CIRCULATORY ADJUNCTS

Newer Methods of Cardiopulmonary Resuscitation

Charles F. Babbs, MD, PhD

Conventional cardiopulmonary resuscitation—what has come to be called standard CPR—has changed little since first introduced in the early 1960s by Kouwenhoven et al.[1] Standard CPR requires chest compressions at a rate of 80 to 100 compressions/min in conjunction with mouth-to-mouth rescue breathing.[2] Positive pressure ventilations (PPVs) are given at an overall rate in the range of about 8 to 12 ventilations/min, depending on whether one is doing one- or two-rescuer CPR. Refinements of standard CPR since its introduction in the 1960s have included increasing the rate of chest compression from 60 compressions/min to 80 compressions/min or more, which research shows matters little,[3] and recently decreasing the tidal volume of the PPVs under certain circumstances.[2, 4] Elimination of the carotid artery pulse check in the year 2000 guidelines has abolished unnecessary delay in starting chest compressions by lay rescuers. Otherwise, external CPR today is performed in approximately the same way as it was in the 1960s. In keeping with the engineering maxim that "if you keep on doing what you're doing, you'll keep on getting what you've got," success rates for standard CPR have changed little over the past 40 years.[5–9] Between 15% and 40% of all victims of nontraumatic cardiac arrest are resuscitated, and approximately 5% to 10% live to leave the hospital.

Despite lack of significant change on the surface, a neglected community of researchers has made substantial progress in the laboratory and in the clinic during this same 40-year period. Results have lead to much fundamental understanding of the circulatory physiology and hemodynamics of cardiac arrest and resuscitation. A variety of alternative forms of CPR have been proposed and tested in animals and in humans. All of these alternative methods apply more kinetic energy to the victim, either by way of an additional rescuer or some type of mechanical device. More energetic forms of CPR include high-impulse CPR, interposed abdominal compression–CPR (IAC-CPR), active compression-decompression CPR (ACD-CPR), and vest CPR. These derivatives of standard CPR have come to be known as *circulatory adjuncts*. By the

The author has no financial interest, consulting arrangement, or research contract with Ambu, CPRxLLC, or any other resuscitation device company that could be construed as a conflict of interest.

From the Department of Basic Medical Sciences, Purdue University, West Lafayette, Indiana

year 2000, a convergence had occurred between laboratory knowledge about mechanisms of blood flow and clinical experience with the various adjuncts. This long-delayed synthesis of research findings has led to *improved* forms of practical resuscitation (able to generate more blood flow and systemic perfusion pressure during cardiac arrest through the efforts of the rescuers) based on sound basic science principles. If there is one certain truth in resuscitation science, stemming from the pioneering work of Redding[10], Ralston et al,[11] Weil and Tang,[9] and Kern et al,[12] it is this: Improved coronary and systemic perfusion pressures during cardiac arrest and CPR lead to improved immediate resuscitation success and improved likelihood of longer-term, neurologically intact survival.

The improved hemodynamics, however, do not come without a price, because more physical work has to be done by human or machine to generate more forward flow of blood. Accordingly, adjuncts may not be appropriate for the lone rescuer faced with a collapsed coworker or loved one. Nevertheless, the time has come for serious consideration of alternative forms of resuscitation when adequate equipment and trained personnel are available, for example, in the emergency department of a hospital. This article focuses on the relevant physiology of cardiac arrest and CPR that makes possible the generation of systemic perfusion pressure substantially greater than that provided by standard CPR. Thereafter, selected practical aspects of applying currently approved circulatory adjuncts in a hospital setting are discussed.

MODERN PHYSIOLOGY OF BLOOD FLOW DURING CARDIOPULMONARY RESUSCITATION

At least three different mechanisms can move blood during cardiac arrest and CPR: (1) the cardiac pump, (2) the thoracic pump, and (3) the abdominal pump. The cardiac pump mechanism was the first to be recognized by the original discoverers of closed-chest CPR.[1] This pump mechanism is operative to the extent that external chest compression squeezes the cardiac ventricles between the sternum and the spine. As a result, forward blood flow occurs through the aortic and pulmonic valves without mitral or tricuspid incompetence. In particular, when the cardiac pump mechanism is operative, the aortic valve is open, and the mitral valve is closed during chest compression.[13] The cardiac pump mechanism is also operative during open-chest cardiac massage.

The thoracic pump mechanism was discovered in the 1980s as a result of clinical observation by Criley et al[14, 15] of cough CPR and extensive laboratory studies at Johns Hopkins University, led by Rudikoff et al[16] and Weisfeldt.[17] This pump is operative to the extent that chest compression causes a global rise in intrathoracic pressure sufficient to force blood from the pulmonary vasculature, through the heart, and into the periphery. When the thoracic pump mechanism is operative, the mitral and the aortic valves are open simultaneously during chest compression.[18–20] The heart acts as a conduit rather than a pumping chamber.

The abdominal pump mechanism was discovered independently by workers in Great Britain, Japan, Israel, and the United States in the latter half of the 20th century.[21–26] This mechanism has two components: (1) arterial and (2) venous. The arterial component of the abdominal pump mechanism is operative to the extent that abdominal compression forces blood from the abdominal aorta into the periphery against a closed aortic valve. Thus, the aortic valve is closed during abdominal compression. The venous component of the abdominal pump mechanism is operative to the extent that external abdominal pressure forces blood from the inferior vena cava through the tricuspid valve into the right ventricle (or through the tricuspid and pulmonic valves into the pulmonary vasculature). In this case, the right heart valves are open during abdominal compression.

The operation of these three pumps—cardiac, thoracic, and abdominal—is related to the fundamental architecture of the cardiovascular system. The physiology of the pumps has been demonstrated in relatively simple mathematical models that represent the essential features of the human cardiovascular system.[24, 27–31] Successful circulatory ad-

juncts generate greater blood flow than standard CPR either by enhancing the operation of one of these pumps or by simultaneously invoking the action of multiple pumps. High-impulse CPR,[32–34] for example, aims to enhance the action of the cardiac pump mechanism. Vest CPR[35, 36] aims to enhance the action of the thoracic pump mechanism through the action of a pneumatic vest that is inflated and deflated rapidly at a rate of 60 to 150 times/min. ACD-CPR[37, 38] aims to improve filling of the either the cardiac pump or the thoracic pump by creating negative pressure in the thorax between chest compressions. IAC-CPR[39–43] aims to invoke the abdominal pump together with either chest pump. IAC-CPR and its derivative, Lifestick (Datascope, Fairfield, NJ) CPR,[44] aim to combine thoracic and abdominal pumps alternately, in such a way that the thoracic pump primes the abdominal pump during one half of the cycle and the abdominal pump primes the thoracic pump during the other half of the cycle.

Regarding the question of the effectiveness of these various adjuncts, it would be difficult to compare and contrast all such methods in a single animal or clinical model; however, it is reasonable to use a mathematical model of the circulation that includes all three pumps.[31] Such a model can be used to simulate the effects of various circulatory adjuncts in exactly the same circulatory system, without model-to-model variation. It also serves to demonstrate the relevant cardiovascular physiology of CPR.

An interesting feature of the circulatory model used in this article is the small number of assumptions required to obtain realistic results.[31] These are limited to the existence of compliant vessels and resistive vascular beds, the definition of compliance ($\Delta V/\Delta P$), normal anatomy (the arrangement of connected vessels and cardiac chambers), and a linear relation between flow and pressure (Ohm's Law: flow = pressure/resistance).[28–31] Although much more complex models of the circulation can be created, only these basic assumptions are needed to demonstrate the three mechanisms of blood flow during CPR and the relative utility of various circulatory adjuncts. Circulatory systems that have these properties behave similarly, including those of large and small people and large and small experimental animals. The author of this article uses the simplest realistic model of CPR physiology to illustrate the actions of the basic pump mechanisms and to demonstrate selected circulatory adjuncts. The exact values of vascular compliances and resistances and other technical details of the model, which can be implemented in a spreadsheet, are fully described elsewhere.[31]

As a point of reference and calibration, Figure 1 illustrates pressures throughout a simplified cardiovascular system for a nonarrested circulation of a hypothetical 70-kg man. In this instance, cardiac pump generates left ventricular pressures (Ppump) of 122/2 mm Hg at a heart rate of 80 beats/min (bpm). Systemic arterial blood pressure is 119/82 (the data point representing the exact minimum diastolic pressure at 82 mm Hg is not plotted on the chart), mean arterial pressure is 95 mm Hg, and cardiac output is 5.0 L/min. These are classical textbook values for the normal human circulatory system.[31] Note the essentially normal arterial pulse waveforms and low systemic venous pressures.

Figure 2 illustrates the action of a pure cardiac pump CPR in the same circulatory model during cardiac arrest. Steady-state conditions are shown after stable pressures have been achieved by 20 prior compressions. In this simulation, only the right and left ventricles of the heart are compressed at a rate of 80 compressions/min with a half sinusoidal waveform having a peak pressure of 60 mm Hg, a typical value reported in the literature of standard CPR[29]. There is no intrinsic myocardial contractility in this system, and there is no pump priming effect of atrial contraction (which in some circumstances could exist for a few minutes in witnessed cardiac arrests). The cardiac pump produces reasonable aortic pressures and very small venous pulsations. Especially important to note are the low right-sided central venous pressures throughout the compression cycle. There is substantial coronary perfusion pressure (aortic to right atrial gradient) throughout the cycle. Forward flow is 2.5 L/min, and systemic perfusion pressure is 47 mm Hg. This state of affairs represents idealized classical external CPR in which "the heart is squeezed between

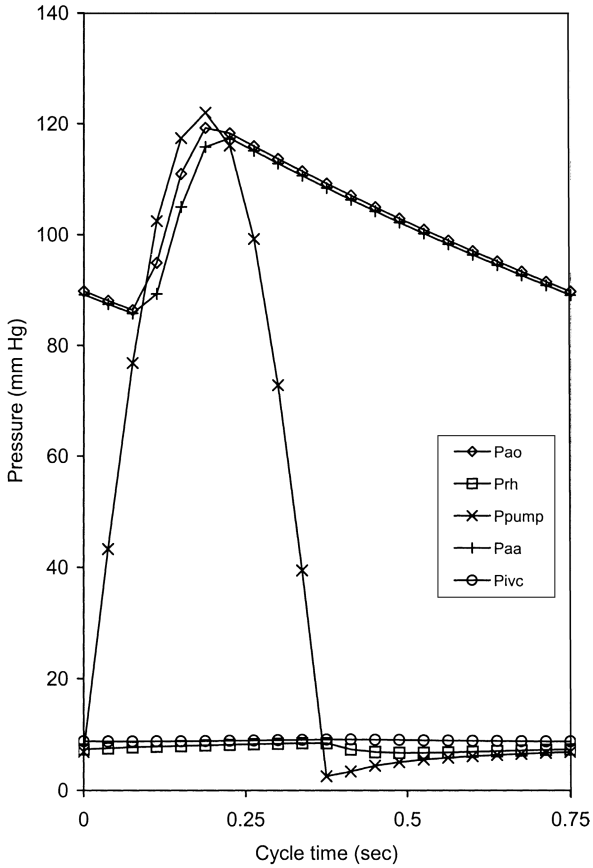

Figure 1. Pressures in a mathematical model of the normal adult human circulation with the cardiac ventricles beating. The heart rate is 80 beats per minute (bpm). Pressures are plotted as a function of cycle time for the thoracic aorta (Pao), the right atrium (Prh), the intrathoracic pump, here the left ventricle (Ppump), the abdominal aorta (Paa), and the inferior vena cava (Pivc). SPP is mean systemic perfusion pressure (SPP) = Pao − Prh. Normal flow: flow = 5.0 L/min; SPP = 95 mm Hg.

the sternum and the spine."[45] It is also a reasonable representation of open-chest CPR with manual cardiac compression, which works by a pure cardiac pump mechanism. A similar state of affairs can occur in children (and young pigs[46, 47]), who have small compliant chest walls.

Figures 1 and 2 were generated using positive applied extravascular pressures during the compression phase and zero extravascular pressure during the relaxation phase. A relatively recent concept in the physiology of CPR is the use of active decompression, rather than simple relaxation, between chest compressions. Decompression can be accomplished by the use of plungerlike devices (discovered accidentally using a real toilet plunger![37, 48]) or by sticky adhesive pads that make contact with the skin of the anterior chest or abdomen, such as those incorporated into the Lifestick.[44] This approach is known as *active compression-decompression CPR* (Figure 3). Currently, active decompression of the chest during CPR can be accomplished using a specially designed plunger applied to the human sternum,[46, 49–51] which is sold commercially in Europe as the Ambu CardioPump (Ambu Inc., Glostrup, Denmark).

Figure 4 illustrates the steady-state effect of active decompression of the chest to

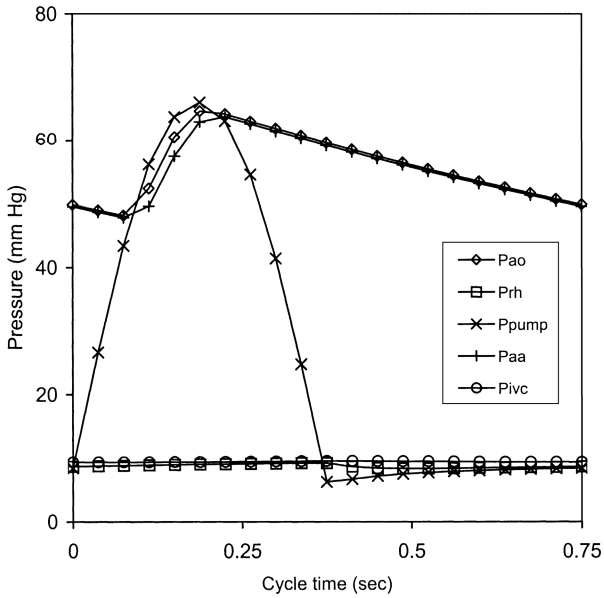

Figure 2. Pressures in a mathematical model of the normal adult human circulation during cardiac arrest and Cardiopulmonary resuscitation (CPR) with a pure cardiac mechanism. The compression rate is 80 per minute. Cardiac compression CPR: flow = 2.5 L/min; SPP = 47 mm Hg.

−20 mm Hg, the maximum reported in the literature.[50, 52, 53] This particular simulation is for cardiac pump CPR. Combining positive and negative chest pressures has a salubrious effect upon hemodynamics. Cardiac filling is enhanced during the negative pressure phase, so that greater stroke output can be achieved on the next positive pressure phase. Note in Figure 4 the particular times near 0.55 seconds in the cycle when pump pressure is substantially less than right heart pressure. At this stage, enhanced pump filling occurs. The result of enhanced pump filling is greater forward flow and greater perfusion pressures: 3.2 versus 2.5 L/min and 61 versus 47 mm Hg. When more energy is applied to the ar-

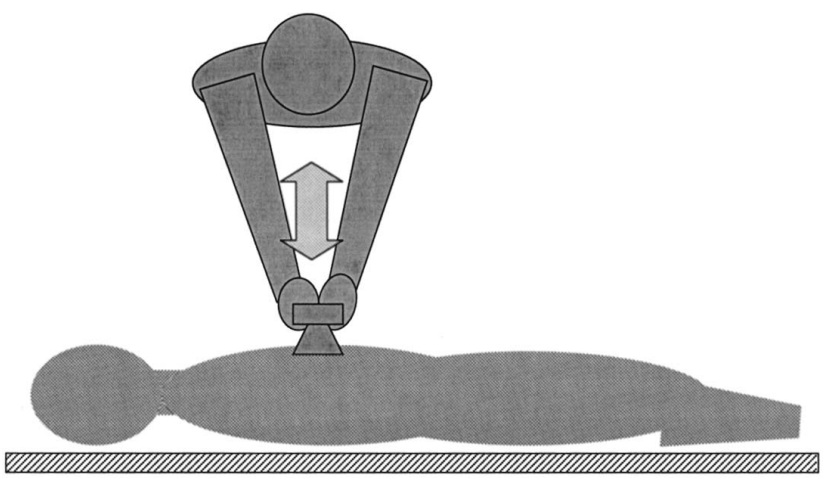

Figure 3. Active compression-decompression CPR (ACD-CPR) using a plungerlike device.

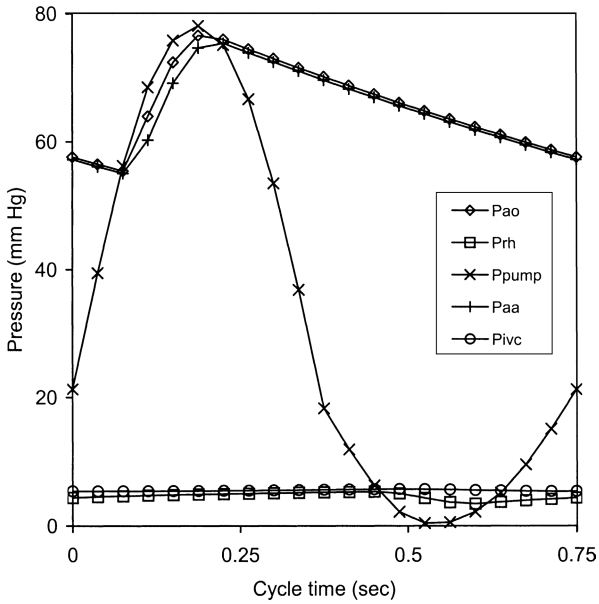

Figure 4. Pressures in a mathematical model of the normal adult human circulation during cardiac arrest and ACD-CPR with a pure cardiac pump mechanism. The compression rate is 80 per minute. Maximal chest compassion pressure is +60 mm Hg. Maximal decompression pressure is −20 mm Hg. Cardiac compression-decompression CPR: flow = 3.2 L/min; SPP = 61 mm Hg.

rested circulation in this manner, forward flow improves. A potential advantage of the use of negative and positive pressure phases, rather than simply greater positive pressure, is that it is less traumatic to the victim. Also, needless compression of pumping chambers that are already empty is avoided. The use of a decompression phase compliments the use of a compression phase for all three pump mechanisms in CPR (see later discussion).

When it works, the cardiac pump mechanism is the most effective and natural of the three pumps in CPR. Its operation in external CPR, however, depends on good mechanical coupling between the sternum and the heart. In most adults, the coupling of chest compression to the heart is indirect, and a thoracic pump mechanism tends to predominate.[20, 35, 54] Thoracic pump CPR has a quite different set of pressure profiles. Figure 5 illustrates the action of a pure thoracic pump. In this simulation, all intrathoracic blood–containing chambers are pressurized equally at a rate of 80 per minute with a peak pressure of 60 mm Hg, as before. This state of affairs happens in broad-chested adults. It also happens during vest CPR, in which a pneumatic vest encircles the chest to produce pulses of compression from all sides simultaneously.

In thoracic pump CPR, forward flow occurs even though the heart is not being squeezed between the sternum and the spine. Coronary blood flow and systemic blood flow occur when aortic pressure is greater than systemic venous or right heart pressure. As shown in Figure 5, positive coronary and systemic perfusion pressures occur mostly between compressions, rather than during compressions. Because of the tendency toward equalization of aortic and venous pressures during compressions, forward flow with the thoracic pump mechanism tends to be less, other factors being equal, than with the cardiac pump mechanism. In a thoracic pump model of Figure 5, forward flow is 0.94 L/min, and systemic perfusion pressure is 18 mm Hg.

If a decompression phase is added (Fig. 6), perfusion pressures are somewhat increased, but to a lesser extent than with cardiac pump CPR. In this instance, forward flow is 1.14 L/

min and systemic perfusion pressure is 22 mm Hg. The difference in the responsiveness of the thoracic and cardiac pumps to negative pressure priming may be responsible for some of the discrepancies in the reported literature regarding the benefits of ACD-CPR in various models.[31] The simulated improvement in coronary perfusion pressures with ACD-CPR (61 mm Hg for a cardiac pump model and 22 mm Hg for a thoracic pump model) are in keeping with the range of measured coronary perfusion pressures in human patients during ACD-CPR. In one recent study,[51] the generation of −20 mm Hg intrathoracic pressure during ACD was ensured with the use of an inspiratory impedance valve. These investigators found coronary perfusion pressures of 40 to 45 mm Hg. These results are consistent with those of Figures 4 and 6, if one takes the modern consensus view[20, 35, 54] that the operative pump mechanism in different animal and clinical models is a combination of the cardiac and thoracic pumps.

Figure 7 illustrates the steady-state action of a pure abdominal pump. In this simulation, there is no chest compression. Abdominal compression begins at time zero. Only the abdominal aorta and vena cava are compressed at a rate of 80 compressions/min with a pressure of 110 mm Hg, values reported in the literature of IAC-CPR.[39, 55, 56] Manual compression leading to periaortic pressures this high can be tolerated without pain by a conscious person.[57] Artificial circulation can indeed be created by abdominal compression only, as first observed experimentally by Rosborough et al.[58] During abdominal compression, blood is squeezed from the aorta to the periphery by the positive intra-aortic pressure acting against a closed aortic valve. During abdominal relaxation, the aortic valve opens, allowing filling of the aortic pumping chamber from the left heart

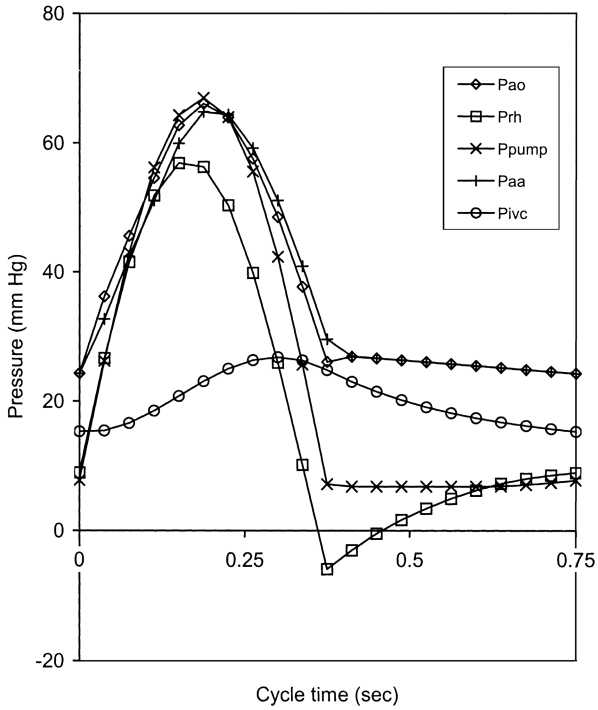

Figure 5. Pressures in a mathematical model of the normal adult human circulation during cardiac arrest and CPR with a pure thoracic pump mechanism. The compression rate is 80 per minute. Maximal chest compression pressure is +60 mm Hg. Thoracic compression CPR: Flow = 0.93 L/min; SPP = 18 mm Hg.

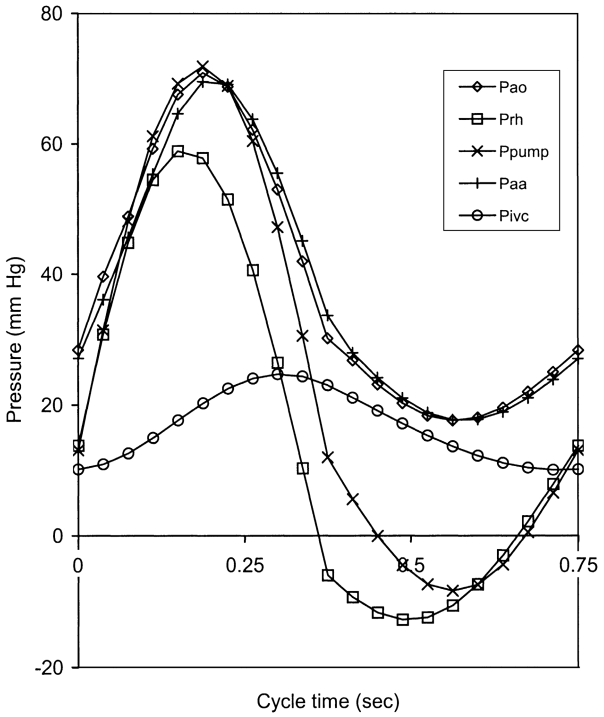

Figure 6. Pressures in a mathematical model of the normal adult human circulation during cardiac arrest and ACD-CPR with a pure thoracic pump mechanism. The compression rate is 80 per minute. Maximal chest compression pressure is +60 mm Hg. Maximal chest decompression pressure is −20 mm Hg. Thoracic compression-decompression CPR: flow = 1.1 L/min; SPP = 22 mm Hg.

and lungs. Only one valve is needed to achieve forward flow, as can be demonstrated by deliberately rendering the other three heart valves incompetent.[29] It is important to note that the abdominal aortic pressure waveform at 0 to 0.2 seconds in the cycle leads the thoracic aortic pressure waveform in time, indicating retrograde flow in the aorta. Pump pressures change very little, because the chest pump is acting as a passive conduit. There is a positive coronary perfusion pressure during most of the compression cycle. Forward flow is 1.3 L/min and coronary perfusion pressure is 25 mm Hg.

Figure 8 shows the potential benefit of adding active abdominal decompression (−20 mm Hg pulses) to abdominal compression. Such a manipulation is possible using a sticky adhesive abdominal pad, and has been implemented practically in the experimental Lifestick device. In this simulation, there is still no chest compression. The abdominal pump is filling when chest pump pressure is greater than thoracic aortic pressure near 0.4 seconds in the cycle. At this time, the low resistance aortic valve is open. With active abdominal decompression, the filling pressure differences are more obvious than when abdominal decompression is absent. During subsequent abdominal compression positive pressure in the aorta, which is greater than that in the chest pump, closes the aortic valve. The slower rise of right heart pressures compared with inferior vena cava pressures is associated with differences in the capacitances of these structures. Near 0.3 seconds of the cycle, the chest pump, including the heart and lungs, fills when right heart pressure exceeds chest pump pressure. Thus forward flow occurs around the entire circuit with abdominal compression–decompression alone. As in the case of the cardiac and thoracic pumps, the addition of active decompression enhances abdominal pump performance (see Fig. 8).

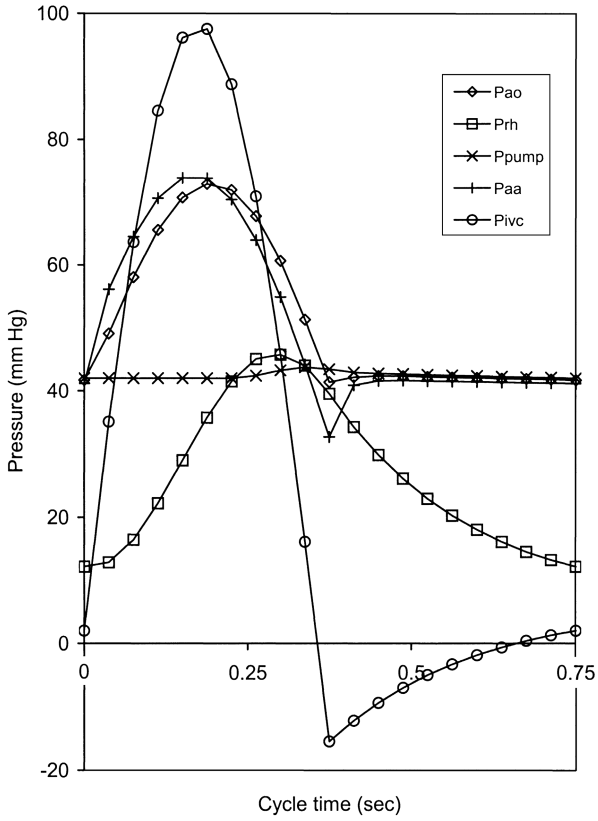

Figure 7. Pressures in a mathematical model of the normal adult human circulation during cardiac arrest and CPR with a pure abdominal pump mechanism. The compression rate is 80 per minute. Maximal abdominal compression pressure is +110 mm Hg. Abdominal compression CPR: flow = 1.3 L/min; SPP = 25 mm Hg.

COMBINING CHEST AND ABDOMINAL PUMPS

Given this theoretical background, it is a small conceptual leap to imagine combining chest and abdominal pump mechanisms. The simplest and most practical such combination is called *interposed abdominal compression–CPR* (Fig. 9). IAC-CPR invokes the abdominal pump together with either the cardiac or thoracic pump. The trick is to compress the abdomen during chest diastole, thereby operating two pumps together in the same compression cycle. Alternation of chest and abdominal compression has the practical advantage of avoiding trauma to the liver and has been proved extremely safe in animal and human trials.[43] Although other timing and phasing schemes have been investigated,[23, 30, 44] the simplest and most studied form of IAC-CPR involves a 50% duty cycle for both chest and abdominal phases. That is, the compression cycle is divided into two equal duration phases, in which an extra rescuer manually compresses the abdomen during the relaxation phase of chest compression.[26, 42] With alternating chest and abdominal compression, the aortic valve is open during chest compression and closed during abdominal compression. Chest pump outflow during chest compression fills the aorta, priming the abdominal pump. Abdominal-to-thoracic caval flow during abdominal compression fills the right heart and pulmonary vasculature, priming the chest pump.[31] Thus the two pumps work in concert. Improved venous return increases stroke output of the chest pump, in conjunction with either the thoracic pump

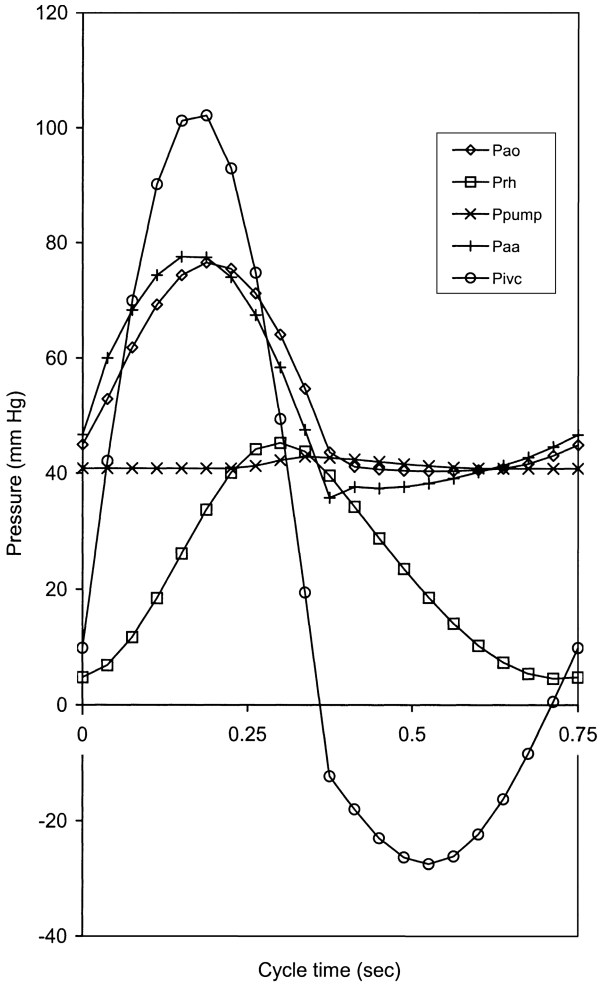

Figure 8. Pressures in a mathematical model of the normal adult human circulation during cardiac arrest and ACD-CPR with a pure abdominal pump mechanism. The compression rate is 80 per minute. Maximal abdominal compression pressure is +110 mm Hg. Maximal abdominal decompression is −30 mm Hg. Abdominal compression-decompression CPR: flow = 1.6 L/min; SPP = 30 mm Hg.

mechanism or direct cardiac compression. Abdominal compression also produces counterpressure on the abdominal aorta, inducing retrograde flow toward the heart and brain between chest compressions.[59] This abdominal pump action on the arterial side increases flow in a manner analogous to an intra-aortic balloon pump.[29, 43]

The effectiveness of such combined chest and abdominal compression was discovered empirically[22, 26] long before the theoretical physiology had been worked out.[27–31] Indeed the positive hemodynamic effects of IAC during CPR have been confirmed in 20 of 21 animal studies using canine and porcine models.[43, 60] After encouraging laboratory studies, clinical trials of IAC-CPR were performed. Three randomized clinical trials of IAC-CPR for in-hospital cardiac arrest have shown statistically significant improvement of outcome measures.[42, 61–62] One randomized trial of prehospital IAC-CPR, combined, when possible, with standard CPR in the field, showed no difference in outcome.[63] These studies are summarized in Table 1. Pooled analysis of all available data for pre-

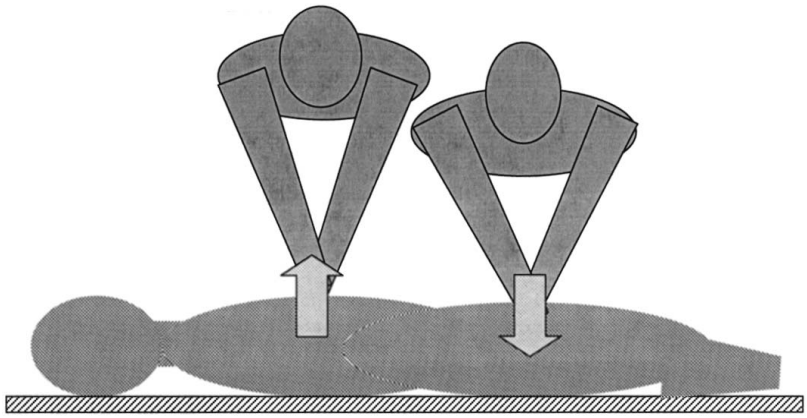

Figure 9. Interposed abdominal compression (IAC) CPR (IAC-CPR).

hospital and in-hospital resuscitations shows statistically significant improvement in the return of spontaneous circulation with IAC-CPR. When only in-hospital studies are examined, the effect of IAC becomes much greater and is highly statistically significant. Pooled data from two studies that examined long-term, neurologically intact survival following in-hospital resuscitations show a positive benefit of IAC-CPR compared with standard CPR. Thus strong preclinical and clinical evidence supports the use of IAC-CPR for in-hospital resuscitations.

For the purpose of the present discussion, the hemodynamic benefits of IAC-CPR can be demonstrated in the same mathematical model with which basic physiology is investigated. Figure 10*A* illustrates the addition of 110 mm Hg IACs to 60 mm Hg peak thoracic compressions in a cardiac pump model. In this instance, the cardiac and abdominal pump mechanisms are combined. (Even though more external force is applied to the chest than to the abdomen in practice, typical intrathoracic pressure pulses are less than intra-abdominal pressure pulses because of the stiffness of the chest wall.) The double arterial pressure peaks in Figure 10*A* attest to the effects of the double pump approach. Systemic perfusion pressure is sustained throughout both phases of the compression cycle. Enhanced filling of the chest pump is evident at cycle times near 0.7 seconds. Filling of the abdominal pump chamber (ie, the aorta) can be seen at cycle times near 0.125 seconds. The increased forward flow in this model 3.6 L/min with IAC versus 2.5 L/min for cardiac pump CPR. Systemic perfusion pressure is 68 mm Hg versus 47 mm Hg for cardiac pump CPR. Thus the ratio of perfu-

Table 1. RESULTS OF CLINICAL STUDIES OF INTERPOSED ABDOMINAL COMPRESSION–CARDIOPULMONARY RESUSCITATION VERSUS STANDARD CARDIOPULMONARY RESUSCITATION

Outcome Measure	Studies	IAC-CPR (%)	Standard CPR (%)	*P* Value
Return of spontaneous circulation in- or out-of-hospital	Mateer[63]	40/145 (28)	45/146 (31)	.54
	Ward[61]	6/16 (38)	3/17 (18)	.19
	Sack[42]	29/48 (60)	14/55 (25)	.00014
	Sack[62]	33/67 (49)	21/76 (28)	.0067
	All 4 studies	108/276 (39)	83/294 (28)	.0056
Return of spontaneous circulation after in-hospital resuscitation	Ward[61]	6/16 (38)	3/17 (18)	.19
	Sack[42]	29/48 (60)	14/55 (25)	.00014
	Sack[62]	33/67 (49)	21/76 (28)	.0067
	All 3 studies	68/131 (52)	38/148 (26)	.000003
Survival to discharge, neurologically intact after in-hospital resuscitation	Ward[61]	1/16 (6)	0/17 (0)	.3017
	Sack[42]	8/48 (17)	3/55 (5)	.0700
	Both studies	9/64 (14)	3/72 (4)	.0453

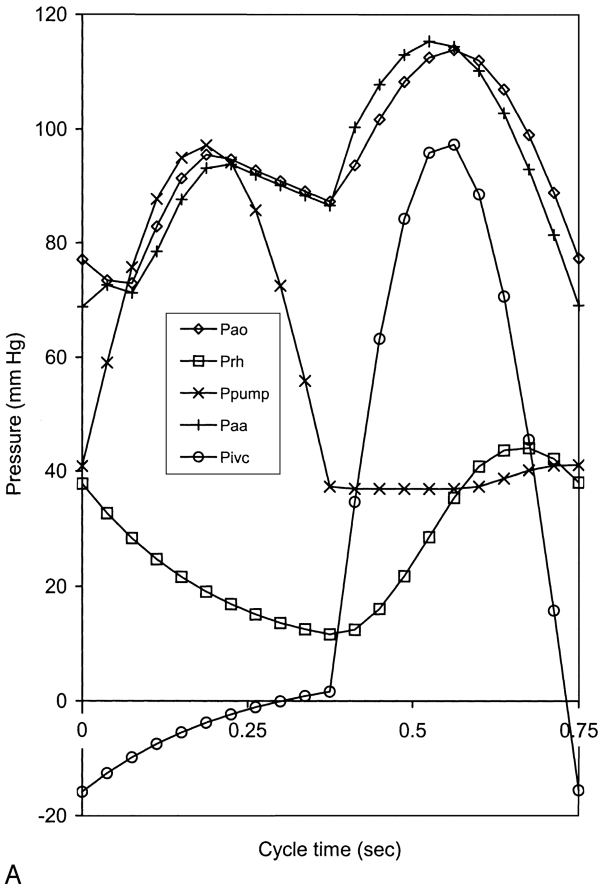

Figure 10. *See legend on opposite page*

sion during IAC-CPR to perfusion during standard CPR is 1.44 in a pure cardiac pump model.

Figure 10*B* illustrates the addition of 110 mm Hg IACs to 60 mm Hg peak thoracic compressions in a pure thoracic pump model, illustrating similar effects of the double pump approach when the thoracic and abdominal pump mechanisms are combined. The increased forward flow in this model is 2.1 L/min with IAC versus 0.93 L/min for thoracic pump CPR. Systemic perfusion pressure is 39 mm Hg versus 18 mm Hg for thoracic pump CPR. Thus, there is a roughly 2-fold ratio of perfusion during IAC-CPR to perfusion during standard CPR in a thoracic pump model versus a roughly 1.5-fold ratio of perfusion during IAC-CPR to perfusion during standard CPR in a cardiac pump model. If one takes the modern consensus view[20, 35, 54] that the operative pump mechanism in different animal and clinical models is a combination of the cardiac and thoracic pumps, these simulation results agree perfectly with the aggregate results of laboratory and clinical research on IAC-CPR, in which systemic perfusion pressure or carbon dioxide excretion served as measures of overall forward flow.[26, 43, 59, 61] These research studies found a 1.5- to 2-fold improvement in perfusion with the addition of IAC.

The safety of IACs, as reviewed previously,[43] has been well documented in 426 humans, 151 dogs, and 14 pigs. Only one isolated case report of traumatic pancreatitis in a child describes local trauma from abdominal compression during CPR.[64] These data compare favorably with the well-known and frequent incidence of rib fracture and pulmonary contusion from chest compression during CPR.[65–67] Increased emesis and aspiration from IAC have not been reported, and there

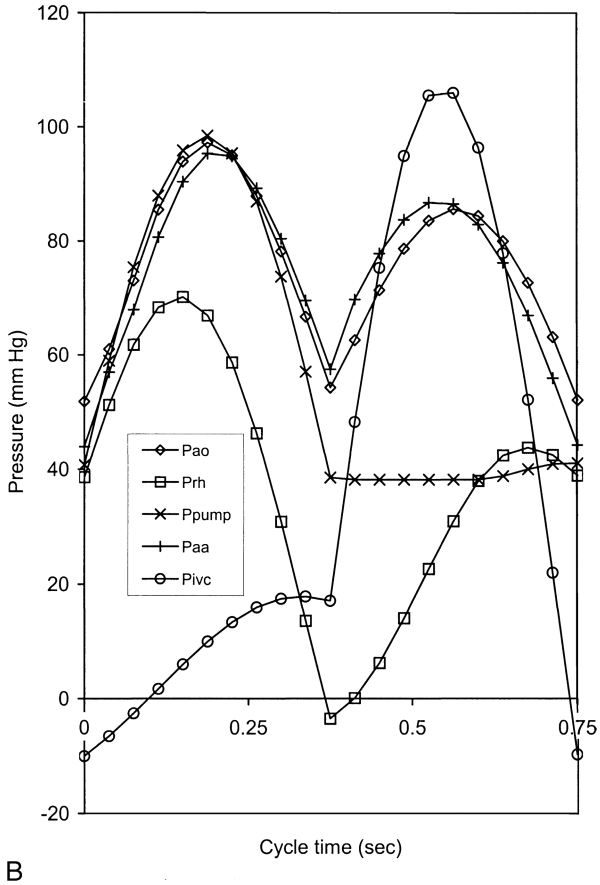

Figure 10. *(Continued).* Pressures in a mathematical model of the normal adult human circulation during cardiac arrest and IAC-CPR. The compression rate is 80 per minute. Maximal chest compression pressure is +60 mmHg. Maximal abdominal compression pressure is +110 mm Hg. *A*, IAC-CPR with a pure cardiac pump mechanism: flow = 3.6 L / min; SPP = 68 mm Hg. *B*, IAC-CPR with a pure thoracic pump mechanism: flow = 2.1 L/min; SPP = 39 mm Hg.

is even evidence that if positive abdominal pressure is applied during ventilations from the beginning of an arrest, the rate of gastric inflation before endotracheal intubation is reduced.[68]

Can we do even better? Theory suggests that the answer is "yes." Ordinary IAC-CPR, as studied experimentally over the past 20 years, includes only positive pressure compressions without active decompression. A logical extension of this line of thought is to examine full four-phase CPR, which includes active compression and active decompression of the chest and the abdomen. All four phases can be accomplished in a single cycle, because chest compression and abdominal decompression can be accomplished simultaneously in the first half of the cycle, and abdominal compression and chest decompression can be accomplished simultaneously in the second half of the cycle.

Recently a commercial venture has attempted to implement this concept in a device called the Lifestick (Datascope, Fairfield, NJ) (Fig. 11). The Lifestick is a mechanical device under development, with which one person can perform both chest and abdominal CPR. Compression is applied through a rocker arm with adhesive pads attached to the chest and the abdomen. The broad chest

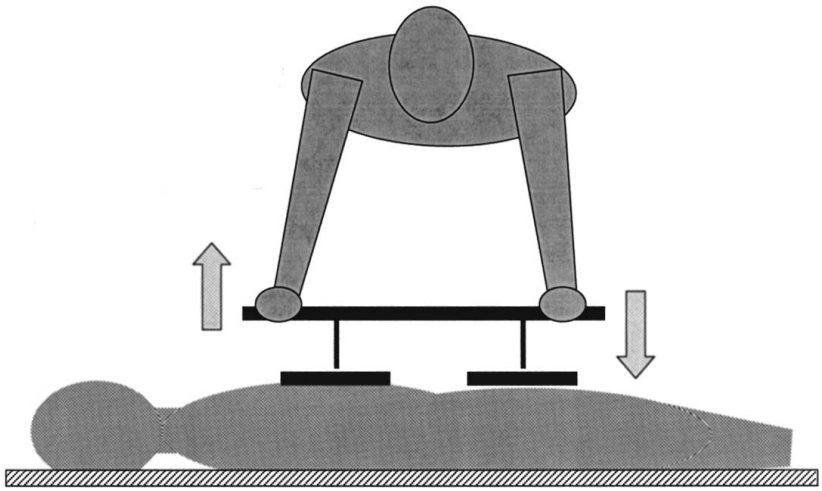

Figure 11. Four-phase CPR with a Lifestick (Datascope, Fairfield, NJ).

compression pad of early models precludes cardiac pump mechanism but also obviates rib fracture. Because the chest and abdominal pads are adhesive, it is possible to obtain a measure of upward retraction of the chest during the abdominal compression phase and vice versa by a rocking motion of the device. Such a device in principle has the advantage of permitting active compression and decompression of both chest and abdomen.[31]

The hemodynamic potential of such four-phase CPR is illustrated in Figure 12. Figure 12*A* shows cardiovascular pressures during cardiac pump and abdominal pump CPR with active compression and decompression. The four-phase method produces dramatically large pressure fluctuations in the chest and abdominal vascular compartments, although the external compression forces are no different than those used for standard CPR or conventional IAC-CPR, and the applied external decompression forces are substantially less than the compression forces. Nearly normal systemic perfusion pressures are obtained throughout the cycle. There is no restriction of positive coronary and systemic perfusion pressure to the diastolic phase. Forward blood flow is 4.6 L/min, approaching the textbook value of 5.0 L/min for the normal circulation in a 70-kg man. These levels are a far cry from the 20% of normal forward flow that has come to be expected from standard CPR.[69-70] Routine practical implementation of full four-phase CPR has yet to be achieved, partly as a result of legal and commercial wrangling unrelated to science and engineering; however, animal studies with a prototype Lifestick device are encouraging.[44]

The forgoing survey of resuscitation physiology suggests a significant potential for improvement of current CPR through the application of combinations of positive and negative pressure to the chest and the abdomen. The most recent American Heart Association guidelines[2] for CPR and emergency cardiovascular care permit the judicious use of two advanced forms of CPR at the discretion of a physician under conditions in which adequate equipment and trained personnel are available. These approved adjuncts include IAC-CPR and ACD-CPR. As so-called "class IIb" interventions, IAC- and ACD-CPR are considered within the standard of care, which reasonably prudent physicians can choose to use.[71] IAC-CPR and ACD-CPR are considered optional or alternative interventions by most experts. Technically these adjuncts could be performed together under the guidelines in selected clinical settings to achieve at least a three-phase CPR, which has hemodynamic benefits nearly as great as four-phase CPR.[31] This new state of affairs, in which emergency department physicians have considerable professional discretion, brings us to consider

practical aspects of implementing the new CPR adjuncts.

PRACTICAL ASPECTS OF ACTIVE COMPRESSION-DECOMPRESSION CARDIOPULMONARY RESUSCITATION

Active compression-decompression CPR requires use of a plungerlike device, sold as the CardioPump or ResQ-Pump (CPRx LLC, Minneapolis, MN). With a two-handed grip on the handles of the device, a rescuer can apply active compression and decompression. Teams in Europe, especially in Paris, have been trained to use the CardioPump with good results, including improved survival at 1 year after resuscitation compared with standard CPR.[72] Some studies have reported mixed results with the CardioPump,[73–77] perhaps as a result of less intensive training.[78–79] Those considering use of ACD-CPR are advised to train well in the use of the device and repeat training often.[78] Training mannequins and other educational materials are available from Ambu Inc. (Glostrup, Denmark).

At the time of this writing, the Ambu CardioPumps are not approved for use in the United States by the US Food and Drug Administration. They are commercially available outside the United States and are manufactured by Ambu, Inc. and marketed in the Western Hemisphere by CPRx LLC (Minneapolis, MN). The CardioPump weighs 0.58 kg and costs about $300. The vacuum cup is 135 mm in diameter and is made of soft silicone rubber. The cup can be removed for cleaning and for autoclave sterilization after use. The vacuum cup is not electrically conducting and does not interfere with transchest electrical defibrillation; it has been designed to adhere to a variety of chest anatomies, including wet hairy skin, without losing contact with the chest wall during decompression. Earlier models did not work well on women with larger breasts, [80–81] and the cup may still seal poorly in about 10% of women.

Rescuer fatigue is a known problem with ACD-CPR, because more energy is needed to perform ACD than to do active compression alone.[79, 82] Back fatigue is definitely reduced if the rescuer either kneels beside the thorax of the victim or works astride the victim on his or her knees. A blanket or pillow under the knees of the rescuer may allow the rescuer's arms and back to be straighter when using the CardioPump device.[72] Because the handle is offset from the chest by about 15 cm, the rescuer's shoulders need to be higher over the victim than in conventional CPR. If the patient is on a table or raised surface, a stool may be needed by the rescuer to provide the necessary elevation.

While using a CardioPump, the arms should be straight and the back of the rescuer as close to vertical as possible. The elbows should be locked. Wrists should be firm, such as in gripping a tennis racquet. In this position, the rescuer can work effectively by raising and lowering the hips against gravity. As the rescuer's hips are lowered in the kneeling position, the weight of the rescuer's body can be used to apply compression. As the rescuer's hips are raised, the quadriceps muscles of the anterior thigh can work to apply decompression while the rescuer's arms remain straight.*

As fatigue sets in, rescuers tend to revert to former habitual methods of chest compression without decompression, and the benefits of ACD-CPR are lost. Remembering to lift up during each cycle is the most challenging aspect of using a CardioPump device. A force gauge on the handle indicates the target ranges of compression and decompression force for chests of *soft*, *normal*, or *stiff* characteristics. The gauge provides useful biofeedback. The duty cycle for ACD-CPR is 50% compression time, 50% decompression time. Further details of ACD-CPR methodology and training are described by Wik et al.[78]

*The reader is encouraged to try this motion in the kneeling position while palpating the quadriceps, gluteus maximus, and hamstring muscles. Note that when the hips are raised when kneeling, the leg is extended and the knee joint by the quadriceps and the thigh is extended at the hip joint by the hamstrings. Posterior compartment (hamstring) muscles and the gluteus maximus muscles are also active in the kneeling position. Reliance on the large anterior and posterior thigh muscles and gluteus muscles minimizes fatigue and keeps the exercise aerobic for either male or female rescuers. These same muscles also should be used as much as possible in the standing position to minimize fatigue. Upper-body strength is not required, once rescuers learn to use leg muscles and *not* back muscles. Nevertheless, adequate personnel need to be available so that frequent changes can occur every 3 to 5 minutes to avoid fatigue.

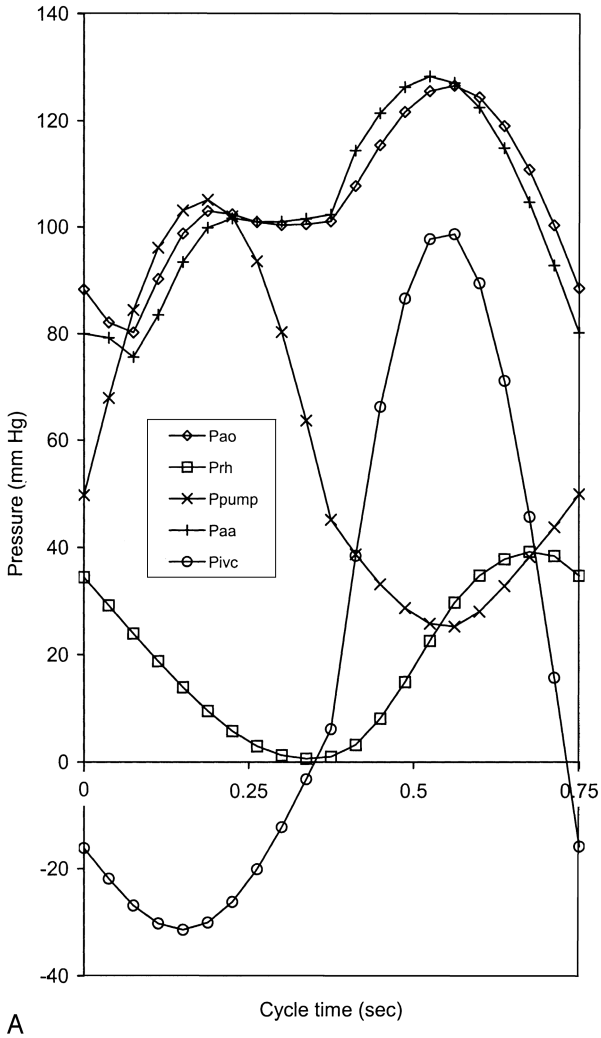

Figure 12. *See legend on opposite page*

A major potential improvement in ACD-CPR is the use of an inspiratory impedance valve[46] for victims with an established endotracheal tube.[51] This valve fits on the end of the endotracheal tube and prevents "decompression of the decompression," that is, an inrush of air into the trachea during the active decompression phase. The resulting increase in the magnitude of the intrathoracic decompression encourages even more chest pump priming. The valve incorporates a *pop-off* feature that is set to open when the intrathoracic pressure decreases below -22 cm H_2O. The valve prevents inflow only when intrathoracic pressure is zero to -22 cm H_2O with respect to atmospheric pressure. The valve does not interfere with normal PPVs or with vigorous spontaneous negative pressure ventilations, if the patient begins to gasp strongly enough to create at least 22 cm H_2O negative pressure. If a pulse returns, however, the inspiratory impedance valve should be removed immediately and the ventilation source reattached to the airway to allow normal spontaneous breathing. The inspiratory impedance valve is available commercially as

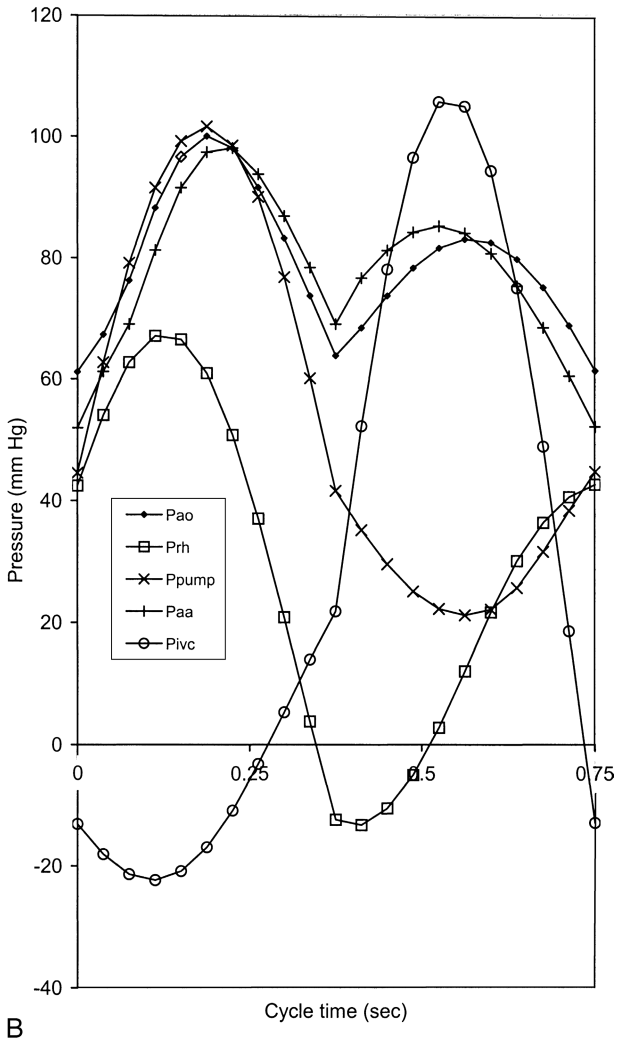

Figure 12. Pressures in a mathematical model of the normal adult human circulation during cardiac arrest and four-phase CPR. The compression rate is 80 per minute. Maximal chest compression pressure is +60 mm Hg. Maximal abdominal compression pressure is +110 mm Hg. Maximal chest decompression pressure is −20 mm Hg. Maximal abdominal decompression pressure is −30 mm Hg. *A*, Four-phase CPR with cardiac pump: flow = 4.6 L /min, SPP = 86 mm Hg. *B*, Four-phase Lifestick CPR with thoracic pump: flow 2.7 L/min; SPP = 51 mm Hg.

a resuscitation device called the Resusci-Valve or ResQ-Valve (CPRx LLC, Minneapolis, MN). The valve is a disposable device that costs between $30 and $50.

PRACTICAL ASPECTS OF INTERPOSED ABDOMINAL COMPRESSION—CARDIOPULMONARY RESUSCITATION

From a practical standpoint, the simplest, least expensive, and longest studied CPR adjunct is IAC-CPR. This method is essentially deviceless. There is no equipment cost, and there is no requirement for FDA approval. IAC-CPR can be performed by two hands of a third rescuer, as shown in Figure 9. IAC-CPR is especially appropriate for in-hospital resuscitations, where the dictum "When extra hands are free, do IAC" may well apply. IAC-CPR is performed most easily when the rescuers compressing the chest and abdomen are on opposite sides of the patient. Because the most favorable clinical results have been obtained when IAC-CPR is applied from the beginning of resuscitation, early application of the technique is to be encouraged. Use of IAC-CPR as a last-ditch effort after prolonged, failed advanced cardiac life support (ACLS) is not recommended and has not been shown to be effective.[61] Obviously, IAC is not recommended for pregnant women, persons suspected of having an abdominal aortic aneurysm, or persons known to have had recent abdominal surgery.

The abdominal compression technique in IAC-CPR involves rhythmic, manual compression of the mid-abdomen. The abdominal compression point is located in the midline, halfway between the xiphoid process and the umbilicus. The recommended force of abdominal compression is that sufficient to generate approximately 110 mm Hg external pressure on the abdominal aorta and vena cava and is equivalent to that required to optimally palpate the aortic pulse in a patient or volunteer when the heart is beating normally.[83] The technique for compression of the abdomen is similar in most respects to the technique for compression of the chest.

Hand Position and Compression Technique

The umbilicus should be visible and not covered by the compressing hands. Just as chest compression is applied making contact with the heel of one hand, which is covered by the heel of the other hand, abdominal compression is applied in the same manner. The heel of the rescuer's bottom hand should lie along the midline of the abdomen. This midline position keeps the main force of compression over the abdominal vena cava and aorta. The fingers may be either extended or interlaced but should be kept off the abdomen.

A straight-armed abdominal compression technique, similar to that used for chest compression, is ergonomically effective. Because the abdomen is composed of soft tissues without the bony resistance of the rib cage, adequate compressions do not require strong muscular effort. In practical training, students learn the force required to produce safe and effective 110 mm Hg pressure pulses within the abdomen using a folded blood pressure cuff and gauge (see later discussion). The depth of abdominal compression is not unlike that of chest compression, 1.5 to 2 inches, in a normal sized, nonobese adult. For obese patients, greater compression depth may be necessary. Compressions can be delivered to the abdominal compression point straight down in the vertical plane, or to maximize aortic counterpulsation, from the left side of the victim at 11° left of vertical.[83] In principle, when IAC-CPR is performed properly, the carotid or femoral pulse demonstrates double peaks, denoting both chest and abdominal compressions.[31, 55] The clinical utility of assessing a double pulse in this setting, however, has not been investigated.

Coordination of Chest and Abdominal Compressions

The rhythm of abdominal compression is natural and easy to learn. If the chest compressor counts "one-*and*-two-*and*-three . . . ," the abdominal rescuer applies pressure during "*and*," and releases pressure during each counted number. The release of abdominal

pressure between chest compressions is needed for chest and abdominal pump mechanisms to work together[31] and also to avoid entrapment of the right lobe of the liver under the sternum when the chest is compressed. Thus, abdominal pressure should be applied whenever chest compressions are relaxed, and abdominal pressure should be relaxed whenever chest compressions are applied. Abdominal pressure can maintained during ventilations to minimize gastric inflation if no endotracheal tube is in place.[68] The most widely used and tested duty cycle is 50% chest/50% abdominal compression time. There is some theoretical advantage to 30% chest/70% abdominal compression time[30], but this variation has not yet been tested in humans.

If two-rescuer CPR has been established and a third rescuer becomes available, this person should take a position on the opposite side of the patient from the second rescuer who is performing chest compressions, locate proper hand position, sense the rhythm of chest compressions, and begin then IACs. If rescuers doing chest and abdominal compressions wish to switch positions (abdominal compressions are less tiring, because there is no ribcage resistance), it is natural for them to slide headward and footward, respectively, during a ventilation, because they are on opposite sides of the patient.

Training Individuals To Do Interposed Abdominal Compression–Cardiopulmonary Resuscitation

A detailed analysis of the ergonomic complexity of IAC (available from the author on request) suggests that it is no more complicated to learn and perform than is opening an airway. The following modifications of whole body mannequins for teaching three-rescuer IAC-CPR are performed easily and are inexpensive:

1. Add extra foam rubber to the lower thoracic compartment of the mannequin and to the abdominal compartment to simulate subcostal soft tissues.
2. Wrap a standard blood pressure cuff around a towel and place it in the abdominal compartment beneath the abdominal compression point (5 cm headward of the umbilicus) in the midline. Bring the tubing, aneroid manometer gauge, and squeeze bulb out at beltline of the mannequin so that the gauge is visible to the trainees. Tape target pressure markers on the manometer dial at 120 mm Hg. Inflate cuff to a resting pressure of 10 mm Hg.
3. During practice sessions coach trainees to hit the target pressure during IAC. The issue of how hard to press has been addressed in detail elsewhere.[83]

If a mannequin is not available, a rolled blood pressure cuff placed inside a rolled towel is a useful training model. Successful practice sessions can be conducted in as little as 5 minutes. The following sequence may be used:

1. The abdominal rescuer takes position on opposite side of victim from chest compressor.
2. Chest compressor says, "You press here (pointing to the abdominal compression point) whenever I release."
3. Chest compressor counts "one-*and*-two-*and*-three. . . ." Abdominal rescuer applies pressure during "*and*."
4. Start with slow-motion practice, and then gradually increase to a normal compression rate. With this kind of practice session, health care professionals can be taught to perform IAC with little time or trouble.

Combining Interposed Abdominal Compression with Active Compression-Decompression–Cardiopulmonary Resuscitation

In selected research settings, there is good reason to investigate combining the methods of ACD and IAC-CPR to create three-phase CPR. Such a method is practically achievable with current technology, omitting only the fourth phase, active abdominal decompres-

sion, from full four-phase CPR. Advance experience with the two techniques separately, is prudent before attempting to combine them. Moreover, full consideration of the following warnings and caveats is in order.

WARNINGS ABOUT THE GENERAL USE OF CIRCULATORY ADJUNCTS

Because of the requirement for special training and sometimes special equipment, advanced forms of CPR are best performed in a hospital by trained health care providers.

Endotracheal intubation is prudent before instituting CPR adjuncts. Most studies of IAC-CPR have required that an endotracheal tube be in place for fear of regurgitation in response to IAC (a fear not substantiated by data[43]). Similarly, an endotracheal tube is desirable if ACD-CPR is performed with an inspiratory impedance valve. The valve can be used with a tightly sealed facemask, but extra care must be taken to avoid leaks.

It is extremely important in one's enthusiasm for CPR adjuncts *not* to delay electrical ventricular defibrillation when ventricular fibrillation (VF) is present. New data confirm the old[84, 85]: that if VF has been present for less than 4 to 5 minutes, electrical defibrillation is clearly the treatment of choice. For longer down-times however, there is evidence that a vasopressor drug plus effective CPR, which produces good coronary perfusion pressure, can improve resuscitation success. As long as defibrillation is not delayed, enhanced CPR methods, such as IAC-CPR, can potentially benefit patients in the hands of trained health care providers.

If initial shocks are not successful for VF, the rescuer should institute adjuncts early—*not* as a last resort after failed ACLS. Adjuncts do not work on dead people. Their use after failed ACLS will only lead to unjustified cynicism.

With more powerful and effective CPR modalities, one can expect an increased number of discharge survivals[72, 86]; however, one also must expect an increased number of individuals who survive only briefly. It is probably an unavoidable result of multiplied probabilities that, in addition to greater numbers of lives saved, there also could be greater numbers of people who are resuscitated and subsequently die a day or so later. It is even possible, although not yet shown by published data, that more effective techniques of resuscitation might produce increased numbers of individuals resuscitated to a lingering vegetative state. Clinical experience with the new modalities will be needed to determine which individuals are at greatest risk for this most undesirable outcome. IAC- or ACD-CPR cannot change underlying pathology or control the quality of care after resuscitation. There is however, theoretical, experimental, and clinical evidence that these improved, more energetic, and more aggressive resuscitation methods can improve blood flow during CPR, and in turn the probability of neurologically intact short- and long-term survival.

SUMMARY

Principles of cardiovascular physiology tell us that during cardiac arrest and CPR, forward flow of blood can be generated by external compression or decompression of either the chest or the abdomen. Standard CPR utilizes only one of these modes—chest compression—and generates roughly 1 L/min forward flow in an adult human,[45] which is 20% of normal cardiac output. IAC-CPR uses two of these modes—chest compression and abdominal compression—and generates roughly twice the forward flow, or 2 L/min in an adult human.[61] ACD-CPR uses two of these modes—chest compression and chest decompression—and also generates roughly twice the forward flow as standard CPR,[51] although the results are somewhat model dependent. The studies by Sack et al[86] with IAC-CPR and by Plaisance et al[72] with ACD-CPR suggest that when methods that double perfusion are employed methodically, resuscitation outcome in terms of short- and long-term survival are also roughly doubled.[72, 86] This state of affairs is fortunate, because it is possible that factors, such as severe underlying disease or the quality of postresuscitation care, could blunt or cancel positive effects of improved blood flow during the brief resusci-

tation period. Theoretically, full four-phase CPR, including active compression and decompression of both chest and abdomen, is capable of generating 4 L/min forward flow or greater, which is 80% of normal, and there is a reasonable prospect of achieving 100% of normal flow under conditions in which all four phases are optimized.[87] Standard CPR is clearly not the ultimate form of external CPR. There is real, credible evidence that substantial improvements in resuscitation methods and results will be possible in the next decade.

References

1. Kouwenhoven WB, Jude JR, Knickerbocker GG: Closed-chest cardiac massage. JAMA 173:1064–1067, 1960
2. Cummins RO: Guidelines 2000 for cardiopulmonary resuscitation and emergency cardiovascular care: International consensus on science. Circulation 102(suppl I):I-1–I-384, 2000
3. Fitzgerald KR, Babbs CF, Frissora HA, et al: Cardiac output during cardiopulmonary resuscitation at various compression rates and durations. American Journal of Physiology 241:H442–H448, 1981
4. Idris AH: Reassessing the need for ventilation during CPR. Ann Emerg Med 27:569–575, 1996
5. Jude JR, Kouwenhoven WB, Knickerbocker GG: Cardiac arrest: Report of application of external cardiac massage on 118 patients. JAMA 178:1063–1070, 1961
6. Kuisma M, Alaspaa A: Out-of-hospital cardiac arrests of non-cardiac origin: Epidemiology and outcome. Eur Heart J 18:1122–1128, 1997
7. Cobb LA, Werner JA, Trobaugh GB: Sudden cardiac death II: Outcome of resuscitation, management, and future directions. Modern Concepts of Cardiovascular Disease 49:37–42, 1980
8. Eisenberg MS, Hallstrom A: Long-term survival after out-of-hospital cardiac arrest. N Engl J Med 306:1340–1343, 1982
9. Weil MH, Tang W: Cardiopulmonary resuscitation: A promise as yet largely unfulfilled. Disease-a-month 43:429–501, 1997
10. Redding JS: Abdominal compression in cardiopulmonary resuscitation. Anesthesia and Analgesia 50:668–675, 1971
11. Ralston SH, Voorhees WD, Babbs CF: Intrapulmonary epinephrine during cardiopulmonary resuscitation: Improved regional blood flow and resuscitation in dogs. Ann Emerg Med 13:79–86, 1984
12. Kern KB, Ewy GA, Voorhees WD, et al: Myocardial perfusion pressure: A predictor of 24-hour survival during prolonged cardiac arrest in dogs. Resuscitation 16:241–250, 1988
13. Mair P, Furtwaengler W, Baubin M: Aortic-valve function during cardiopulmonary resuscitation. N Engl J Med 329:1965–1966, 1993
14. Criley JM, Blaufuss AH, Kissel GL: Cough-induced cardiac compression: A self-administered form of cardiopulmonary resuscitation. JAMA 236:1246–1250, 1976
15. Criley JM, Niemann JT, Rosborough JP, et al: The heart is a conduit in CPR. Crit Care Med 9:373–374, 1981
16. Rudikoff MT, Maughan WL, Effron M, et al: Mechanisms of blood flow during cardiopulmonary resuscitation. Circulation 61:345–352, 1980
17. Weisfeldt ML: Physiology of cardiopulmonary resuscitation. Ann Rev Med 32:435–442, 1981
18. Feneley MP, Maier GW, Gaynor JW, et al: Sequence of mitral valve motion and transmitral blood flow during manual cardiopulmonary resuscitation in dogs. Circulation 76:363–375, 1987
19. Gall F: Incompetence of the atrioventricular valves during cardiac massage. J Cardiovasc Surg 6:356–360, 1965
20. Paradis NA, Martin GB, Goetting MG, et al: Simultaneous aortic, jugular bulb, and right atrial pressures during cardiopulmonary resuscitation in humans: Insights into mechanisms. Circulation 80:361–368, 1989
21. Rainer EH, Bullough J: Respiratory and cardiac arrest during anaesthesia in children. BMJ 1024–1028, 1957
22. Ohomoto T, Miura I, Konno S: A new method of external cardiac massage to improve diastolic augmentation and prolong survival time. Ann Thorac Surg 21:284–290, 1976
23. Beyar R, Kishon Y, Kimmel E, et al: Intrathoracic and abdominal pressure variations as an efficient method for cardiopulmonary resuscitation: Studies in dogs compared with computer model results. Cardiovasc Res 19:335–342, 1985
24. Babbs CF, Geddes LA: Effects of abdominal counterpulsation in CPR as demonstrated in a simple electrical model of the circulation. Ann Emerg Med 12:247, 1983
25. Coletti RH, Kaskel PS, Cohen SR, et al: Abdominal counterpulsation (AC): A new concept in circulatory assistance. Trans Am Soc Artif Intern Organs 28:563–566, 1982
26. Ralston SH, Babbs CF, Niebauer MJ: Cardiopulmonary resuscitation with interposed abdominal compression in dogs. Anesthesia and Analgesia 61:645–651, 1982
27. Beyar R, Kishon Y, Sideman S, et al: Computer studies of systemic and regional blood flow during cardiopulmonary resuscitation. Medical & Biological Engineering and Computing 22:499–506, 1984
28. Babbs CF, Weaver JC, Ralston SH, et al: Cardiac, thoracic, and abdominal pump mechanisms in CPR: Studies in an electrical model of the circulation. Am J Emerg Med 2:299–308, 1984
29. Babbs CF, Ralston SH, Geddes LA: Theoretical advantages of abdominal counterpulsation in CPR as demonstrated in a simple electrical model of the circulation. Ann Emerg Med 13:660–671, 1984
30. Babbs CF, Thelander K: Theoretically optimal duty cycles for chest and abdominal compression during external cardiopulmonary resuscitation. Acad Emerg Med 2:698–707, 1995
31. Babbs CF: CPR techniques that combine chest and abdominal compression and decompression: Hemodynamic insights From a spreadsheet model. Circulation 100:2146–2152, 1999
32. Maier GW, Newton JR, Wolfe JA, et al: The influence of manual chest compression rate on hemodynamic support during cardiac arrest: High-impulse cardiopulmonary resuscitation. Circulation 74(suppl IV):IV-51–IV-59, 1986

33. Maier GW, Tyson GS, Olsen CO, et al: The physiology of external cardiac massage: High-impulse cardiopulmonary resuscitation. Circulation 70:86–101, 1984
34. Babbs CF: High-impulse compression CPR: Simple mathematics points to future research. Acad Emerg Med 1:418–422, 1994
35. Halperin HR, Tsitlik JE, Guerci AD, et al: Determinants of blood flow to vital organs during cardiopulmonary resuscitation in dogs. Circulation 73:539–550, 1986
36. Halperin HR, Tsitlik JE, Beyar R, et al: Intrathoracic pressure fluctuations move blood during CPR: Comparison of hemodynamic data with predictions from a mathematical model. Ann Biomed Eng 15:385–403, 1987
37. Tucker KJ, Idris A: Clinical and laboratory investigations of active compression-decompression cardiopulmonary resuscitation [editorial]. Resuscitation 28:1–7, 1994
38. Tucker KJ, Khan JH, Savitt MA: Active compression-decompression resuscitation: Efects on pulmonary ventilation. Resuscitation 26:125–131, 1993
39. Babbs CF, Tacker WA: Cardiopulmonary resuscitation with interposed abdominal compression. Circulation 74(suppl IV):37–41, 1986
40. Babbs CF: Interposed abdominal compression-CPR: A case study in cardiac arrest research. Ann Emerg Med 22:24–32, 1993
41. Einagle V, Bertrand F, Wise RA, et al: Interposed abdominal compressions and carotid blood flow during cardiopulmonary resuscitation: Support for a thoracoabdominal unit. Chest 93:1206–1212, 1988
42. Sack JB, Kesselbrenner MB, Bregman D: Survival from in-hospital cardiac arrest with interposed abdominal counterpulsation during cardiopulmonary resuscitation. JAMA 267:379–385, 1992
43. Sack JB, Kesselbrenner MB: Hemodynamics, survival benefits, and complications of interposed abdominal compression during cardiopulmonary resuscitation. Acad Emerg Med 1:490–497, 1994
44. Tang W, Weil MH, Schock RB, et al: Phased chest and abdominal compression-decompression: A new option for cardiopulmonary resuscitation. Circulation 95:1335–1340, 1997
45. DelGuercio L, Feins NR, Cohn JD, et al: Comparison of blood flow during external and internal cardiac massage in man. Circulation 31(suppl I):171–180, 1965
46. Lurie KG, Coffeen P, Shultz J, et al: Improving active compression-decompression cardiopulmonary resuscitation with an inspiratory impedance valve. Circulation 91:1629–1632, 1995
47. Lindner KH, Pfenninger EG, Lurie KG, Schurmann W, Lindner IM, Ahnefeld FW: Effects of active compression-decompression resuscitation on myocardial and cerebral blood flow in pigs. Circulation 88:1254–1263, 1993
48. Lurie KG, Lindo C, Chin J: CPR: The P stands for plumber's helper. JAMA 264:1661, 1990
49. Cohen TJ, Tucker KJ, Lurie KG, et al: Active compression-decompression: A new method of cardiopulmonary resuscitation: Cardiopulmonary Resuscitation Working Group. JAMA 267:2916–2923, 1992
50. Chang MW, Coffeen P, Lurie KG, et al: Active compression-decompression CPR improves vital organ perfusion in a dog model of ventricular fibrillation. Chest 106:1250–1259, 1994
51. Plaisance P, Lurie KG, Payen D: Inspiratory impedance during active compression-decompression cardiopulmonary resuscitation: A randomized evaluation in patients in cardiac arrest. Circulation 101:989–994, 2000
52. Wik L, Naess PA, Ilebekk A, et al: Effects of various degrees of compression and active decompression on haemodynamics, end-tidal CO2, and ventilation during cardiopulmonary resuscitation of pigs. Resuscitation 31:45–57, 1996
53. Sunde K, Wik L, Naess PA, et al: Effect of different compression–decompression cycles on haemodynamics during ACD-CPR in pigs. Resuscitation 36:123–131, 1998
54. Chandra NC: Mechanisms of blood flow during CPR. Ann Emerg Med 22:281–288, 1993
55. Berryman CR, Phillips GM: Interposed abdominal compression-CPR in human subjects. Ann Emerg Med 13:226–229, 1984
56. Lindner KH, Ahnefeld FW, Bowdler IM: Cardiopulmonary resuscitation with interposed abdominal compression after asphyxial or fibrillatory cardiac arrest in pigs. Anesthesiology 72:675–681, 1990
57. Babbs CF: Interposed abdominal compression-cardiopulmonary resuscitation: Are we missing the mark in clinical trials? [editorial]. Am Heart J 126:1035–1041, 1993
58. Rosborough JP, Niemann JT, Criley JM, et al: Lower abdominal compression with synchronized ventilation: A CPR modality. Circulation 64(IV):303, 1981
59. Voorhees WD, Niebauer MJ, Babbs CF: Improved oxygen delivery during cardiopulmonary resuscitation with interposed abdominal compressions. Ann Emerg Med 12:128–135, 1983
60. Babbs CF, Sack JB, Kern KB: Interposed abdominal compression as an adjunct to cardiopulmonary resuscitation. Am Heart J 127:412–421, 1994
61. Ward KR, Sullivan RJ, Zelenak RR, et al: A comparison of interposed abdominal compression CPR and standard CPR by monitoring end-tidal PCO2. Ann Emerg Med 18:831–837, 1989
62. Sack JB, Kesselbrenner MB, Jarrad A: Interposed abdominal compression CPR and resuscitation outcome during asystole and electromechanical dissociation. Circulation 86:1692–1700, 1992
63. Mateer JR, Steuven HA, Thompson BM, et al: Prehospital IAC-CPR versus standard CPR: Paramedic resuscitation of cardiac arrests. Am J Emerg Med 3:143–146, 1985
64. Waldman PJ, Walters BL, Grunau C: Pancreatic injury associated with interposed abdominal compressions in pediatric cardiopulmonary resuscitation. Am J Emerg Med 2:510–512, 1984
65. Clark DT: Complications following closed-chest cardiac massage. JAMA 181:337–338, 1962
66. Nagel EL, Fine EG, Krischer JP, et al: Complications of CPR. Critical Care Medicine 9:424, 1981
67. Silberberg B, Rachmaninoff N: Complications following external cardiac massage. Surg Gynecol Obstet 119:6–10, 1964
68. Babbs CF, Schoenlein WE, Lowe MW: Gastric insufflation during IAC-CPR and standard CPR in a canine model. Am J Emerg Med 3:99–103, 1985
69. Standards and guidelines for cardiopulmonary resuscitation (CPR) and emergency cardiac care (ECC). JAMA 244:453–509, 1980
70. Silver DI, Murphy RJ, Babbs CF, et al: Cardiac output during CPR: A comparison of two methods. Crit Care Med 9:419–420, 1981
71. Cummins RO: Introduction to the international guidelines 2000 for CPR and ECC. Circulation 102(suppl I):I-1–I-11, 2000

72. Plaisance P, Adnet F, Vicaut E, et al: Benefit of active compression-decompression cardiopulmonary resuscitation as a prehospital advanced cardiac life support: A randomized multicenter study. Circulation 95:955–961, 1997
73. Panzer W, Bretthauer M, Klingler H, et al: ACD versus standard CPR in a prehospital setting. Resuscitation 33:117–124, 1996
74. Schwab TM, Callaham ML, Madsen CD, et al: A randomized clinical trial of active compression-decompression CPR versus standard CPR in out-of-hospital cardiac arrest in two cities. JAMA 273:1261–1268, 1995
75. Luiz T, Ellinger K, Denz C: Active compression-decompression cardiopulmonary resuscitation does not improve survival in patients with prehospital cardiac arrest in a physician-manned emergency medical system. J Cardiothorac Vasc Anesth 10:178–186, 1996
76. Stiell IG, Hebert PC, Wells GA, et al: The Ontario trial of active compression-decompression cardiopulmonary resuscitation for in-hospital and prehospital cardiac arrest. JAMA 275:1417–1423, 1996
77. Mauer DK, Nolan J, Plaisance P, et al: Effect of active compression-decompression resuscitation (ACD-CPR) on survival: A combined analysis using individual patient data. Resuscitation 41:249–256, 1999
78. Wik L, Schneider T, Baubin M, et al: Active compression-decompression cardiopulmonary resuscitation: Instructor and student manual for teaching and training: Part II: A student and instructor manual. Resuscitation 32:206–212, 1996
79. Wik L, Mauer D, Robertson C: The first European pre-hospital active compression-decompression (ACD) cardiopulmonary resuscitation workshop: A report and a review of ACD-CPR. Resuscitation 30:191–202, 1995
80. Rabl W, Baubin M, Haid C, et al: Review of active compression-decompression cardiopulmonary resuscitation (ACD-CPR): Analysis of iatrogenic complications and their biomechanical explanation. Forensic Sci Int 89:175–183, 1997
81. Klintschar M, Darok M, Radner H: Massive injury to the heart after attempted active compression-decompression cardiopulmonary resuscitation. Int J Legal Med 111:93–96, 1998
82. Baubin M, Schirmer M, Nogler M, et al: Rescuer's work capacity and duration of cardiopulmonary resuscitation. Resuscitation 33:135–139, 1996
83. Babbs CF: IAC-CPR: Are we missing the mark in clinical trials? Am Heart J 126:1035–1041, 1993
84. Yakaitis RW, Ewy GA, Otto CW, et al: Influence of time and therapy on ventricular defibrillation in dogs. Crit Care Med 8:157–163, 1980
85. Marwick TH, Case C, Siskind V, et al: Adverse effect of early high-dose adrenaline on outcome of ventricular fibrillation. Lancet 2:66–68, 1988
86. Sack JB, Kesselbrenner MB, Bregman D: Survival from in-hospital cardiac arrest with interposed abdominal counterpulsation during cardiopulmonary resuscitation. JAMA 267:379–385, 1992
87. Babbs CF: Efficacy of interposed abdominal compression-cardiopulmonary resuscitation (CPR), active compression and decompression-CPR and Lifestick CPR: Basic physiology in a spreadsheet model. Crit Care Med 28:N199–202, 2000

Address reprint requests to

Charles F. Babbs, MD, PhD
Department of Basic Medical Sciences
Purdue University
West Lafayette, IN 47907–1246

e-mail: babbs@purdue.edu