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PREFACE

Almost ten years have passed since this series made its first appearance. During this time, many frontiers of veterinary medical science have been represented. The question has been asked as to a unifying theme. The answer lies in the dual function of veterinary science, to further human as well as animal welfare, and to provide the experimental models for human medicine. From these facts emerges the concept of comparative medicine.

This point is illustrated by the subjects treated in this seventh volume of *Advances in Veterinary Science*. In today's world, veterinary cardiology, with its unique opportunity for pathologic follow-up, could become a major factor in the struggle against heart disease, the foremost killer of man. After many years on the sideline, veterinary dermatology emerges as a specialty. Parasitology is breaking the traditional chains of taxonomy and therapy and is attacking parasitic disease by an entirely new, immunological approach. For every domesticated species, including man, respiratory diseases represent the problem of highest morbidity, economic loss, etiologic complexity, and therapeutic nihilism. The analysis of shipping fever of cattle is a case in point and, at the same time, it emphasizes the possibility of common etiologic factors impinging upon multiple species. That ultimate control of an infectious disease in one species depends upon control in other species is brought out by the chapter on tuberculosis. In our concern with man-made poisons, we are often unaware of possible toxic by-products from otherwise innocuous fungi. The section on mycotoxicoses is probably the first comprehensive review in the English language.

The editors wish to acknowledge with thanks the continuous support and guidance of the Advisory Board and the Publishers in the preparation of this volume.

C. A. Brandly
E. L. Jungherr

March, 1962

CONTENTS

Enterotoxemia-like Conditions among Sheep in South Africa

B. C. Jansen and R. A. Alexander

Nonspecific or Cross-Sensitivity Reactions to Tuberculin in Cattle

Alfred G. Karlson

Skin Diseases

F. Kral

Shipping Fever of Cattle

S. K. Sinha and F. R. Abinanti

Mycotoxicoses

JOSEPH FORGACS AND W. T. CARLL

The Study of Cardiac Dynamics and Its Clinical Significance*†

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I. Introduction

The aim of the diagnosis of disease lies in the detection of functional disturbances of the organs, as early as possible and in quantitative terms. For the diagnosis of cardiac disturbances, exact examination of the cardiac dynamics is important.

The physiology of cardiac function has been rather well known for many years. Clinicians, however, could do little with the findings of the physiologists because of the technical difficulties involved in applying the methods of investigation to patients. Modern technical advances aid greatly in the investigation of cardiodynamics, so that they also can be used in routine clinical practice. With these aids, the accuracy of diagnosis and prognosis has been fundamentally improved, so that the effect of therapy can be followed with greater objectivity.

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* Research supported by the Swiss National Fund for the Promotion of Scientific Investigation (Credit #688).

† This chapter was translated by Dr. D. K. Detweiler and J. Cushmore, School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pennsylvania.

II. Methods of Study

In the investigation of cardiodynamics, the following diagnostic aids are above all necessary:

1. Electrocardiography
2. Phonocardiography
3. Oscillography of the arteries and veins (sphygmography)
4. Intracardiac and intravascular blood pressure measurement
5. Pneumography

Pneumography is significant in the study of cardiac activity, because of the strong influence of respiration on the action of the heart.

III. Time Relationships of Cardiac Dynamics

The following account is restricted, primarily, to the time and pressure relationships of the cardiac cycle. First the time relationships and then the pressure relationships will be discussed.

1. Activation of the Heart

The activation process of the heart of the dog is best known (Scher *et al.*, 1953, 1955a, b; Scher and Young, 1956; Prinzmetal *et al.*, 1953, 1954, 1956; Rothman *et al.*, 1954; Durrer and van der Tweel, 1957; Sodi-Pallares, 1956). In man and in many other species (e.g., the cat, rabbit, guinea pig, etc.) the course of excitation is probably similar. This follows from the circumstance that the electrocardiographic and vectorcardiographic curves in these mammals and in man are very similar.

Considerable variation of the electrocardiographic and vectorcardiographic patterns from the other mammals is shown by the horse and the cow. From this, it must be concluded that the mode of activation in these animals is different from that of the dog (Fig. 1) (Kisch *et al.*, 1952; Spörri and Detweiler, 1956; Van Arsdel *et al.*, 1959).

a. Activation of the Atria

It is well known that in the dog the wave of excitation spreads from the sinuatrial node in the right atrium in all directions, that is, like drops of liquid on a piece of blotting paper. The musculature of the atrial wall is simultaneously excited throughout its entire thickness. The left atrium is activated somewhat later than the right, and the last to be reached by the wave of excitation are those parts of the muscle which are located farthest from the sinuatrial node, i.e., the apices of the auricles.

The activation of the atria in equine and bovine hearts is completely analogous to that of the canine heart (Spörri and Detweiler, 1956).

b. Activation of the Ventricles

The activation of the ventricular musculature is an extremely complex process, which cannot be entered into in complete detail here. In principle, the wave of excitation in the ventricular muscle of the canine heart spreads from the inside to the outside, e.g., the subendocardial muscular layers are activated before the subepicardial layers. Those parts which are more in the middle of the ventricles are excited earliest; somewhat later the apical parts, and finally, the basal regions of the ventricular musculature become activated. Kisch and co-workers (1952)

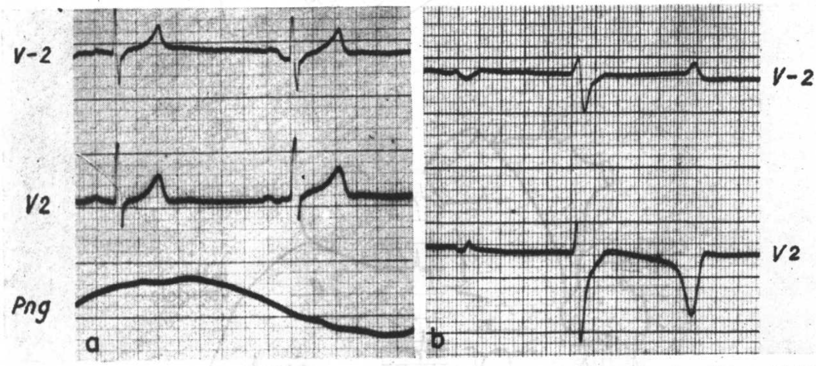
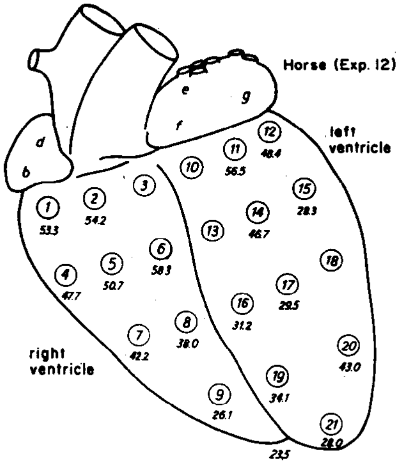


FIG. 1. a. ECG (leads V-2, V 2) and pneumogram (PNG) of a dog (Dog 11/59). b. ECG (leads V-2, V 2) of a horse (Horse 13/59). The electrocardiograms were registered against the Wilson central terminal electrode. Lead V-2: Exploring electrode in the 5th right intercostal space at the height of the olecranon. Lead V 2: Exploring electrode in the 5th left intercostal space at the height of the olecranon. Time intervals: light ordinates = 0.02 sec.; dark ordinates = 0.1 sec. The canine ECG shows a predominantly positive QRS and the equine, a predominantly negative QRS. The QRS of the horse and dog, therefore, are almost like reflected images of each other.

suggest that, because of the almost reverse behavior of the initial ventricular wave of the electrocardiogram (ECG) in the horse and ox in comparison with the dog (predominantly negative initial wave in the horse and the ox, predominantly positive initial wave in the dog, using analogous ECG leads; compared in Fig. 1), the direction of excitation in the ox is not from endocardium to epicardium, but spreads from the epicardium to the endocardium. Experimental investigation of these questions led to the conclusion that in the horse and the ox, at least in the subepicardial muscular layers, the wave of excitation spreads from the inside out (Spörri and Detweiler, 1956). The spread of excitation in the subendocardial myocardial layers could not yet be explained. In these regions, the excitation seems almost to



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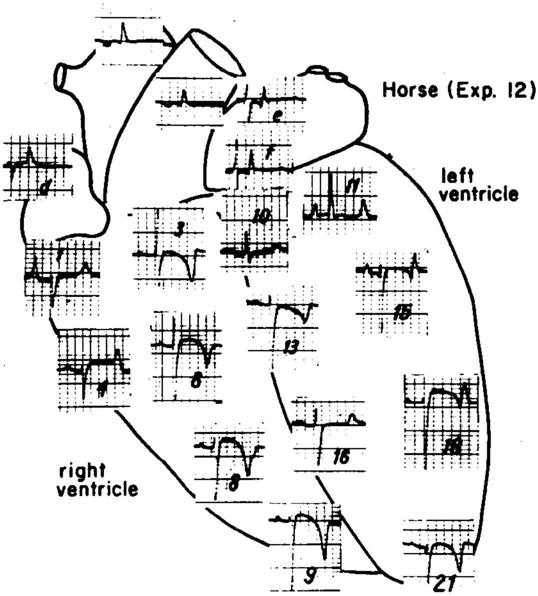


b

FIG. 2. a. Activation of the equine cardiac surface. The figure shows the time of arrival of the wave of excitation on the cardiac surface of the left side of the right and left ventricles. The numbers under the circles which mark the positions of recording electrodes on the myocardium correspond to the time interval, expressed in milliseconds, between the beginning of the QRS complex and the arrival of the wave of excitation at the particular section. The wave of excitation reaches the apex of the right ventricular surface earliest (23.5 milliseconds after the beginning of the Q-wave of the ECG). The base of the heart is activated last.

b. Electrograms (EG) of the cardiac surface of a horse registered against the Wilson central terminal electrode. In the apical region, the EG is almost purely negative, and contrarily, the basal EG is strongly positive.

appear simultaneously everywhere or, at least partly, to be propagated from the outside inward.

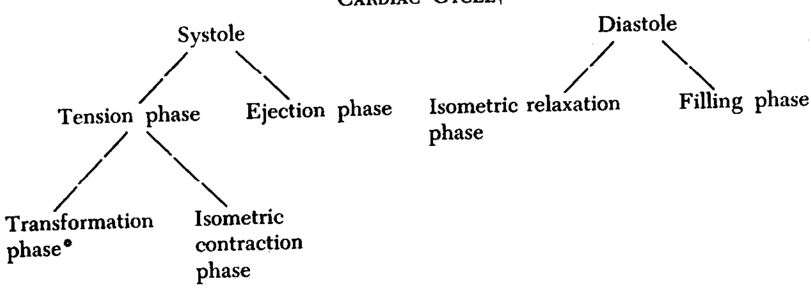
On the cardiac surface, the wave of excitation appears regularly first at the apex of the right ventricle, then in the apical region of the left ventricle, somewhat later in the central regions of the right and left ventricles, and finally at the base of both ventricles. The wave of excitation, therefore, moves along the cardiac surface apparently from apex to base. It can be said with reasonable certainty that the last regions of the myocardium to become excited do not lie on the cardiac surface, but probably in the basal areas of the ventricular septum.

Figure 2b shows electrograms of an equine heart, with the exploring electrode applied directly to the cardiac surface and paired with the Wilson central terminal. The initial ventricular wave in the apical and central regions of the heart is predominantly negative, and in the basal area, on the other hand, predominantly positive. This is an indication that the activation of the cardiac surface of the ventricular myocardium occurs in an apicobasal direction.

2. Normal Phases of the Cardiac Cycle

Since ancient times, the heartbeat has been divided into two main phases, systole and diastole. Newer investigations have led to a further differentiation. Systole is subdivided into the phases of tension and ejection, and diastole into the phases of isometric relaxation and filling. Since the basic researches of Hess (1920), however, we know that the tension phase is not a single, uniform event. At the present time, it is further divided into at least a transformation phase* and an isometric contraction phase.

Cardiac Cycle†



* Also called the "period of fractionate summation," summation phase, or entrant phase.

† *Translator's Note*: In the English literature, the scheme proposed by Wiggers for the divisions of the cardiac cycle is usually employed, while the author has used an equally valid scheme, often found in the German literature. Although both schemes agree in essentials, they differ somewhat in details and the respective ter-

a. Transformation Phase

If one examines simultaneously recorded electrocardiograms and pressure curves of the ventricle, it is immediately obvious that the initial ventricular waves of the QRS complex take place earlier than the rise in intraventricular pressure (Fig. 3). The period of delay between the beginning of the electrical phenomena and the rise in pressure can be termed the period of electropressor latency (Cerletti and Weissel, 1952).

The question now arises as to whether during this electropressor latency there are really no mechanical changes induced in the ventricles. Investigations in recent years have shown that mechanical events do occur during this phase, specifically, changes in the form of the heart. During diastole, the ventricles are relaxed and therefore lengthwise

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minologies are not exactly the same. The two schemes have been compared recently by E. Schuetz ("Physiologie des Herzens," pp. 291 and 292, Fig. 150a and b, Springer, Berlin, 1958). In translating the German description into English, the equivalent terms from Wiggers' scheme have been used wherever possible. The original German terminology and English equivalents are listed in the following for comparison:

Herzzyklus	Cardiac cycle
Systole	Systole
Diastole	Diastole
Anspannungsphase*	Tension phase*
Umformungsphase*	Transformation phase*
Druckanstiegsphase*	Isometric contraction phase*
Austreibungsphase	Ejection phase
Entspannungsphase	Isometric relaxation phase
Füllungsphase	Filling phase

Note on terms indicated by asterisks:

In Wiggers' scheme, the term *isometric contraction phase* includes the entire *Anspannungsphase,* which is further subdivided into an *Umformungsphase* and *Druckanstiegsphase* in the German scheme. In this translation, the term *isometric contraction phase* is restricted to the steep or rapid pressure rise phase (*Druckanstiegsphase*) of the ventricular pressure curve. The term *Anspannungsphase* is translated as *tension phase*. The term *Umformungsphase* is translated as *transformation phase*. In the Wiggers' terminology the first phase is not considered as separate from the isometric contraction phase, but rather as the beginning of isometric contraction. In 1927, the possibility that entry of fractional contractions might be involved led to the introduction of the term "entrant phase" for the corresponding period (C. J. Wiggers, Advances in the Study of Basic Cardiodynamic Problems *in* "Reminiscences and Adventures in Circulation Research," pp. 228-242, Grune and Stratton, New York, 1959). This period has also been called the "period of fractionate summation" or "summation time" in English [G. R. Graham *in* "Atlas of Intracardiac Pressure Curves" (O. Bayer and H. H. Wolter, eds.), pp. 10 and 60, Thieme, Stuttgart, 1959 (English translation)].

ovoid in shape. At the beginning of systole, the muscle fibers contract and the ventricular wall becomes smaller, without lessening the capacity of the chambers or without a significant change in pressure. This is possible because the ventricles assume a more spherical shape. A sphere, it is known, has the least surface area in relation to its volume. Right at the beginning of the systole, therefore, the ventricles merely assume another

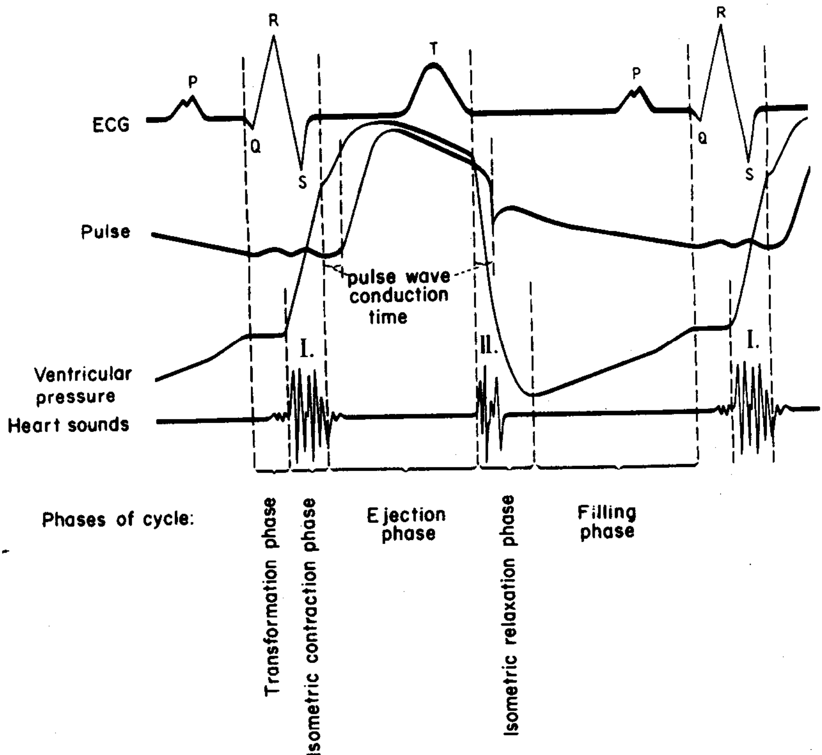

FIG. 3. Phases of the cardiac cycle (explanation in text).

shape without expelling any blood, that is, without significantly raising the blood pressure. This first phase of the systole is therefore called the transformation phase* (Holldack and Wolf, 1956).†

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* See Translator's Note, pp. 5 and 6.

† Cerletti and Weissel (1952) distinguish still another period, that of electromechanical latency. This period signifies the time from the beginning of the first potential difference of the ventricular ECG (beginning of the QRS complex) to the onset of the first detectable mechanical action of the ventricles. The period is difficult to determine exactly for the patient. From the studies of Nazzi *et al.* (1954), it is considered as the time from the beginning of the QRS complex of the ECG up to

When the smallest possible surface of the ventricle is reached, the contraction of the muscle fibers comes to a sudden stop, since blood is incompressible. This abrupt cessation of muscular shortening, in other words, this firm tension of the ventricular wall around the incompressi-

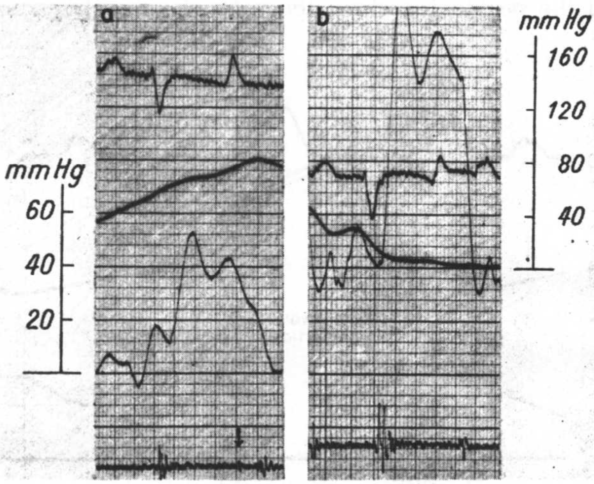

FIG. 4. Pressure curves of a cow (Cow 11/58) with increased pressure in the systemic circulation following administration of a calcium preparation containing amphetamine. From top to bottom: ECG, pneumogram, ventricular pressure curve (a, right ventricle; b, left ventricle), phonocardiogram (PCG). In (a) the first heart sound occurs significantly earlier than the rise of the pressure curve of the right ventricle. In (b) the first heart sound begins simultaneously with the rise of the left ventricular pressure curve. From this, it may be concluded that the beginning of the first heart sound is caused by vibrations associated with closure of the mitral valve. In (a) the PCG shows a slight vibration (arrow) at the moment of descent of the right ventricular curve, which seems to be the second heart sound, but occurs only after the plateau, during the descending limb of the pressure curve. The slight vibration in the PCG which actually precedes the second heart sound by approximately 0.07 sec. may mark the closure of the pulmonary valve. The real second heart sound indicates closure of the aortic valve, for in (b) the second heart sound occurs exactly with the beginning of the sharp descent of the pressure curve of the left ventricle.

ble contents of the chamber, leads to a vibration of the heart, particularly of the atrioventricular valves, which close at this moment. The

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the onset of the very first vibrations (initial segment) of the first heart sound. The initial segment of the first heart sound is believed to be produced by the movements of the ventricle during the transformation phase. Since the electromechanical latent period appears to be of a somewhat constant duration under normal as well as pathologic conditions, it is diagnostically of little significance. In our present study, it will simply be considered as part of the transformation phase.

vibration of the heart at the end of the transformation phase produces the main segment (audible part) of the first heart sound (Fig. 3).

Thus, the duration of the transformation phase can be easily determined. It lasts from the first appearance of the electrical potential (the beginning of the QRS in the ECG) to the closing of the atrioventricular valves, therefore up to the first large vibration of the first heart sound in the phonocardiogram (PCG). If the ECG is recorded simultaneously with the ventricular pressure curve, this phase can be measured from the beginning of the QRS complex of the ECG to the rapid rise of the pressure curve (Fig. 3). This method of measurement is in fact more dependable than using the PCG, since both ventricles do not contract in exact synchrony, and therefore one cannot, from the PCG alone, determine which vibrations denote the closing of the mitral valve, and which represent the closing of the tricuspid valve. At the present time, the beginning of the main vibration of the first heart sound in the horse, ox, and dog is normally considered to be the closing of the mitral valve (Fig. 4b).

b. Isometric Contraction Phase

At the moment when the transformation phase is terminated, the ventricular pressure begins to rise rapidly (Fig. 3). But still at this point no blood is expelled from the ventricles. This phase is called the isometric contraction phase. It lasts up to that moment when the pressure in the ventricles reaches or slightly exceeds that of the aorta or the pulmonary artery. It includes, therefore, the time interval from the closing of the atrioventricular valves to the opening of the semilunar valves. The end of this phase, i.e., the opening of the semilunar valves, is indicated on the rising ventricular pressure curve often in the form of a notch, or a sudden change of the rise in pressure. This is usually in the form of a delay, but also, occasionally, it is in the form of an acceleration (for example, see Fig. 5) in the upper part of the ascending pressure curve (Figs. 3 and 5).

This phase can be measured, therefore, from the beginning of the rapid rise of the ventricular pressure curve to the aforementioned notch, or sudden change in the gradient slope of the pressure curve.

The isometric contraction phase of the left ventricle can also be ascertained without the registration of the ventricular pressure curve. This is very significant because the technique of recording pressure curves of the left ventricle is not so advanced that it can be routinely applied in clinical diagnosis.

As already mentioned, the ejection phase, as the last systolic subphase, follows the isometric contraction phase. The ejection phase of