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A PROPOSAL FOR THE DYNAMIC ANALYSIS OF CIRCULATORY SHOCK

Kenneth E. Woodward

Timothy G. Barila, Lt.Col. (MC) USA

1 March 1963



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* was proposed.

ABSTRACT

The customary empirical models of circulatory shock largely ignore the dynamic character of the human circulatory systems. ~~It is proposed that a dynamic analysis~~ may give better insight into the system's functional state than the usual pressure, flow or volume assessments. This proposal is based on the hypothesis that myocardial forces are minimal and tightly bounded for large variations in blood flow. ~~Further,~~ myocardial energies are minimal for these same flows. Circulatory shock is suggested to be a state in which the heart is required to operate beyond this minimal force-energy region. In so doing the system may malfunction if correction is not prompt, or the heart may overwork and fail prematurely.

1. THE NEED

Past attempts to describe the human cardiovascular system in both normal and abnormal functional states, such as shock, have resulted primarily in the development of word and/or empirical models. These relatively simple models have often proved to be an adequate basis for clinical treatment, but occasionally such treatment has caused the illness either to persist or intensify. This indicates that existing cardiovascular models are not totally adequate.

In recent years, attempts have been made to develop mathematical and analog models that consider the contributions and responses of individual elements on a system basis. These hold much future promise but unfortunately are not yet available. When they do become available, they will undoubtedly be considerably more complex than existing ones and may require new techniques, apparatus, and personnel with strong mathematical and engineering backgrounds in addition to prerequisite medical competence.

A major difference between promised and existing models is that the latter largely ignore the dynamic character of the system. The usual time measurements of blood pressure, flow, volume and oxygen debt, when influenced by biological, emotional, chemical, physical and other factors, fail to recognize the individual contributions of heart rhythms, the dynamic responses of the circulations, the character and kinds of loads imposed on the heart and control phenomena. Future models will undoubtedly consider some or all of these.

It would seem, then, that a need exists for models that consider the dynamic character of the system and that can be implemented by present techniques and personnel. It is the purpose of this report to suggest a simple approach for the dynamic analysis of the cardiovascular system with particular emphasis on circulatory shock. The proposal is based on the yet unproved observation of reference 1 that myocardial forces expended by the heart may be minimal and bounded, and the energies also minimal for the pressures and flows generated.

2. DEFINITION OF SHOCK

It is not intended that this report be a treatise on shock, but it seems necessary to consider definitions briefly. Unfortunately the nature of shock is not so well understood that a single viewpoint exists. There are many, of which two are presented. Guyton (ref 2) offers the following definition for shock. "Circulatory shock is an abnormal condition in which the cardiac output is reduced so much that the tissues of the body begin to deteriorate for lack of nutrition. Even the cardiovascular system itself -- the heart musculature, the walls of the blood vessels, the vasomotor center, and other parts -- begins to weaken so that the shock itself often becomes progressively worse and worse." ... "Some physiologists consider a person to be in shock any time the cardiac output falls even slightly below normal while others consider a person to be in shock only when the cardiac output falls so low that the circulatory system itself begins to deteriorate because of the low cardiac output."

Shock, according to Guyton, can be divided into two major stages, progressive and nonprogressive. In nonprogressive shock, cardiac output is deficient, "but not deficient enough to cause a vicious cycle of cardiovascular deterioration." Alternatively progressive shock is that which exceeds the point at which the circulatory system starts to deteriorate. The latter type results in death unless treatment can cause reversal.

3. CHARACTERISTICS OF SHOCK

From a pathological viewpoint, a person in shock may have decreased metabolism, muscular weakness, lowered body temperatures, impaired mental functions, and reduced renal function (ref 2, p 481). However, the comments of Hardaway (ref 3) appear to be more pertinent to the physical circulatory factors of immediate concern. He says that nonprogressive or reversible shock "has been attributed to a disparity in volume between the blood and the vascular bed. This may be secondary to blood or plasma loss due to hemorrhage, etc, or to expansion of the vascular bed. In fact, this has frequently been offered as a definition of shock. However, it is possible to have shock in the presence of a normal or increased blood volume and marked vasoconstriction. It is possible to have shock with a normal or increased blood pressure. It is possible to have shock with either a cold, clammy skin or a warm, pink skin."

The characteristics to be emphasized, because of their dynamic effect on the system, are marked changes in vasomotor tone, blood or fluid volume, and the capacity of the vascular bed. These may result in altered blood pressures. While all are apparently related, they are also related to many other parameters largely ignored, in a definitive sense, by existing models. The system changes usually appear to be secondary effects rather than primary causes with the exception of lost fluid volume.

4. NATURE OF SHOCK

The lack of a precise definition for shock may of itself create problems in attempting to understand the nature of shock and its treatment. However, within the broad definition for shock, it appears that at least two schools of thought exist that attempt to explain its basic nature. They are complementary rather than separate and distinct.

Hardaway (ref 3) is representative of one school. He says "the common denominator in shock is not a decreased blood volume, or increased vascular bed, low blood pressure, vasodilatation, vasoconstriction, acidosis, a toxin, or failure of the heart, but a decreased capillary perfusion, and that this is frequently brought about by clotting of the blood in capillaries and other small vessels aided by associated vascular spasm.

"Reversible shock results from a decreased cardiac output due to a decreased venous return. In hypovolemic shock this may be due to insufficient blood to fill the vascular system. However, in normovolemic shock, such as that due to endotoxin, incompatible blood, or thrombi, it is due to mechanical blockage of the flow of blood in various capillaries due to thrombi and associated vasospasm.

"This occurs in the liver causing trapping of blood in the portal system. Thrombi also prevent blood from flowing adequately through other organs. Peripheral vasospasm contributes to the blocking of flow. Thus blood does not reach the vena cava in adequate amounts causing a low vena caval pressure and inadequate return of blood to the right atrium. Capillary thrombi in the lungs also block the flow of blood causing a pulmonary artery hypertension and an inadequate venous return to the left heart. All of these changes are reversible and blood pressure returns to normal when blood volume is restored or when the thrombi are washed out with the activation of endogenous heparin and fibrinolysin.

"However, if these capillary thrombi remain in place long enough, they may cause local tissue necrosis in the area which the capillary nourishes. Then when the clots are washed away and the blood pressure returns to normal levels, the tissue necrosis remains. If this is sufficient, especially in the liver and kidneys, death of the animal results." Hardaway in reference 4 observes that "blood-clotting abnormalities seem to be a constant finding in all types of shock."

According to experimental evidence by Crowell as reported by Guyton (ref 2), the blood flow in shock becomes extremely sluggish, "but tissue metabolism continues so that large amounts of acid, either carbonic acid or lactic acid, continue to empty into the blood. Only a few minutes of very sluggish flow in some tissues can lower the capillary pH to the agglutinating level, and minute vascular plugs develop in the small vessels." This supports Hardaway's views.

The other school of thought attributes the ultimate demise of the subject in shock to failure of the heart muscle. Guyton, who holds this view, says "it should especially be remembered that in the final stages of shock the myocardium always eventually fails. Therefore, all the other causes of progression in shock eventually lead to myocardial failure itself before death occurs." Guyton, in contrast to Hardaway, suggests that capillary thrombi "can be one of the causes of progression of shock" but apparently not the final or ultimate cause of death.

Guyton (ref 2, p. 482) summarizes his understanding of shock as follows: "Shock results from inadequate cardiac output," consequently any factor reducing cardiac output can also cause shock. He groups these into two categories; (1) those that decrease the ability of the heart to pump blood and (2) those that tend to decrease the venous return." The first category includes those factors contributing to heart damage that prevent the heart from pumping adequate quantities of blood. In the second, factors reducing venous return "including (a) diminished blood volume, (b) decreased vasomotor tone, or (c) greatly increased resistance to blood flow, can also result in shock."

5. CAUSES FOR PROGRESSION OF SHOCK

In those instances where shock cannot or does not reverse, the following causes according to Guyton contribute to increasing the depth of shock.

(1) Myocardial failure - the diminished output from the heart causes a diminished blood supply to the heart which further reduces the heart's output, etc.

(2) Vasomotor failure - the lack of adequate nutrition depresses the vasomotor center resulting in less and less activity and finally total inactivity. The loss of control by the sympathetic nervous system "can cause such great dilatation of the venous reservoirs that blood will fail to return to the heart adequately; this results in further progression of the shock."

(3) Vascular failure - the dilatation of portions of the vascular system such as the arterioles and veins apart from vascular dilatation caused by vasomotor failure. The dilatation of the arterioles can reduce arterial pressure, reducing blood flow to the heart and brain, and hence intensify shock. Venous dilatation can cause pooling in the veins, depressing cardiac output.

(4) Thrombosis of the minute vessels - the development of thrombi in the minute vessels of the circulatory system caused by sluggish blood flow and the action of metabolic acids (carbonic or lactic). Red cell agglutination results and if not eliminated in time, local necrosis of vital organs takes place.

(5) Failure of tissue use of oxygen - the decreased ability of the body tissues to use oxygen following lengthy periods of shock.

6. CLINICAL TREATMENT

In treating shock, the intent is apparently to increase cardiac output. This first involves correcting the initial cause of shock (if known and necessary). If after such correction is accomplished shock persists, then other steps can be taken to elevate cardiac output. Apparently the two most common approaches involve restoration of blood or fluid volume and the use of sympathomimetic drugs to restore controlled vasomotor tone. Reference 5 discusses vasomotor tone in detail.

In the case of cardiac shock where the heart is failing, drugs (such as digitalis) are administered to strengthen the heart in addition to vasoconstrictors such as nor-epinephrine to sustain arterial pressures.

7. INADEQUACIES OF CLINICAL MODELS

It appears that circulatory shock emphasizes the need for system models that consider the dynamic character of the cardiovascular system usually ignored in present models. The loads imposed on the heart by the circulatory system possess qualities of resistance, capacitance, and inertia which might possibly establish a required dynamic response to rhythms of the heart. Descriptions of shock suggest these are changing during shock. The concept of peripheral resistance, for example, illustrates the need for more refined models. Peripheral resistance is obtained by dividing blood flow into blood pressure (analogous to Ohm's law). This quantity is widely used to describe system as well as organ functions. What the quantity actually represents may be questionable since it does not identify the inertance or capacitance that is known to exist. The derived loads in analog simulations of the human system are much more complex.

The time measurements of blood pressure, flow, and volume produce a panoramic picture of the functioning (or malfunctioning) circulatory system but fail to relate quantitatively vasomotor tone, blood pressure, volume, flow, and other pertinent variables. The importance of these measurements seems to relate generally to their relative magnitudes and trends. The significance of the shape of a single pulse pressure wave, for example, is presently not known. In the diagnosis of systems in shock the usual approach is to measure the deviations of pressure, etc, from "normal" and institute treatment to restore these quantities to normal values. The treatment frequently involves correction of properties affecting the dynamic performance of the system (example vasomotor tone) but the adequacy of the treatment in restoring system normalcy is again measured in steady state quantities.

To the design engineer who is concerned particularly with periodic exciting forces whose frequencies lie in close proximity to those of the

object subjected to such forces, the dynamic response of the object is of considerable importance. Small changes in either the frequencies of the exciting forces or the natural frequencies of the object can cause significant changes in system response. The authors believe the human system may be a system where such dynamic changes can be very significant.

8. THE RESONANT ARTERIAL SYSTEM

Reference 1 suggests that very important reasons exist for a beating heart whose pulse rate lies close to but lower than the natural frequencies of the elastic arterial system. The importance of this can be more easily appreciated when the dynamic principles governing the spring-actuated pendulum clock are examined. The clock operates because the potential energy in a wound spring is caused to act at precisely the right moment on the swinging pendulum. The pendulum is then forced to swing a necessary distance before a subsequent impulse can act on the pendulum. Yet the clock can operate for days or even weeks on relatively small energy investments because minute amounts of energy and correspondingly small forces are operating for each swing of the pendulum. The clock operates then because the force is caused to act in tune with the natural frequency of the pendulum. Any tampering with the natural frequency of the pendulum or the periodicity of the force can, if great enough, stop the clock. It is suggested that the human arterial system may employ these same resonant principles on a system basis to achieve its functional character (ref 1).

The usual simplified graphical representation for a simple mass-spring system is shown in figure 1. The mass is caused to vibrate by periodic exciting forces at the base. This system can be used to examine the laws governing the clock and simple resonant systems in general. If the mass is required to vibrate at a known fixed excursion as the frequency of the exciting force is varied from zero to some value three times higher than the undamped natural frequency of the spring-mounted mass, the maximum exciting forces required are as shown in figure 2. The curve denoted by $\gamma = 0$ signifies zero damping while the $\gamma = 1$ curve represents critical damping.

The law describing the unique characteristic of pendulum clocks is approximated by the curve $\gamma = 0$. It says that where damping is small, a pendulum that is pushed by a periodic force whose frequency is equal to the natural frequency of the pendulum, can be caused to swing by a very small force. Below this condition ($\omega/\omega_r = 1$) the force curve approaches one as a limit; above it the curve increases without bound. The human cardiovascular system may find these characteristics to be of importance.

Figure 3 describes energy losses due to friction for the same system operating under the fixed displacement criteria. Both increasing amounts of damping and higher forcing frequencies cause higher and higher energy

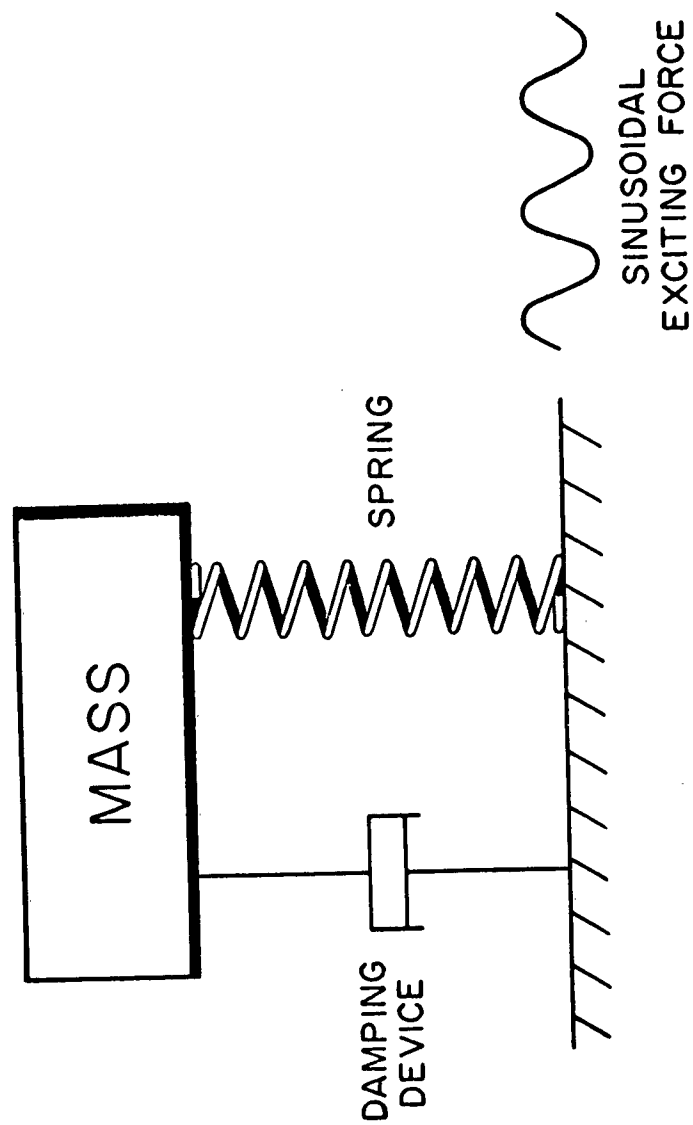


Figure 1. Damped mass—spring system.

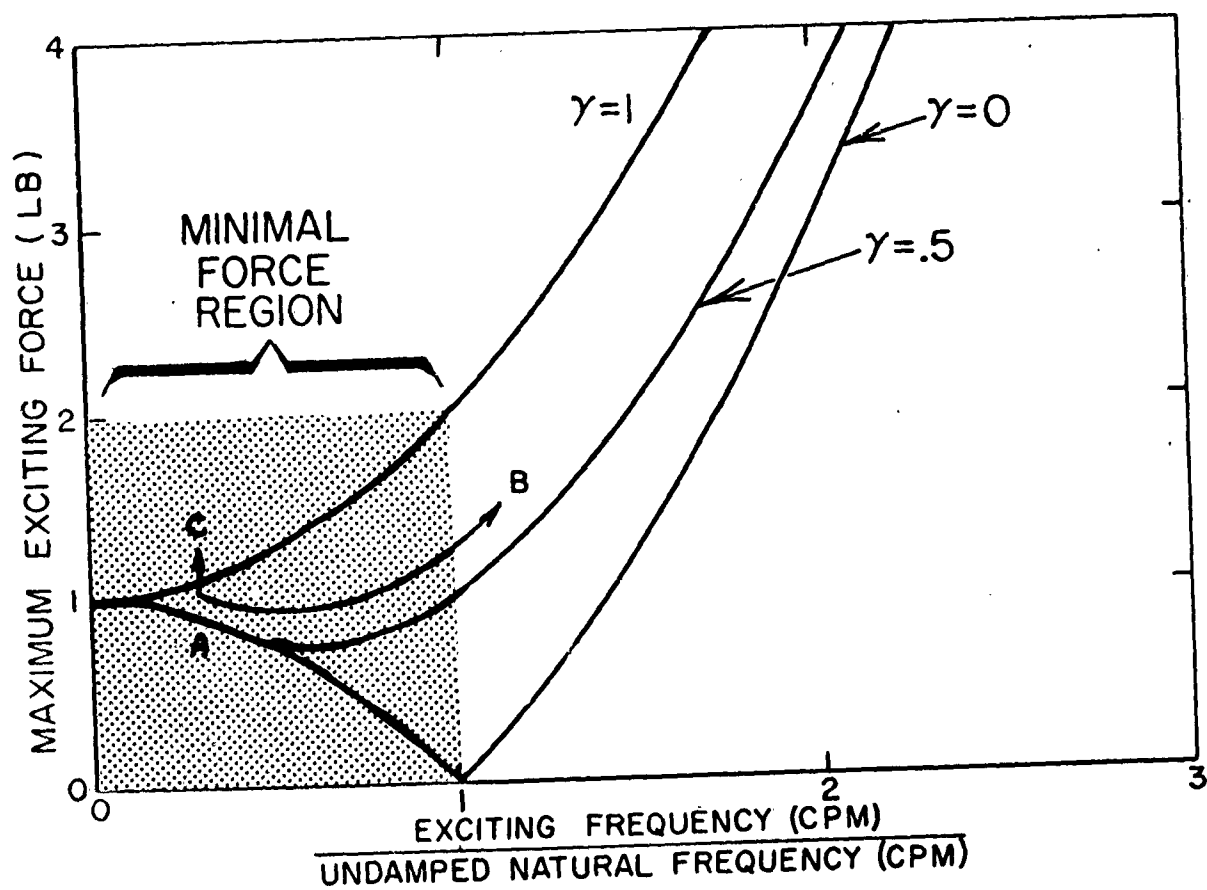


Figure 2. Force characteristics in a resonant system (for a constant mass excursion).

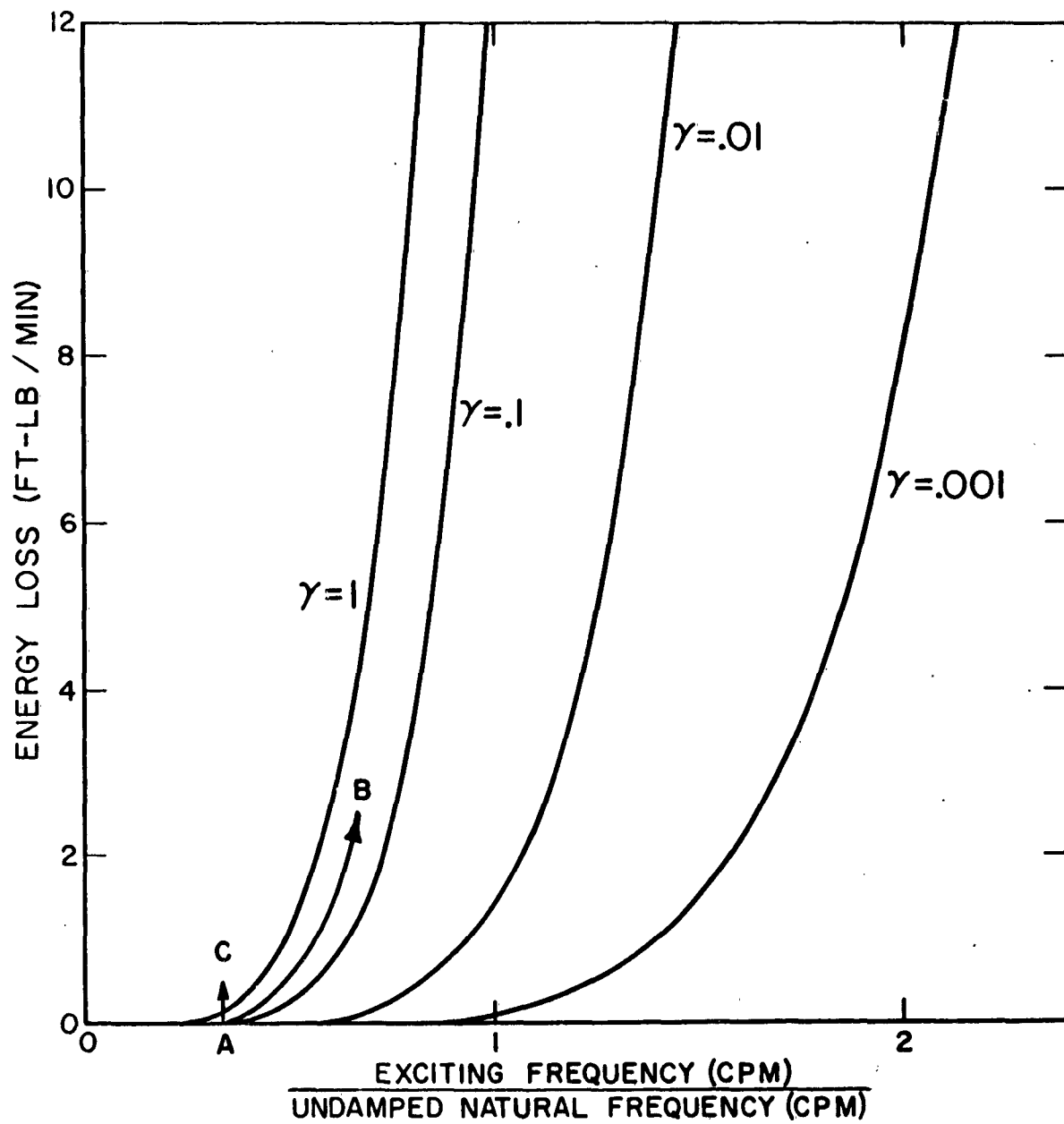


Figure 3. Energy loss in a resonant system (for a constant mass excursion).

losses in achieving the same fixed displacement. Again this should be recognized as a possible design consideration in the human system.

In the human system, the heart approximates a constant-stroke, variable-frequency device forcing its periodic efflux into an elastic circulatory system. In the hope that an analogy existed between the device of figure 2 and the human system, two similar plots for the human arterial system were developed. They are figures 4 and 5 (from ref 1). Reference 1 establishes a more elaborate background for these but the essential point is that the frequency ratios bounding the operation of the cardiovascular system correspond to the region in simple resonant systems where maximum exciting forces are minimal and bounded and energies are minimal. This implies that the human system in which the periodic muscular exciting forces of the heart rhythmically forcing blood into a responding elastic arterial system is doing so with minimal bounded force and minimal energy. Any significant disturbance to the periodicity of the force or the natural responsiveness of the arterial system can force the myocardial forces and energies to much higher values. If this condition persists without correction and if it is sufficiently great, the heart may overwork and die prematurely or other system malfunctions may occur. Conversely, if under these changed conditions the heart cannot exert sufficient force and energy even for short periods, inadequate flow will result with its consequences.

Emphasis, therefore, is directed toward appreciation of the importance of a correct dynamic relationship between the rhythmic force and the responding elastic system when minimal force and energy expenditures are to be realized in a resonant system. Further it should be recognized that in a system such as the human cardiovascular system, which might benefit from resonant phenomena, blood pressures, and flow volumes cannot necessarily measure in a total sense the rightness of system performance. Finally, appreciation for the sensitivity of this proper dynamic relationship to relatively minor changes in the dynamic character of the system is needed. If the upper bounding frequency ratio of figure 5 is much higher than one, small changes to either the frequency of the pulsed force or the natural frequency of the system will be relatively unimportant since the percentage of cardiac work will change very little. But because the system may be operating near resonance, the same small changes to either have a much more pronounced effect on cardiac work.

9. SHOCK EFFECTS ON RESONANCE

Presuming that the design of the human circulatory system is predicated on minimal force-energy relationships, it is possible to discuss the system in shock on an intuitive basis. Predominant alteration of the circulatory system in shock states seem to involve changes in:

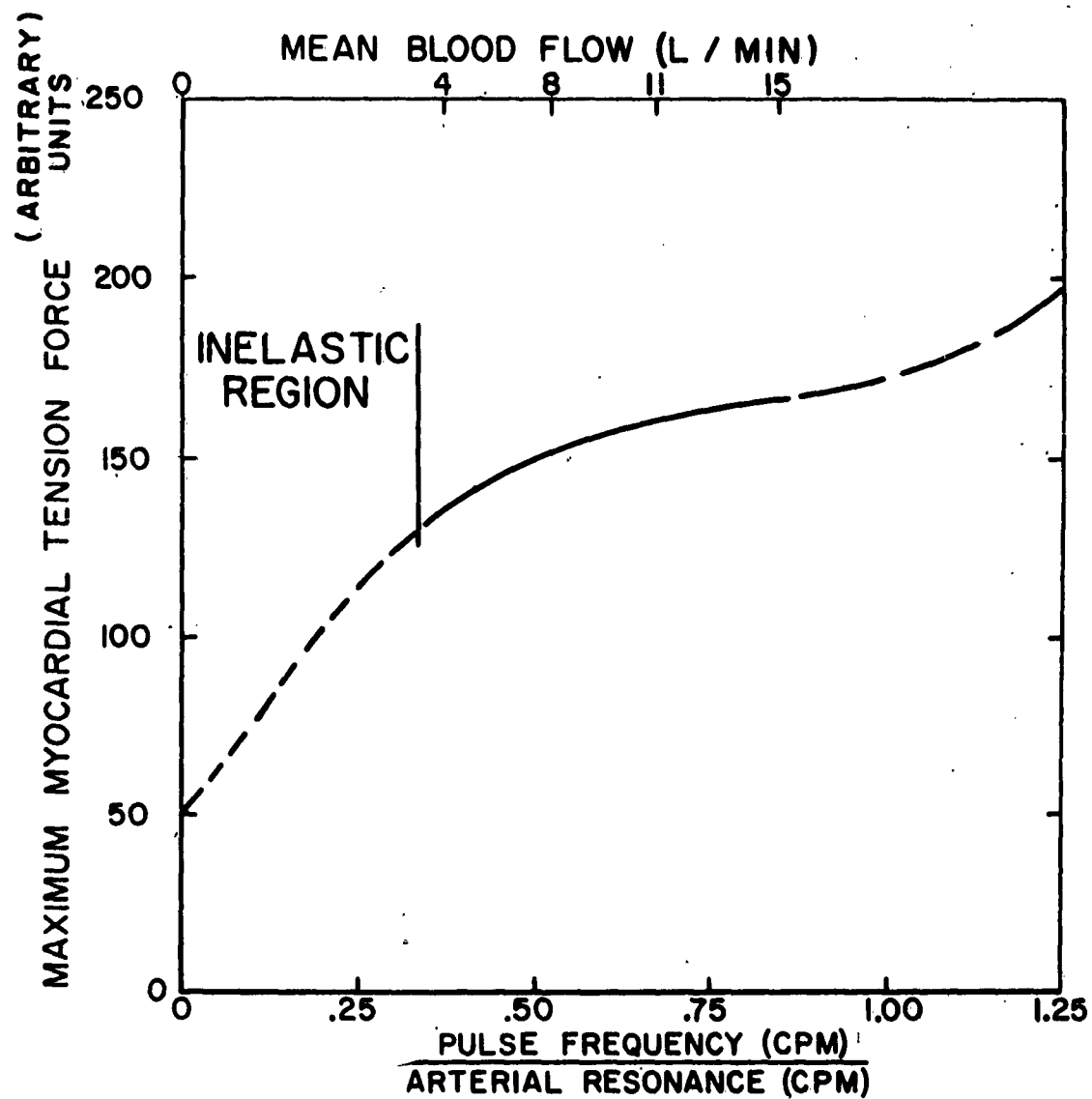


Figure 4. Myocardial force characteristics (left heart).

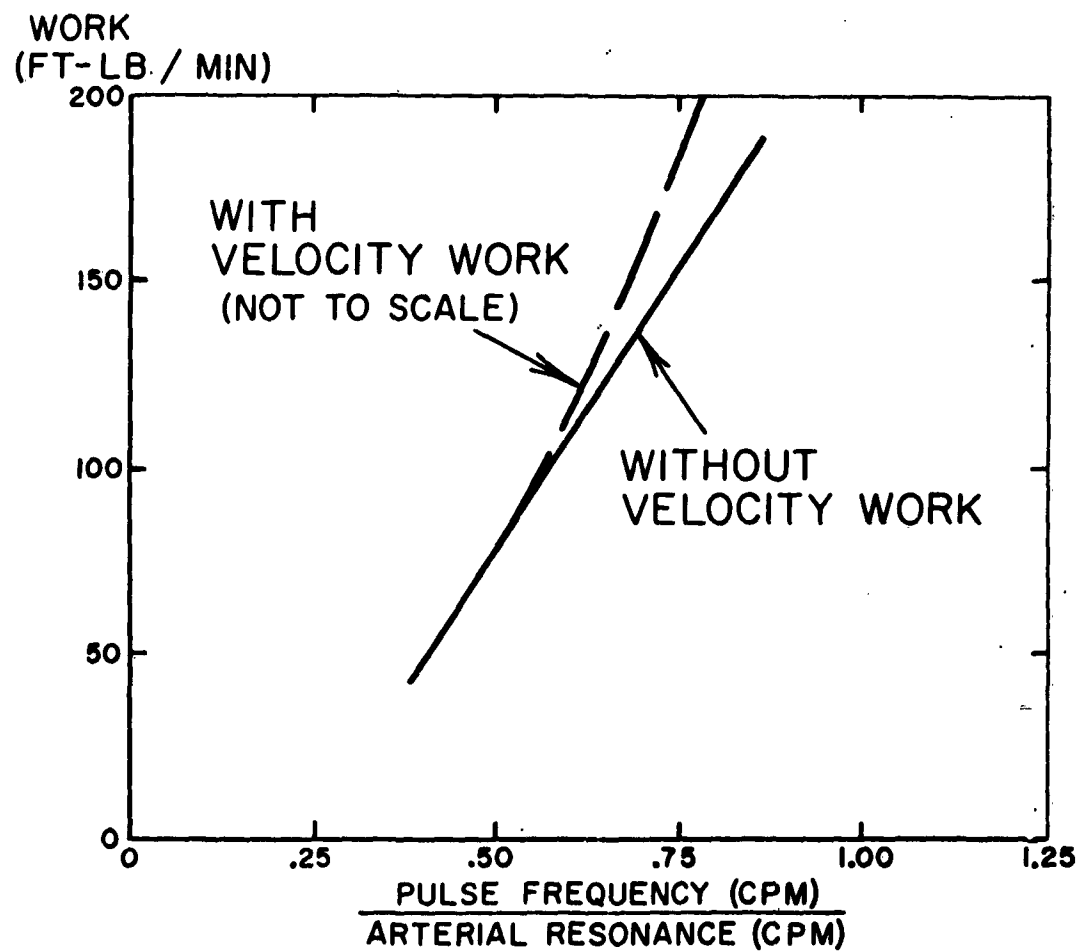


Figure 5. Cardiac work.

- (1) Vasomotor tone
- (2) Fluid volume
- (3) Myocardial force
- (4) Vascular bed capacity

Manifestations of these are changes in flow rates and blood pressures. Other physical manifestations of shock such as altered body temperatures, flushing, etc, are not considered because of their probable less pronounced effect on the minimal bounded force premise.

The axisymmetrical natural frequencies of the arterial system are directly influenced by vasomotor tone, fluid volumes, and the capacity of the vascular bed. Changes to any of these can cause changes to the arterial natural frequencies.

If the minimal force-energy premise holds true and if it is described by curves as simple as those in figures 2 and 3, then the curves C-A-B suggest the probable extremes of the consequences. Two cases are suggested:

(1) In the situation where pulse rate remains constant but arterial resonance decreases (due to decreased vasomotor tone, fluid volume or an increased vascular bed capacity), the frequency ratio describing the system increases (curve A-B). As a consequence myocardial force and energy must increase. If this increase is sustained and great enough, heart failure can occur. The chief reason for this increase would be a shift in phase between the periodic driving force and the driven fluid efflux.

(2) For the case where pulse rate changes are enough to maintain a constant frequency ratio in the face of altered arterial resonances, myocardial forces may increase along line AC if necessary. Again heart failure might occur if the situation was severe and persisted. However, it would seem that a situation of this sort is less dangerous than that described in case (1) since the increases in myocardial force and energy are more limited.

Possibilities for functional situations lying between AB and AC seem likely. Pumping situations to the far left of AC seem less likely because of control center goals of adequate tissue perfusion.

From a dynamic viewpoint it is easy to visualize heart failure in shock if the basic argument holds that minimal force-energies are the normal functional state. Changes in the dynamic status of heart rhythms or circulatory responses to cause the system to operate outside this normal state may cause intolerably large changes in inertial and possibly damping forces.

In treating shock patients, it might be surmized that restoration of a minimal force-energy state is the goal desired -- not a normal blood

pressure for example, although normal blood pressures and volumes are a manifestation and requirement of the state. Again on an intuitive basis, it seems that clinical treatment generally strives for this but it is not recognized as such.

10. THE PROPOSAL

In considering these few rather simple but possibly different facets of what may happen to human circulatory systems in shock, it should be made clear that the dynamic reactions or changes are not necessarily a first cause but rather a result of different causative factors. Further the fact that the proper dynamic relationship between the heart and arterial system can be restored does not imply that all is well. It would be presumed that the cause should first be removed and in sufficient time to preclude sustained and intolerable change. But it is suggested that a dynamic analysis of the system may give better insight into the functional state of the system than the usual pressure, flow or volume assessments. It is therefore proposed that:

(1) Consideration be given to the dynamic relationships between the beating heart and the responding arterial system in both normal and shock states to determine if a significant or measurable difference exists.

(2) A first step be the measurement of a frequency ratio in which the numerator is the pulsing frequency of the heart and the denominator is the natural frequency of the arterial system measured at the root of the aorta and at points distal to the root. Cardiac work performed during normal and shock states might be compared and related to this frequency ratio. Myocardial force might be similarly compared. This report does not attempt to define natural frequency nor suggest techniques for measurement. Neither is it suggested that the natural frequency of the arterial system can be simply defined.

(3) Treatment techniques be tried which attempt to restore the frequency ratio of (2) to normal values presuming a significant difference is found.

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Circulatory system	Circulatory system	Circulatory system
Circulatory shock	Circulatory shock	Circulatory shock

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