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Cardiac, Thoracic, and Abdominal Pump Mechanisms in Cardiopulmonary Resuscitation: Studies in an Electrical Model of the Circulation

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ABSTRACT

To investigate alternative mechanisms generating artificial circulation during cardiopulmonary resuscitation (CPR), an electrical model of the circulation was developed. Heart and blood vessels were modeled as resistive-capacitive networks; pressures in the chest, abdomen, and vascular compartments as voltages; blood flow as electric current; blood inertia as inductance; and the cardiac and venous valves as diodes. External pressurization of thoracic and abdominal vessels, as would occur in CPR, was simulated by application of half-sinusoidal voltage pulses. Three modes of creating artificial circulation were studied: cardiac pump (CP), in which the atria and ventricles of the model were pressurized simultaneously; thoracic pump (TP), in which all intrathoracic elements of the model were pressurized simultaneously; and abdominal pump (AP), in which the abdominal aorta and inferior vena cava of the model were pressurized simultaneously. Flow was greatest with the CP, less with the TP, and least with the AP mechanism. However, the AP could be practically combined with either the CP or TP by interposition of abdominal compressions between chest compressions (IAC-CPR). Our model predicts that this combined method can substantially improve artificial circulation, especially when cardiac compression does not occur and chest compression invokes only the thoracic pump mechanism.

Key words: blood flow, CPR, IAC-CPR sudden cardiac death, ventricular fibrillation

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INTRODUCTION

Any study of improved cardiopulmonary resuscitation (CPR) is complicated by the existence of at least two distinct mechanisms for the generation of forward blood flow by chest compression during cardiac arrest[1-3]. One is the cardiac pump mechanism, which is similar to that of open chest CPR in that the heart is presumed to be squeezed between the sternum and the spine. With the chest closed, however, the pressure is applied both to the cardiac ventricles and to other mediastinal structures including the atria, the esophagus, and some of the great vessels. In this sense, even idealized closed chest cardiac massage is less selective than open chest ventricular massage and in particular causes high venous pressure spikes during the compression phase [4-7], which are absent during open chest CPR [6,7]. Considerable evidence published over the last five years suggests that pressure pulses developed during chest massage are even more widely distributed throughout the thorax. In the limiting case, the pressure throughout the chest is uniformly elevated by chest compression, invoking the thoracic pump mechanism of CPR.

The thoracic pump mechanism of closed chest CPR [1,3,8] is believed to involve pressurization of the entire pulmonary vascular bed in such a way as to squeeze blood from the lungs, through the left side of the heart, and into the periphery, even in the absence of direct heart compression. Venous valves, collapse of veins entering the thorax, and a competent pulmonic valve prevent retrograde flow. Compression of the left side of the heart is not required, and the left side of the heart is said to function as a passive conduit [9].

Yet a third mechanism for generation of blood flow during CPR has been suggested by the work of Rosborough [10] who showed that abdominal compression and ventilation alone can produce significant perfusion during ventricular fibrillation in dogs. Concurrently, Ralston et al. [11] and Voorhees et al. [12] found that abdominal counterpulsation during otherwise standard CPR essentially doubled cardiac output [11], diastolic arterial pressure, and oxygen uptake [12] in anesthetized dogs with ventricular fibrillation. The technique was termed IAC-CPR to indicate interposed abdominal compressions, which can be applied in the field with the bare hands of an additional rescuer. Hence, abdominal compression may provide a third means to generate artificial circulation during CPR.

To study the interactions of chest and abdominal compression in CPR, we developed a simple resistive-inductive- capacitive model of the circulation, in which flow can be generated by compression of either the heart, the entire thorax, the abdominal aorta, or the abdominal veins (by applying voltage pulses to particular sets of capacitors), and in which phased chest and abdominal compression can be simulated. Such electrical models of the circulation permit easy testing of assumptions and manipulation of system parameters to test hypotheses. Such models can serve as a guide to experimentation, suggesting certain interesting experiments and eliminating the need for others. Because such models are much simpler than intact animals, it is easier to evaluate and understand their behavior and the contribution of individual components to the observed results.

In a previous article [13] we described the influence of compression force and peripheral vascular resistance when abdominal counterpulsation is combined with thoracic pump CPR in such a model. This subsequent research was conducted with the following objectives:

1. To characterize the artificial circulation developed by cardiac compression, generalized thoracic compression, and abdominal compression in a simple electronic model of the human circulatory system.

2. To identify the effects of compression frequency for each of the three pump mechanisms.

3. To compare the effects of IAC when combined with cardiac compression versus generalized thoracic compression.

4. To identify the importance of abdominal aortic versus abdominal venous compression in the efficacy of IAC.

MATERIALS AND METHODS

Development of a Circulatory Model

We constructed a simplified electrical analog of the circulation, shown in Figure 1. The great vessels and cardiac chambers are modeled as capacitors, and capillary beds are modeled as resistors. The flow of electric current around the circuit (arrows) represents the flow of blood, and the action of the arterial and venous inductors models the inertia of the blood columns in these larger, longer vessels. Normal cardiac and venous valves are modeled as diodes, which permit flow of current in only one direction. Definitions of the symbols for circuit elements are provided in the figure legend.

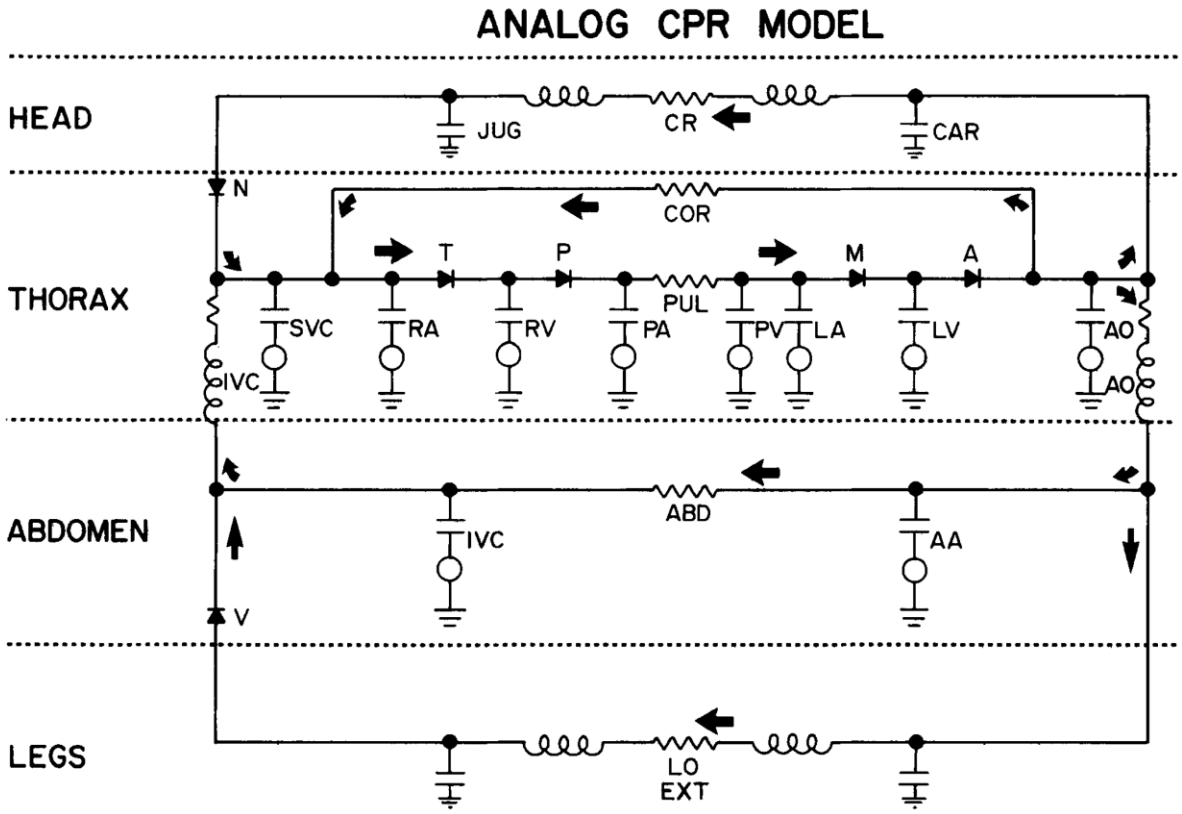

FIGURE 1. Circuit diagram of the model. Elements corresponding to vessels in the head, thorax, abdomen, and legs are identified. Capacitors (-\|\|-) model large vessel compliance; inductors (-eeee-) model blood inertia; resistors (-/\/\/\-) model capillary beds; and diodes (-\|>\|-) model valves. Voltage sources (-O-) are applied between earth ground and the thoracic and abdominal capacitors to model chest and abdominal compression. Arrows indicate direction of current flow. Abbreviations identifying specific vascular elements in alphabetical order are as follows: AA, abdominal aorta; A, aortic valve; ABD, abdominal capillaries; AO, thoracic aorta; CAR, carotid artery; CR, cranial capillaries; COR, coronary capillaries; LA, left atrium; LO EXT, lower extremity capillaries; LV, left ventricle; M, mitral valve; N, Niemann's valve at the thoracic inlet; P, pulmonic valve: PA, pulmonary artery; PUL, pulmonary capillaries; PV, pulmonary veins; RA, right atrium; SVC, superior vena cava; T, tricuspid valve; V, venous valves in legs.

Application of external pressure to blood-containing structures is modeled by the application of voltage pulses between specific capacitors and ground potential, which represents zero (ambient atmospheric) pressure. Table 1 lists the conversion factors for pressure to voltage, flow to current, compliance to capacitance, and inertance to inductance. The conversion factors for capacitance and inductance were further scaled so that the time course of current flow in the model during one-tenth millisecond simulated the flow of blood through the vascular tree in one second. Thus, a compression rate of 1/sec is represented by a frequency of 10 kHz in the model. This frequency transformation by a factor of 10,000 permitted the use of routinely available electronic hardware, avoiding the need for either extremely large capacitances or extremely high voltages. Monitoring of pressure and flow in the model could still be accomplished with the aid of a storage oscilloscope.

TABLE 1. Cardiovascular Variables and Their Electrical Analogs

Physiologic Variable (units)	Electrical Analog (units)	Conversion Factor in Model
Pressure difference (mm Hg)	Voltage difference (volts)	1 mm Hg = 0.5 volt
Blood flow (l/min)	Current flow (amps)	1 l/min = 0.5 ma
Resistance (mm Hg/l/min)	Resistance (ohms)	1 mm Hg/l/min = 1000 Ω
Compliance (ml/mm Hg)	Capacitance (farads)	1 ml/mm Hg = 60 μF 1 ml/mm Hg = 0.006 μF*
Inertance (g/cm^4)	Inductance (henrys)	1 g/cm^4 = 12.59 H 1 g/cm^4 = 1.259 mH*

* Includes 1 Hz to 10,000 Hz frequency transformation.

Current leaving the right side of the heart passes through the pulmonary system, first through the pulmonary artery capacitance, then the pulmonary capillary resistance, and then the pulmonary venous and left atria1 capacitance before entering the left ventricle (Figure 1). Current leaving the left side of the heart through the aortic valve can return to the right atrium via one of four pathways, representing the vascular beds of the head and neck, myocardium, abdomen, and lower extremities. The coronary circulation is modeled as a simple resistive pathway between the thoracic aorta and the right atrium. Separate, pulsatile voltage sources can be applied between ground and any of the vascular capacitors to mimic external compression of these structures during CPR.

A particular value of blood volume was simulated by charging all of the parallel vascular capacitances to a chosen DC voltage, corresponding to the equilibrium, zero-flow pressure in the circulatory system (20 mm Hg) that would exist during cardiac arrest without CPR. Because of the linear relationship between charge and voltage across a simple capacitor, the simulated blood volume (charge) could be determined from the relationship, $V_0 = C_{TB}P_z$, where V_0 is the zero pressure (unstressed) volume of the circulatory system--3,500 ml in most stimulations, C_{TB} is total body capacitance--74 ml/mm Hg in most stimulations, and P_z is zero flow mean circulatory pressure during cardiac arrest--about 20 mmHg. This simulated volume was held on the vascular capacitors for the 120 milliseconds needed to simulate 20 minutes of CPR. Similar electrical models of the circulation have been described by Guyton and coworkers [14] and by others [15-18] (Granick JL Personal communication, March 10, 1976).

To maintain simplicity of design and allow straightforward interpretation of results, many important aspects of classic physiology were specifically not included in the model. For example, the influence of myocardial wall tension of coronary vascular resistance was omitted, since there are no data available to specify such an effect during operation of any of the three mechanisms. Although the dynamic compliance of arteries and veins is known to decrease measurably as the vessels become more distended, the model included constant compliances during any one stimulation. (Reduced venous compliance with volume loading has been stimulated separately by changing the venous capacitance and zero-flow pressure and repeating the simulation [13].) Instead, this simple model was limited to the "plumbing aspects" of the circulation--the movement of blood through resistive elements connected by elastic conduits in a closed circuit similar to the circulatory system--in order to determine how these primary physical elements can be compressed to produce an artificial circulation.

Operation of the Model

To simulate closed chest CPR, similar scaled pressure pulses were applied either to the four cardiac chambers (cardiac pump mechanism); to the four cardiac chambers, superior vena cava, thoracic aorta, and the pulmonary arterial and venous capacitances together (thoracic pump mechanism); or to the abdominal aorta and inferior vena cava of the model (abdominal pump mechanism). For convenience, half-sinusoidal voltage waveforms were used to approximate the rise, peak, and fall in pressure caused by external compressions. When abdominal compression was combined with cardiac or thoracic compression, it was always 180 degrees out of phase with chest compression.

TABLE 2. Initial "Normal" Values of 25 Passive Components of the Model

Name	Physiological Value	Electrical Value
Resistance	(mm Hg/l/min)	(ohms)
Pulmonary vascular bed	2	2000
Coronary vascular bed	263	260,000
Cephalic vascular bed (brain, face, neck)	92	92,000
Abdomen	30	30,000
Lower extremities	142	140,000
Aorta	1.5	1,500
Vena cava	2.2	2,200
Inductances	(g/cm^4)	(mH)
Aorta	15	20
Inferior vena cava	2	2.5
Carotids (2)	15	20
Jugulars (4)	5	6
Iliacs (2) artery and vein	18	23
Femorals (4) artery and vein	38	49
Capacitances	(ml/mm Hg)	(nF)
Systemic arteries	1.56 (SV/PP)	(9.4)
Thoracic aorta	0.78 (50%)	4.7
Abdominal aorta	0.39 (25%)	2.3
Carotids	0.23 (15%)	1.4
Femorals	0.16 (10%)	0.9
Pulmonary arteries	4.17 (SV/PP)	25
Systemic veins	46.9 (30 × SV/PP)	(281)
Superior vena cava	7.0 (15%)	42
Inferior vena cava and abdominal	23.4 (50%)	141
Jugulars	11.7 (25%)	70
Femorals	4.7 (10%)	28
Pulmonary veins	7.8 (1/6 systemic veins)	47
Cardiac chambers:		
Right ventricle	2.5 (SV/(SP-EDP))	15
Left ventricle	0.54 (SV/(SP-EDP))	3.3
Right atrium	5.0	30
Left atrium	5.0	30
Total capacitance	73.5	(440)

Abbreviations: SV = stroke volume, PP = pulse pressure, EDP = end diastolic pressure.

Table 2 shows the initial values of the 25 passive components used to simulate the three modes of CPR. Typical values of resistance and capacitance of vascular beds were selected with reference to published literature and scaled to represent a hypothetical 70-kg man with a normal, resting cardiac output of 5 L/min, arterial blood pressure of 120/80 mm Hg, and pulmonary artery pressure of 25/10 mm Hg. Other assumptions that were made in selecting initial values of resistance, capacitance, and inductance of the model were as follows for normal, resting conditions:

1. For the purpose of computing normal peripheral resistance, pulmonary vascular resistance, and ventricular compliances, normal right atrial pressure is 0 mm Hg and normal left atria1 pressure is 5 mm Hg.

2. Heart rate is 80/min (so that stroke volume is 62.5 ml).

3. Cardiac output in mL/min is distributed as follows: head and neck 1,000; myocardium 350; lower extremities 650; kidneys, skin, and abdominal viscera, 3,000.

4. Total systemic and pulmonary arterial compliances are equal to stroke volume divided by left- or right-sided pulse pressure.

5. Systemic arterial compliance is distributed as follows: thoracic aorta 50%, abdominal aorta 25%, carotids 15%, femorals 10%.

6. Systemic venous capacitance is 30 times aortic capacitance and distributed as follows: superior vena cava 15%; inferior vena cava, abdominal veins, liver and spleen 50%; jugular and cranial veins 25%; femoral veins 10%.

7. Pulmonary venous compliance is 1/6 of systemic venous compliance.

8. Inertance of longer arteries and veins is computed by the expression $L = \rho l/(n\pi r^2)$; where ρ = blood density (1 g/ml), 1 is vessel length in cm, n is the number of parallel vessels (e.g., 2 carotids), and r is vessel radius.

9. Right atrial compliance is twice that of right ventricular compliance and equal to left atria1 compliance.

The exact magnitudes of these quantities during actual cardiac arrest and CPR in human beings are not known at present. As a working approach to the modeling problem, therefore, we have begun with the presumed normal values indicated in Table 2, and then performed simulations with other values to answer specific questions concerning variations in venous capacitance, arterial capacitance, and peripheral vascular resistance [13].

Because we have previously reported that flows in such a model are linearly related to both chest and abdominal pressure by an expression of the form

$$\text{flow} = \alpha P_{chest} + \beta P_{abdomen},$$

the pressure applied to the chest was typically standardized at 80 mm Hg (40 volts) and the pressure applied to the abdomen was typically standardized at 100 mm Hg, values we regard as being clinically reasonable. The duty cycle of chest and/or abdominal compression was always 50%. Compression frequencies ranging from 0 to 120/min were simulated.

The intravascular pressures developed in the model were determined by measuring the voltage with respect to ground potential, using a Tektronics Model D-15 storage oscilloscope and a Tektronics P6015 high voltage probe. In a typical experiment, the model was "charged" by setting the arterial and venous beds to an electrical potential representing 20 mm Hg zero flow equilibrium pressure. Then, thoracic and/or abdominal voltage sources were activated, and the stimulated pressure was recorded on the storage oscilloscope.

Flow in the cranial, coronary, and caudal circuits could be determined by measuring the mean, steady-state voltage developed across peripheral resistance elements and then applying Ohm's law. Cardiac output was determined as the sum of flows in the four peripheral vascular circuits.

RESULTS

Figure 2 illustrates waveforms for simulated aortic and central venous blood pressures during operation of the cardiac, thoracic, and abdominal pumps. In these figures the full horizontal scale represents one compression cycle. The left half of the tracing represents the compression phase. In this comparison the peak applied pressure was 80 mm Hg for all three pumps. During cardiac pump CPR (Fig. 2, top) a positive arteriovenous pressure difference exists through both compression and relaxation phases. During thoracic pump CPR (Fig. 2, center) aortic and central venous pressure rise together during the compression phase, and there is significant systemic perfusion pressure only during the relaxation phase.

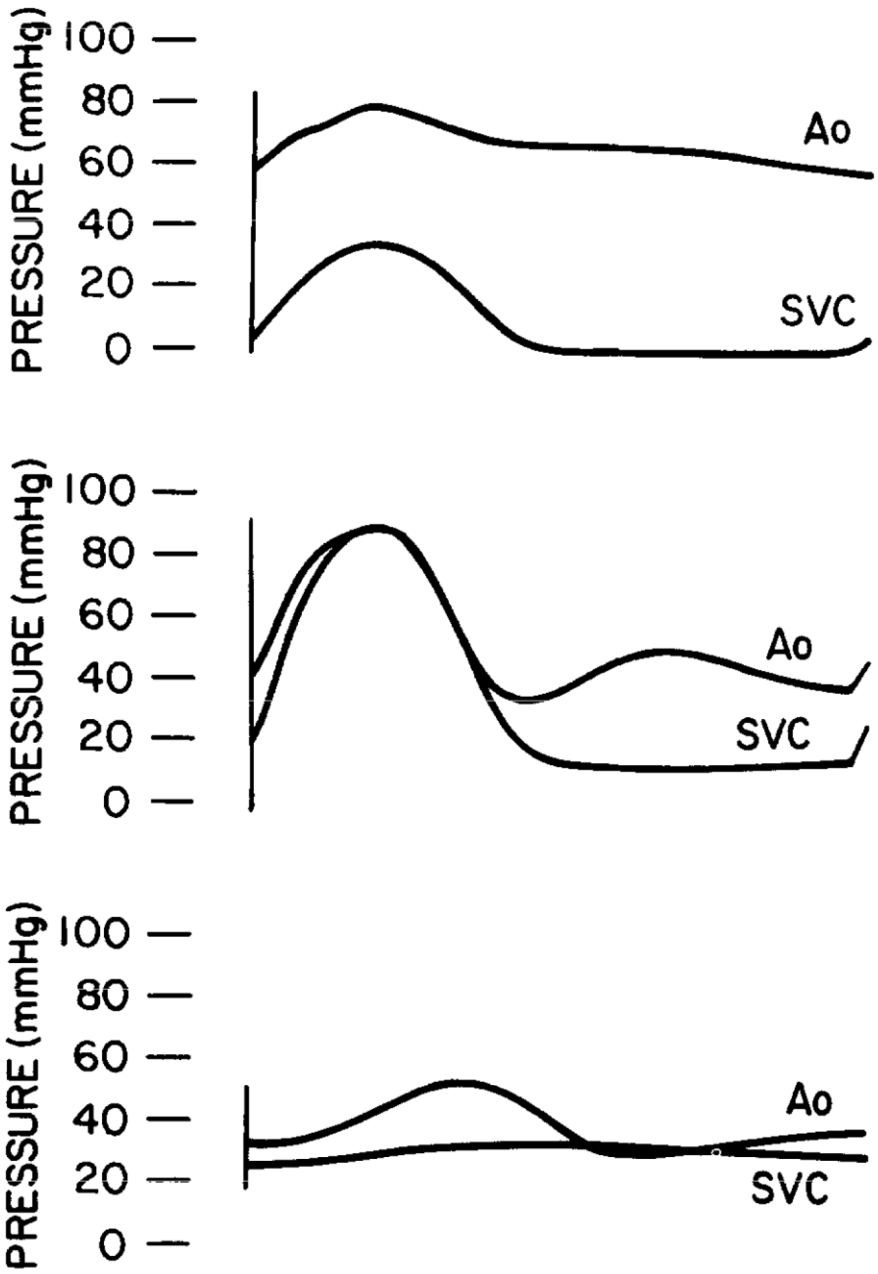

FIGURE 2. Oscilloscope traces showing pressures in the thoracic aorta (Ao) and superior vena cava (SVC) during simulation of cardiac pump CPR (top), thoracic pump CPR (center), and abdominal pump CPR (bottom). Peak extravascular pressure was 80 mm Hg in all cases. The horizontal time base represents one compression relaxation cycle.

During abdominal compression only (Fig. 2, bottom) pressure in the thoracic aorta rises above the zero flow, mean systemic pressure more than does the central venous pressure. As a result, positive systemic perfusion pressure is generated by abdominal compression alone. Comparing the three pump mechanisms, one sees that at the same driving pressure (80 mm Hg) the arteriovenous pressure difference generated is greatest with the cardiac pump only, intermediate with the thoracic pump only, and less still with the abdominal pump only.

Figure 3 illustrates artificial cardiac output and flows to the heart and brain generated by each of the three pump mechanisms as a function of compression frequency. In general, the cardiac pump is more effective than the thoracic pump, which is in turn more effective than the abdominal pump. Interestingly, outputs of the thoracic and abdominal pumps are relatively independent of compression frequency, unlike the output of the cardiac pump. The small differences in flow in the plateau regions of these curves were consistently reproducible, and seem to represent resonance effects.

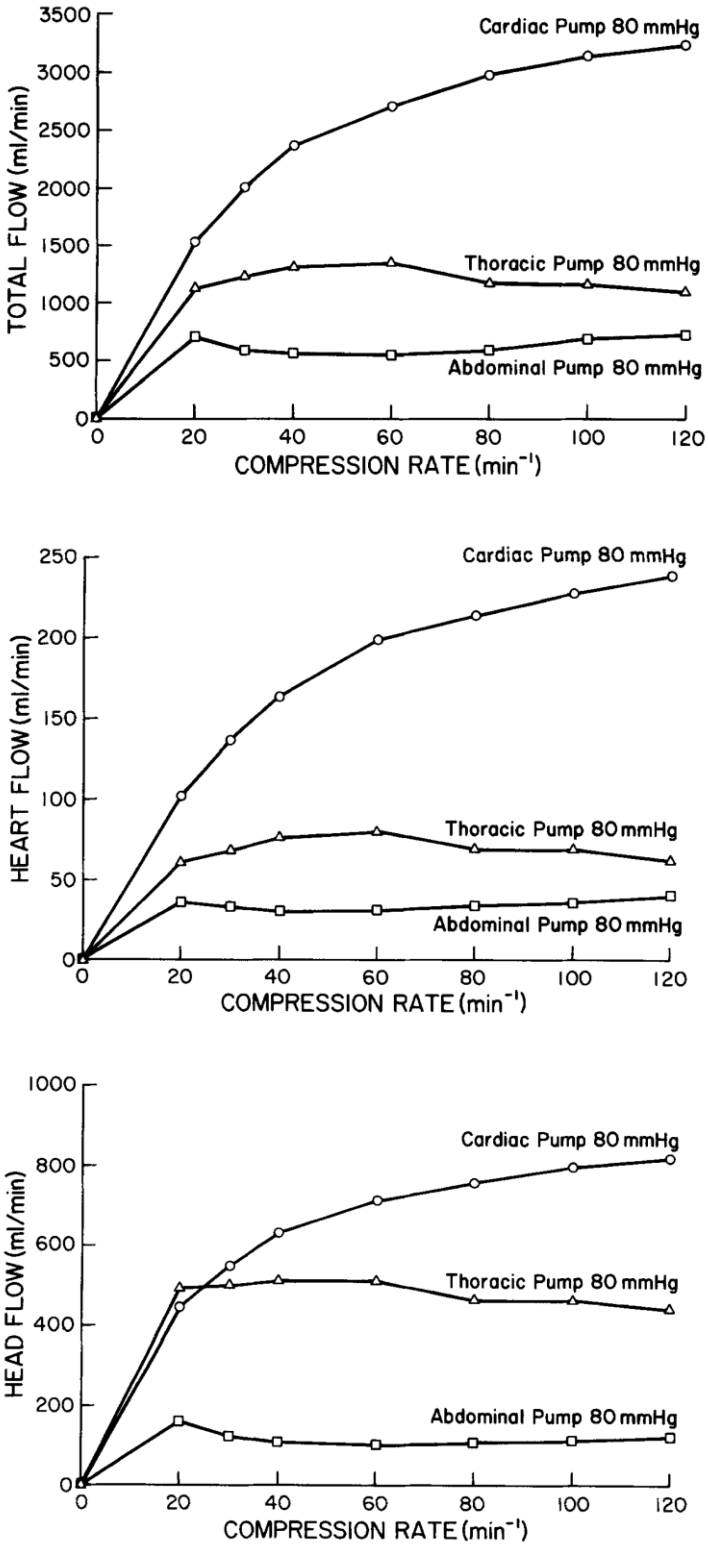

FIGURE 3. Flow generated by three mechanisms at various frequencies. Top, total flow, center, myocardial flow, bottom, cranial flow.

Because the abdominal pump can be combined at will with either of the other two mechanisms, these combinations were studied. Interposed abdominal compression (IAC) at 100 mm Hg, combined with thoracic pump CPR, improves total flow, myocardial flows, and cranial flow at all compression frequencies (Fig. 4). When IAC at 100 mm Hg is added to cardiac pump CPR, there is improved flow for all compression frequencies except 100-120/min, as shown also in Figure 4. Since the flow generated by thoracic pump CPR is substantially less than that generated by cardiac pump CPR, the percentage improvement in flow by IAC is greater when combined with the thoracic pump mechanism. In the case of flow to the head (Fig. 4, bottom) the effect of thoracic pump CPR is more similar to that of cardiac pump CPR, because of the action of Niemann's jugular venous valves [13].

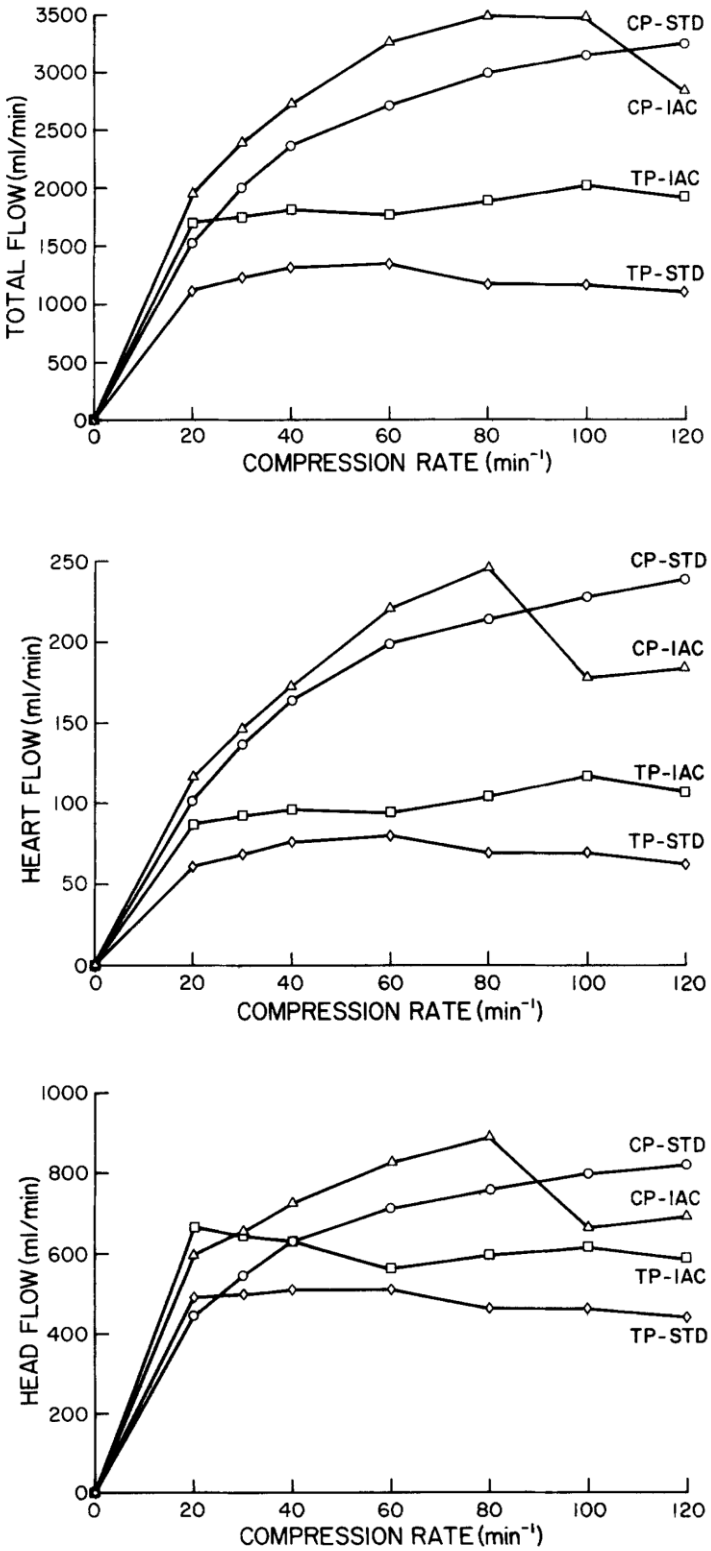

FIGURE 4. Effects of adjunctive abdominal pumping in cardiopulmonary resuscitation (CPR) by means of abdominal counterpulsation combined with cardiac and with thoracic pumps. Top, total flow, center, myocardial flow, bottom, cranial flow, as functions of rate for a simulated circulation. CP = cardiac pump mechanism, TP = thoracic pump mechanism, STD = standard CPR, IAC = CPR with interposed abdominal compressions.

The data in Table 3 compare the effects of abdominal venous compression versus abdominal aortic compression combined with thoracic pump CPR, using cardiac output and coronary perfusion as end-points. In these simulations, counterpressure was either applied or not applied to the abdominal aorta and inferior vena cava of the model in the four possible combinations: aortic counterpressure only, caval counterpressure only, both, and neither. The results in Table 3 suggest that the flow augmentation by IAC is caused in part by aortic and in part by caval counterpulsation, with the greatest flow occurring when counterpressure is applied to both vessels. Coronary flow, however, is nearly as great with abdominal aortic counterpulsation alone as with combined aortic and caval counterpulsation during thoracic pump CPR. Venous counterpulsation alone, however, does slightly increase coronary perfusion in comparison with that achieved by unassisted thoracic pump CPR.

TABLE 3. Flows in ml/min for Thoracic Pump CPR (P_{chest} = 80 mm Hg) with Various Combinations of Abdominal Aortic and Caval Counterpulsation at 100 mm Hg

Inferior Vena Caval Counterpulsation	Abdominal Aortic Counterpulsation			
	Off		On	
	Total Flow	Coronary Flow	Total Flow	Coronary Flow
Off	1,161	68	1,406	102
On	1,603	73	1,874	105

DISCUSSION

By modeling a complex biological system appropriately, an investigator can explore the consequences of certain fundamental assumptions about how the system operates. In the present study we assumed that external pressures can impel blood to flow through resistive-capacitive networks forming a closed circuit similar to the mammalian circulatory system.

The investigation revealed three specific mechanisms for generating an artificial circulation, which we have called the cardiac, thoracic, and abdominal pumps. The artificial circulation produced by cardiac pump CPR is substantially better than the artificial circulation produced by thoracic pump CPR, which is in turn substantially better than the artificial circulation produced by abdominal pump CPR, when all three pumps are tested at the same driving pressure (80 mm Hg). It seemed most practical, then, to employ the abdominal pump as an adjunct to either the cardiac or thoracic pump mechanism, whichever is operative during chest compression. The result is a type of "three-man" CPR in which chest and abdomen are alternately compressed--a technique we have termed IAC-CPR to indicate interposed abdominal compressions. The abdominal contribution to flow seems to add independently to flow generated by either chest

pump mechanism with which it is combined, and is relatively greater when combined with thoracic pump than with cardiac pump CPR, because flows generated by the thoracic pump (TP) are relatively less. The model predicts that TP-IAC-CPR at 80/min will generate about 40% (2 liters/min) of normal resting cardiac output in human beings. This value is nearly identical to that obtained by Voorhees et al. [4] in dogs (approx 40 ml/kg/min), assuming a value for normal resting cardiac output in the dog of 100 ml/kg/min.

One message that seems to emerge from the present research is that every reasonable attempt should be made to invoke a cardiac pump mechanism in order to achieve better perfusion. The available approaches, however, carry identifiable risks. One possible way to increase the likelihood of cardiac compression is to increase the force of chest compression. A recent report from our laboratory [19] demonstrated the neglected importance of chest compression force in generating forward flow during ventricular fibrillation in dogs. In this study, the depth of chest compression had to exceed a critical threshold value, averaging 2.3 cm, before measurable cardiac output (greater than 0) was obtained. It is possible that the increase in cardiac output as compression exceeded this threshold value was due, in part, to increasing amounts of cardiac compression. One might speculate, therefore, that the chances of invoking a cardiac pump mechanism are increased when greater compression force is applied to the sternum in the midline.

One certain way of establishing a cardiac pump CPR is to open the chest surgically and perform direct manual cardiac massage. This approach has been advocated recently, much more strongly than in the past [7,20] because of accumulating evidence that standard CPR may generate inadequate perfusion of the heart in animals [21,22] and that present CPR techniques produce little improvement in resuscitation rates and long-term survival in human beings [23,24].

Either of these methods of invoking a cardiac pump mechanism, however, is associated with increased risk as well as increased benefit, and the optimum parameters of the benefit/risk equation are far from being well defined. Increased sternal compression force, which is though probably desirable, can result in trauma to abdominal organs, especially the liver [19]. More research is needed to delineate the risk/benefit tradeoffs of augmented compression force. Open chest CPR, of course, is a surgical procedure requiring subsequent repair in an operating room, and the number of rescuers presently trained to do open chest CPR is quite small. Accordingly, we are left at present with no safe and certain way of performing cardiac pump CPR in most cardiac arrest victims.

Indeed, most available evidence suggests that CPR as currently performed in adult human beings works by the thoracic pump mechanism. In this regard, the ultrasonic studies of Rich [25] and his co-workers reveal either that the heart is not compressed by closed chest massage or that the right ventricle is compressed somewhat but the left ventricle is not. Thus, closed chest cardiac compression seems to be a distant and elusive goal rather than an everyday clinical reality.

Granted that we must live with thoracic pump CPR under most conditions, the contribution of abdominal pumping provided by IAC may be crucial in maintaining the viability of the heart and brain during prolonged resuscitation attempts. This conclusion is especially pertinent to survival if it can be demonstrated *in vivo* that abdominal counterpulsation improves coronary perfusion

during CPR. Recent work by Ralston and her co-workers [11] in our laboratory indicates that return of circulation after 20 minutes of experimental CPR is almost certain if myocardial perfusion is greater than 25 ml/min/100 g and almost impossible if myocardial perfusion is less than 15 ml/min/100 g. One might therefore conceive of a "survival threshold" for myocardial perfusion near 20 ml/min/100 g that must be exceeded if the heart is to withstand prolonged resuscitation efforts. Under these circumstances, even modest improvements in flow generated by the abdominal pump mechanism may be lifesaving.

The efficacy of IAC-CPR in the electronic model is similar to that reported in canine models. [11,12,26]. Such agreement tends to confirm an assumption underlying much published resuscitation research that the cardiovascular systems of dogs and human beings are fundamentally similar, despite overall differences in scale and certain relative differences in linear dimensions such as those of the hind legs. Canine models of CPR are occasionally criticized for a variety of anatomical differences that are thought to be important in resuscitation, including a high-riding liver, keel-shaped chest, narrow sternum, and mobile mediastinum [27], and one might argue that such species-specific features are responsible for the favorable results of IAC that have been obtained in dogs [11,12], (White BC, personal communication, fall 1983). However, if this proposition were true, one would not expect to observe flow augmentation by IAC in an electrical model, which is completely independent of such anatomic subtleties. Indeed, a major motivation for creating such a model was to determine whether or not the benefits of the abdominal pump mechanism produced by IAC were explained by the fundamental resistive, capacitive, and inertial properties of circulatory systems. The answer to this question appears to be "yes," and one may expect to observe benefits of IAC-CPR in the circulatory systems of both dogs and human beings.

CONCLUSIONS

Three known mechanisms can be employed to generate an artificial circulation during cardiac arrest and CPR. In order of effectiveness they are the cardiac pump, the thoracic pump, and the abdominal pump. The cardiac pump mechanism is invoked in a minority of cases by actual closed chest cardiac massage. It differs from the action of the normally beating heart in that right ventricular pressures are equal to or greater than left ventricular pressures, there is no Starling mechanism, and pulmonary venous pressures are high. Nonetheless, the cardiac pump mechanism can generate positive central arteriovenous pressure differences and vital organ perfusion approximately half normal. The thoracic pump mechanism is invoked by maneuvers that generate generalized intrathoracic pressure pulses, including cough-CPR, CPR with simultaneous chest compression and ventilation at high airway pressure (SCV-CPR), and probably most cases of ordinary clinical CPR in adults as currently practiced. The thoracic pump mechanism is substantially less efficient than the cardiac pump mechanism (although cerebral perfusion tends to be maintained when Niemann's valve is operative) [4,13]. With thoracic pump CPR, central arteriovenous pressure differences and coronary perfusion occur only during chest recoil.

The abdominal pump mechanism can function independently, but it is best used as an adjunct to thoracic pump CPR. During IAC-CPR, compression of the abdominal aorta helps to perfuse the

heart and the brain by raising the central arteriovenous pressure difference. Counterpulsation of abdominal venous elements helps to prime either of the chest pump mechanisms in a manner presumably analogous to the action of the cardiac atria when the heart is beating normally. When on the basis of clinical judgment there is no practical or safe way to invoke cardiac pump CPR, and one is left with the thoracic pump mechanism, the addition of interposed abdominal compressions may substantially improve perfusion and in selected cases could make the critical difference necessary for immediate survival.

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