The mechanical nature of the heart as a pump

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An extracorporeal heart replacement pump has been developed which mimics cardiac function without any adjustments or controls. A comparison of the function of this pump with that of the heart indicates that its mechanical characteristics are the same as those of the heart. The inadequacy of conventional basic hemodynamic principles to explain the findings shakes the very foundation of cardiovascular physiology. A new, more realistic concept of basic hemodynamics is presented.

The mechanical pump (Fig. 1) has three basic hydraulic characteristics. It will be shown that the heart, in all of its complexity, also has only these three essential basic pump characteristics: (1) It fills passively (it does not suck at its inlet). (2) Its atria allow continuous uninterrupted venous inflow. (3) It has an intermittent or pulsatile outflow. Incomplete understanding of these three characteristics of the heart is evident in past medical writings.

Pulsatile blood flow was known to be of benefit in 1937, when Swindle[1] showed that it prevented intravascular agglutination, and allowed diffuse generalized perfusion of tissue. He indicated that the transfer of materials between the extravascular space and the capillary bed is facilitated by pulsatile blood flow. Wilkens, Regelson and Hoffmeister[2] have reviewed the evidence of the importance of pulsatile blood flow. Lillehei[3] showed by motion pictures that there was a decrease in the number of vessels with flow in them as the time increased using conventional nonpulsatile cardiopulmonary bypass. The other two characteristics of the heart have received less consideration. Norton,[4] Liotta,[5,7] and Seidel[6] and their associates, in recent attempts at cardiac replacement, have used nonsucking pumps. They use them to "prevent undue negative pressure in the vena cava and allow the heart replacement filling rate to be determined by the venous pressure." Wiggers and Katz[8] give evidence that the atria have a significant effect in normal physiology. Liotta,[9] Akutsu,[10] and Atsumi[11] and their colleagues reported the incorporation of atria in cardiac replacement pumps, but do not indicate whether these atria allow uninterrupted venous inflow. Most investigators in the field of mechanical heart replacement use pumps which are deficient in one or more of these three characteristics.[12-19] The importance of the understanding of these three basic mechanical characteristics to clinical cardiology, cardiac surgery, and the develop-

ment of a mechanical heart replacement makes this report timely.

Description of pump

The cardiac replacement pump (Fig. 1) consists of a ventricle which passes through three synchronized cam-driven plungers. The outside plungers are ¼ inch wide and act as inlet and outlet valves. The 8-inch wide center plunger acts as an impeller. The proximal 4 inches of the ventricle above the inlet valve acts as an atrium. The pump has three mechanical characteristics:

I. Passive filling. The nonsucking nature of the pump is due to the characteristics of the ventricle. A cutaway diagram in Fig. 2 shows the thin rubber ventricle with a flat cross section. This flat configuration prevents any sucking by rebound to a
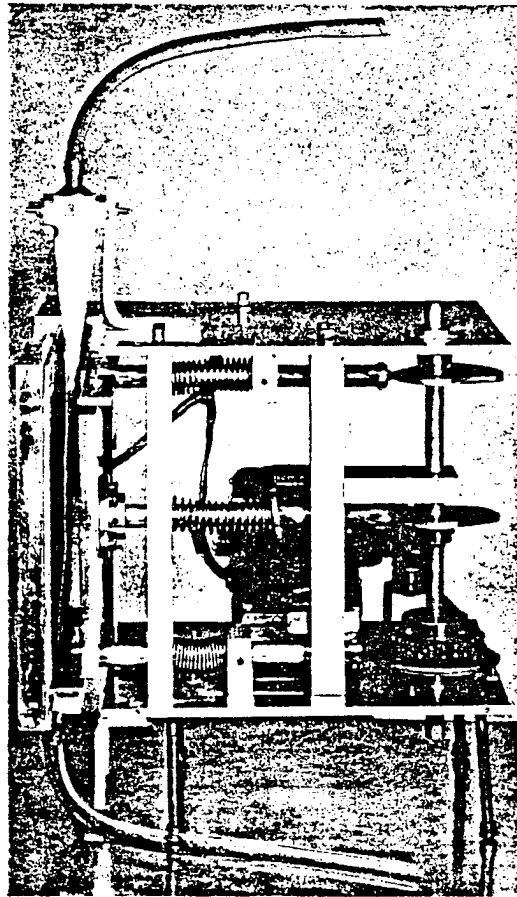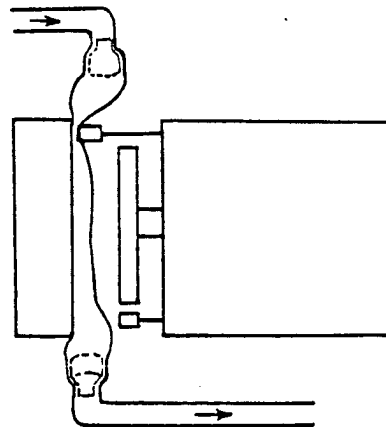

Fig. *1A*. A pulsatile nonsucking pump.

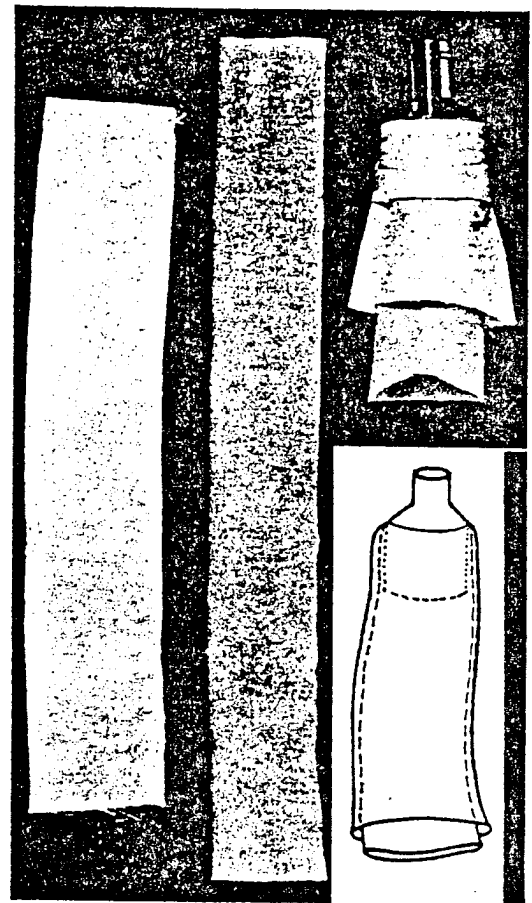Fig. *1B*. Diagram of the pulsatile nonsucking pump.

Fig. *2.* Construction of nonsucking pump ventricle. *Inset:* Cutaway diagram of pump ventricle.

round cross section after the ventricle has
been compressed. A cloth covering is added
for strength. Fig. 3*A* shows the lack of
diastolic sucking of such a ventricle in
comparison to the sucking of the resilient
tubing used in conventional roller and
finger pumps when their inlets are occluded.
The output range at 80 compressions per
minute with up to 12 cm. of venous filling
pressure from tubing of ½-inch I.D. is
from 0 to 4,200 c.c. per minute. Non-

sucking pumps, in contrast to ones that
suck at their inlets, do not produce a given
flow rate but merely pump out any fluid
that comes in, within their operating range
(Fig. 3*B*). If 50 c.c. per minute runs into
the pump, 50 c.c. per minute is pumped out.
If 4,200 c.c. per minute runs in, 4,200 c.c.
per minute is the output. On the other
hand, the output of sucking pumps is
dependent upon the speed or rate of the
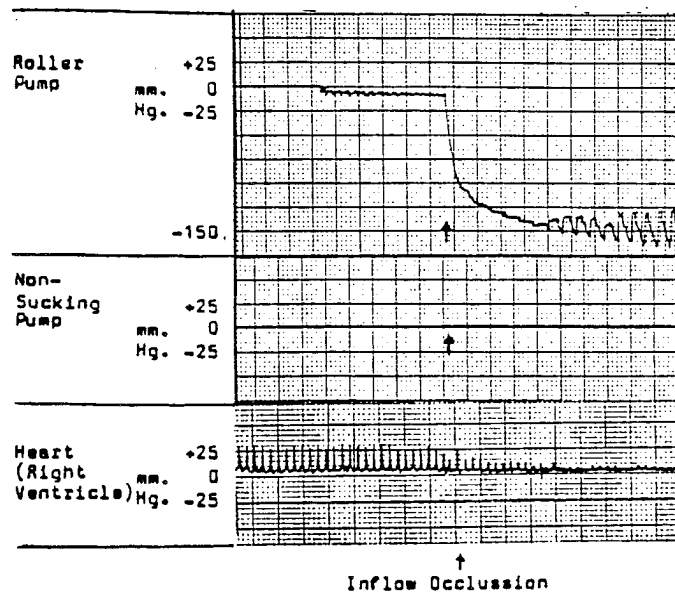pump. Changes in the rate of the pump

Fig. 3*A*. Absence or presence of negative pressure generated by pumps after inflow occlusion.
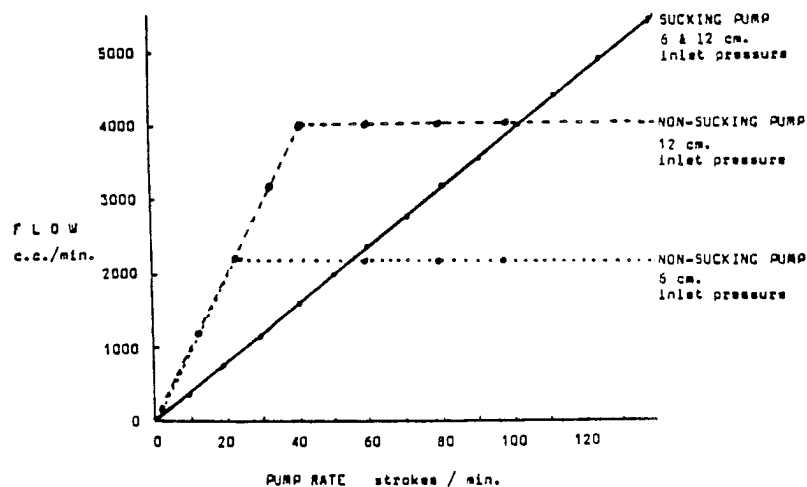

Fig. 3*B*. Pump output with sucking and nonsucking pumps with variation in pump rate and inflow pressure.

with nonsucking pumps merely change the stroke volume not the minute output. Nonsucking pumps are passive to their output. The flow rate is completely dependent upon outside factors which determine the flow rate to the pump. Fig. 3*B* shows findings obtained by changing the pump rates and inflow pressures of these two types of pumps. This illustrates the difference in their output regulation.

II. Atrial effect. The hydraulic effect of the "atrium" is that it allows uninterrupted inflow of fluid from the veins to the pump. A procedure which demonstrates the mechanism of this feature is shown by Figs. 4 and 5.

In Fig. 4,A, a tube ½ inch in diameter is placed into a reservoir of water with the tubing outlet 12 cm. below the level of the fluid. The fluid in the bottle at the
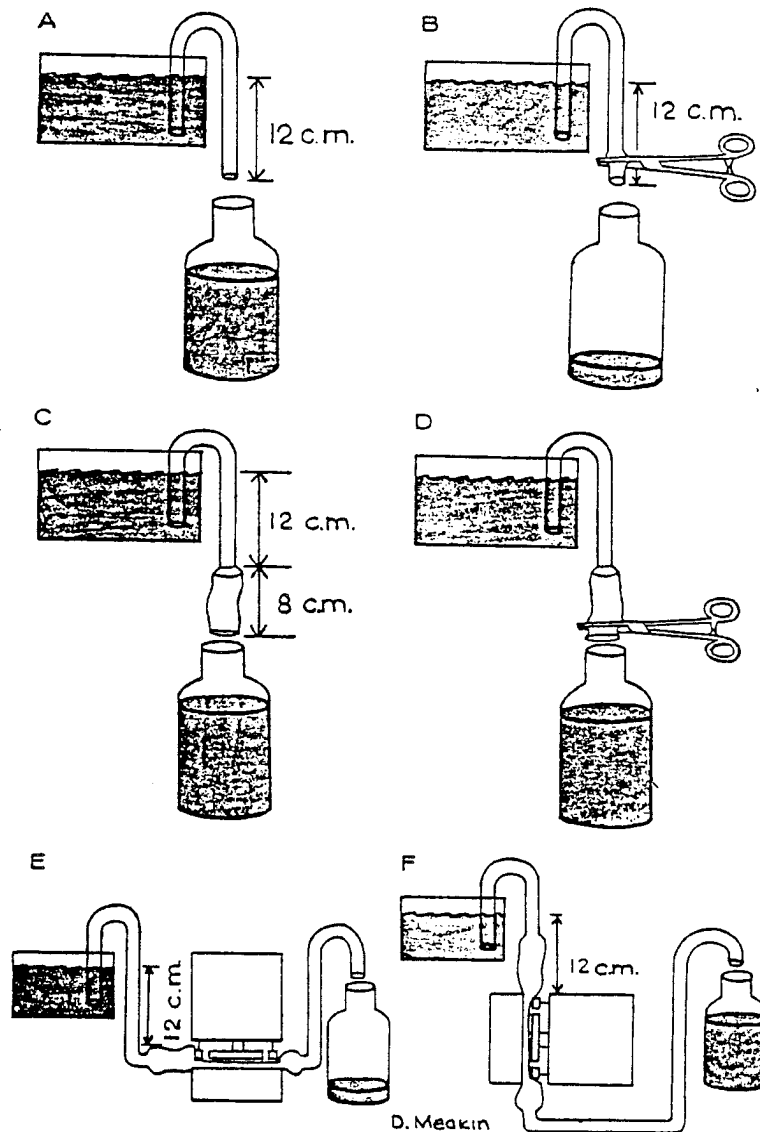

Fig. 4. *A*, Flow of fluid through ½-inch tubing at 12 cm. of pressure for 1 minute. *B*, Interrupted flow with tubing unclamped for a total of 45 seconds. *C*, Continuous flow with an "atrium." *D*, Interrupted flow with an "atrium." *E*, Nonsucking pump flow rate with 12-cm. inlet pressure with no atrial effect. *F*, Nonsucking pump flow rate with 12-cm. inlet pressure using an "atrium."

tubing outlet represents the 4,200 c.c. per minute that will flow in such a system. If the flow is now periodically interrupted, by clamping the tubing 80 times a minute for occlusion periods totaling only a fourth of the time, the flow is reduced to 800 c.c. per minute (Fig. 4,*B*). This decrease in flow is more than that caused by the absence of flow during the periods of interruption, since each period of interruption is followed by a period of decreased flow while the fluid is accelerating to its original rate (Fig. 5). If the frequency of the interruption is very great, there is almost no flow even though the tubing is open most of the time. The area beneath the flow velocity lines of Fig. 5 indicates the relative flow rates in continuous and interrupted flows at the same pressure.

The two systems represented by *A* and *B* of Fig. 4 are now modified to include a 4-inch segment of thin distensible rubber tubing at the tube outlets (Fig. 4,*C* and *D*). If fluid is allowed to flow in an uninterrupted manner, the flow rate, as before, will be 4,200 c.c. per minute (Fig. 4,*C*). It is shown, however, in Fig. 4,*D* that interruption of outflow in such a system does not reduce the minute flow rate as it did in Fig. 4,*B*. It remains 4,200 c.c. per minute. This is because dilation of the distensible segment allows the flow in the half-inch tubing to continue during periods of clamping. The flaccid tubing acts as an "atrium" which distends when its outlet is occluded and is emptied by gravity drainage and by fluid flowing through it when the clamp is removed. *E* and *F* of Fig. 4 show the effect of flow rates with a nonsucking pump with and without this atrial effect. In Fig. 4,*F*, the 4-inch segment of flaccid distensible tubing is above the pump inlet, so that it distends when the inlet valve is closed

Fig. 5. Comparison of continuous and interrupted flow rates and volumes. Shaded area represents volume.

and empties when it opens. This allows the pump to put out 4,200 c.c. instead of the 800 c.c. per minute seen in Fig. 4,*E*, wherein the pump systole interrupts the flow of fluid in the tubing. In Fig. 4,*E*, the position of the atrium eliminates the gravity emptying when the valve is open by eliminating the atrial effect and results in a decreased (800 c.c. per minute) flow rate. To duplicate the 4,200 c.c. flow rate with the interruption of flow without such an atrium required the elevation of the inlet pressure to 48 mm. Hg. Thus, if there are no atria, it is necessary to have four times the normal venous pressure to get the same output, from a nonsucking pump, as when atria are present.

III. Pulsatile outflow. A pulsatile outflow at a fixed rate of 80 per minute is provided by intermittent compression of the ventricle by the cam-operated press. Ventricular systole occupies a fourth of the cycle.

Procedures

Twenty seven mongrel dogs were used. In 15 dogs, both the left and right sides of the heart were bypassed with nonsucking ventricles placed in the pulsatile pump. The dog's own lungs were thus used for oxygenation. The heart was exposed through a median sternotomy. Tubing with a $\frac{3}{8}$-inch I.D. drained blood from each atria to its respective pump. Infusion from the two pumps was through pulmonary artery and aorta cannulae having an I.D. of $\frac{1}{4}$ inch. The pump inlets were placed at the level of the inferior aspect of the atria in order to have the prosthesis in the same hydrostatic position as the dog's own heart. Bypass was established by turning on the pump, unclamping the venous drainage tubes, and then fibrillating the heart. Termination of bypass was by defibrillating the heart, clamping the atrial cannulae, and then turning off the pumps. Bypass periods up to 14 hours were maintained.* No adjustment or regulation of the pump rate was made at any time.

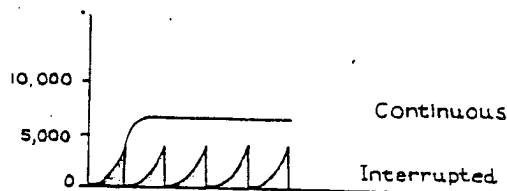Eleven other dogs were put on cardiopulmonary bypass using gravity drainage

to a bubble oxygenator and a pump with a single ventricle. The inlet of the ventricle was placed at the desired level of blood in the oxygenator. Bypass was utilized for periods up to 2 hours. No pump adjustments or regulations were made.

One attempt was made to perform a right heart bypass alone using a single ventricle and pumping from the right atrium to the pulmonary artery. This bypass left the lungs and left ventricle functioning.

To correlate findings from these procedures with normal physiology, mean vascular pressure experiments are included later under the heading *Cardiac Output*.

Findings

Fifteen dogs placed on cardiac bypass with two pulsatile nonsucking pumps with atria were observed to maintain normal mean arterial blood pressure and to react in a normal way to stimuli which normally cause alterations in blood pressure and circulatory rate. They also maintained normal blood volume balance between the systemic and pulmonary circuits. *This duplication of cardiac action occurred without any pump adjustments being made.*

Figs. 6, 7, and 8 from Dog No. 14 are typical of tracings obtained from all of the animals on two-pump bypass. Fig. 6 shows the arterial blood pressure after the onset of bypass, with the same mean pressure being maintained as before bypass. The systolic pressure on bypass is higher and the diastolic pressure is lower because the pump rate is slower than the heart rate had been before bypass. The pump, being slower, has a greater stroke output at the same flow rate. Fig. 7 shows the decrease in arterial blood pressure, while on bypass, after 400-c.c. hemorrhage, followed by the increase in blood pressure after administration of epinephrine. The epinephrine effect wore off in approximately 5 minutes. The resulting hypotension was then gradually corrected by giving back the 400 c.c. of blood. Fig. 8 shows the effect of Arfonad on the blood pressure in the same animal while on bypass. Six minutes after hypotension from Arfonad, epinephrine again increased the blood pressure. These changes in arterial blood pressure parallel, in each instance, the changes in the rate of blood flow as observed by the amount of di-

astolic distention of the pump ventricles. All of the changes demonstrated here were independent of the pump, since it was not adjusted but was passive and pumped out whatever came to it.

Blood volume balance between the pulmonary and systemic circuits was maintained at all times in every case without any need for adjustment of the pump. This was evident since no pulmonary edema or hypoxia developed, such as that which occurs so easily with sucking-pump systems. To demonstrate the effectiveness of this nonsucking pumping system in preventing pulmonary edema from imbalance of the two pumps, the following experiment was performed during two of the procedures. Blood was removed from the aorta and injected into the pulmonary artery in increments of 25 c.c., until an amount equal to more than the entire volume of blood had been exchanged over a 10-minute period. This caused no change in compliance and no pulmonary edema or evidence of any imbalance in the volumes between the two systems. The distensibility and resistance of the two systems determined the blood volume balance between them. For example, if the pulmonary and systemic circuits are in equilibrium, there is essentially equal right and left output. If for any reason there is a shift of blood from the systemic circuit to the pulmonary compartment, the mean systemic pressure will fall and the mean pulmonary pressure will rise. This causes a slowing down of blood returning to the right heart, and a speeding up of blood coming to the left heart. Since both pumps put out whatever comes to them, the left ventricular output then exceeds the right until the old equilibrium is again established. This is simply hydraulic cause and effect and is not dependent on any reflex or adjustment when nonsucking pumps are used. This explains why in intact circulatory systems there can be loss of fluid by loss of water in exhaled air, gain of fluid from the gastrointestinal tract, addition of blood by transfusion to the systemic circuit, and large left-to-right or right-to-left shunts with no imbalancing of the volume equilibrium between the two circuits. By contrast, pumps which suck at their inlets have their output determined by their rate, so that
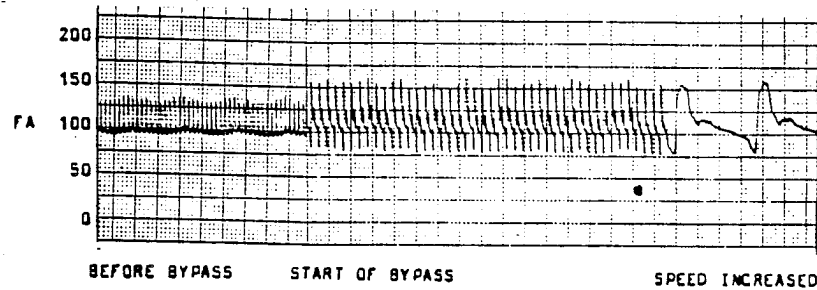
Fig. 6. Arterial blood pressure of Dog No. 14 before bypass and after onset of cardiac bypass with two pumps, using the lungs for oxygenation.
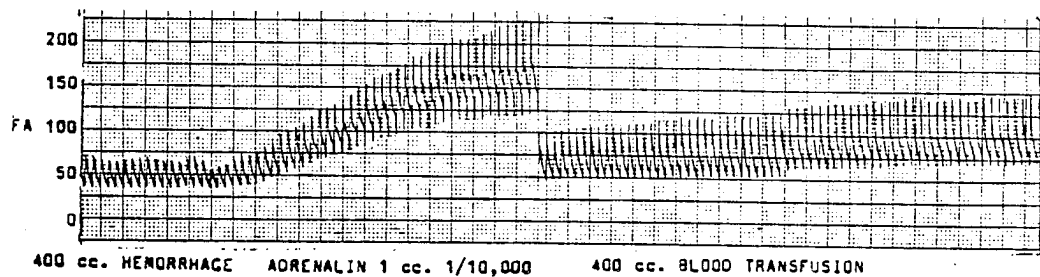

Fig. 7. Arterial blood pressure in Dog No. 14 with the cardiac replacement pump, showing the effect of hemorrhage, adrenaline, and blood replacement without use of pump controls.
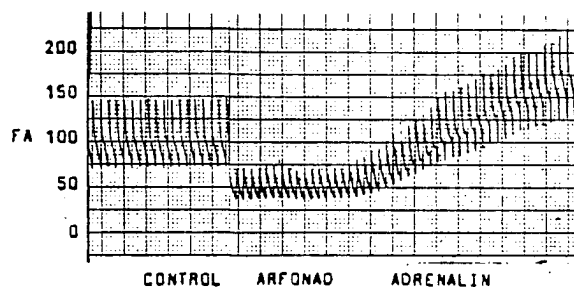

Fig. 8. Effect of Arfonad on blood pressure of Dog No. 14 with cardiac replacement pumps.

constant right and left pump rate alterations are necessary to maintain blood volume balance. Any inequality in pumping rate or stroke volume of the two pumps progressively drains one compartment and overdistends the other. Also, the addition of fluid to, or subtraction of it from, a compartment or a shunt is not followed by an automatic shift to or from the other compartment, so that no restoration of equilibrium occurs. So far, attempts to bypass the heart with two sucking pumps have required complicated sensing devices

and controls, and still have been deficient in satisfactory control of volume equilibrium. With nonsucking heart replacement pumps the normal volume-equilibrating mechanism between the pulmonary and systemic circuits is a function of the vascular bed, and is automatic, with no pump regulation.

The 15 dogs in this group had cardiac replacements with perfusions for as long as 14 hours. The ventricles would last 8 to 14 hours, then rupture and end the experiment. Thus, in all but one animal no at-

tempt at survival was made. In that animal, a short bypass of 2 hours was terminated because of a leak at a connection in the arterial line. This dog's wounds were repaired and he recovered and was a long-term survivor. Experience with pumping parallel with the left ventricle was obtained during the course of these studies. Also, short periods of perfusion with left ventricular bypass alone were utilized prior to going on complete heart bypass. There was no alteration in normal blood flow in these situations, since whatever blood did not go to the left ventricle was automatically pumped by the mechanical pumps, and vice versa.

The second group of dogs, in which a single pump and a Travenol oxygenator primed with blood were used, demonstrated all of the above-mentioned responses seen with two pumps. The nonsucking pump *without regulation* would maintain a constant level of blood in the oxygenator at the level of the pump inlet. This eliminated any hazard of pumping bubbles or exhausting the supply of blood in the oxygenator. Here again, any change in blood volume in the animal or any change in vascular tone due to vasopressors or dilators was reflected in a corresponding change in circulatory rate and arterial blood pressure. Loss of blood resulted in low arterial blood pressure and low blood flow rate, as shown by a decrease in ventricular filling and the smaller size of the blood stream in the oxygenator helix. Replacement of blood caused restoration of circulatory rate and arterial blood pressure. Vasopressors caused increased flow rate and elevated arterial blood pressure. The converse was true with Arfonad.

This pump set up with an oxygenator allowed normal circulation and circulatory responses to physiologic stimuli without need for controls. After the first 7 animals had been lost as we learned the method and overcame the technical problems, there were 4 successive survivors.

The one experience with right heart bypass alone demonstrated a serious hazard due to such a procedure. After ½ hour of pumping, manipulation of the heart caused ventricular fibrillation. The replacement pump continued its output, but the left heart, in arrest, did not. Thus, the

lungs were quickly damaged beyond recovery by a large shift of blood from the systemic circuit. In contrast to the above-mentioned experience, incidental ventricular fibrillation is not such a hazard with left heart bypass, since the systemic circuit can accommodate any shift of blood volume from the lungs.

The heart as a pump

The mimicking of normal circulation by a nonsucking, continuous-inflow (atrial effect), intermittent-outflow pump directs attention to these three factors as being the essential mechanical characteristics of normal heart function. If the heart is, in effect, like the replacement pump, its nonsucking characteristic would make it a passive organ, pumping out merely what comes to it, with no regulation over its output and with no control over the volume of blood between the lungs and systemic circuits. This is opposite to conventional concepts of cardiac output, which must then be replaced with new ones.

I. Evidence that the heart is a nonsucking pump. The heart muscle, like other muscle in the body, does not expend energy to forcibly elongate. The heart, therefore, cannot forcibly generate pressure negative to its surroundings.

Evidence that the heart does not suck is commonplace in cardiac catheterization data. Fig. 3.4 shows that the heart does not suck significantly at its inlet. A pressure recorded from a catheter placed in the right ventricle falls to zero when the venae cavae and coronary sinus are occluded. The tracing shows that no significant negative pressure results. Negative intrathoracic pressure occurs with forceful inspiration, but at that time the intracardiac pressure is not negative to the intrathoracic pressure. Here the chest is sucking but the heart is not.

Investigators in the past have tried to show that the heart does suck. Bloom[20] noted that a beating excised rat heart would jet propel itself in a tub of water and concluded that this showed that the filling at diastole was from sucking. This work demonstrates that the heart can develop a very small negative pressure under some conditions. This does not conflict with the concept that the heart in its normal func-

tion is a nonsucking pump. Normally, the end-systolic volume in the heart and the rapid filling from normal inflow pressure never allow the heart to develop any negative pressure. In Bloom's study there is no determination that would indicate more than an insignificant cardiac output when the heart, in this abnormal situation, acts as a sucking pump.

The heart then is a hollow muscular organ similar to the urinary bladder. It expels what comes to it, with no regulation of the volume that goes through it in a given time. Therefore, with the nonfailing heart, the circulation rate is determined by extracardiac factors. Also, the balance between the pulmonary and systemic compartments is determined by extracardiac factors.

II. Atrial effect. Normal atria produce their benefit to circulation by allowing continuous, uninterrupted venous flow to the heart in a way similar to that shown with the replacement pump. Henderson,[21] in 1906, proposed that atria were not force pumps because they had no inlet valves. Wiggers and Katz[3], Mitchell, Gilmore and Sarnoff,[22] and, more recently, Mitchell, Gupta and Payne[23] have shown that atrial systole does contribute markedly to cardiac output. They, therefore, concluded that the atria were, in effect, force pumps.

Compatibility of these two apparently divergent observations is found in the function of the atria of the replacement pump. Past explanations of atrial function have been merely a description of the sequence of events. The replacement pump shows the physiologic benefit of these events.

At the onset of ventricular systole, as seen by venous cineangiograms and observation at operation, the relatively empty atria allow continuation of venous flow that would otherwise be stopped by closure of the A-V valves at ventricular systole. During ventricular diastole, venous blood flow is through the atria to the ventricles. This flow takes with it the stored atrial blood as well as the volume displaced by the atrial contraction. The atrial contractions which occur just before the A-V valves close leave the atria relatively empty and distensible so that they can accept the venous flow during the ensuing ventricular contraction. Blood from the atrial contraction goes to the ventricles, being propelled there by the forward velocity of the incoming venous blood and the pressure gradient between the veins and the ventricles. During contraction the caliber of the atrial lumen is never narrowed to the extent of increasing the resistance to flow from the veins to the ventricles. Atria are,
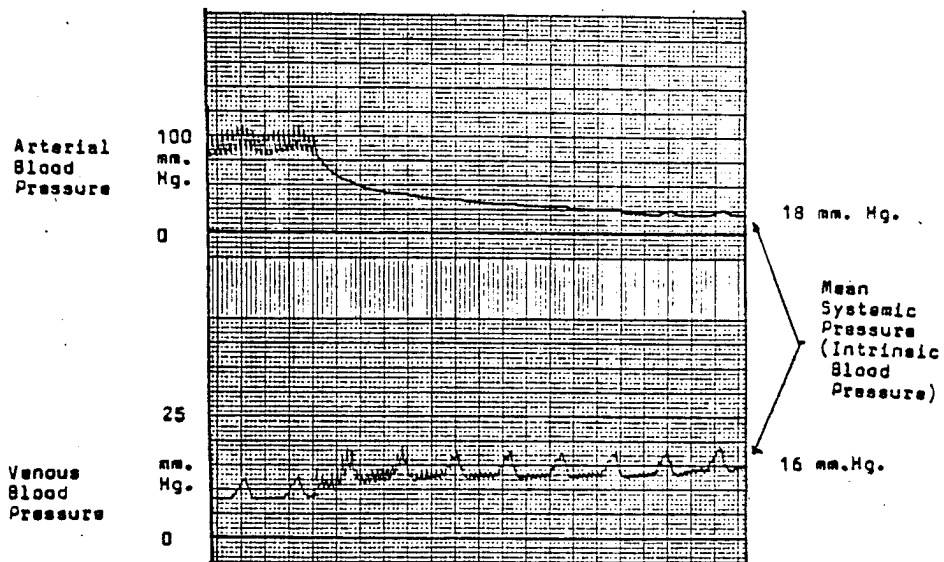

Fig. *9*. Arterial and venous blood pressure equilibration to a mean vascular pressure of approximately 16 mm. Hg after fibrillation of the dog's heart.

therefore, intermittent storage chambers which prevent interruption of venous flow at each systole. Without this atrial effect, interruptions during ventricular contractions would markedly reduce cardiac filling and circulation rate. This atrial effect explains the greater cardiac output in patients with sinus rhythm than in those who have atrial fibrillation. Absence of this atrial effect explains the decreased or marginal rate of blood flow obtained by any method of cardiac massage for cardiac arrest in the supine position. Cardiac massage was observed to interrupt venous flow as the ventricles were compressed. Cardiac pacemakers with atrioventricular synchronization produce their benefit not merely by their changes in rate in a normal fashion, but by their synchronized effect that restores the uninterrupted venous blood flow, thereby allowing normal cardiac output.

III. Pulsatile output is an obvious characteristic of the heart. Evidence of others that pulsatile blood flow is necessary to give diffuse perfusion of tissue is fortified by our 3-year clinical experience with pulsatile blood flow during cardiopulmonary bypass. Increased urinary output, improved electroencephalographic patterns during rewarming of hypothermic perfusions, and increased tolerance to long perfusions have been apparent by the utilization of pulsatile pumps.

The heart, like the mechanical pump, is a pulsatile-outflow, continuous-venous-inflow, nonsucking pump. It is a passive pump which pumps out what comes to it, with no regulatory effect on circulatory rate or blood volume balance between the pulmonary and systemic circuits.

Cardiac output (a new insight based on the concept of the nonsucking passive heart)

With the new realization that the non-failing heart is passive to the regulation of cardiac output, an investigation of the factors that do regulate and determine this passive cardiac output was made.

In any such hydraulic circuit in which the circulating pump pumps out what comes to it, the flow rate is determined by two factors—mean hydraulic pressure in the circuit and resistance to flow. In the vascular system these would be "mean vascular pressure" and "resistance to blood flow."

$$\text{Flow rate} = \text{function} \frac{\text{Mean Vascular Pressure}}{\text{Resistance}}$$

or

$$F = f \frac{MVP}{R}$$

1. Mean vascular pressure. In 1850, E. H. Weber[24] made a model elastic vascular tree. It contained a pump with valves and elastic tubing for arteries and veins, and a sponge for a capillary bed. Manometers were placed on the arterial and venous sides. With the pump at rest a "hydrostatic mean pressure" was present because the enclosed fluid exerted a pressure on the elastic system. He postulated a "hydrokinetic mean pressure" or mean pressure in the system with the fluid in motion. He concluded that the hydrostatic and hydrokinetic pressures were equal because, if the arterial pressure was elevated, the venous pressure was reduced by the same increment.

E. H. Starling,[34] in 1897, confirmed Weber's work and used the term "mean systemic pressure."

Bolton and then Starr,[35] in 1940, showed that blood pressure was elevated after death from congestive heart failure (static blood pressure).

Guyton, Polizo and Armstrong,[26] in 1954, observed transient increases in mean vascular pressure (mean circulatory filling pressure) after the injection of adrenaline or the addition of fluid to the vascular system.

In 1954, R. M. Anderson[27] introduced an indirect method of measuring mean vascular pressure (intrinsic blood pressure) during life. He postulated that the mean vascular pressure existed in a system which was constantly gaining and losing fluid. This pressure was, therefore, dependent on the balance between energy forcing fluid into the vascular system and the resistance to flow out of the vascular system: P = f (E. R.). Changes in the tone of the vascular container caused only transient changes in pressure while the size of the container was changing. Shock,

some types of hypertension, and congestive failure were defined in terms of "mean vascular pressure."

STUDY OF EFFECT OF MEAN VASCULAR PRESSURE ON CARDIAC OUTPUT. In anesthetized dogs, in various physiologic states, circulation was stopped by inducing ventricular fibrillation by a relatively long low-voltage shock. As cardiac output stopped, the arterial pressure fell and the venous pressure rose, so that they approached the same pressure as the fluid became stationary. The resulting mean vascular pressure in the vascular system was recorded. The circulation was then restarted by defibrillating the heart. After fibrillation, 10 to 15 seconds was all that was necessary for the pressure to equilibrate, except for a very small gradient. Fig. 9 shows a normal mean vascular pressure of 16 mm. Hg in a dog with normal arterial blood pressure. The dog was then resuscitated, and repetition of fibrillation after recovery periods consistently gave a pressure of 16 mm. Hg. This has been shown in our laboratory to be the normal mean vascular pressure in dogs. Fig. 10 shows the blood pressure response after intravenous injection of 1 c.c. of 1-10,000 epinephrine. After a maximal response of elevated blood pressure had been obtained, the heart was fibrillated and the mean vascular pressure was re-

corded at 32 mm. Hg, or twice normal. Fig. 11 shows low mean vascular pressure after loss of blood. The same mean vascular pressure and blood pressure responses were obtained in animals with hearts replaced by nonsucking pumps. In these cases, the flow rates, as seen by increases and decreases in diastolic pump filling, correlated with elevations and decreases in arterial blood pressure. This shows that the increased circulatory rate (increased cardiac output) after the use of epinephrine is due to a rise in mean vascular pressure from increased tone of the vascular system. The cardiac effect of epinephrine in increasing the heart rate and the strength of contraction prevents the heart from failing in this situation. However, the heart remains passive in so far as determining the circulatory rate.

2. *Peripheral resistance.* The circulatory rate is inversely proportional to changes in peripheral resistance. The previous data (Figs. 6-11) support the concept that blood pressure elevation from vasopressors is due to increased mean vascular pressure and not to an increase in resistance. The blood pressure elevation is concurrent with increased circulatory flow rate instead of a decreased rate that would accompany a blood pressure elevation from increased arteriolar resistance. The mechanical effect
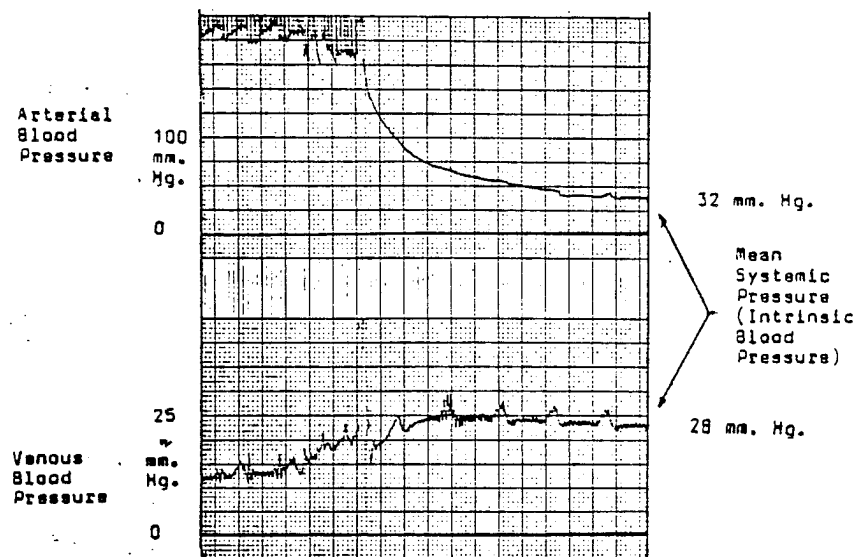

Fig. *10.* Dog arterial and venous blood pressure equilibration. An elevated mean vascular pressure after administration of adrenaline.

of exercise increases cardiac output by decreasing peripheral resistance. This occurs when the contracting muscles around veins containing valves pump blood through channels the resistance of which would otherwise slow the flow.

The cardiac effect of drugs, changes in heart rate, and Starling's law in new perspective

Increased cardiac contraction in response to drugs, increased strength of myocardial contraction to increased diastolic filling (Starling's law), and increased heart rate are all observed in situations of increased cardiac output. They have been misunderstood to be the cause of increased cardiac output because they occurred concomitantly with it. With the previous findings it is seen that with a pump which has a fixed rate and one that cannot react to drugs, etc., circulatory responses are kept intact. They occur without any of the above-mentioned responses. These three are simultaneous responses to situations with increased circulatory rate and do have a physiologic benefit in the conservation of energy expended by the heart. If the heart contracted with maximum strength all of the time, the circulation rate would vary in a normal fashion to all situations, but this would be a great waste of energy

most of the time. If, however, the heart contracted at minimal strength all of the time, it would be in failure in high-output situations. The responses which increase the strength of myocardial contraction insure adequate strength to prevent failure during rapid circulation and the conservation of energy by the heart during periods of slow circulation.

Augmentation of this experimental evidence is found in the following clinical examples of the independence of the output and heart rate: Distance runners may have slow heart rates in the presence of high output. Variation in cardiac output occurs with fixed heart rates from cardiac pacemakers. Tachycardia may occur in hypovolemia with low output. High-output hypertension may occur with a slow heart rate.

It is seen, therefore, that the rate of circulation (cardiac output) is regulated by the extracardiac factors: mean vascular pressure and vascular resistance. Changes in heart rate and strength of contraction insure absence of failure at high outputs. The stroke volume of the nonfailing heart is inversely proportional to heart rate for any given circulatory rate. Stroke volume is determined by these two factors rather than being a determinant of them.
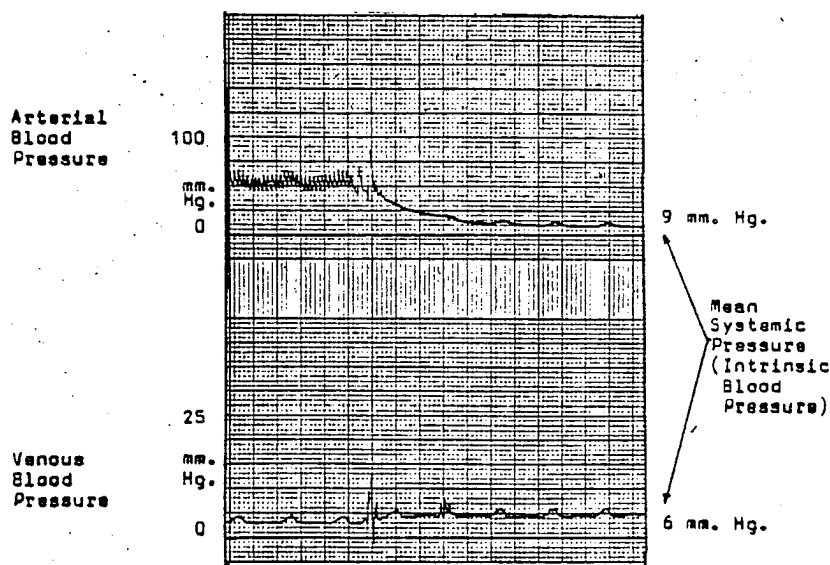
The nonfailing heart, in all of its com-

Fig. *11*. Dog arterial and venous blood pressure equilibration to a low mean vascular pressure after loss of 400 c.c. of blood.

plexity, is a passive pump, pumping out what comes to it without any regulation over the rate of circulation.

Conclusions

1. A cardiac replacement pump which duplicates cardiac function in all physiologic states with no controls or adjustments has been used in dogs.

2. The essential characteristics of the heart and also of this replacement pump have been demonstrated: (1) They are nonsucking pumps, (2) they have a pulsatile outflow, and (3) there is uninterrupted venous flow into them.

3. A new concept of basic hemodynamics is presented which gives better insight into the passive nature of the heart in the regulation of cardiac output and of blood volume between the lungs and systemic circuits.

4. The atria are found to have their physiologic benefit in allowing continuous venous flow to the heart in the presence of an intermittent pumping system.

5. Cardiac output (in the presence of a nonfailing heart) is shown to be determined completely by extracardiac factors.

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