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Anatomy of the Heart

The Importance of Imaging Techniques Correlations

The surface electrocardiogram (ECG) in both the acute and chronic phase of ischemic heart disease (IHD) may give crucial information about the coronary artery involved and the area-at-risk for myocardial injury. Knowing the anatomy of the heart, especially the ventricular walls and coronary tree, helps to understand the various ECG patterns present in IHD. However, the electric activity of the heart is influenced by numerous cardiac and non-cardiac factors. Structural (chamber size, hypertrophy, presence of diffuse fibrosis, or localized scar, presence of pericardial effusion, etc.), functional and metabolic (ischemia, post-ischemic stage, inflammation) aspects and diseases of the conduction system (CS) can all modify the ECG. Non-cardiac factors include the position of the heart in the chest (horizontal position in obese patients, vertical in patients with emphysema, altered anatomy after pulmonary surgery, atelectasis, or pneumothorax), the distance of the electrodes from the heart and the width of the chest wall (effusion, adipose tissue, radical mastectomy, etc.), electrolyte imbalance and drug effects. This information together with the ECG-clinical correlation is very important for diagnosis and risk stratification, as will be demonstrated in this book.

For centuries, since the pioneering works of Vesalio, Leonardo da Vinci, Lower, and Bourgery-Jacob, pathology has been a unique method to study the anatomy of the heart. Since the end of the nineteenth century, the visualization of the heart *in vivo* has been possible by **X-ray examination**. The last 40–50 years started the era of invasive imaging techniques with **cardiac catheterization,** including **coronary angiography,** and non-invasive imaging techniques, first with **echocardiography** and later with **radionuclide studies**, **computed tomography** (CT) **and cardiovascular magnetic resonance** (CMR) imaging. These techniques have opened up new horizons to study, not only the anatomy of the heart, coronary arteries and great vessels, but also myocardial function, metabolism and perfusion, and the characterization of the valves, pericardium, etc.

From an ECG standpoint, **coronary angiography** (Figure 1.1) is especially important in the acute phase for diagnosing the disease and correlating the site of occlusion with the ST-segment deviations. It is also useful in the chronic phase of the disease. However, in the chronic phase of Q-wave myocardial infarction (MI) the ECG does not usually predict the state of the coronary tree, because revascularization has often modified the characteristics of the occlusion responsible for the MI (Basso and Thiene 2006). Moreover, coronary angiography is insensitive for detecting minor collaterals that could attenuate ischemia. Coronary angiography does not give information about the myocardium and, thus, cannot directly assess the extent and severity of ischemia, especially in the acute phase of acute coronary syndromes (ACSs).

In patients with non- ST-elevation ACS, the culprit lesion is not always identifiable

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by angiography, especially in patients without active ischemia during the time of angiography or with multi-vessel disease. Cine angiography of the left ventricle (LV) may give information for identifying hypokinetic or akinetic areas, but in practice, echocardiography is the preferred technique for this purpose. After the acute phase of myocardial ischemia, hypo-/akinetic myocardial segments usually corelate with the extent of tissue injury (Shen, Tribouilloy, and Lesbre 1991a,b; Takatsu et al. 1988; Takatsu, Osugui, and Nagaya 1986; Warner et al. 1986).

Computed coronary tomography angiography (CTA) is an alternative to invasive angiography to explore the coronary artery anatomy, especially in situations with low likelihood of need for percutaneous coronary

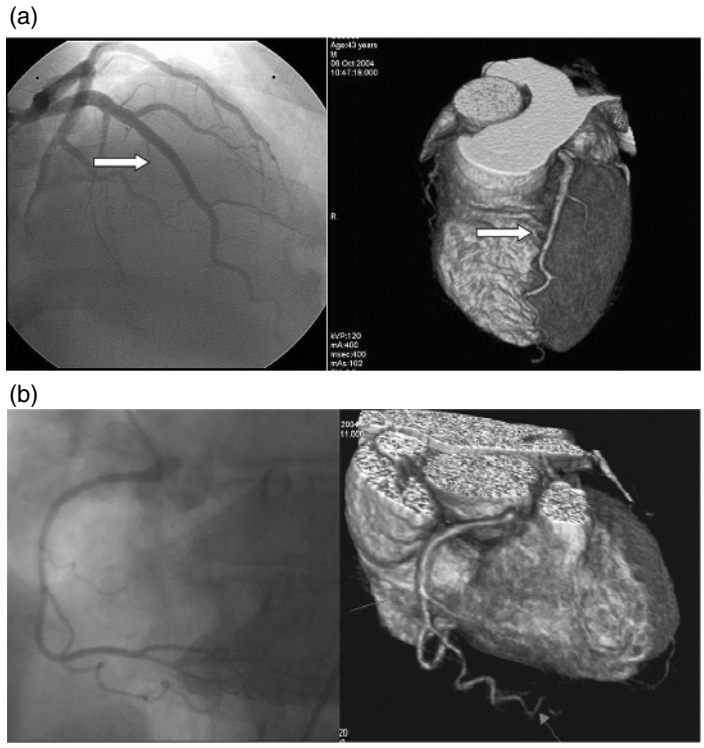
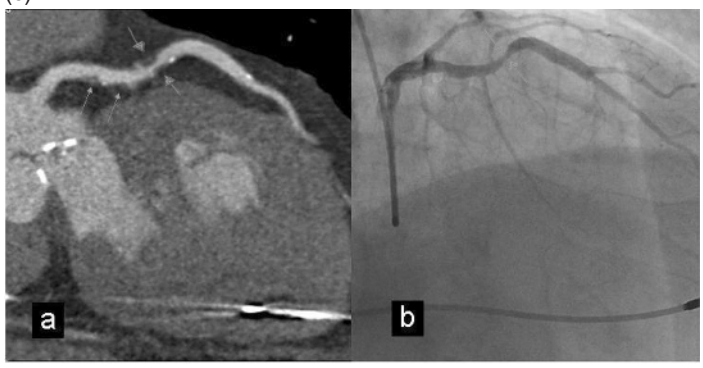


Figure 1.1 (A) Normal case: coronary angiography (left) and three-dimensional CTA (right) showing normal left anterior descending (LAD, arrow) and left circumflex (LCX) coronary arteries. The latter is partially covered by the left appendix in CTA. (B) Normal case: coronary angiography (left) and CTA (right) showing a normal dominant right coronary artery (RCA). (C) An 85-year-old man with atypical chest pain: (a) Maximal intensity projection (MIP) of CTA with tight mid-LAD stenosis that correlates perfectly with coronary angiography (b). (D) Similar case as (C) but with the stenosis in the proximal RCA. (a–d) CTA and (e) coronary angiography. (E) A patient with tight stenosis in the LCX before a bifurcation. (a) and (b) CTA and (c) coronary angiography. (F) These images show that CTA may also demonstrate the presence of stenosis in distal vessel branches, in this case in the posterior descending branch of the RCA. (a–b) CTA and (c) coronary angiography. (G) These images show that CTA (a, b) may delineate the length of a total occlusion and visualize the distal branches (see arrows in (b)). Collateral flow from the LAD to the RCA may be better visualized with CTA than with conventional coronary angiography (c: here only the RCA is shown). (H) A 42-year-old patient with a stent implanted in the LAD six months before. The patient complained of atypical chest pain and underwent CTA. The MIP images of CTA (a–c) show no significant restenosis, but some plaque formation in the left main trunk (d, circle) that was not well seen in coronary angiography (e). The degree of luminal obstruction by the plaque can be exactly measured by intravascular ultrasound (IVUS) (f). The ECG showed mildly inverted T waves in V1–V3 during follow-up (see Plate 1 in color plates).

(c)



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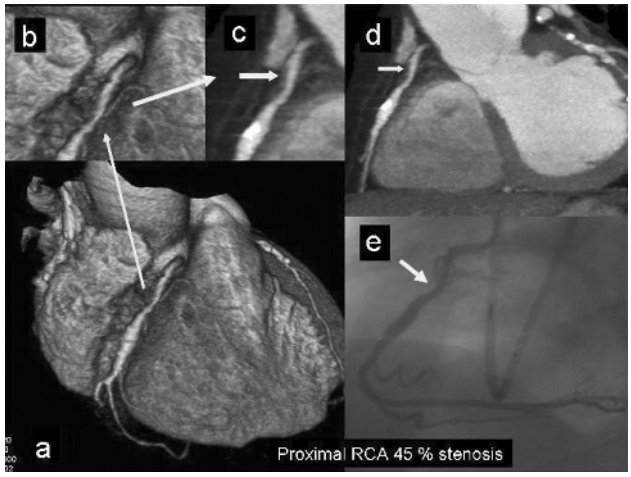

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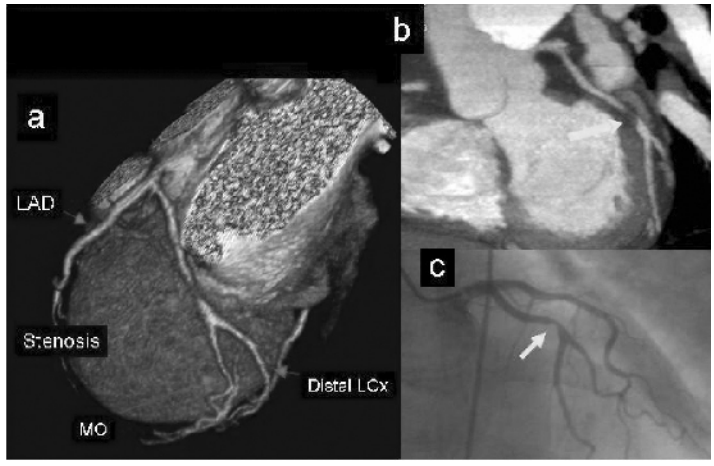

Figure 1.1 (Continued)

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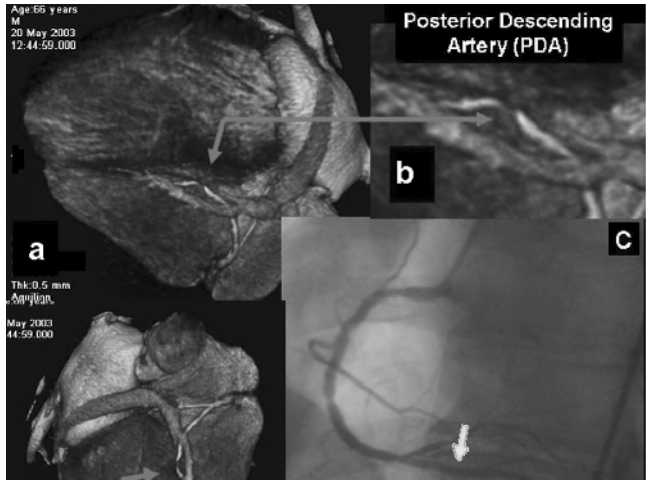

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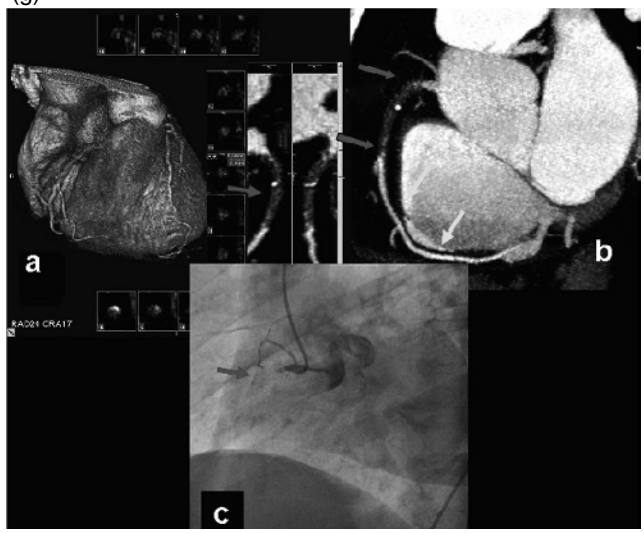

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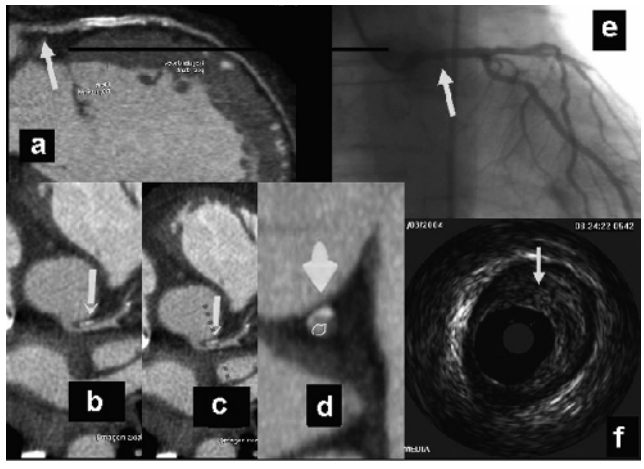
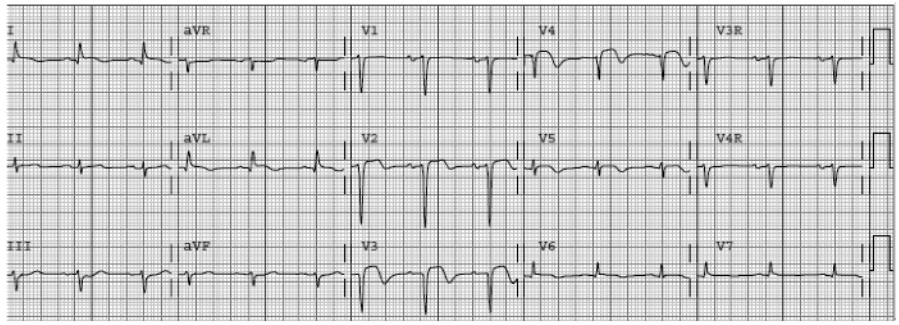


Figure 1.1 (Continued)

intervention (PCI) (Figure 1.1). CTA may provide additional diagnostic information for clinical decision-making in contemporary stable chest pain patients with intermediate pre-test probability for IHD (Hoffmann et al. 2017). In additional to narrowing, CTA can provide information on coronary distribution, including abnormalities in the origin and course of the arteries, and presence of coronary aneurysms. However, the accuracy of assessing the severity of coronary stenosis in highly calcified lesions and within stents is reduced.

The era of modern non-invasive cardiac imaging started with **echocardiography**, which is very convenient for out-patient and bedside evaluation. Echocardiography plays an important role, especially in the acute phase, for evaluation of LV function and mechanical complications of acute MI (Figures 1.2, 11.2, and 11.3). The method is also of clear value in chronic IHD patients for the study of LV function; both global and regional wall motion abnormalities can easily be detected (Bogaty et al. 2002; Matetzky et al. 1999; Mitamura et al. 1981). Both the non-ST-elevation acute coronary syndrome (NSTE-ACS) and ST-elevation MI (STE-ACS) guidelines highlight the importance of transthoracic echocardiography in the diagnosis, differential diagnosis and risk stratification in ACS patients (Roffi et al. 2016; Steg et al. 2012). Visualization of a regional decrease in systolic movement of the inner layers of the LV and a decrease in myocardial thickening are the main criteria for the diagnosis of myocardial ischemia (Figure 1.2) (Leischik et al. 2016). Yet, echocardiography cannot always differentiate between an old infarction and acute ischemia. Echocardiography may aid in decision-making in the emergency room when the ECG findings are inconclusive, since regional wall-motion abnormalities occur within minutes following coronary occlusion, well before necrosis. However, lack of regional wall motion abnormalities cannot rule out acute ischemia, especially in patients with left ventricular hypertrophy (Neuman et al. 2004). Growing evidence of the use of tissue Doppler and strain imaging in acute myocardial ischemia is emerging, but the methods are not yet ready for large-scale routine use in the diagnostic workup of patients with suspicion of ACS (Smiseth et al. 2016). However, echocardiography tends to overestimate the area-at-risk, and thus its reliability is good but not excellent. Stress echocardiography and especially nuclear imaging (**single-photon emission computed tomography**, SPECT) have been extensively used for the detection of perfusion abnormalities during a stress test. Also, nuclear imaging at rest has been used for detecting perfusion defects in patients presenting with ACS, especially for the estimation of the ischemic area-at-risk in patients with STE-ACS (Leischik et al. 2016; Gallik et al. 1995; Huey et al. 1988; Zafrir et al. 2004) (Figure 1.3). These methods are useful in cases with dubious precordial pain with a positive exercise test without symptoms. However, in some cases (non-transmural infarction) the extension of the infarction may be underestimated while in patients with left bundle branch block (LBBB), there may be false-positive results.

The most recent imaging techniques are **CMR** imaging (Figure 1.4) and the already mentioned **CTA** (Figure 1.1). CMR, which may also be used for perfusion and function studies of the myocardium, gives the best "*in vivo*" anatomic information about the heart. Thus, this technique, especially with gadolinium injection – contrast-enhanced CMR (CE-CMR) – is very useful for identifying and locating MIs, as well as for determining transmurality comparable to pathological studies (Bayés de Luna et al. 2006a; Cino et al. 2006; Moon et al. 2004; Salvanayegam et al. 2004; Wu et al. 2001). This is the reason that CE-CMR has become the gold standard technique for studying correlations between ECG findings and infarcted myocardial areas in the chronic phase of coronary artery disease (CAD) (Bayés de Luna et al. 2006a; Cino et al. 2006; Engblom et al. 2002, 2003). Also according to the location of hyperenhancement areas in CE-CMR, the technique can distinguish between ischemic and non-ischemic etiologies (Figure 1.5). The sequence of evolving transmural MI can be studied (Mahrholdt et al. 2005a,b). In addition, CMR can reveal



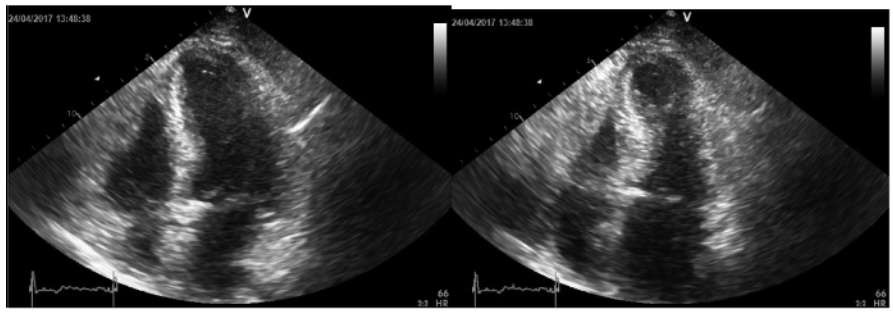


Figure 1.2 ECG-echocardiography correlation. Post-reperfusion ECG of an 82-year-old patient with LAD occlusion. The ECG shows anteroapical STE-ACS. There is ST-segment elevation in I and aVL: although the occlusion was in the mid-LAD, there was one or more diagonal branches distal to the occlusion (Eskola et al. 2007) End-diastolic (left) and end-systolic (right) apical long-axis views show hypokinesia of the apex and compensatory hyperkinesia of the basal septum and lateral wall. Strain rate imaging (bull's eye) clearly delineates the region with apical dyskinesia (see Plate 2 in color plates).

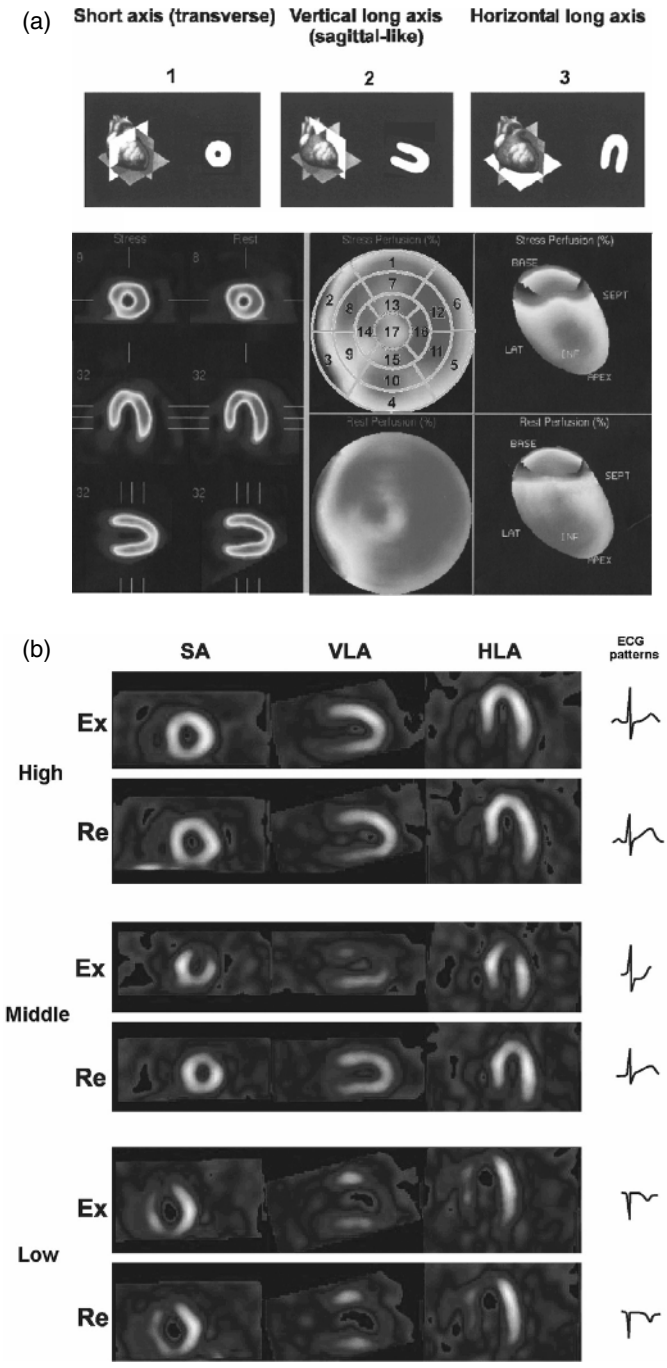


Figure 1.3 Examples of exercise test – nuclear imaging (SPECT) correlation. (a) Above: Observe the three heart planes with their transections (see Figure 1.4b) used by nuclear medicine experts (and other imaging techniques) to transect the heart: (1) short-axis (transverse) view (SA), (2) vertical long-axis view (VLA) (oblique sagittal-like), and (3) horizontal long-axis (HLA) view. The short-axis transections are at the mid-apical level (see Figure 1.8). The segmentation of the heart used in this book is shown (Cerqueira, Weissman, and Disizian 2002). In the middle is also a bull's eye image presented. This patient has normal left ventricular perfusion. (B) Above: In the three planes (SA, VLA, and HLA): (A) normal uptake at rest (Re) and during exercise (Ex) can be observed. Middle: Abnormal uptake only during exercise of segments 7, 13, and 17 (see Figure 1.8) in a patient with ischemia produced by distal involvement of a non-wrapping LAD. The basal part of the anterior wall of the LV is not involved. Below: abnormal uptake during rest and exercise in a patient in the chronic post-MI phase caused by distal occlusion of very long LAD that wraps the apex involving part of the inferior wall (segments 7, 13, and 17 and also 15) (see Figure 1.8). In this case abnormal uptake is also evident in the rest image (see plate 3 in color plates).

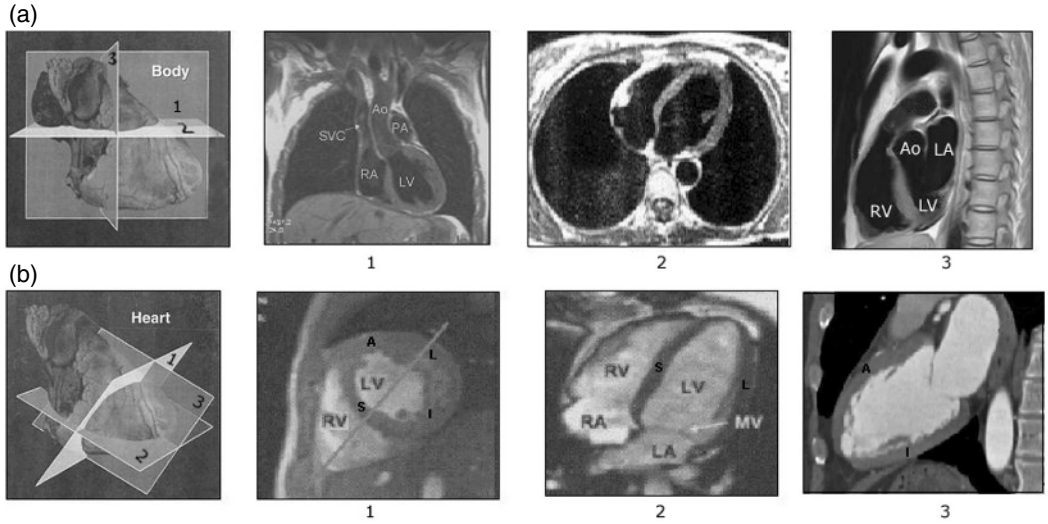


Figure 1.4 CMR imaging. (a) Transections of the heart following the classical human body planes: (1) frontal plane, (2) horizontal plane, and (3) sagittal plane. (b) Transections of the heart following the heart planes that cut the body obliquely. These are the planes used by the cardiac imaging experts: (1) short-axis (transverse) view, in this case at mid-level (see b (1)); (2) horizontal long-axis view; (3) vertical long-axis view (oblique sagittal-like). Check the great difference between the sagittal plane according to human body planes (a (3)) and the heart planes (b (3)). (b) shows the four walls of the heart with the classical names: septal (S), anterior (A), lateral (L) and inferoposterior. Currently, the inferoposterior wall is for consistency named inferior (I) (see Figure 1.8).

inflammatory processes (Saeed et al. 2017). Apart from being the most accurate modality for the assessment of old infarct scars, cardiac CMR has a central role in the setting of STE-ACS. T2-weighted imaging can depict zones with edema 2–7 days after reperfusion. Edema is considered as the equivalent of the ischemic area-at-risk in acute total coronary occlusion. Thus, CMR is the first imaging technique to show the ischemic area-at-risk even after reperfusion has occurred (Mangion 2016). Thus, for the first time we can correlate ECG changes during the acute phase of STE-ACS with the extent and location, and maybe even the severity, of myocardial ischemia. Nevertheless, there is no published data to correlate ECG and CMR findings from the acute phase of STE-ACS in large patient populations and with different locations of culprit lesions. When assessing CMR findings in CAD, one has to take into consideration the variability in coronary artery anatomy (Rinta-Kiikka et al. 2014). CMR can demonstrate post-reperfusion microvascular obstruction (no-reflow) and myocardial hemorrhage and using late gadolinium sequences for assessing the extent of infarction and the edema score for the size of the area at risk, salvage index can be reliably assessed (Mangion et al. 2016). The reproducibility of CE-CMR with time, especially after the acute phase, is very good. It also has the advantage of not producing radiation. The current limitation of CMR, which will probably be solved in the next few years, is the detailed study of the coronary tree. Currently, this may be performed non-invasively by CTA (see above Figure 1.1).

1.1 The walls of the Heart and their Segmentation as Assessed by CMR (Figures 1.4–1.13)

In the past, different imaging modalities used different terms to describe the various segments of the LV (see Figures 1.4–1.13).

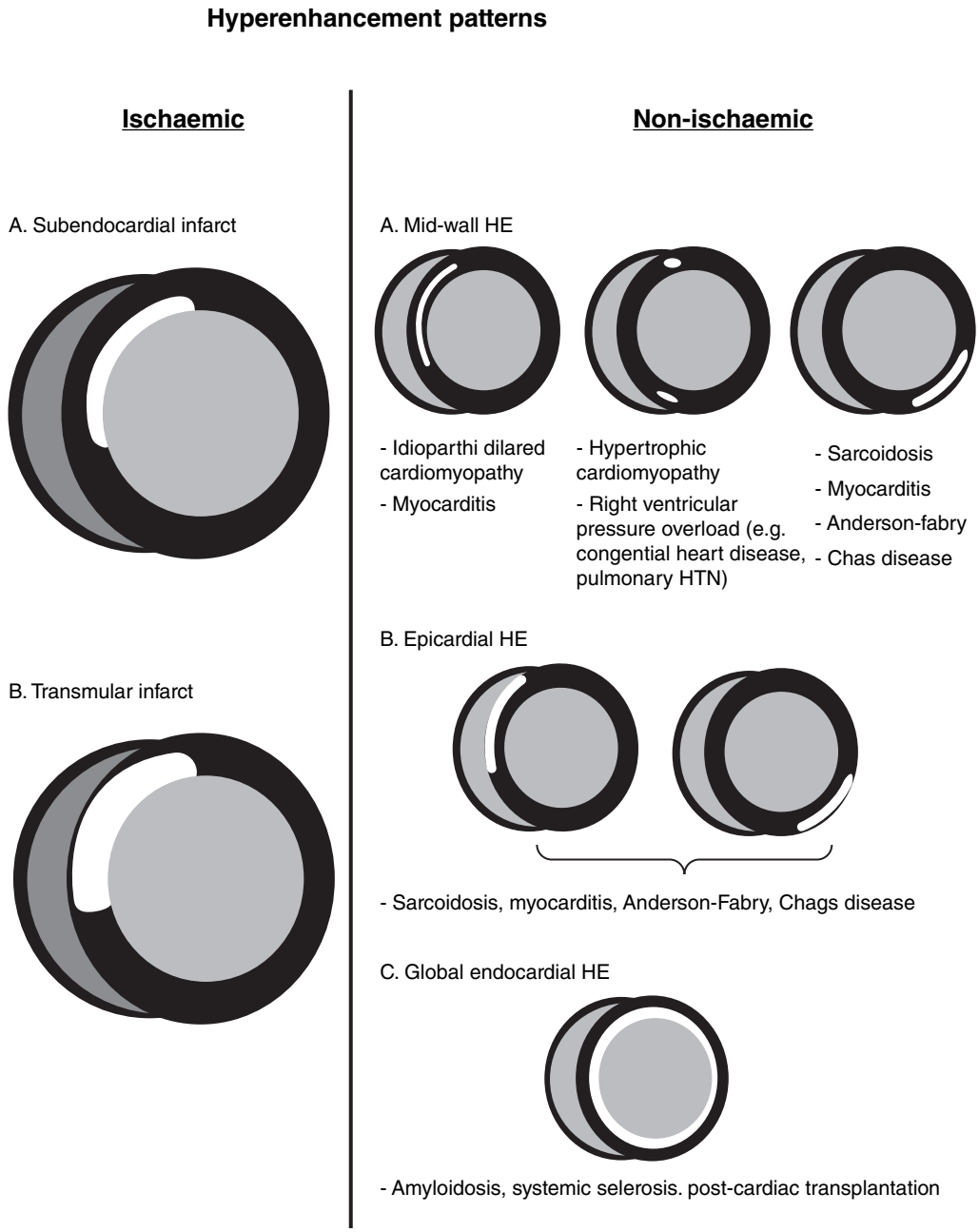


Figure 1.5 CMR hyperenhancement patterns found in clinical practice. If hyperenhancement is present, the subendocardium should be involved in patients with ischemic disease. Isolated mid-wall or subepicardial hyperenhancement strongly suggests a "non-ischemic" etiology. *Source:* From Marhrholdt 2005a.

It led to confusion when the different modalities were compared. The heart is located in the central-left part of the thorax (lying on the diaphragm) and is oriented anteriorly, with the apex directed forwards (Figure 1.4).

The LV is cone shaped. Although its borders are imprecise, classically (Myers et al.

1948a,b; Myers, Howard, and Stofer 1948c), it has been divided, except in its most inferior part of the apex, into four walls, till very recently named septal, anterior, lateral, and inferoposterior. In the 1940s–1950s the inferoposterior wall was named just posterior (by Goldberger) (Figure 1.6a), probably because it was considered opposed to the anterior wall. Later on (Perloff 1964), only the basal part of this wall, which was thought to bend upwards, was considered to be the posterior wall (Figure 1.6b). Therefore, it was named "true posterior" and the rest of the wall as "inferior" (Figure 1.6). According to that, for more than 40 years the terms "true" or "strict posterior infarction," "-injury" and "-ischemia" have been applied, when it was considered that the basal part of the inferoposterior wall was affected. The committee of the experts of the International Society of Computerized ECG (Macfarlane and Veitch Lawrie 1989), in accordance with the publications of Selvester and Wagner, named these walls anterosuperior, anterolateral, posterolateral, and inferior, respectively. However, this nomenclature has not been popularized, and the classical names (Figure 1.7a) are still mostly used in the majority of papers (Roberts and Gardin 1978), ECG books (Figure 1.7b–d), task forces (Surawicz et al. 1978) and statements (Hazinsky, Cummis, and Field 2000).

Later on, in the era of imaging techniques, the heart was transected into different planes (Figure 1.7) and different names were given to the walls of the heart by echocardiographers and experts in nuclear medicine. However, the consensus of the North American Societies for Imaging (Cerqueira, Weissman, and Disizian 2002) divided the LV into 17 segments and **4 walls**: **septal**, **anterior**, **lateral,** and **inferior** (Figures 1.8 and 1.9). Therefore, the word "posterior" has to be omitted. Figures 1.8 and 1.9 show the 17 segments into which the four LV walls are divided (6 basal, 6 medial, 4 inferior, and the apex), and the right side of Figure 1.9 shows the walls of the heart with their corresponding segments on a polar "bull's eye" map, as used by specialists in nuclear medicine. In the rest of the book we will use this current terminology.

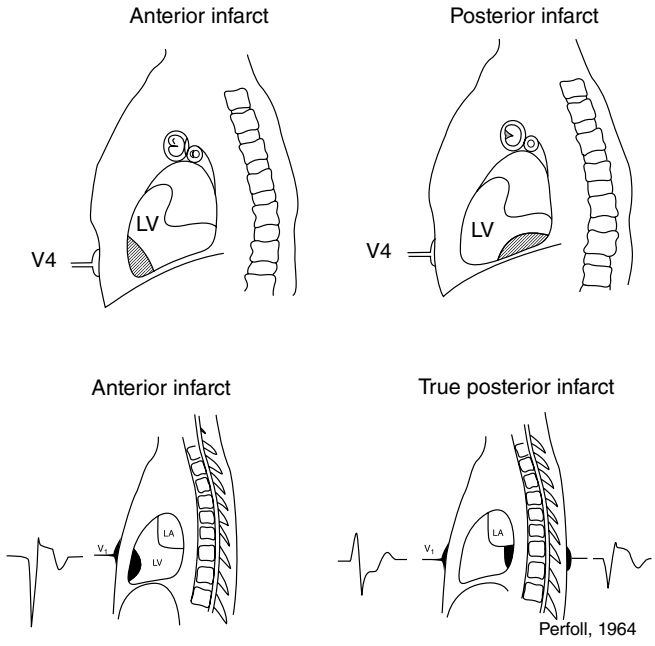


Figure 1.6 Above: The concept of anterior and posterior infarction according to Goldberger. Below: The concept of anterior and true or strict posterior infarction according to Perloff (1964). The other part of the wall that lies on the diaphragm was later named inferior.

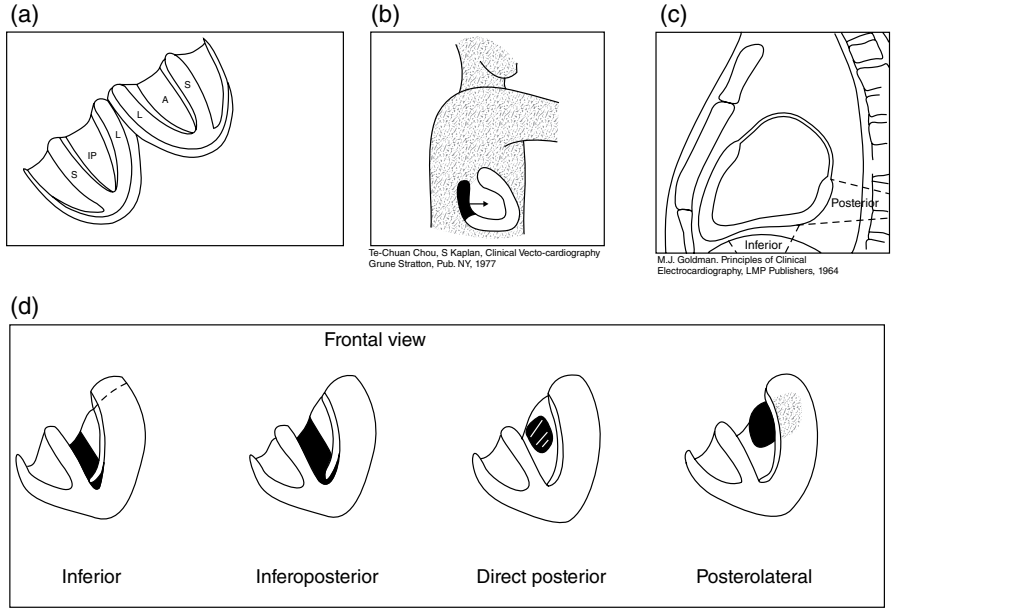


Figure 1.7 (a) The left ventricle may be divided into four walls that till very recently were usually named anterior (a), inferoposterior (IP) or diaphragmatic, septal (S) and lateral (L). However, according to the arguments given in this book, we consider that the "inferoposterior" wall has to be named just "inferior." (b–d) Different drawings of the inferoposterior wall (inferior + posterior walls) according to different ECG textbooks (see inside the figure). In all of them the posterior wall corresponds to the basal part of the wall lying on the diaphragm that was thought to bend upwards. It was considered that the heart was located strictly in an anteroposterior position in the thorax (Figures 1.11a). CMR gives us the information that the inferoposterior wall lies flat, even in its basal part, in around two-third of cases (Figure 1.12), which means that the heart is always located in an oblique position (Figure 1.11b,c). *Source*: Kennedy R.J. Varrialle P, Alferito JC. Textbook of vectocardiography. Harper and Row. N.J. 1970

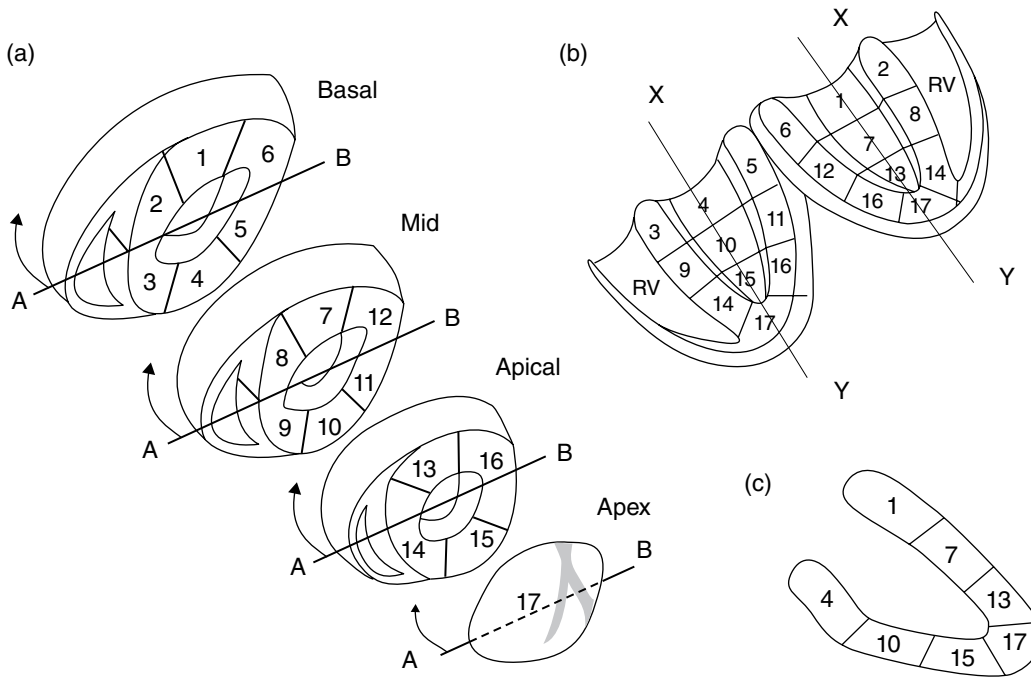


Figure 1.8 (a) Segments into which the heart is divided, according to the transverse (short-axis view) transections performed at the basal, mid, and apical levels. The basal and medial transections delineate six segments each, while the apical transection shows four segments. Together with the apex, they constitute the 17 segments in which the heart can be divided according to the classification performed by the American imaging societies (Cerqueira, Weissman, and Disizian 2002). (b) View of the 17 segments with the heart open in a horizontal long-axis view, and (c) vertical long-axis (sagittal-like) view seen from the right side. Figure 1.13 shows the perfusion of these segments by the corresponding coronary arteries.

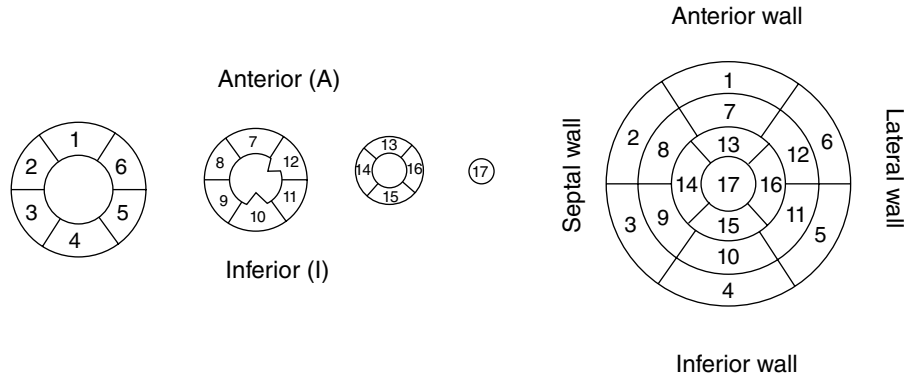


Figure 1.9 Images of the segments into which the left ventricle is divided according to the transverse transections (short-axis view) performed at the basal, mid, and apical levels, considering that the heart is located in the thorax in a posteroanterior and right-to-left position. Segment 4, inferobasal, was classically named posterior wall. The basal and medial transections delineate six segments each, while the apical transection shows four segments. Together with the apex, the left ventricle can be divided into 17 segments. Note, in the mid-transection, the location of the papillary muscles is shown. To the right, all 17 segments in the form of a polar map (bull's eye), just as it is represented in nuclear medicine reports.

Thanks to CMR correlations (Figure 1.10), there is evidence that the sagittal view of the heart is, in respect to the thorax, located with an oblique right-to-left inclination rather than in a strictly anteroposterior position. This helps us to understand how the RS (R) or predominant ST-segment depression patterns in V1 is the consequence of infarction of the lateral, not the inferobasal, segment (classical posterior wall) (Figure 1.11). However, it must be kept in mind that in the majority of individuals, except for the very lean ones (see Figure 1.12c), the part of the basal inferior wall, that can be really posterior, depolarizes late and therefore, cannot generate Q waves when infarcted (segment 4, or inferobasal).

The usefulness of invasive and non-invasive imaging techniques and their correlations with ECG in IHD:

- Non-invasive imaging techniques, especially SPECT, are very useful in detecting perfusion defects during the exercise test.
- In this book, we will point out the importance of ECG-coronary angiography correlations to identify the coronary artery occlusion site and the myocardial area-at-risk.
- The role of coronary angiography, and especially in chronic IHD of CTA, will be discussed.
- In chronic MI we will emphasize the importance of the ECG-CMR correlations to identify and locate the area of infarction.
- ECG is very useful in the coronary care unit (CCU) and is also used routinely in the chronic phase.
- X-ray examination still plays a role, especially in the acute phase (cardiac enlargement and pulmonary edema), and in the detection of aneurysms and calcifications, visualization of pacemaker leads, etc.

In order to clarify the terminology of the walls of the heart, a committee appointed by ISHNE (the International Society for Holter and Non-invasive Electrocardiography) made the following recommendations (Bayés de Luna 2006c):

1) Historically, the terms "true" or "strictly posterior" MI have been applied when the basal part of the LV wall that lies on the diaphragm was involved. In echocardiography the term "posterior" was replaced by "inferolateral", some noninvasive imaging reports still use the term "posterior" or "posterolateral". It is the consensus of this report **to abandon the term "posterior" and to recommend that the term "inferior" be applied to the entire LV wall that lies on the diaphragm**.

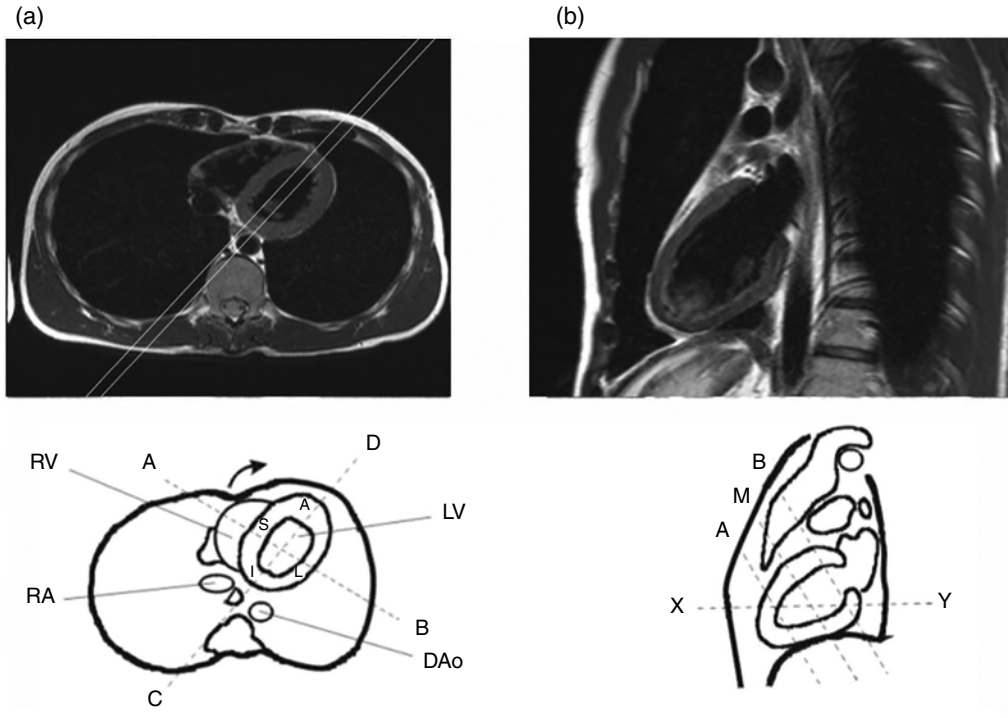


Figure 1.10 CMR imaging. (a) Thoracic horizontal axial plane at the level of the "*xy*" line of the drawing on the right side of the figure. The four walls can be adequately observed: anterior (A), septal (S), lateral (L), and inferior (I), represented by the inferobasal portion of the wall (segment 4 of the Cerqueira et al. statement) that bends upwards in this case (b). The infarction vector generated principally in segments 4 (basal inferior) and 10 (mid inferior); in the case of very lean individuals (Figure 1.12C) this region faces lead V3 and not V1 (line CD). On the contrary, the vector of infarction that arises from the lateral wall, from segments 5 (basal inferolateral) and 11 (mid inferolateral) faces V1 and therefore explains an RS morphology in this lead (line BA). (b) According to the transection, following the vertical longitudinal axis of the heart (line CD in (a)), we obtain a sagittal oblique view of the heart from the left side. These four walls, anterior, inferior (inferobasal), septal, and lateral, are clearly seen in the horizontal axial plane (a), and two walls, anterior and inferior including the inferobasal segment, in a sagittal-like plane (b).

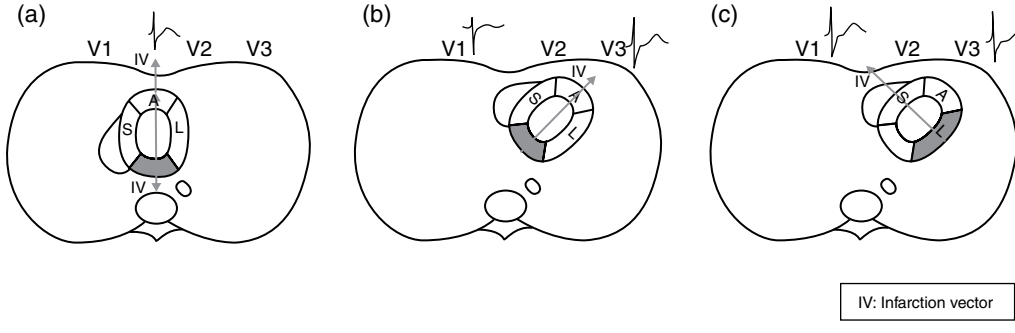


Figure 1.11 (a) The posterior (inferobasal) wall as it was wrongly considered to be placed. With this location an infarction vector of inferior infarction (segments 4 [basal inferior] and 10 [mid inferior] in case of very lean individuals) faces V1–V2 and explains the RS pattern in these leads. (b, c) The real anatomic position of inferior (inferobasal) and lateral wall infarctions. The mean QRS vector in infarctions of the inferobasal and mid-segment in lean individuals faces V3–V4 and not V1, and this may contribute to the RS pattern seen in these leads. On the contrary, the vector of infarction of the lateral wall faces V1 and may explain an RS pattern in this lead.

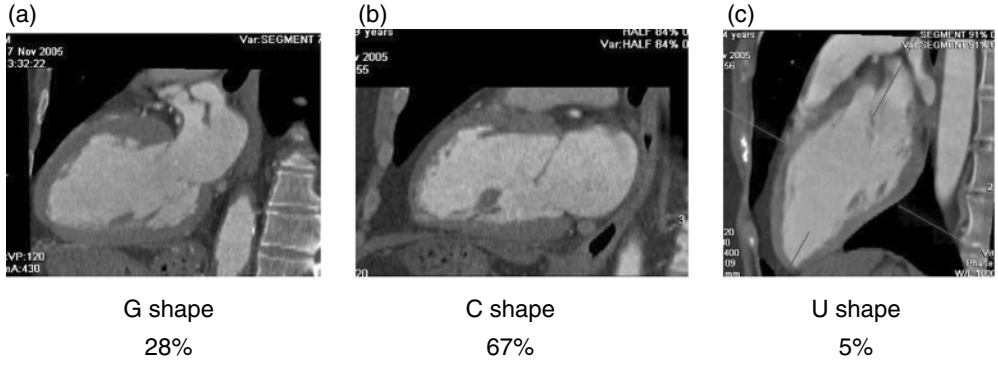


Figure 1.12 Sagittal-oblique view in an individual with normal body build (a) (G shape), with horizontal heart (b) (C shape) and in a very lean individual (c) (U shape). We have found that the inferior wall does not bend upward in C shape (two-third of the cases), and only in very lean individuals with U shape, the largest part of the wall is posterior (5% of the cases) (c).

2) Therefore, the **four walls of the heart are named anterior**, **septal**, **inferior,** and **lateral**. The basal and mid segments are further divided into six (anteroseptal, anterior, anterolateral, inferolateral, inferior and inferoseptal). This decision regarding the change in terminology achieves agreement with the consensus of experts in cardiac imaging appointed by American Heart Association (AHA) (Cerqueira, Weissman, and Disizian 2002) and thereby provides great advantages for clinical practice.

1.2 The Perfusion of the Walls of the Heart and the Conduction System

The myocardium and conduction system (CS) are perfused by the right coronary artery (RCA), the left anterior descending coronary artery (LAD) and the left circumflex coronary artery (LCX). Figure 1.1 shows the typical correlation of coronary angiography and CTA in the normal coronary tree and in some anatomic variants.

Figures 1.13b–d shows the perfusion that the different walls with their corresponding segments receive from the three coronary arteries. The areas with common perfusion are colored in gray in Figure 1.13a. Figure 1.13e shows the correlation of ECG leads with the bull's eye image (Bayés, Fiol, and Antman 2008).

1.3 The Myocardial Areas Perfused by three Coronary Arteries these areas were Described by Candell-Riera et al. (2005) and Gallik et al. (1995)

1.3.1 LAD (Figure 1.13b)

It perfuses the anterior wall, especially via the diagonal branches (segments 1 [basal anterior], 7 [mid anterior], and 13 [apical anterior]), the anteroseptum, a portion of inferoseptum (mainly the basal segments), via the septal branches (segments 2 [basal anteroseptal], 8 [mid anteroseptal], and part of 14 [apical septal], 3 [basal inferoseptal], and 9 [mid inferoseptal]). Segment 14 is perfused by the LAD, sometimes shared with the RCA, and also parts of segments 3 and 9 are shared with the RCA. Segments 12 (mid anterolateral) and 16 (apical lateral) are sometimes perfused by the second and third diagonals and sometimes by the second obtuse marginal branch of the LCX. Frequently, the LAD perfuses the apex and part of the inferior wall,

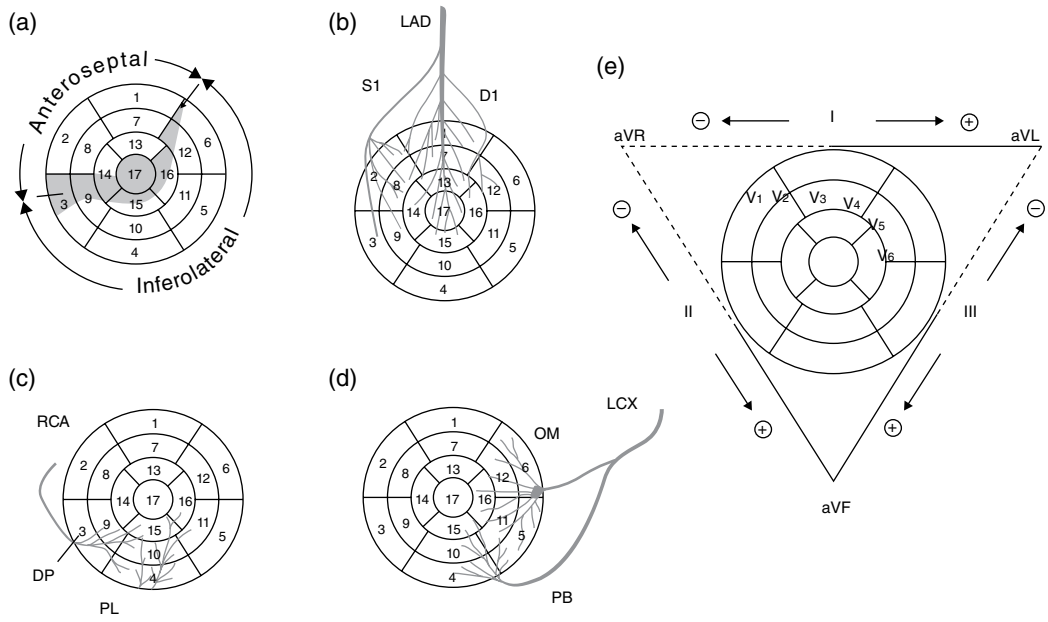


Figure 1.13 According to the anatomical variants of coronary circulation, there are areas of shared variable perfusion (a). The perfusion of these segments by the corresponding coronary arteries (b–d) can be seen in the "bull's eye" images. For example, the apex (segment 17) is usually perfused by the LAD but sometimes by the RCA or even the LCX. Segments 3 (basal inferoseptal) and 9 (mid inferoseptal) are shared by the LAD and RCA, and also a small part of the mid-low lateral wall is shared by LAD and LCX. Segments 4 (basal inferior), 10 (mid inferior), and 15 (apical inferior) receive their blood supply from the RCA or the LCX, depending on which of them is dominant (the RCA in >70–80% of individuals). Segment 15 often receives blood from the LAD. (e) Correspondence of ECG leads with the bull's-eye image. Abbreviations: LAD, left anterior descending coronary artery; S1, first septal branch; D1, first diagonal branch; RCA, right coronary artery; PD, posterior descending branch; PL, posterolateral branch; LCX, left circumflex coronary artery; OM, obtuse marginal branch; PB, posterobasal branch (usually named posterolateral).

as the LAD wraps around the apex in over 80% of cases (segment 17 [apex] and part of segment 15 [apical inferior]).

1.3.2 RCA (Figure 1.13c)

This artery perfuses, in addition to the right ventricle (RV), the inferior portion of the septum (part of segments 3 [basal inferoseptal] and 9 [mid inferoseptal]). Usually, the more basal part of the septum receives double perfusion (LAD + RCA conal branch). Segment 14 (apical septal) corresponds more to the LAD, but it is sometimes shared by both arteries (see before). The RCA perfuses a large part of the inferior wall (segment 10 [mid inferior] and parts of 4 [basal inferior] and 15 [apical inferior]). Segments 4 (basal inferior) and 10 (mid inferior) can be perfused by the LCX if this artery is dominant (in ~10–15% of all cases), and at least part of segment 15 (apical inferior) is perfused by the LAD if this artery is long. Parts of the lateral wall (segments 5 [basal inferolateral], 11 [mid inferolateral] and 16 [apical lateral]) may, on certain occasions, pertain to RCA perfusion if it is very dominant. Sometimes segment 4 (basal inferior) receives double perfusion (RCA + LCX). Lastly, the RCA perfuses segment 17 (apex) if the LAD is very short.

1.3.3 LCX (Figure 1.13d)

The LCX perfuses most of the lateral wall – the anterior basal part (segment 6 [basal anterolateral]) and the mid and distal parts of the

lateral wall shared with the LAD (segments 12 [mid anterolateral] and 16 [apical lateral]) and the inferior part of the lateral wall (segments 5 [basal inferolateral] and 11 [mid inferolateral]) sometimes shared with RCA. It also perfuses, especially if it is the dominant artery, a large part of the inferior wall, especially segment 4 (basal inferior), on rare occasions segment 10 (mid inferior), and part of segment 15 (apical inferior) and the apex (segment 17).

The double perfusion of some parts of the heart explains that these areas may be at least partially preserved in case of occlusion of one artery, and that in case of necrosis, the involvement is not transmural.

If one coronary artery is acutely occluded, one of the main zones of the heart will be involved in acute ischemia or MI (Figure 1.13a): (i) the **inferolateral** zone, which encompasses all the inferior wall, a portion of the inferior part of the septum and most of the lateral wall (**occlusion of the RCA or the LCX**); or (ii) the **anteroseptal** zone, which comprises the anterior wall, the anterior part of the septum and often a great part of inferior septum and part of the mid-low anterior portion of the lateral wall (**occlusion of the LAD**). However, a**ccording to recent findings by Allencherril et al. (2018), there is a need for a change of concepts of this terminology. Their observations from ECG-CE-CMR correlation in STEMI patients do not support the existence of isolated basal anteroseptal or septal infarction. Therefore, anteroapical infarction is a more appropriate term than anteroseptal infarction**.

In general, the LAD, if it is large, as is seen in over 80% of cases, tends to perfuse not only the apex, but also part of the inferior wall (Figures 1.1 and 1.13).

The occlusion of a coronary artery may affect only one wall (anterior, septal, lateral, or inferior) or, more often, more than one wall. ACSs and infarcts in their chronic phase, which affect only one wall, are uncommon. Even an occlusion of the distal part of a coronary artery usually involves at least two walls. For example, the distal LAD affects the apical part of the anterior wall but also the apical part, even though small, of the septal, lateral, and inferior wall (Bogaty et al. 2002), and the distal LCX generally affects part of the inferior and lateral walls. In addition, an occlusion of the diagonal branch, although fundamentally affecting the anterior wall, often also involves the mid anterior part of the lateral wall, and even occlusion of the first septal branch, or a subocclusion of the LAD encompassing the septal branches, involves part of the septum and often a small part of the anterior wall. Probably, occlusion of the oblique marginal (OM) branch (part of the lateral wall) or distal branches of a non-dominant RCA or LCX (part of the inferior wall) involves only one single wall.

In fact, **whether ACSs or established infarctions involve one or more walls has relative importance. What is most important is their extension, related mainly to the site of the occlusion and to the characteristics of the coronary artery (dominance, etc.)**. Naturally, on the basis of all that was previously discussed, large infarcts involve a myocardial mass that usually corresponds to several walls, but the involvement of several walls is not always equivalent to a large infarct. For instance, the apex, although part of various walls, is equivalent to only a few segments. Therefore **knowing what segments are affected allows us to better approximate the true extension of the ventricular involvement** (Cerqueira, Weissman, and Disizian 2002). Lastly, although in many cases multivessel coronary disease exists, this does not signify that a patient has suffered more than one infarct.

Consequently, in order to better assess the prognosis and the extent of ACSs, and infarcts in the chronic phase, it is very important in the acute phase to establish the correlation between the ST-segment deviations/T-wave changes and the site of occlusion and the area-at-risk, and in the chronic phase between leads with Q waves and the number and location of the LV segments infarcted (Figures 1.8 and 1.9).

The perfusion of the conduction system structures is as follows:

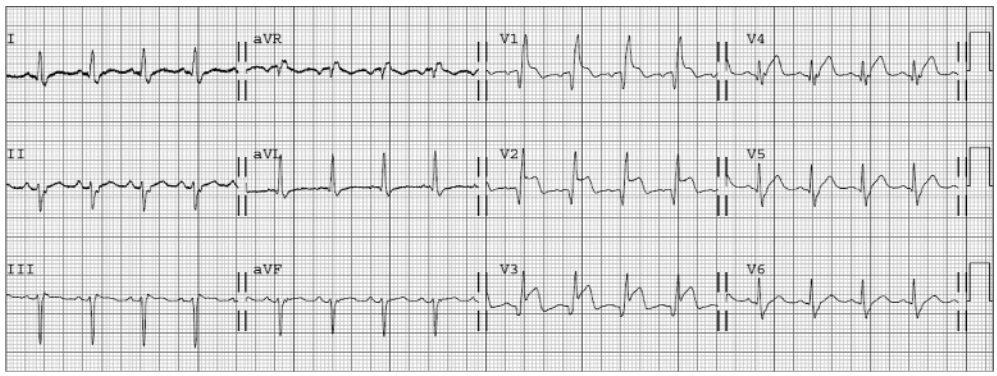
a) **The sinus node** and the sinoatrial zone by the RCA or the LCX (approximately 50% each)
b) **The atrio-ventricular (AV) node** by the RCA in 90% of cases and by the LCX in 10% of cases
c) **The right bundle branch and the anterior subdivision of the left bundle branch** by the LAD
d) **The inferoposterior division of the left bundle branch** by septal branches from the LAD and the RCA, or sometimes the LCX
e) **The left bundle branch trunk** receiving double perfusion (RCA + LAD)

This information is useful in understanding when and why bradyarrhythmias and/or intraventricular conduction abnormalities may occur during an evolving ACS (see "Arrhythmias and intraventricular conduction blocks" in ACS).

1.4 Self-Assessment. Case Reports. Chapter 1

Case 1

A 70-year old woman with a 2-day history of malaise, weakness, nausea, and vomiting. She reported crushing chest pain with dyspnea on effort, which subsided with rest, for several weeks. The ECG in the emergency department showed anterior ST elevation with right bundle branch block and left anterior fascicular block. Coronary angiography showed a subtotal occlusion of the proximal LAD with TIMI 1 flow. Primary PCI was performed with implantation of a drug-eluting stent. The post-reperfusion ECG (Figure) shows persistence of the ST elevations and conduction disorders. Despite reperfusion therapy, there was hemodynamic instability and metabolic acidosis with need for vasoactive support with noradrenaline. Echocardiography showed septal hypokinesis.

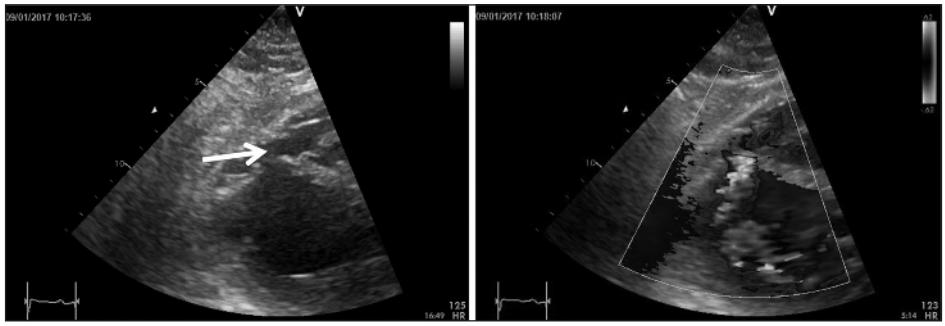


Question/statements

1) Should the patient have a new intervention because of treatment failure?
2) Persistent ST elevations are caused by norepinephrine.
3) The persistence of ST elevation indicates a (missed) mechanical complication of the infarction.
4) The ECG shows inappropriate sinus tachycardia.

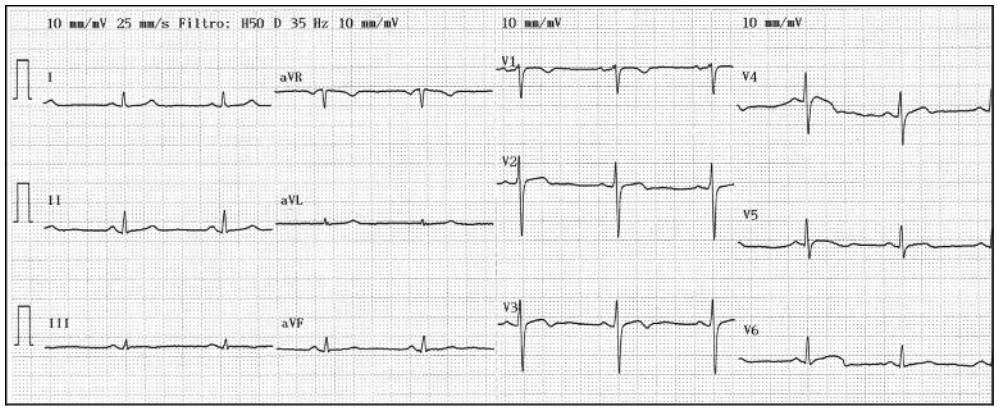
Correct answer: 3

The persistence of ST elevations and hemodynamic instability should raise the suspicion of a post-infarction mechanical complication. The echocardiogram should be repeated in several projections. Next figure shows a rupture of the interventricular septum (arrow) that had gone unnoticed. In this case the ECG pattern was an important key to raise the suspicion of an important complication of the disease.

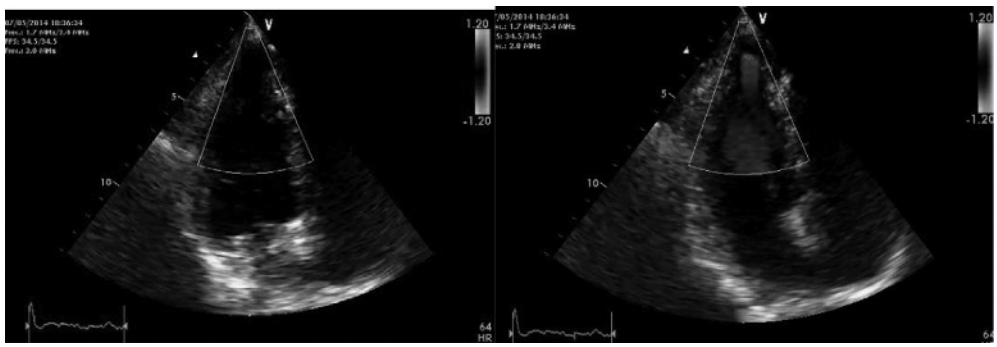


Case 2

A 60-year old woman with epigastric discomfort. Her medical history included a treadmill exercise test several years earlier because of negative T waves in V2 to V4 found on a routine ECG (Figure). The ECG findings were considered a normal variant in a woman and the treadmill test was negative. The ECG shows sinus rhythm with mild biphasic T waves in V2 to V4.



An echocardiogram was performed (left diastole, right systole)

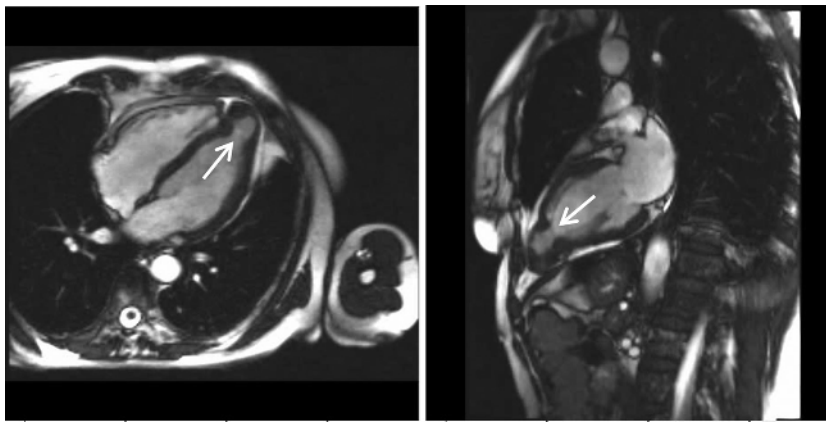


Suggestions for diagnosis:

1) Congenital apical aneurysm
2) Apical aneurysm secondary to undiagnosed silent infarction
3) Apical congenital diverticulum
4) Apical hypertrophic cardiomyopathy

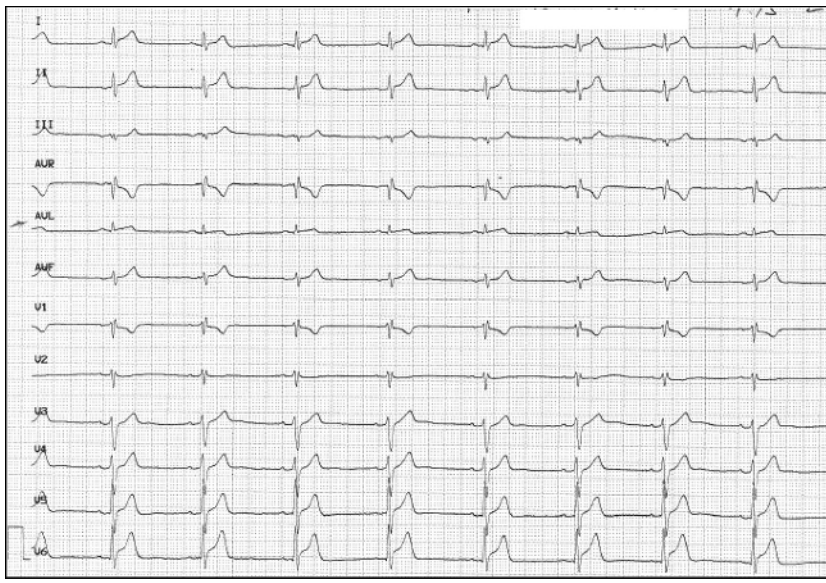
Correct answer: 1

The echocardiogram (of intermediate quality) detected an apical aneurysm. The ventricular aneurysm with a paradoxical expansion during systole and end-systolic flow pattern from the ventricle to the aneurysm was better demonstrated with CMR (next figure, arrows) (Fiol M, et al. Int J Cardiovasc Imaging. 2015;31: 1261–1262). A "normal variant" is uncommon, but still possible, at the age of 60.

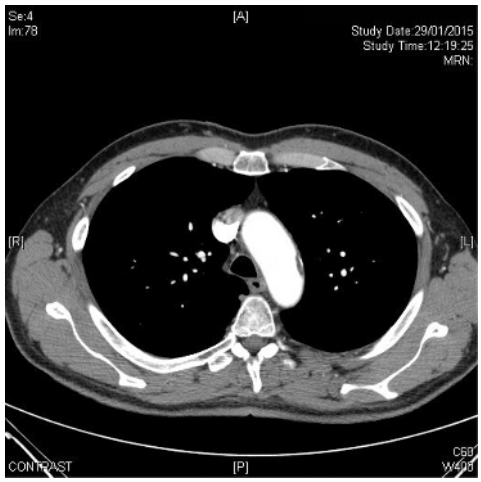


Case 3

A 68-year-old diabetic patient presented with precordial pain radiating to the back. The ECG (Figure) was interpreted as non-diagnostic. An aortic CT was performed to rule out aortic dissection.



CT (Figure) shows an aortic ulcer. The patient was admitted to the CCU and continued to have pain.

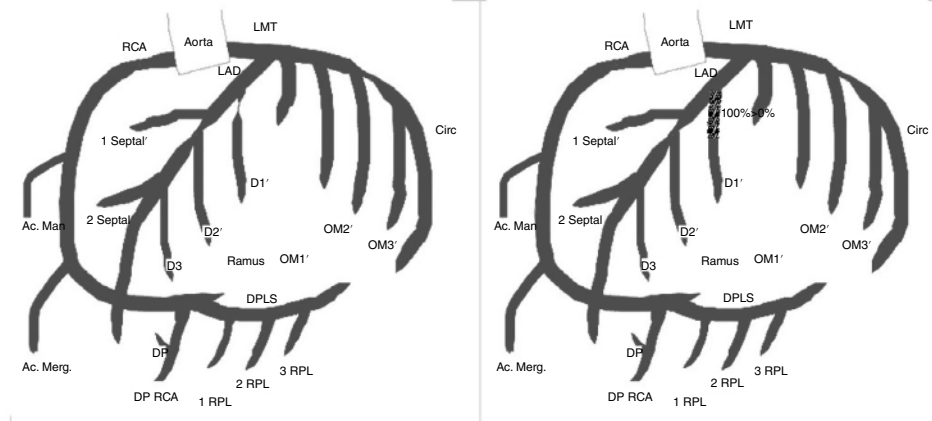


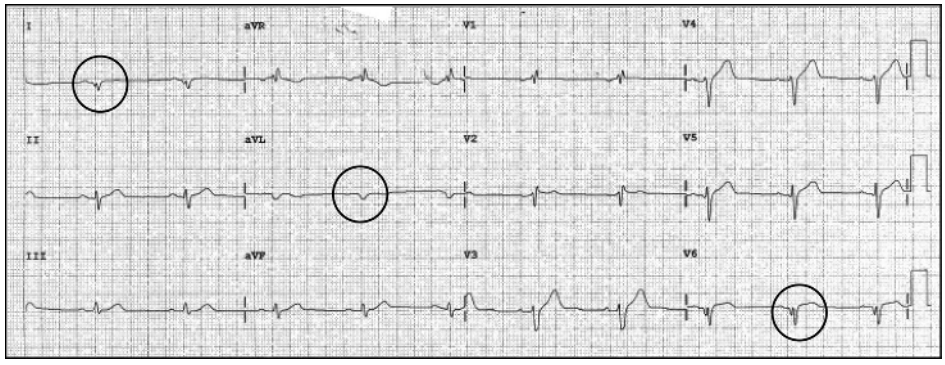
Suggestions for diagnosis:

1) The ulcer has progressed to dissection and the CT should be repeated
2) The ulcer has progressed to dissection involving the coronary ostium
3) STE-ACS has gone unnoticed and coronary angiography is indicated
4) Pain is due to peptic ulcer. Endoscopy is indicated

Correct answer: 3

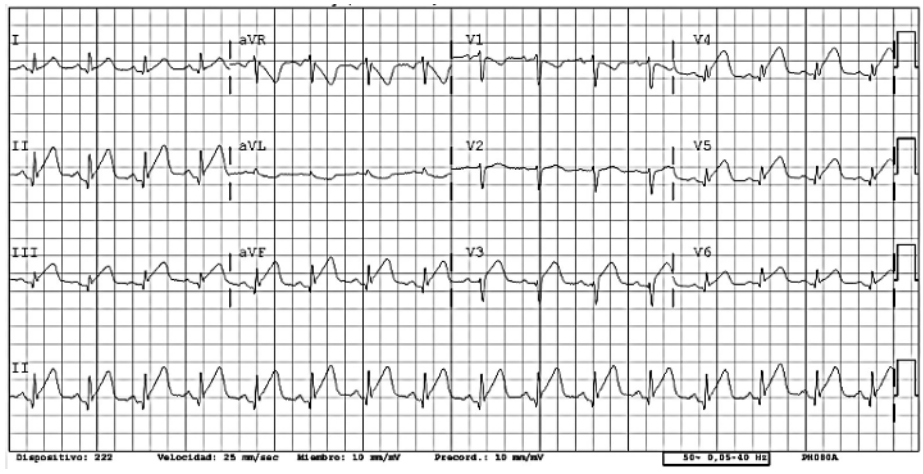
STE-ACS has gone unnoticed and coronary angiography should be performed. This is a demonstrative case of an early ECG pattern of a first diagonal branch occlusion (minimal ST elevation in I, II, aVL, V5-V6). The presence of an aortic ulcer indicated an acute aortic syndrome, which made the diagnosis more challenging. The follow-up ECG (Figure) shows new Q waves in I, aVL, and V6. The diagram shows the location of the lesion within the coronary tree (left) and the situation after stenting (right).



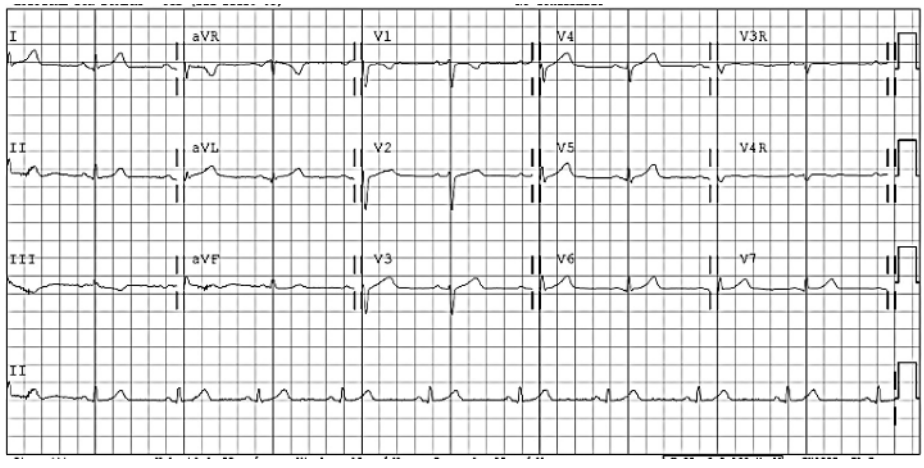


Case 4

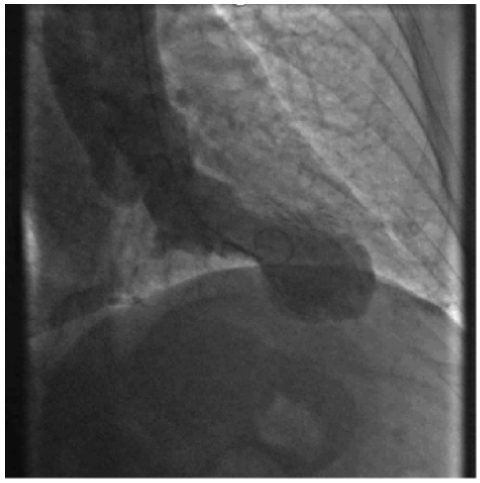
A 58-year old woman with retrosternal pain radiating to the back. The ECG (Figure) shows sinus rhythm, PR depression in the inferior leads, ST elevations in I, II, III, aVF, and V3-V6 and ST depression in aVR and V1.



The second ECG (Figure) shows spontaneous resolution of the ST/T changes



A coronary angiography (Figure) was performed.

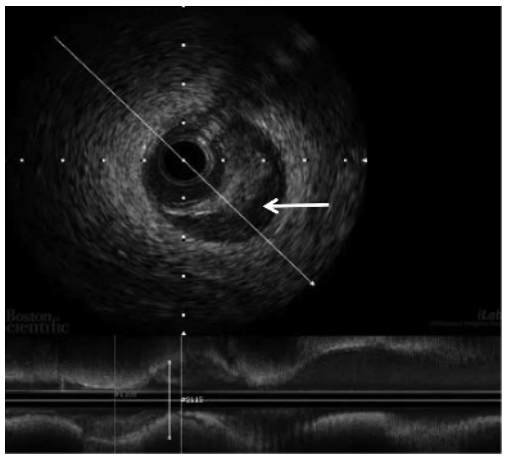


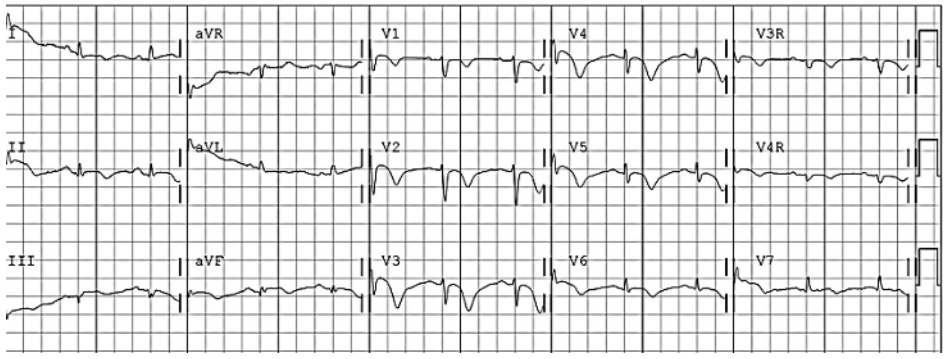
Suggestions for diagnosis:

1) It is STE-ACS by thrombotic occlusion and spontaneous or medication-induced reperfusion of the mid-LAD or coronary spasm
2) It is a spontaneous coronary dissection
3) It is Takotsubo
4) All can be true

Correct answer: 4

It could be an aborted infarction due to the administration of antiplatelet drugs or a coronary spasm resolved with nitroglycerin. On the other hand, the image of the ventriculography is typical of Takotsubo and it is a woman, although relatively young, and without a triggering factor. Although the first ECG can be mistaken for acute pericarditis, the rapid resolution of the changes suggests ischemia. Spontaneous coronary dissection is more frequent in women and often produces a variable degree of coronary occlusion. In this case, intravascular ultrasound was indicated to clarify the etiology, and it showed a clear dissection (Figure, arrow). The ECG hours later showed typical post-reperfusion negative T-waves (Figure). A CMR performed days later showed late enhancement in the apex (Figure, arrow)




I
aVR
V1
V4
V3R
II
aVL
V2
V5
V4R
III
aVF
V3
V6
V7