Concomitant ventilatory and circulatory functions of the diaphragm and abdominal muscles

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Submitted 26 May 2010; accepted in final form 30 August 2010


Concomitant ventilatory and circulatory functions of the diaphragm and abdominal muscles. J Appl Physiol 109: 1432–1440, 2010. First published September 2, 2010; doi:10.1152/japplphysiol.00576.2010.—Expulsive maneuvers (EMs) caused by simultaneous contraction of diaphragm and abdominal muscles shift substantial quantities of blood from the splanchnic circulation to the extremities. This suggests that the diaphragm assisted by abdominal muscles might accomplish ventilation and circulation simultaneously by repeated EMs. We tested this hypothesis in normal subjects by measuring changes (Δ) in body volume (Vb) by whole body plethysmography simultaneously with changes in trunk volume (Vtr) by optoelectronic plethysmography, which measures the same parameters as whole body plethysmography plus the volume of blood shifts (Vbs) between trunk and extremities: Vbs = ΔVtr - ΔVb. We also measured abdominal pressure, pleural pressure, the arterial pressure wave, and cardiac output (Qc). EMs with abdominal pressure ~100 cmH2O for 1 s, followed by 2-s relaxations, repeated over 90 s, produced a “stroke volume” from the splanchnic bed of 0.35 ± 0.07 (SD) liter, an output of 6.84 ± 0.75 l/min compared with a resting Qc of 5.59 ± 1.14 l/min. Refilling during relaxation was complete, and the splanchnic bed did not progressively empty. Diastolic pressure increased by 25 mmHg during each EM. Between EMs, Qc increased to 7.09 ± 1.14 l/min due to increased stroke volume and heart rate. The circulatory function of the diaphragm assisted by simultaneous contractions of abdominal muscles with appropriate pressure and duration at 20 min−1 can produce a circulatory output as great as resting Qc, as well as ventilation. These combined functions of the diaphragm have potential for cardiopulmonary resuscitation. The abdominal circulatory pump can act as an auxiliary heart.

cardiopulmonary resuscitation; cardiac output; venous return; splanchnic circulation; expulsive maneuvers

Our laboratory recently found that, during quiet diaphragmatic breathing, 50–75 ml of blood were expressed from the splanchnic vascular bed by the increase in abdominal pressure (Pab) with diaphragmatic descent, while the fall in pleural pressure (Ppl) inflated the lungs (1). We thought we had discovered a generally unrecognized function of the diaphragm. However, we found that, in 1733, Stephen Hales clearly described blood shifts from the splanchnic vascular bed to the extremities when Pab increased (8). Nevertheless, that the diaphragm has both a ventilatory and a circulatory function during quiet breathing is not widely recognized, even though the beneficial hemodynamic effects of diaphragm contractions induced by phrenic pacing have been described in dogs (10) and in humans (17). As long as Ppl falls and Pab rises with breathing, both functions occur simultaneously. We also found that increases in Pab resulting from expulsive maneuvers (EMs) performed by simultaneous contractions of the diaphragm and abdominal muscles augmented the circulatory function of the diaphragm dramatically. Ramp increases in Pab caused shifts in blood from the splanchnic circulation to the extremities in the order of 5 ml/cmH2O increase in Pab (1), so that, with a ramp increase to 100 cmH2O, 0.5 liter of blood could be shifted, more than the usual single transfusion. We were able to measure the time course of splanchnic blood emptying following square-wave increases in Pab (1) and found that it initially came from a fast compartment with a time constant of ~0.6 s, which we assumed to be the liver, followed by a much slower compartment. Filling was well described by a single compartment, also with a time constant of ~0.6 s. The volume of blood coming from the fast compartment varied between individuals from 250 to 600 ml. Based on these data, we calculated that a 1-s “systole” with a Pab of 100 cmH2O (76 mmHg), followed by a 2-s “diastole”, should produce a “stroke volume” of 250–400 ml and, with a frequency of 20 min−1, an output of 5–8 l/min.

High-Pab values are known to stop femoral venous return. In the literature, this has been assumed to result from inferior vena cava obstruction (13, 14, 22), which, in turn, should reduce venous return to the heart and lower blood pressure. However, we have proposed an alternative explanation, namely, that an increase in Pab increases splanchnic blood output through the hepatic vein, increasing inferior vena caval pressure (Pivc) where the hepatic vein enters, and abolishing the gradient in pressure producing flow in the inferior vena cava distal to the entry of the hepatic vein (1). In other words, the circulatory function of the diaphragm produces an oscillatory composition of inferior vena caval blood. During inspiration, splanchnic venous return is favored, whereas, during expiration, venous return of blood below the entry of the hepatic vein is favored.

In this paper, we tested the hypotheses that EMs can produce a splanchnic circulatory output as great as resting cardiac output (Qc), while at the same time arresting femoral venous return by abolishing the energy gradient producing flow upstream from the entrance of the hepatic vein into the inferior vena cava. We did this by measuring 1) the splanchnic vascular output during repeated EMs with pressures and timing as

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We measured Vbs during EMs that increased Pab to the extremities. WBP is insensitive to such blood shifts. Therefore: Vtr changes for the same reasons and by the same amount, but lungs and by gas heating, humidification, compression, and expansion. Vt changes for the same reasons and by the same amount, but it can also change if there are blood shifts (Vbs) between the trunk and the extremities. WBP is insensitive to such blood shifts. Therefore:

\[ \Delta Vt - \Delta Vb = Vbs \]

We measured Vbs during EMs that increased Pab to \( \sim 100 \text{ cmH}_2\text{O} \), while Ppl swings remained essentially constant. This was performed with a 1-s contraction and a 2-s relaxation at a frequency of 20 min \(^{-1}\). During the repeated EMs, we measured the arterial pressure wave continuously with a finger cuff (Portapres, TNO-BMI, Amsterdam, The Netherlands), and calculated Qc using the pulse contour method (20). As this method depends on the distribution of circulation to remain the same for each measurement, we only used the technique in the 2-s interval between the EMs, because of the cessation of femoral venous return. In a separate series of experiments, we measured brachial, subclavian, and carotid arterial and femoral venous blood velocity with an echo-Doppler probe (LOGIQ Book XP, GE Healthcare, Chalfont St. Giles, UK) during breathing and ramp increases in Pab with the subjects lying semirecumbent on a bed.

**Subjects and ethics.** The project was approved by the ethics committee of the INRCA Hospital. We studied seven normal subjects, one woman and six men, aged between 28 and 77 yr. They were trained laboratory personnel, experienced in respiratory maneuvers, who volunteered for the experiment. All gave informed consent after personal risks were explained and the benefits of the research were outlined. They were free to refuse or withdraw at any time without penalty. All studies conformed to the latest revision of the Declaration of Helsinki. Except for one subject with mild well-controlled seasonal asthma who was receiving no medication at the time of the experiment, none had known cardiac or respiratory disorders. One male subject had juvenile diabetes mellitus, which was well-controlled by diet and insulin. Their anthropometric characteristics are given in Table 1.

**Measurements.** \( \Delta Vb \) was measured with the subject sitting inside a home-made, transparent, air-conditioned, variable-flow WBP (1), wearing a nose-clip and breathing room air through a mouthpiece and flowmeter, which connected the subject to the exterior of the box. The flow in and out of the body box due to \( \Delta Vb \) was measured by a pneumotachometer mounted in the top of the box, which connected the box’s interior with the room. The box was antifiltered and had a frequency response flat to 40 Hz, with negligible phase shift up to 10 Hz. The flow signal was integrated digitally to obtain \( \Delta Vb \). Vtr was measured by OEP, which provided three-dimensional tracking of displacements of 89 reflective markers attached to the skin anteriorly and posteriorly between the level of the clavicles and the pubis. The volume of the trunk was measured from the marker positions using Gauss’ theorem (5). Light refraction through the walls of the WBP was corrected by calibrating the OEP system moving markers at known distances inside the WBP.

OEP is based on a motion analyzer that uses charge-coupled device (CCD) sensors mounted on TV cameras equipped with infrared lighting (2, 5). The system provides an electronic shutter to avoid shape distortion of the markers on the CCD. The “active period” of the CCD is only 1 ms, a duration very short, which represents the maximum theoretical time for sampling markers’ movement. All subsequent image processing analysis does not introduce any further delay or time-dependent effect. As the frequency content of respiratory maneuvers is \( \approx 20 \text{ Hz} \), it can be assumed that OEP provides an “instantaneous” measurement of respiratory kinematics. \( \Delta Vt \) was tracked continuously, along with \( \Delta Vb \) and Vbs obtained by subtracting \( \Delta Vb \) from \( \Delta Vt \).

Esophageal and gastric pressures were measured by standard balloon catheter probes attached to two piezo-resistive pressure transducers (RCEM250DB, Sensortechines, Puchheim, Germany) and were used as indexes of Ppl and Pab, respectively. The esophageal balloon contained 1 ml of air, and the gastric balloon 2 ml. Flow at the mouth was measured by another flowmeter (Sensormedics Vmax, Yorba Linda, CA) using a hot wire anemometer, which measured gas velocity in a tube of known cross-sectional area.

A Portapres system was used for continuous measurement of the arterial pulse wave under a finger cuff. From this curve, systolic, diastolic, pulse, and mean arterial pressures were obtained. During breathing at rest and between EMs, Qc was calculated from the arterial pressure wave using the pulse contour method (20). In a separate series of experiments performed with the subject semirecumbent on a bed, an echo-Doppler probe was used to measure blood velocity in the femoral vein to determine what happened to femoral venous velocity during an EM.

**Protocol to measure splanchnic circulatory output.** With the subject sitting comfortably inside the WBP, the OEP cameras tracked the three-dimensional displacements of the reflective markers through the transparent walls of the box, so that \( \Delta Vt \) and \( \Delta Vb \) were measured simultaneously and continuously. We estimated Qc at rest and during the 2-s interval between the EMs. Ppl and Pab were estimated by measuring esophageal and gastric pressures, respectively. Measurements of \( \Delta Vb \), \( \Delta Vt \), the arterial pressure wave, flow at the mouth, Pab, and Ppl were made during a 3-min period of quiet breathing, followed by a series of breaths taken primarily with the inspiratory muscles of the rib cage, so that change in Pab with breathing were voluntarily minimized. Then the subject performed at least three ramp increases in Pab, up to values of 140 cmH2O, with Pab increasing quasi-linearly with time. Finally, the subject performed a series of

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Age, yr</th>
<th>Sex</th>
<th>Weight, kg</th>
<th>Height, cm</th>
<th>BMI, kg/m²</th>
<th>BSA, m²</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>42</td>
<td>M</td>
<td>76</td>
<td>176</td>
<td>24.54</td>
<td>1.92</td>
</tr>
<tr>
<td>2</td>
<td>33</td>
<td>F</td>
<td>85</td>
<td>167</td>
<td>30.48</td>
<td>1.94</td>
</tr>
<tr>
<td>3</td>
<td>77</td>
<td>M</td>
<td>100</td>
<td>172</td>
<td>33.8</td>
<td>2.12</td>
</tr>
<tr>
<td>4</td>
<td>28</td>
<td>F</td>
<td>75</td>
<td>172</td>
<td>25.35</td>
<td>1.88</td>
</tr>
<tr>
<td>5</td>
<td>43</td>
<td>M</td>
<td>70</td>
<td>168</td>
<td>24.8</td>
<td>1.79</td>
</tr>
<tr>
<td>6</td>
<td>38</td>
<td>M</td>
<td>92</td>
<td>180</td>
<td>28.4</td>
<td>2.12</td>
</tr>
<tr>
<td>7</td>
<td>30</td>
<td>M</td>
<td>70</td>
<td>178</td>
<td>22.09</td>
<td>1.87</td>
</tr>
</tbody>
</table>

M, male; F, female; BMI, body mass index; BSA, body surface area.
EMs by changing Pab as close to square-wave increases and decreases as possible with 1-s contractions and 2-s relaxations. These were continued for 90 s, and at least three series of repeated EMs were recorded.

Throughout all of these maneuvers, Vbs was measured continuously by \( \Delta V_{\text{tr}} - \Delta V_b \), so that we measured the blood shifts during quiet breathing, with and without increases in Pab, during ramp increases in Pab and postramp recovery, and throughout the repeated EMs. We particularly wished to know the stroke volume of each maneuver, whether refilling was complete during the 2-s relaxation, and whether, as the EMs progressed, there seemed to be progressive emptying of the splanchnic vascular bed, with a decreasing response to the increases in Pab.

Statistical analysis. Unpaired and paired t-tests were performed to compare, respectively, individual and grouped data between relaxation and EM periods, between rest and relaxation intervals during EMs trials, and between values of Pab at which femoral venous flow stopped and restarted during quiet breathing and ramp EMs. All data are expressed as means \( \pm \) SD. The level of significance was set at 0.05.

RESULTS

Figures 1 and 2 are an example of the results in one subject. In Fig. 1, A and B, the top two traces are \( \Delta V_b \) and \( \Delta V_{\text{tr}} \) superimposed. Below these, in order from top down, are Vbs, Pab, and Ppl. Figure 1A shows Vbs during quiet breathing. With normal diaphragmatic breathing, Vbs are in phase with Pab with a stroke volume of \( \sim 50 \)–70 ml. When the changes in Pab were minimized, Vbs became virtually negligible. During the ramp increases in Pab shown in Fig. 1B, Vbs as a function of time was sigmoid shaped, indicating that, as Pab increased, blood flow out of the splanchnic vascular bed increased with it, reached a maximum, and then decreased as Pab became maximal. These results confirm what our laboratory has already reported (1).

Figure 2A illustrates the whole series of EMs over the 90-s period. From top down, tracings are Vbs, Pab, Ppl, and arterial blood pressure. Points to be noted are that, with the first EM, there was a large blood shift of 0.35 liter, and refilling during the 2-s relaxation was incomplete. With all subsequent EMs, however, the amount of blood pumped out was completely restored during relaxation. Ppl was essentially unchanged from that during quiet breathing. In this particular instance, the “stroke volume” averaged \( \sim 0.39 \) liter, with a circulatory output of 6.62 l/min. With successive maneuvers, there was little or no effect on stroke volume, nor an upward trend in Vbs, which would be expected if the splanchnic vascular bed were gradually emptying during the 90-s run. At the onset of the EMs, there was an increase in arterial pressure, and during the repeated maneuvers there was a further increase in arterial pressure, most evident for the diastolic value.

Figure 2B shows three EMs, shown by the arrows in Fig. 2A, taken early, midway through, and near the end of the run. The resulting blood shifts were well described by single exponentials superimposed on the Vbs tracing with a time constant of \( \sim 0.17 \) s.

Figure 3 shows the time constants for emptying calculated for each EM for each of four subjects where this was possible. All time constants for all EMs in each subject were \( \sim 0.2 \) s, and there was no tendency to change over time. Thus every stroke volume in each subject was well modeled by a single exponential as a linear capacitor emptying through a linear resistor in response to a square-wave forcing function. When ramp emptying was fitted by an ideal response of a linear time-invariant system of the first order to a ramp input, the fitting provided an average time constant, \( \tau_{\text{ramp}} = 0.67 \pm 0.56 \) s (\( r^2 = 0.975 \)).

In Table 2, mean values \( \pm \) SD are given for the increase in Pab during the repeated EMs, the resulting Vbs, the total cycle time, (i.e., EM time plus relaxation time), duty cycle, splanchnic circulatory output per minute, and resting Qc for the five subjects in whom these data were available. In the two other subjects, loss of visibility of reflective markers during the EMs made the estimates of Vtr unreliable. The splanchnic stroke volume averaged 0.35 \( \pm \) 0.07 liter, the total cycle time 3.10 \( \pm \) 0.14 s, and the increase in Pab 103.4 \( \pm \) 15.8 cmH2O, resulting in a mean splanchnic circulatory output of 6.84 \( \pm \) 0.75 l/min compared with a resting Qc of 5.63 \( \pm \) 1.27 l/min.

Table 3 gives means \( \pm \) SD for maximal systolic, diastolic, and mean arterial pressures during the EMs and their minimal values during relaxation. Systolic pressure rose from 142.6 \( \pm \) 18.8 mmHg during the relaxation interval to 155.5 \( \pm \) 22.4 mmHg during the EM (\( P < 0.001 \)). The corresponding diastolic pressures were 77 \( \pm \) 7.8 and 102.4 \( \pm \) 10.5 mmHg (\( P < 0.001 \)). The minimal and maximal mean arterial pressures were 97.8 \( \pm \) 11.6 and 111.7 \( \pm \) 13.0 mmHg, respectively (\( P < 0.001 \)). The diastolic pressures rose by 25 mmHg or 32.9 cmH2O, while Pab rose by 103.4 cmH2O. Thus, assuming that Pab is the perivascular pressure, there was an average reduction of intra-abdominal arterial transmural pressure of 70.5 cmH2O during the EMs.

The Pab at which femoral venous flow stopped and restarted during quiet breathing and ramp increases and decreases in Pab are shown in Table 5.

DISCUSSION

Our demonstration that repeated EMs produced a splanchnic vascular output of 6.84 \( \pm \) 0.75 l/min validates our hypothesis that, with appropriate pressures and timing, the splanchnic circulation can provide an output equaling resting Qc. This is basically an augmentation of the circulatory function of the diaphragm, which is greatly enhanced by the action of the abdominal muscles and inhibited by the antagonistic actions of the rib cage muscles. The fact that the EMs increased blood pressure and Qc, even though femoral venous return ceased, is incompatible with the postulate that high Pab collapsed the inferior vena cava entirely. Rather, it supports our hypothesis that femoral venous flow ceases when the splanchnic venous output raises Pivc at the entrance of the hepatic vein, to the extent that the energy gradient producing flow upstream from the entry of the hepatic vein is abolished (1). For this to happen, Pivc has to be greater than it was before the EM. This, in turn, would increase the pressure gradient from Pivc to the right atrium, increasing Qc, even though venous return up-
Fig. 1. A: tracings during spontaneous quiet breathing and minimization of inspiratory increases in abdominal pressure (Pab). Top: changes in body volume ($\Delta V$; solid line) and the volume of the trunk (Vtr; dashed line) measured simultaneously showing the ventilatory pattern. The difference between Vtr and Vb gives the volume of blood shifted from the splanchnic vascular bed to the extremities (Vbs) by the circulatory function of the diaphragm shown in the middle tracing. Blood shifts (60–75 ml) from the splanchnic bed are in phase with Pab variations during spontaneous breathing, but, when $\Delta Pab$ was minimized, the blood shifts abruptly changed both phase and magnitude. Bottom: Pab and pleural pressure (Ppl). B: Vbs (top), Pab (middle), and Ppl (bottom), all as a function of time during an expulsive maneuver during a ramp increase in Pab.
Fig. 2: A: results during a series of expulsive maneuvers (EMs). The tracings from top down are Vbs, Pab, Ppl, and arterial pressure. The three arrows labeled square waves 1, 2, and 3 indicate 3 EMs taken early, midway through, and near the end of the run. B, top: thick solid lines are single exponentials fitted to the three blood shift tracings chosen in A at the beginning (square wave 1), halfway through (square wave 2), and toward the end (square wave 3) of the repeated expulsive maneuvers. Listed for each curve are the $r^2$ values and time constants for each exponential fit. Bottom: tracings are the “square wave” Pab that produced each blood shift.
stream from the entry of the hepatic vein in the inferior vena cava fell to zero. This is precisely what we found.

Some readers may doubt the ability of OEP to make such accurate measurements of Vbs. However, OEP has been extensively validated (2, 3, 5, 9) when used without combining it with WBP. The optical distortion introduced by the transparent walls of the body box between the markers on the subject and the video cameras that detected their motion was corrected by moving a bar holding markers placed at known distances within the box and then applying the algorithms for camera calibration (Thor2, BTS, Milan, Italy). To demonstrate that they were valid, we measured solids of known volume inside the box with an error always less than ±0.3% (1). Furthermore, when the changes in Pab were minimal during rib cage breathing, OEP and WBP measurements should be identical. Figure 1A shows that this was the case. If the splanchnic stroke volume of ~70 ml during quiet breathing during diaphragmatic descent were due to calibration differences between OEP and WBP, they would not have changed during rib cage breathing. The fact that Vbs virtually disappeared during rib cage breathing validates our measurements of Vbs (1) and proves that Vbs are dependent on Pab.

Table 2. Increases in Pab, Vbs, cycle period, duty cycle, estimated splanchnic output, and cardiac output during repetitive expulsive maneuvers

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>ΔPab, cmH2O</th>
<th>Vbs, liter</th>
<th>Cycle Period, s</th>
<th>Duty Cycle, %</th>
<th>Splanchnic Output, l/min</th>
<th>Cardiac Output at Rest, l/min</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>94.7 ± 10.1</td>
<td>0.442 ± 0.115</td>
<td>3.1 ± 0.1</td>
<td>27.8 ± 2.4</td>
<td>8.67 ± 2.13</td>
<td>5.13 ± 0.54</td>
</tr>
<tr>
<td>4</td>
<td>97.0 ± 1.8</td>
<td>0.280 ± 0.020</td>
<td>3.0 ± 0.0</td>
<td>29.2 ± 3.5</td>
<td>5.16 ± 0.62</td>
<td>3.82 ± 0.41</td>
</tr>
<tr>
<td>5</td>
<td>131.7 ± 0.8</td>
<td>0.270 ± 0.071</td>
<td>3.2 ± 0.5</td>
<td>29.4 ± 0.9</td>
<td>7.56 ± 0.58</td>
<td>5.54 ± 0.29</td>
</tr>
<tr>
<td>6</td>
<td>97.3 ± 1.0</td>
<td>0.370 ± 0.014</td>
<td>3.0 ± 0.1</td>
<td>24.7 ± 0.9</td>
<td>7.13 ± 0.29</td>
<td>6.62 ± 0.17</td>
</tr>
<tr>
<td>7</td>
<td>96.3 ± 1.7</td>
<td>0.385 ± 0.021</td>
<td>3.3 ± 0.0</td>
<td>27.6 ± 1.9</td>
<td>6.84 ± 0.75</td>
<td>5.63 ± 1.27</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>103.4 ± 15.8</td>
<td>0.349 ± 0.073</td>
<td>3.1 ± 0.1</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Values are means ± SD. ΔPab, change in abdominal pressure; Vbs, blood shift volume; duty cycle, ratio between contraction time and relaxation time × 100.

The remainder of this discussion will be an exploration of the possibility that repeated EMs of appropriate timing and pressure might be an effective means of CPR. The fact that we could produce splanchnic vascular outputs by these maneuvers of 5.16–8.67 l/min suggests strongly that augmentation of the circulatory function of the diaphragm could be more effective than sternal compression. We found that, with rapid increases in Pab, the blood initially came from a rapid emptying compartment with a time constant, previously measured (1) of ~0.6 s. The refilling time constant was also ~0.6 s.

To use this information for CPR, the following requirements should be met: 1) Ppl must remain low during the increases of Pab to avoid the effects of a Valsalva maneuver on venous return; 2) the blood should come from the fast emptying compartment; 3) the timing and pressure during the EMs should aim for a stroke volume of at least 250 ml; 4) the stressed blood volume of the liver must be sufficient to meet these requirements; 5) the “diastolic” interval between systoles should allow complete refilling; 6) compression of the inferior vena cava leading to flow limitation must be avoided; 7) the liver and other splanchnic viscera should not be traumatized; 8) the risk of retrograde flow through the splanchnic arteries must be minimized; and 9) the risk of diaphragmatic and abdominal muscle fatigue must also be minimized.

The proposed method of cardiopulmonary resuscitation meets all of these requirements in healthy subjects. It consists of a rapid increase in Pab to ~100 cmH2O (76 mmHg) sustained for 1 s, keeping Ppl constant, followed by a rapid relaxation for 2 s more. Normal subjects who used this timing during voluntary EMs had abdominal stroke volumes of 270–440 ml. With a frequency of 20 min⁻¹, this provided a splanchnic circulatory output that was generally greater than the resting Qc. After the first maneuver, refilling of the splanchnic vascular bed was complete, and there was no tendency for the abdominal stroke volume to decrease with time. There was no evidence that the EMs produced any damage to the abdominal contents.

We now describe the mechanics of flow between the splanchic vascular bed and the rest of the systemic circulation produced by EMs during circulatory arrest. Imagine the situation in which all blood flow stops. The blood pressure would equalize throughout the systemic circulation and fall to the mean circulatory pressure. This pressure is determined solely by the product of
blood volume and the compliance of the vasculature. During normal circulation, it is situated in the small veins. According to the concepts of Guyton et al. (7), the mean circulatory pressure relative to right atrial pressure (Pra) is the pressure producing venous return. During circulatory arrest, an EM will increase the mean circulatory pressure in the splanchnic vascular bed relative to Pra. How is this done?

Imagine that the blood in the splanchnic vasculature were trapped; i.e., no blood could either leave or enter the splanchnic bed. During an EM as Pab increases, the mean splanchnic circulatory pressure (Pmc,sp) would increase equally with Pab. If now the blood were free to flow through the arteries and hepatic vein, the mean circulatory pressure in the splanchnic bed would be higher than that in the rest of the systemic circulation by an amount equal to the increase in Pab, and blood would flow out of the splanchnic bed by both retrograde flow through the arteries and antegrade flow through the hepatic vein. The hepatic vein flow should be greater than the retrograde arterial flow, because the latter is a higher resistance pathway, and initially the hepatic vein flow would all come from the liver. Because the Pmc,sp increased by the change in Pab, the pressure gradient between it and right atrium could be much higher than the normal mean circulatory-Pra gradient, which produces the normal venous return.

In fact, during a cardiac arrest, it would be the highest pressure in the circulatory system, so there would be a pressure gradient producing flow with Pmc,sp > Pra, where Pao is aortic pressure. This assumes that the retrograde arterial flow out of the splanchnic bed is slow and takes a longer time than hepatic venous flow; i.e., that retrograde arterial flow has insufficient time to raise Pao to a level where it equals Pra.

We have described this as a two-step process, first the increase and maintenance of a high Pab, followed by allowing blood to leave the splanchnic vascular bed, whereas, in reality, if EMs were used as CPR, the increase in Pab and outflow of blood from the splanchnic bed would occur simultaneously. But the crucial points to understand are as follows: 1) that according to the concepts of Guyton et al. (7), venous return is described by the mean circulatory pressure relative to Pra divided by the resistance of the venous system between the small veins, where the mean circulatory pressure is situated, and the right atrium; and 2) in the splanchnic vascular bed, in sharp contrast to the rest of the systemic vasculature, the mean circulatory pressure relative to body surface pressure and Pra can increase simply by increasing Pab. An increase in Pab by 100 cmH2O will increase the Pmc,sp by 76 mmHg, vastly increasing the venous return from the splanchnic bed.

Now think about what happens when Pab decreases back to baseline. Under these circumstances, Pao > Pmc,sp > Pra, the splanchnic vascular bed will refill and Pao − Pra will provide coronary flow. If sufficient time is available before the next increase in Pab, the refilling will be complete, and coronary perfusion hopefully sufficient. This is the reason that we chose an EM of 1 s and a relaxation time of 2 s. Probably a 0.5-s EM and a 2.5-s relaxation would be even better. Figure 2 reveals that the blood shifted reached a plateau after 0.5 s. The splanchnic vascular output during a 0.5-s EM would be almost as great as a 1-s EM. It would decrease the duty cycle from 0.333 to 0.167 s and bring the tension-time index of the myocardium to within 30% of control.

Table 3. Psys, Pdia, and MAP during expulsive maneuvers averaged during contraction time (high Pab) and relaxation time (low Pab)

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Psys Low Pab (mmHg)</th>
<th>Psys High Pab (mmHg)</th>
<th>Pdia Low Pab (mmHg)</th>
<th>Pdia High Pab (mmHg)</th>
<th>MAP Low Pab (mmHg)</th>
<th>MAP High Pab (mmHg)</th>
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<tbody>
<tr>
<td>1</td>
<td>143.8 ± 5.3</td>
<td>155.9 ± 8.3*</td>
<td>83.7 ± 2.6</td>
<td>109.3 ± 8.5*</td>
<td>111.0 ± 6.6</td>
<td>114.7 ± 6.7</td>
</tr>
<tr>
<td>4</td>
<td>166.2 ± 6.7</td>
<td>179.8 ± 6.5*</td>
<td>5.1 ± 4.9</td>
<td>108.4 ± 7.5*</td>
<td>108.6 ± 5.3</td>
<td>123.5 ± 8.5*</td>
</tr>
<tr>
<td>5</td>
<td>151.5 ± 9.3</td>
<td>174.6 ± 16.5*</td>
<td>75.9 ± 5.1</td>
<td>112.0 ± 9.1*</td>
<td>95.8 ± 6.7</td>
<td>124.1 ± 10.2*</td>
</tr>
<tr>
<td>6</td>
<td>115.5 ± 6.8</td>
<td>126.7 ± 5.6*</td>
<td>74.4 ± 3.6</td>
<td>92.5 ± 6.0*</td>
<td>87.3 ± 3.9</td>
<td>98.7 ± 5.2*</td>
</tr>
<tr>
<td>7</td>
<td>36.1 ± 10.1</td>
<td>140.5 ± 10.3*</td>
<td>65.7 ± 4.8</td>
<td>89.6 ± 5.6*</td>
<td>86.3 ± 6.7</td>
<td>97.3 ± 8.0*</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>142.6 ± 18.8</td>
<td>155.5 ± 22.4*</td>
<td>77.0 ± 7.8</td>
<td>102.4 ± 10.5*</td>
<td>97.8 ± 11.6</td>
<td>111.7 ± 13.0</td>
</tr>
</tbody>
</table>

Values are means ± SD in mmHg. Psys, systolic pressure; Pdia, diastolic pressure; MAP, mean arterial pressure. *P < 0.001, †P < 0.01, ‡P < 0.05 vs. corresponding relaxation time period.

Table 4. Cardiac output, stroke volume and heart rate at rest and averaged during relaxation intervals between repeated expulsive maneuvers

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Cardiac Output, l/min</th>
<th>Stroke Volume, ml</th>
<th>Heart Rate, beats/min</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>Square waves</td>
<td>Rest</td>
</tr>
<tr>
<td>1</td>
<td>5.03 ± 0.33</td>
<td>6.94 ± 2.42*</td>
<td>70.93 ± 3.73</td>
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<tr>
<td>4</td>
<td>6.95 ± 0.40</td>
<td>8.75 ± 0.79*</td>
<td>6.94 ± 5.03</td>
</tr>
<tr>
<td>5</td>
<td>4.12 ± 0.20</td>
<td>5.90 ± 0.77*</td>
<td>1.34 ± 3.26</td>
</tr>
<tr>
<td>6</td>
<td>5.34 ± 0.41</td>
<td>6.25 ± 0.50*</td>
<td>57.6 ± 3.20</td>
</tr>
<tr>
<td>7</td>
<td>6.53 ± 0.33</td>
<td>7.63 ± 1.10*</td>
<td>9.24 ± 5.25</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>5.59 ± 1.14</td>
<td>7.09 ± 1.14*</td>
<td>69.2 ± 9.5</td>
</tr>
</tbody>
</table>

Values are means ± SD. *P < 0.001, †P < 0.01 vs. corresponding resting period. §Nonsignificant.
increase and decrease in Pab stopped and restarted during quiet breathing and slow increases and decreases in Pab.

Values of Pab at which femoral venous flow stopped; Pab, start, Pab at which femoral venous flow restarted; Ramp, slow increases and decreases in Pab. *P < 0.001 vs. Pab, stop.

did not occur. If a vascular waterfall had developed, the splanchic outflow would not have been described by a single exponential. We presume the capacitor is the liver, and the resistor is the hepatic venous outflow tract. If this interpretation is correct, as seems likely, it is reasonable to conclude that hepatic stressed volume was sufficient to provide all of the splanchic stroke volume.

During the repeated EMs, we measured time constants of ~0.2 s for all maneuvers in each subject. This is in contrast to the time constants our laboratory reported previously of ~0.6 s (1) during single as opposed to repeated square waves and measured as 0.67 s during ramp increases in Pab. We are uncertain as to why this discrepancy exists, but speculate that repeated maneuvers may distend the hepatic venous outflow tract, making the hepatic stressed blood volume more readily available to the rest of the systemic circulation.

In patients with a nonbeating heart, we believe that square-wave magnetic stimulation of the diaphragm and abdominal muscles could provide circulatory outputs similar to those reported here. Under these circumstances, the heart becomes a resistor in the circuit, and, during the EM, the high pressure in the splanchic vascular bed will produce flow through the right side of the heart, pulmonary vasculature, and the left side into the aorta. With Pra greater than Pao during EMs, coronary perfusion will cease. However, during “diastoles”, Pra should fall to a value close to Ppl, while Pao should remain elevated and retrograde flow blocked by aortic valve closure. What the coronary perfusion pressure (the difference between Pao and Pra) would be remains to be determined experimentally, as does the circulatory output from the splanchic vascular bed during cardiac arrest. Animal experiments have begun to answer these questions, but, to date, insufficient data have been obtained to provide useful answers.

Magnetic stimulation could also be used for negative pressure ventilation, if diaphragmatic contraction were just slightly stronger than abdominal muscle contraction. Thus CPR by repeated EMs could provide both circulation and negative pressure ventilation simultaneously. To our knowledge, no existing method of CPR accomplishes this. In fact, positive pressure ventilation has been shown to have adverse effects on the circulatory output (18).

In pilot unpublished studies conducted in normal volunteers, we have been able to achieve the desired Pab values through square-wave magnetic stimulation of the phrenic nerves, inducing diaphragm contractions that were synchronized with pneumatic abdominal compression. Adequate pressures were also produced by square-wave magnetic stimulation of the lower thoracic roots that produced abdominal contractions.

Synchronous stimulation of both sets of muscles has not yet been tried, but, when it is, increasing Pab by 100 cmH2O should not prove difficult, at least in healthy subjects.

To conclude, we believe that abdominal compression keeping Ppl constant, while increasing Pab to ~100 cmH2O for 0.5–1 s, followed by a 2.5–2 s “diastole”, might be an effective means of CPR, which could simultaneously ventilate the lung by decreasing Ppl. However, whether or not this can provide adequate coronary perfusion pressure remains to be determined. Furthermore, the discontinuous nature of the Qc produced by this approach (as opposed to the quasi-continuous flow produced by high-frequency chest wall compressions during CPR) could allow important drops in mixed-venous
blood oxygen saturation to occur. Whether or not this is the case and whether or not this has clinical consequences will have to be determined by animal studies. Of note, it is known that repeated coughing during asystolic cardiac arrest can maintain consciousness (6, 15), and our experiments provide an explanation for this interesting phenomenon. Our laboratory has previously demonstrated that outflow from the splanchnic blood reservoir is controlled by Pab (1), and in this paper we confirm and extend these findings. During quiet breathing with diaphragm descent, the diaphragm serves two functions: the first to ventilate the lung; and the second to shift blood from the splanchnic vascular bed to the extremities. With simultaneous contraction of abdominal muscles, such as occurs during exercise (3), the circulatory function of the diaphragm can be considerably enhanced. The circulatory function of the diaphragm has previously described (1, 8, 10). Under appropriate circumstances, the diaphragm’s circulatory function combined with abdominal muscle contraction can act as an abdominal circulatory pump, capable of acting as an auxiliary heart. When needed, this should be clinically useful.

REFERENCES