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NOTE: For coverage of blood flow to the brain, see "The Cerebral Circulation" by S. S. Kety, chapter LXXI, pp. 1751–1760, Neurophysiology section of the *Handbook* series.

CHAPTER 23

Functional anatomy of cardiac pumping[1]

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CHAPTER CONTENTS

ALTHOUGH THE PURELY MECHANICAL NATURE of cardiac pumping is taken for granted by modern scientists, this view has not always been accepted in the past. Only during the last hundred years were the forces of muscle contraction finally stripped of the 'vis vitalis' and ascribed exclusively to energy transformation according to the laws of physics and chemistry. In this historical process, the heart which had been formerly thought of as the seat of emotions, was deprived of all metaphysical connotations and became an organ of purely mechanical function just as the skeletal muscle. It is of interest to trace briefly the emergence of this concept (160, 161).

During the age of the pyramids (3000–2500 B.C.) an unknown Egyptian clearly recognized the heart as the center of a system of distributing vessels and associated the pulse with the cardiac beat. The Greek philosopher Alcmaeon of Croton (about 500 B.C.) distinguished the veins from the arteries and asserted that the seat of sensation was not in the heart but in the brain. The function of the heart as a pump was apparently expressed for the first time by Plato (427–347 B.C.) when he stated: it "pumps particles as from a fountain into the channels of the veins, and makes the stream of the veins flow through the body as through a conduit." Hippocrates (493–423 B.C.) had described the cardiac valves, the ventricles and the great vessels, but he did not refer to the pumping action, which he might have taken for granted. For Aristotle (384–322 B.C.) the heart was the seat of "innate heat" and also of the soul. This notion was probably based on the observation that death results from dissection of the beating heart. However, from his studies on the embryonic chick heart Aristotle may have had knowledge of the pumping function. Erasistratus (310–250 B.C.), who described the aortic valves, pulmonary valves, and chordae tendineae, and Galen of Pergamon (131–201 A.D.) both stated that the heart is a pressure-suction pump. Their view was founded mainly on the assumption that during diastole blood was sucked into the ventricles by active enlargement of the cardiac walls [discussed by Ebstein (40), Böhme (14)]. They also believed that blood is expelled backward into the caval veins during ventricular systole. The first definite statement concerning the continued forward flow of blood from the right ventricle through the lungs into the left heart was made by Ibn an-Nafis (1210–1299 A.D.). The first scientist of the Renais-

[1] The results of some recent experiments of the authors and their colleagues are quoted in this paper. This work was supported in part by USPHS grant H-3796, and grants from the Life Insurance Medical Research Fund and the Georgia Heart Association.

sance who recognized the heart as a hollow muscle and probably as a pump was the artist-engineer, Leonardo da Vinci (1452–1519 A.D.), who stated: "The heart is a principal muscle, in respect of force, and it is much more powerful than the other muscles" [Keele (90)]. However, it remained to William Harvey (1578–1657) to prove that the heart, and not the liver, is the center of the vascular system and that it propels the blood unidirectionally by its rhythmical contractions as would the repeated strokes of a man-made pump. The microscopic proof of the muscular nature of the heart was brought by Niels Stenson (1638–1686), who demonstrated that the substance of the heart is composed of fibers, membranes, arteries, veins, and nerves just as is the substance of other muscles. Once this important point had been firmly established, it became customary to consider the heart as a pump, to develop analogies with mechanical systems of fluid transfer, and to apply to the myocardium the increasing knowledge about skeletal muscle contraction. The present chapter is a rather general and classically oriented treatment of the mechanical function of the heart. It attempts to provide an understanding of the anatomical structures, while avoiding teleological oversimplification as well as useless controversies about functions.

The role of the heart consists of providing the body tissues with a continuous stream of blood. The heart fulfills this function by converting potential energy (primarily chemical energy, secondarily energy of position) into kinetic energy, as movement is imparted to the blood ejected from the ventricular cavities. From the standpoint of cellular function at large, it does not matter whether tissue perfusion is brought about by alternate contraction and relaxation of myocardial cells, or by the action of an artificial pump. This concept has been established on a firm experimental basis by the advent of extracorporeal circulation techniques, whereby a mechanical pump substituted for the human heart can fully support the circulation. Thus the heart can be looked upon as a pump inserted in the circulatory system and its function can be described by analogy with purely mechanical systems.

Mechanical pumps are divided into two main classes: kinetic pumps and positive displacement pumps. In the former class, kinetic energy is added to the fluid by the forced rotation of an impeller (fig. 1*A*). In the latter class, the fluid is progressively displaced from a suction inlet to a discharge opening. Two kinds of positive displacement pumps need to be mentioned here. In rotary pumps (fig. 1*B*), moving members trap a portion of the fluid in a chamber of pliable tubing and conduct it toward the outlet. The segment of tubing occluded acts as a valve to prevent backflow. In reciprocating pumps (fig. 1*C*) a cavity limited by two valves is subjected to the action of a piston or diaphragm. As the piston moves back and forth, fluid is drawn in through the suction valve and forced out through the discharge valve.

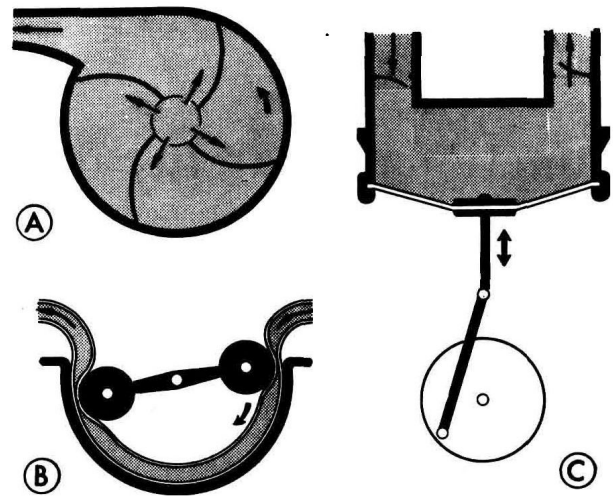


FIG. 1. Mechanical analogues for some pumping principles embodied in the heart. *A:* kinetic pump in which energy is added to the fluid by the rotation of an impeller. *B:* rotary pump in which fluid is propelled through squeezing a resilient tube by means of rollers mounted on a rotating arm. *C:* reciprocating pump in which fluid is displaced by the back and forth movement of a diaphragm while valves give direction to the stream.

The action of the heart in some invertebrates can be compared to that of rotary pumps, since forward movement of fluid is obtained by peristaltic movements of the walls. In the mammalian heart also some degree of blood propulsion may be accomplished on the "progressive cavity principle" as in rotary pumps, particularly the displacement caused by the wringing action of the ventricles. However, cardiac action in vertebrates most closely resembles that of reciprocating pumps. It is characterized by pulsatile action, by the presence of valves, and by the capability of the pump to be adjusted in terms of either speed, or volume displacement, or of speed and volume displacement simultaneously. Although the design of the heart has nothing in common with that of kinetic (centrifugal) pumps, its control displays two characteristics for which kinetic pumps are appreciated in technology: namely that the volume

output is directly related to the input pressure, and is inversely related to the pressure head against which the pump works. Like centrifugal pumps, the heart has the tendency to deliver a higher flow as more blood is fed into it at the atrial level; it also provides a lower flow when the resistance to ejection in the vascular system increases.

A close look at mechanical pumps for cardiac substitution throws a light on built-in features of the natural heart that one easily takes for granted. Adequate perfusion of an adult human organism under all possible conditions requires that:

1) The heart be able to move blood volumes ranging from 3 to 30 liters per min and to pump against pressures up to 300 mm Hg.

2) Even at maximal cardiac output, the flow velocity must not exceed the limit of tolerance for mechanical trauma to blood corpuscles through turbulence, friction, or cavitation (1–2 m/sec).

3) The relationship between stroke volume and stroke rate must not deviate much from an optimum which is set by the elastic properties of the cardiac walls, the time needed for efficient transformation of potential into kinetic energy and by the lowest flow velocity compatible with the output required.

4) The valves must easily open during their flow phase, yet be competent and prevent regurgitation of blood during their holding period.

5) The regulation of the pumping action must be automatically controlled through sensing elements with feedback mechanisms which adapt the output to the tissue demands [see also Wagner (153)]. These control mechanisms must integrate hemodynamic data (e.g., perfusion flow, arterial and venous pressures) and metabolic data (e.g., arteriovenous oxygen difference) to maintain viable conditions.

Considering these points in more detail, one must first emphasize the pumping capacity of the heart. As 3 to 30 liters per min of blood is pumped by the left ventricle into the systemic circulation, practically the same amount is ejected by the right ventricle into the pulmonary vascular bed. Furthermore, the atria have some pumping function of their own, so that the combined pumping of all the chambers of the human heart is in the order of 7 to 70 liters per min, depending upon the state of muscular activity. A range of this magnitude (1:10) is not easily obtained in artificial pumps and, when it is reached, it is at the price of considerable sacrifices in mechanical efficiency (ratio of work produced to fuel consumed). On the contrary, the mechanical efficiency of the heart does not seem to be very closely related to cardiac output. The extended scale of activity over which the heart can perform is certainly facilitated by the elastoviscous properties of the cardiac walls. The cavities are distensible over a wide range of volume increments without much increase in intraventricular or intraatrial pressures [see fig. 2, and Little (99)]. Therefore the heart can easily accommodate and deliver varying stroke volumes even if the stroke frequency remains unchanged. Furthermore the time needed for the transformation of chemical into mechanical energy apparently comprises only a fraction of the systole. At a constant stroke volume the heart can increase its minute output simply by beating faster and shortening the pause between the strokes without affecting the energy conversion processes. The limiting factor of cardiac output at high heart rates is not an encroachment on the time needed for energy conversion but an encroachment on the time needed for filling the pump chambers (ventricular filling phase).

Another fundamental difference between artificial pumps and the heart is that in the former a force is applied from the outside to activate a part or the entire wall of the pump chamber, whereas in the latter the force is developed within the wall of the pump chamber itself by small elements, the muscle fibrils, which alternately shorten and lengthen. Furthermore, since the heart is surrounded by other resilient structures in the thorax, there is an interaction of the physical forces developed in the myocardium and those developed either passively or actively in these structures [Pfuhl (129, 130), Blair & Wedd (12)]. For example, during ventricular contraction and ejection the elastic forces of the lungs oppose to a small extent the diminution of the ventricular size, whereas during ventricular relaxation the same forces of the lungs enhance slightly the expansion of the ventricles. These forces are said to be negligible as compared with the intravenous filling pressures (60, 64). Mechanical effects are exerted upon the rhythmical form changes of the heart by such structures as the pericardium, the attachments of the heart to the large vessels, the sternum, the mediastinal tissues, and the diaphragm through its changes in position during respiration or because of varying degrees of abdominal filling. The complexity of these forces, in terms of direction and magnitude, and their continuous changes during the cardiac and the respiratory cycle make it presently impossible to evaluate quantitatively the contribution of extracardiac structures to cardiac pumping. Nevertheless, their importance is demonstrated by the possibility of pumping blood solely by the action

of external forces on the heart [Hosler (81), Stephenson (150)]. In closed-chest cardiac massage, vigorous pressure on the lower part of the sternum causes ejection of the ventricular content into the large arteries. Conversely, when pressure is released, the recoil is sufficient to permit the venous pressure to fill the ventricles again [Kouwenhoven *et al.* (92)]. In this manner, a sufficient, though subnormal, cardiac output can be maintained in the absence of any myocardial activity. This points again to the fact that, in principle, it does not matter whether the propulsion of blood through the body is brought about by the contraction of cardiac fibers or by any other suitable forces applied to the blood contained in the ventricles.

MACROSCOPIC STRUCTURES

A great deal of commonly accepted knowledge about cardiac pumping is derived from purely morphological considerations. Although conclusions reached in this manner have occasionally proved to be correct, morphological reasoning often leads to fallacious functional interpretations of structural findings. In the case of the heart, physical vector analysis of all the mechanical forces involved is especially difficult because of the great complexity of the anatomical structures and of the perplexing geometry of cardiac filling and emptying. We have only a limited knowledge of the sequence of events as they occur during muscular contraction and relaxation within various parts of the myocardium. In this particular section an attempt is made to describe the macroscopic structures of the heart with reference to their probable function as deduced from the anatomical observations. A topographic anatomical description of the heart is available in standard texts (51, 95, 98, 101).

Composition of Cardiac Tissues

The myocardium is the most important structure of the heart because its contraction causes the blood to flow. However, it should be realized that only part of the cardiac walls consists of muscle fibers, and that within the muscle fibers, the contractile substance is limited to the fibrils. Indeed about half of the heart's weight is made of noncontractile material such as the sarcolemma in the muscle fibers, connective tissue in the heart skeleton, tendons and valves, and finally blood vessels, lymphatics, and nerve fibers. All these elements are interwoven with the muscle fibers or closely connected to them (45, 59). During cardiac contraction or relaxation, they are deformed and resist to some degree the shortening or lengthening of the myofibrils.

Little is known about the mechanical effects of the coronary vessels upon the function of the ventricles. Though relatively inconspicuous in a "dead" heart, they appear heavily engorged with blood in the live organ. In fact, since 5 to 10 per cent of the cardiac output passes through the coronary system, a significant mass of the beating heart consists of circulating blood contained within the anatomical bounds of the epicardium. During heavy exercise the coronary blood supply is probably so great that one might look upon the myocardium as a spongy structure of muscle fibers suspended like chains of islands in a lake of blood. In the past it has been postulated frequently that the degree of filling of the coronary vascular bed affects in some form the ventricular contraction.

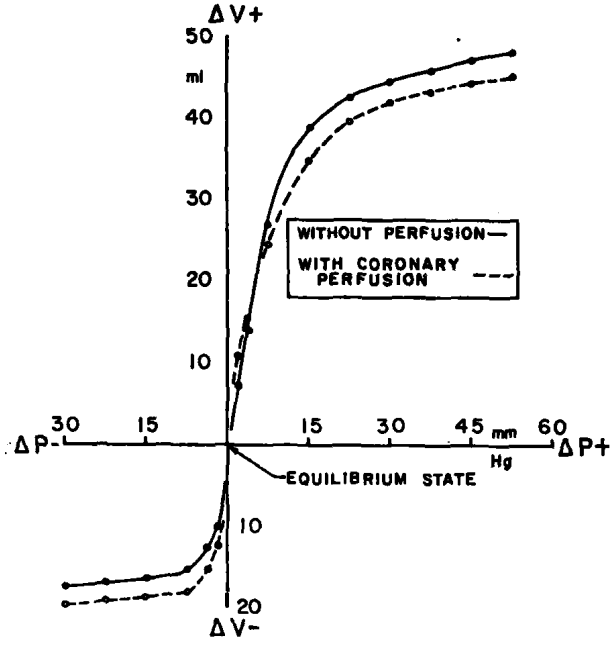


FIG. 2. Left ventricular pressure-volumes curves of a dog heart illustrating the changes resulting from coronary perfusion. The freshly excised heart of a 13.5-kg dog was submerged in Locke's solution and assumed its elastic equilibrium state (zero transmural pressure, origin of the coordinates) upon cessation of spontaneous contraction. The curves were obtained by addition or reduction of the intraventricular volume [Brecher & Kissen (22)]. The origins of the coordinates for the perfused and unperfused heart were arbitrarily superimposed. At negative (and up to +5 mm Hg) intraventricular pressures the ventricle accommodated a greater volume with coronary perfusion than without. At pressures above +5 mm Hg the ventricle accommodated less fluid with coronary perfusion than without (Horres *et al.*, unpublished data).

Most of these postulates were of speculative nature. For example, Donders (39) stated that "the blood which enters at the end of systole into the coronary arteries seems to cause a slight active expansion of the heart, especially of the ventricles." This view was originally formulated in 1855 by Brücke (26) and also advocated by Luciani (102). Based on X-ray kymograph studies, a modern modification of the same hypothesis was presented by Cignolini (34) without conclusive evidence. However, recent work by Salisbury *et al.* (141) indicates that the filling of the coronary bed affects the ventricular distensibility. There are indeed significant differences in the ventricular pressure volume relationship depending upon whether the coronaries are perfused or not [Brecher *et al.* (24)]. In figure 2 the S-shaped pressure volume curve of the ventricle with an empty coronary bed (solid line) is different from that obtained during coronary perfusion (broken line). This shift of the curve when the coronary bed is perfused indicates that the perfused heart accommodates more fluid at low intraventricular pressures and less fluid at high intraventricular pressures. Around the elastic equilibrium state (zero transmural pressure) the perfused heart is somewhat stiffer than the nonperfused heart. The effect of varying degrees of engorgement of the coronary bed upon the distensibility of the beating ventricle during the different phases of the cardiac cycle is still unknown.

The heart skeleton, the chordae tendineae, and the cells of the Purkinje system are noncontractile, yet are functional components of the myocardium. The heart skeleton (fig. 3) is represented by four interconnected fibrous rings of dense connective tissue, which surround the orifices of the great vessels. The musculature of the ventricles and atria, the roots of the large vessels, and the heart valves are attached to this skeleton, which also anchors the tendinous endings of the ventricular muscle (see below). An important function of the cardiac skeleton is to provide a firm basis for the attachment of the cardiac valves. Another function, though less frequently mentioned, is to aid in keeping the orifices open during the phases of blood inflow and outflow. During ventricular activity, the orifices undergo changes in form which probably involve also the cardiac skeleton as indicated in the different outlines of the orifices during systole and diastole in figures 4, 5, and 6. By inserting a finger through the atrial appendage in the intact beating heart, one can easily verify that the atrioventricular valve rings become smaller during ventricular contraction and larger during relaxation. This observation, which has not been substantiated by precise measurements as yet, indicates that the fibrous tissues of the heart skeleton are passively deformed by myocardial contraction and thereby store energy which is released

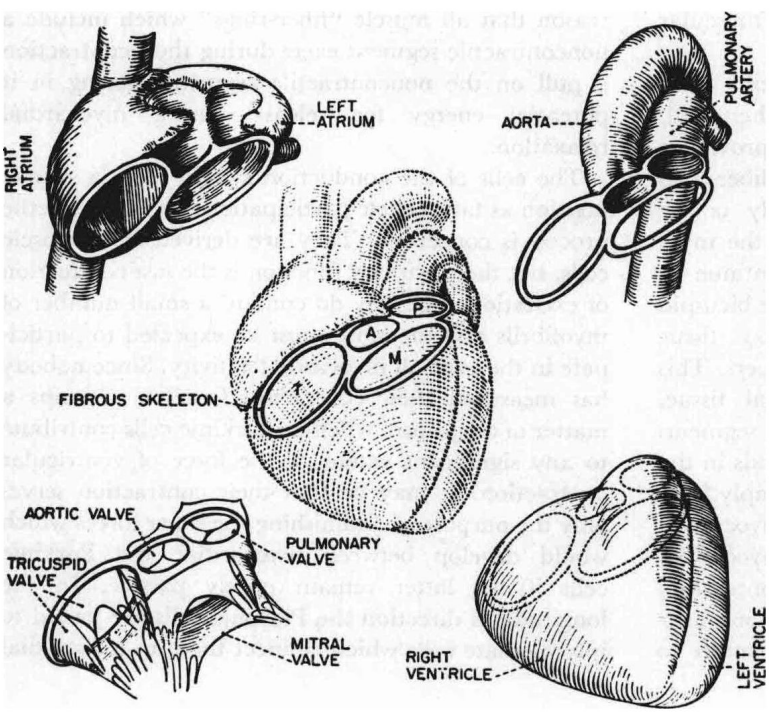


FIG. 3. Anatomic components of the heart depicting the relation of the fibrous skeleton to the heart chambers and arterial roots. The trunks of the aorta and pulmonary artery as well as the atria are fastened to the cranial aspect of the four annuli fibrosi, whereas the ventricles are attached to the caudal aspect. [From Rushmer (139).]

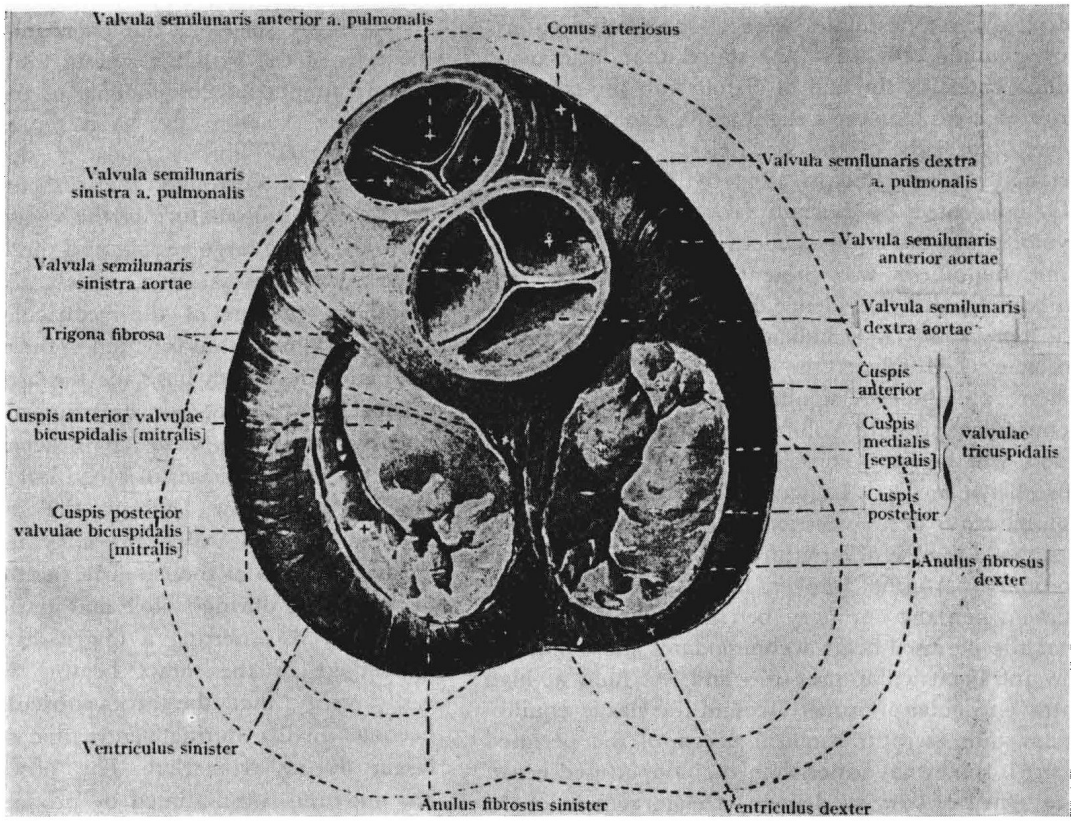


FIG. 4. Base of the human ventricles seen from their cranial aspect after the atria have been removed. The shape of the ostial orifices in the state of contraction differs significantly from the shape in the state of relaxation, as indicated by the dashed lines. [From Spalteholz (148).]

by elastic rebound at the beginning of muscular relaxation.

Many strands of myocardial fibers end with tendinous tissues. Yet one cannot compare them with skeletal muscle, since there is no bone to provide a fixed attachment. In reality, all myocardial fibers end on other myocardial fibers either directly or by insertion of connective tissue. For instance the myocardial fibers of the papillary muscles continue as chordae tendineae, which in turn lead via the bicuspid and tricuspid valve leaflets and the fibrous tissue of the heart skeleton to other myocardial fibers. This arrangement forms a circle of myocardial tissue, although with inclusion of a tendinous segment. Other myocardial fibers, such as many strands in the left ventricular deep bulbospiral bundles, simply form a circle. Since, in the final analysis, all myocardial fibers pull directly or indirectly on other myocardial fibers, the concerted effect of their contraction diminishes each heart cavity more or less concentrically [see also Hawthorne (67)]. It also stands to reason that all muscle "fiber-rings" which include a noncontractile segment exert during their contraction a pull on the noncontractile segment, storing in it potential energy for release during myocardial relaxation.

The cells of the conduction system have a special position as far as their participation in the contractile process is concerned. They are derived from muscle cells, but their primary function is the fast conduction of excitation. Yet they do contain a small number of myofibrils and therefore must be expected to participate in the over-all myocardial activity. Since nobody has measured their contractile force, it remains a matter of conjecture whether Purkinje cells contribute to any significant extent to the force of ventricular contraction. It may be that their contraction serves only the purpose of diminishing the shear forces which would develop between myocardial and Purkinje cells if the latter remain purely passive. In the longitudinal direction the Purkinje cells are joined to intermediate cells which connect them to myocardial

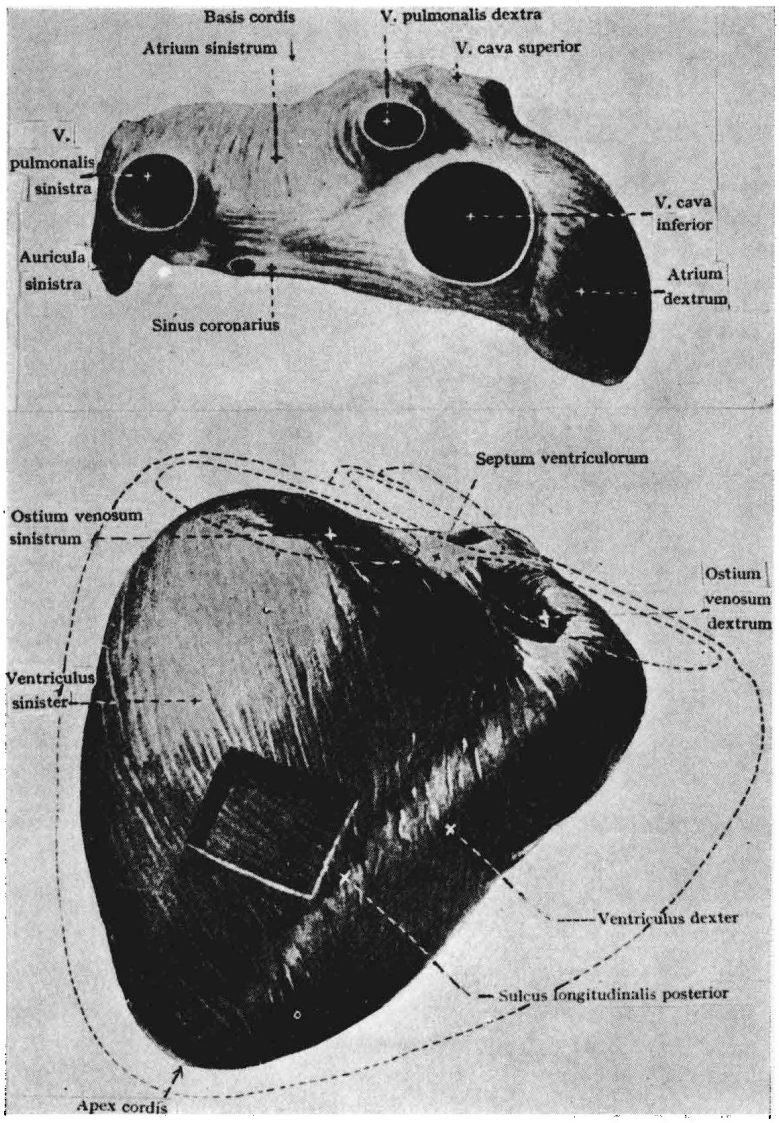


FIG. 5. Superficial muscle layers of the maximally contracted human heart, viewed from the caudal aspect after separation of the atria (above) from the ventricles (below). The ostia of the contracted ventricles can be compared with their state in the relaxed ventricles (dashed lines). The changes in ventricular configuration during relaxation are also indicated by dashed lines. [From Spalteholz (148).]

cells. These intermediate cells contain an increasingly larger number of myofibrils as they approach the true myocardial cells. Merely judging from morphological evidence, they must contribute to some extent to the over-all contractile process.

Architecture of the Ventricular Myocardium

Since the ventricles perform more pumping action than the atria, the architecture of the ventricular myocardium has attracted most of the attention of functionally oriented anatomists. Despite extensive description by MacCallum (108), Mall (111), Mönckeberg (114), Benninghoff (10), Robb & Robb (136), Spalteholz (148), and Lev & Simkins (97), much confusion still prevails. Opinions vary because it is difficult to dissect clearly the complexly arranged, intertwined and crisscrossing discrete muscle bands. Consequently, it is even harder to derive from the anatomic findings a picture of the direction of maximal pull of each muscular component, not to mention the concerted action of several components.

Many of the muscle bands encircle both left and right ventricles. According to the most commonly accepted terminology, one distinguishes four different muscles, the course of which can be best understood from semischematic drawings: the superficial bulbospiral (fig. 7), superior sinospiral (fig. 8), deep sinospiral (fig. 9), and deep bulbospiral muscle (fig. 10.)

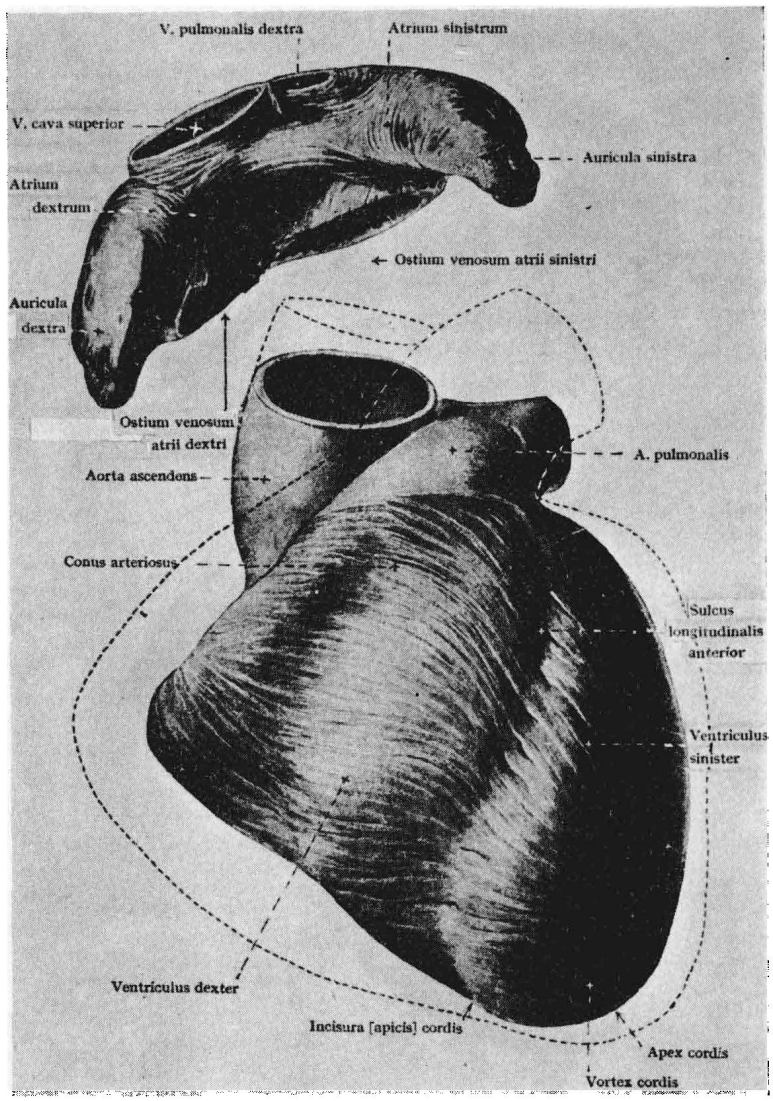
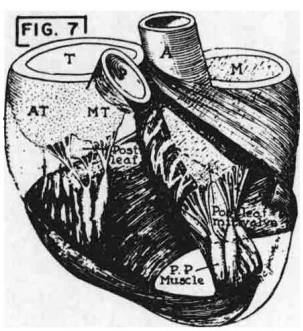
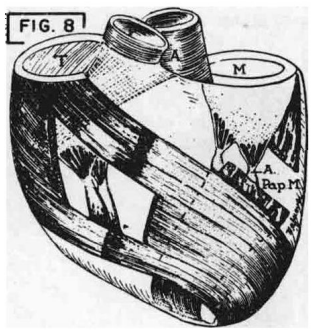
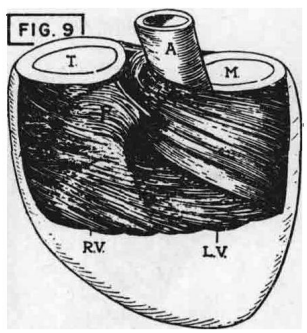


FIG. 6. Superficial muscle layers of the maximally contracted human heart, seen from the ventrocranial aspect after separation of the atria (above) from the ventricles (below). The position changes of the great vessels and the ventricle outlines during relaxation are indicated by dashed lines. [From Spalteholz (148).]

According to Benninghoff (10), who uses a somewhat different classification, there are three interconnected systems which intersect rectangularly: *a*) the outer longitudinal fibers which connect to the outer contour fibers at the ostia; *b*) the ring fibers which encircle the entire chamber and curve around to form fibers of the ventricular septum; *c*) the internal longitudinal fibers which run from the contour fibers toward the apex (figs. 11 and 12). Benninghoff (10) analyzed the function of these various bundles on the basis of careful comparative anatomical studies and in vivo observations. He emphasized the concept that crossing of the fiber layers at right angles results in an over-all reduction of the cavity size, as first postulated by Carl Ludwig. Each of the three systems affects the entire heart and at the same time each of the ventricular cavities. They act in such a manner that a reduction of the heart chambers does not occur equally in all directions but in such a manner and sequence that the cavities are emptied toward their outflow tracts. The evolution proceeded as follows: in lower vertebrates (fish, amphibia) there are no tendinous elements and all muscle bundles are ring shaped. In the mammalian heart secondary valves (atrioventricular) are formed from which the connective tissues of the fibrous rings of the atrioventricular valves and of the chordae tendineae originate and become inserted into the course of the ring-shaped muscle. The fibrous rings become connected to the roots of the arteries and form the solid trigona fibrosa, which furnish new insertions for many myocardial fibers (see fig. 4). In the evolutionary process the







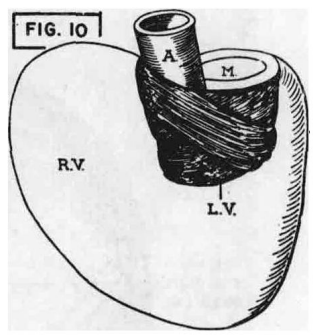


FIG. 7. The superficial bulbospiral muscle as seen from the front of the human heart. A = Aorta; M = mitral orifice; P = pulmonary artery; T = tricuspid orifice; AT = anterior leaflet of tricuspid valve; MT = medial leaflet of tricuspid valve. A V-shaped section is cut from those fibers encircling the left ventricle subendocardially, so that the mitral valve may be seen. A similar band on the right is not sketched in. [From Robb & Robb (136).]

FIG. 8. The superior sinospiral muscle as seen from the antreior surface of the heart. Symbols as in fig. 7. Again the subendocardial layer has been cut through in order to show deeper structures. The window in the right ventricular wall shows the fibers from the trabeculated area running up to the anterior and medial leaflets of the tricuspid valve. In both of these superficial muscles, blood vessels follow the muscle strands as they encircle the apex. [From Robb & Robb (136).]

FIG. 9. The deep sinospiral muscle as seen from the front. Note the division of the muscle at the posterior inter-ventricular sulcus, with fibers passing anteriorly to form most of the basal two-thirds of the septum; these septal fibers lie just distal to the band of the left head of origin at the base of the aorta. Symbols as in fig. 7. [From Robb & Robb (136).]

FIG. 10. The deep bulbospiral muscle, a powerful sphincter encircling the left ventricular base and enclosing both the aorta and the mitral orifice within its sweep. [From Robb & Robb (136).]

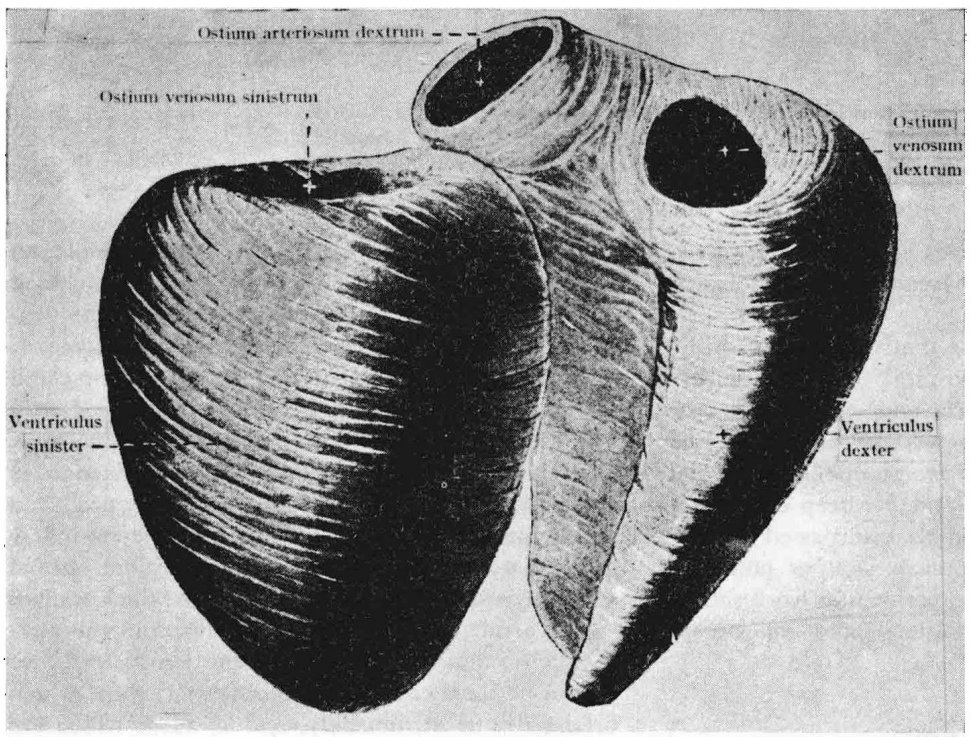


FIG. 11. Human ventricular myocardium after removal of the superficial muscle layers (seen from the caudal aspect). [From Spalteholz (148).]

spongiosa (spongy network of muscle fibers) is gradually reduced by the increasing compacta (solid tissue of muscle fibers). The phylogenetic remainders of the spongiosa are the muscular trabeculae, which are only moderately developed in the mammalian heart and are almost completely replaced by compacta in the bird heart. In this respect the birds represent the highest functional development. According to Benninghoff (10) the spiral course of the muscle bundles toward the heart skeleton and the

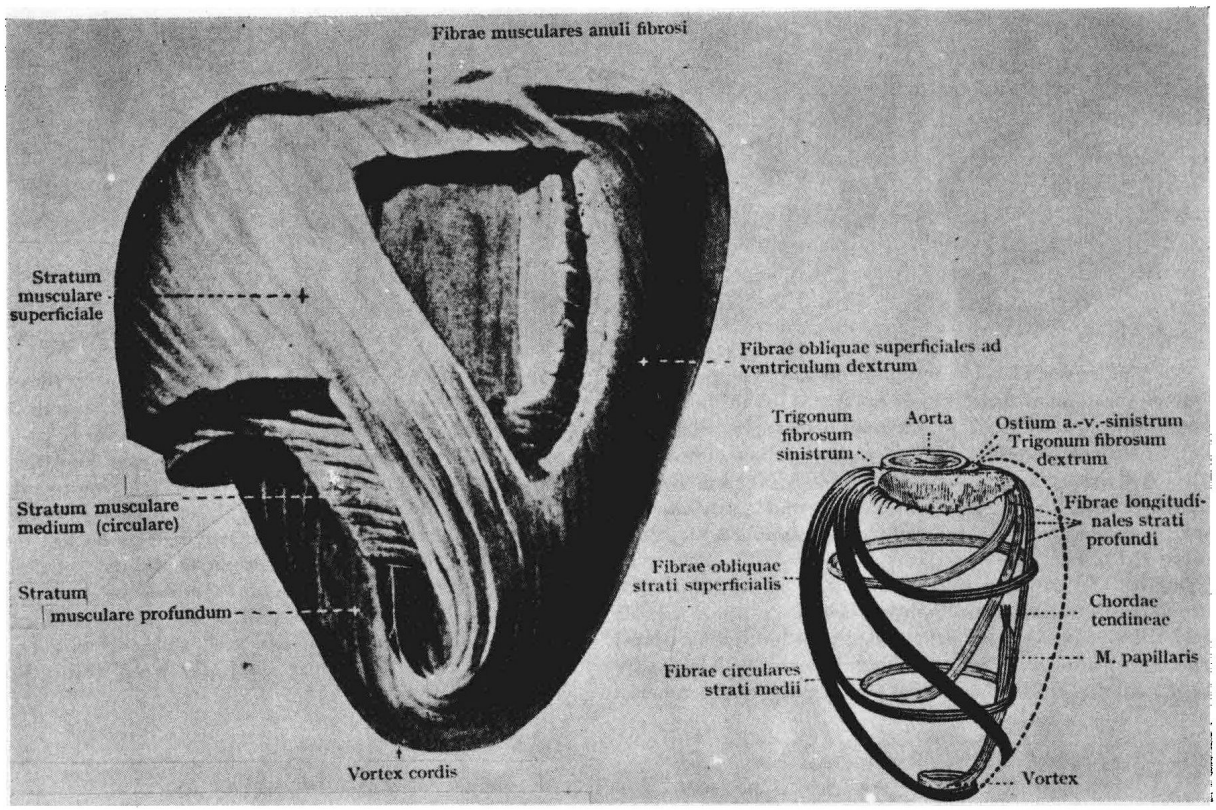


FIG. 12. Course of the left ventricular muscle fibers. *Left:* preparation of human heart after partial removal of the superficial and medial muscle layers (seen from the dorsal aspect). *Right:* schematic presentation of the course of the muscle fibers as viewed from the dorsal aspect. [From Spalteholz (148).]

vortex formation near the apex are more pronounced in the mammalian than in reptile and bird hearts (see fig. 12, right).

Rushmer (139) points out that the division of the heart musculature into "sinospiral" or "bulbospiral" bundles is rather arbitrary and complicates the functional analysis. He suggests the division of the ventricular musculature into two groups of myocardial bundles, the spiral muscles and the deep constrictor muscles (fig. 13). He states in his unsurpassed description, the "functional anatomical analysis points to the direction physiological experimental work should pursue to verify . . . postulations and to obtain quantitative measurements."

Architecture of the Atrial Myocardium

The atria supply blood to the ventricles through three mechanisms: *1*) passively, during the first part of their diastole, by serving as blood collecting chambers as long as the atrioventricular valves are closed by the high ventricular pressure; *2*) still passively, during the second part of their diastole, by serving as channels to permit the passage of blood from the systemic or pulmonary veins into the ventricles once the atrioventricular valves are opened; *3*) actively, during atrial systole, by contracting and thereby pushing some blood into the ventricles shortly before the ventricular myocardium begins to contract. Since usually only a small fraction (10–30%) of the blood for ventricular filling is actively propelled by the atrial musculature and the resistance to inflow into the ventricular cavity is negligible, the normal atrial myocardium does not need to be thick walled.

The arrangement of the muscle fibers in the atria is much simpler than that in the ventricles. Two groups of fibers can be distinguished: *1*) those which belong to one atrium only, and *2*) those which are common to both atria (151).

Group 1: The fibers which lie in the wall of each atrium form muscle rings around the entrance orifices, i.e., the pulmonary veins in the left atrium and the coronary and caval veins in the right atrium. These annular fibers may act as sphincters, possibly

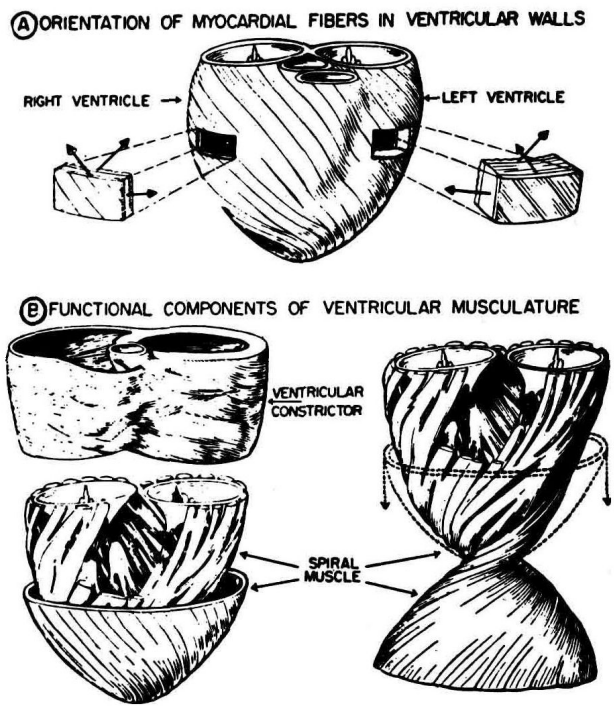


FIG. 13. Muscular structures of the ventricles diagrammatically arranged so as to reveal their functional components. *A:* blocks of tissue removed from the walls of the ventricles are composed of three layers of muscle. The myocardial fibers in these layers are oriented roughly in the three general directions indicated by the arrows. *B:* from a functional point of view, the ventricles are formed of two sets of myocardial bundles: *a*, the internal and external layers of spiral muscle, which enclose *b*, the ventricular constrictor muscles. The internal and external investments of the ventricular chambers are composed of the same muscle bundles, which are strongly twisted at the vortex and spiral in opposite directions from the apex toward the base. [From Rushmer (139).]

impeding, though not completely blocking, the backflow of blood into the veins during atrial systole. Looped fibers are also found which run from the anterior to the posterior segments of the atrioventricular junction, directly beneath the endocardium. At many places these fibers bulge into the atrial cavities forming various ridges which are most conspicuous at the inner walls of the atrial appendages, where they are named musculi pectinati from their resemblance to a comb.

Group 2: The fibers common to both atria are less numerous and lie superficially with respect to the proper fibers of each individual atrium. They consist of two thin muscle sheets which extend in a transverse direction from one atrium to the other. They can be subdivided into anterior and posterior fascicles. The muscle fibers of the atria and ventricles are separated by connective tissue except at one place, known as the atrioventricular bundle or bundle of His.

The atrial cavity is surrounded by the thin myocardial fibers of both groups arranged in layers which are partly parallel and partly crisscrossed. The concerted action of all fibers is that, upon their contraction, they diminish the size of the atrial cavity and push blood into the region of least resistance, i.e., primarily into the ventricles, secondarily into the venous orifices. In addition to the main atrial cavity, there is an adjoining cavity formed by the lumen of the atrial appendage, also called "auricle" because of its resemblance to a little ear. The function of the auricles is unknown. Excision of the auricles in various operative procedures does not influence the circulation noticeably. Yet one cannot state bluntly that the atrial appendages have no function at all, since in a complex system, such as the heart, the function of a missing part may often be taken over or substituted by increased activity of other components. The mere presence of the atrial appendages results in an increase in the cardiac reserve. According to Benninghoff (10) and Böhme (14), the atrial appendages fill the space which is created within the pericardial sac during ventricular systole, as the ventricles eject blood into the large arteries and decrease in size. During this period the atrial appendages accommodate a considerable amount of blood. This blood is immediately available at the beginning of the rapid ventricular filling phase to be transferred into the ventricular cavities.

PRESSURE AND FLOW EVENTS DURING THE CARDIAC CYCLE

Historically the cardiac cycle was first divided into "systole," or period of contraction, and "diastole," or period of relaxation of the ventricles. It was soon recognized that the terms systole and diastole should refer equally to the atrial contraction and relaxation, although the ventricular events were most conspicuous in the gross observation of cardiac activity. Since the atrial contraction precedes that of the ventricle, terminological difficulties arose as to which systole was meant in describing the time sequence of cardiac events. As knowledge about the heart's action increased, it was also deemed necessary to subdivide the cardiac cycle in greater detail [see also Mackenzie (110)]. With the advent of methods for precise pressure recording from the cardiac chambers and great vessels, the ventricular pressure tracings

became the deciding guidelines for characterizing the phases of the cycle. The generally adopted subdivisions of Wiggers (156) stem from this era. Since other landmarks of cardiac activity such as flow, volume changes, or biochemical processes were difficult to record adequately, they were only correlated with the pressure curves at a later date.

It is still impossible to subdivide the cardiac cycle according to the most important physiological events: the blood flow into and out of the cavities. The approximate beginning and end of systolic ejection can be determined from simultaneous pressure tracings in a ventricle and in an arterial outflow tract. However, the precise timing of flow is only possible through direct recording of flow either at the root of the aorta or at the pulmonary artery [see also Moscovitz & Wilder (117)]. The recent advent of refined flowmeters will probably necessitate some adjustments in the original Wiggers scheme of the cardiac cycle. For the time being it is still preferable to retain the well-established scheme and to fit modifications into it, rather than to advocate a completely new one [see also Horowitz (80)].

Figure 14 [modified from Wiggers (156, 159)] illustrates in schematic form the sequence of pressure events during the cardiac cycle in the left ventricle, left atrium and aorta, and the volume changes in the combined ventricles [from Henderson (69)]. For time correlation, tracings of the heart sounds and of the electrocardiogram are added. This composite chart is mainly based on curves obtained in animal experiments.

The cycle is divided into two periods, systole and diastole. The former begins with the rise of ventricular pressure caused by ventricular contraction (fig. 14, 1) and ends at the onset of myocardial relaxation, 4, at the point when ejection actually ceases. This point then also represents the beginning of the diastole. The systolic period is subdivided into 1–2, isovolumetric ventricular contraction (50 msec); 2–3, maximum ventricular ejection (90 msec); and 3–4, reduced ventricular ejection (130 msec). The diastolic period is subdivided into 4–5, isovolumetric ventricular relaxation (120 msec), which includes a phase occurring just prior to the incisura and formerly called protodiastole (40 msec), plus the phase formerly known as isometric relaxation (80 msec); 5–6, rapid ventricular filling (110 msec); 6–7, slow ventricular filling or diastasis (190 msec); and 7–1, ventricular filling by atrial contraction (60 msec).

Numerous other cyclical events occur with each heart beat. They are correlated timewise with the phases of the pressure-volume cycle as follows.

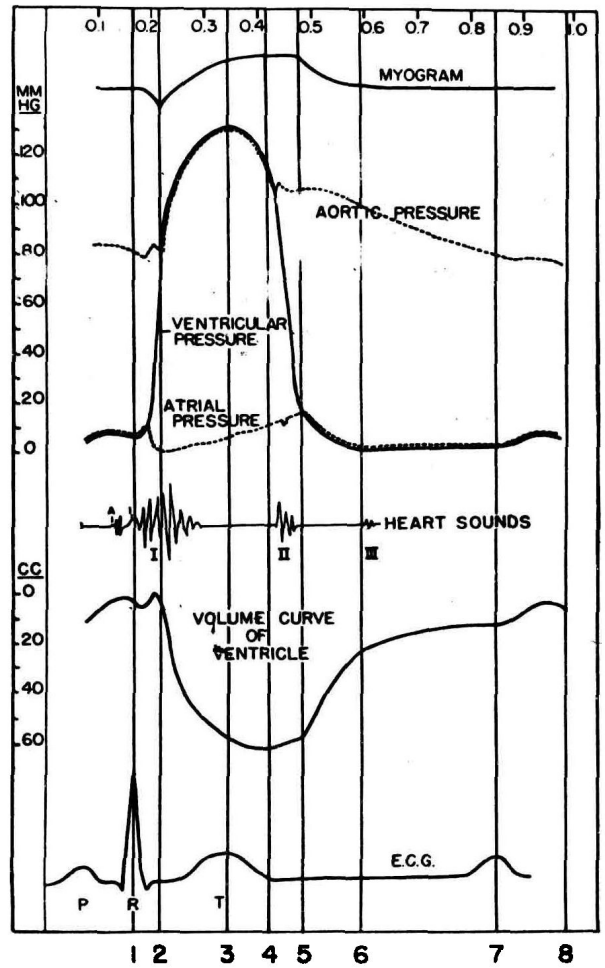


FIG. 14. Scheme of the cardiac cycle. Time, totaling 1 sec, on upper margin. Numbers under lower margin indicate beginning and end of phases. Period of ventricular systole lasts from 1 to 4, period of ventricular diastole lasts from 4 to 1. Detailed description in text. [Figure (but not numbers in text) slightly modified from Wiggers (159).]

1–2: *Isovolumetric ventricular contraction.* During this phase the myocardium builds up tension and this gives a fast rise of intraventricular pressure without change in the volume of blood contained in the ventricular cavity. The intraventricular pressure must rise to the level of the diastolic pressure prevailing in the aorta (or pulmonary artery) before blood can be ejected from the ventricles during the next two phases. The term "isovolumetric contraction" suggested by Rushmer (139) should supersede the older term "isometric contraction," since at the beginning of this phase there is an actual shortening

of the fibers of the papillary muscles and trabeculae carneae which results in a tension of the chordae tendineae, and an approximation of the atrioventricular valves (139). Simultaneously, there is a passive stretching of the other still relaxed myocardial layers, mainly those of the outer walls of the heart [see also Hawthorne (67), Anzola (4), and Burton (29)]. The older term "isometric contraction" had the misleading implication that all myocardial fibers contract simultaneously and isometrically from the very start. Since in fact some muscle fibers shorten whereas others are passively lengthened during this phase, while the intraventricular volume remains constant, the term isovolumetric contraction provides a more accurate description than isometric contraction. Apparently instrumentation has not yet been refined sufficiently to decide whether or not there is in this phase a brief "latent relaxation" of cardiac muscle fibers as there exists in skeletal muscle fibers.

The shortening of the ventricle in the longitudinal axis results in a descent of the atrioventricular junction which in turn expands the atrial cavities. This leads to a precipitous lowering of the atrial pressure (fig. 14) which is often observed even before the ventricle ejects blood. The ventricular muscle fibers contract in a successive order, probably following the same time sequence as their depolarization (75, 142). As a consequence the blood contained in the ventricular cavity is pushed from the apex region toward the center of the ventricle and moves thereby closer to the outflow tract. The subsequent ejection from the ventricles can be looked upon as a continuation of the intraventricular movement of blood which already starts before the semilunar valves open. At the same time the ventricular cavity changes from a cylindrical to a more spherical shape, which from the energy standpoint represents a more economical way of discharging the ventricular content, once the aortic diastolic pressure is overcome. As pointed out by Rushmer (139), the asynchronous contraction of the ventricular myocardium readily explains the brief upward deflection at the beginning of isovolumetric contraction in the ventricular volume curve described by Wiggers (156) in fig. 14. This was formerly interpreted as an artifact in the recording.

Some arbitrariness is involved in determining accurately the start of isovolumetric contraction. In all pressure tracings the upward movement begins slowly in the form of a rounded curve. There is no abrupt beginning, inflection, or break. This becomes especially evident if one records the pressure events by drawing out the time axis with fast moving paper as can be easily done today with electrical recording apparatus. The rounded beginning of the upward limb results from the combined effect of *a*) the contraction of the papillary muscles, and *b*) the simultaneous passive distention of some of the muscle fibers in the ventricular wall. Whenever the transfiguration of the ventricle causes a detectable rise of intraventricular pressure, then by convention the ventricular isometric contraction is said to begin. The fact that the different strands of myocardial fibers contract in sequence rather than simultaneously may also explain the great variability of the slopes of the pressure tracings in the early part of isovolumetric contraction.

The steepness of the slope during isovolumetric contraction is predominantly determined by the forcefulness of the fiber contraction. If the difference between the end-diastolic ventricular and end-diastolic aortic pressure remains unchanged, the duration of the ventricular isovolumetric contraction is shortened by sympathetic or sympathomimetic stimulation and lengthened by agents or conditions which depress the sympathetic control of the heart [Cotton & Maling (35), Gleason & Braunwald (54); see also Reeves *et al.* (133)]. Thus in forcefully contracting ventricles, the slope will be steeper than in feebly contracting preparations.

The atrioventricular valves close approximately at the beginning of isovolumetric contraction; the opening of the semilunar valves marks the end of this phase. The precise moment of the valve actuation is difficult to establish experimentally (discussed in the section on heart valves). In the interval between closure of the atrioventricular valves and opening of the aortic and pulmonary valves, the blood contained in the ventricular cavities is temporarily isolated from the fluid columns in the atria and arteries. However, the ventricular content does not remain still (10). In fact the blood which rushed into the ventricles at high velocity during diastole may aid in expanding the ventricular cavities. Since the inflow is primarily directed toward the apex, it is this part of ventricular wall which could be preferentially expanded. As the papillary muscles and trabeculae carneae begin to contract, the movement of the blood is deviated toward the outflow tract. This change in direction of flow is favored anatomically by the fact that the axis of the inflow tract and that of the outflow tract form an angle. In other words, the inflowing blood probably does not come to a complete standstill in order to reverse its direction of flow for ejection into the

arteries, but rather it keeps flowing in a curve from the main direction of the inflow tract toward the outflow tract. This translocation of blood within the ventricle during the isovolumetric phase is energy preserving. In fact, there seems to be rather little turbulence and not always complete mixing of blood during this "intraventricular" streaming from the inflow side to the outflow region. This explains why the streamlining of flow in the venous circulation is not always completely interrupted by the passage of blood through the ventricle. For example, the systemic venous blood is transferred into the pulmonary arteries in such manner that superior caval blood reaches predominantly the right lung and inferior caval blood the left lung [see also Bucher *et al.* (27)]. Obviously, the possibility of incomplete mixing deserves attention when samples of so-called mixed venous blood are drawn.

How much does the velocity of the blood flow decrease during the transit in the ventricle? In the resting organism with a slow heart rate, the velocity of blood streaming into the ventricle toward the end of diastole is rather small, as may be surmised from the fairly flat portion of the ventricular volume curve. When the cardiac output is elevated, the velocity of the intraventricular flow during isovolumetric contraction will probably increase for two reasons: *1*) the velocity of end diastolic ventricular inflow increases through a shortening of diastole due to high heart rates and through a more forceful atrial contraction; *2*) the transit time through the ventricle is shortened by the more powerful and often shorter myocardial contraction. Such higher intraventricular flow velocities under sympathetic activity could then result in a better energy conservation by not letting the speed of blood flow slow down too much before ventricular ejection begins again.

[It is the feeling of the editors that there is not sufficient evidence to show that continued translocation of blood within the ventricular cavity during isovolumetric contraction could contribute significantly to the subsequent ejection. Ed.]

2–4: *Rapid and reduced ventricular ejection.* As soon as the pressure in the ventricular cavities exceeds that in the aorta or the pulmonary artery, the blood is suddenly ejected. Although flow is created by a difference between the intraventricular and arterial pressures, an inspection of pressure curves alone, simultaneously recorded from the ventricle and the root of the artery, furnishes only meager information about the rate of volume flow and its time course. However, from simultaneously recorded flow and

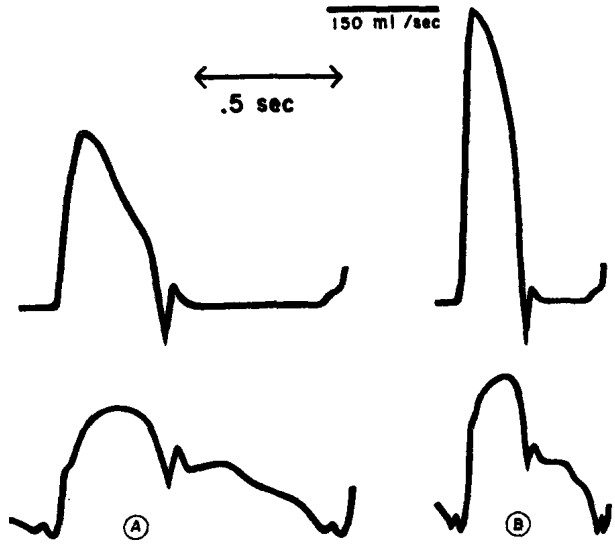


FIG. 15. Phase relationships between pressure and flow as revealed by simultaneously recorded curves from the ascending aorta of a conscious dog. *Upper tracings:* rate of volume flow measured with a permanently implanted electromagnetic flowmeter. *Lower tracings:* aortic pressures obtained through a permanently implanted cannula leading to a strain gauge manometer. *A:* curves from the quiet reclining animal. *B:* curves from the animal running behind a car during moderate exercise. [Original curves by the courtesy of Frederick Olmstead, Cleveland Clinic, Cleveland, Ohio (personal communication, 1961).]

pressure curves in the aorta or in the pulmonary artery, the process of ventricular ejection is now fairly well understood [Wetterer (155)]. The ejection starts abruptly (fig. 15). The blood column in the root of the aorta, which is practically stationary at the end of diastole and during isovolumetric contraction, is rapidly accelerated and pushed toward the periphery. The greatest flow acceleration occurs during the steeply ascending limb of the aortic pressure curve, so that the highest flow rate (peak of the flow curve) is actually reached prior to the summit of the pressure curve. When the flow then becomes less rapid, the phase of reduced ejection is said to begin. The border between rapid and reduced ejection is quite arbitrary. When only pressure and cardiometer curves were available [Wiggers (156)], it was difficult to determine from the gradual leveling off of the downward limb of the volume curve when the rapid ejection started to slow down. The summit of the ventricular pressure curve was thought to indicate the end of rapid ejection (fig. 14). It is now known that the flow slows down earlier, since the peak of the flow curve definitely precedes the peak of the ventricular or aortic pressure curve (upper tracings