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Marc Gertsch is a true expert in the ECG and its analysis. In this small book, intended for medical students, house-staff, residents, and coronary care/intensive therapy unit nurses, the author eliminates much of the ritual explanation of the derivation of the electrocardiogram and, instead, provides a pragmatic and practical approach to its interpretation. The book explores important principles of electrocardiology and importantly electrocardiographic traces are consistently related to clinical scenarios and real-life cases.

The art of ECG interpretation is often shrouded in mystique but an important principle that Professor Gertsch uses throughout this book is to base his explanation of the ECG findings on evidence from the literature. In this way the “secret” of the ECG is thoroughly exposed and it becomes possible to use the technique to reach accurate and practical diagnoses.

These days many physicians rely on the automatic analysis of an ECG by a computer chip on board the ECG recording machine. It has even been suggested that such “diagnosis” is now so correct that it might be possible not print the tracing itself. This would reduce the physician’s familiarity and understanding of the ECG still further. As with the modern echocardiogram, we may expect to see only the reports of studies, and it will be left to our memories to relate the report to similar examples of findings seen in the past. Professor Gertsch’s book dispels this notion by explaining how the simple way in which an ECG may be interpreted and the essential aspect of making that interpretation within a clinical context, something that no ECG machine yet attempts to do!

Above all Marc Gertsch is a teacher, and a demanding teacher at that. He expects his students to learn and with this book he has provided the material for study. It is a pleasure and an education to read.

A. John Camm
Professor Clinical Cardiology
St George’s University of London
UK
Preface

This book is designed for medical students and colleagues (including general practitioners, internists, and cardiologists) who need a short, practical book to help them correctly interpret more than 90% of all ECGs they see on a daily basis. Although this is a particularly ambitious goal for such a compact ECG book, important theoretic basics, as well as ventricular and atrial vectors, are included.

The knowledge needed to correctly identify diagnostic ECG features, including arrhythmias, requires extensive experience obtained over many years. Why then an “evidence-based” book? Because the cardiologist must be experienced in all fields of cardiology: in the ambulatory section using Echo/Doppler, ambulatory, and exercise ECGs; at the emergency station; and in the coronary care unit. The interventional cardiologist, who has performed thousands of heart catheterizations, coronary angiographies, and pacemaker implantations, and has moved beyond field into invasive electrophysiology, has a very critical attitude toward ECGs. Such cardiologists always respect the limits of ECGs when compared with other approaches, such as echocardiographic or coronary angiographic findings.

Another argument for an “evidence-based” book is that I read or re-read more than 2000 citations, of which approximately 650 have been included in this book. The evidence was improved by adding detailed descriptions of the coronary artery and left ventricular angiographic findings for every of the thirty ECG patterns of myocardial infarction, as well as by case reports/short stories of predominantly spectacular cases.

Designed to sit alongside my earlier publication, The ECG: A Two-Step Approach to Diagnosis, references to that book are given if the details cannot be found in any other ECG book (e.g., a long list of the etiology of electrolyte disturbances, the differentiation of myocardial infarction stages based on four principally different nomenclatures, and the ECG documentation of all possible false limb lead poling in electric left and vertical hearts).

It remains to thank my young academic collaborator Christoph Obrecht for extensive logistic help and meticulous corrections, to my excellent scientific designer Willy Hess, to Sandra Fabiani, Senior Editor at Springer Heidelberg with
whom all this began, to Barbara Chernow from Chernow Editorial Services Inc., New York, and to Grant Weston, Senior Editor at Springer London for constructive and pleasant collaboration.

Marc Gertsch, MD
June 2008
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The following abbreviations are used regularly throughout the text:

- AAI pacing: Atrial inhibited atrial pacing
- ACE: Angiotensin-converting enzyme
- acPE: Acute pulmonary embolism
- AF: Atrial fibrillation
- AJT: Automatic junctional tachycardia
- AMI: Acute myocardial infarction
- AP or acP: Action potential
- APB: Atrial premature beat
- $\tilde{\text{QRS}}_p$: Mean QRS axis in the frontal plane
- ASD: Atrial septal defect
- AV: Atrioventricular
- AVR: Aortic valve replacement
- AVNRT: Atrioventricular nodal reentrant tachycardia
- BVH: Biventricular hypertrophy
- CABG: Coronary artery bypass graft
- CAD: Coronary artery disease
- CHD: Coronary heart disease
- CK = CPK: Creatine kinase
- CK-MB: Myocardial-bound creatine kinase
- COPD: Chronic obstructive pulmonary disease
- Coro: Coronary angiogram/coronary angiography. (In most cases, “coro” also includes left ventricular angiography/angiogram.)
- CPK: Creatine phosphokinase
- CPR: Cardiopulmonary resuscitation
- CT: Computed tomography
- CX: Circumflexa (circumflex branch of the left coronary artery)
- DC: Direct current
- DD: Differential diagnosis
DDD  Dual-chamber dual-inhibited (pacing)
DDD(R)  Dual-chamber dual-inhibited rate responsive (pacing)
ECG  Electrocardiogram
Echo  Echocardiogram/echocardiography (in most cases color Doppler is integrated)
EF  Ejection fraction (in most cases of the left ventricle)
EPI/EPS  Electrophysiologic investigation/study
HOCM  Hypertrophic obstructive cardiomyopathy
Htx  Xeno-transplantation of the heart
ICD  Implantable cardioverter defibrillator
INR  International normalized ratio (for oral anticoagulation)
LA  Left atrium/left atrial
LAD  Left anterior descending coronary artery = left anterior descending branch of the left coronary artery
LAD  Left axis deviation (ÅQRSF < −30°)
LAFB  Left anterior fascicular block (= left anterior “hemiblock”)
LBBB  (Complete) left bundle-branch block
LCA  Left coronary artery
LPFB  Left posterior fascicular block (= left posterior “hemiblock”)
LV  Left ventricle/left ventricular
LVH  Left ventricular hypertrophy
MET  Metabolic equivalents
MET  Maximal exercise test
MI  Myocardial infarction
MAS attack  Morgagni-Adams-Stokes attack
MRI  Magnetic resonance imaging
NSAID  Nonsteroidal antiinflammatory drug
PA  Pulmonary artery
PE  Pulmonary embolism
PET  Positron emission tomography
PJRT  Permanent junctional reciprocating tachycardia
PTCA  Percutaneous coronary transluminal angioplasty
RA  Right atrium/right atrial
RBBB  (Complete) right bundle-branch block
RCA  Right coronary artery
RV  Right ventricle/right ventricular
RVD  Right ventricular dysplasia
RVOT  Right ventricular outflow tract
SA  Sinoatrial
SACT  Sinoatrial conduction time
SN  Sinus node
SNRT  Sinus node recovery time
SPECT  Single photon emission computed tomography
SR  Sinus rhythm
SVPB  Supraventricular premature beat
SVT  Supraventricular tachycardia
SVTab  Supraventricular tachycardia with aberration
VPB  Ventricular premature beat
VSD  Ventricular septal defect
VT  Ventricular tachycardia
VVI  One-chamber ventricular (pacemaker)
VVI(R)  Ventricular inhibited ventricular rate-responsive (pacing)
WPW syndrome  Wolff-Parkinson-White syndrome
Chapter 1
Some Theoretic Aspects

Because every physician, and even more so, every student in medicine, is aware of the important theoretic basics of Electrocardiology [as anatomy and the impulse conduction system, correlation between ion flows and the intracellular action potential (AP) of a single myocardial working cell, lead systems, and the nomenclature of the heart cycle], repetition in this short ECG Manual is renounced. Only some lesser-known basics are provided. Moreover, the reader will find a section about the determination of the frontal QRS axis (AQRS).

Atrial vectors are discussed in Chapter 3, “The Normal Electrocardiogram and Its (Normal) Variants.” The ventricular vectors, however, are discussed in this chapter, and a simplified scheme of the left ventricular vectors is provided that is especially useful in understanding bundle-branch blocks and the fact that the human electrocardiogram (ECG) generally represents a “levogram.”

Differences Among Heart Cells

In Figure 1.1a–c, the main differences of AP among (a) a working cell, (b) a conduction cell, and (c) a sinus node cell are demonstrated. In working fibers, phase 4 of the AP remains stable and isoelectric, respectively (Figure 1.1a). In contrast, conduction fibers have a slow depolarization during phase 4, called slow spontaneous phase 4 (diastolic) depolarization. This also represents an inherent characteristic of a pacemaker cell and explains the potential capacity of a conduction cell to act as pacemaker cell. If the cell is not depolarized by an electrical stimulus before reaching the threshold at the level of approximately −70 mV, it spontaneously depolarizes (Figure 1.1b). This fact is important for the understanding of arrhythmia, e.g., in premature and escape beats and rhythms.

A ventricular premature beat is generated by a diseased Purkinje cell (or a group of fibers) that shows a faster “spontaneous phase 4 depolarization” than the sinus node. Thus, the premature beat falls in too early (as the term describes it), disturbing the normal rhythm.

In contrast, an escape beat falls in too late, visually. For example, in the case of complete infrahisian atrioventricular block, asystole would occur, without a
Some Theoretic Aspects

Figure 1.1  

a. Action potential (AP) of a single working heart muscle cell.  
b. AP of a single conduction cell.  
c. AP of a single sinus node cell

ventricular escape beat (or rhythm). Because no electric stimulus reaches the Purkinje fibers, their “spontaneous phase 4 depolarization potential” reaches the threshold and produces an AP, an ordinary depolarization. The Purkinje fibers substitute the absent rhythm in a beneficial manner, at a lower rate.

As mentioned, the shape of the AP between conduction cells and pacemaker cells is not principally different. However, a relatively fast “slow spontaneous phase 4 depolarization,” associated with a short AP (both resulting in a relative high rate), allows the pacemaker fibers to depolarize first. In normal conditions, the fibers of the sinus node have the shortest AP, thus dominating the heart rhythm (Figure 1.1c).

Ventricular Vectors and Their Simplification

A vector is a theoretic model for an electric force. The different types are P vectors, QRS vectors, ST vectors, and T vectors. The concept of vectors, especially of the QRS vectors, with their amplitude and their directions in all three dimensions, is of
great importance for understanding the scalar ECG, in normal and some pathologic conditions (such as bundle-branch blocks, fascicular blocks, left and right ventricular hypertrophy, and myocardial infarction).

**Simplified QRS Vectors**

The instantaneous vectorial interpretation, described here and later presented in a simplified manner, also considerably facilitates memorization of important ECG patterns (Figure 1.2). In normal conditions [and pathologic conditions, with the exclusion of left bundle-branch block (LBBB) and corrected transposition of the great arteries], ventricular excitation begins in the middle part of the interventricular septum on the left side and spreads out throughout the septum, from left to right. This first QRS vector (or septal vector), vector 1, lasts approximately 15 ms, is generally directed to the right, anteriorly and slightly downward, and corresponds to the small Q wave in leads I and V5/V6. In other leads (for instance in V1/V2), the same vector leads to a small R wave, because of projection.

Afterward, the apical part of the left ventricle is depolarized, followed by the excitation of the main portions of the left (and right) ventricle. The great second QRS vector, vector 2, lasts approximately 60 ms, is generally directed to the left, inferiorly and mostly slightly posteriorly, and corresponds to the tall R waves in leads I and V5/V6 and the deep S waves in V2/V3. The great left ventricular main vector completely swallows the small simultaneous right ventricular, vector 2a, produced by the depolarization of the right ventricle, with its muscle mass 15 times

![Figure 1.2 Simplified QRS vectors](image-url)
smaller than the left ventricle muscle mass. In regard to ventricular depolarization (and repolarization), the human ECG represents a “levogram.” Right ventricular activation is only visible in the ECG in conditions that increase the right ventricle vector (in right ventricular hypertrophy) or delay right ventricle excitation [in right bundle-branch block (RBBB)].

The remaining small upper ventricular parts (of the high lateral wall of the left and right ventricle and the upper part of the septum) are excited last. The third small QRS vector, vector 3, lasts approximately 15 ms, points generally superiorly, to the right and posteriorly and leads in the ECG to the small S wave in lead I/V6 and to the last part of the S wave in lead V2/V3.

The QRS configuration in an ECG lead depends on the variations of the frontal QRS axis (the variations in the horizontal plane are of minor degree), and on the projection of the three above-mentioned ventricular vectors on the different ECG leads in the frontal and horizontal plane. A QRS complex may also be an RS complex, a QS complex, or a simple R wave, and so on.

**ST and T Alterations in the Different Stages of Ischemia**

The nomenclature of the different grades of ischemia is shown in Figure 1.3. The expressions for ischemia used below represent electrocardiographic terms, and include the different grades of hypoxia of (mostly left) ventricular myocardium.

The slightest grade of ischemia manifests as high and peaked T waves and is called *subendocardial ischemia*. It has the same morphologic alteration found in moderate hyperkalemia. A higher grade of ischemia leads to the pattern of symmetric negative T waves. The term *subepicardial ischemia* is sometimes used. This same alteration is found in many conditions other than ischemia (see Chapter 17, “Alterations of Repolarization”). An even higher grade of ischemia leads to depression of the ST segment and is called *subendocardial lesion*. This alteration is also rather nonspecific and is seen in conditions such as left ventricular hypertrophy and in patients receiving digitalis. ST depression is the best marker for ischemia during exercise.

These three grades of ischemia are reversible in many patients. The highest grade leads to extensive elevation of the ST segment (monophasic deformation) and is called *transmural lesion* or *transmural injury*. This ECG pattern is only reversible in the case of vasospastic angina (Prinzmetal angina) and in other rare conditions. In approximately 99% of transmural lesions, ischemia persists and myocardial infarction (necrosis) develops, with the appearance of new Q waves. Minor degrees of ST elevation are seen in pericarditis, early repolarization, and other conditions, all in the absence of true ischemia (see Chapter 17).

It is important to mention that, theoretically, in all grades of electrocardiographic ischemia, the grade is highest in the subendocardial layers of the (left) ventricular myocardium.
Determination of the Mean Frontal QRS Axis

The triangle of Einthoven, shown in Figure 1.4 (in Figure 1.5 integrated in the circle of Cabrera), is the basis for the calculation of $\Delta QRS_F$. Einthoven’s triangle and Cabrera’s circle represent a reasonable arbitrarily determined construction of a system of frontal leads and of degrees, subdivided in positive and negative degrees, where the cardiac vectors, especially the direction of the $\Delta QRS_F$ complex, can be placed.

In the German literature, the following inexact nomenclature is frequently used:

$\Delta QRS_F$ more positive than $+120^\circ$ = Überdrehte (QRS) Rechtslage
$\Delta QRS_F$ between $+120^\circ$ and $+90^\circ$ = Rechtslage
$\Delta QRS_F$ between $+90^\circ$ and $+60^\circ$ = Steillage
$\Delta QRS_F$ between $+60^\circ$ and $+30^\circ$ = Mittellage or Indifferenzlage
$\Delta QRS_F$ between $+30^\circ$ and $-30^\circ$ = Linkslage
$\Delta QRS_F$ less than $-30^\circ$ (more negative than $-30^\circ$) = Überdrehte Linkslage
Figure 1.4 Einthoven’s triangle

Figure 1.5 Cabrera’s circle
In the English literature, only left axis deviation (more negative than −30°; this means between −30° and ±150°) and right axis deviation (more positive than +120°; this means between +120° and ±150°) are clearly defined. A vertical electric frontal QRS axis is approximately +90°, and a left frontal QRS approximately 0°.

In any case, it is more exact to calculate the ÅQRS$_F$ with a precision of approximately ±10°. This can be performed within several seconds, after some practice. It is important to mention that we always consider the plane of the positive and negative portions of QRS and not the amplitude.

Examples

Example 1 (Figure 1.6a)
The QRS in lead aVF is isoelectric. This means that the ÅQRS$_F$ is perpendicular (at an angle of 90°) to aVF. Therefore, ÅQRS$_F$ is +90° or −90°. Lead I shows a positive QRS, so ÅQRS$_F$ is 0°. Looking for an overall isoelectric QRS is the fastest method for determining ÅQRS$_F$.

Example 2 (Figure 1.6b)
In lead I, the QRS is slightly more positive than negative, in aVL, the QRS is a bit more negative than positive, and leads II and aVF show almost the same QRS configuration. ÅQRS$_F$ is calculated at approximately +80°.

Example 3 (Figure 1.6c)
QRS in I is a bit more negative than positive, but significantly more negative in aVL. The QRS is more positive than negative in II and aVF, but almost purely positive in III and nearly isoelectric in aVR. ÅQRS$_F$ is approximately +120°.

Example 4 (Figure 1.6d)
QRS is more positive in III and aVF than in II, more negative than positive in I, completely negative in aVL, and only slightly positive in aVR. ÅQRS$_F$ is approximately +110°.

In calculating the ÅQRS$_F$ in the presence of complete RBBB, the mean frontal left ventricular QRS vector is always. The right ventricular mean frontal QRS vector does not (or at best by some degrees) influence the ÅQRS$_F$ except in the presence of considerable right ventricle hypertrophy or in the presence of RBBB. In right ventricular hypertrophy, we can generally only estimate the influence on ÅQRS$_F$. In RBBB, the activation of the left and right ventricle occurs practically one after the other. Therefore, we do not consider the whole QRS but only the first 60–70ms, so we get an approximate calculation of the ÅQRS$_F$. This is not always easy.

In LBBB, any calculation of the ÅQRS$_F$ is misleading and useless. An LBBB deforms the entire ventricular excitation so excessively that the amount of ÅQRS$_F$ that would be present without LBBB cannot be determined. We have made bad calculations ourselves, in contrast to a calculated ÅQRS$_F$ without LBBB.
In conclusion, the reader will soon recognize that $\hat{\text{AQ}RS}_F$ in the Einthoven leads (I, II, III) does not fully coincide with $\hat{\text{AQ}RS}_F$ in the Goldberger leads (aVR, aVL, aVF) and may differ between 5° and 15°. This means that $\hat{\text{AQ}RS}_F$ is always an approximate value. Moreover, Einthoven’s triangle is in fact not an isosceles triangle but one with a shorter branch above, and with two longer branches, directed slightly to the left, according to the mean frontal heart position (Figure 1.7).
Figure 1.7  Original and modified Einthoven’s triangle
Chapter 2
Practical Approach

There are beginners in electrocardiogram (ECG) analysis who are fascinated by a special pattern (e.g., a bundle-branch block or a striking Q wave) and thereby overlook other abnormalities. The best way to avoid similar errors is to analyze an ECG systematically, step by step. However, for experienced ECG interpreters, loss of concentration can also occur for a variety of reasons (see Short Story).

Short Story

About 10 years ago, an ECG with obvious preexcitation (ECG 2.1), diagnosable by any dentist, was shown to the author by a very good-looking young colleague. The author’s concentration was focused on the “wrong” subject and he interpreted the ECG as being normal.

Practical Approach

The practical approach includes:

- Analysis of rhythm
- Morphologic analysis of p, QRS, ST, and T (U) waves. The measurements of the PQ interval and of the QT (QTc) interval are included
- Definitive ECG diagnosis
1. Analysis of rhythm

Step 1: Rhythm regular or irregular?
   a. Regular: in most cases, normal SR
      Pathologic regular rhythms: escape rhythms; some forms of supraventricular tachycardias; VT
   b. Irregular: the most frequent cause of irregularity is regular SR with supraventricular and ventricular premature beats. Complete irregularity of the R-R intervals: atrial fibrillation

Step 2: Normal (sinusal p) wave present? → SR. If not:
   a. Abnormal (nonsinusal) p waves present: atrial rhythm
   b. No p waves: AV junctional rhythm
   c. Replacement of p waves by other atrial waves: atrial flutter or atrial fibrillation
1. Analysis of rhythm (continued)

Step 3: Rate (of the ventricles)? Eventually rate of the abnormal (nonsinusal) p waves or flutter waves?

Step 4: PQ interval? If we measure the PQ interval, we will not only recognize a prolongation or shortening of the PQ time, but also the following:
   a. every p is conducted, or not every p wave is conducted: in the 3 forms of AV block 2°
   b. no p wave is conducted. This means that atria and ventricles are working independently from each other, in the presence of AV block 3° = complete AV block
   c. p waves are twisting around the QRS complexes: in the special forms of AV dissociation

Step 5: QRS duration normal (≤90 ms) or prolonged?
   QRS ≥120 ms: pattern of BBB
   a. Supraventricular rhythm/tachycardia with aberration.  
   b. Ventricular origin of the rhythm (with AV dissociation):
      • Low rate: ventricular escape rhythm
      • Medium rate: accelerated idioventricular rhythm
      • High rate: VT

Note: Typical pathologic findings are italicized.
AV, atrioventricular; BBB, bundle-branch block; SR, sinus rhythm; VT, ventricular tachycardia.

2. Detailed analysis of morphology

Step 1: P

1. Normal (sinusal)? (p duration 90–110 ms)
   Note: A negative p in lead I and (often) a positive p in lead aVR means “false poling” of the upper limb leads in 99% of the cases

2. Pathologic p waves
   a. p duration ≥110 ms, accentuated terminal negativity in lead V1: Left atrial enlargement
   b. p voltage ≥2.5 mm in leads III and aVF: Right atrial enlargement
   c. Summation of 2a and 2b: Bialtrial enlargement

Step 2: QRS

1. Frontal QRS axis = ÄQRSF? (DD of different ÄQRSF values, see Chapter 3)
2. Broad QRS?
   a. Typical configuration for aberration: RBBB (QRS ≥120 ms) or LBBB (≥140 ms). More or less typical BBB (≥160 ms): suspicious for severe hyperkalemia.
   b. Typical pattern of bilateral BBB (RBBB + LAFB or RBBB + LPFB)
   c. Atypical BBB-like configuration (QRS ≥140 ms): suspicious for ventricular origin of rhythm, generally with AV dissociation

3. (Formally) pathologic Q or QS waves?
   a. Typical for old MI? (combined with symmetric negative T waves; typical history; risk factors for CHD)
   b. Atypical for old MI? (combined with asymmetric discordant T waves; atypical history; no risk factors for CHD)

DD:
   • Artifact: Q/QS in lead I; R/qR in lead aVR—false poling of limb leads (DD: situs inversus)
   • Normal variant: QS in lead III (QIII) —attributable to projection
   • LVH
   • Preexcitation (QS in III, aVF)
   • Hypertrophic (obstructive) cardiomyopathy
   • LBBB (QS in III, aVF, V1 to V4 with duration ≥140 ms)

(continued)
2. Detailed analysis of morphology (continued)

4. Signs of \textit{LVH or RVH}? (Chapters 5 and 6)
5. Signs of \textit{LAFB or LPFB}? (Chapter 9)
6. \textit{Presence of delta wave}? (with shortened PQ: preexcitation)
7. Presence of notching/slurring? DD: intraventricular conduction disturbance versus \textit{normal variant}
   7a. Normal variant (Chapter 3)
   7b. \textit{Pathologic, e.g., in old MI or left fascicular block} (Chapters 13 and 9, respectively)

Step 3: \textbf{ST}
1. ST elevation?
   1a. Normal variants: ST (in $V_2/V_3$), early repolarization (Chapter 3)
   1b. Pathologic:
      • Typical for \textit{acute MI}: consider other findings; symptoms, history, risk factors for CHD (Chapter 13).
      • Typical for \textit{acute pericarditis}: frontal ST vector about $+70^\circ$—\textit{ST} elevations in leads aVF, II, and I (Chapter 15).
      • Typical for \textit{mirror image of ST depression}; e.g., in LVH/systolic LV overload.
2. ST depression?
   2a. \textit{Ischemic}
   2b. \textit{LVH/LV overload}
   2c. \textit{Related to BBB or other conditions} (Chapter 17)

Step 4: \textbf{T (and U)}
1. Asymmetric T negativity?
   1a. Normal in lead $V_1$; normal in vertical $\dot{A}QRS_F$ in aVF, III(II); normal in left $\dot{A}QRS_F$ in aVL.
   1b. Pathologic in \textit{LVH/LV overload}; \textit{preexcitation}; \textit{BBB}
2. Symmetric T negativity?
   2a. \textit{Often ischemic}, but extensive DD
   2b. \textit{Later stage of pericarditis}; \textit{LVH/LV overload}; \textit{acute pancreatitis}; \textit{drugs}; \textit{others}
   3. High and symmetric T
   3a. \textit{Ischemia} (rare, because short-lasting)
   3b. \textit{Hyperkalemia}
   4. U negativity?
   4b. \textit{Ischemic}; other conditions

Step 5: \textbf{QT}
1. QT prolonged
   1a. \textit{Long QT syndromes}
   1b. \textit{Hypocalcemia}
2. QT shortened: \textit{hypercalcemia}
3. Fusion of T and U: \textit{hypokalemia, long QT syndromes}

Step 6: \textbf{Definitive diagnosis}

\textit{Note}: Typical pathologic findings are italicized.

DD, differential diagnosis; $\dot{A}QRS_F$, frontal QRS axis; AV, atrioventricular; BBB, bundle-branch block; CHD, coronary heart disease; LAFB, left anterior fascicular block; LPFB, left posterior fascicular block; LBBB, left bundle-branch block; RBBB, right bundle-branch block; LV, left ventricle; LVH, left ventricular hypertrophy; MI, myocardial infarction.
### Definitive Electrocardiogram Diagnosis

Take the important normal and pathologic findings from the analysis above and put them into the following scheme:

Note: As mentioned above, an ECG must be interpreted in context with the clinical findings of a patient. Therefore, in this book, age and gender and the clinical diagnosis of the patient are provided for many ECG examples in the text.

<table>
<thead>
<tr>
<th></th>
<th>Example 1</th>
<th>Example 2</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Rhythm/rate</strong></td>
<td>SR, 72 beats/min</td>
<td><em>Atrial fibrillation</em>, medium rate 90 beats/min (maximum 140 beats/min, minimum 40 beats/min)</td>
</tr>
<tr>
<td>p</td>
<td>Normal</td>
<td>–</td>
</tr>
<tr>
<td>PQ</td>
<td>Normal (0.16 s)</td>
<td>–</td>
</tr>
<tr>
<td>ÅQRS&lt;sub&gt;F&lt;/sub&gt;</td>
<td>(+80°)</td>
<td>LAD (−60°): LAFB</td>
</tr>
<tr>
<td>ST</td>
<td>Normal (elevation in V&lt;sub&gt;L&lt;/sub&gt;/V&lt;sub&gt;s&lt;/sub&gt;)</td>
<td>0.12 s, LVH</td>
</tr>
<tr>
<td>T</td>
<td>Normal (negative in III)</td>
<td>Idem</td>
</tr>
<tr>
<td>QT</td>
<td>Normal</td>
<td>Prolonged?</td>
</tr>
<tr>
<td>Special remarks</td>
<td>–</td>
<td><em>Fusion of T and U</em></td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Normal ECG</td>
<td><em>Atrial fibrillation, LAFB, LVH, Hypokalemia?</em></td>
</tr>
</tbody>
</table>

*Note:* Typical pathologic findings are italicized.

LAD, left axis deviation; LAFB, left anterior fascicular block; LVH, left ventricular hypertrophy; SR, sinus rhythm.
Knowledge about the normal electrocardiogram (ECG) and its (normal) variants is enormously important because, when a physician looks at a new ECG, he or she automatically compares it with the normal ECGs stored visually in the posterior part and intellectually in the frontal part of the cerebrum. Therefore, in this chapter, normal components of the ECG and its variants are discussed so that the reader can make accurate comparisons.

**Components of the Normal Electrocardiogram**

**Sinus Rhythm**

The sinus node produces the normal rhythm of the whole heart, resulting in a p wave with the typical atrial vectors (Figure 3.1a and b). Because the sinus node is localized in the right atrium (high laterally at the entry of the superior vena cava), the right atrium is activated first and the left atrium approximately 20 ms later, resulting in a fusion of two p waves, both forming the normal p wave. Its duration is 0.09–0.11 s, best measured in lead II where the beginning and the end of the p wave are clearly visible in most cases.

In the frontal plane, the p wave is positive on all leads except in aVL (where it is negative or biphasic +/−) and in, III, where it may be biphasic +/−. In the horizontal plane, the p wave is only biphasic in V₃ (because the left atrial vector is directed posteriorly and to the left), with a longer part of the (first) positive component than the (second) negative part. In all other precordial leads (V₂ to V₆), the normal p wave is positive. The normal rate of sinus rhythm is 60–100 beats/min (or perhaps better, 50–90 beats/min) (see ECG 3.1). Rates higher than 90–100 beats/min are called sinus tachycardia (normal in emotion and on exercise) and rates less than 50–60 beats/min are called sinus bradycardia (normal in many individuals at rest, especially athletes).

Alterations of the normal p wave are discussed in Chapter 4, “Atrial Enlargement and Other Abnormalities of the p Wave” and in the chapters about arrhythmias.
Figure 3.1  a. Normal atrial vectors and corresponding p waves in the frontal plane. b. Normal atrial vectors and corresponding p waves in the horizontal plane. RAV, right atrial vector; LAV, left atrial vector; pV, p vector
**PQ Interval**

The normal PQ interval measures 0.13–0.20 s (in sinus bradycardia up to 0.21 s) and is determined from the beginning of the P wave to the beginning of the QRS complex.

Prolonged PQ intervals (≥ 0.21 s) were found in 8% of males and 12% of females. In young, healthy students, a 2° atrioventricular block of the Wenckebach type (see Chapter 12, “Atrioventricular Block and Atrioventricular Dissociation”) was detected in 6% of males and 4% of females.
QRS Complex

The QRS complex is quite variable in the frontal plane and largely dependent on the individual’s age. Table 3.1 shows the common frontal QRS axis ($ÅQRS_F$) for approximately 70% of normal individuals. ECG 3.2a–g shows the typical $ÅQRS_F$ for a variety of ages. A sudden change of $ÅQRS_F$ to the left is rare (perhaps occurring in inferior infarction or in a new left anterior fascicular block), but a sudden change of $ÅQRS_F$ to the right can be seen in acute pulmonary embolism.

ECG 3.2  a–d Different frontal QRS axis ($ÅQRS_F$) values by age. a. 18-year-old, $ÅQRS_F +80°$. b. 25-year-old, $ÅQRS_F +75°$. c. 40-year-old, $ÅQRS_F +30°$. d. 54-year-old, $ÅQRS_F +20°$
In contrast to the frontal plane in the horizontal plane, the QRS complex generally is characterized by a striking uniformity. In leads V₁ and V₂, we find an rS complex, a small r and a deep S, especially in V₂. In lead V₁, the transition zone begins—a change from a predominantly negative to a predominantly positive QRS complex. Therefore, in lead V₃, the R wave can have almost the same amplitude as the S wave. From V₄ to V₆, the QRS is positive, with an Rs complex; leads V₃ and V₆ often have a small initial deflection, forming a qRs complex.

ECG 3.2 (continued) e. 60-year-old, ÅQRSₚ 0°. f. 73-year-old, ÅQRSₚ –20°. g. 25-year-old, ÅQRSₚ not determinable. The positive and negative components of the QRS complex have almost the same amplitude in the individual limb leads. This frontal QRS axis is called sagittal axis.