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NEW VERSUS OLD THEORIES OF BLOOD FLOW DURING CPR

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Introduction

Today, the physiology of blood flow during external chest compression appears more complicated than had previously been imagined. New evidence requires that at least two possible mechanisms be considered for generating artificial circulation by external chest compression. The first is the classical mechanism in which the heart propels blood because it is squeezed between the sternum and the spine. The second is a more recently appreciated mechanism in which the entire thorax acts as a pump because of global intrathoracic pressure changes. The first might be called the cardiac pump mechanism and the second might be called the thoracic pump mechanism. In this article, the author will endeavor to describe these two mechanisms, identify their similarities and differences, and discuss their significance in the search for improved CPR techniques.

The cardiac pump mechanism

Most physicians learned this mechanism as basic rescuers. Central chest compression during artificial systole squeezes the heart between the sternum and the spine, causing ejection of blood (Figure 1). Presumably, cardiac compression generates higher pressure in the ventricles than elsewhere in the thorax. The mitral and tricuspid valves close and the semilunar valves open. Blood is ejected into the great arteries, and a positive arteriovenous pressure difference is generated in the thorax. In artificial diastole intracardiac pressure falls, the mitral and tricuspid valves open, and the heart fills from the systemic venous reservoir. The airways vent the thorax somewhat during chest compression, and a small inflow of air occurs when compression is released. The airflow generated, however, is insufficient for adequate gas exchange, and positive pressure ventilation must be applied. Classically, ventilation is applied between chest compressions, in order not to hinder the compression of the heart.

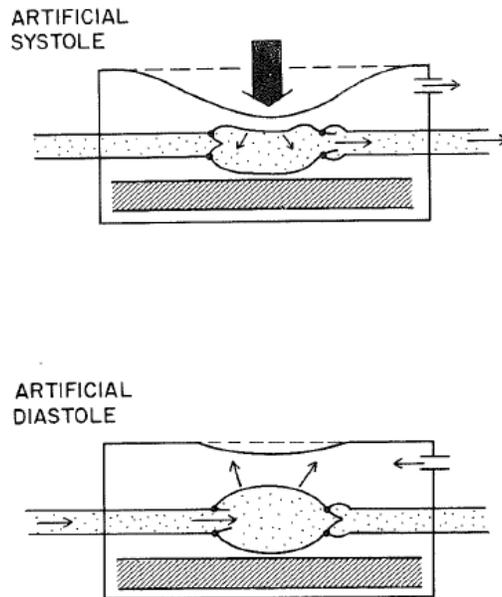


Figure 1. Cardiac pump mechanism for generating blood flow during CPR. During artificial systole, central chest compression squeezes the heart against the spine, forcing blood out. Air is vented from the thorax via the trachea (top right). During artificial diastole chest resiliency creates negative pressure for filling. The site of application of force (broad arrow) is critical. For simplicity only one pumping chamber is shown.

Flow limiting factors for this mechanism include those that limit filling and those that limit emptying of the cardiac pump. Filling of the pump during artificial diastole depends upon the pressure difference between the systemic veins and the right ventricle. Increasing this pressure gradient for cardiac filling during CPR provides a rationale for such measures as the application of negative airway pressure during artificial diastole, fluid loading to increase venous pressure, and physical attempts to decrease systemic venous capacitance, such as abdominal binding or leg elevation. These latter measures increase pressure in the great veins entering the thorax by virtue of the fundamental relationship for elastic structures: $\text{pressure} = \text{volume}/\text{capacitance}$.

Emptying of the cardiac pump depends upon generating a left ventricular pressure somewhat higher than peripheral arterial pressure. Increasing this pressure difference may be accomplished in principle by increasing sternal displacement relative to the ventral-dorsal chest diameter, preventing displacement of the heart to the side perhaps by simultaneous compression and ventilation, decreasing outflow resistance with selective arteriolar dilating agents. (Such drugs must not dilate venules, however, because this action would increase venous capacitance and lower right ventricular filling pressures.)

The thoracic pump mechanism

For this mechanism, the displacement of the sternum and direct cardiac compression are irrelevant; instead, the development of intrathoracic pressure is critical. This mechanism, first suggested by Criley et al.[1] and elaborated by Niemann et al.[2], and is probably operative in cough-CPR. The thoracic pump mechanism also provides the rationale for "new CPR" as described by Chandra et al. [3]. Figure 2 presents the essential features of the thoracic pump mechanism. All blood-containing structures within the thorax are considered to be elastic tubes or chambers which are susceptible to collapse by external pressure. The great systemic veins which carry blood to the thoracic pump are easily collapsed by a small transmural pressure difference; whereas the aorta and its major branches tend to resist collapse. The compression chamber of the thoracic pump includes the right ventricle, the pulmonary vasculature, the left atrium, and the left ventricle, all connected in series. The aortic valve and probably the tricuspid valve are critical to the operation of the thoracic pump, but other cardiac valves are irrelevant or redundant.

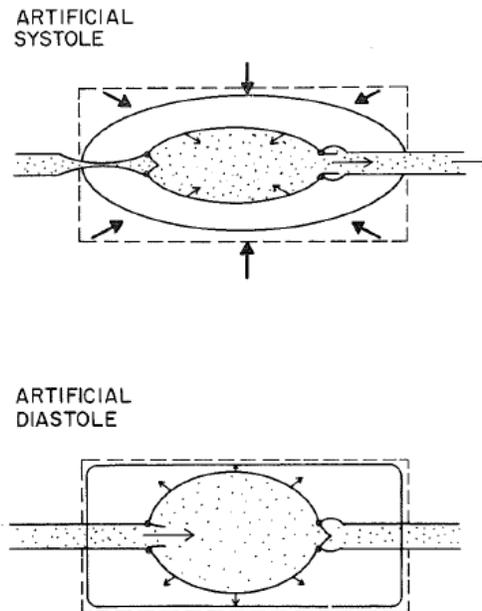


Figure 2. The thoracic pump mechanism for generation of blood flow during CPR. During artificial systole thoracic compression or cough generates intrathoracic pressure, which is vented by arterial outflow to peripheral tissues. During artificial diastole release of intrathoracic pressure allows for filling. The site of application of force is not important.

Operation of the thoracic pump depends on the intermittent generation of global intrathoracic pressure that is greater than prevailing extrathoracic arterial or venous pressures. Such pressure can be developed by thoracic compression in any direction (Figure 2, arrows) as well as by positive pressure ventilation or cough. As intrathoracic pressure rises, both forward and backward flow are possible, until the tricuspid valve closes. The action of the tricuspid valve is complemented by the action of peripheral venous valves. Forward flow occurs through the pulmonary vasculature and the left heart, which acts as a passive conduit. Generation of sufficient global intrathoracic pressures to move blood in this manner requires that venting of thoracic pressure via the airways be minimal, as is certainly the case in cough-CPR and in new CPR [3] with simultaneous ventilation and compression. Forward flow of blood occurs into peripheral arteries during artificial systole for the same reason that forward flow of air occurs during forced expiration [4, 5]. Chest compression or cough raises intrathoracic pressure to equal levels in the heart, lungs, and pulmonary vessels. Blood in the thoracic pump is vented by arteries leaving the thorax, which are relatively resistant to collapse and lead to areas of lower extrathoracic pressure. A pressure gradient is, therefore, established along the arterial channels exiting the thorax (Figure 3).

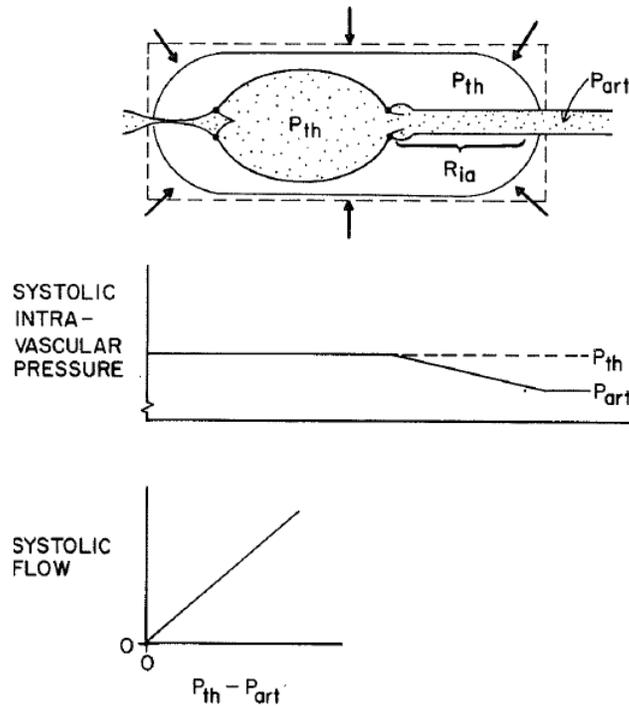


Figure 3. Systolic pressure-flow relationships for the thoracic pump mechanism in its low pressure state of operation, in which flow is not limited by arterial collapse. P_{th} = global intrathoracic pressure, P_{art} = peripheral (extrathoracic) arterial pressure, R_{ia} = intrathoracic arterial resistance. Venting of blood from the thorax into peripheral arteries causes a pressure gradient to develop along the intrathoracic arterial segments, but the pressure difference across the arterial wall is everywhere inadequate to cause arterial collapse. Systolic flow is given by the expression $(P_{th} - P_{art})/R_{ia}$.

The pressure difference between the aortic arch and vessels outside the thorax causes blood flow to the periphery. At the points where the arteries exit from the thorax, the intra-arterial pressure falls to the level of peripheral arterial pressure. Hence, at these exit points, the pressure difference across the arterial walls, tending to cause their collapse, equals the difference between the global intrathoracic pressure and peripheral arterial pressure.

The state of operation of the thoracic pump depends upon the level of intrathoracic pressure. As long as this transmural gradient at thoracic exit points is less than their critical closing pressure, the arteries will remain open. Further increases in intrathoracic pressure will generate increased flow. The value of the thoracic outflow will be given by the difference between intrathoracic pressure and peripheral arterial pressure, divided by the resistance of the arterial segments within the thorax (Figure 3, bottom).

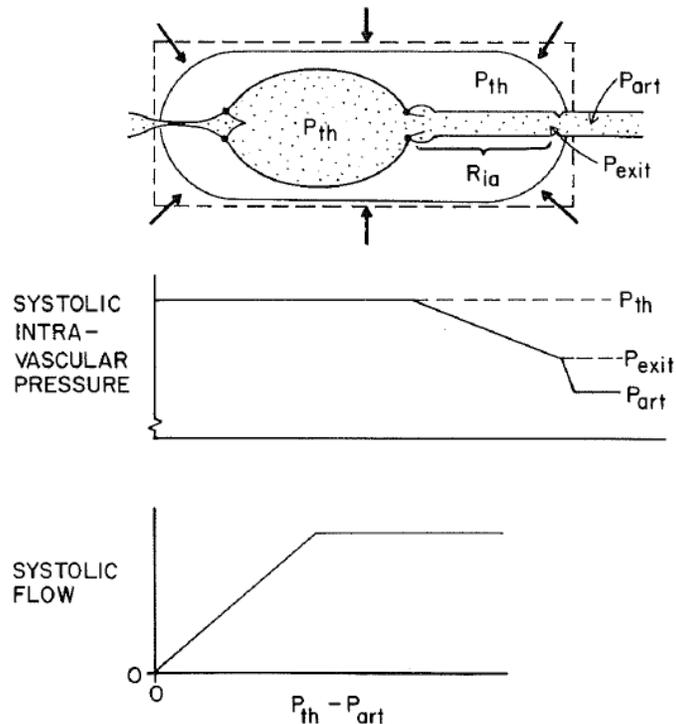


Figure 4. Systolic pressure-flow relationships for the thoracic pump mechanism in its high pressure state of operation, in which flow is limited by partial arterial collapse. P_{th} = global intrathoracic pressure, P_{art} = peripheral (extrathoracic) arterial pressure, P_{exit} = intraarterial pressure at the sites where arteries leave the thorax, R_{ia} = intrathoracic arterial resistance. The potential pressure difference across the arterial wall at the point of exit from the thorax ($P_{th} - P_{exit}$) is greater than the arterial collapse pressure, P_c . P_{exit} is maintained at the level $P_{th} - P_c$, and flow is given by the expression $(P_{th} - (P_{th} - P_c)) / R_{ia} = P_c / R_{ia}$, a constant value.

If more forceful chest compression is applied, the pressure difference across the arterial walls may rise to the critical closing pressure of the arteries, and transition to a second hemodynamic state will occur. In this state, blood flow reaches a maximal level which is independent of the difference between intrathoracic and peripheral arterial pressures (Figure 4). One can appreciate intuitively the reason for flow limitation as follows. If the transmural pressure exceeds the critical level, an artery will collapse. Collapse will stop venting of blood from the artery, allowing intra-arterial pressure to rise, which, in turn, will reverse the collapse. Therefore, intravascular pressure at the arterial exits from the thorax will remain at a value equal to the intrathoracic pressure minus the arterial closing pressure. This constant pressure difference limits thoracic outflow (Figure 4, legend).

In the thoracic pumping mechanism, the positive intrathoracic pressure generated by external chest compression or cough is analogous to the positive intrathoracic pressure generated by forced expiration. The critical closing pressure of arteries is analogous to the critical closing pressure of airways; and the intrathoracic arterial resistance is analogous to airway resistance. The resultant pressure-flow curve, for rate of blood flow during artificial systole, shown in Figure 4 (bottom), is similar to the pressure-flow curve for airflow during forced expiration.

During artificial diastole, intrathoracic pressure is relieved, the tricuspid valve opens, and blood flows into the thoracic pump. The aortic valve closes, so that filling of the pump is accomplished only from the venous capacitance.

Figure 5 illustrates the pattern of pressures and flows in time that one would expect from the operation of the thoracic pump mechanism. Flow is proportional to the difference between thoracic pressure and peripheral arterial pressure, as long as this difference is less than the critical closing pressure of the arteries exiting the thorax. Otherwise, flow is constant at a maximal level. The predicted flow versus time tracings are similar in shape to those presented by Weisfeldt for carotid blood flow during CPR in large dogs, in whom the heart was probably not directly compressed [3].

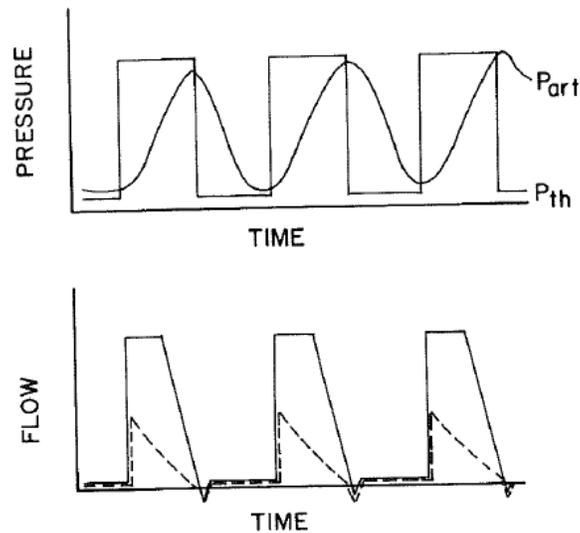


Figure 5. Instantaneous pressure-flow relationships for the thoracic pump mechanism. Hypothetical patterns for intrathoracic pressure, P_{th} , and peripheral arterial pressure, P_{art} , are given in the top tracing as functions of time. Applying the transfer function at the bottom of Figure 4, to the instantaneous values of $P_{th} - P_{art}$ gives the patterns of instantaneous flow from the thorax into peripheral arteries shown in the bottom tracing. The solid flow versus time curve indicates initial flow limitation with each systole due to high intrathoracic pressure. The dashed flow versus time curve indicates no flow limitation at lower intrathoracic pressures. The negative flow notches represent aortic valve closure, followed by zero thoracic outflow during diastole.

The output of the thoracic pump mechanism is limited by factors that limit filling and by factors that limit emptying. Filling of the thoracic and cardiac pumps in CPR is quite similar, except that the input capacitance of the thoracic pump is greater than the input capacitance of the cardiac pump. For both mechanisms the initial rate of venous inflow is determined by the diastolic difference between venous pressure and thoracic pressure and by the resistance to flow offered by the great veins. Hence, the measures previously listed as effective in promoting venous return to the cardiac pump should be equally effective in filling the thoracic pump.

Potential improvements in CPR technique

Theoretically, it is possible to generate blood flow during CPR by two distinct mechanisms. Which is the predominant mechanism in a given victim probably depends upon several factors, including the ventral-dorsal chest diameter, the presence or absence of cardiac enlargement, the stiffness or resiliency of the anterior chest, and whether or not ventilations are applied simultaneously with chest compression. For example, in small dogs weighing less than 10 kg,

positive systolic arteriovenous pressure differences in the thorax during CPR, indicative of direct cardiac compression are usually recorded. The ventral-dorsal chest geometry of these animals models that of children and thinner-chested human adults. In larger dogs, weighing more than 20 kg, the systolic arterio-venous pressure difference is usually negative or zero, and blood flow is probably due to thoracic pumping. The ventral-dorsal chest geometry of these large dogs models that of more barrel-chested human victims. Quite probably, both mechanisms are operative in human CPR to varying proportions. During standard CPR, the dominant mechanism may even vary at different times during the compression cycle; if, for example, effective cardiac compression occurs only at or near the point of maximal chest depression. Preliminary data reported by Weisfeldt indicate some degree of direct cardiac compression in one-fourth to one-third of adult human patients. Hence, it seems best to seek improvements in CPR applicable to both cardiac pumping and thoracic pumping.

Fortunately, from the practical standpoint of basic life support, there is little need to argue which theory describes the "true" mechanism of CPR. The determinants of venous return for both mechanisms are quite similar. Certainly, therefore, steps to promote venous return during basic or advanced life support would be effective regardless of the dominant mechanism, and their further investigation should be encouraged. These include, among others, fluid loading, venoconstrictors, negative diastolic airway pressure, abdominal binding, and leg elevation. Determinants of arterial outflow, although in principle different for cardiac versus thoracic pumping, are difficult to separate in practice. Increasing the amplitude of chest compression to a maximally effective level should improve both means of generating forward flow. The site of chest compression is irrelevant for thoracic pumping but critical for external cardiac massage. Therefore, if one does not know the relative contributions of the two mechanisms in a given victim, it seems reasonable to continue with sternal compression in order to massage the heart if this is possible--especially for victims with thin chests in the ventral-dorsal dimension or with cardiac enlargement. Higher blood flows may be possible to the extent that direct compression of the heart occurs, because the positive pressure so generated in arteries relative to thoracic pressure will tend to prevent their collapse. Decreasing peripheral arteriolar resistance, without increasing venous capacitance, by means of selectively acting drugs offers a third possibility for improving vital organ perfusion without knowledge of the predominant mechanism. Such interventions, which theoretically improve flow during both cardiac and thoracic pumping, would be the most fruitful objects of future research.

After decades of practicing CPR clinically, the mechanisms that produce blood flow are finally beginning to be understood. A large portion, if not the majority of blood flow, may be caused for reasons totally different than those assumed in the development of present CPR standards. Fortunately, many of the practical outcomes of the classical concepts also turn out to be reasonable in view of the newer enlarged understanding. Undoubtedly, improved understanding of mechanisms will lead to continued improvement of CPR standards in the future. The artificial cardiac outputs achievable may even double. There may evolve different standards for generating artificial circulation during basic versus advanced life support. Fortunately, however, it seems that the techniques of basic CPR will require modification much more slowly than these theories.

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