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The Physiology of Circulation: A Reappraisal

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The Heart as a Pump

THE PREVAILING MODERN notion that the circulatory system is a closed system can already be found in the thirteenth century, when Ibn An Nafis wrote that blood circulated throughout the body, following closed pathways in the smaller vessels. William Harvey went a step further, claiming in 1628 that the movement and beating of the heart are the only cause for the circulation of blood through the body [1]. This is the origin of our modern pump model of the heart. Writing in a modern physiology textbook, Antoni formulates the pump model succinctly: "The heart functions as the circulating pump that drives the blood through the vessels. Furthermore, strictly speaking, blood circulation consists of a single cycle into which both halves of the heart are inserted, functioning as motors that drive the blood" [in 15]. With regard to the specific capacity of these motors, Bassenge notes, "Increases in the heart's rate (which ranges from 50 to 200 beats per minute) and force of contraction are incontestably the most significant

factors in cardiac adaptation to exertion. These two factors lead to an increase of up to 30% in the stroke volume" [in 14]. This increase in stroke volume explains the exceptional cardiovascular reserve in endurance athletes with exercise-induced enlarged hearts.

These descriptions of the heart as an organ that pumps the blood throughout the organism are based on the seminal work of Frank and Starling at the close of the nineteenth century. Starling experimented on an isolated dog's heart-lung preparation and formulated in 1895 his famous law of the heart, stating that, within physiological limits, the larger the volume of the heart, the greater the energy of its contraction. This was confirmed by others and in comprehensive experiments on intact animals by Guyton. Guyton writes, "the heart can pump either a small or a large amount, depending on the amount that flows into it from the veins; and it automatically adapts to whatever this load might be as long as the total quantity of blood does not rise above the physiological limit that the heart can pump" [4, p. 156; see also Fig. 10].

On this view, the heart supposedly creates enough force to overcome the existing pressure in the aorta—and in the arteries and capillaries that follow—so that there is still enough momentum available to the venous blood to return and fill the heart. This leads to the conclusion that the heart constantly cycles the blood in a closed system, producing the perfusion of tissues that is vital to life.

When the heart is isolated from the rest of the body, its pumping ability is clearly apparent: up to a point, the heart is able to eject increasing amounts of returning blood (Starling's law). Furthermore, when the rate and the strength of contractions increase, more blood is moved. Indeed, an isolated heart can pump more blood than an intact heart. An isolated frog's heart clearly functions as a pressure-suction pump. It will continue pumping, rhythmically contracting, sucking in and ejecting water for hours.

The pump model can be used to explain many pathological phenomena. Most obvious is cardiac arrest: the heart stops beating and therefore the movement of the blood stops. In heart failure "the quantity of blood pumped by the heart each minute (cardiac output) is insufficient to meet the body's normal requirements for oxygen and nutrients" [11, p. 87].

Since 1958, it has been possible to counteract an excessively slow heart rate by installing a pacemaker. This device registers the heart's intrinsic rate and supplies an electrical stimulus, thus adjusting the rate, should it fall to unacceptably low levels. Since 1954 we have seen not only the emergence of heart-lung-machines, such as are used during open heart surgery, but also of a number of left ventricular assist devices that can support the failing heart and give it time to recover. Total artificial hearts enable patients waiting for a heart transplant to bridge the critical time until a suitable donor heart is found. Such devices use various forms of pumps, which directly or indirectly support the movement of the blood in the circulation. Thus the inadequate rate and force of contraction of a weakened heart can be corrected separately by means of an artificial pacemaker and a mechanical pump, respectively. The pacemaker is a substitute for the natural heart rate, while the artificial heart replaces both the contraction rate and the force of the natural heart.

The pump model of the heart can apparently explain myriad facts of normal and pathological physiology, and the advent of technological devices such as the pacemaker and artificial heart bolster this model. The question is, does it truly provide an adequate understanding of all the observable physiological phenomena?

Complex Reality

It is usually assumed that because a pump can replace the heart, the heart is in fact a pump. As long as one can supply enough mechanical force to maintain a cardiac output and hence adequate tissue perfusion the most important premise of the circulation appears to be satisfied. In reality, however, when the heart is replaced with an artificial device, one encounters several problems that in themselves reveal some of the basic principles of circulatory physiology. William DeVries, the creator of the first implanted artificial heart, made an unexpected observation after implanting the device into four different patients [2]. He observed that when systolic, diastolic, and mean blood pressures are increased, the *cardiac output actually decreases* (see Figure 1). This is the opposite of what one would expect if the circulation is impelled by an artificial pump. In the early postoperative period it was

relatively easy to control the cardiac output by increasing or decreasing the heart rate. However, the actual optimal cardiac output for the particular patient was not known and one patient suffered seizures early on due to presumed overperfusion of the brain. This was corrected by reducing the cardiac output to levels thought to be below "normal" for his size.

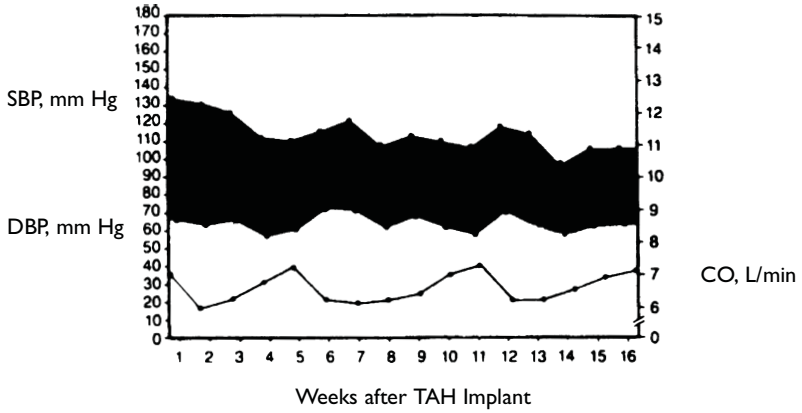

Fig. 1: The effect of pressure changes produced by an artificial heart on cardiac output (CO). The black area represents artificially altered systolic and diastolic blood pressure amplitudes at a rate of 80 beats per minute. Below, in liters per minute, is blood flow velocity. Increasing the pumping pressure does not increase flow velocity but decreases it. Above a specific blood pressure level, the flow of blood through the entire body cannot be affected by increasing the pumping pressure. Time (on the abcissa) means weeks after implantation of a TAH (total artificial heart) beating at 80 beats per minute [2].

After about forty days the doctors attempted to increase cardiac output by increasing the pumping rate. Since, however, the circulatory system had adapted to its new, postoperative state, this intervention acted as a disturbance. As a result, the blood vessels dilated (reflex vasodilation) and cardiac output soon returned to its original level, a level appropriate to the body's needs. This mismatch between arbitrarily introduced cardiac output and the organism's needs may have been the cause of kidney insufficiency and a host of other problems such as strokes.

Since the blood came into contact with an artificial material there was an increased tendency toward clot formation and vessel blockage (thrombus formation and embolism) on the one hand, and toward

destruction of the red blood cells with kidney insufficiency on the other. Because all patients had to be given blood thinners, there was also a substantial risk of internal hemorrhage. Out of four patients discussed by DeVries, one died of bleeding into the chest cavity on the tenth postoperative day. The second patient lived 112 days and died of circulatory failure and massive infection. The third and the fourth patients lived 620 and 482 days respectively and died of overwhelming infection. At the present time (Fall 2001), doctors in the United States, England, and Germany continue to develop and test various types of pumps and small turbines that support weak heart function as well as pumps that completely replace the heart.

Observations of patients with artificial pacemakers also show that a simple pump model of the heart does not do justice to the phenomena. The pacemaker stimulates the heart to beat more rapidly. When the pacemaker induces excessively rapid beating (so-called pacemaker tachycardia), both aortic pressure and the strength of heart contractions increase [5]. However, the volume of blood flowing through the heart per minute (the cardiac output) *remains the same.* Even when the heart rate is doubled or tripled, cardiac output remains the same as it is at rest (Figure 2).

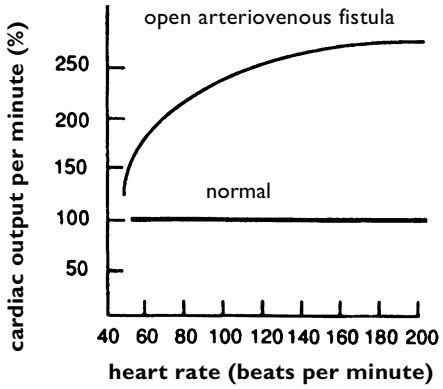

Fig. 2: Effect of an artificial pacemaker on intact circulation in a dog. When the heart rate is elevated artificially (pacemaker tachycardia), cardiac output per minute does not increase but remains the same (100% on the graph). Increasing the heart rate does not produce a corresponding increase in blood flow velocity, which remains the same [7]. The effect of increased heart rate is evident only when a short circuit occurs through insertion of an arteriovenous shunt and blood circumvents the organs that would otherwise receive the flow. (See the curve for the open arteriovenous fistula, which competes with natural circulation to the organs.)

Similarly, there is no increase of cardiac output in otherwise healthy young individuals during an episode of racing heartbeat (paroxysmal tachycardia). In both cases the amount of blood ejected during each heartbeat (the stroke volume) is diminished, so that the volume of blood returning to the heart is also smaller, keeping the output constant. If, on the other hand, the heart rate drops below normal (bradycardia), there is a concomitant increase in stroke volume and also, up to a limit, unchanged cardiac output.

We know that the heart is always capable of approximately doubling its stroke volume (from about 70 ml to 140) should the need arise, say, due to sudden exertion. But if a sudden, critical drop in heart rate (from 50 to 25 beats per minute, for example) occurs when the body is at rest, the heart does not automatically double its stroke volume, although doing so would maintain normal levels of cardiac output and hence also of vital perfusion.

From these observations, we can conclude that at faster heart rates an implanted artificial heart does not automatically produce a greater output than it does at resting rates. Similarly, when the heart rate is increased either naturally or by means of an artificial pacemaker, the natural heart does not function as a pump that drives blood circulation as a whole. The question arises: why is it impossible, at rest, to artificially increase cardiac output and organ perfusion by increasing the force (contractility) of the heart muscle, blood pressure, heart rate, or stroke volume, when we know that in endurance athletes such increases constitute the reserve needed to boost the performance of the heart? We will attempt to answer this question in the following section (see page 59 ff.).

Known treatments for diseases associated with either excessive cardiac strength or weakness (hypertension and cardiac insufficiency, respectively) also call into question a simple pump model of heart function:

* Although hypertension symptoms (an overly strong heart) and symptoms of cardiac insufficiency (an overly weak heart) usually occur separately, or at least at different times in the same patient, why is it that they can also appear together?

* Digitalis is a cardiac stimulant and should extend the lives of patients with chronic heart insufficiency, since it increases the heart's perfor-

mance. But extensive studies of therapeutic efficacy tell us that long-term administration of digitalis does not extend the lives of patients.

* Why is long-term drug therapy successful (as measured in terms of life expectancy) when it reduces resistance to the flow of blood?

* Why can the same medications, such as diuretics or ACE (angiotensin converting enzyme) inhibitors, be used in conditions of both excessive strength and weakness of the heart? [9]

* Why can lives be prolonged in both high blood pressure and heart failure patients with beta blockers that reduce the heart's force of contraction? [12, 13]

* Why is blood pressure often normal in patients with cardiac insufficiency and poor perfusion?

* Why do we also see poor perfusion in patients with hypertension, even when the onset of the illness is relatively recent and arteriosclerosis has not yet caused narrowing of the vessels?

* Why can blood pressure and heart rate vary greatly without altering perfusion, even though (as mentioned at the beginning) reputable authors uphold the view that increases in these levels constitute the reserve mechanisms of the blood's circulating pump?

Answering these questions simply and clearly becomes increasingly difficult when we consider the plethora of scientific discoveries with regard to both cardiac function and circulatory and metabolic processes in the capillaries. It is time to reassess the relationship between the heart and the movement of the blood in the light of a few important facts. Our further discussion will address the specific but not yet universally acknowledged effect of the tissues on blood circulation.

The Heart and Peripheral Circulation

Since calling the heart a pump always means that it pumps blood throughout the *entire* circulatory system, we will consider the circulatory system in its entirety and not merely the sections just before and just after the heart. Let's begin by investigating whether it is possible to confirm *in vivo* that the pumping effect of the heart persists throughout the blood's entire cycle from and back to the heart.

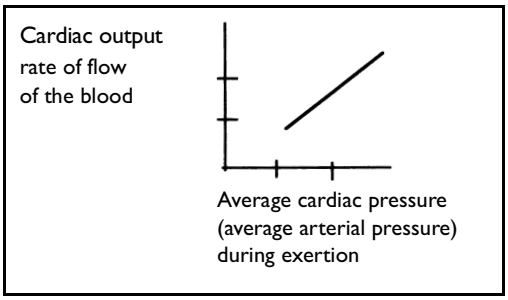

Fig. 3: During exertion, cardiac output increases as pressure increases.

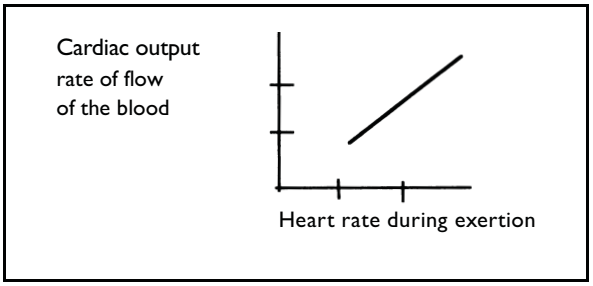

Fig. 4: During exertion, cardiac output increases as the heart rate increases.

When someone runs quickly, blood pressure in the body increases moderately. The heart beats approximately three times as fast, and five times as much blood (20 liters per minute) moves through it and the body (see Figures 3 and 4). Such extensive change is possible during exertion because resistance to the flow of blood decreases (see Figures 5 and 6). The entire channel in which the blood flows—the total peripheral cross-section—increases. According to physiology texts, the volume of blood per unit of time that flows through the muscles can increase by a factor of 15 to 25 during exertion, depending on the individual's fitness level [15]. The flow to certain other parts of the body, such as the kidneys and intestines, is reduced, while flow to the brain remains unchanged.

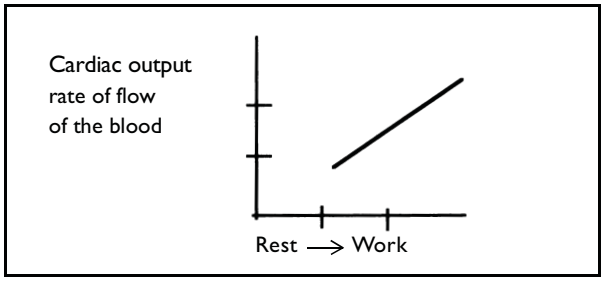

Fig. 5: Cardiac output increases with exertion.

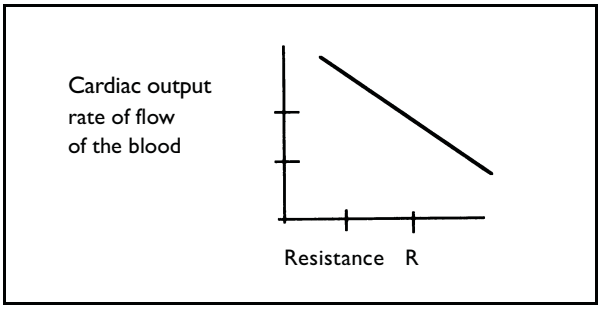

Fig. 6: Cardiac output decreases in proportion to the increase in resistance to the flow of blood.

The blood's flow velocity is ultimately determined, then, by the driving force, on the one hand, and the degree of resistance, on the other. When resistance decreases, blood flow increases greatly, both peripherally and centrally (i.e., through the heart).

This overview of cardiac output is not fully adequate for our purposes, because heavy bodily exertion and accelerated respiration interfere with the assumed causal relationship between heart activity and the movement of the blood. Heavy breathing (which causes pressure in the chest to fluctuate above and below atmospheric pressure) and vigorous limb movements (which enhance the pumping effect of muscle activity on the blood stream) influence central and peripheral circulation respectively. What we want to know, however, is how the heart, and only the heart, affects the flow of blood, but we want to see how it

does so in its natural interaction with the rest of the body, rather than in isolation, as in a heart-lung machine.

What happens when cardiac output is too small or too large? When the heart beat weakens and the heart rate slows, producing lower pressures, cardiac output also decreases until ultimately circulation ceases when the heart stops beating. If, on the other hand, the heart beats faster and produces higher pressures while the body remains at rest, blood flow never amounts to 10 or 20 liters per minute, even though pumping pressure and rates are sufficient to produce such a flow, just as they are during exertion (see Figure 7). This is not what we would expect of a circulating pump. We have already seen that increasing the rate of the implanted artificial heart does not increase cardiac output (see Figure 1).

Similarly, increasing the rate of an artificial pacemaker during rest does not increase cardiac output. All these phenomena run counter to the notion that the heart drives the blood through the entire circulation. We must look beyond the heart to understand blood flow.

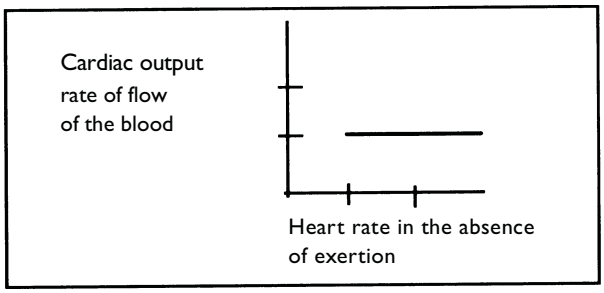

Fig. 7: In the resting state, without exertion, cardiac output remains the same even though blood pressure and heart rate (the basic means for boosting output) increase.

It is a well-known physiological fact that, within certain limits, tissues and organs regulate their own blood flow independent from fluctuations in arterial blood pressure. This principle of autoregulation in tissues and organs occurs because widening or narrowing of the vessels (which is stimulated hormonally) modulates resistance. This modulation, in turn, is regulated by a complex of local and central processes. When vessels open, blood flows more easily, when they constrict, blood

flow is restricted (higher resistance). Moreover, central venous pressure always rises when the volume of blood increases. In evaluating his experiments, Guyton comes to this conclusion:

> Therefore, there is no reason to believe that in the normal circulation, increasing the heart pumping capacity above normal will increase the cardiac output or the arterial pressure to higher than normal values. On the other hand, when increased amounts of blood return from the veins, an increase in pumping capacity of the heart often then becomes essential to keep up with the returning blood. [5, see also 3]

The amount of blood entering the right side of the heart ultimately depends on central venous pressure. Guyton performed an experiment whereby a rapid transfusion of blood was given in the amount equal to 40% above the dog's normal blood volume. This resulted in an increase of cardiac output by 100%. [3,5]. During diastole, the normally relaxed ventricles offer little resistance to the force of the instreaming blood. If larger amounts of blood are returning to the heart, then the heart fills with more blood. Since, according to Starling's law, the degree of filling (end-diastolic volume) determines the force of the subsequent contraction, a larger amount of returning blood increases cardiac output.

We see now that peripheral circulation, which is regulated in relation to the needs of the organs and the body as a whole, plays a major role in determining overall blood flow and, ultimately, cardiac output. Physiological facts lead us beyond the notion of the heart as a central circulating pump that of its own accord drives the blood through the entire circulatory system.

Why Is There No "Excessive Pumping Syndrome?"

Although the pumping action of the heart is supposed to be its most significant contribution to blood circulation, excessive pumping —heart-induced hyper-dynamic syndrome— is not known to occur. According to Starling's law of the heart, increased ventricular filling causes increased expansion of the ventricles, which in turn increases

the force of the subsequent heart beat and the amount ejected. Thus the Frank-Starling mechanism should permit the development of a positive feedback loop, as follows:

1. A sudden increase in the force of contraction (in paroxysmal tachycardia, for example) causes an increase in ejection volume.
2. Increased ejection leads to increased perfusion and thus to increased vascular filling with a subsequent decrease in resistance due to widening of the vessels (the radius increases), which in turn causes an increase in the flow of blood returning to the heart.
3. Increased venous return into the heart increases stretching of the ventricles, leading to an increase in ejection volume and contractility.
4. Together with an increasing heart rate, this all adds up to a heart-induced hyperdynamic or hypercirculation syndrome.

The drop in peripheral resistance coupled with enhanced heart performance (Starling's law) would soon lead to a vicious cycle and a collapse of circulation. Fortunately, there are numerous regulatory functions that, together with autoregulation at the level of individual organs, prevent such occurrences. And even when disturbances in autoregulation do occur, they are not accompanied by heart induced hyperdynamic states.

When we observe cardiac insufficiency in greater detail, we encounter an additional paradox. When the heartbeat becomes too weak and less blood is ejected, the central venous pressure rises instead of dropping. (It is normally between 2 to 12 cm of water and increases to as high as 20 cm.) Venous filling adjacent to the heart also, strangely enough, increases. Seeing that blood pressure and heart rate do not always correlate with cardiac output, or with the actual amount of circulating blood, are there other factors that do?

Blood Flow and Oxygen

Our experience of circulation during moderate exertion, when heart rate and respiration increase markedly, is very different from how we experience a racing heart and rapid respirations when we are

at rest. The volume of blood that flows through the heart in each case can be measured, and a measurable factor that rises and falls in proportion to this volume is indeed known. It is the amount of oxygen consumed by the tissues. The amount of blood flowing through the heart is directly proportional to the amount of oxygen consumed in the entire body (see Figure 8). Peripheral circulation moves large amounts of blood only in areas where large amounts of oxygen are being consumed. Cardiac output is, therefore, determined by the metabolic demands of the tissues.

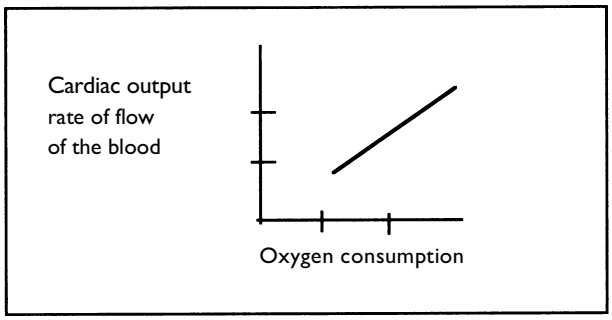

Fig. 8: Cardiac output always increases as oxygen consumption increases.

During movement, our muscles consume a great deal of oxygen, and a correspondingly greater volume of blood per minute moves through both the heart and the active muscles, while during rest we consume less oxygen and move less blood. As already mentioned, depending on individual fitness levels, muscle perfusion during exercise can increase by a factor of 15 to 25.

Keeping the foregoing discussion in mind, let's ask again: why is there no change in cardiac output when heart rate and blood pressure increase at rest? We can explain this phenomenon by the fact that oxygen consumption remains the same. Likewise, we can now understand why the syndrome of excessive pumping of blood or hyperkinetic circulation does not occur at rest, since the ultimate stimulus for increase in cardiac output is tissue oxygen consumption. The relation between oxygen consumption in the tissues and cardiac output is linear. When oxygen consumption increases, both the heart

rate and cardiac output increase. By the same token, in the case of hypothermia (decreased body temperature), tissue metabolism and oxygen consumption decrease proportionally. When the core body temperature falls to 30° C, the basal metabolic rate drops by 50%. Cardiac output decreases by the same percentage.

What Moves the Blood?

As long as we assume circulation is a closed system, it seems logical to view the heart as the circulating pump. If, however, we recognize the fact that tissue metabolism ultimately determines the flow of blood, the pump model no longer suffices. It is true that circulation must be a relatively closed system in order to prevent escape of red blood cells from the capillaries into the tissues. But functionally, circulation must be open to allow the passage of oxygen and carbon dioxide and also the exchange of the nutrients, electrolytes and water to and from the tissues. In other words, the extravascular tissues continually produce the intravascular blood volume, which ultimately allows the heart to function.

The exchange of substances occurs through the capillary walls. Every organ system in the body has its own specific type of capillaries, which range from the ones with tightly closed walls (e.g, in the brain) to the type with porous walls (e.g. the kidney, bone marrow). The type of capillaries determines the amount and the type of filtration and fluid exchange occurring. Being open to the tissues is a vital function of the capillaries. They ensure the constant flow of oxygen and nutrients (400 g glucose in 24 hours) into the tissues and the removal of toxins and lymph (3 liters in 24 hours, or significantly more at higher filtration pressures, as is especially apparent in the kidneys). In the capillaries, therefore, a certain volume of fluid is lost to the tissues in conjunction with the processes of filtration, diffusion, and active transport of substances. As a result, there is a volume deficit after the capillaries in the venules.

The capillaries themselves do not actively direct this flow of fluid into the tissues. It results in part from the hydrostatic pressure that is maintained in the arterial circulation. The higher the pressure, the greater the volume of fluid that filters out of the capillaries into the tissues. This phenomenon, which occurs in all tissues and organ systems of the body, reduces venous filling and with it central venous pressure.

In other words, the blood pressure produced by the heart is the cause of filtration, that is, of the loss of fluids from the circulation into the tissues. Instead of the heart causing the venous system to fill, which it would do in the closed system model, it actually reduces venous filling.

Each minute the kidneys filter 650 ml of blood and form 120 ml of primary urine, which amounts to 170 liters per day, all of which is reabsorbed into the blood stream, except for the 1.5 liters of urine we produce each day. Along with water, the kidneys also reabsorb 1500 grams of sodium chloride, 160 grams of glucose and 0.4 grams of albumin, which are also returned to the circulation. Various products of tissue metabolism such as carbonic acid, phosphates and sulfates, together with water, form anew as products of oxidative metabolism (300 ml in 24 hrs). They are actively transported into the venous ends of the capillaries against the hydrostatic pressure.

There is a physiological watershed in the capillaries that is created by the two opposing forces of suction and pressure. At the arterial end of the capillary the hydrostatic pressure (residual of arterial pressure) forces the fluids out, while at the venous end the oncotic pressure (water-binding force) of blood plasma proteins draws fluids from the tissues back into the capillary.

The venous return directly or indirectly depends on the metabolic activity of the tissues. Since the fluid loss at the arterial side of the capillary is greater than capillary refill at the venous end, the lymphatic system prevents the accumulation of excess fluids in the tissues. Through an intricate system of lymphatic capillaries and progressively larger vessels, it collects the excess, protein rich fluid, which amounts to about three liters per day, and returns it via the lymphatic ducts to the great veins near the heart. Greater venous return fills the large veins and increases venous pressure. This is the force that fills the heart and the greater the amount of venous return, the greater the force of ventricular contraction. The filling pressure of the heart is therefore not a residual of the arterial pressure, but is the result of the metabolic activity of the whole organism.

In this context it is helpful to compare the work performed by the whole body with that of the heart. In order to sustain its basic vital functions, the human body consumes at rest on average 250 cubic centimeters of oxygen per minute (basal metabolic rate). Expressed as

metabolic work this is equivalent to 60 watts (joules per second). This is the energy required to sustain the muscular tone and to support normal workings of the inner organs and the ongoing production of blood. As an inner organ, the heart also requires a certain amount of oxygen, which is in the order of 10 cubic centimeters per 100 grams of heart tissue. If we take the average weight of the heart to be around 250 grams, the amount of oxygen consumed by the heart would be some 25 cubic centimeters, or about one tenth of the basic metabolic needs of the body. Now, about 20% of oxygen consumed by the heart is used for basal metabolism and only about 5% (1 Watt) for the work involved in beating (this can increase up to 20% during exertion). This is the work the heart carries out during contraction. Surprisingly, 60 to 70% of oxygen consumed is turned into heat. Thus we see that most of the heart's work does not result in mechanical force but in the production of warmth. This warmth infuses into the bloodstream and helps to warm the rest of the body.

The body as a whole expends much more energy—carries out more work—than the heart (60 watts compared to 1 watt). A portion of this work is expended by the tissues in producing and moving blood into the capillaries and then through the veins. We can now readily understand why the central venous pressure increases when the heart is failing and is not decreased, as one would expect in pump failure (see Figure 9). The tissues, and not the heart, are determining how much blood flows into the right side of the heart, out to the lungs and then back to the left side of the heart.

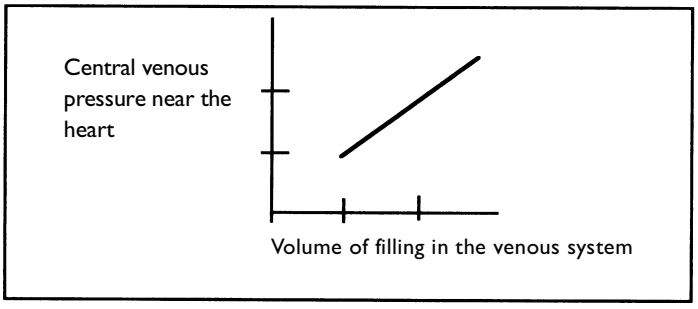

Fig. 9: The more the circulatory system is filled with blood, the greater the pressure that drives the blood into the heart and the greater the volume of flow through the heart (cardiac output).

The volume of blood does not depend only on the fluid dynamics at the level of the capillaries, but also on the presence of its cellular components, which constitute about 45% of blood volume. By far the most numerous are the red blood cells, which are produced by the bone marrow and find their way into the circulation via porous (fenestrated) capillaries. They, too, significantly contribute to the filling of the circulation. In short, the tissues essentially produce the innumerable components of the blood.

The volume of blood in the vascular system at any given moment is determined both by the arterial pressure—whose effect is subtractive or volume reducing—and by the metabolic work of the tissues, whose effect is additive, or filling. Let's look again at the kidney as a particularly good example. A certain amount of arterial pressure is required for the kidneys to filter from the blood about 170 liters of primary urine per day. At the cost of considerable metabolic work, the kidneys then separate out the metabolic waste products and secrete them in the final volume of urine of some 1.5 liters. The remainder is returned back to the blood. We might say, therefore, that the heart and arteries empty the capillaries, while the tissues constantly fill them with cells and plasma.

A delicate balance is maintained between the two processes. Should this balance be disturbed, either overfilling of the circulation or tissue swelling (edema) would ensue. The volume of fluid that leaves the capillaries and flows back to the heart may be equal to that which originally entered them, but the components of the blood are changed and being continually renewed. The blood volume that moves straight through is always less at the end of the capillaries than at the beginning, because a certain volume is lost to the tissues. This volume is replaced by the metabolic activity of the tissues.

The organs of the body are continually producing substances and fluids out of the substances and fluids they take in from the blood. In turn, the organs give substances and fluids back into the blood. In other words, the blood is being continually renewed. A rough tally of all the blood-derived body fluids (primary urine or renal ultrafiltrate, gastrointestinal and pancreatic juices, bile, saliva, cerebrospinal fluid, lymph, sweat and insensible perspiration from the skin and mucous membranes) amounts to some 200 liters per day. Since the volume of blood circulating through the body is about four to five liters and about

sixty percent of the blood's volume, or two and one half liters, consists of plasma (the fluid, noncorpuscular component of blood), the volume of plasma is renewed approximately 80 times during a twenty-four-hour period. This is a remarkable turnover. Furthermore, about 400 grams of glucose are taken from the blood during the same time.

We can confirm the importance of this unfamiliar aspect of the blood by imagining what would happen if all the water, glucose, sodium, etc. that filter into the tissues under pressure were not returned to the capillaries. Should the kidneys, for example, cease returning the processed primary urine back to the circulation for only 8 minutes, there would be a volume deficit of one liter in the venous circulation, a drop in central venous pressure of about 7 cm of water, and also a corresponding drop in cardiac output.

This dynamic view of circulation makes it clear that the heart does not function like a pump that propels the blood in a circle back to the heart. *Rather, the force that causes the blood to flow into the heart is the result of work performed by the tissues continually replenishing the fluid volume of the blood.* The cause of blood flow is found not in the heart in the body's center but in all the locations on the periphery where the fluid volume of the blood is produced (see Figure 10).

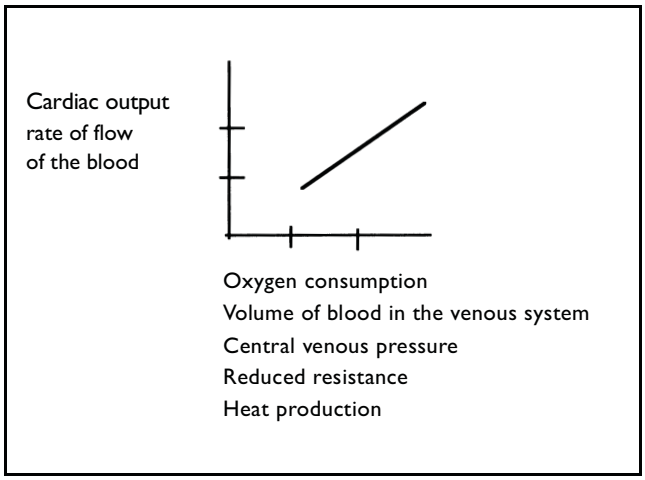

Fig. 10: Cardiac output is directly proportional to oxygen consumption, the volume of blood in the venous system, central venous pressure, the size of the organism, and the body's warmth output. It is inversely proportional to resistance to flow.

We can bring this knowledge gained through a dynamic view of physiology into connection with our experiential relation to bodily processes. In the resting human body, blood moves through the heart at a rate of four liters per minute. In contrast, when we run fast, this flow increases to 10 to 20 liters per minute, and we breathe heavily. We have learned that what moves the blood is not the heart but the increase in work performed in the tissues, and we can sense the connection when we breathe heavily during exertion, which causes a great increase in oxygen consumption.

Who ultimately causes this increase in work, however? Whose intention makes it happen? Who makes me run fast? Only myself. Upon reflection, I realize that I, as an intentional, embodied being, always move just as much blood as I need (in addition to basic metabolic demands) to carry out a specific action. We also see how our feelings alter the flow of blood, so that we blush with shame and turn pale with anger. Both my intention (which is realized in the form of an outer activity) and the accompanying feeling directly affect the velocity of my bloodstream. Inner experiences, rather than running parallel to bodily processes as psychophysical parallelism contends, directly influence vital physical processes such as blood flow velocity. Because I exert myself deliberately, consciously, and purposefully, my heart beats faster to accommodate how fast my blood needs to flow. In rest and sleep, on the other hand, the functions of body are more indirectly serving the I, helping us to regenerate for new activity. Blood flow is not some independent mechanism, but totally bound up with our whole being.

The Heart's Place in the Circulatory System

We have arrived at a totally new perspective on blood flow. Because the body as a whole, rather than the pumping heart, produces filling and pressure levels near the heart, the heart receives the inflow of blood without creating the force driving it. So what is the function of the heart with regard to this inflow? According to our conclusions thus far, the heart creates the appropriate resistance to the inflow of blood. In the living organism, the heart functions alternately to create a weak resistance to flow (diastole) and a strong resistance (systole). When a

great deal of blood flows, for example, either in the heart or elsewhere in the circulatory system, resistance to flow is reduced at that location.

Unaccustomed as this thought may be, recent research in cardiology has recognized its great importance. It has been established that a healthy heart essentially offers very little resistance to diastolic filling. When the heart relaxes during early diastole, it actually produces negative pressure, drawing in the blood. The relaxing, flexible heart also accommodates to the incoming volume of blood, which is a critical function. Should this ability fail, as is the case in ventricular wall stiffness, disordered movement (dyskinesia), or any other form of diastolic dysfunction, the blood backs up before the heart with a resulting increase in central venous or pulmonary vein pressures. The backing up of the blood and insufficient filling of the heart then become the cause of heart failure. In other words, when the heart is healthy it produces little resistance to the inflowing blood. A too rapid or slow heart beat, or a too high or extremely low blood pressure are signs of illness.

Since the flow of blood through the heart depends on total metabolic activity, the heart must adapt its function to allow large amounts of blood to pass quickly through it during strenuous exercise. The author carried out an echocardiographic study of heart function during the so-called isovolumetric phase of the heartbeat [9,10]. This is the part of the heart cycle when both aortic and mitral valves are closed and no blood flows in or out of the ventricles. As the heart rate increases, the length of the isovolumetric phase becomes shorter. At the rate of 140 beats per minute, a new hitherto undescribed event of the cardiac cycle was observed. For a brief time, both the mitral and aortic valves open simultaneously and, due to flow inertia, the blood was already flowing out of the left ventricle, before the inflow was completed. This means there was no longer an isovolumetric phase, but rather a continual flow of blood through the heart.

Today we can we observe the flow pattern of blood through the heart using sophisticated imaging techniques [7]. One sees a spiral pattern, whereby the blood flows down through the mitral valve towards the apex, where the direction of flow reverses towards the left ventricular outflow tract. Furthermore, at a higher heart rate, with the disappearance of the isovolumetric phase, the blood in the atrium also continues to flow towards the closed mitral valve, thus pushing the valve rings

towards the apex of the heart. Kilner, et al. suggest that during exercise and rapid blood flow, the looping pattern of blood in the heart might allow a sling-like effect, enhancing the outflow of blood [7].

In 1972 the surgeon Fontane carried out the first operation on newborns who had such a badly formed right side of the heart that it did not function. Without taking any theory into account, Fontane operated on the babies and connected the right atrium directly to the pulmonary artery, thereby bypassing the right ventricle. (This is known as the Fontane operation.) The first of these patients are now nearly 30 years old. While they cannot exert themselves as fully as people with a normal right side of the heart, they carry out productive lives. Evidently, the beating right side of the heart is not absolutely necessary for blood flow. This supports the view we have developed, which sees the driving force of venous return not in the heart, but in the whole metabolism.

If we are looking for a model of the heart's function, then the hydraulic ram is more appropriate than the central circulating pump (see Appendix B and Schad's chapter in this book). The inflow of water into the ram is not produced by the hydraulic ram itself, but is propelled and elevated by the inertia of the flowing stream. This "hydraulic ram effect," may explain the phenomenon of "second wind" experienced by endurance athletes in which cardiac output is enhanced due to decreased resistance to blood flow. If the heart is unable to accommodate the increased inflow in the face of greatly increased diastolic pressure, the blood can pool in the heart causing acute dilation and possibly death. This may account for sudden deaths occurring in athletes.

The author of this chapter is presently exploring the possibility of supporting weak heart function through a hydraulic ram. In this model there would be no need for an external power source, since the hydraulic ram, implanted into the chest, would be driven by the force of the chest, belly and diaphragm muscles. A description and film of this model can be viewed at www.lauboeck.de.vu.

To sum up: the heart regulates cardiac output by varying resistance to the driving force of the blood produced by the periphery. This force originates in the dynamic fluid exchange in the capillaries, which precisely regulates the amount of venous filling. It does not originate in the heart.

Conclusion

The prevailing view in conventional science and medicine is that the heart serves as the pump that drives blood circulation. This view sees the heart as functioning in a closed vascular system, where it exerts pressure on the blood, causing it to circulate.

But this model cannot explain many phenomena. Paradoxically, both the volume of blood that returns to the heart and central venous pressure *increase* when the heart's pumping action weakens and moves less blood. Another startling fact is that the heart is momentarily capable of increasing the volume of blood that flows through it, but never does so on its own accord. Furthermore, although pump-driven cyclical flow depends on the force and rate of pumping, the phenomenon of an "excessive pumping syndrome" is unknown in the resting body.

These and many other phenomena we have discussed, lead us to look to the organism as a whole in our search for the causes of blood movement. Oxygen consumption in the tissues is the only measurable factor that has been shown to increase consistently in proportion to the flow of blood. But what is the connection between combustion in the tissues and the force that drives circulation? In reality, the vascular system is not closed; in the capillaries it is functionally open. A tally of amounts of fluid withdrawn from the blood by all the tissues of the body adds up to roughly 200 liters in 24 hours. Thus the entire fluid, non-corpuscular volume of the blood (2.5 liters) is replaced approximately 80 times a day. The tissues of the body constantly expend energy to produce the volume of blood that flows in the vascular system. Blood production by the tissues expands the vascular system, thus producing the pressure that fills the heart with blood. In contrast, the arterial pressure produced by the heart—which is customarily seen as the driving force of circulation—actually reduces the filling of the capillary system.

If the heart is not a pump that drives the whole circulatory cycle, what does it do? The answer to this question is not one that our current scientific understanding of the subject would expect. The function of the heart, with regard to the blood returning in the veins, is to regulate resistance. The blood always flows faster when the venous return increases and the total resistance to inflow exerted by the heart

and vessels decreases. This phenomenon also explains not only the increased pressure and accumulation of blood in the central veins as the heart grows weaker, but also the efficacy of therapies that reduce resistance to the flow of blood.

Contrary to popular assumption, the force that drives the blood into the heart is not the force of the heart's pumping action persisting throughout the circulatory system and all the way back to the heart. In reality, the magnitude of filling in the venous system, and thus central venous pressure approaching the heart, is due to tissue activity, which produces the volume of blood present at any given moment. The organism as a whole constantly replaces blood fluid, diffusing it into the venous system through the capillary membranes. And this process is bound up with individual activity.

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