CPR Techniques That Combine Chest and Abdominal Compression and Decompression

Hemodynamic Insights From a Spreadsheet Model

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Background—This study was done to elucidate mechanisms by which newer cardiopulmonary resuscitation (CPR) techniques, including interposed abdominal compression (IAC), active compression-decompression (ACD), and Lifestick CPR, augment systemic perfusion pressure and forward flow and to compare the 3 techniques in the same test system.

Methods and Results—Mathematical models describing hemodynamics of the adult human circulation during cardiac arrest and CPR were created and exercised by use of spreadsheet software. Assumptions of the models are limited to normal human anatomy and physiology, the definition of compliance (volume change/pressure change), and Ohm’s law (flow = pressure/resistance). Standard CPR generates 1.3 L/min forward and 25 mm Hg systemic perfusion pressure. In otherwise identical models, IAC-CPR generates 2.4 L/min and 45 mm Hg; ACD-CPR, 1.6 L/min and 30 mm Hg; and Lifestick CPR, which combines IAC and ACD, 3.1 L/min and 58 mm Hg. Augmented CPR techniques work by enhanced priming of either chest or abdominal pump mechanisms.

Conclusions—Adjunctive maneuvers, combined with conventional chest compression, can produce substantial hemodynamic benefit in CPR by credible physiological mechanisms. (Circulation. 1999;100;2146–2152.)

Key Words: blood flow ▪ cardiopulmonary resuscitation ▪ computers ▪ heart arrest ▪ Lifestick ▪ mechanics

Two mechanical techniques have emerged from animal and clinical studies as potentially effective means of augmenting perfusion during external cardiopulmonary resuscitation (CPR). The first is the addition of interposed abdominal compression (IAC) to otherwise standard CPR.1–3 The second is CPR with active compression and decompression (ACD) of the chest.4–6 Reviews of these techniques are found in References 7 and 8. During IAC-CPR, positive pressure is applied to the abdomen in counterpoint to the rhythm of chest compression, so that the abdomen is being compressed when chest pressure is relaxed. During ACD-CPR, positive and negative pressures are applied alternately to the chest by means of a “plunger” that forms a seal with the anterior chest wall. Both methods improve hemodynamics in animal studies of electrically induced ventricular fibrillation.9,10 Both improve CO2 excretion as a measure of effective systemic perfusion in human resuscitation.3,11,12 Three randomized clinical trials of IAC-CPR compared with standard CPR2,11,13 have found statistically significant benefit, and 1 early trial found no difference.14 Four randomized clinical trials of ACD-CPR have found improved outcome.5,6,15,16 and 4 other trials have found no difference.17–20 Most recently, Lifestick CPR21 has become the subject of active research. The Lifestick is a 2-handed device that is able to apply IAC and ACD-CPR simultaneously by alternately compressing and decompressing the chest and the abdomen through adhesive pads. These 3 CPR adjuncts are illustrated in Figure 1.

Using a mathematical model of CPR hemodynamics, the nomenclature for which is given in Table 1, the author has explored the possibility that IAC, ACD-CPR, and Lifestick CPR work by similar mechanisms. The hypothesis is that improved filling of the thoracic aorta and right heart can be accomplished either by positive pressure in the abdomen or by negative pressure in the chest and conversely, that improved filling of the abdominal aorta can be achieved either by positive pressure in the chest or by negative pressure in the abdomen. Improved pump filling, in turn, leads to higher stroke output, systemic perfusion pressure, and systemic blood flow.

The approach to the present research was to create and test a mathematical model of CPR hemodynamics based on fundamental principles of cardiovascular physiology. Such a model is independent of the many confounding factors present in laboratory studies and in clinical trials. These include varying patient populations, downtime, drug therapy, central venous pressure, peripheral vascular resistance, underlying disease, chest configuration, and body size, as well as varying rescuer size, skill, strength, consistency, prior

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training, and bias. Mathematical models also allow exact control of the dominant hemodynamic mechanism of CPR (thoracic pump in large subjects versus cardiac pump in small subjects). This approach facilitates quantitative comparison of various resuscitation techniques in exactly the same test system.

**Methods**

**Anatomic Parameters**

To capture the essence of CPR hemodynamics, one may solve the family of differential equations describing pressures and flows in the simplified fluidic system shown in Figure 2. The human circulation is represented by 7 compliant chambers connected by resistances through which blood may flow. The compliances correspond to the thoracic aorta, abdominal aorta, lumped superior vena cava and right heart, lumped abdominal and lower extremity veins, carotid arteries, and jugular veins. In addition, the chest compartment contains a pump representing the pulmonary vascular and left heart compliances between the pulmonic valve and the aortic valve. This pump may be configured to function either as a heartlike cardiac pump, in which applied pressure squeezes blood from the heart itself through the aortic valve, 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. Conductance pathways with nonzero resistances, R, connect the elastic compartments. R, R, and R are large and represent resistances of the systemic vascular beds of the head, heart, and trunk and legs. R, R, and R are small and represent in-line resistances of the great vessels. R and R are the small input and output resistances of the chest pump in series with the aortic and pulmonic valves. 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.

**Physiological Parameters**

Parameters describing a textbook normal “70-kg man” (Table 2) are used to specify values of the compliances and resistances shown in Figure 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/resistance) into cranial, thoracic, and caudal components reflects textbook distributions of cardiac output to various body regions.

**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 $C = \frac{\Delta V}{\Delta P}$, where $C$ is compliance and $\Delta P$ is the incremental change in pressure within a compartment as volume $\Delta V$ 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 change in pressure across resistance $R$ as flow $i$ occurs. In Figure 2, currents $i$, (carotid), $i$, (aortic), $i$, (systemic), $i$, (venous), $i$, (jugular), $i$, (pump input), and $i$, (pump output) are shown for clarity, with positive directions specified by arrows.

**Extrathoracic Components**

Applying these basic concepts with reference to Figure 2 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 versus time waveforms in each of the 7 compartments. Beginning, for example, with the abdominal aorta,


\[ \Delta P_{AA} = \Delta P_{abd} + \frac{1}{C_{AA}}(i_a - i_s) \Delta t = \Delta P_{abd} + \frac{\Delta t}{C_{AA}} \left[ \frac{1}{R_a}(P_{aa} - P_{AA}) - \frac{1}{R_c}(P_{AA} - P_{IVC}) \right]. \]

Here, \( \Delta P_{abd} \) represents the change in external pressure applied to vessels in the abdominal compartment during IAC and Lifestick CPR. For standard CPR, \( \Delta P_{abd} \) is assumed to be zero. The next term represents the increase in abdominal aortic pressure caused by net inflow of blood during the small time interval \( \Delta t \). Substitution for currents \( i_a \) and \( i_s \) by use of Ohm’s law completes the expression.

Similarly, the pressure changes in other extrathoracic vascular compartments are given by Equations 2 through 4, as follows.

\[ \Delta P_{IVC} = \Delta P_{abd} + \frac{1}{C_{IVC}}(i_s - i_i) \Delta t = \Delta P_{abd} + \frac{\Delta t}{C_{IVC}} \left[ \frac{1}{R_i}(P_{IVC} - P_{Rh}) - \frac{1}{R_c}(P_{IVC} - P_{BH}) \right]. \]

**TABLE 2. Nominal Normal Values of Model Parameters**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Symbol</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Compliances, L/mm Hg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thoracic aortic</td>
<td>( C_{a} )</td>
<td>0.000936</td>
</tr>
<tr>
<td>Chest pump</td>
<td>( C_{p} )</td>
<td>0.012</td>
</tr>
<tr>
<td>Right heart and superior vena cava</td>
<td>( C_{rh} )</td>
<td>0.0145</td>
</tr>
<tr>
<td>Abdominal aorta</td>
<td>( C_{aa} )</td>
<td>0.000468</td>
</tr>
<tr>
<td>Inferior vena cava</td>
<td>( C_{ivc} )</td>
<td>0.0234</td>
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<tr>
<td>Carotid artery</td>
<td>( C_{car} )</td>
<td>0.000156</td>
</tr>
<tr>
<td>Jugular veins</td>
<td>( C_{jug} )</td>
<td>0.00936</td>
</tr>
<tr>
<td>Resistances, mm Hg/L/sec</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pump output (aortic valve)</td>
<td>( R_{o} )</td>
<td>10</td>
</tr>
<tr>
<td>Pump input (tricuspid valve)</td>
<td>( R_{i} )</td>
<td>10</td>
</tr>
<tr>
<td>Carotid arteries</td>
<td>( R_{c} )</td>
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<td>Head+arm resistance</td>
<td>( R_{h} )</td>
<td>6470</td>
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<tr>
<td>Jugular veins</td>
<td>( R_{j} )</td>
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<tr>
<td>Aorta</td>
<td>( R_{a} )</td>
<td>25</td>
</tr>
<tr>
<td>Subphrenic vena cava</td>
<td>( R_{v} )</td>
<td>25</td>
</tr>
<tr>
<td>Subphrenic organs</td>
<td>( R_{s} )</td>
<td>1704</td>
</tr>
<tr>
<td>Coronary vessels</td>
<td>( R_{ht} )</td>
<td>11400</td>
</tr>
<tr>
<td>Peak applied pressures, mm Hg</td>
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<td></td>
</tr>
<tr>
<td>Chest compression</td>
<td>( P_{maxch} )</td>
<td>+60</td>
</tr>
<tr>
<td>Chest decompression</td>
<td>( P_{maxcd} )</td>
<td>-20</td>
</tr>
<tr>
<td>Abdominal compression</td>
<td>( P_{maxac} )</td>
<td>+110</td>
</tr>
<tr>
<td>Abdominal decompression</td>
<td>( P_{maxad} )</td>
<td>-30</td>
</tr>
</tbody>
</table>

**Thoracic Components**

Corresponding expressions for the 3 thoracic components of the model are as follows.

\[ \Delta P_{aa} = \Delta P_{chenv} + \frac{1}{C_{aa}}(i_a - i_i - i_h) \Delta t = \Delta P_{chenv} + \frac{\Delta t}{C_{aa}} \left[ \frac{1}{R_c}(P_{aa} - P_{ao}) - \frac{1}{R_{ao}}(P_{ao} - P_{aa}) \right]. \]

\[ \frac{\Delta t}{C_{aa}} \left[ \frac{1}{R_c}(P_{aa} - P_{ao}) - \frac{1}{R_{ao}}(P_{ao} - P_{aa}) \right], \]

where \( N = 1 \) normally and \( N = 0 \) during cough or intrathoracic pressure pulses (when \( P_{BH} = P_{ao} \)).

**Numerical Methods**

**Integration**

Standard spreadsheet programs, such as Microsoft Excel, are ideal for implementing numerical integration of Equations 1 through 7 to obtain pressures in all 7 compartments as a function of time. To simulate a resuscitation, one can create a spreadsheet in which pressures in each compartment at any point in time are computed from the pressures at the preceding time point and the corresponding \( \Delta P \)'s—that is,

\[ P(t + \Delta t) = P(t) + \Delta P(t). \]

One begins with a uniform pressure of 10 mm Hg in all compartments of the arrested circulation and applies phased external pressure pulses to the chest and abdominal compartments, as desired. The incremental changes in compartment pressure computed from Equations 1 through 7 are used to construct a marching solution for successive small increments of time, \( \Delta t \), typically 0.001 to 0.0005 second. Use of a time increment that is too coarse results in unstable oscillations of computed pressures. Decreasing the value of \( \Delta t \), however, can always return stability. In the models used for the
present research, each row of the spreadsheet represents a time point. Pressures for row n + 1 are computed from values in row n by use of Equation 8 and compartmental ΔP values from Equations 1 through 7. At a minimum, 15 columns are needed to represent time from the onset of CPR, absolute pressures P1 through P7 in the 7 compartments of the model, and incremental pressures ΔP1 through ΔP7. For convenience, it is useful also to include columns for systemic perfusion pressure; the applied pressure waveform; the sine function, sin(ωt); ΔP chest; and ΔP Abd.

**Applied Pressures for Chest and Abdominal Compression**

P chest and P Abd represent driving intrathoracic and intraabdominal pressures applied to outer surfaces of blood vessels in the chest and abdomen of the model. Although any arbitrary function or waveform can be used to represent the imposed chest and abdominal pressures in external CPR, the present studies used half-sinusoidal functions, defined as follows and sketched in Figure 3. To represent chest compression,

\[
P_{\text{che}}(t) = \max[0, \sin(\omega t)]P_{\text{max che}}.
\]

If IAC-CPR is being simulated, then

\[
P_{\text{abd}}(t) = \max[0, -\sin(\omega t)]P_{\text{max abd}}.
\]

and if active compression-decompression CPR is being simulated,

\[
P_{\text{adc}}(t) = \min[0, \sin(\omega t)]P_{\text{max adc}}.
\]

In simulations of abdominal decompression during 4-phase Lifestick CPR,

\[
P_{\text{aad}}(t) = \min[0, -\sin(\omega t)]P_{\text{max aad}}.
\]

**Thoracic Versus Cardiac Chest Pump Mechanisms**

To explore the influence of the thoracic pump versus the cardiac pump mechanisms that can impel blood during cardiac arrest and chest compression, a factor 0 ≤ Tpq ≤ 1 is introduced, and a pressure equal to the product of P chest and Tpq is applied to the thoracic aorta and superior vena cava to create a continuum of hybrid pump mechanisms ranging from pure cardiac pump (Tpq = 0) to pure thoracic pump (Tpq = 1). When Tpq = 1, all intrathoracic structures, including the great veins and thoracic aorta, experience a uniform “global” intrathoracic pressure rise, as originally conceived by Rudikoff and coworkers.\(^{28}\) When Tpq = 0, only the pump compliance, Cp, is pressurized, as if the heart alone, and not the great vessels, were compressed between the sternum and the spine, as originally conceived by Kouwenhoven et al.\(^{29}\) Intermediate values of the thoracic pump factor allow models approximating the present understanding,\(^{25,30,31}\) in which for small animals and children, blood is impelled in external CPR predominantly by the cardiac pump mechanism (for example, Tpq = 0.25), whereas in larger animals and adult humans, blood is impelled predominantly by the thoracic pump mechanism (for example, Tpq = 0.75).

**Test Cases and Validation**

The spreadsheet code was validated by solution of 12 simple test cases for very small or very large values of the resistances and compliances and by establishment of a model of the normal adult circulation using Tpq = 0. This model had an aortic blood pressure of 120/82 mm Hg and a cardiac output of 4.9 L/min for a heart rate of 80 bpm, closely approximating the textbook normal values of 120/80 mm Hg and 5.0 L/min.

**Results**

**Alternative CPR Methods in Adult Human Models**

Figure 4a, 4b, and 4c illustrates 5-channel pressure records after 20 cycles of CPR for the normal human circulatory model as shown in Table 2 during a simulation of continuous standard CPR, IAC-CPR, or ACD-CPR, respectively. In Figure 4, the peak positive abdominal pressure for IAC-CPR is 110 mm Hg, and the maximal negative intrathoracic pressure for ACD-CPR is −20 mm Hg, approximating published values for the 2 techniques.\(^{1,2,32–35}\) In this model, the thoracic pump factor is 0.75 to simulate an adult patient in whom the thoracic pump mechanism is dominant but there is some degree of selective cardiac compression.\(^{25}\) The parameters in Table 2 were used for this and all subsequent simulations, unless explicitly stated otherwise. Figure 4a illustrates steady-state pressures generated by standard CPR.
Comparison of the pressure waveforms in Figure 4a and 4b shows the mechanism of +110 mm Hg interposed abdominal compression CPR. The abdominal venous pressure pulse induces increased right heart filling pressure during IAC and consequent faster pump emptying during chest compression compared with standard CPR. Faster pump emptying is caused by larger pressure gradients across the input valve from times 0.54 to 0.67 second of the cycle. Faster pump emptying is caused by the Starling characteristic of the pump associated with greater filling and, in turn, larger pressure gradients across the aortic valve during ejection. With the addition of IAC, cardiac output increases from 1.3 to 2.4 L/min, and mean systemic perfusion pressure (SPP = PAo - Pivc) is mean systemic perfusion pressure in mm Hg. Flow is forward flow in L/min. See Table 1 for abbreviations.

**IAC-CPR**

Comparison of the pressure waveforms in Figure 4a and 4b shows the mechanism of +110 mm Hg interposed abdominal compression CPR. The abdominal venous pressure pulse induces increased right heart filling pressure during IAC and consequent faster pump emptying during chest compression compared with standard CPR. Faster pump emptying is caused by larger pressure gradients across the input valve from times 0.54 to 0.67 second of the cycle. Faster pump emptying is caused by the Starling characteristic of the pump associated with greater filling and, in turn, larger pressure gradients across the aortic valve during ejection. With the addition of IAC, cardiac output increases from 1.3 to 2.4 L/min, and mean systemic perfusion pressure (SPP = PAo - Pivc) is mean systemic perfusion pressure in mm Hg. Flow is forward flow in L/min. See Table 1 for abbreviations.

**ACD-CPR**

Figure 4c shows steady-state pressure waveforms for -20 mm Hg ACD-CPR. Reduced pump pressure (pulmonary vascular pressure) during diastole promotes faster pump filling from ~0.45 to 0.60 second into the cycle. Cardiac output is increased from 1.3 to 1.6 L/min, and mean systemic perfusion pressure is increased from 25 to 30 mm Hg compared with standard CPR. The ACD-induced decrease in central venous pressure offsets the ACD-induced decrease in thoracic aortic pressure, so that augmented perfusion pressure is maintained.
Figure 5. Mean systemic perfusion pressure (SPP) generated by standard and augmented CPR techniques in models with varying mechanisms of blood flow. Effects of chest compression vary according to the thoracic pump factor, ranging from 0 (no compression of the thoracic aorta and superior vena cava) to 1.0 (same pressure applied to the thoracic aorta and superior vena cava as is applied to the heart).

The effects on systemic perfusion pressure of IAC and ACD are similar to those reported for studies in experimental animals and in human patients. Compared with standard CPR, 110 mm Hg IAC produced an 85% increase in total flow. In the same model, 20 mm Hg ACD produced a 23% increase in total flow. The present results in an independent mathematical model confirm that the positive findings in animal studies and most clinical studies are valid and are based on the fundamental anatomy and physiology of the circulatory system.

Four-Phase Lifesick CPR

Although less well studied, Lifesick CPR is a recently developed technique to combine the effects of IAC and ACD. The sticky, self-adhesive compression pads of the Lifesick permit active compression and decompression of both the chest and the abdomen. Accurate simulation of Lifesick CPR is difficult, because actual values of negative intra-abdominal pressure have not yet been reported. If one estimates maximal decompression-phase pressure in the abdomen to be –30 mm Hg, the results in Figure 4d are obtained. With this possible 4-phase technique, mean systemic perfusion pressure is 58 mm Hg. Total forward flow is 3.1 L/min—2.5 times that of standard CPR. Study of the pressure waveforms in Figure 4d reveals that in 4-phase CPR, negative inferior vena cava pressure draws blood out of the chest from 0 to 0.3 second into the cycle, widening the systemic perfusion pressure. Positive inferior vena cava pressure from 0.33 to 0.67 second promotes excellent pump filling.

Influence of Chest Pump Mechanisms

Systemic perfusion pressures obtained by chest and abdominal compression are dependent on the degree to which blood is impelled by cardiac compression versus global intrathoracic pressure fluctuation. In Figure 5, mean systemic perfusion pressure is plotted as a function of the thoracic pump factor for 4 possible CPR techniques: standard, IAC, ACD, and 4-phase Lifesick CPR. Maximal compression or decompression pressures are those listed in Table 2. Although perfusion pressures for the augmented CPR techniques are always better than those for standard CPR, the ratios of experimental to standard perfusion pressures vary with the thoracic pump factor. The relative benefit of IAC-CPR compared with standard CPR, evident in Figure 5, appears to be greater in a pure thoracic pump model than in a pure cardiac pump model. Conversely, the relative benefit of ACD-CPR appears to be greater in a pure cardiac pump model. The apparent benefit of ACD-CPR is especially model-dependent and may be greater in small-animal models, such as beagles, which permit more cardiac compression, than in larger-animal models, including humans. This effect might well explain the generally more dramatic and favorable results with ACD in animal models compared with the overall mixed results observed in humans.

Discussion

Mathematical models provide a good way to synthesize knowledge about complex systems in new and interesting ways and to explore assumptions about how the systems operate. The present mathematical model provides an independent test confirming the efficacy of adjunctive diastolic-phase maneuvers to augment perfusion during CPR. It offers a convenient and low-cost way to compare various CPR adjuncts in exactly the same test system, eliminating the need to extrapolate published results from one animal or clinical model to another. The results confirm that compression and decompression of either the chest or the abdomen can move blood in cardiac arrest. Importantly, the positive effects of IAC-CPR, ACD-CPR, and 4-phase Lifesick CPR can be predicted from fundamental principles of cardiovascular physiology—the definition of compliance and Ohm’s law.

Analysis of pressure waveforms suggests that these techniques function primarily by pump priming. In IAC-CPR, the chest pump is primed by positive pressure in the abdomen during thoracic recoil. In ACD-CPR, the chest pump is primed by negative diastolic pressure in the chest that draws blood centrally from extra-thoracic veins. In 4-phase Lifesick CPR, these effects are combined so that negative thoracic and positive abdominal pressures prime the chest pump. In turn, positive thoracic and negative abdominal pressures prime the abdominal pump.

Conclusions

The present mathematical model, based on fundamental aspects of cardiovascular physiology, provides a recapitulation and synthesis of abundant experimental and clinical evidence suggesting that adjunctive compression and decompression of the chest and abdomen can improve current standard CPR. Systemic perfusion pressure achievable with IAC alone is approximately double that of standard CPR. Systemic perfusion pressure achievable with full 4-phase CPR might possibly exceed 3-fold that of current standard CPR. Improved perfusion during IAC-CPR may not necessarily lead to better long-term survival, especially when the underlying rhythm is asystole or electromechanical dissociation. For the fraction of cardiac arrest victims who can be saved, however, these techniques, performed by trained healthcare providers, are valid and practical alternatives to standard CPR and have a rational place in resuscitation protocols of the 21st century.
References

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