AN INTRODUCTION TO

ELECTROCARDIOGRAPHY

by LEO SCHAMROTH

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FRCP (Glasg), FACC, FRS (SAf)

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SIXTH EDITION

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Foreword

Dr Schamroth publishes this little book on modern electrocardiography at the insistent demand of students and graduates whom he has instructed, and who consider that he should make available for general circulation a method of teaching electrocardiography applicable to clinical practice which is simple, revealing and based upon sound electro-physiological principles.

Simply written, and amply illustrated with many meaningful diagrams, Dr Schamroth's book is a contribution to the rational appreciation and interpretation of electrocardiographic abnormalities by students and practitioners whose training in the physiological aspects of cardiac action has been no more profound than is provided by the undergraduate curriculum.

Present-day literature on electrocardiography is forbidding in its complexity for anyone who has not had specialized training. This book is a welcome addition to electrocardiographic literature, because by its simplicity and correctness it invites understanding.

G. A. Elliott
Professor of Medicine
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South Africa

Preface to the Sixth Edition

The basic design of this book remains essentially the same as that of the previous editions. It is directed primarily at the beginner, and its aim is simplicity. The emphasis remains on deductive rather than empirical electrocardiographic interpretation.

The whole text has been appreciably revised. New sections have been added on: ventricular fusion complexes, structural nodal disease—the so-called sick sinus syndrome, reciprocal rhythms, the supraventricular tachycardias, the Q–T interval and technical standardization. Many of the diagrams and illustrative electrocardiograms have been replaced, and new ones added.

L. Schamroth

Johannesburg, January 1982

Contents

SECTION 2. DISORDERS OF IMPULSE FORMATION

SECTION 3. DISORDERS OF IMPULSE CONDUCTION

SECTION 4. THE SECONDARY DISORDERS OF RHYTHM

SECTION 5. CORRELATIVE ESSAY

Preface to the First Edition

This volume, it should be stated at the outset, makes no pretensions to be a complete or comprehensive treatise on electrocardiography, nor does it seek to supplant the major works on the subject. It is rather a stepping-stone to the fuller and more detailed study of a most important branch of medical science.

The student, introduced for the first time to the intricacies of electrocardiography, is frequently bewildered, sometimes overwhelmed, by complicated methods of presentation. It is the beginner who has been kept continuously in mind in the writing of this book, and the primary object throughout has been to give him a working knowledge of the subject. Consequently, a certain amount of licence has been taken with a view to clarifying the various processes. Theoretical considerations have been reduced to a minimum, emphasis being placed on the practical aspects. The text has been illustrated as profusely as possible with sketches, a clear drawing invariably being worth pages of script.

Clarity of presentation has thus been the author's aim; if he should succeed in dispersing a few clouds from his readers' minds, his efforts will not have been in vain.

L. Schamroth

Johannesburg, May 1956

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It is my pleasure to express my thanks to:

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PART I
ABNORMALITIES OF THE P-QRS-T AND U DEFLECTIONS

Basic Principles
Myocardial Death, Injury and Ischaemia
Bundle Branch Block
Ventricular Hypertrophy
Drug and Electrolyte Effect
The P Wave: Atrial Activation
The Electrical Axis
Hypothermia
Electrical Alternans
The Q–T Interval

Chapter 1
Basic Principles

THE ACTION OF THE GALVANOMETER

When an electrical impulse flows **towards** a unipolar electrode, or the positive electrode of a bipolar lead, the galvanometer will record a **positive** or upward deflection (Diagram A of Fig. 1).

When an electrical impulse flows **away** from a unipolar electrode, or **away** from the positive electrode of a bipolar lead, i.e. towards the negative pole of a bipolar electrode, the galvanometer will record a **negative** or downward deflection (Diagram B of Fig. 1).

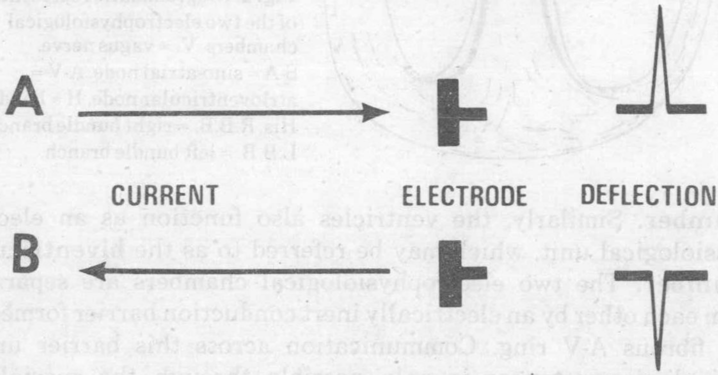

Fig. 1. Diagrams illustrating the effect of current direction on the positive electrodes of a galvanometer.

THE 'TWO CHAMBER' CONCEPT

It is stating the obvious to say that the heart is a four-chambered organ. It is not often appreciated, however, that, in an electrophysiological sense, the heart consists of only two chambers: one formed by the atria, and the other formed by the ventricles (Fig. 2). The two atria function as a single electrophysiological chamber—an electrophysiological unit: there is no electrical boundary between them, and both are activated by a single activation process. This functional electrophysiological unit may be referred to as the **bi-atrial**

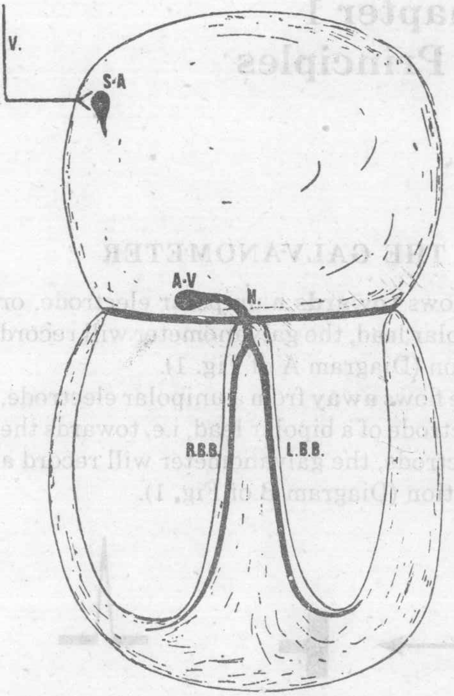

Fig. 2. Diagrammatic representation of the two electrophysiological chambers. V. = vagus nerve. S-A = sino-atrial node. A-V = atrioventricular node. H = bundle of His. R.B.B. = right bundle branch. L.B.B. = left bundle branch.

chamber. Similarly, the ventricles also function as an electrophysiological unit, which may be referred to as the **biventricular chamber.** The two electrophysiological chambers are separated from each other by an electrically inert conduction barrier formed by the fibrous A-V ring. Communication across this barrier under normal circumstances is only possible through the specialized conducting system formed by the A-V node, the bundle of His, the bundle branches and their ramifications.

THE DOMINANCE OF THE LEFT VENTRICLE

The free wall of the left ventricle, and the interventricular septum have relatively thick walls (large muscle masses) and together constitute a uniform ring of muscle or chamber—the anatomical left ventricle (Fig. 3). It is quite evident from a cross-section of the ventricles that the interventricular septum and the free wall of the left ventricle constitute an anatomical continuum (Fig. 3). The free wall of the right ventricle is, in effect, merely a thin anatomical appendage of the left ventricle.

The interventricular septum also contracts functionally with the free wall of the left ventricle, constituting the main haemodynamic pump of the heart. The right ventricle functions principally as a conduit.

From the electrocardiological viewpoint, the left ventricle is also the dominant chamber. For example, anterior wall myocardial infarction refers principally to infarction of the interventricular septum. The interventricular septum thus, in effect, constitutes the 'electrical' anterior wall of the biventricular chamber, whereas the thin free wall of the right ventricle constitutes the anatomical anterior wall of the biventricular chamber.

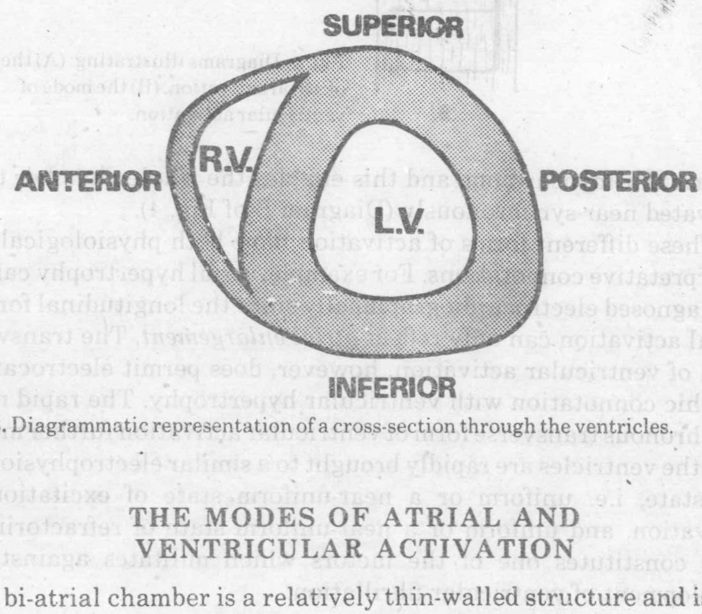

Fig. 3. Diagrammatic representation of a cross-section through the ventricles.

THE MODES OF ATRIAL AND VENTRICULAR ACTIVATION

The bi-atrial chamber is a relatively thin-walled structure and is not equipped with the highly specialized conducting system of the ventricles. Activation of the bi-atrial chamber therefore occurs **longitudinally** and by **contiguity,** spreading from its point of origin in the S-A node to engulf the whole chamber, each fibre in turn activating the adjacent fibre (Diagram A of Fig. 4).

Activation of the ventricles is effected through the specialized and highly efficient conducting system which transmits the supraventricular impulse very rapidly to all the endocardial regions of the chamber. The muscle is then activated from endocardial to epicardial surfaces through the terminal ramifications of the conducting system. Excitation therefore occurs **transversely** through the

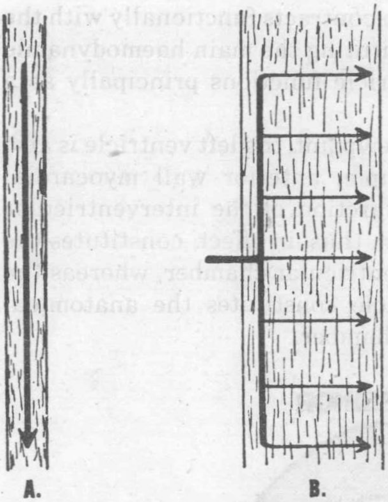

Fig. 4. Diagrams illustrating: (A) the mode of atrial activation. (B) the mode of ventricular activation.

ventricular myocardium, and this enables the whole chamber to be activated near-synchronously (Diagram B of Fig. 4).

These different forms of activation have both physiological and interpretative connotations. For example, atrial hypertrophy cannot be diagnosed electrocardiographically since the longitudinal form of atrial activation can only reflect *atrial enlargement*. The transverse form of ventricular activation, however, does permit electrocardiographic connotation with ventricular hypertrophy. The rapid near-synchronous transverse form of ventricular activation further means that the ventricles are rapidly brought to a similar electrophysiological state, i.e. uniform or a near-uniform state of excitation or activation, and uniform or a near-uniform state of refractoriness. This constitutes one of the factors which militates against the development of ventricular fibrillation.

THE NOMENCLATURE OF THE ELECTROCARDIOGRAPHIC DEFLECTIONS

The electrocardiographic deflections are arbitrarily and sequentially named P, QRS, T and U. The P wave reflects atrial activation. The QRS complex reflects ventricular activation. The T wave reflects ventricular recovery. The genesis of the U wave is still controversial.

Note: An initial downward deflection after the P wave is termed a *q* wave. An initial upward deflection after the P wave is termed an *r* wave. The ensuing deflections are named by the succeeding alphabetical letters. Large, or relatively large, deflections are labelled with capital or upper-case alphabetic letters; small, or relatively small, deflections are labelled with non-capital, small or lower-case letters. Thus, a deep wide Q wave followed by a small r wave—as illustrated by Diagram C of Fig. 22—is labelled a Qr complex. A totally negative QRS deflection without ensuing positivity—as illustrated by Diagram D of Fig. 22—is labelled a QS complex. A small q wave followed by a tall or relatively tall R wave—as illustrated by Diagram A of Fig. 22—is labelled a qR complex. A small initial r wave followed by a deep or relatively deep S wave—as illustrated by lead V1 in Fig. 8—is labelled an rS complex. When the QRS complex reflects two positivities as occurs, for example, in right bundle branch block, the second positivity is termed an R′, for example an rSR′, an RsR′, or an RR′ complex.

ACTIVATION OF THE VENTRICLES

In an electrocardiological sense, the ventricles are composed of three muscle masses: the interventricular septum, the free wall of the right ventricle (the right ventricular muscle mass), the free wall of the left ventricle (the left ventricular muscle mass) (Fig. 5).

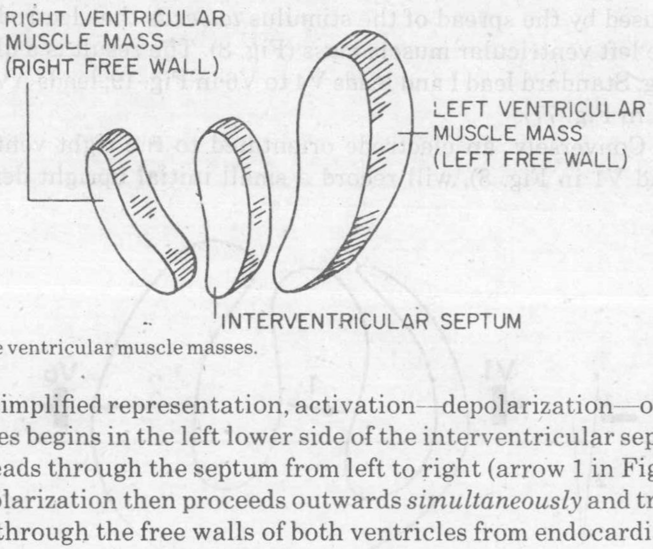

Fig. 5. The ventricular muscle masses.

In a simplified representation, activation—depolarization—of the ventricles begins in the left lower side of the interventricular septum and spreads through the septum from left to right (arrow 1 in Fig. 6).

Depolarization then proceeds outwards *simultaneously* and transversely through the free walls of both ventricles from endocardial to epicardial surfaces (arrows labelled 2 in Fig. 7).

The free wall of the left ventricle has a larger muscle mass—and hence a larger potential electrical force—than the free wall of the

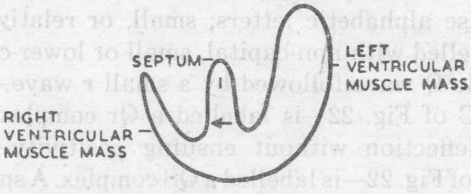

Fig. 6. First stage of depolarization.

Fig. 7. Second stage of depolarization.

right ventricle (Figs. 3 and 5). Consequently, as depolarization of both free walls occurs simultaneously, the larger left ventricular forces counteract the smaller right ventricular forces. The result is a single force directed from right to left (arrow 2 in Fig. 8). Thus, *for convenience*, depolarization of the ventricles may be represented in simplified form as a small initial force from left to right through the septum, followed by a larger force from right to left through the free wall of the left ventricle (Fig. 8). These forces have magnitude and direction and are thus vectors.

An electrode orientated to the left ventricle (e.g. lead V6 in Fig. 8). will record a small initial downward deflection (a small q wave) caused by the spread of the stimulus *away* from the electrode through the septum, followed by a larger upward deflection (a tall R wave) caused by the spread of the stimulus *towards* the electrode through the left ventricular muscle mass (Fig. 8). The result is a qR complex (e.g. Standard lead I and leads V4 to V6 in Fig. 19; leads AVL, V5 and V6 in Fig. 77).

Conversely, an electrode orientated to the right ventricle (e.g. lead V1 in Fig. 8), will record a small initial upright deflection (a

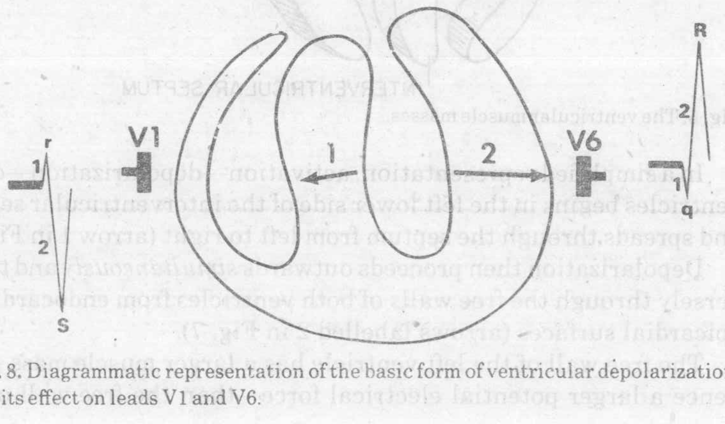

Fig. 8. Diagrammatic representation of the basic form of ventricular depolarization and its effect on leads V1 and V6.

small r wave) caused by the spread of the stimulus *towards* the electrode through the interventricular septum, followed by a larger downward deflection (a deep S wave) caused by the spread of the stimulus *away* from the electrode through the free wall of the left ventricular muscle mass (Fig. 8). The result is an rS complex (e.g. leads V1 and V2 in Fig. 19; leads V1 and V2 in Fig. 77).

NOMENCLATURE AND LOCATION OF THE ELECTRODE LEADS

Each electrocardiographic lead has a positive pole or electrode and a negative pole or electrode, which could theoretically be orientated in any relationship to the heart. By convention, however, there are 12 lead placements. These are:

Standard lead I.
Standard lead II.
Standard lead III.
Lead AVR.
Lead AVL.
Lead AVF.
Leads V1 to V6.

Standard leads I, II and III are bipolar leads (see Appendix and Chapter 7).

Leads AVR, AVL, AVF and V1 to V6 are unipolar leads (see Appendix and Chapter 7).

BASIC ORIENTATION OF THE LEADS

The 12 conventional leads may be divided electrophysiologically into two groups, one being orientated in the frontal plane of the body, and the other in the horizontal plane (see Chapter 7 and Fig. 116).

Standard leads I, II and III, and leads AVR, AVL and AVF are orientated in the **frontal** or **coronal plane** of the body.

The precordial leads—leads V1 to V6—are orientated in the **horizontal** or **transverse plane** of the body.

The frontal plane leads

Standard leads I, II and III

The orientation of the Standard leads is described in Chapter 7.