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Chapter 3
Abdominal counter pressure in CPR: What about the lungs?
An in silico study

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The appendix of chapter 2 equally applies to the model used in this chapter.
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Abstract

The external pumping action in CPR should generate sufficient flow and pressure, but the pump must also be ‘primed’ by ongoing venous return. Different additions to standard CPR are in use just for this purpose. Active decompression of the thorax (ACD-CPR) to ‘suck in’ venous blood has proven successful, but, theoretically, compression of venous reservoirs in the abdomen should be even more effective. We compared different techniques for improved CPR with specific attention to the pulmonary circulation. We did our comparisons ‘in silico’ rather than ‘in vivo’ in a well-evaluated computer model.

Methods: We used an adapted version of Babb’s computer model for CPR, reprogrammed in Matlab®. 1) We compared standard chest compression-only CPR (CO-CPR) and ACD-CPR to CPR with interposed abdominal compression (IAC-CPR). 2) Since the thorax/heart configuration differs between patients, and consequently the way blood is propelled by the chest compressions, we checked the influence of the ratio thoracic/cardiac pump effectiveness.

Results: 1) Only IAC-CPR leads to physiological values for mean aortic pressure and cardiac output. 2) However, since the whole heart is in the pressure chamber of the compressed thorax, pulmonary artery pressure rises to about the same level as aortic pressure. In practice, this might lead to pulmonary edema during and after CPR, unless 3) Intra-abdominal compression pressure is strictly limited; simulations indicate that intra-abdominal pressure should not exceed 30-40 mmHg.

Conclusions: IAC-CPR outperforms the other techniques in achieving good aortic pressure and cardiac output. However, abdominal pressure should be limited.
Introduction

Mechanical adjunct devices which do more than just compressing the thorax to improve CPR have been proposed and tried in various studies (1-6). Despite the initial high hopes of the inventors, to date these CPR techniques fail to give consistently better outcomes than standard CPR (S-CPR) (7-9). In 2010 the AHA published its updated guidelines (10) for how and when to perform Cardiopulmonary Resuscitation (CPR). The new guidelines devote only 1.5 out of 330 pages to the option of more complex, possibly machine-supported, modalities of CPR in view of the lack of supporting clinical evidence (10,11). One wonders why praxis is lagging behind, since it makes perfect sense, theoretically, to improve venous return by intermittent abdominal in counter phase with thoracic compressions (IAC-CPR).

In early 2011 the Lancet published a large randomized, multicenter trial that shows improved outcome of CPR when the effect of chest compressions is supported by mechanical devices (12), these are: A hand-held suction cup with handle placed on the thorax to support active thoracic recoil in the relaxation phase (ACD-CPR) and an impedance-threshold valve (ITV) to connect to a facemask or advanced airway access that would not open until intrathoracic pressure would fall below – 16 cm H2O pressure. The latter was in place to promote venous return during the supported chest recoil phase. The Lancet study thus encourages adjuncts to S-CPR. It showed that ACD-CPR with ITV gave the same survival rate, but better neurological function than S-CPR (12). The only significant adverse effect where intervention- and S-CPR groups differed was in the prevalence of pulmonary edema: 11% in the intervention group (94/840) compared to 8% (62/813) in the SCPR group. This inspired us further to elaborate on our earlier modeling work (13), looking more specifically into the pulmonary effects of increased venous return during CPR.

For the present study, we used a computer model to simulate compression-only (CO-CPR) following the new (2010) guidelines, i.e. 100 compressions per minute, no breaks for chest inflation; next ACD-CPR with and without ITV and, additionally, CPR with Interposed abdominal compression (IAC-CPR). The aim was to explore favorable and potentially unfavorable hemodynamic changes produced by augmented CPR techniques compared to standard manual CPR, specifically looking at the effects of improved venous return. For the purpose of this theoretical study, we used a well-known mathematical model of the circulation and the application of CPR, which has been developed and extensively published by Babbs (14-16). A computer model allows analyzing the effects of alternative CPR techniques on many aspects of the cardiovascular and respiratory systems at the same time.
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In the experimental laboratory, this would require sacrificing many experimental animals and in the clinic, it would be next to impossible. In the model we checked the effects on systemic and pulmonary pressures, ventricular pressures, flow to vital organs and so on. Our working hypothesis was that the CPR techniques with supported venous return may have side effects which prevent them from reaching their full beneficial effect.
Methods

Model description

The computer model used is essentially Babbs’ circulatory model, programmed in Matlab® as we used it in earlier studies; details are in (13,15). In short, the model simulates a 70-kg adult, it includes four heart chambers, the pulmonary circulation, the thoracic aorta feeding an upper body compartment and an abdominal compartment, the latter feeds the lower body (legs, buttocks) compartment. CPR is modeled by the application of external forces to the various compartments. Figure1A gives a mechanical representation of the model, Figure1B its electrical analogue as used in the calculations; Table 1 in the Appendix to chapter 2 summarizes the most important model parameters; Table 1A gives the resistances in the model, Table 1B the compliances, initial volumes and unstressed volumes in all compartments. The model supposes a constant blood volume of 4.36 liters, the subject starts in the condition of cardiac arrest, where blood flow has stopped and the blood volume is distributed over the various compartments in relation to their volume compliance. The mean filling pressure in the systemic circulation was chosen to be 5 mmHg (17) that of the pulmonary circulation 8 mmHg while supine.

Figure 1 A: Schematic of the circulation under CPR. ACD: Alternating Compression-Decompression, IAC: Interposed Abdominal Compression, ITV: Impedance threshold valve, impeding inflow of air at negative intrathoracic pressures. NV: Niemann’s valve, preventing reverse venous flow from the thorax to head and neck. VV: venous valves in the legs, preventing reverse venous flow from the abdominal compartment. The (upper) arterial side is connected by lumped Starling resistors to the (lower) venous side.
When CPR is started, chest compression simultaneously increases intrathoracic and mediastinal pressure, the first one working on all compartments in the chest, the second one leading to compression of the heart between sternum and spine. Both contribute as
‘circulatory pumps’ to the generation of flow and blood pressure. We followed Babbs’ model in attributing 75% of the CPR effect to the ‘thoracic pump’ and ‘25% to the ‘cardiac pump’ effects (15). As this choice influences the result, we also checked various other values of this factor.

Abdominal compression is supposed to lead to an immediate pressure increase within the abdomen, compressing in particular the capacity veins feeding the right heart, but also the abdominal aorta, giving the effect of an aortic balloon pump, and changing diastolic runoff. Babbs’ original model does not include abdominal wall mechanics, which in general is much less of a hindrance to externally applied pressures. Therefore, we assumed a homogeneous pressure in the abdominal cavity, following the set time pattern not bothering about how much pressure was applied to the outside to generate this inside pressure.

The model does not take displacement of the diaphragm into account, neither during thoracic nor abdominal compression. Computed pressures in the aorta (minus right atrial pressure) lead to organ flow, depending on the estimated resistances of the various organs (brain in particular). To get a realistic estimate of coronary flow, we suppose that no flow passes while the heart is being compressed.

**CPR techniques**

We simulated three different CPR techniques: first chest-compression only CPR (CO-CPR) as applied by one rescuer and Active Compression-Decompression CPR (ACD-CPR) with and without an impedance—threshold valve (ITV) as in the Lancet study (12). Furthermore, we implemented one technique that combines thorax - with abdomen compression in the relaxation phase to support venous return, i.e. Interposed Abdominal Compression CPR (IAC-CPR). Two rescuers together, one administering thorax compression and the other one compressing the abdomen in counter phase, can administer this (3,18).

In keeping with the new guidelines, a compression frequency of 100/min is used for all techniques; no time is devoted to ventilation. The chest is compressed by a force of 400 N, or around 40 kilo’s, which clinically relates to a depth of 5.1 cm; non-overlapping half-sinusoids with 50% duty cycle are used (15,19) as external pressure waveforms. A force of 150 N supports active decompression of the thorax in ACD-CPR; abdominal compression is supposed to result in (up to) 100 mmHg intra-abdominal pressure in IAC-CPR. The latter two are also shaped as half-sinusoids, with 50% duty cycle in exact counter phase to the thorax compressions.
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Results

Effects of various CPR techniques

The successive columns of Table 1 show that with increasing complexity of the applied technique the numbers get better: higher aortic pressures, higher CO. Indeed, by using IAC-CPR almost physiological levels for mean aortic pressure and cardiac output can be reached. Figure 2 demonstrates how this is obtained: in the phase of abdominal compression, thoracic aortic pressures rise again due to increased systemic vascular resistance during diastolic runoff. In line with the definitions for the working heart, we have defined diastolic pressures as those pressures at the start of thorax compression.

![IAC-CPR abdomen compression: 40mmHg](image)

Figure 2: Aortic ($P_{a0}$, red line) and peripheral pulmonary venous pressures ($P_{ppv}$, thin blue line) during Interposed Abdominal Compression CPR (IAC-CPR) at a compression rate of 100/min, abdominal compression pressure: 40mmHg, thoracic pump factor of 0.75.

Table 1 also shows that ACD-CPR, indeed, gives better results than CO-CPR, only slightly improved by the addition of an ITV. However, none of these 3 techniques gives very satisfactory pressures or cardiac output, while the 4th technique IAC-CPR, can easily overdo it: at increasing abdominal pressures the forced venous return leads to overly increased values...
CPR-optimisation and the lungs

for ventricular end-diastolic and pulmonary artery pressures. All numbers indicate that pulmonary capillary pressures will be above normal plasma colloid osmotic pressures (around 25-30 mmHg (20)), which may cause acute pulmonary edema. However, one may well ask to what extent these results are due to choices made in the modeling process, in particular the division between cardiac pump and thoracic pump. In the computations for Table 1 a thoracic pump factor of 0.75 was used.

Table 1. Blood pressures and flows for the tested CPR-techniques. Thoracic pump factor is 0.75

<table>
<thead>
<tr>
<th>Ventilation/ITV</th>
<th>CO-CPR</th>
<th>ACD-CPR</th>
<th>ACD-CPR</th>
<th>IAC-CPR</th>
<th>IAC-CPR</th>
<th>IAC-CPR</th>
<th>AC-CPR</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No ITV</td>
<td>With ITV</td>
<td>No ITV</td>
<td>No ITV</td>
<td>No ITV</td>
<td>No ITV</td>
<td>No ITV</td>
</tr>
<tr>
<td>Chest comp/decomp (force in N)</td>
<td>400/-</td>
<td>400/150</td>
<td>400/150</td>
<td>400/-</td>
<td>400/-</td>
<td>400/-</td>
<td>400/-</td>
</tr>
<tr>
<td>Abdominal compression</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>40</td>
<td>80</td>
<td>100</td>
<td>Continuous 40</td>
</tr>
<tr>
<td>P_{ao} (sys/dia; mean)</td>
<td>47/22; 30</td>
<td>52/27; 35</td>
<td>55/28; 37</td>
<td>61/34; 46</td>
<td>75/45; 62</td>
<td>83/52; 71</td>
<td>66/41; 49</td>
</tr>
<tr>
<td>P_{tv} (sys/end-dia)</td>
<td>48/7</td>
<td>53/8</td>
<td>56/10</td>
<td>62/26</td>
<td>76/44</td>
<td>84/53</td>
<td>67/27</td>
</tr>
<tr>
<td>CO (l/min)</td>
<td>1.3</td>
<td>1.5</td>
<td>1.7</td>
<td>2.0</td>
<td>2.8</td>
<td>3.2</td>
<td>1.4</td>
</tr>
<tr>
<td>P_{pa} (sys/dia; mean)</td>
<td>41/7; 18</td>
<td>41/7; 17</td>
<td>42/6; 18</td>
<td>62/27; 39</td>
<td>82/47; 58</td>
<td>92/57; 68</td>
<td>62/27; 39</td>
</tr>
<tr>
<td>P_{tv} (sys/end-dia)</td>
<td>42/4</td>
<td>41/4</td>
<td>43/1</td>
<td>63/25</td>
<td>83/47</td>
<td>93/57</td>
<td>62/22</td>
</tr>
<tr>
<td>P_{ppv} (sys/dia; mean)</td>
<td>33/7; 16</td>
<td>31/7; 14</td>
<td>34/5; 15</td>
<td>53/26; 35</td>
<td>72/44; 53</td>
<td>81/53; 62</td>
<td>53/27; 36</td>
</tr>
<tr>
<td>Q_{heart} (ml/s)</td>
<td>0.8</td>
<td>0.9</td>
<td>1.0</td>
<td>1.0</td>
<td>1.2</td>
<td>1.4</td>
<td>0.8</td>
</tr>
<tr>
<td>Q_{head} (ml/s)</td>
<td>5.3</td>
<td>6.0</td>
<td>6.6</td>
<td>8.5</td>
<td>11.8</td>
<td>13.6</td>
<td>6.1</td>
</tr>
</tbody>
</table>

All pressures are in mmHg. Pao: aortic pressure; Plv: left ventricular pressure; Ppa: pulmonary arterial pressure; Prv: right ventricular pressure; Pppv: peripheral pulmonary veins; sys: systolic pressure; dia: diastolic pressure; end-dia: end-diastolic pressure. CO: cardiac output; Qheart: coronary blood flow; Qhead: blood flow to neck and head; AC-CPR: abdominal compression CPR (the pressure is no longer interposed, but continuous).
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Cardiac vs thoracic pump

The ‘cardiac pump’ theory supposes that forward blood flow is caused by direct compression of the heart under the sternum (with blood flow similar to an intact circulation); while the ‘thoracic pump’ theory supposes that blood flow is secondary to changes in intrathoracic pressure (21). The exact contribution of either pump mechanism in a specific case is unknown (21-24), and may depend on thorax-heart configuration: a deep thorax translating CPR-pressure more into a thoracic pump effect, a flat (or child’s-) thorax more into a cardiac pump effect. Therefore, we tested how the alternative attribution of ‘thoracic pump’ or ‘cardiac pump’ might influence the results. In the model a thoracic pump factor 0 implies a pure cardiac pump, 1 a pure thoracic pump.

Figure 3 Top: Effects of thoracic pump factor in CO-CPR (cardiac pump factor + thoracic pump factor = 1); a higher thoracic pump fraction leads to lower aortic pressures and higher pulmonary pressures. A thoracic pump factor = 0 is comparable to direct cardiac massage. Chest compression force: 400 N.

Bottom: Effects of thoracic pump factor in IAC-CPR; a higher thoracic pump factor has little effect on aortic pressures and leads to higher pulmonary pressures. Chest compression Force: 400 N; abdominal compression pressure: 100 mmHg.
Figure 3 top shows the hemodynamic effects on CO-CPR when the thoracic pump factor changes from 0 to 1: diastolic and mean aortic pressures (left panel) decrease and all pulmonary pressures (right panel) increase. Figure 3 bottom shows the same for IAC-CPR, (in the computations a peak abdominal pressure of 100 mmHg was assumed). Here the effects on aortic pressures are less outspoken, much more those on peripheral pulmonary venous pressures. Even at a pure cardiac pump effect (thoracic pump factor = 0) the pressure in the pulmonary capillaries will be too high at this abdominal pressure. Therefore we chose to look for an optimal abdominal pressure that would prevent acute pulmonary edema.

**Optimization of IAC-CPR**

If mean pulmonary capillary pressure exceeds plasma colloid osmotic pressure (around 25-30 mmHg), pulmonary edema may be expected to occur. To prevent this, we tried a range of abdominal pressures, from 0 to 100 mmHg, at a thoracic pump factor of 0.75. Figure 4 shows that Pppv (the model’s approximation of pulmonary capillary pressure) exceeds a value of 30 mmHg when abdominal compression pressure is higher than 30 mmHg; at this point CO is 1.8 L/min and mean aortic pressure around 43 mmHg.

![Figure 4](image)

**Figure 4** Effects of abdominal compression pressure in IAC-CPR. Thoracic pump factor is set to 0.75; chest compression force: 400 N; abdominal compression pressure: 0-100 mmHg. The point of 30 mmHg (mean) peripheral pulmonary venous pressure is shown as upper limit above which pulmonary edema due to hydrostatic pressure may occur. At that level mean aortic pressure is ca. 43 mmHg and cardiac output 1.8 L/min.
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Discussion

This study shows that CO-CPR may be improved upon: better systemic pressures and cardiac output can be reached by improving venous return and increasing lower body vascular resistance as in IAC-CPR. However, these improvements come at a price: the lungs are at risk. In the early papers where IAC-CPR was applied, the authors looked specifically for damage to abdominal organs (4, 25). They took great care in how and how much pressure was applied, and thereby they were able to prevent abdominal trauma. We have shown that peripheral pulmonary venous pressures may run very high during IAC-CPR (Figure 3) with the inherent risk of pulmonary edema, which may prevent successful resuscitation or lead to protracted problems after return of spontaneous circulation. In an experimental study in dogs Kern et al (26) showed that IAC-CPR led to 3/10 cases of pulmonary edema vs. 1/10 in 2 alternative —chest compression only - CPR techniques. In a critical review of the clinical use of IAC-CPR Ward (9) ascribed the variability in its outcome to its inconsistency to improve coronary perfusion pressure (CPP) compared to S-CPR. Although he did not mention pulmonary edema, he did notice that at various ways to exert abdominal compression the induced abdominal pressures rise above 100 mmHg, even up to 150 mmHg. The resulting increase in right atrial pressure was supposed to be the cause of decreased CPP. In our study we did not find a diminution of CPP under IAC-CPR compared to CO-CPR, even while accounting for the aortic to right atrial pressure drop and setting cardiac perfusion to zero in the phase of thorax compression.

As to the damage of the lungs that is hypothesized in our study: what is lacking to make our case, are systematic data from thorax X-ray after successful CPR and/or autopsy data on lungs after unsuccessful attempts. In the early years of CPR a few reports have been devoted to the problem of pulmonary edema (27, 28). Probably more data are available in many clinics where the more intense machine-supported CPR is in use for unshockable cardiac arrest, but except for a few publications (29, 30)it seems that detailed analysis and reporting of pulmonary pathology is not part of standard CPR follow-up.

In this study, we have shown that increased venous return by active decompression of the thorax, but even more by abdominal compression leads to better organ flow in the absence of a spontaneous rhythm. However, there seem to be limits to ‘better pressures and better venous return’: the wall of the right ventricle might be endangered when venous return is increased too much with ensuing increased diastolic pressures in the ventricle. Moreover, the high pressures in the pulmonary circulation may lead to acute pulmonary edema, shifting the
cause of death from cardiac arrest to suffocation. Of course, this prospect may not
discourage rescuers from starting CPR in the best possible way; pulmonary edema can be
resolved over time, after return of spontaneous circulation. This complication that might be
present immediately after successful CPR should be recognized and properly treated.

In view of the two competing mechanisms that explain the effects of CPR: the thoracic pump
and the cardiac pump, one might argue that active abdominal compression in fact adds a
third pump, in series with the thoracic and cardiac pump (31). As is the case with two
locomotives that combine forces to pull a heavy train, one should take care that not one
locomotive is doing all the pulling or pushing, including the other one. In the case of the two
CPR-pumps together, our modeling points to the lungs as being ‘caught in the middle’ This
might call for a new look at CPR, for instance applying the compression/suction cup to the
abdomen and reversing the ITV, so as to block outflow of air up to a certain pressure. That
might prevent many of the known dangers of present-day CPR, like broken ribs, cardiac
contusion and pulmonary edema. Alternatively a G-suit-like device may be applied to the
abdomen and legs. In many ambulances these are available as inflatable supports to stabilize
broken legs or to support the circulation after extensive loss of blood. Rubal et al. (32)
studied the effects of constant G-suit inflation in healthy subjects by left and right heart
catheterization. They showed that inflation pressures >40 mmHg can significantly increase
both cardiac and pulmonary pressures. In a swine model of CPR Lottes et al. (33) tested the
effect of a constantly inflated G-suit. They applied G-suit pressures up to 200 mmHg which
resulted in improvements like those obtained by vasopressor drugs. In our model a
continuous intra-abdominal pressure of around 40 mmHg (Table 1, last column) gives about
the same pressures, systemic and pulmonary, as IAC-CPR at the same abdominal value.
However, flows are considerably less: these are comparable to CO-CPR.

A short survey of available human and animal experimental data shows a consistent lack of
measurements on the low-pressure side of the circulation. We consider our study to be
successful when in follow-up experimental CPR-studies more often a Swan-Ganz catheter is
used.

**Study limitations:**
This computer model is based on anatomy and physiology, combined with experimental data
when these were available. Elaborate studies, implementing other options to improve CPR,
have been published by Babbs (16,34,35). In animal experiments higher aortic pressures than
in our simulations have sometimes been observed (36). However, those were acute
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experiments in animals with healthy hearts (and lungs), probably not comparable to the ‘average’ human heart suddenly going into cardiac arrest due to a build-up of underlying cardiac pathology. What is not modeled is the effect of abdominal compression on displacement of the diaphragm and thereby compression of the thoracic contents (lungs and heart).

No attempts have been made here to incorporate the effects of oxygenation by mouth-to-mouth breathing or otherwise. However limited, a model is just as good as the assumptions that were input to it. In particular the supposed linearity of many anatomical and physiological systems is a simplification to keep models practical. Since this particular model covers a large range of pressure- and volume changes, differences between model and outcome in praxis must exist.

Conclusions:
This study shows that there is room for improvement of CPR by proper choice of parameters for thorax and/or abdominal compression. However, this has its limits: more is not always better. Too much venous return, combined with effective thorax and heart compression may unduly increase pulmonary pressure, thereby exposing the lungs to the risk of acute edema. Post-CPR checks of pulmonary damage by X-ray or autopsy should be a standard follow-up on CPR, be it successful or unsuccessful.

References
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CPR-optimisation and the lungs


