THE INTERRELATIONSHIP OF FACTORS CONTROLLING CARDIAC OUTPUT

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ABSTRACT

Our understanding of the mechanics of circulation may be broadened by testing old and new concepts on a new hydraulic model made possible by a unique pump which resulted from the evolution of open heart surgical equipment. Findings in human physiology which have corollaries in the model can be analyzed more easily in the model, with the resulting conclusions transferable with reasonable validity. Factors thought to control cardiac output were tested. The results indicate that there are two separate and distinct sets of control factors, with only one set being operative at a time. It is the set that is potentially limiting cardiac output the most that is the determinant at any time. Past paradoxes in our perception of human circulatory physiology are accommodated by the resulting concept.

Key Words: Cardiovascular Dynamics, Cardiovascular Function, Circulation Rate Control, Cardiac Output, Mean Vascular Pressure.

INTRODUCTION

A hydraulic model of the cardiovascular system, upon which new and old physiologic concepts about circulation may be tested, has been a by-product of the development of cardiac bypass equipment used in surgery.

The duplication of many physiologic cause and effect phenomena is made possible in the model by an unique pump which has closely mimicked heart function in a large number of animal experiments (1) and human clinical applications (2).

Most variables and reflexes thought to effect cardiac output can be tested in the model. Study of the model has the advantage over studying "in vivo" preparations as one or more variables at a time can be
changed in the model without the uncertainty that some hidden, undetected change might have occurred that would invalidate conclusions.

Study of the hydraulic model also has the advantage over study of a computer or mathematical model, as fluid mechanics eliminates the possibility of overlooked or exaggerated evaluation of factors in a very complex hydraulic system.

Findings using the model have resulted in a concept of cardiac output regulation that accommodates paradoxes in explaining:

1) Why pulse rate alteration does not always effect cardiac output:
   a) When altering pacemaker rates in patients with non-failing hearts (3, 4).
   b) When decreasing pulse rates with propranolol in normal subjects (5).
   c) When increasing pulse rates during hypovolemic, neurogenic and endotoxic shock (6, 7, 8, 9).

2) Why strength of myocardial contraction alteration does not always alter cardiac output:
   a) When digitalis is given to normal subjects (10).
   b) When propranolol decreases myocardial contractility in non-failure states (5, 11).
   c) When intra-aortic balloon augmenting myocardial contraction causes no increase in cardiac output in non-failure states (12).

3) Why total vascular resistance increase causing essential hypertension is not accompanied by a decrease in cardiac output (13, 14).

4) Why "afterload" increase from aortic stenosis (15) or coarctation of the aorta (16) is unaccompanied by a decrease in cardiac output.

5) Why "pre-load" increase during heart failure fails to increase cardiac output.

THE MODEL

Figures 1 and 2 show the major components of the simulated cardiovascular system.

Pumps: The unique simulator heart pumps share three characteristics of the heart that allow them to closely approximate cardiac function (1, 2): they are non-sucking at their inlets, allow uninterrupted venous flow to their inlets, while providing pulsatile outflow to the arteries.

Each pump consists of an atrial chamber and a ventricular chamber with inlet and outlet ball valves (Fig. 3). These pumping chambers have rigid Lucite saucer-shaped outer shells (Fig. 4A) with mirror image rubber diaphragm inner shells (Fig. 4B). These pumping chambers are opposed
by similar shaped impeller chambers which also consist of rigid Lucite outer shells (Fig. 4E) and inner rubber diaphragms (Fig. 4D). A 1/4-inch thick Lucite gasket (Fig. 4C) placed between the opposing impeller and pumping chambers has side holes that make free communication between the room air and the space between the opposing diaphragms.

The pumps are driven by alternately delivering compressed air and suction to the impeller chambers. An electronic regulator triggers solonoid valves to control the pulse rate and ratio of systole to diastole. Reducer valves regulate peak pressure and onset to peak time.

1) Non-sucking characteristic: The Lucite gasket with side holes lets room air between the pumping and impeller diaphragms during forceful evacuation of the impeller chamber during ventricular diastole. The negative pressure in the impeller chamber is thus applied to the room air instead of to the pumping diaphragm.

2) Uninterrupted inflow to the pumps is provided by the atria.
Atria emptied during ventricular diastole accommodate venous flow during ventricular systole, thus preventing interruption of flow during ventricular systole. Then during ventricular diastole, atria empty the accumulated blood into the ventricles without venous interruption. A rigid "Y" segment of the atrium between the inlet veins and the inlet valves prevents interruption of venous flow by the blocking of a completely emptied atrium that would otherwise occur at the end of atrial systole.

3) Pulsatile outflow is provided by alternate positive and negative air pressure being delivered to the impeller chambers. The pulse rate is variable from 1 to 160 compressions/min. with a pressure from 1 to 30 p.s.i.

The Simulated Vascular Network consists of a systemic and pulmonary network in series with the two pumps interposed. Major vessels are silicone tubing, while the capillary beds are made of more compliant Penrose drain material.
Figure 3. Assembled model heart pump, side and top views.

Metering valves for altering resistance are incorporated in the venous and arterial tubing (Fig. 1,e). Pressure-sensitive transducers (Fig. 2, #5, #6 & #8), connected to a direct writing recorder and electromagnetic flowmeter and probes, are used to monitor pressures and flow. A "transfusion" reservoir permits alteration in the fluid volume within the system. Weights, placed on a plate resting on the capillary and venular portions of the model, are used to vary the compliance of the system.

PROCEDURES

The effect on pump output from modifying one circulation variable at a time, while keeping others constant, was measured using the model. The variables tested were: 1) mean vascular pressure, 2) pump rate, 3) strength of pump contraction, and 4) resistance in the vascular network.

Experiment #1. Relationship of Mean Vascular Pressure to Pump Output.

("Mean vascular pressure" is the resting pressure in the system that results from the fluid volume and the compliance of the system. Mean vascular pressure was measured with the pumps turned off with the pressure equalized in all parts of the model).

Mean vascular pressure was changed by adding fluid, leaving compliance, the other factor, unchanged. Starting with a mean vascular pres-
Figure 4. Disassembled model heart pump.
A - Atrial and ventricular pumping chamber shell and valves.
B - Rubber pumping compartment diaphragm.
C - Spacer with side holes which allow free movement of ambient air to the space between the impeller and pumping diaphragms.
D - Silicone rubber impeller diaphragm.
E - Impeller chambers with atrial and ventricular connectors for suction and compressed air.

sure at zero, with 2,000 cc. volume in the model, appropriate amounts of fluid were added to change the mean vascular pressure in steps of 2 cm. water pressure. The amounts added were predetermined from a volume pressure curve made while adding fluid to the system with the pumps off (Fig. 5). Factors kept constant besides compliance were pump rate of 80 beats/min., impeller pressure of 16 p.s.i. and the resistance.

Findings:

1) Figure 6 shows a direct correlation between pump output and mean vascular pressure over the range of 0 to 20 cm. of water pressure.
2) At no time during the range of pressure studied did the ventricles fill maximally at diastole.
3) At no time was there any interruption in venous flow by the intermittent closing of the inlet valves.

Experiment #2. Pump Rate Correlation with Pump Output.

The pump rate in the model was varied from 0 to 170 beats/min. with other variables remaining constant (Fig. 7). The mean vascular pressure was at 12 cm. water pressure, with the impeller pressure at 14 p.s.i. and the resistance kept constant.

Findings:

1) At pump rates between 0 and 20/min.:
Figure 5. Mean vascular pressure - Volume relationship in the circulation model.

Figure 6. Pump output correlation with mean vascular pressure. Such variations in mean vascular pressure result from either volume or compliance changes within the system.
Figure 7. Relationship of Pump Rate to Flow. Composite tracing of flowmeter measurement above 20 beats/min. and mechanical measurement below 20 beats/min. where the flowmeter was non-operative.

a) The pump output correlated in a linear way with the pump rate (Fig. 7, A to B).
b) The ventricles filled to capacity at each ventricular diastole.
c) The atria filled to capacity during each ventricular systole.
d) Venous flow was interrupted during each ventricular systole.

2) At pump rates between 20 and 40/min.:
a) The pump rate correlated in a non-linear way with the pump output (Fig. 7, B to C).
b) The ventricles were filled slightly less than capacity at ventricular diastole.
c) The atria were filled to capacity at the end of ventricular systole.
d) Venous flow was slowed but not completely interrupted before the end of ventricular systole.

3) Increasing pump rate from 40 to 110:
a) Caused no increase in pump output (Fig. 7, C to D).
b) Was associated with progressive decrease in ventricular end-diastolic volume.
c) Caused progressive decrease in atrial end-diastolic volume.
d) Was unaccompanied by any interruption in venous flow.

4) Increasing the pump rate from 110 to 170 (Fig. 7, D to E) was accompanied by a progressive increase in residual air pressure in the ventricular impeller after ventricular systole which:
a) Progressively decreased pump output.
b) Impeded ventricular filling at ventricular diastole.
c) Caused venous flow interruption at ventricular systole.

Experiment #3. Ventricular Impeller Force Correlation with Pump Output.

The ventricular impeller pressure was varied from 0 to 20 p.s.i., in increments of 2 p.s.i., while keeping other variables constant. The
pump rate was 80, the mean vascular pressure was 12 cm. water pressure and all resistance remained unchanged.

Figure 8. Relationship between ventricular impeller pressure and pump output.

Findings:

1) At impeller pressures between 0 and 10 p.s.i. there was:
   a) Linear correlation between impeller pressure and pump output (Fig. 8, A to B).
   b) The ventricles were never completely emptied at ventricular systole.
   c) The ventricles were completely filled at ventricular diastole.
   d) Venous flow to the pumps was interrupted at each ventricular systole.

2) At impeller pressures between 10 and 18 p.s.i. there was:
   a) No increase in the output as the pressure increased (Fig. 8, B to C).
   b) Maximal ventricular emptying at systole.
   c) Sub-maximal filling of the ventricles at diastole.
   d) No venous flow interruption at any time.

3) As the impeller pressure was progressively increased above 18 p.s.i. (Fig. 8, C to D) there was progressively incomplete evacuation of compressed air in the ventricle impeller during diastole which:
   a) Progressively decreased pump output.
   b) Impeded ventricular filling at diastole.
   c) Caused venous flow interruption at ventricular systole.

Experiment #4. Relationship of Resistance to Pump Output

Resistance at a variety of sites was changed in the model's vascular network while maintaining other factors constant with the mean vascular pressure at 12 cm. water, pump rate at 80 and an impeller pressure at 16 p.s.i. There was a marked difference in response to a given resistance, depending upon where the resistance was placed in the model.
Responses fell into two categories which became more obvious the closer the resistance was to either the inlet or outlet of a pump. Therefore, resistance was studied in two situations: near a pump outlet and near a pump inlet.

Experiment #4a. Resistance Near a Pump Outlet.

A metering valve was placed 20 cm. from the left heart homologue pump outlet (Fig. 2, #9) across which a pressure gradient could be monitored by transducers (Fig. 2, #8), one on each side of the valve.

![Figure 9. Relationship of outflow resistance to pump output.](image)

- A - No resistance across outlet metering valve.
- B - Beginning pressure gradient indicating addition of minimal resistance.
- C - 100 mm Hg gradient manifesting moderate outflow resistance with normal flow.
- D - Marked resistance with marked flow reduction.

Findings:

1) As the metering valve was progressively closed (Fig. 9) the superimposed arterial pressure tracings which demonstrated no gradient initially (Fig. 9, A to B), separated (Fig. 9, B to D) as resistance was added.

2) With increase in outflow resistance up to 100 mm Hg gradient there was no drop in pump output (Fig. 9, B to C).

3) Further increase in gradient from 100 to 250 mm Hg did cause a corresponding drop in flow (Fig. 9, C to D).

4) In the range where there was no drop in flow (Fig. 9, B to C) the left ventricle did not fill maximally, but did empty maximally, and there was no interruption of venous flow at any time during the pumping cycle.

5) However, when the gradient became great enough so that the flow slowed (Fig. 9, C to D), the left ventricle progressively emptied less completely, and the residual volume resulted in complete left ventricular filling at diastole and interruption of venous inflow at each systole.

6) Whenever the left ventricle was being filled completely at diastole, increasing the mean vascular pressure caused no concomitant
increase in pump output.

7) As slowing of flow occurred (Fig. 9, C to D) there was gradual increase in a greater than previous volume in the pulmonary circuit at the expense of the systemic circuit. The greater the slowing, the more the volume equilibrium shifted to the simulated pulmonary circuit.

Experiment #4b. Resistance Near a Pump Inlet.

A metering valve was placed 20 cm. from the left pump's inlet (Fig. 2, #7) across which a pressure gradient could be monitored by way of transducers (Fig. 2, #6). The metering valve was progressively closed. The resulting inflow resistance magnitude is represented by the pressure gradient across the valve. Figure 10 shows the effect of increasing inlet resistance as the two superimposed "CVP" lines (Fig. 10, A to B) become separated (Fig. 10, B to C).

![Figure 10. Relationship of inflow resistance to pump output.](image)

A to B - No resistance at pump inflow.
B to C - Flow progressively decreases as inflow resistance increases.

Findings:

1) There was a direct inverse correlation between pump output and inflow resistance to the pump (Fig. 10, B to C) over the whole range studied. This is in marked contrast to outflow resistance of the previous experiment (Fig. 9, B to C).

2) As inlet resistance increased there was progressively less filling of the ventricles at diastole. Maximal emptying did occur at each ventricular systole, and venous flow remained uninterrupted at all times.

3) Progressive increase in left pump inflow resistance shifted the fluid volume equilibrium between the pulmonary and systemic circuits toward the pulmonary bed.

4) The pump with the greatest inflow resistance was the major determinant of the output of both.
Discussion: Relationship of Inflow and Outflow Resistances To One Another and To Pump Output

In that the vascular system is a circle, every point is both upstream from a pump inlet and downstream from a pump outlet. Therefore, every resistance point may offer some inflow and some outflow impediment to flow. The magnitude of impediment, however, that a given resistance offers to flow has one other factor; the amount of vascular compliance upstream from that resistance point.

The less the compliance of the bed upstream from a resistance point, the greater is the outflow impediment, and the less is the inflow impediment. Conversely, the more compliant the bed upstream from a resistance point, the less is the outflow impediment and the greater is the inflow impediment.

The pump's ability to expend energy to overcome outflow impediment, and its inability to expend energy to overcome inflow impediment, makes any generalization about the correlation of pump output to resistance alone, without considering upstream compliance, invalid. It is only when upstream compliance is added to resistance that factors are produced that can be correlated with pump output. Therefore, "inflow impediment" and "outflow impediment" will be used to designate the interaction of resistances and upstream compliance, which may be a factor in pump output control in specific situations.

SUMMARY OF MODEL FINDINGS

No single factor correlated with pump output at all times. It was only when a factor was limiting pump output that it correlated with flow. Therefore at all times it was the limiting factors that determined pump output. The limiting factors fall into two mutually exclusive sets of determinants that define two states: "Pump Energy Excess" and "Pump Energy Failure."

Pump Energy Excess is the state where flow is being limited by factors other than pump energy. This state was manifested whenever alteration in pump rate or impeller pressure caused no concomitant change in pump output. Pump energy excess was always associated with uninterrupted venous flow to the pumps and submaximal filling of the ventricles at ventricular diastole. The set of determinants of pump output during pump energy excess is: mean vascular pressure and inflow impediment (Fig. 6, Fig. 7-C to D, Fig. 8-B to C, Fig. 9-B to C).

Pump Energy Failure is the state where flow is being limited by pump energy, in either rate or in strength of impeller pressure, in relation to outflow impediment. This state was manifested whenever change in pump rate, impeller pressure or outlet impediment caused a concomitant change in pump output. Pump energy failure was always associated with interruption of venous flow at ventricular systole and maximal ventricular filling before the end of ventricular diastole. The set of determinants during pump energy failure is: pump rate and/or impeller pressure in relation to outflow impediment (Fig. 7-A to B, Fig. 8-A to
CONCLUSIONS FROM THE MODEL

There are two sets of mutually exclusive determinants of pump output, only one of which can be determining flow at a time. It is the set that is limiting the flow that is the determinant at any time. The power or energy supply available for circulation determines which set at any moment is controlling output:

During Pump Energy Excess:

\[
\text{Pump output} = \frac{\text{Mean vascular pressure}}{\text{Inflow impedance}} \times \frac{\text{Fluid volume}}{\text{System compliance}} \times \frac{\text{resistance}}{\text{and upstream compliance}}
\]

During Pump Energy Failure:

\[
\text{Pump output} = \frac{\text{Pump rate and/or Impeller pressure}}{\text{Outflow impedance}} \times \frac{\text{resistance}}{\text{upstream compliance}}
\]

PHYSIOLOGIC COROLLARIES TO MODEL FINDINGS

There are corollaries in human physiology for all findings in the model. Furthermore, all circulatory findings in man seem to have a counterpart in one or the other of the two hydraulic states demonstrated in the model: pump energy excess and pump energy failure.

I. Myocardial Energy Excess: Pump Energy Excess

This is the hydraulic state where mean vascular pressure and inlet impedance determine the output. Conversely, this is the state where changes in heart rate, strength of myocardial contraction and outlet impedance do not change cardiac output.

A. Cardiac Output Changes Correlate with Mean Vascular Pressure Alterations: (Fig. 6). Mean vascular pressure was first described by Weber more than 100 years ago (17); synonyms include static blood pressure (18), intrinsic blood pressure (19) and mean circulatory filling pressure (20).

During the early stages of hemorrhagic shock, low cardiac output accompanies exsanguination whether or not the heart beats rapidly or strongly (9). Similarly, diminished cardiac output accompanies the decreased blood volume that occurs from dehydration (21) as seen in untreated diabetes mellitus, diabetes insipidus or Addison's disease. In contrast, increase of blood volume by transfusion after hemorrhage increases cardiac output in geometric relation to the volume replaced as long as the vascular tone is unaffected and myocardial function is in excess of the workload (6). Cardiac output above normal levels may develop after over-transfusion or from an abnormally high intravascular
volume such as associated with renal failure (22). High cardiac output also may be associated with high output arterial hypertension when a component of the syndrome is an increased intravascular volume (15).

Cardiac output changes, similar to those in the model, occur where mean vascular pressure is altered by changes in vascular compliance rather than in blood volume. Thus, decrease in cardiac output accompanies any stimulus, reflex or otherwise, which relaxes and thereby increases vascular compliance (23). Vasoactive drugs (e.g., arfonad), metabolic acidosis (24), spinal anesthesia (8), vasovagal syncope (16), and decapitation are all conditions that relax the vascular system and are accompanied by low cardiac output in the absence of any change in blood volume or myocardial energy expenditure. In contrast, high cardiac output results from stimuli that increase vascular tone where there is absence of myocardial energy failure. Circulating catecholamines (after exogenous administration of epinephrine or endogenous secretion by pheochromocytoma (22), hypoxia, hypercarbia (24), changes in body position and exercise (22), fatigue, emotional tension and anxiety (25) and peripheral nerve stimulation all increase vascular tone, thereby increasing mean vascular pressure which may account for the concomitant increased cardiac output.

B. Cardiac Output Changes Due to Inlet Impediment Alterations: (Fig. 10, B to C). The interplay between inlet impedance and cardiac output during cardiac energy excess is manifested in vena caval obstruction (e.g., inflow obstruction during cardiac surgery, mechanical compression during cardiac massage, or direct encroachment by tumors or thromboses), Valsalva maneuver, venous spasm after administration of vasoactive drugs, and the effects of increased blood viscosity (26). Other examples of inlet impedance correlating with decrease in output include increased atrial impedance from atrial fibrillation and nodal rhythm (22). Moreover, with increased valvular impedance from mitral or tricuspid stenosis, cardiac output decreases in proportion to the severity of the narrowed inlet (27). During rapid tachycardia (7), marked ventricular hypertrophy and pericardial tamponade (21, 28) where filling time is short, chamber size is contracted or distensibility of the heart is restricted, respectively, the output is low.

The increase in cardiac output from arteriovenous fistulae may be explained by the lower inlet impedance to the heart as the fistula bypasses the resistance points that are distal to most of the compliant beds. Furthermore, exercise increases cardiac output where muscle vein pumps tend to overcome the inlet impediment to the heart.

C. Factors Which Have Not Been Shown to Effect Cardiac Output During Myocardial Excess: Increasing Heart Rate (Fig. 7, C to D) and/or Strength of Myocardial (Fig. 8, B-C) Contraction and Decreasing Outflow Impediment (Fig. 9, B to C). The model findings have a clinical counterpart with pacemakers in the range where changes in heart rate do not increase or decrease cardiac output (3, 4). Correlation is also seen when stimuli that strengthen myocardial contractility such as digitalis (10) or which decrease contractility such as propranolol, do not alter
cardiac output except when ventricular energy failure is present (5). Furthermore, alterations in outflow impedance such as seen in sub-aortic stenosis, aortic stenosis (15, 22), coarctation of the aorta, aortic rigidity from atherosclerosis, and arteriolar resistance in essential hypertension (14) cause no decrease in cardiac output as long as the heart expends enough energy to effectively eject its contents. The fact that cardiac output in this situation does not change, does not imply that the output impedance increase in the absence of failure does not alter the ejection fraction, ejection time, and myocardial metabolism. The very limited secondary elevation in cardiac output that occasionally occurs during myocardial energy excess by elevating the strength of contraction or decreasing outlet impedance may result in a reduction in end-systolic volume which in such cases may decrease inlet impedance and thus secondarily cause a slight increase in cardiac output. The resulting change being primarily from inlet impedance alteration is therefore not an exception to the model findings.

II Myocardial Energy Failure

The point of myocardial energy failure with the heart is less abrupt than in the more rigid model; otherwise, the findings have their counterparts in the model.

A. Low Heart Rate (Fig. 7, A to B). Clinical counterparts to findings during low pump rate energy failure are seen with the use of cardiac pacemakers for complete heart block (3, 4). In patients with heart rates of 20-30 beats/min. the cardiac output is low and ventricular filling is complete before the end of diastole. If a pacemaker rate progressively increases, cardiac output increases until the ventricles are no longer filled to capacity during diastole. Further increases in pacemaker rate are then without increase in cardiac output.

B. Myocardial Energy Failure in Overcoming Outflow Impediment (Fig. 8, A to B and Fig. 9, C to D). Clinical counterparts to the model are the increase in cardiac output generated when digitalis and other inotropic drugs (29) are administered to patients during myocardial energy failure. Similarly, administration of propranolol reduces cardiac output whenever the strength of contraction is decreased to the point of myocardial energy failure.

Low cardiac output from aortic or mitral insufficiency is from myocardial energy failure. Inlet or outlet valvular insufficiency may restrict cardiac output by wasting so much energy on forcing blood backward that there is myocardial energy failure for forward flow.

Modification of outflow impediment causes cardiac output change during myocardial energy failure. This is seen as a further corollary to model findings. Administration of nitroprusside or other arteriolar relaxing drugs (11) or intra-aortic balloon pumping increases cardiac output by lowering outflow impediment to a level that low myocardial energy expenditure is sufficient to cause more adequate emptying of the
ventricles. Similarly, operative correction of aortic stenosis by valvulotomy or valve replacement (30) derives its benefit by decreasing outflow impediment to a point where myocardial energy is in excess of that used on circulation.

Clinical observations thus correlating with those of the model suggest that myocardial function and its multitude of factors, as they relate to outflow impediment, alter cardiac output only during myocardial energy failure. At all other times myocardial factors provide excess power supply and therefore do not regulate or control cardiac output.

C. Factors That Have Not Been Shown to Effect Cardiac Output During Energy Failure: Mean Vascular Pressure and Inlet Impediment (Experiment 4a, #6). A physiologic corollary is seen during ventricular energy failure where transfusion has no beneficial effect on cardiac output and where surgically creating an arteriovenous fistula for dialysis during heart failure is unaccompanied by an increase in cardiac output.

RESULTING CONCEPT

We may now postulate that:

I. The heart is essentially a non-sucking, continuous inflow, pulsatile outflow pump which therefore, depending upon extracardial factors, may be either active or passive in cardiac output determination.

II. The heart is passive in relation to cardiac output control during pump energy excess, where mean vascular pressure and inlet impediment determine output.

III. The heart is active in determining cardiac output only when it is in pump energy failure, where heart rate and/or strength of myocardial contraction in relation to outflow impediment determine output.

IV. There are two physiologic states which have separate sets of determinants, only one of which can be regulating cardiac output at a time. It is, therefore, always the set that potentially gives the lowest output that limits and therefore becomes the sole determinant of cardiac output.

The two states are:

1) Myocardial energy excess (where mean vascular pressure and inlet impediment to flow determine cardiac output).

2) Myocardial energy failure (where heart rate and/or strength of myocardial contraction and outlet impediment to flow are determinants of cardiac output).

DISCUSSION

The above concept eliminates many of the hard to explain paradoxes
of the past and, therefore, points to reevaluation of the many old concepts. In the new concept, Starling's law becomes an expression of energy conservation during energy excess and has output control implications only during energy failure.

In Poiseuille's law, it is the pressure gradient during myocardial excess and not the cardiac output that changes with alterations in "total vascular" resistance.

Vasomotor tone, with all of its reflex control factors, may alter circulation by changes in mean vascular pressure and inlet impediment, and not merely by changing arteriolar resistance.

The new concept identifies such statements as "cardiac output equals stroke volume times heart rate" and "cardiac output equals venous return" as being synonyms of cardiac output rather than being expressions of determinants.

The new insight makes realization that concomitant changes from stimuli are not necessarily responsible for the resulting circulation change. A heart rate change, for example, may not be the factor responsible for a cardiac output alteration resulting from a stimulus.

Pulmonary congestion from high inlet impediment is an example that the clinical designation "congestive heart failure" is not always an expression of myocardial energy failure.

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