Cardiac

PRESSURES AND PULSES

ALDO A. LUISADA and CHI KONG LIU

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A Manual of Right and Left Heart Catheterization

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Introduction

Catheterization of the right heart was initiated by a German surgeon (Forssmann) in 1929. It is not surprising that a surgeon, and not a physician, was the first to try this technique. At that time, physicians were far less familiar with the use of catheters and intravenous drip methods. The only surprising fact is that a surgeon rather than a urologist may claim to have been the first.

While Forssmann's attempts on himself were considered at the time only an unusual technical exercise, Cournand and his group perfected this method to such a degree that it was gradually accepted and utilized by more and more researchers. Dexter, Lenègre, McMichael, and Burchell, together with their teams, further developed the techniques which are currently used.

The method, as originally described, was based on the oxygen determination of blood samples aspirated from the chambers of the right heart or pulmonary artery, and on pressure measurements. Later, catheterization of the chambers of the left heart through septal openings added interesting data to those already known. Intracardiac electrocardiography, determination of cardiac output by means of the Fick principle, and study of the pulmonary venous pressure by firmly wedging the catheter in a pulmonary arteriole were subsequent additions.

Left heart catheterization is a relatively recent development which will very likely extend the realm of this technique to include a much larger group of patients. Cases with aortic valve lesions (which are outside the realm of right heart catheterization) and cases with mitral valve lesions (in which right heart catheterization gives only indirect data) may be studied by left heart catheterization.

Pressure tracings are usually taken with a conventional film speed which is inadequate for the study of accurate details of the pulse. This small monograph is based on tracings recorded with a somewhat different technique, aiming to study in detail various patterns of the pressure pulses of the heart and vessels.

Cardiovascular physiology, details of technique, formulas used in catheterization, and a study of artifacts have been included in this volume to simplify the task of young cardiologists who may wish to begin working in the fascinating and ever-expanding field of cardiac catheterization.

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ALDO A. LUISADA
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Chicago, 1956

Contents

CHAPTER ONE

The Cardiac Cycle

CONTRACTION OF THE ATRIA

The cardiac cycle starts with *the contraction of the atria*, also called "atrial systole." A wave of contraction follows that of excitation, moving downward from above,[1] thus creating a propulsive wave toward the ventricles. An appreciable backflow toward the large veins is prevented by the initial contraction of the musculature at the opening of the veins, and by the high venous pressure accompanying this phase.

Atrial contraction takes place during that short phase which immediately precedes ventricular contraction, the *presystole*. As the A.V. valves are open during atrial contraction, only a moderate rise in pressure takes place within the atria, and the contraction is mainly revealed by movement of blood (Fig. 1).

Atrial contraction is not indispensable for ventricular filling because the greatest part of this ventricular phase occurs in early diastole. Still, contraction of the atria completes ventricular filling and is one of the factors upon which the normal function of the A.V. valves is based.[1] In rapid heart action and in mitral stenosis, atrial contraction may acquire a much greater importance.

CONTRACTION OF THE VENTRICLES

Initiation of ventricular contraction increases the pressure in the ventricles and closes the *atrioventricular valves* (tricuspid valve in the right heart, mitral valve in the left heart). Immediately afterwards, the contraction of the papillary muscles prevents an eversion of these valves and permits a further rise of pressure to a point equaling and then exceeding the pressures existing in the aorta and in the pulmonary artery. In this short period, the ventricular contraction builds up pressure without causing motion of blood. This short phase is called *the period of tension* or *the period of isometric contraction* because the muscle fibers of the ventricles build up tension steadily even though unable to become shorter.

At the onset of ventricular contraction, the entire myocardium has been excited. Owing to the latency between excitation and contraction, however, those fibers which were excited first start contracting first. Then, more and more fibers contract. For this reason, ventricular pressure rises slowly at first, then very rapidly.[2]

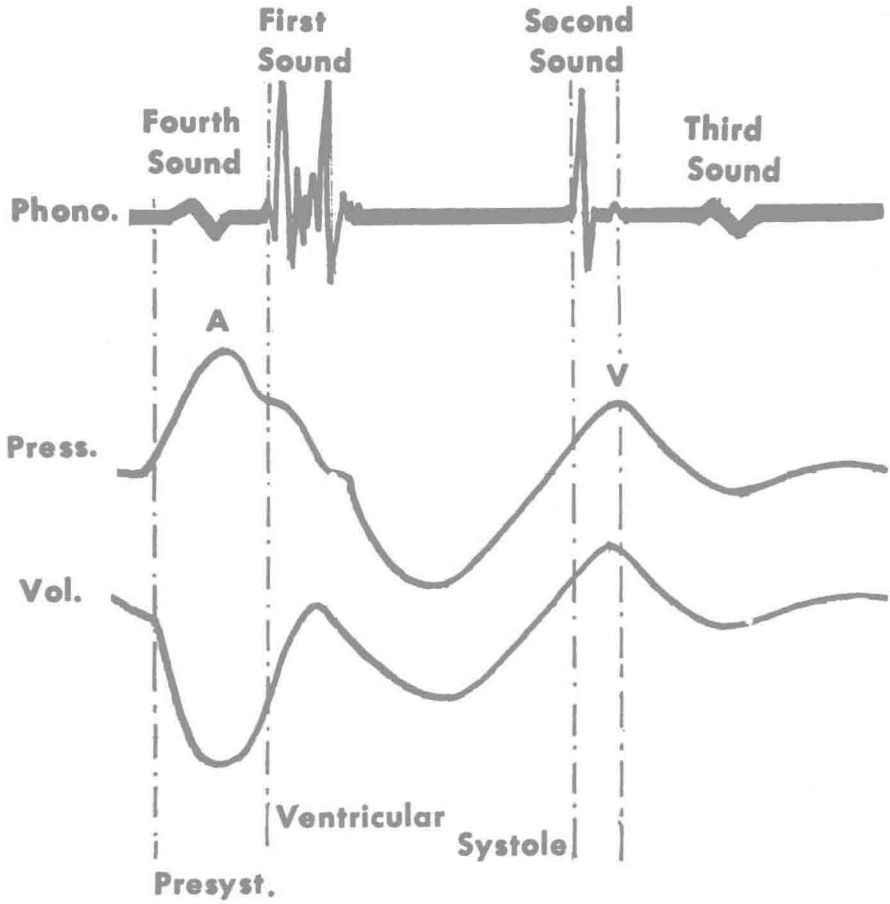

Fig. 1—Changes of atrial pressure (press.) and volume (vol.) during the cardiac cycle, and correlation with heart sounds tracing (phono).

As soon as the ventricular pressure exceeds that of the respective artery, *the semilunar valves open* and *the outflow begins*. During this period of *outflow* or *ejection*, the fibrous septum which supports the A.V. valves is lowered by the contraction of the ventricles (papillary muscles, free ventricular wall, and intraventricular septum). As a consequence, a remarkable increase in size of the atria takes place during systole, causing suction of blood from the veins.

From beginning to end, the ventricular pressure maintains a steady course. Still, during the last part of ejection, outflow is more limited. Therefore, ejection has been divided into two parts: *maximum ejection*, which includes about one half of the time and expels about two thirds of the blood, and *reduced ejection*, which expels about one third of the blood in the last half of the time.[2]

MOVEMENTS OF THE VALVES

Despite their apparently delicate structure, the *flaps* of *the A.V. valves* have considerable strength and resistance. When closed, they do not merely touch, but form a surface contact without folds. Closure is started by the eddy currents and is increased by the ventricular contraction which immediately follows. Eversion is prevented by the chordae tendineae, held by the papillary muscles. The musculature of the septum and

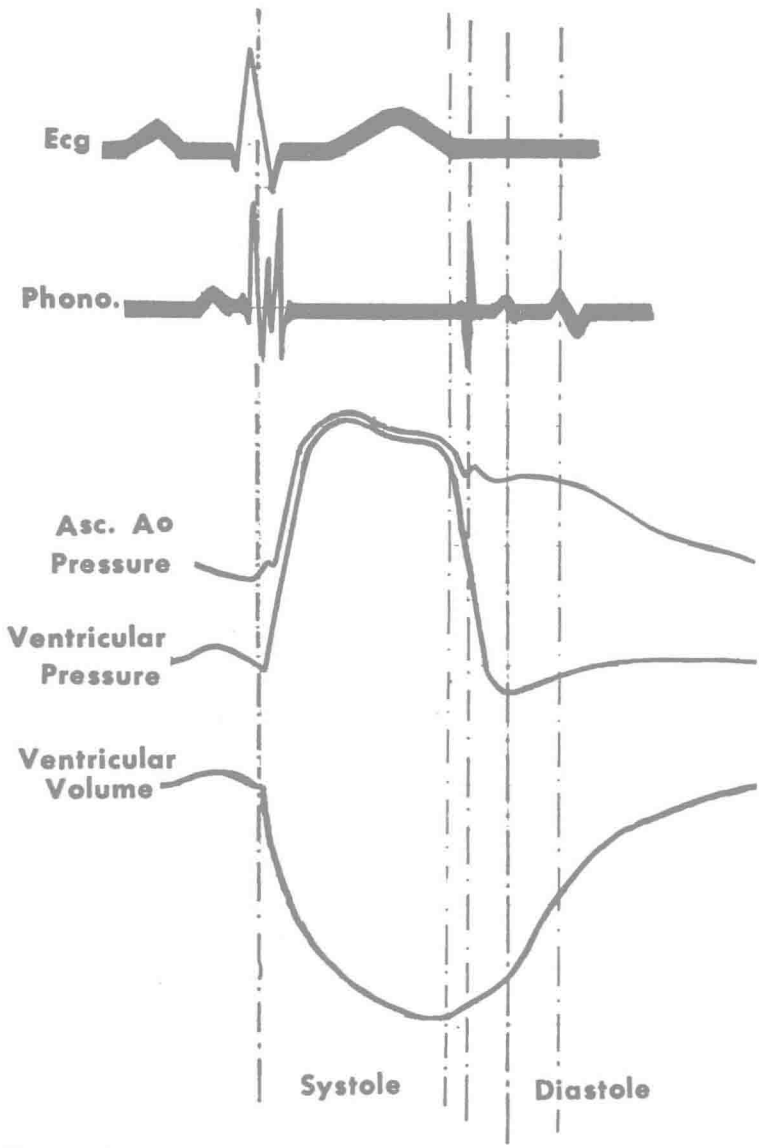

Fig. 2—Changes of ventricular pressure and volume, and pressure in the ascending aorta during the cardiac cycle; correlation with heart sounds (phono) and electrocardiogram (ECG).

of the papillary muscles is the first to contract, insuring a timely closure of the valves. The termination of atrial contraction contributes to the closure of the A.V. valves, because the leaflets are brought into position by the eddy currents set up by the flow through the orifices, and by a reversal of the pressure gradient. This is shown by the temporary valvular insufficiency which frequently develops in cases with incomplete A.V. block. In the event of delayed A.V. conduction, there may be a double closure of the A.V. valves: the first at the end of atrial contraction, the second at the beginning of ventricular systole.

Since the efflux of blood from the branches of the aorta is faster than ejection from the left ventricle, the pressure gradually declines in the aorta during the second part of systole. This drop of aortic pressure may be responsible for a slight drop of pressure in the ventricular curve.

The *semilunar valves of the aorta and pulmonary artery* resemble pockets attached to the wall of the vessel. The blood contained in the pockets keeps the valves away from the wall. Both the reversal of the gradient of pressure created by the sudden cessation of outflow and the eddy currents determine closure of these valves at the end of ventricular systole. Firm attachment of the valves, muscular support from the ventricular base, and lateral apposition prevent any possibility of eversion, in spite of the lack of chordae tendineae.[3]

CHANGES OF CARDIAC DIAMETERS

During ventricular contraction, *all diameters of the heart decrease*: the base is pulled downward and the large vessels are stretched while the apex does not move upwards (Fig. 3). The spiral arrangement of the muscular bundles of the ventricles makes their contraction very efficient, so that the blood is virtually wrung out. It also causes the heart to rotate to the right, pressing the apex more firmly against the chest wall. This, together

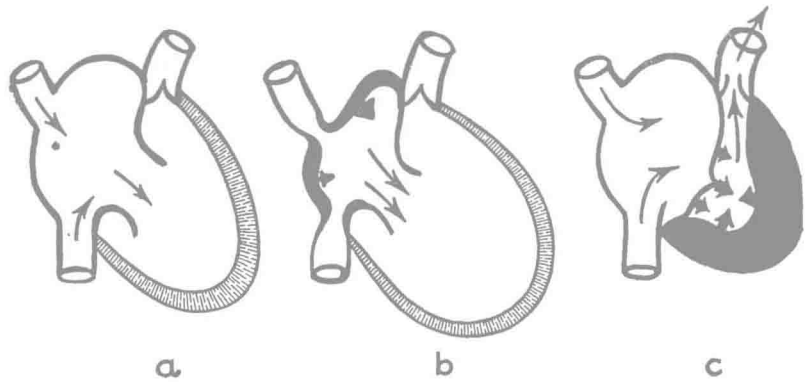

Fig. 3—Schematic changes of cardiac shape during contraction. (a) Mid-diastole (free flow from veins to atria and ventricles). (b) Presystole (atrial contraction completing ventricular filling). (c) Ventricular contraction (expulsion of blood into the arteries; lowering of A.V. floor; suction in the atria and veins).

with the increased firmness of the ventricular mass, is the cause of the so-called *apex beat*. In spite of the progressive contraction of the free ventricular walls and of the septum, a small amount of blood is left within the ventricles even in normal conditions (*residual blood*).

The *interventricular septum* seems to have an important dynamic role; a large part of right ventricular ejection seems to be due to this structure.[4]

VENTRICULAR DIASTOLE

At the end of ventricular systole, ventricular pressure drops to zero. Following an extremely brief interval of latency (so-called *protodiastole*)[1] the semilunar valves of the aorta and pulmonary artery close. A short time interval separates this phase from the subsequent opening of the A.V. valves: the *isometric relaxation period*. Ventricular filling starts after the end of this period, e.g., following the opening of the A.V. valves (Fig. 2).

Filling of the ventricles has the following features:

(a) An initial phase of *rapid passive filling* (*early diastole*). This is caused by the difference in pressure between the full atria and the empty ventricles. At this time, the entire venoatrial reservoir experiences a drop in pressure due to acceleration of the stream after the opening of the atrioventricular valves.

(b) A phase of *slow passive filling* (*mid-diastole or diastasis*). The gradual filling of the ventricles slows down the inflow and a gradual pressure rise takes place in the venoatrial reservoir as well as in the ventricles.

(c) A late phase of *rapid active filling* (*presystole*) caused by the atrial contraction which completes ventricular filling. As soon as the atrial contraction is completed, the ventricles start contracting, because the descending stimulus has already reached the ventricular myocardium.

It should be kept in mind that, during ventricular diastole, the atrium and ventricle of each side of the heart form like a single chamber.

DURATION OF CARDIAC PHASES

The following time intervals, in seconds, may be considered typical of a normal heart with a rate of 68 beats per minute[1]:

Ventricular systole		*Ventricular diastole*	
tension	0.06	protodiastole	0.04
maximum ejection	0.12	isometric relaxation	0.07
reduced ejection	0.16	rapid filling	0.10
		slow filling	0.22
Total	0.34	atrial dynamics	
		dynamic interval	0.05
		atrial contraction	0.06
		total	0.54

Because of the thinness and weakness of the *right* atrial wall and its distensibility, the filling volume of this chamber is about twice that of the *left* atrium.

As atrial contraction lasts but a small fraction of the total cycle (less than one tenth of a second), the atrial wall is relaxed during most of ventricular diastole and during all of ventricular systole. Thus the atria act as a reservoir for the blood coming to the heart.

The traction developed by the ventricular muscles and septum on the atrioventricular junction during systole dilates the atria by causing a phenomenon of suction. This is rapidly transmitted to the venous system and accelerates the flow of blood toward the atria[1, 2] (Fig. 1). This is proven not only by physiologic experiments, but also by the fact that in clinical tracings of normal subjects, recorded from the veins or atria, the pressure drops during ventricular systole.

The *atrial appendages* seem to have little propulsive function and serve as complementary spaces which fill the deep niches at the base of the heart during ventricular systole.

In abnormal conditions, as in cases with rapid heart rate, diastole shortens tremendously. In such cases, atrial contraction may include most or all of diastole and acquire much greater importance.

THE HEART SOUNDS

Auscultation of the normal heart usually reveals two sounds (or tones); occasionally, three. Recording of the normal heart sounds by means of phonocardiography may reveal as many as four sounds.[7]

The *first heart sound* takes place at the beginning of ventricular systole and lasts through the tension period and the beginning of the ejection period. The *second heart sound* is shorter; it takes place at the end of systole, during the phases of protodiastole and isometric relaxation. The name "systolic sounds" has been suggested by one of the authors for these two constantly heard sounds.[8] The other two sounds, less frequently heard, take place during diastole. The name "diastolic sounds" has been suggested for them.[8]

The following dynamic phenomena take place at the time of the heart sounds:

Systolic Sounds

First sound:

Initiation of ventricular systole
Closing of the A.V. valves
Opening of the semilunar valves

Second sound:

End of ventricular systole
Closing of the semilunar valves
Opening of the A.V. valves

Diastolic Sounds

Third sound: Rapid passive filling of the ventricles

Fourth sound: Rapid active filling of the ventricles due to atrial contraction.

The mechanism of production of **the complex of the first sound** has been repeatedly investigated but the conclusions of the various researchers are by no means in agreement.

Several authors suggested a purely muscular origin of the first sound. Others, particularly Dock[9] and Kountz and coworkers,[10] believe that the first sound is due to the sudden tension of the previously slack fibers of the A.V. valves. A theory of mixed origin was advocated by Wiggers,[1] who denied the possibility of separating in the tracing the vibrations caused by the various structures. He postulated the theory that vibrations were set up in the A.V. valves, the chordae, and the ventricular walls.

The phonocardiographic studies of Orias and Braun-Menendez[7] and Rappaport and Sprague[11] led to the view that the first sound is due to four separate factors (atrial, muscular, valvular, and vascular). However, while two larger vibrations were recognized as coinciding with the two main valvular events, no separate vibration or group of vibrations was found to result from muscular contraction.

Experimental studies conducted by one of the authors with Alimurung and Lewis[12] proved: (a) that the two main vibrations of the empty heart are extremely faint and barely appreciable; and, (b) that the first sound is the result of both muscular and valvular factors, a sudden change in muscular tension first closing the A.V. valves and then opening the semilunar valves. This causes a double vibration of the cardiac wall, including high-frequency and low-frequency components, which is further transmitted to the chest wall. Although simultaneous with the action of the valves, these vibrations are likely to arise in both the valvular and the muscular structures as a response to rapid changes in tension and pressure. In other words, the first sound is the audible expression of that complex movement of the heart which is also revealed by the first part of the apical thrust.

The **second sound complex** is caused mainly by the closing of the semilunar valves and the resulting vibrations of the heart and chest wall. However, vibrations of vascular origin[11] and even the opening of the A.V. valves[11, 13] contribute to its formation, at least in certain cases.

The **third sound** arises in the ventricular wall as the result of the vibration caused by the onrush of blood at the moment of rapid passive filling of the ventricles. The third sound has been attributed to valvular vibrations (a theory which is not too likely to be confirmed) or to the apical impact on the chest wall (which may be only a concurrent factor).

The **fourth sound** arises in the ventricular wall and is caused by the blood rushing into the ventricular chambers because of atrial contraction.[7] Earlier vibrations, recorded from the esophagus, can be attributed to the atrial contraction *per se*.

THE ARTERIAL PULSE

The left ventricle empties itself at each beat into the aorta. This vessel stores a portion of the blood received so that neither the pressure nor the flow fall too low before the next ventricular contraction. The aorta offers little resistance to the flow of blood. Its great distensibility, however, gives a variable resistance according to the rate at which the pressure changes, e.g., the heart rate.

When the aortic pressure rises suddenly during the ejection phase of ventricular systole because of sudden penetration of blood expelled from the left ventricle, the aortic volume increases considerably, creating a new space (*aortic reservoir*). When the pressure falls during ventricular diastole, the retraction of the wall can be compared to the reinjection of blood from the reservoir into the aorta, so that the pressure tends to be maintained in spite of the lack of flow from the heart. In addition to this change in the size of the aorta, the *systolic discharge* of the left ventricle succeeds in causing a forward movement of blood. The progressive expansion of the arterial wall from the center to the periphery reveals this movement.[2]

The *pressure wave* caused by the contraction of the heart travels with a speed of 3–4 meters per second in the aorta and 7–14 meters per second in the peripheral arteries. This speed is much greater than the average rate at which the blood flows toward the periphery (14 to 18 cm. per second).[2] The difference disappears, on the other hand, in the capillaries, where the pulsating pressure is converted into steady pressure and flow.

At the closure of the semilunar valves, the recoil of the aorta maintains the onward drive of the blood. At this time, the peripheral arteries are still undergoing distention, but they return to a smaller size as the excess of blood flows through the capillaries.

FUNCTIONS OF THE VEINS

The return of blood through the venous system is due only partly to remaining force after it has passed through one or more capillary systems. Many different mechanisms have been recognized which favor the venous return: (1) contractions of the veins; (2) decreasing pressure in the large veins due to the action of the heart (systolic suction); (3) aspirating effect of the low pressure existing in the thorax which increases during inspiration; and (4) action of skeletal muscles on the nearest veins.

RESPIRATION

Respiratory dynamics have multiple effects on the heart and on the veins. During inspiration, the diaphragm contracts and exerts pressure downwards. As a result, the following changes take place:

(a) The intrathoracic pressure is lowered and the intraabdominal pressure is increased, favoring a flow of blood from the abdomen to the thorax.

(b) The liver is compressed by the diaphragm and "wrung out."

(c) The pericardial sac is expanded and its complementary sinuses open, favoring diastole.

As a result, the blood moves from the inferior cava to the heart mainly during inspiration. The blood of the superior cava, however, shows less marked changes and a more constant course. Still, a remarkable inspiratory collapse of the superficial veins of the neck is frequently observed.