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Abstract

Interposed abdominal compression (IAC)-CPR incorporates alternating chest and abdominal compressions to generate enhanced artificial circulation during cardiac arrest. The technique has been generally successful in improving blood flow and survival compared to standard CPR; however some questions remain. ***Objective:*** To determine "why does IAC-CPR produce more apparent benefit in some subjects than in others? and "what is the proper compression rate, given that there are actually two compressions (chest and abdomen) in each cycle?" ***Method:*** Computer models provide a means to search for subtle effects in complex systems. The present study employs a validated 12-compartment mathematical model of the human circulation to explore the effects upon systemic perfusion pressure of changes in 35 different variables, including vascular resistances, vascular compliances, and rescuer technique. CPR with and without IAC was modeled. ***Results and conclusions:*** Computed results show that the effect of 100 mmHg abdominal compressions on systemic perfusion pressure is relatively constant (about 16 mmHg augmentation). However, the effect of chest compression depends strongly upon chest compression frequency and technique. When chest compression is less effective, as is often true in adults, the addition of IAC produces relatively dramatic augmentation (e.g. from 24 to 40 mmHg). When chest compression is more effective, the apparent augmentation with IAC is relatively less (e.g. from 60 yo 76 mmHg). The optimal frequency for uninterrupted IAC-CPR is near 50 complete cycles per minute with very little change in efficacy over the range of 20 to 100 cycles/min. In theory, the modest increase in systemic perfusion pressure produced by IAC can make up in part for poor or ineffective chest compressions in CPR. IAC appears relatively less effective in circumstances when chest pump output is high.

Key words: Coronary perfusion pressure; Guidelines; Heart arrest; IAC-CPR

1. Introduction

Of the many proposed modifications of conventional external CPR, interposed abdominal compression (IAC)-CPR, has provided relatively consistent positive results in computational and animal models[1, 2] as well as in human clinical trials[3-7] over the past 25 years. IAC-CPR is performed by the application of manual mid-abdominal compressions in counterpoint to the rhythm of chest compressions during cardiac arrest and resuscitation. The abdominal compression point is over the abdominal aorta, halfway between the umbilicus and the xiphisternum. The force of abdominal compression is similar to that required for deep palpation of the aortic pulse in a normal patient, which generates about 100 mmHg internally in the vicinity of the abdominal aorta[8]. By invoking a separate "abdominal pump" mechanism[9], which

is independent of the chest pump mechanism, IAC augments blood perfusion between 50 and 150 percent compared to standard CPR with chest compression only[10, 11]. Remarkably, this increase in blood flow during CPR appears to translate into a rough doubling of resuscitation success in the clinical studies of Sack and coworkers[4, 5]. These investigators found that when IAC-CPR is applied from the beginning of an arrest sequence in randomized patients in a hospital, both immediate survival and long term, neurologically intact survival roughly double.

Despite these generally positive results, a few studies have found no significant difference in hemodynamics between standard and IAC-CPR[12-15]. Indeed one of the original descriptions of IAC-CPR in anesthetized dogs[16] showed a wide range of effects, in which increases in cardiac output ranged from about 10 percent to over 200 percent with the addition of IAC. In a deterministic universe, one would expect that there are some still unappreciated variables or underlying physiologic states that make IAC-CPR more or less effective compared to standard CPR. Identifying these unappreciated factors could allow for more rational application of this promising resuscitation method through improved patient selection, adjunctive drug therapy, or modified technique.

A search for unappreciated critical variables can be done easily using computer models of the circulation, which have become increasingly used in resuscitation research and which are capable of producing results generally congruent with animal and clinical studies[17]. Although obviously lacking in blood-and-guts biology, such models are independent of many confounding factors present in laboratory studies and in clinical trials. These include varying patient populations, cardiac arrest time, drug therapy, underlying disease, chest configuration, and body size, as well as varying rescuer size, skill, strength, consistency, prior training, and bias. Mathematical models also allow exact control of the dominant hemodynamic mechanism of CPR (thoracic pump in large subjects, vs. cardiac pump in small subjects[9, 18]) and are especially suited to exhaustive manipulation of selected variables, while the remaining variables are precisely controlled.

The objective of the present study is to systematically vary 35 parameters in a complex computational model of the human circulation in order to identify situations where IAC is especially effective or especially ineffective compared to standard CPR.

2. Methods

The computational model used here (Figure 1) is an adaptation of that previously published for resuscitation research by this author[19]. The number of vascular compartments is increased from 7 to 12 in order to include explicitly the anatomic details of the pulmonary circulation and a 4-chambered heart (Figure 2). Assumptions are limited to normal human anatomy and physiology, the definition of compliance (volume change / pressure change), and Ohm's Law (flow = pressure / resistance). The model was solved using Microsoft Visual Basic to perform numerical integration of coupled differential equations describing incremental pressure changes in each vascular compartment.

2.1. Anatomical parameters

The human circulatory system is represented by twelve compliant chambers, connected by resistances through which blood may flow, as shown in Figures 1 and 2. Definitions of subscripts indicating particular vascular structures are provided in Table 1. The compliances correspond to the thoracic aorta, abdominal aorta, carotid arteries, femoral arteries, jugular veins, leg veins, superior vena cava, inferior vena cava, right ventricle, pulmonary arteries, pulmonary veins, and left ventricle. The structures within the chest compartment (right heart, pulmonary vascular, and left heart compliances) may be configured to function either as a heart-like cardiac pump, in which cardiac muscle or external pressure squeezes blood from the ventricles through the pulmonic and aortic valves, or as a global thoracic pressure pump, in which applied pressure squeezes blood from the pulmonary vascular bed, through the left heart, and into the periphery[18, 20, 21]. Combinations of these two hemodynamic mechanisms can be modeled as well.

Conductance pathways with non-zero resistances, R, connect the vascular compartments. The values of R_h, R_{ht}, R_s and R_l are large and represent resistances of the systemic vascular beds of the head, heart, trunk, and legs. R_c, R_a, R_j, R_{ia}, R_{iv}, and R_v are small and represent in-line resistances of the great vessels. R_{pc} is intermediate in value and represents the resistance of the pulmonary capillary bed, which is much less than that of the systemic vascular bed. Also included are the small resistances, R_{tv}, R_{pv}, R_{mv}, R_{av}, which represent the inflow and outflow resistances of the tricuspid, pulmonic, mitral, and aortic valves of the heart. Niemann's valves between the chest and jugular veins at the level of the thoracic inlet are actual, but little known anatomic structures that function to block headward transmission of large positive pressure pulses in the chest during cough and also during CPR[22].

2.2. Physiological parameters

Parameters describing a textbook normal "70 Kg man"[23] are used to specify values of the compliances and resistances in Figures 1 and 2. The normal 30-fold ratio of venous to arterial compliance characterizes a circulation in the absence of fluid loading or congestive heart failure. The distribution of vascular conductances (1/Resistances) into cranial, thoracic, and caudal components reflects textbook distributions of cardiac output to various body regions. Details of the rationale for selection of resistance and compliance values are provided in references[9, 23, 24]. The normal diastolic compliance of the left ventricle was taken from Greger and Windhorst's comprehensive textbook of physiology[25]. The diastolic compliance of the right ventricle was estimated as twice that of the left ventricle.

2.3. Solving for pressures in the system

The relationships among the pressures in the various vascular compartments are determined by the definition of compliance and by Ohm's Law. The definition of compliance is $C = \Delta V / \Delta P$, where C is compliance, and ΔP is the incremental change in pressure within a compartment as volume ΔV is introduced. Ohm's Law, which relates flow to pressure and resistance, is

$i = \frac{1}{R}(P_1 - P_2)$, where $P_1 - P_2$ is the instantaneous difference in pressure across resistance R as flow i occurs. In Figure 1 currents i_c (carotid), i_a (aortic), i_s (systemic), i_v (venous), i_j (jugular), i_{ia} (iliac artery), i_l (legs), i_{iv} (iliac veins), i_i (pump input), and i_o (pump output) are shown for clarity, with positive directions specified by arrows. In Figure 2 flows across each heart valve and the pulmonary vascular resistance are shown.

2.3.1. Extrathoracic components

Applying these basic concepts with reference to Figure 1 provides a set of governing finite difference equations that can be used to describe hemodynamics. These equations can be integrated numerically to describe instantaneous pressure vs. time waveforms in each compartment. Beginning, for example, with the abdominal aorta

$$\begin{aligned} \Delta P_{aa} &= \Delta P_{abd} + \frac{1}{C_{aa}}(i_a - i_s - i_{ia})\Delta t \\ &= \Delta P_{abd} + \frac{\Delta t}{C_{aa}}\left[\frac{1}{R_a}(P_{ao} - P_{aa}) - \frac{1}{R_s}(P_{aa} - P_{ivc}) - \frac{1}{R_{ia}}(P_{aa} - P_{fa})\right] . \end{aligned} \tag{1}$$

Here ΔP_{abd} represents the change in pressure applied to outer surfaces of blood vessels in the abdominal compartment during manual abdominal compressions. For simulation of the normal circulation or simulation of standard CPR the value of ΔP_{abd} is zero. The next term represents the increase in abdominal aortic pressure caused by net inflow of blood during the small time interval Δt. Substitution for currents, i_a , i_s , and i_{ia}, using Ohm's Law completes the expression.

Similarly, the pressure changes in other extrathoracic vascular compartments are given by expressions (2) through (6), as follows.

$$\begin{aligned} \Delta P_{ivc} &= \Delta P_{abd} + \frac{1}{C_{ivc}}(i_s - i_v + \max(i_{iv}, 0))\Delta t \\ &= \Delta P_{abd} + \frac{\Delta t}{C_{ivc}}\left[\frac{1}{R_s}(P_{aa} - P_{ivc}) - \frac{1}{R_v}(P_{ivc} - P_{rh}) + \max\left(\frac{1}{R_{iv}}(P_{fv} - P_{ivc}), 0\right)\right] \end{aligned} \tag{2}$$

In (2) the max() function is used to represent the action of venous valves in the femoral and iliac veins that prevent retrograde flow. Max(x, y) is the larger of x or y.

$$\Delta P_{car} = \frac{1}{C_{car}}(i_c - i_h)\Delta t = \frac{\Delta t}{C_{car}}\left[\frac{1}{R_c}(P_{ao} - P_{car}) - \frac{1}{R_h}(P_{car} - P_{jug})\right] \tag{3}$$

$$\Delta P_{jug} = \frac{1}{C_{jug}}(i_h - i_j)\Delta t = \frac{\Delta t}{C_{jug}}\left[\frac{1}{R_h}(P_{car} - P_{jug}) - N\frac{1}{R_j}(P_{jug} - P_{rh})\right], \quad (4)$$

where the Niemann's valve function N=1 normally, and N=0 during cough or intrathoracic pressure pulses (when $P_{rh} > P_{jug}$). Thus the max() function can be used as in expression (2) to implement the one-way valve action of N.

The legs are represented as follows.

$$\Delta P_{fa} = \frac{1}{C_{fa}}(i_{fa} - i_l)\Delta t = \frac{\Delta t}{C_{fa}}\left[\frac{1}{R_{ia}}(P_{aa} - P_{fa}) - \frac{1}{R_l}(P_{fa} - P_{fv})\right] \quad (5)$$

$$\Delta P_{fv} = \frac{1}{C_{fv}}(i_l - \max(i_{iv}, 0))\Delta t = \frac{\Delta t}{C_{fv}}\left[\frac{1}{R_l}(P_{fa} - P_{fv}) - \max\left(\frac{1}{R_{iv}}(P_{fv} - P_{ivc}), 0\right)\right] \quad (6)$$

2.3.2. Thoracic components

Corresponding expressions for the blood containing thoracic structures (Figure 2) are as follows. For the thoracic aorta

$$\begin{aligned}\Delta P_{ao} &= f_{tp}\Delta P_{chest} + \frac{1}{C_{ao}}(i_o - i_c - i_a - i_{ht})\Delta t \\ &= f_{tp}\Delta P_{chest} + \frac{\Delta t}{C_{ao}}\left[E\frac{1}{R_o}(P_{lv} - P_{ao}) - \frac{1}{R_c}(P_{ao} - P_{car}) - \frac{1}{R_a}(P_{ao} - P_{aa}) - \frac{1}{R_{ht}}(P_{ao} - P_{rh})\right],\end{aligned} \quad (7)$$

where f_{tp} is the thoracic pump factor, E = 1 during ejection (aortic valve open), and E = 0 otherwise.

For the superior vena cava and right atrium ("right heart")

$$\begin{aligned}\Delta P_{rh} &= f_{tp}\Delta P_{chest} + \frac{1}{C_{rh}}(i_j + i_v + i_{ht} - i_i)\Delta t \\ &= f_{tp}\Delta P_{chest} + \frac{\Delta t}{C_{rh}}\left[N\frac{1}{R_j}(P_{jug} - P_{rh}) + \frac{1}{R_v}(P_{ivc} - P_{rh}) + \frac{1}{R_{ht}}(P_{ao} - P_{rh}) - F\frac{1}{R_i}(P_{rh} - P_{rv})\right],\end{aligned} \quad (8)$$

where F = 1 during the filling phase (tricuspid valve open) and F = 0 otherwise, and N = 0 during CPR-induced intrathoracic pressure pulses (Niemann's valve closed) and N = 1 otherwise.

Here the thoracic pump factor, $0 \le f_{tp} \le 1$, is used to model the effects of the thoracic pump mechanism in external CPR, as explained in Section 2.4.3. When $f_{tp} = 0$, chest pressure is applied to the cardiac ventricles only (the cardiac pump mechanism). When $f_{tp} > 0$, then 100 times f_{tp} percent of chest pressure is applied also to the thoracic aorta, thoracic vena cava, atria, pulmonary arteries, and pulmonary veins.

For the right ventricle

$$\begin{aligned} \Delta P_{rv} &= \Delta P_{chest} + \frac{1}{C_{rv}}(i_i - i_2)\Delta t \\ &= \Delta P_{chest} + \frac{\Delta t}{C_{rv}}\left[F\frac{1}{R_{tv}}(P_{rh} - P_{rv}) - E\frac{1}{R_{pv}}(P_{rv} - P_{pa}) \right]. \end{aligned} \tag{9}$$

For the pulmonary arteries

$$\Delta P_{pa} = f_{tp}\Delta P_{chest} + \frac{1}{C_{pa}}(i_2 - i_3)\Delta t = f_{tp}\Delta P_{chest} + \frac{\Delta t}{C_{pa}}\left[E\frac{1}{R_{pv}}(P_{rv} - P_{pa}) - \frac{1}{R_{pc}}(P_{pa} - P_{pv}) \right]. \tag{10}$$

For the pulmonary veins

$$\Delta P_{pv} = f_{tp}\Delta P_{chest} + \frac{1}{C_{pv}}(i_3 - i_4)\Delta t = f_{tp}\Delta P_{chest} + \frac{\Delta t}{C_{pv}}\left[\frac{1}{R_{pc}}(P_{pa} - P_{pv}) - F\frac{1}{R_{mv}}(P_{pv} - P_{lv}) \right]. \tag{11}$$

Finally, for the left ventricle

$$\begin{aligned} \Delta P_{lv} &= \Delta P_{chest} + \frac{1}{C_{lv}}(i_4 - i_o)\Delta t \\ &= \Delta P_{chest} + \frac{\Delta t}{C_{lv}}\left[F\frac{1}{R_{mv}}(P_{pv} - P_{lv}) - E\frac{1}{R_{av}}(P_{lv} - P_{ao}) \right]. \end{aligned} \tag{12}$$

2.4. Numerical methods

2.4.1. Integration

The Visual Basic programming language and Microsoft Excel spreadsheets are ideal for implementing numerical integration of expressions (1) through (12) to obtain pressures in all twelve vascular compartments as a function of time. One can create a Visual Basic procedure, in which pressures in each compartment at any point in time are computed from the pressures at the preceding time point and the corresponding ΔP's—that is

$$P(t + \Delta t) = P(t) + \Delta P(t) \quad (13)$$

To model a cardiac arrest and CPR one begins with a uniform pressure such as 5 mmHg in all compartments of the arrested circulation and applies phased pressure pulses internally to the vascular structures of the chest and abdomen. To model the normal circulation as a control, one applies pressures to the cardiac ventricles only. The incremental changes in compartment pressures computed from expressions (1) through (12) are used to construct a marching solution for successive small increments of time, Δt, typically 0.001 sec. Use of a time increment that is too coarse results in unstable oscillations of computed pressures. Decreasing the value of Δt, however, can always return stability.

2.4.2. Pressure waveforms applied to vessels in the chest and abdomen

The input parameters $P_{max\text{-}chest}$ and $P_{max\text{-}abd}$ represent peak external pressures applied independently to the chest vessels and to the abdominal vessels as a result of external compression. These are peak pressures that act directly upon the outer surfaces of blood vessels in the model. In this study half sinusoidal waveforms were used. Between half sinusoidal compressions $\Delta P_{chest} = 0$ and $\Delta P_{abd} = 0$.

2.4.3. Thoracic vs. cardiac chest pump mechanisms

To explore the influence of the alternative pump mechanisms that can impel blood during cardiac arrest and chest compression, the thoracic pump factor, $0 \le f_{tp} \le 1$, is used. A pressure equal to the product of P_{chest} and f_{tp} is applied to intrathoracic compartments other than the right and left ventricles to create a continuum of hybrid pump mechanisms ranging from pure cardiac pump ($f_{tp} = 0$) to pure thoracic pump ($f_{tp} = 1$). When $f_{tp} = 1$ all intrathoracic structures, including the great veins and thoracic aorta, experience a uniform "global" intrathoracic pressure rise, as originally conceived by Weisfeldt, Rudikoff and coworkers [26]. When $f_{tp} = 0$, only the ventricular compliances, C_{rv}, C_{lv}, are pressurized, as if the heart alone, and not the great vessels, were compressed between the sternum and the spine, as originally conceived by Kouwenhoven, Jude, and Knickerbocker [27]. Intermediate values of the thoracic pump factor allow models approximating the current understanding[21, 28, 29], in which for small animals and children blood is impelled predominantly by the cardiac pump mechanism (for example, $f_{tp} \approx 0.25$), whereas in larger animals and adult humans blood is impelled predominantly by the thoracic pump mechanism (for example, $f_{tp} \approx 0.75$ in the "normal adult" model used as a reference).

2.4.4. Model output

The output of the model is a multi-channel record of pressure as a function of time. Standard "normal" values of model parameters are given in Table 2 and in references[9, 24]. Because of the high venous pressures that can occur during CPR or external pulsation, systemic perfusion pressure (SPP = thoracic aortic pressure minus right atrial pressure) is used to describe

hemodynamic benefit. Mean systemic perfusion pressure is the figure of merit used in the present study. Mean SPP was computed for the 21st compression cycle after onset of CPR to allow for steady state conditions to develop.

2.4.5. Test cases and validation

The spreadsheet code was validated by solving simple test cases for very small or very large values of the resistances or compliances and also by establishing a model of the normal adult circulation using $f_{tp} = 0$. This model had an aortic blood pressure of 120/82 mmHg and a cardiac output of 5.1 L/min for a heart rate of 80/min, closely approximating the textbook normal values of 120/80 mmHg and 5.0 L/min.

3. Results

The benefit of IAC is defined as the percentage improvement in mean systemic perfusion pressure when abdominal compressions are turned on in the model and all other variables are held constant. To identify variables in the model that cause the greatest changes in the benefit of IAC, each variable in turn was incremented by 10 percent of its initial normal value and the mean systemic perfusion pressure with and without IAC computed. The sensitivity is defined as the percentage change in the benefit of IAC produced by a 10 percent change in any particular variable. Table 3 shows the results of the sensitivity analysis for variables with sensitivity greater than ±1 percent.

Of the 35 variables studied, including vascular compliances, vascular resistances, and rescuer performance factors, 29 had little effect upon the benefit of IAC. When duty cycle (chest compression time divided by cycle time) was increased from 50 to 55 percent, systemic perfusion pressure decreased by less than 1 percent. When pathological changes in vasomotor tone were simulated by simultaneously increasing vascular resistance and decreasing vascular compliance (making vessels stiffer) throughout the model, as would occur with epinephrine treatment, there was no change in the benefit of IAC. The remaining 6 variables (Table 3) had more substantial effects. When the resistance of the inferior vena cava connecting the abdominal and chest compartments increased by 10 percent, the benefit of turning on IAC decreased by 3 percent. This effect is quite reasonable, because venous return from abdomen to chest is needed for IAC to prime the chest pump. Similarly when right heart compliance increased (i.e. the right heart became stiffer and harder to fill), the benefit of IAC diminished. Right heart compliance might diminish when the right heart becomes engorged with venous blood late in an arrest sequence. It could similarly change with fluid overload or in rare disease states such as constrictive pericarditis. More interesting are the remaining critical variables.

The relative benefit of IAC changes negatively with increasing frequency, which is clearly under control of the rescuer. The full effect of this variable is shown in Figure 3. Here we see that IAC-CPR generates a nearly constant high perfusion pressure across compression rates, while standard CPR shows a decelerating rate effect. The overall maximal effect of IAC is near 40 to 50 complete cycles per minute, including both 40 to 50 chest compressions and the same number

of interposed abdominal compressions. Compared to standard CPR, the relative benefit of IAC is greater at lower compression rates. The results for standard CPR are similar to those reported by Fitzgerald and coworkers[30], the only comprehensive study of chest compression frequency in vivo known to the present author.

The thoracic pump factor has the greatest influence on the benefit of IAC-CPR of any model variable. Details are shown in Figure 4. A thoracic pump factor of 0 indicates pure cardiac compression mechanism. A thoracic pump factor of 1.0 indicates a pure thoracic pump mechanism. Intermediate values indicate hybrid mechanisms. In this model IAC adds a relatively fixed amount of systemic perfusion pressure to whatever chest pump mechanism is operative. Since the cardiac pump mechanism is more effective in producing forward flow, the added benefit of IAC is relatively small when the f_{tp} is zero. This situation would tend to occur in a small child with a compliant chest. Although nearly the same in absolute terms, the benefit of IAC is relatively much greater in when the $f_{tp} = 1$. This situation would tend to occur in an emphysematous or barrel chested adult.

In a similar vein, the maximal internal pressure generated by chest compression ($P_{max\text{-}chest}$) influences the relative benefit of IAC-CPR (Figure 5). As chest compression becomes more vigorous and effective in generating peak intrathoracic pressure, the relative benefit of IAC augmentation becomes less pronounced. IAC does, however, offer benefit at all chest pressures.

Finally, there is the obvious dependence of the benefit of IAC on abdominal compression pressure itself. Clearly, very small abdominal pressures produce less benefit than larger ones. In the present mathematical model, which is a quasi-linear system (except for the cardiac valves), this benefit of greater abdominal pressure increases indefinitely. In an actual person there is a maximal benefit of IAC, which is limited by the "stroke volumes" of the abdominal aorta and abdominal veins, namely the volumes that can be squeezed out by external compression. The present simulations were conducted using a practical value of 100 mmHg for peak abdominal pressure for IAC. This value appears from other research to be close to maximally effective and is also tolerable in an awake, conscious person[31].

4. Discussion

The absolute augmentation of systemic perfusion pressure produced by 100 mmHg interposed abdominal compressions is insensitive to changes in conditions. However, the relative benefit of IAC-CPR compared to standard CPR varies across physiologic states of the victim and across rescuer techniques, because the effectiveness of standard CPR changes. The effectiveness of the chest pump is limited by chest size and stiffness and also by the ability of external compressions to invoke the cardiac pump mechanism, which is more effective than the thoracic pump mechanism. Thus IAC can provide added benefit in any resuscitation, but the effects would be most noticeable when the chest pump mechanism is less effective. Lower chest pump output occurs at lower compression rates and also in larger, more barrel chested adults or other persons in whom the thoracic pump mechanism dominates.

For various computational, animal, and clinical models, the addition of IAC to otherwise standard CPR produces a modest benefit of roughly twofold increase in artificial forward blood flow[7]. In a 70 kg man, this benefit would correspond to an increase from roughly 1 L/min forward flow to roughly 2 L/min forward flow during CPR. The effective "stroke volume" of the abdominal aorta may determine the benefit of the abdominal pump in vivo. For example, if the compression rate is 60/min and the additional forward flow from abdominal compression is 1000 ml/min, the stroke volume of the abdominal aorta is about 16 ml/compression, which is anatomically reasonable for an adult human. A similar argument can be made for the enhanced chest pump filling on the venous side. Since it is easier to compress the abdomen than the chest, this modest benefit of IAC can be expected regardless of the size, strength, or fatigue of the rescuers. In contrast, the effectiveness of conventional sternal compressions can vary greatly with small changes in compression depth[32]. Such changes can occur as a rescuer doing manual chest compressions tires. We now know from the present study that the benefit of chest compressions also depends upon compression frequency and upon which hemodynamic mechanism (cardiac pump vs. thoracic pump) is operative within the chest. The modest but consistent augmentation of perfusion pressure IAC can make up to some degree for the relative inconsistency of chest compressions in practical resuscitations.

Another important result of the present modeling exercise is the observation that there is little influence of compression frequency in IAC-CPR. The optimal continuous compression rate for IAC-CPR is near 40 to 50 complete cycles/min, including 40 to 50 abdominal compressions and the same number of chest compressions. This regime is practical and much less exhausting for rescuers than faster rates. It is also graceful for incorporation into current standards, which specify 80 to 100 compressions per minute for standard CPR. For IAC-CPR the recommended number of total compressions (both chest and abdominal) would be the same. Also, the observation that blood flow with IAC-CPR is not strongly dependent on compression frequency, which has been observed before in models[9, 33], makes the technique more forgiving and user friendly. Here only continuous thoraco-abdominal compression, without ventilatory pauses, was modeled. For detailed discussion of the deleterious effects of ventilatory pauses on SPP, please see references[34-37].

In conclusion, IAC tends to add a constant increment to systemic perfusion pressure during CPR. The benefit of IAC-CPR, therefore, appears to be relatively less when chest pump output is high. The observation that a small minority of animal and clinical studies of IAC-CPR vs. standard CPR found no statistically significant benefit[7] does not necessarily mean that IAC-CPR is perhaps ineffective and that more research is needed. A better interpretation of the literature may be that in these few studies chest compression was done especially well.

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Table 1: Nomenclature

Symbol	Definition
Subscripts	
aa	Abdominal aorta
a	Aorta at level of diaphragm
ao	Thoracic aorta
av	Aortic valve
C, car	Carotid
fa	Femoral artery
fv	Femoral vein
h	Head
ht	Heart
ia	Iliac artery
iv	Iliac vein
ivc	Inferior vena cava
j, jug	Jugular
l	Legs
lv	Left ventricle
mv	Mitral valve
pa	Pulmonary arteries
pc	Pulmonary capillaries
pv	Pulmonic valve
rh	Right heart and superior vena cava
rv	Right ventricle
s	Systemic circulation below diaphragm
tv	Tricuspid valve
v	Portal and systemic veins at level of diaphragm
Variables	
C	Compliance (L/mmHg or ml/mmHg)
E	Pump ejection valve (1=open, 0=closed)
F	Pump filling valve (1=open, 0=closed)
i	Flow or current between compartments (L/sec or L/min)
N	Niemann's valve (1=open, 0=closed)
P	Instantaneous pressure in a compartment (mmHg)
ΔP	Pressure increment during time Δt (mmHg)
R	Resistance (mmHg/(L/sec))
t	Time during a cycle of CPR (sec)
Δt	Time increment (sec)
f_{tp}	Thoracic pump factor (0 – 1)

Table 2: Model parameters

Resistances (>0)

	Value (mmHg/L/sec)	Definition
Rc	60	Resistance of both carotid arteries
Rh	5520	Resistance of the head vasculature
Rj	30	Resistance of both jugular veins
Rtv	5	Resistance of the tricuspid valve
Rpv	10	Resistance of the pulmonic valve
Rmv	5	Resistance of the mitral valve
Rav	10	Resistance of the aortic valve
Rpc	120	Resistance of the pulmonary capillary bed
Rht	15780	Resistance of coronary vessels (heart)
Ra	25	Resistance of the aorta
Rv	25	Resistance of the inferior vena cava
Rs	1800	Resistance of residual systemic vasculature
Ria	360	Resistance of both iliac arteries
Riv	180	Resistance of both iliac veins
Rl	8520	Resistance of leg vasculature

Compliances (>0)

	Value (L/mmHg)	Definition
Crv	0.016	Compliance of the arrested right ventricle
Cpa	0.0042	Compliance of the pulmonary arteries
Cpv	0.0128	Compliance of the pulmonary veins AND left atrium
Clv	0.008	Compliance of the arrested left ventricle
Ccar	0.0002	Compliance of both carotid arteries
Cjug	0.012	Compliance of both jugular veins
Cao	0.0008	Compliance of the thoracic aorta
Crh	0.0095	Compliance of the right atrium and intrathoracic great veins
Caa	0.0004	Compliance of the abdominal aorta
Civc	0.0234	Compliance of the inferior vena cava
Cfa	0.0002	Compliance of both femoral arteries
Cfv	0.0047	Compliance of both femoral veins

Other variables

	Value and units	Definition
Frequency	60/min	Number of cycles per minute for uninterrupted chest and abdominal compression
Duty cycle	0.5	Fraction of cycle time for chest compression
Vasomotor tone	---	Increased vascular resistance and decreased vascular compliance throughout the model
f_{tp}	0.75	Thoracic pump factor (0.75 = adult, 0.25 = child, 1.0 = emphysema, 0 = open chest)
Pinit	5 mmHg	Initial equilibrium pressure of arrested circulation
Pmax-chest	100 mmHg	Maximum chest pressure
Pmax-abd	0 mmHg	Maximum abdominal pressure

Table 3: Sensitivity analysis*.

Parameter	STD SPP (mmHg)	IAC SPP (mmHg)	Benefit (%)	Sensitivity* (%)
Reference	30.7	48.2	56.8	
R_v	30.5	46.9	53.8	-3.0
C_{rh}	30.5	46.9	53.8	-3.0
frequency	31.6	48.7	53.9	-2.9
f_{tp}	26.7	45.0	68.8	11.9
$P_{max\text{-}chest}$	33.8	51.0	50.1	-5.9
$P_{max\text{-}abd}$	30.7	50.1	63.1	6.4

* Sensitivity = percentage change in the benefit of IAC produced by a 10 percent increase the in indicated parameter. The benefit of IAC is defined as the percentage improvement in systemic perfusion pressure with standard parameter values (Table 2). Only parameters with sensitivity more than ±1 percent different from zero are shown.

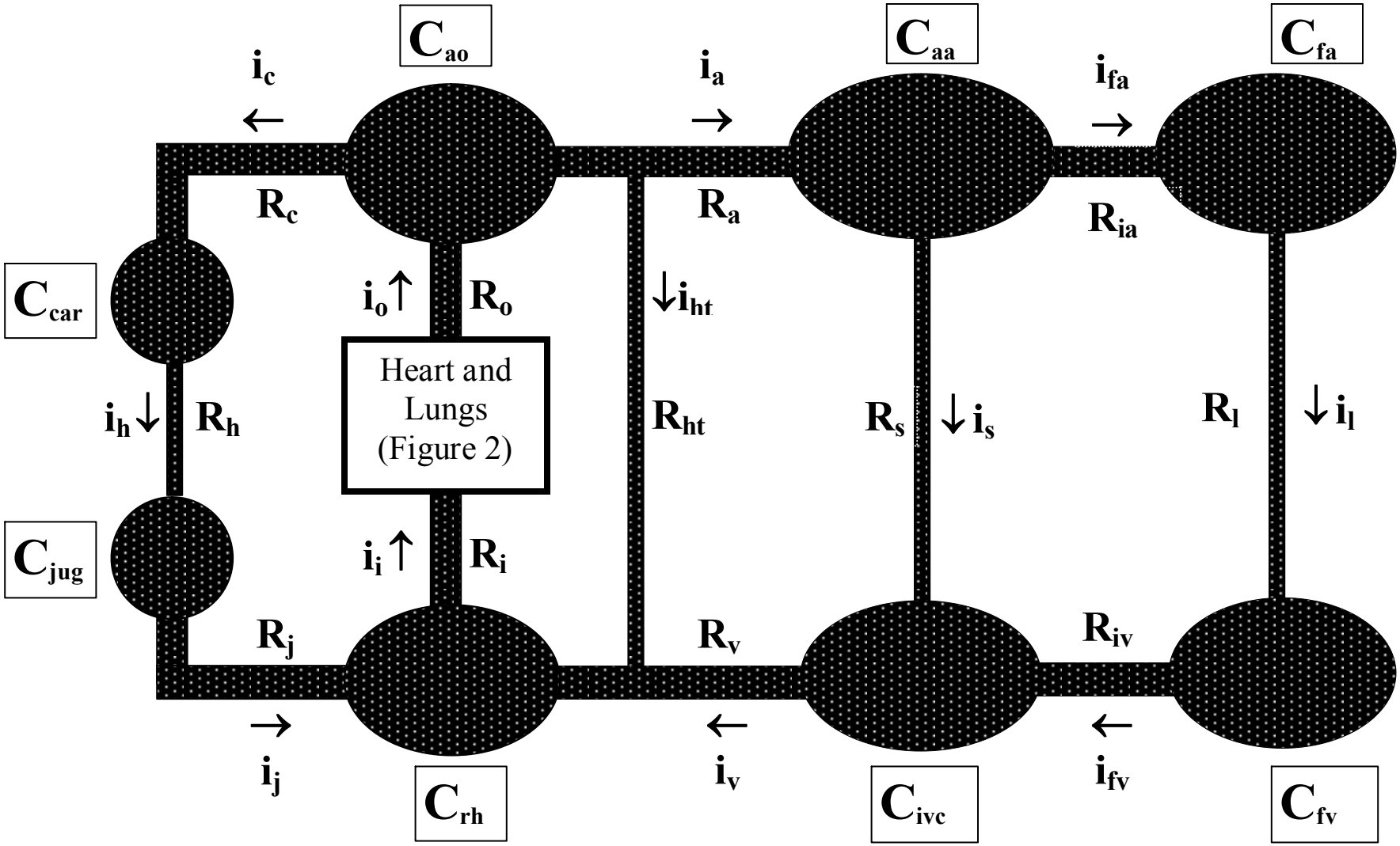

Figure 1. Model of the human circulatory system.

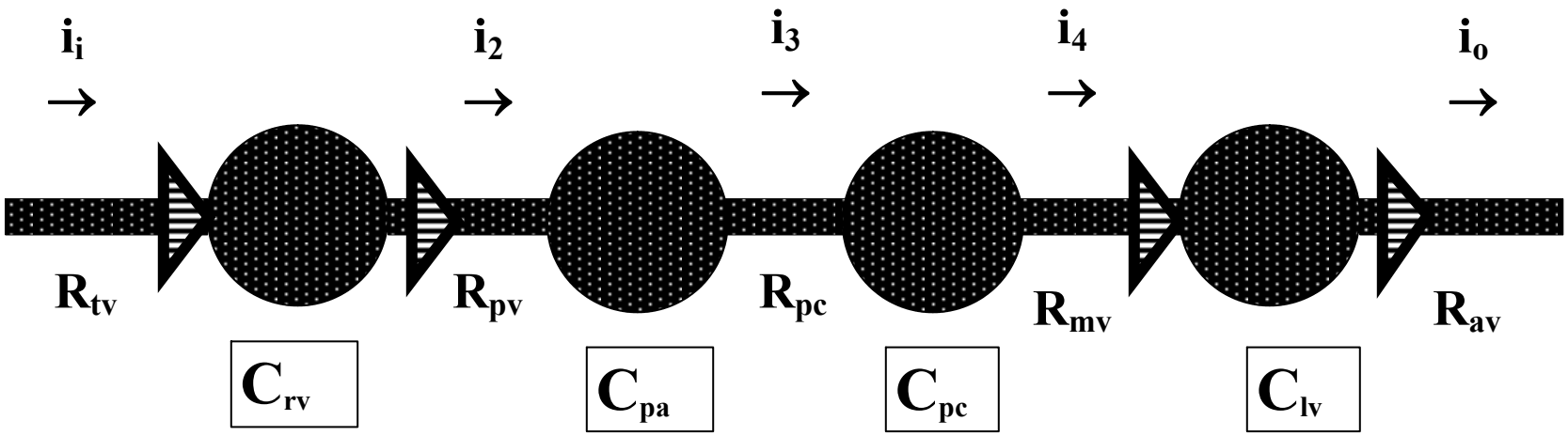

Figure 2. Detailed components within the chest. Triangles indicate heart valves.

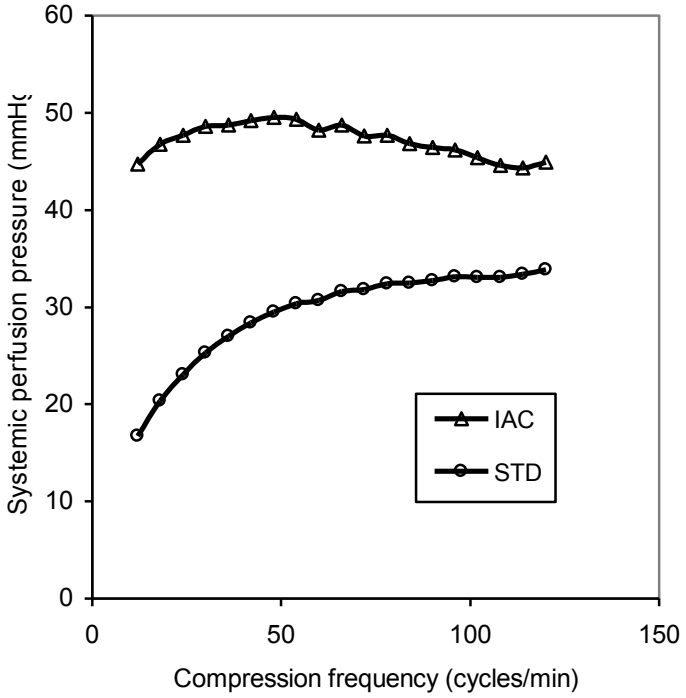

Figure 3. Systemic perfusion pressure for IAC-CPR and standard CPR as a function of compression frequency. Thoracic pump factor is 0.75. Other parameters as in Table 2.

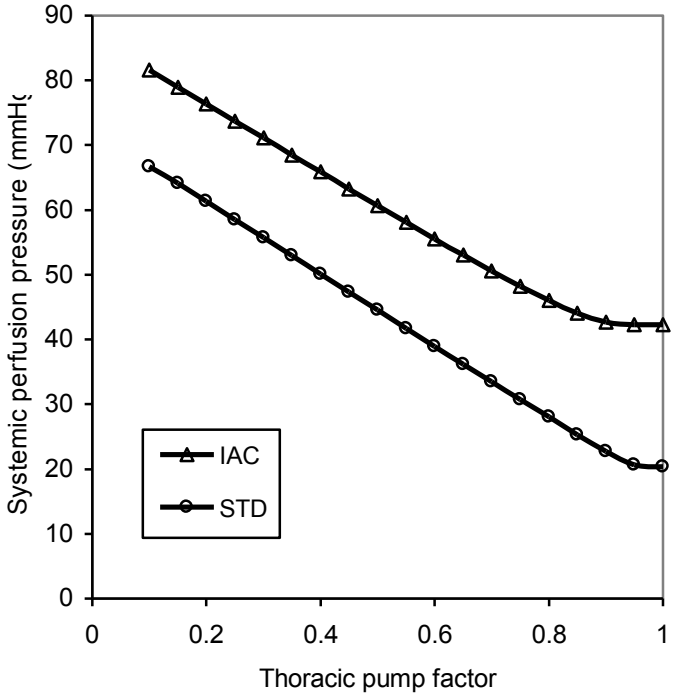

Figure 4. Systemic perfusion pressure for IAC and standard CPR as a function of the thoracic pump factor. Other parameters as in Table 2.

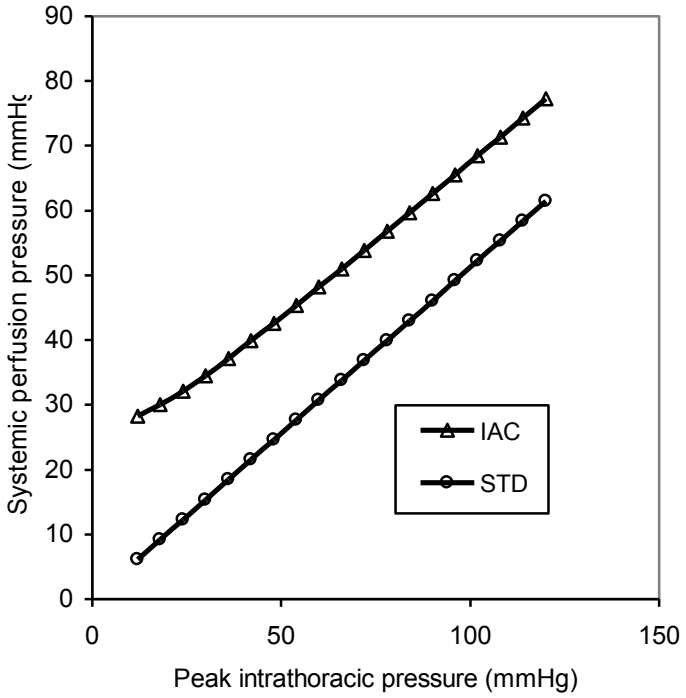

Figure 5. Systemic perfusion pressure for IAC and standard CPR as a function of the peak internal pressure generated by chest compression. Thoracic pump factor is 0.75. Other parameters as in Table 2.