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ABSTRACT

Animal studies and preliminary clinical observations suggest that the addition of interposed abdominal compressions (IAC) to ventilation and chest compression of standard cardiopulmonary resuscitation (CPR) augments blood flow, blood pressures, and immediate survival. To investigate the physical basis for enhanced circulation during IAC-CPR, we developed an electrical model of the circulation. Heart and blood vessels were modeled as resistive-capacitive networks, pressures as voltages, blood flow as electric current, blood inertia as inductance, and the cardiac and venous valves as diodes. External pressurization of the heart and great vessels, as would occur in CPR, was simulated by application by half-sinusoidal voltage pulses between vascular capacitances and ground. Closed-chest CPR was simulated by pressurization of all intrathoracic capacitances. IAC was simulated by similar pressurization of the inferior vena cava and abdominal aorta, 180 degrees out of phase with chest compression. During simulation of CPR, IAC improved cranial and myocardial perfusion at all levels of chest compression pressure by amounts linearly related to peak abdominal pressure, suggesting that the abdomen can function as a second, independent blood pump during CPR. Brain and heart flow were improved further during simulated vasoconstriction in kidneys, abdominal viscera, and extremities. Based on the fundamental properties of the cardiovascular system represented in the model, abdominal counterpulsation provides a rational basis for flow augmentation during CPR.

INTRODUCTION

Manual abdominal counterpulsation involves pressing on the abdomen of a supine subject with one or both hands in counterpoint to ventricular ejection [1]. This manual technique is a noninvasive analog of intra-aortic balloon pumping, a mechanical technique in which inflation of an intra-aortic balloon is triggered during ventricular diastole to expel blood from the aorta into peripheral vascular beds. In animals with depressed cardiac output, manual abdominal counterpulsation augments diastolic pressure in the thoracic aorta and improves coronary flow [l, 2].

This noninvasive technique is also applicable during circulatory arrest supported by cardiopulmonary resuscitation (CPR). Ralston [3] and Voorhees [4] and their coworkers in our laboratory have found that, compared to standard CPR, CPR with manually interposed abdominal compressions (IAC) generates approximately twice the diastolic arterial pressure, cardiac output, and oxygen uptake in dogs with electrically induced ventricular fibrillation. In these studies abdominal pressure, sensed in the bladder of a blood pressure cuff placed over the mid-abdomen, was always less than 150 mm Hg; none of 20 dogs suffered grossly observable intra-abdominal injury. Moreover, stimulation of esophageal regurgitation by abdominal counterpulsation has not been reported by any who have studied it. These observations suggest that IAC could be an effective and safe modification of basic life support.

Confirmation of these promising results in the dog model of CPR is needed in other test systems, as are more comprehensive studies to determine how the technique can be applied optimally. For example, the shape of the curve relating flow augmentation to peak abdominal pressure is not yet known. Moreover the amount of flow augmentation may vary, depending on the physiologic state of the subject, particularly with regard to the degree of peripheral vasodilation, the competence of the cardiac and venous valves, and the relative compliance of veins and arteries. Each of these factors has been reported as crucial to the effectiveness of closed-chest cardiac massage, especially in producing coronary artery blood flow [5-9]. Quite possibly IAC may be relatively ineffective when peripheral resistance is low, when cardiac or venous valves are incompetent, or when arterial or venous compliance is low, and thus IAC may be limited in its clinical application.

These physiologic variables are examined in this study of an electrical model of the circulation. Electrical models of the circulation permit easy testing of assumptions and straightforward manipulation of system parameters to define the conditions for theoretically optimal flow. Because such models are much simpler than intact animals, evaluation and understanding of their behavior are more straightforward, and it is much less likely that the observed results are due to the action of uncontrolled and unappreciated variables. In particular, if flow augmentation by abdominal counterpulsation can be demonstrated in an elementary electrical model of the circulation, then it is likely to represent a species-independent improvement in technique based on the general properties of circulatory systems represented in the model.

Accordingly, we conducted our study with the following objectives:

1. To investigate the effects of IAC during CPR in an electrical model of the circulation;

2. To characterize flow augmentation during IAC as a function of the peak value of abdominal counterpressure;

3. To study the effects of raised or lowered peripheral vascular resistance during CPR with IAC;

4. To study the effects of altered arterial and venous compliance during CPR with IAC; and

5. To study the effects of valve competence versus incompetence during CPR with IAC.

MATERIALS AND METHODS

Circulatory Model

To conduct this research we constructed the simplified electrical analog of the circulation that is shown (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 inertance of blood columns in the larger, longer vessels. Normal cardiac and venous valves are modeled as germanium diodes, which permit flow of current in only one direction. Definitions of the symbols for circuit elements are provided in the figure legend.

Application of external pressure to blood-containing structures was modeled by the application of voltage pulses between specific capacitors and ground potential, which represents zero (ambient atmospheric) pressure. The conversion factors for pressure to voltage, flow to current, compliance to capacitance, and inertia to inductance are listed in Table 1. The conversion factors for capacitance and inductance were scaled further 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 heart 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 capacitors and inductors or extremely high voltages.

Current leaving the right heart passes through the pulmonary system, first through the pulmonary-artery capacitance, then the pulmonary capillary resistance, and then the pulmonary venous and left atrial capacitances, before entering the left ventricle (Figure 1). Current leaving the left heart through the aortic valve can return to the right atrium by one of four pathways -- representing the vascular beds of the head and neck, myocardium, abdomen, and lower extremities.
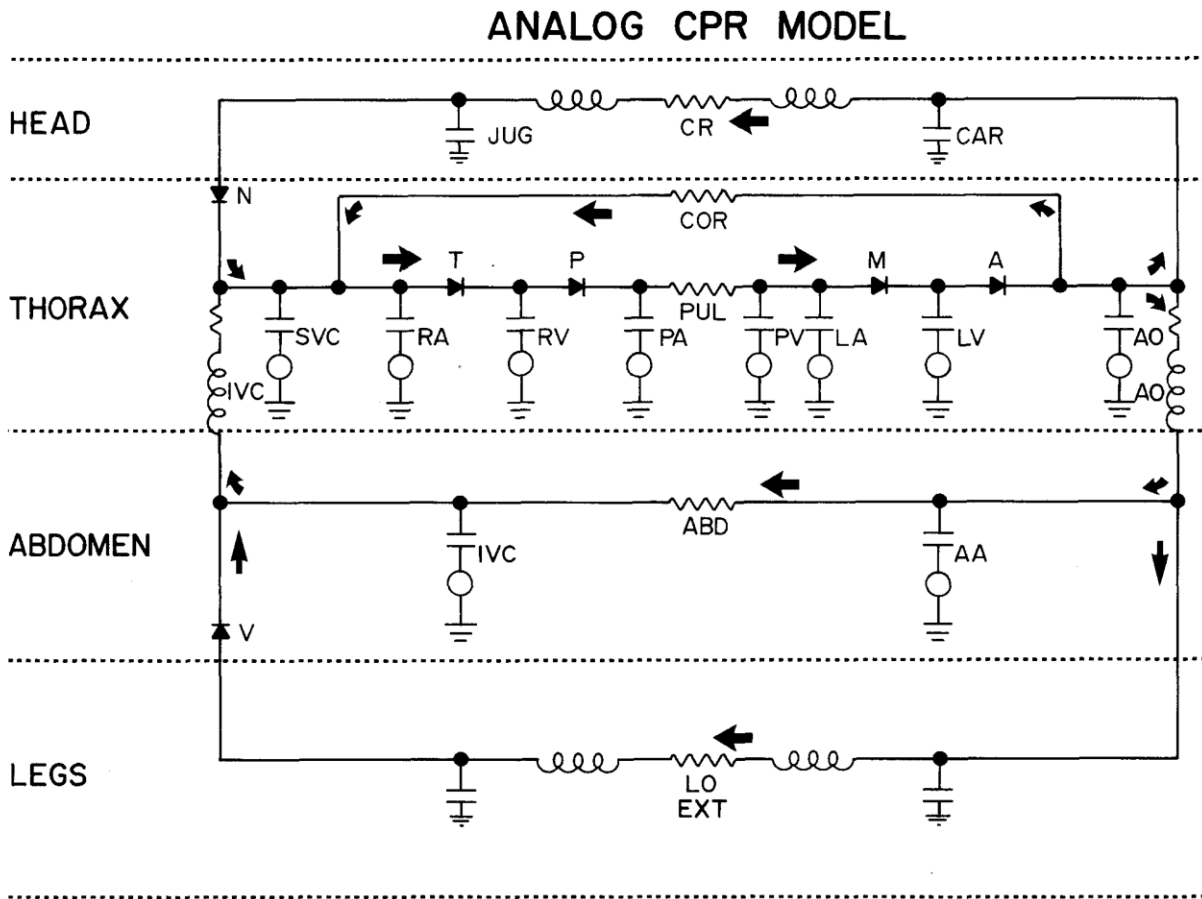
ANALOG CPR MODEL

Fig. 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 (-oooo-) 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: AA, abdominal aorta; A, aortic valve; ABD, abdominal capillaries; AO, thoracic aorta; CAR, carotid artery; COR, coronary capillaries; CR, cranial capillaries; FEM, femoral arteries or veins; IVC, inferior vena cava; JUG, jugular veins; 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; and V, venous valves in legs.

Abdominal and thoracic components of the aorta and vena cava were specified as discrete elements in the model to allow simulation of abdominal compression. The coronary circulation was modeled as a simple resistive pathway between the thoracic aorta and the right atrium. Separate voltage sources could be applied between ground and the thoracic and abdominal capacitors to mimic external compression of these structures during CPR. Similar electrical models of the circulation have been described by Guyton and coworkers [10] and by others [11-14]; none, however, has used such a model to study CPR.

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 V
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 = 1,000 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.

One particular element was added to the model especially for CPR simulations. This element is Niemann's valve (denoted N in Figure 1), a functional venous valve at the level of the thoracic inlet between the jugular vein and the superior vena cava. This valve has been demonstrated by Niemann and Rosborough et al. [15, 16] as well as by Voorhees [17] and by Rudikoff [18] and their coworkers, to be of functional importance during CPR in dogs and in at least one human subject. This particular venous valve remains open during normal quiet breathing, but is closed by pulses of high intrathoracic pressures such as are generated during cough and during CPR. Niemann's valve appears responsible for the diminished venous pressure pulses measured in the jugular veins during CPR as compared to pressure pulses in other systemic veins.

In general the model was planned deliberately to be uncomplicated in order to maintain simplicity of design and to allow straightforward interpretation of results. The influence of myocardial wall tension on coronary vascular resistance was omitted, because there are no data available to specify such an effect during CPR. Although the dynamic compliance of arteries and veins is known to decrease measurably as the vessels become more distended, the vascular compliances of the model were constant during any one simulation. (Altered vascular compliance was simulated separately by changing vascular capacitance and repeating the simulation.)

Values of resistance and capacitance of vascular beds (Table 2) were selected with reference to published literature and were scaled to a hypothetical 70-kg man with a 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 about normal, baseline physiology were made in order to specify initial values of resistance, capacitance, and inductance in the model as follows:

1. For the purpose of defining normal peripheral resistance, pulmonary vascular resistance, and ventricular compliances, normal right atrial pressure is 0 mm Hg and normal left atrial 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 when the heart is beating normally: 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, respectively.

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

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

7. Pulmonary venous compliance is $\frac{1}{6}$ of systemic venous compliance.

8. Inertance of longer arteries and veins is computed by the expression $L = \rho l/(n\pi r^2)$,[31] where ρ is blood density (1 g/mL), l is vessel length in centimeters, n is the number of parallel vessels (eg, 2 carotids), and r is vessel radius in centimeters.

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

Finally small values of resistance, 1 and 2 mm Hg/L/min respectively, were included in series with the inductances of the aorta and inferior vena cava to eliminate short circuits between the abdominal and thoracic sections of the model. Such short circuits could have overloaded the power supply and could have permitted the compliances to become reverse-charged during IAC.

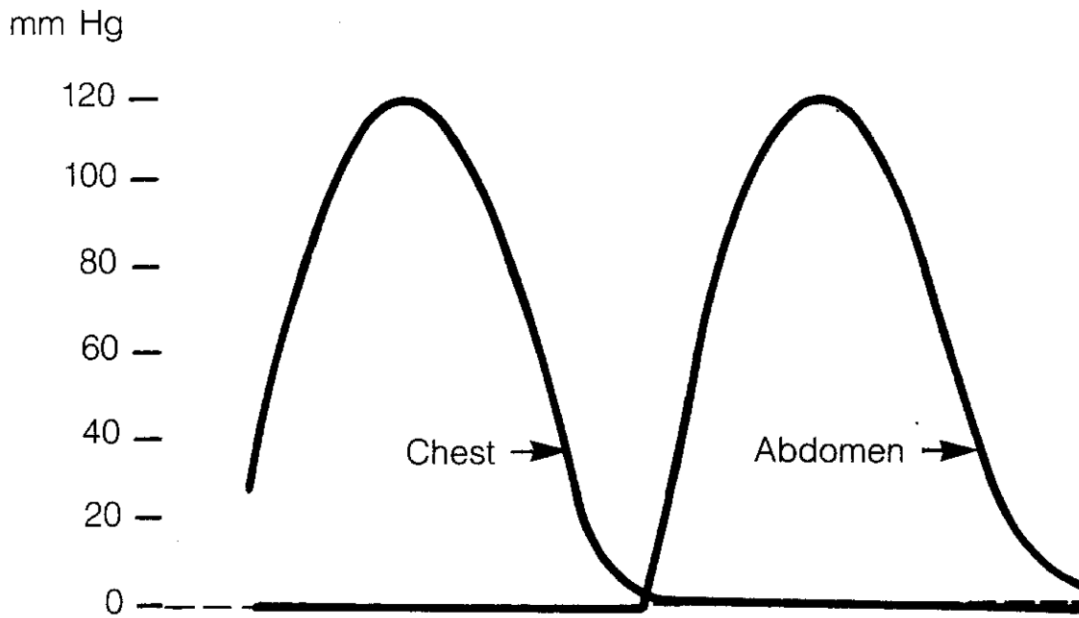*Fig. 2. Specification of model components.*

Fig. 3. Oscilloscope trace showing pressure (voltage) waveforms applied to the thorax and abdomen of the model during simulation of closed-chest IAC-CPR. Maximal peak pressures representing 120 mm Hg are shown. The entire horizontal time base represents one compression cycle.

An interstitial fluid compartment was not modeled, so that any exchange of fluid between intravascular and extravascular compartments during 20 minutes of CPR was not accounted for in the simulations. Finally, the action 'of the renin-angiotensin system in shock was not included. Rather, the model was limited to the "plumbing aspects" of the circulation. It was intended to simulate the movement of blood through resistive elements connected by elastic conduits in a closed circuit. Through manipulation of these primary physical elements, we sought to determine whether there is a way to produce a more effective artificial circulation than is achievable with standard CPR.

On the basis of standard published descriptions of the normal cardiovascular system of a 70-kg man, it was possible to assign specific numerical values to all of the model components (Table 2) according to textbook principles of physiology (Figure 2). Off-the-shelf components with these values (within a tolerance of ± 10%) were then included in the model (Figure 1). This initial configuration was intended to represent an otherwise healthy individual just prior to onset of sudden cardiac death and CPR. Unfortunately, data specifying the magnitudes of vascular resistances and compliances during progression of actual cardiac arrest and CPR in man are not available. Therefore, as a working approach to the modeling problem, we began with the presumed normal values (Table 2), and then performed simulations with a range of other values to answer specific questions.

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	2,000
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
Iliac arteries and veins (2)	18	23
Femoral arteries and veins (4)	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 x SV/PP)	(281)
SVC	7.0 (15%)	42
IVC 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		
RV	2.5 (SV/[SP-EDP])	15
LV	0.54 (SV/[SP-EDP])	3.3
RA	5.0	30
LA	5.0	30
Total Capacitance	73.5	(440)

SV = stroke volume.
PP = pulse pressure.
EDP = end diastolic pressure.

Operation of the Model

In order to simulate cardiac arrest and CPR with our model, first it was necessary to specify the mechanism by which blood flow is generated during CPR. The mechanism believed to generate forward blood flow in most animal models [19] and in most human patients [20, 21] is termed the "thoracic pump mechanism." The thoracic pump mechanism of closed chest CPR [8, 22, 23] involves pressurization of the entire pulmonary vascular bed in such a way as to squeeze blood from the lungs, through the left heart, and into the periphery--even in the absence of direct heart compression. Left heart compression is not required, and the left heart is said to function as a "passive conduit [24]. To model thoracic pump CPR, scaled half-sinusoidal voltage pulses were applied to the four cardiac chambers, superior vena cava, aorta, and pulmonary arterial and venous capacitances together. The cardiac or CPR cycle length was always 0.75 second (75 μsec in the model), corresponding to a compression rate of 80/min, and the duty cycle of chest compression was 50%. To model interposed abdominal compressions, half-sinusoidal voltage pulses exactly 180 degrees out of phase with the thoracic pressure pulses were applied between ground and the abdominal aortic capacitance and the abdominal venous capacitance (Figure 3). Values of abdominal counterpressure ranging from zero to 120 mm Hg were simulated, and the resultant flows were compared with those developed without abdominal counterpulsation.

Measurement of Pressures and Flows

Intravascular pressures in the model were determined by measuring the voltage with respect to ground potential with the aid of a Tektronix Model P6013A high-voltage probe, connected to a Tektronix Model D15 storage oscilloscope (Tektronics, Inc, Beaverton, OR). The relatively high 100-MΩ input impedance of the P6013A probe was required to ensure that leakage of charge (blood volume) through the probe to ground was negligible during the 120 msec necessary to simulate 20 minutes of CPR. In a typical experiment all elements of the model were charged to the same reference potential corresponding to the zero-flow intravascular pressure. This zero-flow potential represents the equal pressure (about 20 mm Hg) that would exist in arteries and veins shortly after onset of ventricular fibrillation without CPR. Then thoracic and abdominal voltage sources were activated and the desired pressure waveform was recorded on the D15 storage oscilloscope with a sweep speed of 20 μsec per division.
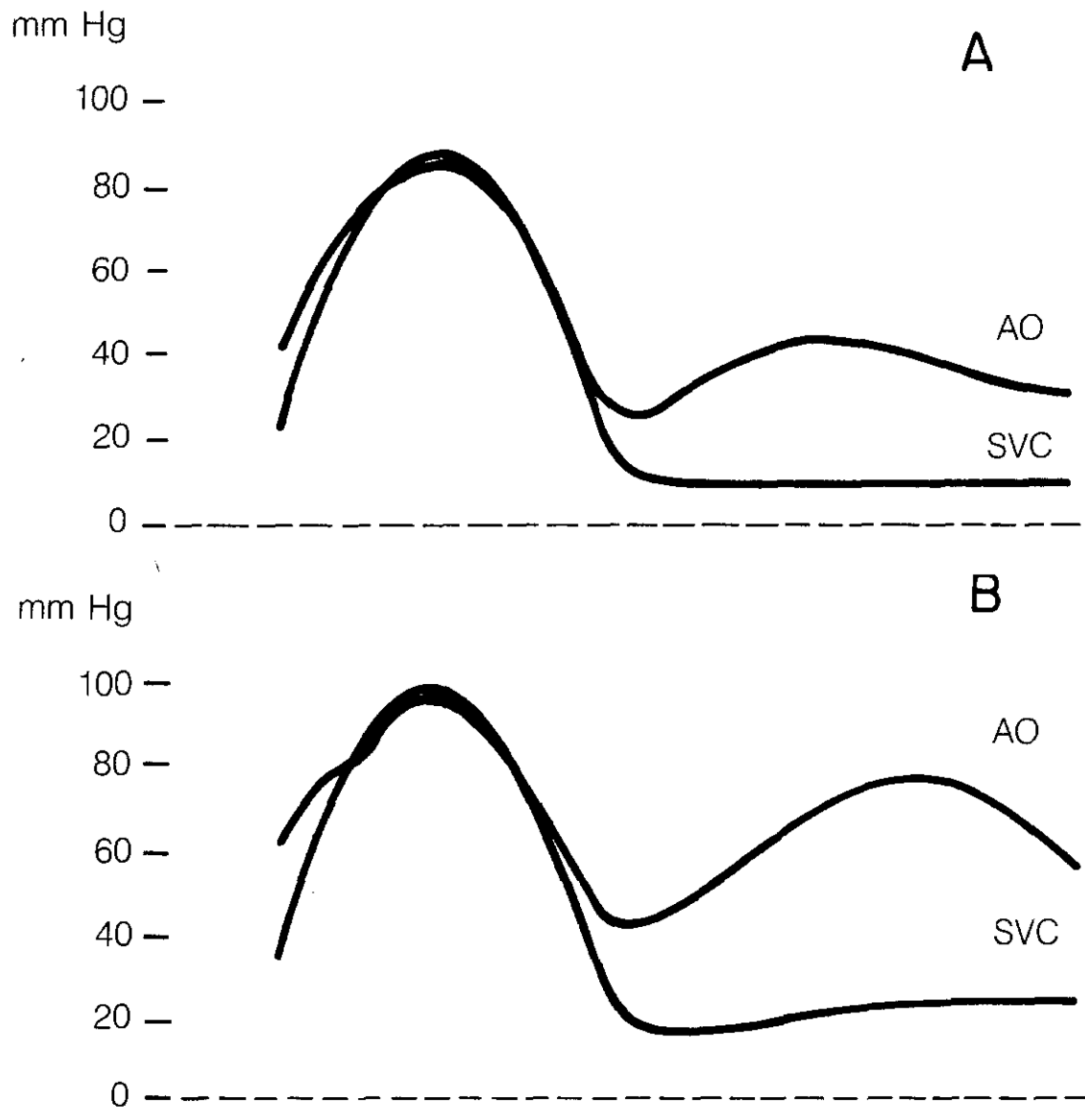
Fig. 4. Oscilloscope trace showing pressures in the thoracic aorta (AO) and superior vena cava (SVC) during simulation of thoracic pump CPR without (A) and with (B) interposed abdominal compressions (IAC). Peak chest pressure was 80 mm Hg and peak abdominal counterpressure was 100 mm Hg in this simulation. The horizontal time base represents one complete cycle. The major effects of IAC occur during diastole (chest recoil); aortic diastolic pressure is raised to a greater extent than is central venous pressure.
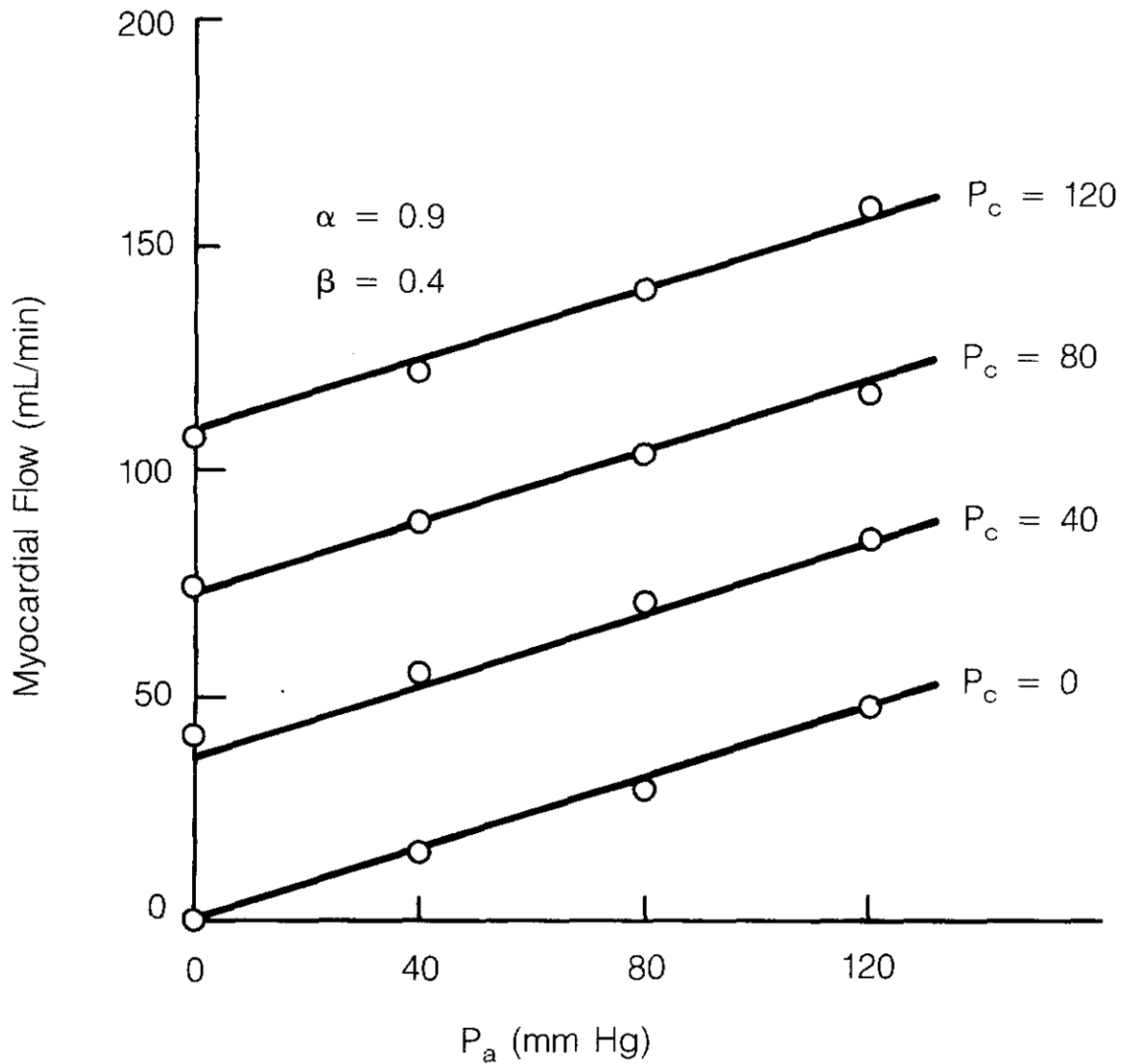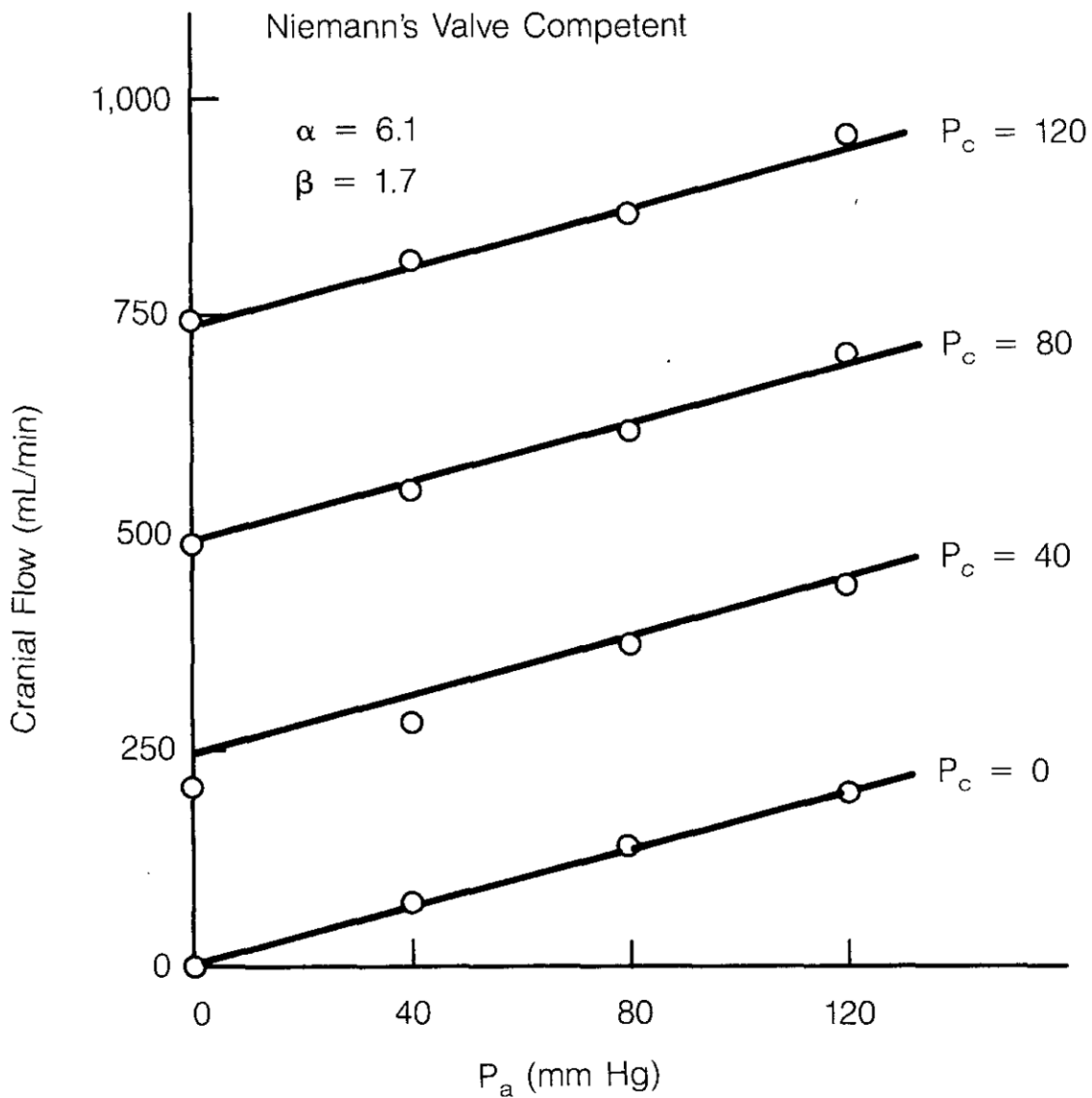
Fig. 5. Effects of varying abdominal counterpressure on myocardial (coronary) flow for various levels of chest compression pressure during simulated cardiac arrest and CPR. P_c represents peak value of chest compression pressure and P_a represents peak value of abdominal counterpressure. Data points represent measured values and straight lines represent the multiple linear regression of the form, flow = $\alpha\, P_c + \beta\, P_a$, with ($\alpha$ = 0.90 and β = 0.40 mL/min/mm Hg.

Fig. 6. Effects of varying abdominal counterpressure on cranial (brain) flow for various levels of chest compression pressure. Details are similar to those of Figure 5.

(A) Simulations were performed with a functional Niemann's valve at the level of the thoracic inlet, in which case supranormal flows were possible. The multiple linear regression coefficients are $\alpha = 6.14$ and $\beta = 1.68$ mL/min/mm Hg.

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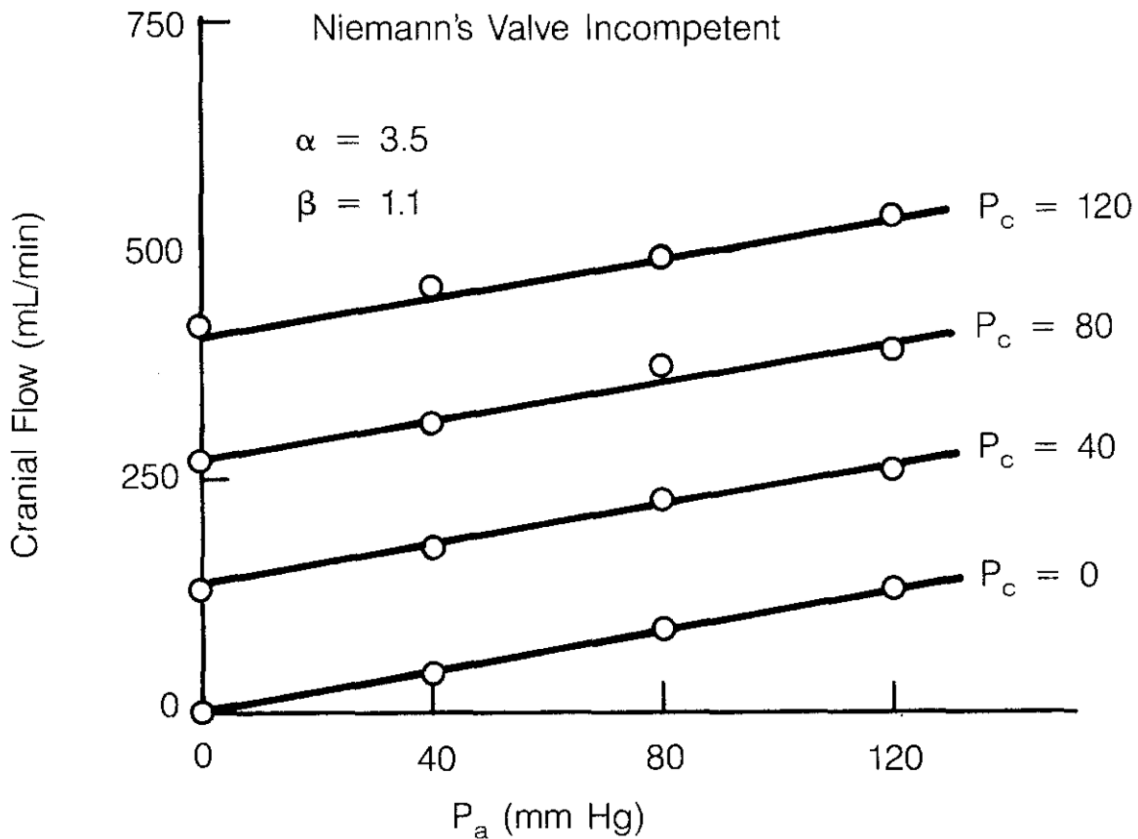
6B

(B) Simulations performed with an incompetent Niemann's valve at the level of the thoracic inlet, in which case the multiple linear regression coefficients were reduced to $\alpha = 3.45$ and $\beta = 1.06$ mL/min/mm Hg.

Flow in the cranial, coronary, abdominal, and lower extremity vascular beds was determined by measuring the mean voltage developed across the vascular resistances and applying Ohm's law. Specifically if resistance (R) represents a capillary bed, and $\hat{e}_1$ is the mean voltage measured with respect to ground on the arterial side of the resistor during one simulation, and $\hat{e}_2$ is the mean voltage measured with respect to ground on the venous side of the resistor during an identical simulation, then the simulated flow through the capillary bed was calculated as:

$$\text{Flow} = (\hat{e}_1 - \hat{e}_2)/R .$$

For such calculations mean voltage was determined as the difference between the waveform recorded with the oscilloscope in the direct coupled mode and the steady-state waveform with

zero mean value recorded with the oscilloscope in the AC coupled mode. Cardiac output was determined as the sum of flows in all four peripheral vascular beds under any given test condition.

Data Analysis

In order to separate the effects of thoracic and abdominal compression pressure on peripheral perfusion of the various vascular beds, a multiple linear regression analysis was performed on flow data obtained with various combinations of chest and abdominal pressure. Multiple linear regression analysis was performed to fit a linear equation of the following form:

Flow = α [chest pressure] + β [abdominal pressure]

by the least-squares method. The regression coefficients α and β were found by solving the normal equations:

$$\sum x_i z_1 = \alpha \sum x_i^2 + \beta \sum x_i y_i$$

and

$$\sum y_i z_1 = \alpha \sum x_i y_i + \beta \sum y_i^2 \ ,$$

where z is flow, x is chest pressure, and y is abdominal pressure [25]. Then the overall effectiveness of abdominal counterpulsation under various simulated physiologic conditions could be compared in terms of the value β and/or the ratio β/α.

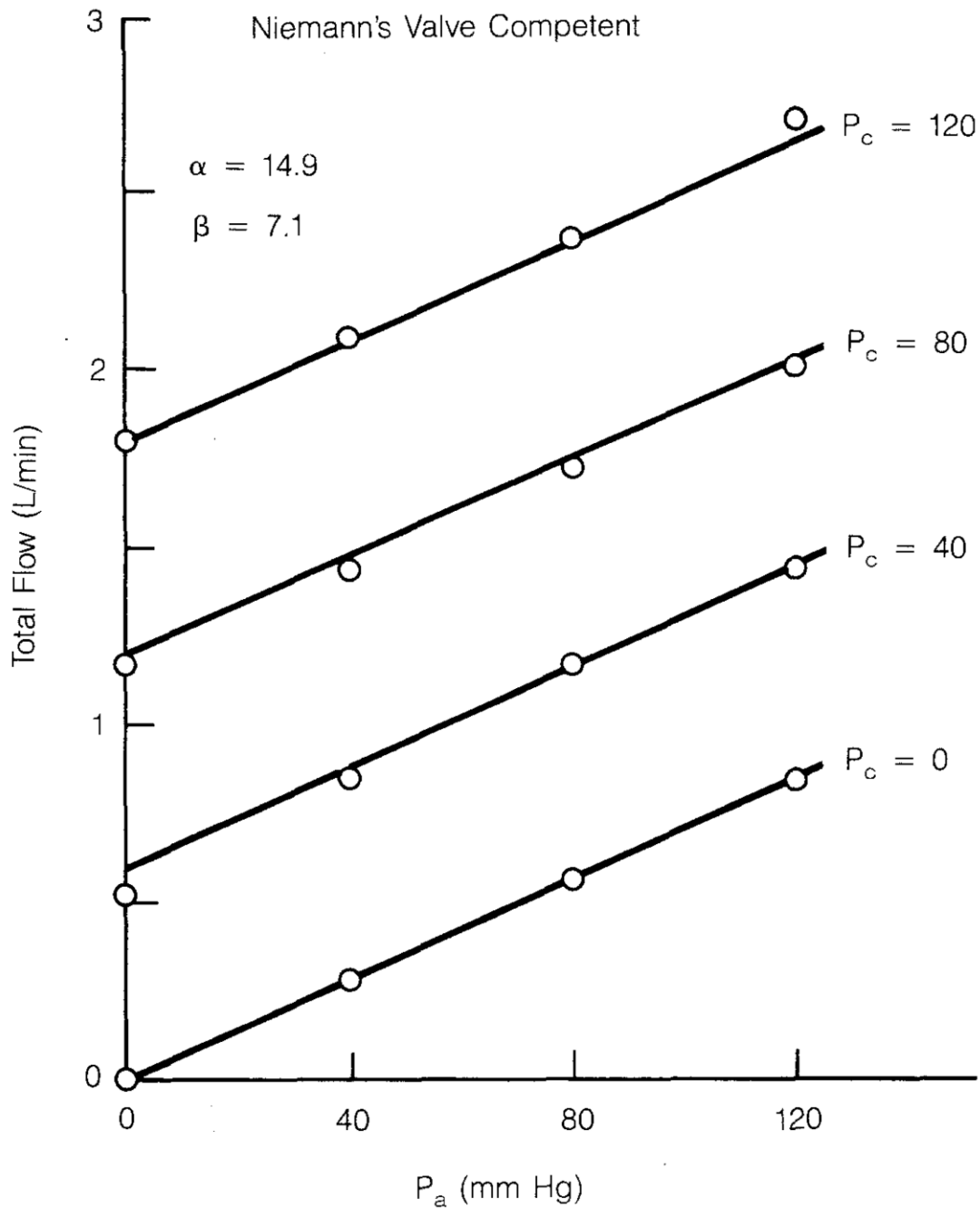*Fig. 7. Effects of varying abdominal counterpressure on artificial cardiac output for various levels of chest compression pressure during simulated cardiac arrest and CPR. Details are similar to those of Figure 5. Data points represent summed values for all vascular beds with Niemann's valve competent. The multiple linear regression coefficients are $\alpha = 14.9$ and $\beta = 7.1$ mL/min/mm Hg.*

RESULTS

Aortic and central venous pressures simulated for thoracic pump CPR are illustrated (Figure 4), with model parameters as indicated (Table 2) and blood volume of 5,000 mL. The full horizontal scale represents one compression cycle. Standard CPR without (Figure 4A) and with (Figure 4B) 100 mm IAC applied to inferior vena cava and abdominal aorta during the second half of the cycle are represented. During the chest compression phase, aortic and central venous pressures were similar. During the chest recoil phase (diastole), an arteriovenous pressure gradient existed, which was augmented during abdominal counterpulsation.

Effect of Abdominal Counterpressure

The families of curves shown (Figures 5-7) reveal the general dependence of blood flow to the myocardium and brain, as well as total flow (cardiac output) on both chest and abdominal pressure in this model. The families of straight lines in each figure were computed from multiple regression analysis, and show that flow increased as a function of both chest pressure (P_c) and abdominal counterpressure (P_a) as described by the following expression:

$$Flow = \alpha P_c + \beta P_a.$$

Even with zero chest pressure (bottom lines), abdominal pulsation generated measurable flow. Because the data points for each chest pressure fell along parallel lines, it appears that blood flow can be generated by two independent mechanisms (the "thoracic pump" and the "abdominal pump"), and that the results are additive. The values of brain perfusion with or without abdominal counterpulsation were dependent on the competence of Niemann's valve (Figures 6A and 6B). When Niemann's valve was competent, protecting the jugular venous bed from the high intrathoracic pressure pulses, then cranial flow exceeding 50% of normal could be established. When Niemann's valve was incompetent, substantially less artificial circulation to the brain could be generated. In either case, however, abdominal counterpulsation improved blood flow to the brain in a predictable way.

Artificial cardiac output generated during CPR was similarly dependent on both chest and abdominal pressure (Figure 7). With the combination of maximal chest and abdominal pressures (each 120 mm Hg), blood flows approaching 50% of normal resting levels could be simulated in the model when normal values of peripheral resistance were simulated.

Effect of Peripheral Vascular Resistance

The effectiveness of both standard CPR and IAC-CPR was found to be critically dependent on caudal (peripheral) vascular resistance. As shown (Figure 8A), coronary flow was enhanced greatly in the presence of high peripheral vascular resistance in the abdomen and lower extremities. In these simulations peripheral resistances changes were modeled by first increasing values of resistance for abdominal viscera and extremities by multiples of 2, 5, 10, and 20, and

then by decreasing values of these resistances by factors of 1/2, 1/5, 1/10, and 1/20. Coronary and cranial resistances were maintained at previous normal levels.
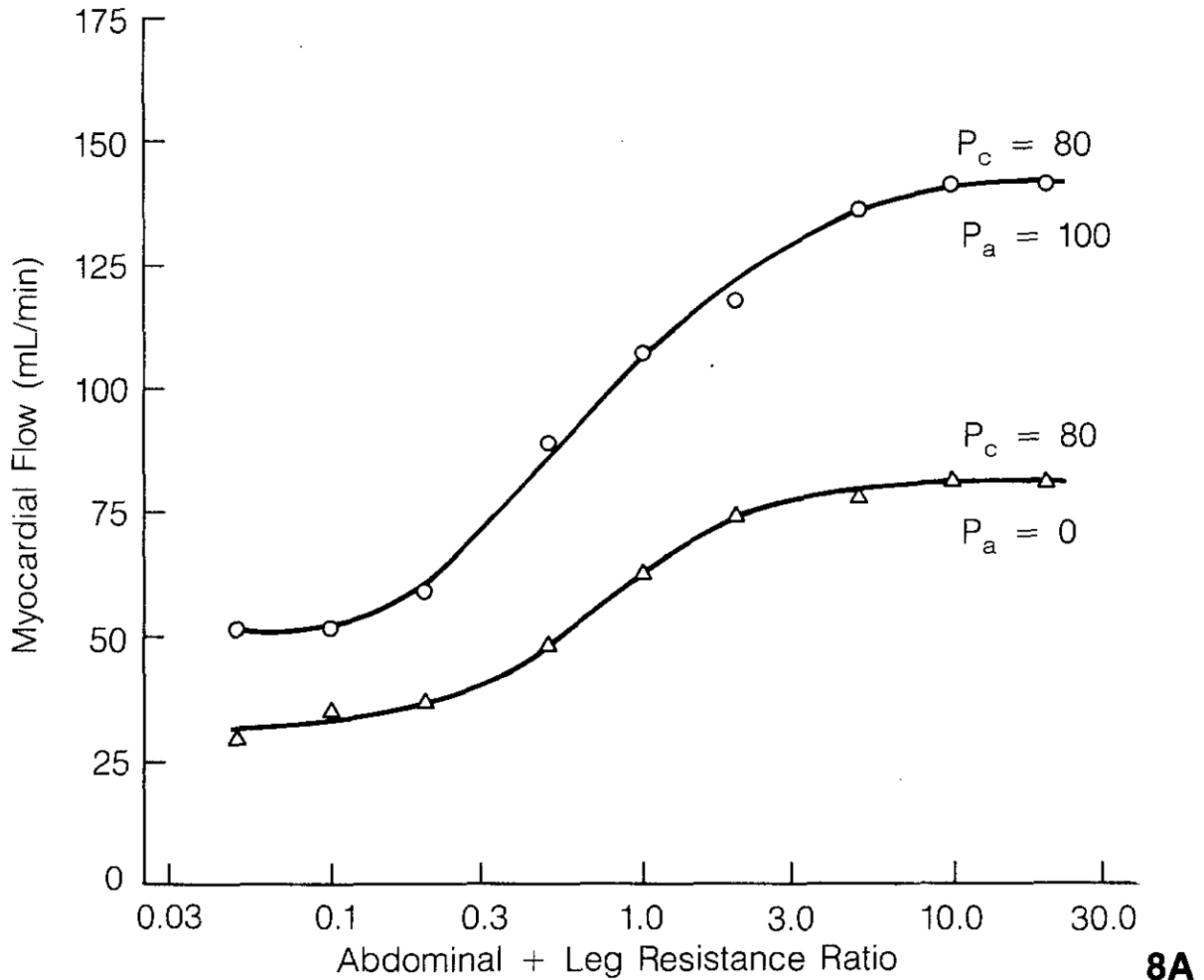

Fig. 8. Simulated effects of epinephrine produced by varying vascular resistance of abdominal and lower extremity beds (caudal resistance). Cranial and myocardial resistances were not changed. Data points represent measured values; 1.0 on the horizontal axis (log scale) represents normal peripheral resistance (Table 2). Smooth curves are hand-drawn trend lines. (A) Coronary flow was nearly doubled by abdominal counterpulsation (P_a = 100) in the presence of supranormal peripheral vascular resistance, and flows approaching 50% of normal resting myocardial perfusion were obtained.
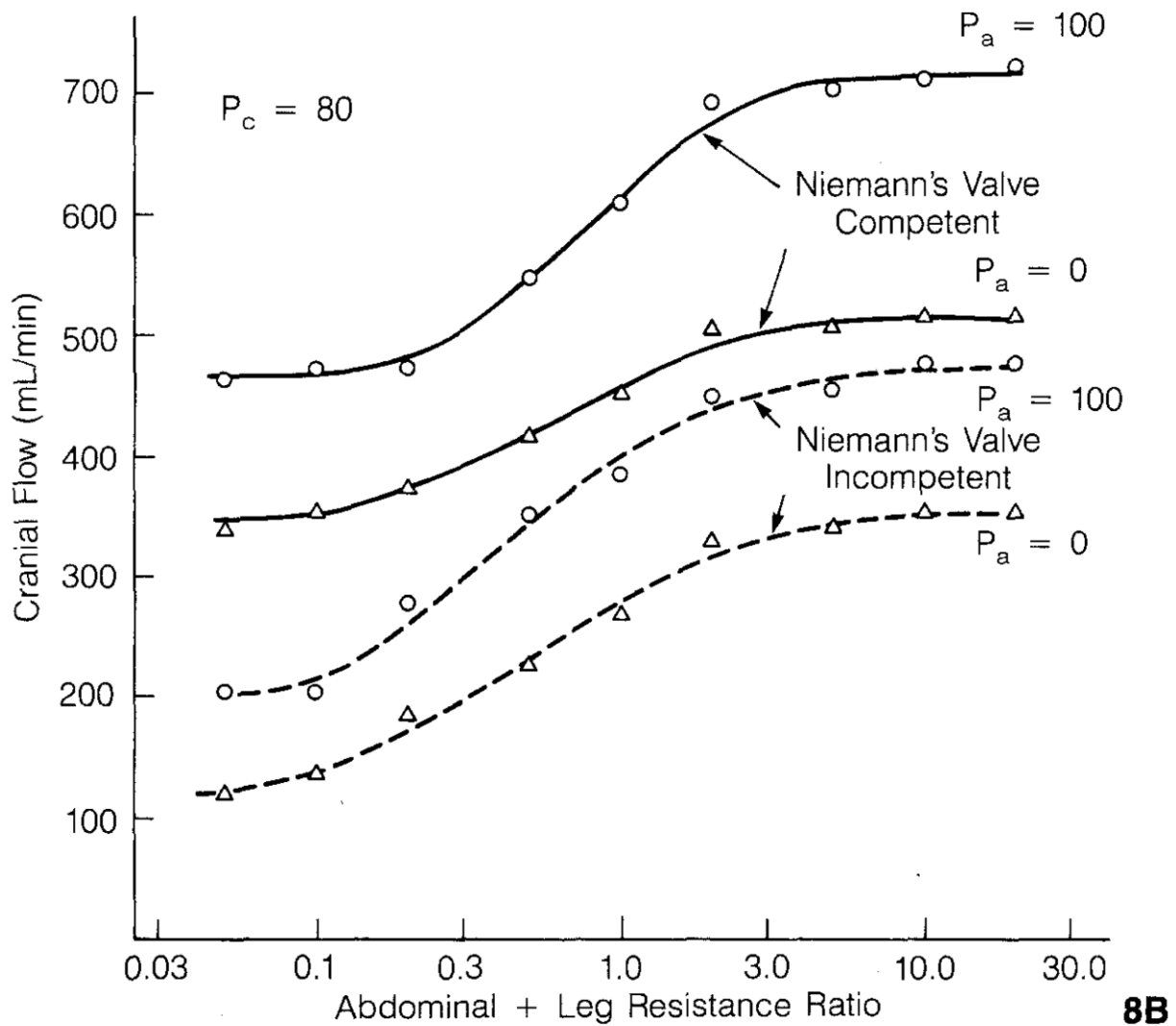
Fig 8 (B) Brain flow was also enhanced with peripheral vasoconstriction. Solid curves were determined with Neimann's valve competent, dashed curves with Niemann's valve incompetent. Brain perfusion approaching normal resting levels was possible in the model when epinephrine effect was combined with abdominal counterpulsation, a result that has recently been confirmed experimentally [30].

The sigmoid curves (Figure 8A) indicate that the maximal beneficial effect of vasoconstriction was obtained when peripheral resistance was five to ten times normal. Under these circumstances there was a synergistic effect of vasoconstriction and abdominal counterpulsation. In the presence of peripheral vasoconstriction, abdominal counterpulsation at 100 mm Hg (upper curve) nearly doubled coronary perfusion.

Similar results were obtained for brain perfusion (Figure 8B). When Niemann's valve was functional (solid curves), supranormal flows were obtained in the model with the combination of abdominal counterpulsation and peripheral vasoconstriction. When Niemann's valve was short-circuited and made incompetent, cranial perfusion generally was reduced, but still was augmented by the combination of abdominal counterpulsation and peripheral vasoconstriction.

Venous versus Arterial Compliance

Venous capacitance was found to be relatively unimportant in determining flow (Table 3); aortic compliance, however, was found to be quite important (Table 4). Coronary flow during IAC-CPR, for example, was greater when abdominal aortic compliance was relatively high and/or thoracic aortic compliance was relatively low. Coronary flow during IAC-CPR was less when abdominal aortic compliance was relatively low and/or thoracic aortic compliance was relatively high. Interestingly, reduced compliance in both the abdominal and thoracic portions of the aorta, as would occur with generalized stiffening of the aorta by atherosclerosis, caused relatively slight reduction in the effectiveness of IAC-CPR.

TABLE 3. Effects of varying venous compliance on coronary and cranial flows (mL/min)

	Abdominal Pressure (mm Hg)	Venous Capacitance 1/10 Normal	Venous Capacitance Normal	Venous Capacitance 10 × Normal
Coronary Flow	0	70	60	56
	100	118	103	97
Cranial Flow	0	450	462	434
	100	585	605	531

TABLE 4. Effects of varying aortic compliance on coronary flow (mL/min)

Thoracic Aortic Capacitance	Abdominal Aortic Capacitance		
	10 × Normal	Normal	0.1 × Normal
10 × Normal	40	40	40
Normal	124	104	89
0.1 × Normal	167	122	111

Valvular Function

In addition to Niemann's valve, the cardiac valves were evaluated to determine whether their competence or incompetence altered flow during IAC-CPR. In general, a competent aortic valve was as effective as all four cardiac valves in maintaining cardiac output and coronary flow. Any combination of competent valves that included the aortic valve also produced maximal or nearly maximal blood flow.

DISCUSSION

By modeling a complex biological system appropriately, an investigator can explore the consequences of certain fundamental assumptions. In this 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. We found that rhythmic external pressure on "thoracic" structures could produce an artificial circulation, which was augmented substantially by the addition of rhythmic compression to the "abdominal" structures. During thoracic pump CPR, which is believed to be operative in most human patients [23], abdominal compression at 100 mm Hg caused a predictable increase in total blood flow, brain flow, and coronary flow during simulated CPR. High peripheral resistance favored flow to the heart and brain.

The results of the regression analysis presented (Figures 5-7) suggest that two independent mechanisms--a thoracic pump and an abdominal pump--can be brought into play to support the circulation during manual CPR. The action of the two mechanisms appears to be strictly additive for a given level of peripheral vascular resistance. Even when chest compression is highly effective, abdominal counterpulsation can further augment flow. When chest compression is ineffective, abdominal compression can offset the deficit in flow and increase perfusion by a large percentage. Indeed Rosborough and coworkers [26] have shown in intact dogs that blood flow can be generated during ventricular fibrillation with ventilation and abdominal compression alone.

Magnitude of Abdominal Pressure

The effectiveness of the abdominal pump mechanism depends on the magnitude of abdominal counterpressure. In the simplified electrical analog that we have studied, the relationship is a linear one, and one may infer from our results that application of very large abdominal pressures may be used to generate normal or even supranormal perfusion. Such extreme extrapolation is probably erroneous for two distinct reasons: the risk of causing abdominal trauma, and the plateau effect associated with collapse of the aorta and abdominal veins in vivo.

Considering the danger of abdominal trauma, one must realize that the total force required to produce enhanced perfusion, roughly 100 mm Hg pressure on the mid-abdomen, is nearly the same as that required to produce effective sternal compression. For example if one considers the area of two spread hands applying abdominal pressure to be 355 cm^2 (18.3 cm x 18.3 cm), the total force required to generate 100 mm Hg abdominal pressure is exactly 100 lb. As a rule of thumb, therefore, one can estimate that application of one pound of force would be required to produce 1 mm Hg manual counterpressure. Although the surface area over which this force is distributed is relatively large compared to the area of the sternum that is compressed during CPR, and although no injury to abdominal viscera has been reported in animal studies of IAC-CPR [3, 4]; application of forces in excess of 100 lb must at some point cause abdominal trauma, and also would be difficult to achieve by smaller individuals weighing less than 100 lb themselves.

A second reason for avoiding very high abdominal pressures is that beneficial flow augmentation is likely to be limited by collapse of the abdominal aorta. If one supposes that the mechanism for flow augmentation involves blood being squeezed from the abdominal aorta into peripheral vessels to provide added stroke volume, it is clear that abdominal pressure in excess of that required to empty the abdominal aorta of blood will not further improve flow. In our modeling studies, we can calculate that the increase in effective stroke volume caused by 100 mm Hg abdominal counterpressure is equal to the additional cardiac output (710 mL/min) divided by the compression rate (80/ min), or 9 mL/cycle. This volume is likely to be less than the volume contained in the abdominal aorta of an adult human being at the beginning of abdominal compression. If one were to apply much higher pressures to the abdominal compartment of the model, however, the calculated increase in stroke volume for the electrical model might be greater than the expected abdominal aortic volume, and thus it would be unlikely that flow augmentation to such a degree could be obtained in vivo. Accordingly we believe that results obtained in the model with abdominal counterpressure in the range of 100 to 120 mm Hg will correspond to the maximal flow augmentation that one can reasonably expect in clinical practice.

Myocardial and Cranial Perfusion

Perfusion of the myocardium during prolonged CPR seems to be especially important for immediate success of resuscitation. Recently one of us (SHR) has found that myocardial flow is an excellent predictor of survival after 20 minutes of CPR in dogs [7]. In particular, when myocardial flow was greater than 25 mL/min per 100 g tissue, all animals survived; when myocardial flow was less than 15 mL/min/100 g, no animal survived experimental CPR. There may be a threshold of myocardial perfusion near 20 mL/min/100 g that must be exceeded for effective resuscitation, in which case even modest improvements in coronary perfusion produced by abdominal counterpulsation could be lifesaving.

Several investigators, including Ditchey [9] and Luce [27] and their coworkers, have expressed some concern that the coronary vascular bed can be perfused at all by conventional closed-chest CPR. Granted the thoracic pump mechanism, in which systolic arterial and venous pressures are essentially equal in the aorta and right atrium, coronary flow can occur only during diastole (i.e. chest recoil). Our modeling studies confirm that with the closed-chest thoracic pump mechanism the heart can be perfused during the chest recoil phase, provided that sufficiently high intrathoracic pressure pulses are generated. However, if inadequate flow is generated by thoracic compression alone (as might occur in stiff-chested or barrel-chested individuals), abdominal counterpulsation at 100 mm Hg may improve coronary perfusion substantially. Such improvement seems especially likely when one includes in the analysis the critical closing pressure of the myocardial capillary beds. In this case the aorto-atrial pressure difference must exceed a certain critical value (10 to 15 mm Hg) before any perfusion occurs.

The importance of peripheral vasoconstrictors in enhancing coronary and cerebral perfusion, as classically described by Redding and Pearson [5, 6, 28], was confirmed emphatically in our model. Epinephrine especially, which is likely to produce coronary vasodilation as well as peripheral vasoconstriction [29], is likely to produce an effect in vivo similar to selective caudal vasoconstriction in the electrical model. In general, peripheral vasoconstrictors seem to be synergistic with abdominal compression, and combined use of the two therapeutic adjuncts is a strategy ripe for further investigation. Indeed, a study by Walker et al. [30] in dogs has demonstrated normal resting brain flows, measured by a thermal clearance technique, during experimental CPR with abdominal counterpulsation at 100 to 120 mm Hg and epinephrine infusion at 10 mg/kg/min.

Of course any physical or electrical model such as that described in our study embodies a compromise between the advantages of simplicity and the advantages of complexity. Simple models are easy to interpret and understand. They succeed if they capture the essence of the phenomena under study. The quantitative predictions of such models can always be improved by making them more complex. Our model, for example, could have been made more realistic by including the differences between systolic and diastolic compliances of the cardiac chambers and aorta, the volume dependency of venous compliance, the critical closing pressures of myocardial and other capillary beds, the partial transmission of the intrathoracic pressure to the cranial cavity with resultant effects on cerebral perfusion pressure, the partial transmission of intrathoracic pressure to the abdominal cavity through motion of the diaphragm and vice versa,

the tendency of the inferior vena cava to collapse during chest and/or abdominal compression, and the influence of endogenous catecholamines, the metabolism of which is specified as a function of blood flow through the lungs and liver. These and other refinements of the model have been suggested by well-meaning colleagues. Our own bias, however, is strongly in favor of simpler models that provide insight into physical mechanisms, because the inclusion of each refinement would have required yet another assumption, and because IAC-CPR has already been studied in live animal models that include all of the previously mentioned complexities.

CONCLUSION

Our electrical simulations identify a theoretical and physical basis for the beneficial effects of abdominal counterpulsation and identify the abdominal pump as an independent flow generating mechanism during CPR. Because preliminary trials of IAC-CPR in 20 animals [3, 4] have shown no evidence for esophageal regurgitation, liver laceration, or other abdominal trauma as a result of manual abdominal compression at 120 mm, the theoretical benefits of IAC-CPR are likely to be achievable in vivo with acceptable risk. Enhancement of vital organ perfusion seems to be independent of peculiar anatomical differences between man and experimental animals, because the electrical model is totally independent of chest geometry. If IAC-CPR can be performed in human beings with low risk of abdominal injury, it would seem to be a valid and simple means of improving the effectiveness of CPR in a manner entirely compatible with existing protocols for basic and advanced life support, and without the use of additional mechanical equipment.

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