Chapter 1 The Electrical Activity of the Heart

Basic concepts

The heart is a pump that sends blood to every organ in the human body. This is carried out through an electrical stimulus that originates in the sinus node and is transmitted through the specific conduction system (SCS) to contractile cells.

During the rest period, myocardial cells present an equilibrium between the positive electrical charges outside and the negative charges inside. When they receive the stimulus propagated from the sinus node, the activation process of these cells starts. **The activation** of myocardial cells is an electro-ionic mechanism (as explained in detail in Chapter 5) that involves two successive processes: **depolarization**, or loss of external positive charges that are substituted by negative ones, and **repolarization**, which represents the recovery of external positive charges.

The process of depolarization in a contractile myocardial cell starts with the formation of a depolarization dipole comprising a pair of charges (-+) that advance through the surface cell like a wave in the sea, leaving behind a wave of negativity (Figure 1.1A). When the entire cell is depolarized (externally negative), a new dipole starting in the same place is formed. This is known as a dipole of repolarization (+-). The process of repolarization, whereby the entire cell surface is supplied with positive charges, is then initiated (Figure 1.1B).

The expression of the dipoles is a vector that has its head in the positive charge and tail in the negative one. An electrode facing the head of the vector records positivity (+), whereas when it faces the tail it records negativity (–) (Figures 1.1–1.3; see also Figures 5.24, 5.25, and 5.28). The deflection originating with the depolarization process is quicker because the process of depolarization is an active one (abrupt entry of Na ions, and later Ca) and the process of repolarization is much slower (exit of K) (see Chapter 5, Transmembrane action potential).

If what happens in one contractile cell is extrapolated to the left ventricle as the expression of all myocardium,

we will see that the repolarization process in this case starts in the opposite place to that of depolarization. This explains why the repolarization of a single contractile cell is represented by a negative wave, whereas the repolarization of the left ventricle expressing the human electrocardiogram (ECG) is represented by a positive wave (Figure 5.28) (see Chapter 5, from cellular electrogram to human ECG).

How can we record the electrical activity of the heart?

There are various methods used to record the electrical activity of the heart. The best known method, the one we examine in this book, is electrocardiography. An alternative method, rarely used in clinical practice today but very useful in understanding ECG curves and therefore helpful in learning about ECGs, is vectorcardiography.

The latter and other methods will be briefly discussed in Chapter 25. These include, among others, body mapping, late potentials, and esophageal and intracavitary electrocardiography. In addition, normal ECGs can be recorded during exercise and in long recordings (ECG monitoring and Holter technology). For more information about different techniques see Chapter 3, The Future of Electrocardiography or consult our book *Clinical Arrhythmology* (Bayés de Luna 2011), and other ECG reference books (Macfarlane and Lawrie 1989; Wagner 2001; Gertsch 2004; Surawicz *et al.* 2008) (see page X).

What is the surface ECG?

The ECG is the standard technique used for recording the electrical activity of the heart. We can record the process of depolarization and repolarization through recording electrodes (leads) located in various places.

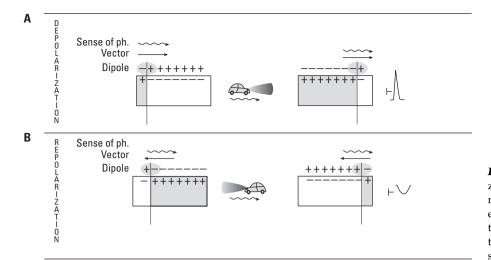


Figure 1.1 Depolarization and repolarization of the dipole in an isolated myocardium cell. We see the onset and end of the depolarization and repolarization processes and how this accounts for the positivity and negativity of corresponding waves (see text and Chapter 5).

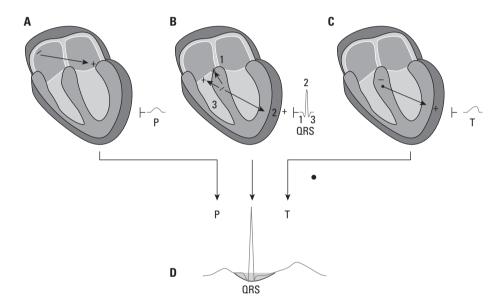


Figure 1.2 The origin of P, QRS, and T deflections. When an electrode faces the head (+) of a vector of depolarization (P, QRS) or repolarization (T), it records positivity. When an electrode faces the tail of a vector (-), it records negativity. Atrial repolarization is hidden in the QRS (shadow area) (see text and Chapter 5).

The depolarization process of the heart, atria and ventricles (see Chapter 5 and Figures 5.16 and 5.18) starts with the formation of a dipole of depolarization (-+), which has a vectorial expression (→→) that advances through the surface of the myocardium and seeds the entire surface of the myocardial cells with negative charges. A recording electrode facing the head of the vector records positivity (Figure 1.2). Later, the repolarization process starts with the formation of a repolarization **dipole** (+-), which also has a vectorial expression. During this process the positive charges of the outside surface of the cells are restored.

These two processes relate to specific characteristics of the atria and ventricles (Figure 1.2). The process of atrial depolarization, when recorded on the surface of the body in an area close to the left ventricle (Figure 1.2), presents as a small positive wave called the P wave (^). This is the expression of the atrial depolarization dipole (vector). The process of ventricular depolarization, which occurs later when the stimulus arrives at the ventricles, usually presents as three deflections (♠), known as the QRS complex, caused by the formation of three consecutive dipoles (vectors). The first vector appears as a small and negative deflection because it represents the depolarization of a small area in the septum and is usually directed upwards and to the right and recorded from the left ventricle as a small negative deflection ("q"). Next, a second important and positive vector is formed, representing the R wave. This is the expression of depolarization in most of the left ventricular mass. The head of this vector faces the recording electrode. Finally, there is a third small vector of ventricular depolarization that depolarizes the upper part of the septum and right ventricle. It is directed upwards and to the right and is recorded by the recording electrode in the left ventricle zone as a small negative wave ("s") (Figure 1.2).

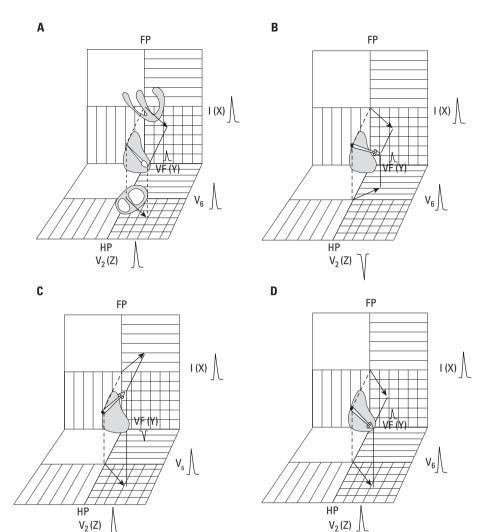


Figure 1.3 Four locations of a vector and their projection in frontal (FP) and horizontal planes (HP). A and B have the same projection in FP but not in HP. C and D has the same projection in HP but not in FP. Different positive and negative morphologies appear according to these projections. The locations of the orthogonal leads X, Y and Z perpendicular to each other are similar to I, VF, and V2 leads. Vertical lines correspond to the positive hemifields of VF and V2, and horizontal lines correspond to the positive hemifields of leads I and V6. FP lead I (X) = 0° ; VF (Y) = $+90^{\circ}$; HP $V2 (Z) = +90^{\circ}; V6 = 0^{\circ}.$

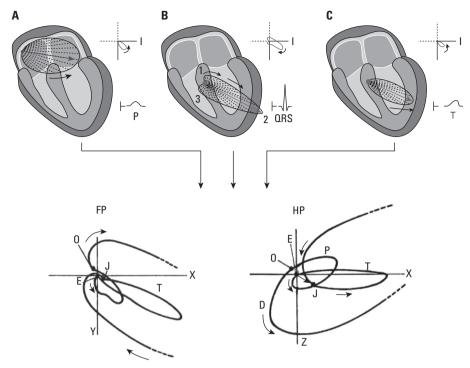
After depolarization of the atria and ventricles, the process of repolarization starts. The repolarization of the atria is usually a smooth curve that remains hidden within the QRS complex. The ventricular repolarization curve appears after the QRS as an isoelectric ST segment and a T wave. This T wave is recorded as a positive wave from the left ventricle electrode because the process of ventricular repolarization, as already mentioned and later explained in detail (see Chapter 5, From cellular electrogram to the human ECG and Figures 5.24 and 5.25), appears very differently from what happens in an isolated contractile cell (see Figure 5.9). Repolarization starts on the opposite side to that of depolarization. Thus, the recording electrode faces the positive part of the dipole, or head of the vector, and will record a positive deflection, even though the dipole moves away from it (Figures 1.2C; see also Figures 5.24 and 5.25). Therefore, repolarization of the left ventricle in a human ECG (the T wave) is recorded as a positive wave, just as occurs with the depolarization complex (QRS) in leads placed close to the left ventricle surface (\rightarrow).

The successive recording of the ECG is linear and the distance from one P–QRS–T to another can be measured in time. The frequency of this sequence is related to heart rate.

The heart is a three-dimensional organ. In order to see its electrical activity on a two-dimensional piece of paper or screen, it must be projected from at least two planes, the frontal plane and the horizontal plane (Figure 1.3).

The shape of the ECG varies according to the location (lead) from which the electrical activity is recorded. In general, the electrical activity of the heart is recorded using 12 different leads: six on the frontal plane (I, II, III, VR, VL, VF), located from +120° to -30° (the VR is usually recorded in the positive part of the lead that is located in -150°) (see Figures 6.10 and 6.11), and six on the horizontal plane (V1–V6) located from +120° to 0° (see Chapter 6, Leads and Figures 6.10 and 6.13).

Each lead has a line that begins where the lead is placed, 0° for lead I or +90° for lead VF in the frontal plane (FP) and 0° for lead V6 and +90° for lead V2 in the horizontal plane (HP), for example (see Figure 6.10), and ends at the opposite side of the body, passing through the



EO = P loop; OJ = QRS loop; JE = T loop; OJ = ST vector

Figure 1.4 The origin of P, QRS, and T loops. The vectorcardiographic curve is the union of the heads of multiple vectors that form during the consecutive processes of depolarization and repolarization (see text and Figure 5.23).

center of the heart. By tracing each perpendicular line that passes through the center of the heart, we may divide the electrical field of the body into two hemifields for each lead, one positive and one negative (Figure 1.3). A vector that falls into the positive hemifield records positivity, while one that falls into the negative hemifield records negativity. When a vector falls on the line of separation between hemifields, an isodiphasic curve is recorded (see Chapter 6, Figures 6.14 and 6.16).

The different vectors are recorded as positive or negative depending on whether they are projected onto positive or negative hemifields of different leads (Figures 1.3 and 1.5). This is a key concept for understanding the morphology of ECG curves in different leads and is explained in Chapter 6 in more detail (Figure 6.14).

The ECG and its different morphologies can be explained using the following sequence:

Dipole \rightarrow Spatial vectors \rightarrow Projection in frontal (FP) and horizontal (HP) planes

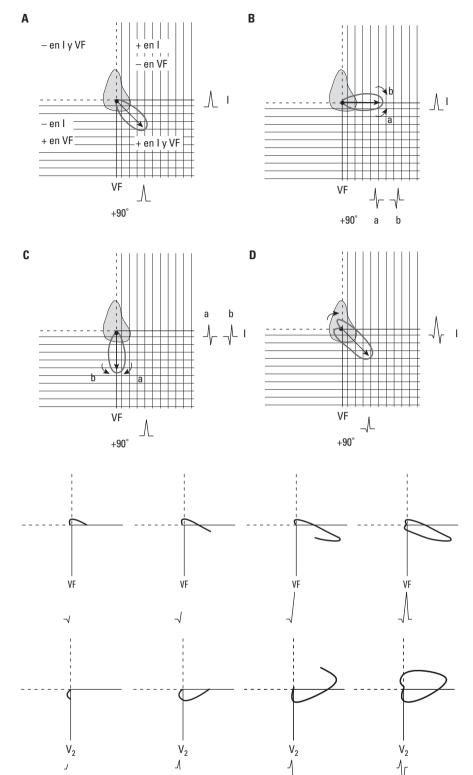
What is vectorcardiography?

The vectorcardiogram (VCG) is the closed curve or loop that records the entire pathway of an electrical stimulus from the depolarization of the atria (P loop) and ventricles (QRS loop) to the repolarization of the ventricles (T loop). These loops are recorded in FP and HP, as well

as in the sagittal plane. Made of the joined heads of the multiple vectors that form during the consecutive processes of depolarization and repolarization of the heart (Figure 1.4), VCG loops are obtained from three orthogonal (perpendicular to each other) leads, X, Y, and Z, which are placed in positions similar to those of leads I, VF, and V2, respectively (see Figure 1.3 and Chapter 25).

The VCG curve is a plot of voltage against voltage of the different waves generated by the heart (P, QRS, T loops), and therefore it is not possible to measure the time between the beginning of the P loop and the beginning of the QRS loop (PR interval), or the beginning of QRS and the end of the T loop (QT interval). However, we can interrupt the loops of P, QRS, and T by cutting the tracing every 2.5 ms, which allows the duration of each loop to be measured (see Figures 10.6–10.10 and 10.22–10.25).

One advantage of the VCG is that the different rotations of the loop can be visualized, which is important to know if the stimulus follows a clockwise or counter-clockwise rotation when one complex or wave is diphasic. Figure 1.5B shows how the mean vector of a loop directed to $+0^{\circ}$ that falls within the limit between the positive or negative hemifields in lead "Y" (VF) may present a $+-(\sqrt[1]{})$ or a $-+(\sqrt[1]{})$ deflection. The direction of the mean vector of the loop does not solve one important problem: a +- deflection is normal, but a -+ deflection may be the expression of myocardial infarction. The correct morphology will be shown by the direction of loop rotation, however (Figure 1.5). In addition, the mean vector of the QRS loop, which



We see how a morphology may be +- or -+ with the same vector but a different loop rotation (B and C) (A and B). The recording of the initial and terminal deflections of qRs are well understood with the correlation of the loop and hemifields in D (see I and VF).

Figure 1.5 The concept of the hemifield.

Figure 1.6 Correlation of a vectorcardiographic loop with an electrocardiographic morphology in VF and V2.

expresses the sum of all vectors of depolarization, does not indicate the direction of the small initial and final forces when these forces are opposed to a mean vector (Figure 1.5). However, the small part of the loop (beginning

and end) that falls in the opposite hemifield of the main vector can explain the complete ECG morphology with initial (q) and final small (s) deflections (Figures 1.5 and 1.6; see also Figures 7.10 and 7.11).

The VCG can be described using the following sequence: The head of multiple vectors \rightarrow Spatial loops \rightarrow Projection in FP and HP

ECG-VCG correlation

Bearing in mind the abovementioned information, it is clear that to better understand the morphology of an ECG we must consider the stimulus pathway through the heart (VCG loop) in different normal and pathological conditions and identify the projection of these loops in FP and HP. It is important to understand how the different parts of the loop that fall into the positive or negative hemifields of each lead correspond to the different deflections of an ECG curve (Figures 1.5 and 1.6; see also Figures 4.60 and 4.61) (ECG–VCG correlation). This allows the ECG curves to be drawn from the VCG loops and vice versa.

The key concepts around how ECG curves can be obtained from the VCG loops and vice versa (ECG–VCG correlation) are defined using the following sequence: $\label{eq:property} \mbox{Dipole} \rightarrow \mbox{Vector} \rightarrow \mbox{Loop} \rightarrow \mbox{Projection in different hemifields} \rightarrow \mbox{ECG patterns}$

Why do we record ECG curves and not VCG loops?

Although ECG-VCG correlation is used in this book to explain how the different ECG patterns are produced, the recording of vectorcardiographic loops for diagnostic purposes is rarely used in clinical practice at present. There are many reasons for the superiority of ECG curves over VCG loops, the main ones being as follows:

- The established diagnostic criteria of ECG in different pathologies are more defined and agreed-upon, compared with the VCG criteria. They are also easier to apply. Furthermore, it has not been clearly demonstrated by experts in ECG/VCG interpretation that VCG criteria provide more diagnostic information than that taken from ECGs, even in an era when VCG criteria were most used (Simonson *et al.* 1967; Rautaharju 1988; Van Bemmel *et al.* 1992).
- VCG loops do not show an appreciation of time (PR and QT interval), as previously mentioned.
- The ECG curves–VCG loop correlation gives us all the detailed information obtainable from VCG loops. In fact, if the origin of the ECG curve interpretation based on the projection of VCG loops in the positive and negative hemifields of different leads (ECG–VCG

correlation) is understood and used, we are able to derive the same information that VCGs provide by just looking at the ECG. For example, it has been reported (Benchimol et al. 1972) that VCGs are essential for the diagnosis of superoanterior fascicular block (hemiblock) associated with inferior myocardial infarction (MI). However, as discussed in Chapter 13, the same information can be obtained from the ECG if we recognize the exact pathway of the stimulus in both cases (inferior MI alone or associated with hemiblock) and we make a good ECG-VCG correlation (see Figure 13.98). Furthermore, many details provided by amplified VCG loops (the degree of ST shifts, onset of pre-excitation, characteristics of the P wave, etc.) can also be obtained from surface ECGs through amplification of the ECG waves, if necessary (see Figures 9.20 and 13.24) (Bayés de Luna 1998; Bayés de Luna and Fiol-Sala 2008).

- The VCG is not useful in the diagnosis of arrhythmias. Even information about the ectopic P wave may be correctly obtained from ECG curves.
- Computerization of ECG data and not of VCG has become dominant and, despite current limitations, it has a great future. However, as we see later on (Chapter 3, Limitations) it is necessary to improve the results with better technology and the inclusion of new data (clinical setting, etc.).
- The ECG is used more than the VCG for estimating the size of an MI (Selvester QRS scoring system) (Selvester *et al.* 1972; Wagner *et al.* 1982). However, in the era of ECG-imaging correlations it is necessary to improve the methodology of QRS score measurement to obtain a better correlation with contrast-enhanced cardiovascular magnetic resonance measurements (see later and Chapter 3, Limitations).
- ECG and not VCG patterns have already been correlated with imaging techniques, especially coronagraphy and contrast-enhanced cardiovascular magnetic resonance (CE-CMR). The correlation of ECG patterns with coronagraphy has allowed us to better locate the occlusion and determine the severity of ischemia in different types of acute coronary syndromes (leads with different ST shifts) (Sclarovsky 1999; Wellens et al. 2004; Bayés de Luna and Fiol-Sala 2008). It is also possible to obtain better classification and location of Q-wave MI (leads with abnormal Q or R waves as mirror image) using CE-CMR-ECG correlation (Bayés de Luna et al. 2006a, 2006b; Cino et al. 2006; Bayés de Luna 2007; Rovai et al. 2007; Bayés de Luna and Fiol-Sala 2008; Van der Weg et al. 2009). Similar correlations have not been done with VCG loops. Currently, a good estimation of infarction size using CE-CMR has been obtained (Kim et al. 1999; Moon et al. 2004). However, the correlation of infarct size measurement performed by surface ECG (QRS scoring system) (Selvester et al. 1972) with CE-CMR is not very consistent, and the CE-CMR shows larger values than the QRS score estimation (Weir et al. 2010). We hope that in the future

it will be possible to improve these results with new equations (see Chapter 3, The future). Good results have also recently been shown by Montant *et al.* (2010) using a contrast-enhanced three-dimensional echocardiography compared with CE-CMR to identify and quantify myocardial scars (positive and negative predictive value (PV) >90%).

- Young physicians should realize that ECG-VCG correlation is a basis for better understanding ECG curves. This does not mean that they need to know specific VCG criteria, such as the number of milliseconds the loop is going up and down, because these data obtained from the VCG does not add too much diagnostic information. Therefore, a recorded VCG loop alone is not clinically efficient. However, it is important to remember that the ECG-VCG correlation is a key point for better understanding of how ECG curves are originated (see below).
- Currently, there are very few devices that still correctly record VCG curves. At the Electrocardiology Congress held in 2009, titled with the subheading "VCG symposium," it was decided that this subheading should be suppressed (Macfarlane 2009). "Signum temporis" stated the first organizers (Sobieszczańska and Jagielski 2010). The number of VCG papers published in Medline in the 1970s and 1980s reached more than 800 per decade; today, in the first decade of this century, there are fewer than 60.
- It appears that the VCG loops taken from the orthogonal leads do not give much more information from a clinical point of view than a conventional 12-lead surface ECG. They are also time consuming and need special devices. Although we presume that VCG devices will no longer serve as an independent tool in the future, the VCG loops are very useful for teaching purposes and for some diagnostic, prognostic, and research purposes (Kors et al. 1990, 1998; Rautaharju et al. 2007; Pérez Riera 2009; Lazzara 2010). It may be that incorporating VCG loops synthesized directly from 12-lead surface ECG recordings would be an interesting option (see Chapter 3).

Why do we use ECG-VCG correlations to understand ECG patterns?

Electrocardiography and vectorcardiography are two methods for recording the electrical activity of the heart. As explained above, the ECG is a linear curve based on the positive and negative deflections recorded when an electrode faces the head or the tail of a depolarization and repolarization dipole, the expression of which is a vector, from leads placed in frontal and horizontal planes. The VCG is a loop that represents the outline of the joining of multiple dipoles (vectors) formed along the electrical stimulus pathway through the heart. The projection of these loops in frontal and horizontal planes is a closed curve that is different in morphology from the linear

curves of an ECG. Both ECG curves and VCG loops, however, are completely connected so that the ECG curve may be easily deduced from the VCG loop, and vice versa (see ECG–VCG correlation, Figures 1.5 and 1.6). As already mentioned, this approach is considered to be the best way to understand both the normal ECG and all the morphological changes that different pathologies introduce to the ECG.

The correlation between VCG loops and projection of this on different hemifields to understand the ECG pattern (**dipole** \rightarrow **vector** \rightarrow **loop** \rightarrow **hemifield sequence**) will no doubt remain a cornerstone of the teaching of the ECG (Grant and Estes 1952; Sodi-Pallares and Calder 1956; Cooksey *et al.* 1957; Cabrera 1958; Bayés de Luna 1998; Gertsch 2004).

- 1. The deduction of the ECG from the VCG loops is crucial to better recognizing how both normal ECG curves and the many ECG changes found in different heart diseases and under special circumstances originate.
- 2. Although the deductive method for teaching electrocardiography is fundamentally based on the correlation that exists between ECG curves and VCG loops, VCG criteria are not used for diagnosis.
- 3. In the majority of diagrams used to show the usefulness of VCG loop–ECG wave correlations, the pathway of the electrical stimulus is represented as a curve with a continuous line. When we record original tracings, however, dashes every 2.5 ms in the VCG loops are shown. Examples of this may be seen throughout this book (see, for example, Figures 11.25, 11.36, and 11.40).

References

Bayés de Luna A. Textbook of Clinical Electrocardiography. Futura, 1998.

Bayés de Luna A. New heart wall terminology and new electrocardiographic classification of Q-wave myocardial infarction based on correlations with magnetic resonance imaging. Rev Esp Cardiol 2007;60:683.

Bayés de Luna A. *Clinical Arrhythmology*. Wiley-Blackwell, 2011. Bayés de Luna A, Fiol-Sala M. *Electrocardiography in Ischemic Heart Disease*. Blackwell Futura, 2008.

Bayés de Luna A, Wagner G, Birnbaum Y, et al. A new terminology for left ventricular walls and location of myocardial infarcts that present Q wave based on the standard of cardiac magnetic resonance imaging: A statement for healthcare professionals from a committee appointed by the International Society for Holter and Noninvasive Electrocardiography. Circulation 2006a;114:1755.

Bayés de Luna A, Cino JM, Pujadas S, et al. Concordance of electrocardiographic patterns and healed myocardial infarction

- location detected by cardiovascular magnetic resonance. Am I Cardiol 2006b:987:443.
- Benchimol A, Desser KB, Schumacher J. Value of the vectorcardiogram for distinguishing left anterior hemiblock from inferior infarction with left axis deviation. Chest 1972;61:74.
- Braunwald E, Bonow RO, Mann DL, Zippes D, Libby P. *Heart Disease*, 11th edn. Elsevier Saunders, 2012.
- Cabrera E. *Teoría y práctica de la Electrocardiografía*. Edic INC México, La Prensa Médica Mexicana, 1958.
- Camm J, Luscher TF, Serruys PW (eds). The ESC Textbook of Cardiovascular Medicine. Blackwell. 2006.
- Cino J, Pons-Lladó G, Bayés de Luna A, *et al*. Utility of contrastenhanced cardiovascular magnetic resonance (CE-CMR) to assess how likely is an infarct to produce a typical ECG pattern. J Cardiovasc Magn Reson 2006;8:335.
- Cooksey J, Dunn M, Massie E. Clinical Vectorcardiography and Electrocardiography, 2nd edn. YearBook MP, 1957.
- Fuster V, Walsh RA, Harrington RA (eds). Heart's. 13th edition. McGraw-Hill, 2010.
- Gertsch M. The ECG: A two step approach for diagnosis. Springer, 2004.Grant R, Estes EH. Spatial Vector Electrocardiography. Blakston Co., 1952.
- Kim RJ, Fieno D, Parrish T, *et al*. Relationship of CE-CMR to irreversible injury, infarct age and contractile function. Circulation 1999;100:1992.
- Kors JA, Van Herpen G, Sitting AG, et al. Reconstruction of the Frank VCG from the standard ECG leads. Eur Heart J 1990;11:1083.
- Kors JA, De Bruyne MC, Hoes AW. T axis as an independent indicator of risk of cardiac events in elderly people. The Lancet 1998;352:361.
- Lazzara R. Spatial vectorcardiogram to predict risk for sudden arrhythmic death: Phoenix risen from the ashes. Heart Rhythm 2010;1614.
- Macfarlane PW. Interview with Peter W Macfarlane by Ljuba Bacharova. J Electrocardiol 2009;42:223.
- Macfarlane PW, Lawrie TDV (eds). Comprehensive Electrocardiography. Pergamon Press, 1989.
- Montant P, Chenot F, Gaffinet C, et al. Detection and quantification of myocardial scars using CE-3D-echocardiography Circulation CV Imag 2010;3:415.
- Moon JC, De Arenaza DP, Elkington AG, et al. The pathologic basis of Q-wave and non-Q-wave myocardial infarction: a cardiovascular magnetic resonance study. J Am Coll Cardiol 2004;44:554.

- Pérez Riera A. Learning easily Frank vectorcardiogram. Editora Mosteiro Sao Paulo, 2009
- Rautaharju PM. A hundred years of progress in electrocardiography. 2: the rise and decline of vectorcardiography. Can J Cardiol 1988;4:60.
- Rautaharju P, Prineas R, Zhang Z-M. A simple procedure for estimation of the spatial QRS/T angle from the standard 12-lead ECG. J Electrocardiol 2007;40:300.
- Rovai D, Di Bella G, Rossi G, et al. Q-wave prediction of myocardial infarct location, size and transmural extent at magnetic resonance imaging. Coronary Artery Dis 2007;18:381.
- Sclarovsky S. Electrocardiography of Acute Myocardial Ischaemic Syndromes. Martin Dunitz, 1999.
- Selvester RH, Wagner JO, Rubin HB. Quantitation of myocardial infarct size and location by electrocardiogram and vectorcardiogram. In Snelin HA (ed.) *Boerhave Course in Quantitation in Cardiology*. Leyden University Press, 1972, p. 31.
- Simonson E, Tune N, Okamoto N, et al. Vectorcardiographic criteria with high diagnostic accuracy. Z Kreislaufforsch 1967;56:1243.
- Sodi-Pallares D, Calder R. New Bases of Electrocardiography. Mosby, 1956.
- Sobieszczańska M, Jagielski J. The International Society of Electrocardiology: A 50 year history originated in Poland. J Electrocardiol 2010;43:187.
- Surawicz B, Knilans TK, Te-Chuan Chou. Chou's Electrocardiography in Clinical Practice, 6th edn. Saunders, 2008.
- Van Bemmel JH, Kors JA, van Herpen G. Combination of diagnostic classifications from ECG and VCG computer interpretations. J Electrocardiol 1992;25(suppl):126.
- Van der Weg K, Bekkers S, Gorgels A, *et al.* The R in V1 in non-anterior wall infarction indicates lateral rather than posterior involvement. Results from ECG/MRI correlations. Eur Heart J 2009;30(suppl):P2981.
- Wagner GS (ed.) *Marriott's Practical Electrocardiography*, 10th edn. Lippincott Williams & Wilkins, 2001.
- Wagner G, Freye C, Palmer ST, et al. Evaluation of QRS scoring system for estimating myocardial infarction size. Circulation 1982:65:347.
- Weir R, Martin T, Wagner G. Comparison of infarct size and LVEF by CE-CMR and ECG scoring in reperfused anterior STEMI. J Electrocardiol 2010;43:230.
- Wellens H, Doevedans P, Gorgels A. *The ECG in Acute Myocardial Infarction and Unstable Angina*. Kluwer Academic, 2004.