VECTORCARDIOGRAPHY

PHYSICAL BASES AND CLINICAL PRACTICE

J. BOUTKAN

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FOREWORD

There are many reasons for welcoming the appearance of the present book on vectorcardiography, still a fairly new diagnostic method.

In the first place it deals with the fundamentals of vectorcardiography, and this treatment can be described as "popular" in the best sense of the word. Here a medical man is talking to his colleagues in clear language, without at the same time giving offence to the physicist; on the contrary, the physicist will be satisfied with these explanations – and that, unfortunately, cannot always be said about other books on the subject.

The clinical section bears witness to the critical approach and great experience of the author. Vectorcardiograms in heart disease are discussed with reference to numerous examples and readily understandable diagrams. The physician, desirous of acquainting himself with the method, with a view to employing it himself, will find readable and trustworthy guidance in this part of the book.

It is to be hoped that the book will encourage many cardiologists to adopt vectorcardiography in addition to electrocardiography in their daily practice.

Utrecht, 1965

Prof. H. C. Burger

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CHAPTER 1

INTRODUCTION

Electrocardiography has found a permanent place in clinical work as an invaluable means of diagnosis. Nevertheless, those who are not in daily contact with this work will find it difficult to interpret an electrocardiogram and moreover, descriptions of electrocardiograms are often misunderstood. It is unfortunate that at a time when heart diseases are becoming increasingly prevalent, the results of what is properly an exact and objective method of examination are not capable of interpretation by the majority of medical men. Apart from electrocardiography, the now well developed technique of vectorcardiography is regarded by many as something new and even more difficult to understand. Our own experience has shown, however, that this is quite unfounded, since a vectorcardiogram in many cases simplifies interpretation of the electrical phenomena of the heart and moreover, may well lead to a better understanding of "conventional" electrocardiography.

These considerations have led to the preparation of a course in vectorcardiography, given at the Philips' Health Centre, and now presented here in the form of a monograph. The first part of this course deals with the fundamentals of vectorcardiography and electrocardiography, and with the relationship between the two methods. Emphasis has been laid on the general concepts of image space and lead vector, as these constitute a physically sound basis for the relationship between the ECG and the VCG.

The second part is mainly concerned with clinical practice.

The author would like to express his gratitude to Professor H. C. Burger for his helpful criticism of the treatment of physical subjects.

CHAPTER 2

ELECTROPHYSIOLOGY

In vectorcardiography and electrocardiography the electrical phenomena generated by the heart and conducted through the body are recorded on the external surface of the body, i.e. the skin. The ultimate form of the vectorcardiogram or electrocardiogram thus obtained is determined by the following factors:

1. The electrical phenomena taking place within the tissue of the heart muscle itself.
2. The conductivity, size and shape of the body in which the heart is functioning.
3. The conductivity of the blood in the cavities of the heart.

Now, in order to understand the manner in which the electrical phenomena in the heart muscle originate, it is necessary to know something about the electrophysiology of the muscle cell.

In a muscle cell or a muscle fibre of the myocardial syncytium we can distinguish a cell content and an enveloping membrane. As long as the cell is intact and is in the unactivated condition, no electrical phenomena are apparent at the surface of the cell. It is possible, however, to pass a microelectrode through the membrane to the interior of the cell (Fig. 1). By this means it has been found that the potential inside the cell is negative

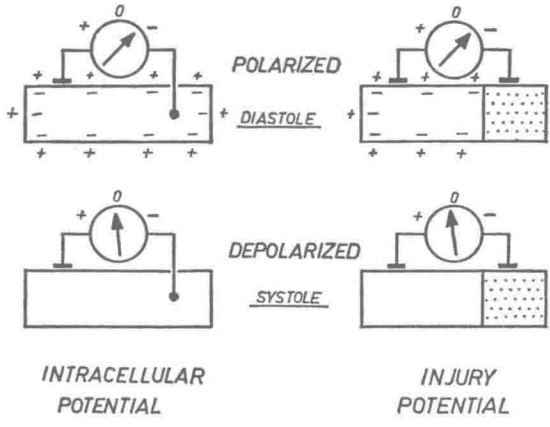

Fig. 1 Membrane potential. Upper diagrams: determination of intracellular negativity by means of intracellular microelectrode (*L*), and by means of an electrode placed over an injured part of the cell, indicated by dotted area. (*R*) Lower diagrams: after depolarization the potential difference has disappeared.

relative to the exterior of the cell membrane. Hence there is a potential difference between the interior and exterior of the cell membrane, which is called the *membrane potential.* A cell in this condition is said to be *polarized.* The membrane potential can be represented diagrammatically by placing a number of minus signs along the inner side of the membrane and a similar number of plus signs along the outer side, in the manner of an electrical double layer.

There is also another method of measuring the membrane potential. Part of an intact muscle cell is damaged, with the result that the membrane potential at that point becomes zero. A window is thus, as it were, provided into the interior of the cell. It is found that an electrode placed on the damaged portion of the cell records a negative potential relative to another electrode placed on the exterior of the intact portion of the cell, and this potential is accordingly termed the *injury potential.* Both methods thus demonstrate that the interior of the cell is negative relative to the exterior, i.e. in the case of the non-excited cell. In this non-excited condition the heart muscle is in diastole.

It appears that upon excitation of the cell, during systole, the membrane potential mentioned above suddenly disappears and that for a brief interval a potential difference of the opposite sign is produced; in other words the interior of the cell becomes positive relative to the exterior. On return to the state of rest the original membrane potential is restored; this phenomenon is called *repolarization.* When at rest, during diastole, the cell thus is in the *polarized state* again. The disappearance of the membrane potential on activation, during systole, is known as *depolarization,* the activated cell being then in the *depolarized state.*

This sequence of electrical phenomena can be presented diagramatically as an "electrogram" of an isolated heart muscle fibre, the potential difference changes between either side of the cell membrane during one cycle being shown. (Fig. 2). The base line showing the potential of the polarized cell in diastole, when the interior of the cell is negative relative to the

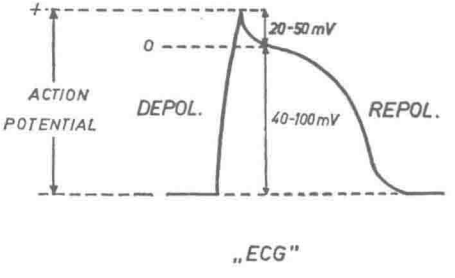

Fig. 2 Curve of action potential. Rapid depolarization curve with final overshoot of 20–50 mV, and much slower repolarization curve of characteristic shape.

surface of the cell, will therefore be below the electrical zero potential level. When the cell is activated, polarization disappears and the interior of the cell becomes positive relative to the surface. The result is a sharply rising line to a point above the electrical zero level. The return to the resting state, repolarization, is represented as a downward deflection to the original starting level. The repolarization does not take place as rapidly as depolarization and this downward deflection is accordingly less steep than the upward deflection.

The membrane potential in the resting state varies from 40 to 100 mV. The potential differences occurring on depolarization of the cell, the so-called *action potentials*, are 20 to 50 mV larger. This may be seen from that part of the curve which lies above the electrical zero level.

It should be noted, however, that the potential differences measured at the surface of the body when taking an ECG or VCG are very much smaller, namely only a few millivolts. Partly this is because at any instant electrical impulses in the myocardium are travelling along several paths and there is a tendency for them to cancel each other out when summated. Then again, the blood in the cavities of the heart is a conductive medium, as are the tissues outside the heart, and tends to act as a short circuit.

Needless to say, considerable research has been carried out in order to demonstrate the manner in which the electrical phenomena described above are produced. Obviously, the different ions surrounding the cell membrane have a direct bearing on all this. In fact, the ions both inside and outside the cell membrane are distributed in accordance with a certain pattern. The concentration of positively-charged potassium ions within the cell is much

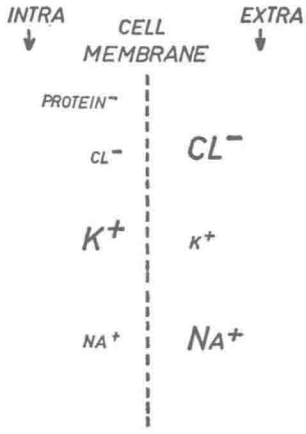

Fig. 3 Relative ion concentrations on either sides of the cellular membrane indicated by the size of the symbols.

higher than that outside the cell, whereas positively-charged sodium ions and negatively-charged chloride ions show a much higher concentration outside the cell than within (Fig. 3). It was originally thought that the potassium ion in particular played an active part, but in the light of modern methods of research, e.g. using radio-active tracers, it may be said that both the chloride and the potassium ions are able to pass through the cell membrane with little difficulty. If it were a question of simple diffusion the concentration of these ions on either side of the membrane would be equal.

In practice, however, the distribution of these ions indicates a mechanism which maintains a negative potential within the cell, causing the positive potassium ions to be drawn, as it were, into the cell, with the expulsion of chloride ions from within. The behaviour of the chloride and potassium ions is thereby wholly passive.

2.1 The sodium pump

In the process which maintains a negative potential within the cell the sodium ion plays an important part. The concentration of this ion is higher outside the cell membrane than inside and if simple diffusion were obtained, a state of equilibrium would result. It is assumed that a mechanism exists whereby the majority of the sodium ions passing into the cell are "smuggled" out again in a non-ionised state: this process is known as the *sodium pump* or *active transport* (Fig. 4). As the permeability of the cell membrane to sodium ions is relatively low (one-thirtieth of the permeability to potassium ions) the "smuggling" process can keep ahead of the inward flow of sodium ions, thus maintaining a low concentration in the cell. Outside the cell membrane the "smuggled" ions are liberated and are free to take part in the diffusion process again. The difference in the concentration of the positively charged

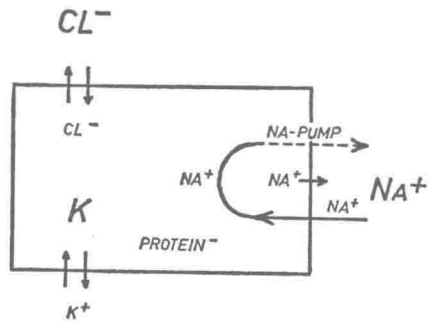

Fig. 4 The sodium pump. The dashed arrow indicates the non-ionic flux.

sodium ions maintained in this manner results in a negative potential of the interior of the cell relative to the outside.

Now this difference in potential has a direct bearing on the distribution of the potassium and chloride ions which, due to the intracellular negative state are respectively attracted and repulsed; the behaviour of these ions themselves is passive. Hence the difference in potential is reduced, but not entirely levelled out.

As the concentration of potassium ions rises within the cell and falls outside it, the "diffusion pressure" is increased in the outward direction, so that ultimately a distribution of these ions is produced such that the "diffusion pressure" and the attraction of the negative cell content are maintained in a compensated state. A similar theory applies to the chloride ions.

Note: The substances histamine and serotonine amongst others would appear to take part in the mechanism of the sodium pump; acetylcholine is also involved in varying the permeability of the cell membrane, with calcium ions as a regulating element.

The resting state of the polarized cell is destroyed immediately the cell is activated. Depolarization (activation) may be the result of the application of a negative charge which has the effect of reducing the positivity at the exterior of the cell, this being accompanied by a drop in the membrane potential. It may be assumed that this reduction in the membrane potential results in an increase in the permeability of the membrane to sodium ions, thus facilitating their passage into the cell; the inward flow is due to the higher concentration outside and the lower concentration within the cell. The consequence of this transport of sodium ions is a further drop in the membrane potential, with the ultimate result that the permeability of the membrane to sodium ions is increased by a factor of as much as 500 for a short time, i.e. to a level far beyond the permeability to potassium ions. The sodium ions are thus able to pass into the cell more freely than the potassium ions can be driven out; a surplus of positive ions is thus temporarily produced in the cell, the interior of which is then positively charged with respect to the exterior until such time as the equivalent number of potassium ions has passed out of the cell. The free influx of sodium ions counteracts the action of the sodium pump.

When depolarization is complete, the permeability to sodium ions is again lessened and the sodium pump resumes its function. As the sodium ions pass out of the cell, potassium ions are once more free to enter and this continues until the resting state, i.e. complete polarization is achieved (repolarization).

Polarization can be represented diagrammatically as a mechanical analogy in the manner shown in Figs. 5A and 5B, in which the potential is expressed as a pressure. The cell is depicted as an enclosed space. A pressure gauge indicates the pressure in the cell by analogy with the intracellular potential. The potassium and sodium ions are shown in balloons. Each has an intracellular and an extracellular balloon connected by ducts the diameter of

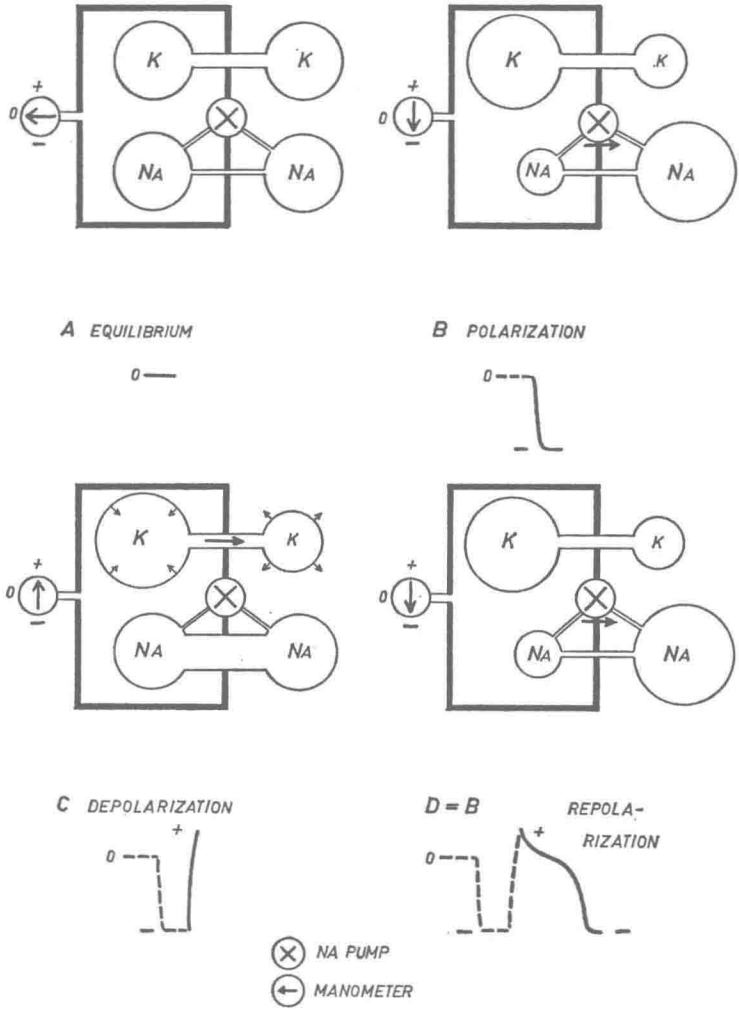

Fig. 5 Ionic flux and sodium pump represented as a mechanical model. The size of the balloons indicates the ion concentration, the width of the connecting tubes the permeability. The manometer registers the intracellular pressure, analogous to the intracellular potential.

which denotes the permeability of the cell membrane; the balloons for the sodium ions are, moreover, shown connected to the sodium pump. In the state of affairs shown in Fig. 5A the pump is not working, the intracellular and extracellular potassium and sodium ions are present in equal numbers and there is thus no difference in pressure as between the inside and outside of the cell.

In Fig. 5B the sodium pump is in action, thus exhausting the intracellular balloon and blowing up the external one. Within the cell a negative pressure is produced, causing potassium ions to be drawn in; this continues until the pressure in the intracellular potassium balloon reaches the point where it can dilate no further. This pressure is analogous to the "diffusion pressure". In this state the intracellular pressure is lower than the pressure outside, as shown by the negative reading of the pressure gauge. This, then represents the state of the polarized cell at rest; the cell interior reveals a negative potential with respect to the outside. Depolarization is shown in Fig. 5C Here, the duct between the sodium ion balloons is seen to be very wide, indicating increased permeability, considerably wider than that between the potassium ion balloons. Due to the width of this duct, equilibrium of the sodium ions sets in more rapidly than that of the potassium ions with their relatively narrower duct. So there is, as it were, a positive pressure in the cell, as shown by the positive reading of the pressure gauge.

The return to reduced permeability of the cell membrane to sodium ions and the resumption of work by the sodium pump produces the situation shown in Fig. 5D, namely, the polarized resting state. The curves drawn below the figure illustrate the pressure as registered by the pressure gauge; it should be noted, however, that the state of equilibrium shown in Fig. 5A does not actually occur in the process of depolarization and repolarization, but is merely intended to clarify our argument.

2.2. Propagation of the stimulus in the muscle fibre

Fig. 6A represents part of an unactivated muscle fibre (diastole). We have seen above that in this state the cell wall is polarized, the interior being negative. Let us suppose that a stimulus is applied to the left hand side of the fibre. This results in an activated area at that point, the polarity of which is now reversed, i.e. the interior is positive and the exterior negative. At the boundary between the activated and non-activated areas there is a transitional zone about 0.5 mm in thickness, that is, very thin compared with the dimensions of the heart. This transitional zone represents the *excitation wave*

front. Fig. 6B depicts the situation as a longitudinal section and Fig. 6C shows this in three dimensions. If we now consider only the potentials at the *surface* of the fibre it will be seen that the non-activated part is positive with respect to the activated part. It will be clear that it is just these potential ratios at the surface of the muscle fibre which are of importance in electro- and vectorcardiography, for it is these surface potentials from which, via the surrounding tissues the ECG or VCG is taken from the body surface.

It can be proved that physically, the condition depicted in Figs. 6B and 6C (provided the fibre is a long one) is equivalent to Fig. 6D, in which the excitation wave front is represented as an electrical double layer, the positive side of which corresponds to the non-activated part of the fibre.

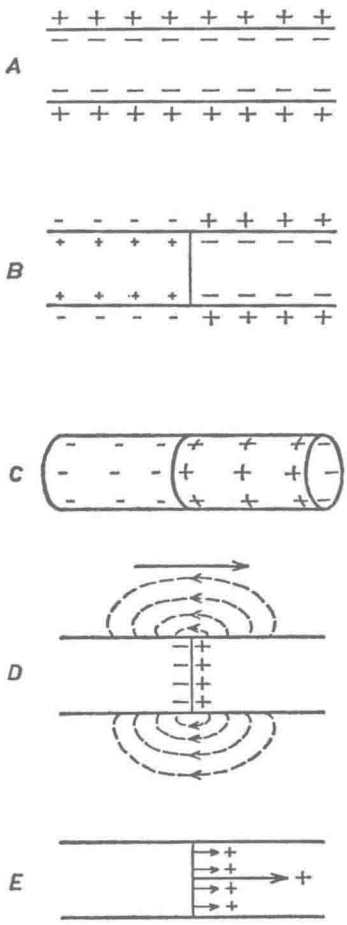

Fig. 6 Depolarization wave front proceeding from left to right through a muscle fibre.
Fig. A Polarized state.
Fig. B Left hand portion of fibre depolarized, the polarity across the membrane is inverted.
Fig. C Spatial representation of the same situation as in Fig. B, the outer surface potential of the stimulated part being negative with respect to that of the non-stimulated part.
Fig. D Depolarization wave front symbolized as an electrical double layer; the dashed lines indicate electrical flow in the surrounding tissue, the solid arrow indicates the direction in which the wave front is propagated.
Fig. E The small arrows represent the vectors symbolizing the individual dipoles, the large arrow is the resultant vector of the wave front.

Now the elements with which the muscle fibre is surrounded in the body are electrical conductors, which means that an electrical connection exists between the positive and negative parts on each side of the excitation wave front (the short-circuiting factor already mentioned). Hence, positive ions will drain away from the positive non-activated part – particularly in the immediate vicinity of the wave front – to the negative activated part, and negative ions in the opposite directions, resulting in a drop in the positive potential (broken lines in Fig. 6D). The membrane potential at this point accordingly also drops and this in turn constitutes excitation. In this way the excitation wave front propagates itself to the positive side (Fig. 6D); the arrow indicates the direction of propagation. In other words, when a muscle fibre is activated, the excitation is propagated in the form of an *excitation wave front*, the front of which is positive, and the rear negative.

2.3. Depolarization and repolarization wave front

We have seen above that when a stimulus is applied to a muscle fibre it is propagated as the depolarization wave front from the point of excitation.

Now what happens when the wave front has passed? The part of the muscle immediately behind the wave front is then in the activated or depolarized state. It immediately begins to return to a state of rest, or *repolarization*, but this takes place much more slowly than the depolarization itself.

If we look at Fig. 2 once more we see that the "overshoot" as from the zero level disappears quite quickly, but that the activation remains at a relatively high level for a while, before decaying fairly rapidly. In view of this it is clear that the activated state must persist along a certain length of the muscle fibres behind the depolarization wave front.

Repolarization, in the same way as the more rapid depolarization, can also be regarded as a wave front, namely the *repolarization wave front*, between the activated (depolarized) and recovered (repolarized) muscle fibre. The repolarization process, however, is more gradual and the "transition zone" is thus necessarily wider than in the case of depolarization.

When a muscle fibre is activated, the following events will pass a given point in succession:

1. a sharp depolarization wave front.
2. an activated zone.
3. a more vaguely defined repolarization wave front.

This sequence of events is illustrated in Fig. 7. From top to bottom the activated zone with its two wave fronts is seen progressing from left to right.

D = depolarization wave front with sharply defined distribution of potential.
R = repolarization wave front with more diffuse potential distribution.
The dotted area shows the activated zone.

The electrical phenomena resulting from the events taking place in the muscle fibre as recorded by means of a sensitive, low-inertia millivoltmeter connected to an electrode near the muscle fibre and to another "indifferent"

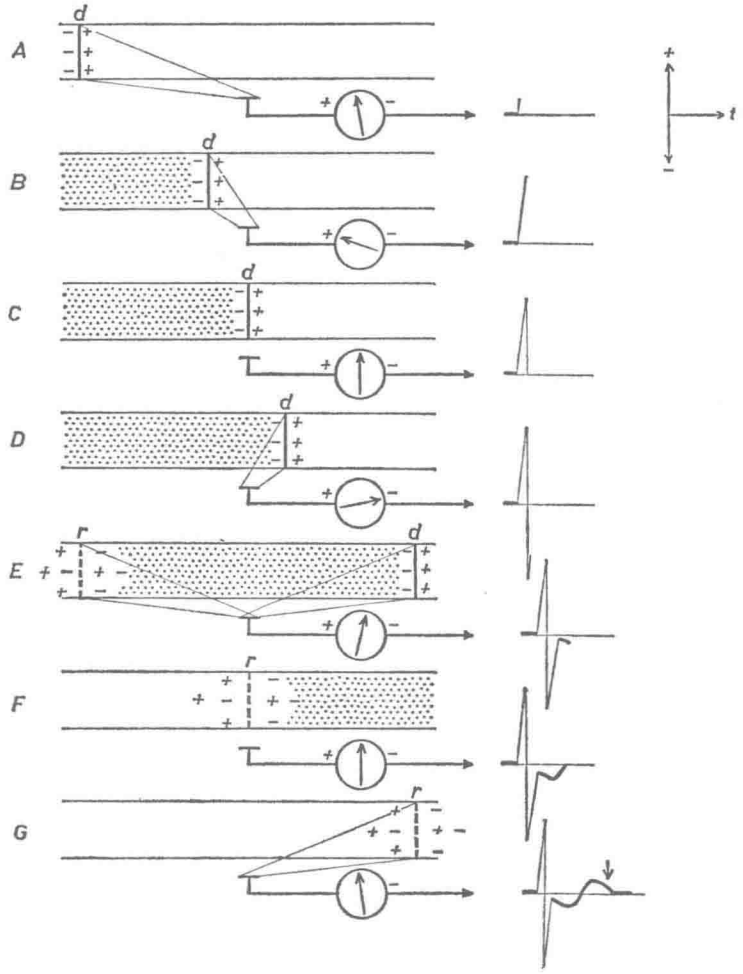

Fig. 7 Deflections registered by an exploring electrode near a muscle fibre during the passage of a depolarization and a repolarization wave front. (from left to right). d = depolarization wave front, r = repolarization wave front. The dotted area represents the activated part of the muscle fibre.