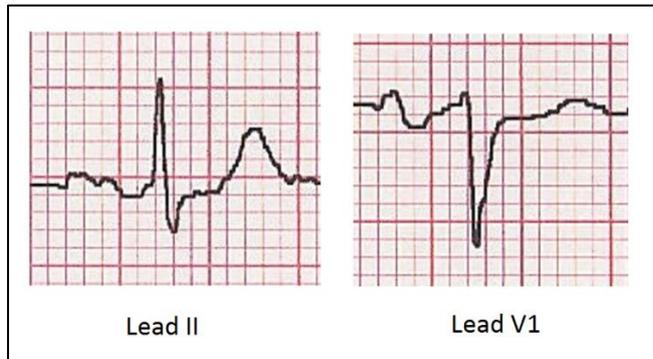
Figure 7. Left atrial abnormality. There is a bifid P-wave in lead II, with more than one small square separating the peaks. In lead V1, the negative component is greater than one small square wide and deep.

In lead V1, a similar separation in right- and left-atrial components occurs. The P-wave in lead V1 is often biphasic, with the first (positive) part representing right atrial depolarisation, and the terminal (negative) part representing activation of the left atrium (Hampton, 2013a). In LAE, the terminal portion of P-wave becomes wider and deeper than normal (Houghton and Gray, 2014). A width and depth of at least 1 small square has been suggested as the criteria for LAE (Garcia, 2015; Kitvungvan and Spodick, 2009).

In the past, the P-wave associated with LAE was referred to as 'P-mitrale' because of its association with mitral valve disease (Burns, 2011). Although widely known and understood, this name is not appropriate to our modern understanding of the ECG. Mitral valve disease is not the most common cause of LAE, and LAE is not the only cause of this P-wave appearance. Bayes de Luna et al (2012) suggest that atrial conduction block, in the absence of LAE, is a more common cause of P-wave widening and morphology change. This is supported by a study by Tsao et al (2008) that compared ECG and MRI findings in people with and without LAE. Only 35% of people with abnormal P-waves had LAE at MRI, suggesting that the ECG has poor specificity for LAE. In light of this, Kitvungvan and Spodick (2009) suggest that the ECG findings typically described as LAE should be called 'interatrial block' instead. Current practice guidelines acknowledge the place of electrical disease in P-wave abnormality, but recommend the term 'left atrial abnormality' as it encompasses both structural and electrical causes of ECG change (Hancock et al, 2009).

Combining voltage and non-voltage criteria

Although non-voltage criteria are often seen in LVH, the low specificity of left atrial and ST-T abnormalities means that they are not considered diagnostic without voltage changes (Hancock et al, 2009). Nonetheless, they are widely appreciated as additional diagnostic clues that help to

confirm a diagnosis (Garcia, 2015; Hampton, 2013a, Houghton and Gray, 2014). ST-T abnormalities also have a prognostic role, given their association with increasing cardiovascular risk (Schocken, 2014). Various diagnostic tools have been proposed that utilise non-voltage as well as voltage criteria, the best known of which is Romhilt-Estes scoring system (Romhilt-Estes, 1968) (*box 3*). Despite its increased complexity, a recent study using MRI to validate ECG criteria suggests that its sensitivity is no better than simpler criteria such as Sokolow-Lyon or Cornell, with a similar specificity (Jain et al, 2010).

Criteria	Points
R or S in limb leads ≥ 20mm **OR** S in V1 or V2 ≥ 30mm **OR** R in V5 or V5 ≥ 30mm	3
ST-T abnormalities without digitalis	3
ST-T abnormalities with digitalis	1
Negative component of P-wave in V1 at least 1mm in depth and 40ms in duration	3
Left axis deviation	2
QRS duration ≥ 90ms	1
Delayed intrinsicoid deflection in V5 or V6 > 50ms	1

Box 3. The Romhilt-Estes scoring system for LVH. A score of four indicates that LVH is probable, a score of 5 or more is diagnostic for LVH.

Right ventricular enlargement

Right ventricular (RV) enlargement results from pressure or volume overload in much the same way as LV enlargement (Haddad et al, 2008). The principal cause is pulmonary hypertension, although it also occurs in pulmonary valve stenosis, and pulmonary embolism (Hampton, 2013a).   On the ECG, it is usually referred to as right ventricular hypertrophy (RVH) (Hancock et al, 2009). RVH is much less common than LVH, and its ECG features have been less well studied (Whitman et al, 2014). Both voltage and non-voltage criteria have been identified, however all are limited by poor sensitivity and specificity (Harrigan and Jones, 2009).

The principal voltage criteria in RVH is a dominant R-wave in lead V1 (Houghton and Gray, 2014). By dominant, we mean that the R-wave is greater than the S-wave (Hampton, 2013a). This can also be expressed as an R:S ratio of greater than 1 (Garcia, 2015). This is an unusual finding on the ECG, as lead V1 commonly has a small R-wave, and a deep S-wave.

Lead V1 sits over the RV (Houghton and Gray, 2014). In the normal heart, left to right depolarisation of the septum moves towards this lead, creating a small R-wave. The subsequent depolarisation of the ventricles is dominated by the thicker-walled LV, so the predominant vector moves away from lead V1, causing a deep S-wave (Hampton, 2013a). In severe RVH, the RV mass may exceed that of the LV, shifting the predominant vector of depolarisation to the right and towards lead V1 (Hancock et al, 2009). This creates a dominant R-wave in the right sided precordial leads. Because the amount of hypertrophy needed to alter the predominant vector is large, many people with mild RVH will have a normal ECG, and hence sensitivity of the ECG for RVH is low (Whitman et al, 2014).

In addition to poor sensitivity, specificity is also an issue with this ECG finding. A tall R-wave in lead V1 is a normal variant, and is also seen in other conditions including posterior myocardial infarction, pre-excitation, and right bundle branch block (Hampton, 2013a; Houghton and Gray, 2014). Additional ECG features must therefore be sought when making a diagnosis of RVH. These include:

- QRS duration less than 0.12s (to exclude right bundle branch block)
- Right axis deviation ≥ 110 degrees
- Deep S-waves in leads V5 and V6
- Secondary ST-T abnormalities (strain)
- Right atrial abnormality

(Harrigan and Jones, 2009; Hampton, 2013b)

The secondary ST-T abnormalities that develop in RVH are similar to those in LVH in that the typical pattern is down-sloping ST depression, with asymmetrical T-wave inversion (Hancock et al, 2009). In RVH, however, the strain pattern is seen in the right sided precordial leads (V1 to V3), and often the inferior leads (Hampton, 2013b). This is because these are the leads overlying the hypertrophied RV (Garcia, 2015). *Figure 8* illustrates many of the key features of RVH.
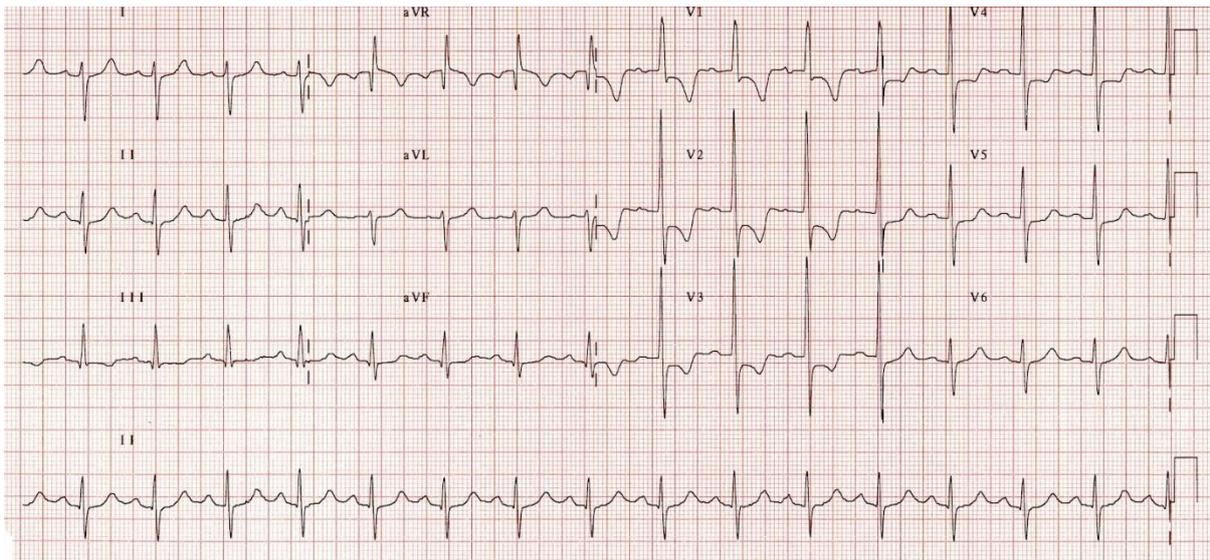
Figure 8. RVH with strain. Note the dominant R-waves in V1-3, and the deep S-waves in V4-6. There are secondary ST-T abnormalities in leads V1-4, and right axis deviation.

Right atrial abnormality

Right atrial abnormality refers to changes in the size and shape of the P-wave that occur in diseases affecting the right side of the heart (Houghton and Gray, 2014). It is commonly associated with RVH, and often reflects enlargement of the right atrium due to increased filling pressures in the RV (Haddad et al, 2008). This may occur acutely in pulmonary embolism, or over a longer period in chronic disease such as pulmonary hypertension and chronic obstructive pulmonary disease (Hampton, 2013a). Other causes of right atrial enlargement include congenital heart disease, and tricuspid stenosis, although these are less common (Harrigan and Jones, 2009).

The characteristic finding in right atrial abnormality is a tall, peaked P-wave in the anterior and inferior leads (Harrigan and Jones, 2009) (*figure 9*). In the inferior leads (II, III and aVF), the height of the P-wave is greater than or equal to 2.5mm (Garcia, 2015). P-wave duration is usually normal unless there is congenital heart disease (Hancock et al, 2009). In older texts, the term 'P-pulmonale' is often used, reflecting the association between right atrial enlargement and chronic lung disease (Hampton, 2013a). As with left atrial disease, the preferred terminology today is right atrial abnormality, reflecting the fact that both electrical and structural factors can influence P-wave morphology (Hancock et al, 2009).

Figure 9. Right atrial abnormality in lead II. The P-wave is tall (almost 3mm) and peaked. Note the ST-T abnormality, which also suggests RVH.

Biventricular enlargement

In advanced heart disease, both ventricles may be enlarged (Klabunde, 2012). Because voltages on both sides of the heart will be increased, these changes may cancel each other out to a certain extent (Garcia, 2015). The ECG therefore has particularly poor sensitivity and specificity in this situation (Hancock et al, 2009). The ECG features that might be seen are voltage criteria for LVH plus features of RVH such as deep S-waves in V5 and V6, right axis deviation, and right atrial abnormality (Hancock et al, 2009).

The same principles apply to bi-atrial enlargement, in which case features of both left and right atrial abnormality may be present. In the inferior leads, for example, the P-wave is wide and broadly bifid, but also has a peaked initial component that is at least 2.5mm high (Garcia, 2015).

Conclusion

Enlargement of all four chambers of the heart may be reflected in the 12-lead ECG (Garcia, 2015). LVH is more common, and in consequence has been more widely studied and defined (Rautaharju and Soliman 2014). Numerous voltage criteria for LVH have been proposed, although they are only diagnostic in adults over 35-years old who are non-athletes (Hancock et al, 2009). In this population, all of the published criteria are highly specific but lack sensitivity, suggesting a limited role for the ECG in population screening (Hampton, 2013a). Of the available criteria, the Sokolow-Lyon and Cornell are widely used, and in combination may increase sensitivity (Hancock et al, 2009). They should be combined with a search for non-voltage criteria such as left axis deviation, secondary ST-T abnormalities, and left atrial abnormality (Houghton and Gray, 2014).

RVH is less common, and does not affect the ECG unless disease is severe (Harrigan and Jones, 2009). In consequence, sensitivity of the ECG for RVH is poor (Hampton, 2013a). The principal sign of RVH, a dominant R-wave in lead V1, has poor specificity because of multiple differentials. A diagnosis of RVH must therefore be supported by associated changes such as deep S-waves in leads V5 and V6, right axis deviation, ST-T abnormalities, and right atrial abnormality. The QRS must be narrow as a wide QRS suggests that ECG changes are due to right bundle branch block.

Next month we turn our attention to the signs of myocardial ischaemia and infarction, examining the ECG changes that take place, and placing them in the context of coronary artery anatomy and the pathophysiology of coronary heart disease.

Key points

- Many important cardiovascular and respiratory diseases cause enlargement of the cardiac chambers. This enlargement can often be detected on the 12-lead ECG. Although the specificity of some ECG features is high, sensitivity is generally low, reducing the usefulness of the ECG as a screening tool.

- LVH is the most common and well defined problem. LVH causes an increase in QRS voltages across the precordium, with deep S-waves in leads V1 and V2, and tall R-waves in leads I, aVL, V5 and V6. This may be accompanied by non-voltage criteria such as left axis deviation, secondary ST-T abnormalities, and changes in the duration and morphology of the P-wave.

- Various ECG criteria for LVH have been proposed, based on QRS voltage alone, or in combination with non-voltage criteria. All have high specificity, but low sensitivity. Current guidelines suggest using more than one voltage criteria, as well as evaluating for associated non-voltage features. The Lyon-Sokolow and Cornell criteria are commonly used, and are as sensitive and specific as more complex criteria.

- Left atrial abnormality describes a prolonged P-wave with a widely bifid appearance in the inferior leads, and a broad terminal component in lead V1. It is also known as P-mitrale. Left atrial abnormality is commonly associated with LVH, and is often due to left atrial enlargement. It also occurs as a result of electrical disease, so has poor specificity.

- RVH is much less common, and is usually severe before it causes significant ECG changes. The principal ECG finding is a prominent R-wave in lead V1, however this must be supported by other features due to low specificity. Other findings in RVH include right axis deviation, secondary ST-T abnormalities and right atrial abnormality.

- Right atrial abnormality, also known as P-pulmonale, is often caused by right atrial enlargement and may be seen in association with RVH. It can be recognised by a tall, peaked P-wave in the inferior leads.

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