Clinical Anesthesia

Anesthesia and the Circulation

Leonard Fabian, M.D. / Editon

3/1964

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BLACKWELL SCIENTIFIC PUBLICATIONS
OXFORD

Printed in the United States of America
Library of Congress Catalog Card Number 64-25582

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Chapter 1

Regulation of Cardiac Output and Venous Return*

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* The original investigations reported in this article were supported by research grants-in-aid from the National Heart Institute and the American Heart Association.

Regulation of cardiac output has long been an enigma because of the many factors that can alter it. However, we will begin this analysis of output regulation by seeing what will happen under several different abnormal conditions of the circulation shown in the schema of Figure 1. First, let us squeeze the large venous reservoir. This will force increased quantities of blood into all other parts of the circulation, including an increased quantity of blood into the heart itself, with resultant distention of the cardiac chambers. We know from the early studies of Starling[8, 10] that increased filling of the heart causes increased cardiac output. Thus,

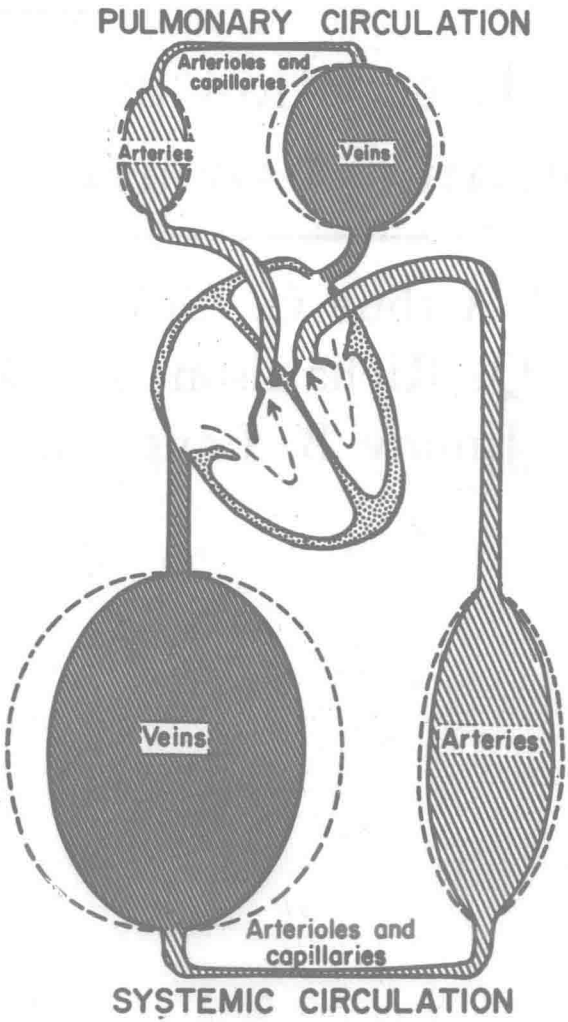

Figure 1. Schema of the circulation showing the distensible reservoirs in the systemic and pulmonary circulations.

this simple translocation of blood from the venous reservoir into other parts of the circulation, particularly into the heart, increases the cardiac output.

Second, let us assume that some obstruction occurs between the venous reservoir and the heart. This will dam blood up in the venous tree, causing the veins to distend; but, at the same time, it will also reduce the quantity of blood in the remainder of the circulation, reducing the quantity of blood in the heart itself. In accord with Starling's description of cardiac activity, one would expect decreased volume of blood in the heart to reduce the cardiac output very severely. And this is the effect that is observed under these conditions.

Third, sympathetic stimulation of the heart itself increases its pumping ability and can therefore increase the rate at which blood flows around the circulatory system.[11] However, there are very definite limits to the rate at which this can occur, because once the pumping ability of the heart has reached a critical level, the veins leading into the thorax and thence into the heart begin to collapse, thus acting as a valve to prevent additional increase in cardiac output.[5] In essence, then, increasing the pumping activity of the heart can increase the cardiac output, but this increase is limited, a limit that is very important, as we shall see later.

The above three changes in the circulatory system are only examples of many different possible changes anywhere in the cardiovascular circuit that can affect cardiac output. It will be the purpose of this chapter to describe the basic mechanisms by which cardiac output is controlled and to try to establish the quantitative importance of each of the factors involved in this control. In a previous monograph on this subject,[4] one of the authors has described the regulation of cardiac output in basic mathematical terms, giving a detailed review of the research work that has led to a rather complex analysis of cardiac output regulation. This chapter will present the subject in a completely different way, basing the discussion on fundamental physiologic and hydrodynamic principles with which all or at least most practicing physicians are familiar.

Definition of "Venous Return"

The term "venous return" has caused a tremendous amount of confusion in the literature on the subject of cardiac output regulation, mainly because the term itself has been used in several different ways. One of the definitions of venous return, and the way in which we will use it in this article, is the rate of blood flow into the heart, which, under most conditions, is exactly equal to cardiac output. The only instance in which these two are not equal occurs when blood is in the actual process of being translocated from the systemic circulation into the central circulation or, in the opposite direction, from the central circulation into the systemic circulation. Under one of these conditions the venous return

might be greater or less than cardiac output for a few beats of the heart. But it usually takes only a few seconds for enough blood to be translocated until the two rates of flow return to a state of equilibrium.

A second usage of the term "venous return" has been to indicate translocation of blood from the systemic circulation into the central circulation. For instance, if we should squeeze the abdomen, a large amount of blood is forced into the heart and lungs. Some authors have said that this represents increased venous return irrespective of whether or not it increases the cardiac output. Under most conditions such translocation of blood into the heart and lungs *does* cause increased cardiac output, but under some conditions large quantities of blood can be translocated into the heart without increasing the cardiac output. Therefore, this usage of the term "venous return" often belies the meaning of the words themselves.

Thus, one of the most important concepts that must be clear at the outset of this discussion is the difference between venous return, which means the rate of inflow to the heart, and translocation of blood from the periphery to the heart, which usually increases venous return but under a few conditions, such as in heart failure, may not do so.

BASIC MECHANISMS IN THE REGULATION OF CARDIAC OUTPUT

There are two basic mechanisms for the regulation of cardiac output. These are: (1) altering the pumping effectiveness of the heart itself and (2) altering the availability of blood to the heart at the right atrium.

The effectiveness of the heart as a pump determines how well the heart will be able to pump the blood attempting to enter the heart from the veins. The heart sometimes becomes much more effective than it is normally, as when it is strongly stimulated by the sympathetic nervous system,[15] or when it is hypertrophied. On the other hand, the effectiveness often becomes greatly depressed, as, for example, following a myocardial infarction.

On the other hand, even if the heart is tremendously effective as a pump and yet blood is not available at the input side of the heart, the cardiac output may still be extremely low. Therefore, equally as important as the effectiveness of the heart as a pump in controlling cardiac output, and under many conditions even more important, is the availability of blood. For instance, following severe hemorrhage or following vasomotor collapse, blood for pumping simply is not available to the heart, so that, as a consequence, the cardiac output may be extremely low even though the effectiveness of the heart as a pump might be as much as twice as great as it is normally.

Therefore, the basic mechanisms of cardiac output regulation will be discussed under two major headings: first, the factors that determine the

pumping effectiveness of the heart and, second, the factors that determine the availability of blood to be pumped.

Pumping Effectiveness of the Heart

The heart under normal conditions pumps 5 to 6 liters of blood per minute, but this same heart, without being stimulated or in any other way affected by outside control, can pump as much as 12 to 15 liters per minute if 12 to 15 liters of blood is made available to the input side of the heart each minute. The reason the heart pumps only 5 to 6 liters per minute is simply that this is the amount of blood that is normally made available to it. It is obvious, therefore, that we cannot express the effectiveness of the heart in terms of the amount of blood the heart actually pumps; instead, it is necessary to state how much blood the heart *could* pump per minute if unlimited amounts of blood were available at the input side of the heart.

The method that has been used since the days of Starling to represent the pumping effectiveness of the heart is the so-called *cardiac function curve.*[10] Figure 2 illustrates several individual cardiac function curves determined by Starling for hearts of dogs in a heart-lung preparation. These curves show that when the right atrial pressure falls to zero,

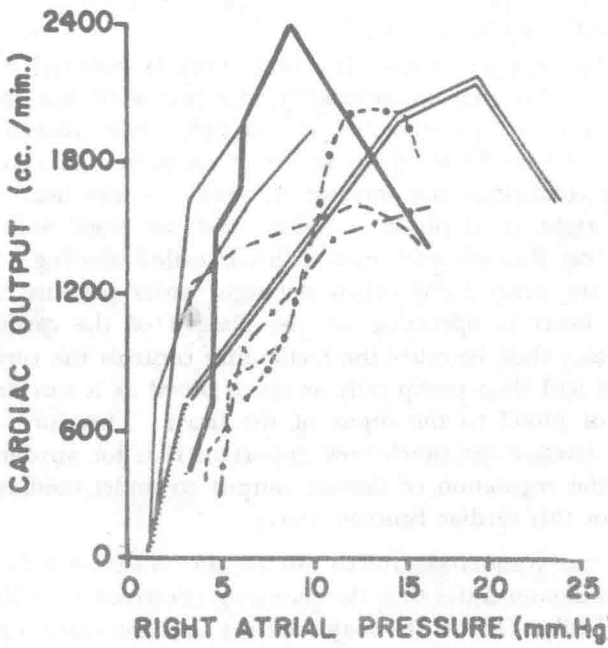

Figure 2. Cardiac function curves obtained in eight heart-lung preparations by Patterson and Starling, depicting cardiac output as a function of mean right atrial pressure. (Redrawn and axes transposed from Patterson and Starling.[10])

the heart will pump absolutely no blood. Then, as the right atrial pressure rises progressively up to 10 mm. Hg, the output of the heart likewise increases markedly, finally reaching a maximum value. Thus, these function curves describe the pumping effectiveness of the respective hearts, showing how much blood each heart could pump at each given right atrial pressure.

Basic Function Curves of the Human Heart. Even though the only truly accurate means for expressing the pumping effectiveness of the heart is to use cardiac function curves, almost no measurements of cardiac function curves have ever been made in the human being. Therefore, such curves for the human being must be extrapolated from those that have been determined in animals and from a large number of isolated measurements of atrial pressure and cardiac output under many different conditions in the human being. By piecing these together we have derived the "family" of cardiac function curves illustrated in Figure 3 for the human being.[4] The heavy black line illustrates the average cardiac output that would be expected from a heart of a young adult human being for each given right atrial pressure. Note that when the right atrial pressure is 0 mm. Hg, the cardiac output is approximately 5 liters per minute. However, an increase in right atrial pressure of only 1 mm. Hg will double the cardiac output, and a decrease in right atrial pressure of about 3 mm. Hg will reduce the cardiac output essentially to zero.

Study of this normal cardiac function curve is essential to an understanding of cardiac output regulation, for we shall see later that so long as the heart is operating in the low right atrial pressure range the heart itself has very little to do with the regulation of cardiac output. Under these conditions any increase in inflow to the heart will simply increase the right atrial pressure slightly, and the blood will be pumped immediately on through the heart. This is called Starling's "law of the heart." On the other hand, when the right atrial pressure is very high so that the heart is operating on the plateau of the cardiac function curve, the heart then becomes the factor that controls the cardiac output, for the heart will then pump only as much blood as it can despite excess availability of blood to the input of the heart. Therefore, once again, it cannot be stressed too much how important it is for anyone wishing to understand the regulation of cardiac output to understand in detail the significance of this cardiac function curve.

Effect of the Basic Strength of the Heart on Pumping Effectiveness. Figure 3 also demonstrates that the pumping effectiveness of the heart can change drastically. One of the major factors that can cause such a change is the basic strength of the heart itself, which can change under many different conditions. For instance, a heart may be weakened by myocardial infarction, by myocarditis, by nutritional disturbances, by toxins, or by

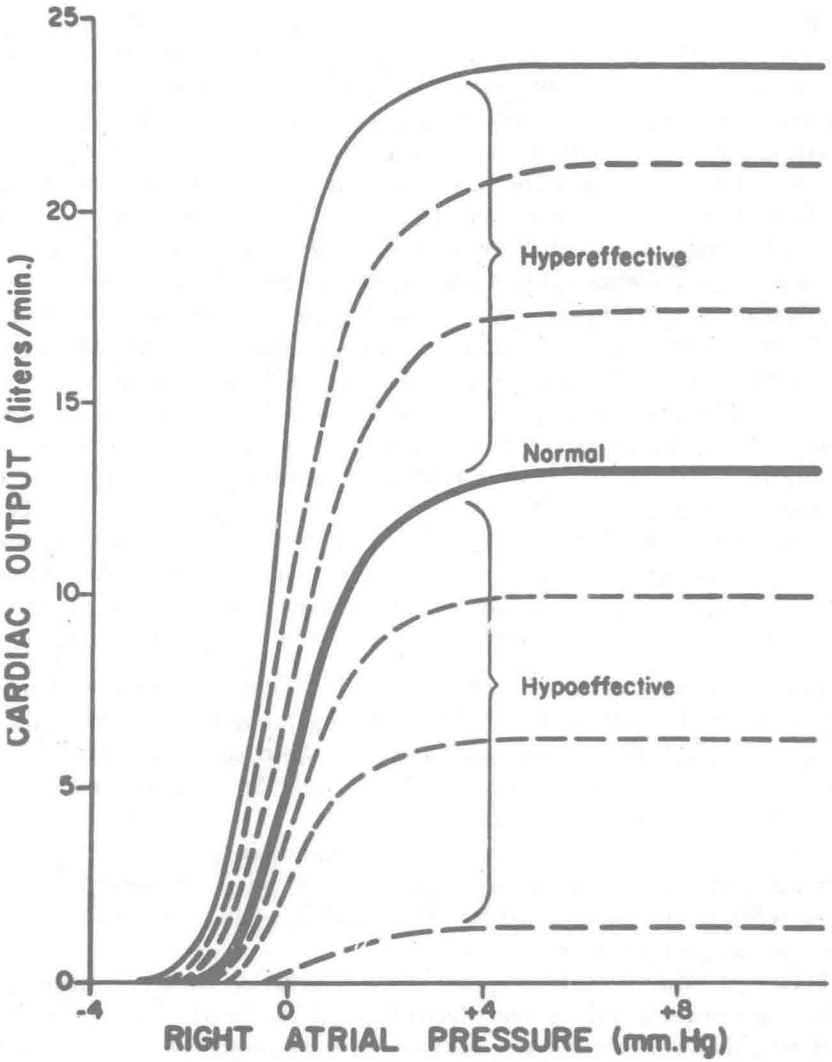

Figure 3. "Family" of cardiac output curves for hypo- and hypereffective hearts. (Reprinted from A. C. Guyton: **Circulatory Physiology: Cardiac Output and Its Regulation.** W. B. Saunders Co., Philadelphia, 1963.)

many other factors. Also, its effectiveness as a pump can be decreased as a result of valvular disorders, even though this might not decrease the strength of the myocardium itself.

On the other hand, in some conditions the heart can become much more effective as a pump than normally, for instance, when it becomes greatly hypertrophied or when it is strongly stimulated by the sympathetic nervous system.

Figure 3 demonstrates that the cardiac function curve becomes depressed and shifted slightly to the right as the heart becomes more and more "hypoeffective" as a pump. This general pattern of changes occurs regardless of the condition that causes the hypoeffectiveness. On the other hand, factors that cause the heart to become "hypereffective" cause the plateau of the curve to rise and the curve to shift very slightly toward the left.

Note especially that even the heart that has lost approximately half its effectiveness as a pump can still pump a normal cardiac output of 5 to 6 liters per minute without any significant rise in right atrial pressure (only about 1 mm. Hg rise). Furthermore, increasing the effectiveness of the heart as a pump, without simultaneously making additional amounts of blood available to the input side of the heart, will hardly affect the cardiac output. The reason for this is that even though the hypereffective heart can pump as much as 25 liters per minute, nevertheless, under normal conditions, only 5 to 6 liters of blood flow into the right atrium each minute from the systemic circulation, which obviously is all the blood that can be pumped. Yet, we shall see that in certain conditions in which the heart becomes excessively loaded, such as in exercise, in A-V fistulae, in hyperthyroidism, and in many other instances, the changes in cardiac pumping ability do play a very significant role in cardiac output regulation.

Effect of Autonomic Stimulation. One of the most important means for changing the effectiveness of the heart as a pump is autonomic stimulation. *Parasympathetic stimulation* decreases both the heart rate and the strength of contraction of the atria. As a result, the total amount of blood pumped by the heart each minute decreases.

Sympathetic stimulation, on the other hand, increases the heart rate and the strength of contraction of both the atria and ventricles. Because of these effects, sympathetic stimulation increases the quantity of blood that can be pumped each minute.

Figure 4 illustrates the approximate effects of different degrees of autonomic stimulation on the effectiveness of the heart as a pump. Note that maximal sympathetic stimulation can increase the effectiveness of the heart as a pump by about 70 per cent. On the other hand, complete block of the sympathetic system decreases the effectiveness from its normal level by about 25 per cent.

Maximal activity of the parasympathetic nerve centers ordinarily decreases the effectiveness of the heart as a pump by as much as 30 to 50 per cent, though under some conditions, such as in the carotid sinus syndrome, it can decrease the effectiveness of the heart as a pump all the way to zero for short periods of time. On the other hand, complete block of the parasympathetic system can probably increase the effectiveness of the heart as a pump by some 20 to 40 per cent, though exact values for this are unknown.