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1994

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Interposed abdominal compression as an adjunct to cardiopulmonary resuscitation

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ABSTRACT

The addition of interposed abdominal compression (IAC) to otherwise standard CPR provides external pressure over the abdomen in counterpoint to the rhythm of chest compression. Interposed abdominal compression is a simple manual technique that can supplement the use of adrenergic drugs to increase both coronary perfusion pressure and total blood flow during CPR. Mechanistically, manual abdominal compressions induce both central aortic and central venous pressure pulses. However, owing to differences in venous versus arterial capacitance, the former are usually greater than the latter, so that systemic perfusion pressure is enhanced. Moreover, practical experience and theoretical analysis have suggested subtle refinements in the hand position and technique for abdominal compression that may further improve the ratio of arterial to venous pressure augmentation. Clinical studies confirm that IAC-CPR can improve perfusion pressures and carbon dioxide excretion during CPR in humans. The incidence of abdominal trauma, regurgitation, or other complications is not increased by IAC. Randomized trials have shown that short-term and long-term survival of patients resuscitated in the hospital by IAC-CPR are about twice that of control patients resuscitated by standard CPR. The technique of IAC has thus evolved to become a highly promising adjunct to normal CPR, which is likely to be implemented in an increasing number of clinical protocols.

Key words: abdomen, blood flow, CPR, review, survival

INTRODUCTION

Even during optimally performed standard cardiopulmonary resuscitation (CPR), total blood flow is only 20% to 35% of normal,[1-4] and coronary perfusion pressure is often less than the 15 to 30 mm Hg gradient that is necessary to permit the return of spontaneous circulation from more than momentary cardiac arrest[2, 5-7] One alternative means to elevate perfusion of vital organs and to augment systemic perfusion pressure, despite low total flow, is the application of interventions that increase peripheral vascular resistance.[8] The positive influence of raised peripheral vascular resistance, produced either by α-adrenergic drugs or by mechanical abdominal binding, was first noted in animal models of cardiac arrest by Pearson and Redding over two decades ago.[5, 9]

Recent efforts aimed at improving resuscitation outcome have continued to focus on pharmacologic and mechanical measures that increase vital organ perfusion through raised peripheral resistance during CPR. Pharmacologically, Redding et al.,[10] Brown et al.,[11] and Stell et al.[12] have explored the ability of higher dose epinephrine and alternative adrenergic drugs, such as methoxamine, to produce peripheral vasoconstriction. The maximal benefit of adrenergic stimulation during cardiac arrest, however, is likely already provided either by high endogenous catecholamine release (three hundredfold normal plasma levels)[13] or by currently recommended doses of exogenous epinephrine. Indeed, two recent large-scale trials[11, 12] have failed to reveal any difference in the outcome of patients receiving high-dose (0.2 mg/kg) epinephrine, compared with patients receiving standard- dose (0.1 mg/kg) epinephrine during CPR.

Mechanically, several groups[5, 14-17] have explored the use of static abdominal binding to inhibit diastolic runoff from the thoracic aorta to nonvital vascular beds, and in turn to raise the aortic-to-right atrial pressure gradient driving perfusion of the heart and brain. The major difficulty with static abdominal binding or continuous abdominal pressure as a means to achieve raised peripheral resistance during CPR is the danger that fatal hepatic trauma may occur as a result of splinting of the liver beneath the sternum at the time that chest compression is applied.[14] The extensive literature on this subject has been reviewed elsewhere in detail[18] and is summarized in Table I. All published investigations report a hemodynamic benefit of abdominal compression for periods of several minutes or more, but there is disagreement about the likelihood of liver damage with intraperitoneal hemorrhage that would be sufficient to make long-term resuscitation impossible.

Table I. Meta-analysis of the safety of static abdominal binding

Studies suggesting CPR with static abdominal pressure causes liver rupture	*Studies suggesting CPR with static abdominal pressure does not cause liver rupture*
Birch[15] 1962; continuous manual pressure, two dogs*	Redding[5] 1971; blood pressure cuff around abdomen, 65 dogs
Harris[14] 1967; continuous manual pressure (upper abdomen), six dogs	Rudikoff[38] 1980; inflatable bladder and adhesive tape from xiphoid to iliac crests, 15 dogs
Bircher[16] 1980; pediatric MAST suit, six dogs	Bircher[54] 1980; pediatric MAST suit 12 dogs
	Chandra[17] 1981; bladder and corset from ribs to iliac crests, 10 patients

*Numbers of dogs refer to animals subjected to abdominal pressurization, excluding control groups. Complete citations to tabulated studies are found in Reference 18.

One especially attractive means of securing the hemodynamic benefits of abdominal pressurization, while maintaining a low risk of visceral trauma, is to compress the abdomen only during the diastolic phase of chest compression. This concept of interposed abdominal compression-CPR (IAC-CPR) is based upon the idea that one can achieve the efficacy of abdominal binding by compressing the abdomen during the intervals between chest compressions, while avoiding entrapment of the liver between the sternum and the spine as the chest is compressed.

Fig. 1 illustrates application of the technique in man. Abdominal pressure is applied by an additional rescuer over the mid abdomen with two hands, beginning at the time of release of one chest compression and ending at the time of onset of the next chest compression. Abdominal pressure is always released when the chest is being actively compressed, permitting the diaphragm and liver to move in response to transphrenic pressure gradients. Abdominal compression is also maintained during ventilatory pauses, thus reducing the possibility of gastric insufflation.[19]
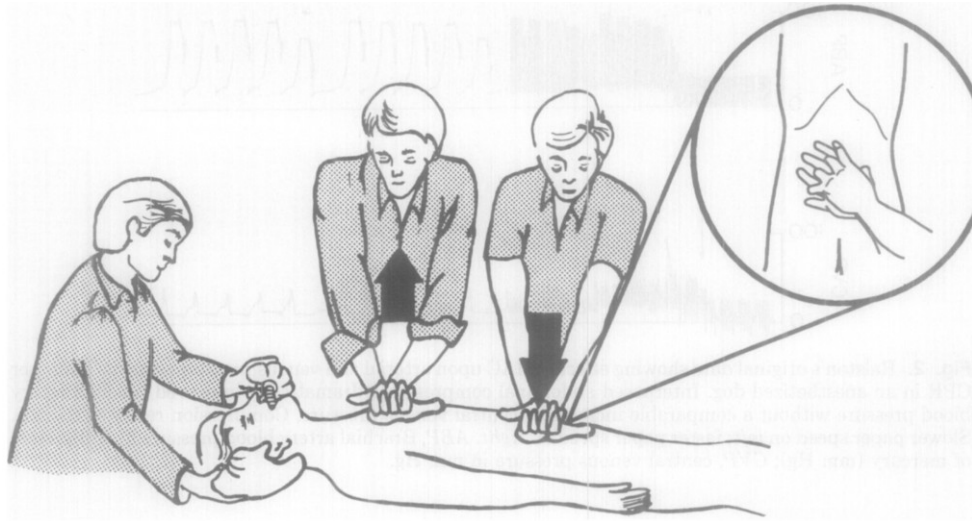
Fig. 1. Artist's conception of three rescuers performing IAC-CPR. For clarity, rescuers are shown on the same side of the victim. With three rescuers, ventilation, chest compression, and abdominal compression ere each performed by a single individual. The central abdominal over-and-underhand position for abdominal compression, illustrated here, is that employed in a recent successful clinical studty[48]; however, this hand position is not necessarily the most effective possible.[34]

Good results have been obtained with negligible trauma in animals using applied abdominal pressures of 120 to 150 mm Hg, sensed by a folded blood pressure cuff connected to an aneroid manometer and placed between the hands of the rescuer and the abdomen.[20, 21] During the past decade, the effectiveness and safety of such manual abdominal counterpulsation, as well as the hemodynamic mechanisms invoked by the technique, have been well explored in both laboratory and clinical settings.

LABORATORY STUDIES

The hemodynamic effects of abdominal counterpulsation were discovered independently by several scattered research teams. Manual compression of the thoracic aorta, alternating with internal cardiac massage, was first described by Molokhia et a1.[22] in an open-chest canine model. These investigators found that the addition of interposed digital aortic compression during open-chest cardiac massage significantly improved coronary sinus blood flow. In a canine model of external CPR, Ohomoto et a1.[23] described an arrangement of two mechanical pistons, one that compressed the closed chest and another that compressed the abdomen. They called the technique "countermassage" and reported that phased abdominal compression, combined with chest compression, improved mean aortic pressure and short term survival in anesthetized dogs with ventricular fibrillation.

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Ralston et al.[20] were the first in North America to discover the ability of manually interposed abdominal compressions to raise systolic and diastolic arterial pressure during external CPR. In pilot studies they originally planned to test a manual version of sustained abdominal binding as an adjunct to Thumper® CPR in dogs (Michigan Instruments, Grand Rapids, Mich.). To avoid liver damage by the powerful downstroke of the gas-powered piston, Ralston et al. deliberately limited application of manual abdominal pressure to periods when chest compression was being released. The originally observed effects upon central arterial and venous blood pressures were dramatic (Fig. 2), and have been repeatedly demonstrated in subsequent studies both in animals and in human subjects.[21, 24-27]
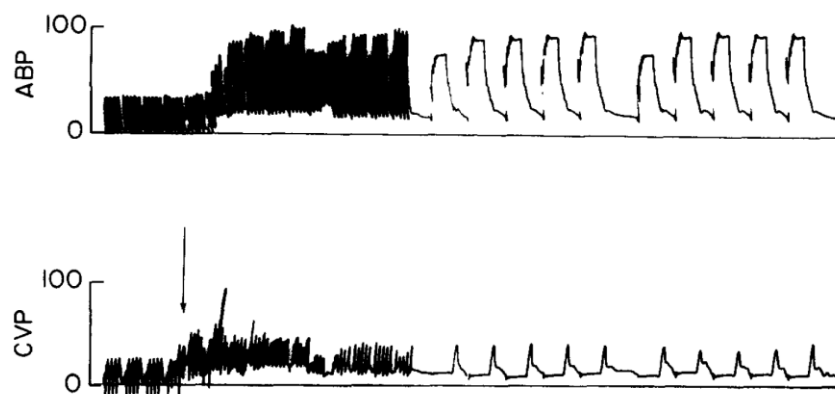

Fig. 2. Ralston's original data showing effects of IAC upon arterial and venous pressures during Thumper CPR in an anesthetized dog. Interposed abdominal compressions dramatically improved brachial artery blood pressure without a comparable increase in central venous pressure. Compression rate was 60/min. Slower paper speed on left; faster paper speed on right. ABP, brachial artery blood pressure in millimeters of mercury (mm Hg); CVP, central venous pressure in mm Hg.

Blood flow augmentation

A subsequent study by Ralston et al.[20] in 10 dogs with electrically induced fibrillation showed that IAC-CPR significantly improved cardiac output and diastolic arterial pressures when combined with pressure-limited ventilation. Voorhees et al.[21] extended this line of research further to show an approximate doubling of cardiac output, diastolic arterial pressure, and diastolic arteriovenous pressure difference, with significant improvement in total body oxygen delivery when IAC was added to standard CPR, performed with constant volume ventilation.

A visual demonstration of flow augmentation by IAC-CPR was provided by contrast angiocardiographic studies in dogs,[18] which confirmed improved hemodynamics during IAC-CPR compared with standard CPR in the same animals. To obtain these radiographs, Babbs and Blevins constructed a special frame for mounting a Thumper® on an X-ray table, allowing spot

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films during CPR in the lateral projection at 4 and 13 seconds after injection of contrast medium into the left ventricle. The films at 4 seconds showed much less regurgitation of contrast into the left atrium and pulmonary veins during IAC-CPR than during standard CPR. The films at 13 seconds showed faster clearance of contrast from both the heart and the aorta during IAC-CPR, documenting greater total blood flow.

Regional perfusion

Several studies have shown that abdominal counterpulsation significantly increases cerebral blood flow during CPR. Walker et a1.[8] measured perfusion of the cerebral cortex using the thermal washout method in a canine model of cardiac arrest. They used both manual chest compression and manual abdominal compression in large supine dogs, following induction of cardiac arrest by intravenous potassium chloride. The chest compression was done in a side-to-side manner, as is accepted practice in veterinary medicine [28] They found that cerebral blood flow averaged 0.06 ml/min/gm in animals receiving standard CPR versus 0.27 ml/min/gm in the animals undergoing IAC-CPR.[8] Voorhees et a1.[3] also demonstrated a significant increase in cerebral blood flow with abdominal counterpulsation in dogs. Animals undergoing IAC-CPR had cerebral blood flow averaging 0.28 ml/min/gm.[3]

Using an electromagnetic flow probe, Einagle et a1.[29] demonstrated a marked increase in carotid blood flow with the use of IAC-CPR. Dogs receiving standard CPR had carotid blood flow only 9 % of control values, whereas dogs receiving IAC-CPR had carotid blood flow averaging 23 % of control values. Interestingly, these investigators found no difference in the results when the timing of abdominal compression was varied within the diastolic phase. Elsewhere, Coletti et a1.[30] demonstrated augmentation of cerebral and coronary blood flow with the use of abdominal counterpulsation during the diastolic phase of normal sinus rhythm. Other work by Coletti et a1.[31] demonstrated improved coronary perfusion pressure with the use of IAC during CPR in a dog model of cardiac arrest.

Survival

In the original study by Ohomoto et a1.,[23] five of five dogs receiving countermassage during 30 minutes of ventricular fibrillation lived more than 5 days, compared with 2 of 10 receiving conventional closed-chest massage. Retrospective analysis by one of us (CFB) showed this difference to be statistically significant by Fisher's exact test (p < 0.01).[32] Subsequently Kern et a1.,[27] who compared IAC-CPR and standard CPR without concurrent epinephrine administration in dogs, found no difference in 24-hour survival with the addition of interposed abdominal compressions to otherwise standard CPR. Additionally, no difference in neurologic deficit scores was seen between dogs receiving IAC-CPR compared with those dogs receiving standard CPR. However, Lindner et a1.[33] demonstrated a significant improvement in the resuscitation of pigs experiencing either asphyxial or fibrillatory cardiac arrest with the addition of IAC. All 14 pigs undergoing IAC-CPR (seven in asphyxial arrest; seven in fibrillatory arrest) survived the initial resuscitation attempt, whereas none of 14 control animals treated with standard CPR survived

despite the continuation of standard CPR for 30 minutes. In this study no epinephrine was administered to pigs during the resuscitation attempts.

HEMODYNAMIC MECHANISMS OF IAC-CPR

Taken together, the various preclinical laboratory investigations of central pressures and flows, regional blood flows, and survival strongly suggest that application of interposed abdominal compression during otherwise standard CPR can, under most circumstances, increase coronary and cerebral perfusion and improve survival, without inducing liver damage or intraperitoneal hemorrhage. Mechanistically, there are several probable ways in which IAC-CPR may facilitate vital organ perfusion during resuscitation from cardiac arrest. In a physical sense manual external compression of the abdomen during IAC-CPR can be considered to have at least two immediate effects: contact compression and hydrostatic compression.[34] Contact compression occurs to the extent that the localized external force applied to the abdominal wall is directly transmitted through intervening tissues to the underlying structures such as the aorta and great veins. Hydrostatic compression occurs to the extent that a generalized rise in intraabdominal pressure is created and transmitted uniformly to all sides of intraabdominal structures.

Contact compression of the abdominal aorta improves aortic diastolic pressure, as in Molokhia's original experiments, and may lead to retrograde flow toward the heart and brain during the release phase of chest compression. This diastolic augmentation provided by IAC-CPR appears to be similar to that of intraaortic balloon counterpulsation. Unlike epinephrine administration, however, IAC improves coronary perfusion pressure without increasing myocardial oxygen consumption (MVO_2)[36]. Alternatively, the use of interposed abdominal compression to supplement exogenous catecholamines during cardiac arrest could improve myocardial oxygen supply sufficiently to negate or cancel epinephrine-induced increases in myocardial oxygen demand.

Hydrostatic compression of intraabdominal veins by IAC may also improve hemodynamics during CPR by priming the thoracic pump mechanism. According to the thoracic pump hypothesis of blood flow during CPR,[37, 38] chest compressions induce global increases in intrathoracic pressures, which are equally transmitted to the heart, lungs, and pulmonary vessels in such a way that the entire intrathoracic blood pool is advanced with each chest compression. During release of chest compression, elastic recoil of the chest wall refills this reservoir from the extrathoracic veins. Interventions that increase extrathoracic venous return, reversing venous and hepatic blood pooling during cardiac arrest, would be expected to increase cardiac output. The hydrostatic compression of intraabdominal veins by IAC, as opposed to the contact compression of the abdominal aorta, may lead to augmentation of venous return to the chest, or "pump priming," analogous to the action of the atria in a normally beating heart.

There may be yet another benefit of raised venous pressures induced by IAC during resuscitation. Based upon previous work by Downey[39] and by Hanley et al.,[40] Howard et al.[25] have suggested that the generation of a given systemic arteriovenous pressure diference produced by abdominal venous compression may better overcome capillary closing pressure, especially in

the myocardium. This secondary effect would be expected to further improve myocardial perfusion.

Many of the forgoing hemodynamic mechanisms of IAC-CPR have been demonstrated clearly in a simple electrical model of the circulation, in which the relevant variables can be isolated and studied either one at a time or in combination.[41, 42] In the electrical model described by Babbs et a1.,[41] the heart and blood vessels were modeled as resistive-capacitive networks, pressures were modeled as voltages, blood flow as electric current, blood inertia as inductance, and the cardiac and venous valves were modeled as diodes. Pressurization of the chest and abdomen, as would occur in IAC-CPR, was simulated by half sinusoidal voltage pulses applied to the vascular capacitances. The model was typically arranged to simulate thoracic pump CPR,[37] in which blood is impelled by compression of all intrathoracic vascular model produced quantitative flow augmentation according to the expression: flow = $\alpha P_{th} + \beta P_a$, where P_{th} = peak intrathoracic pressure, P_a = peak abdominal pressure, and α and β are constants ($\alpha > \beta$). In this expression the left-hand term, αP_{th} , represents the contribution of the thoracic pump and the right hand term, βP_a , represents the independent contribution of the abdominal pump.[42] Expressions of this form (with differing values for α and β) describe simulated blood flow to the heart or brain, as well as total flow in the electrical model. In particular, myocardial perfusion was augmented with IAC at all levels of chest compression by amounts that were linearly related to peak abdominal pressure.

In this same electrical model it was also demonstrated that IAC is expected to be synergistic with peripheral vasoconstriction, such as that produced by catecholamines (Fig. 3). Simulated IAC-CPR with high peripheral resistance values generated flow to the head and neck portion of the model corresponding to roughly 70% of normal, prearrest flow--the highest value obtained in any simulation. Conversely, with very low peripheral resistance, the flow produced by abdominal compression is shunted to nonessential vascular beds.
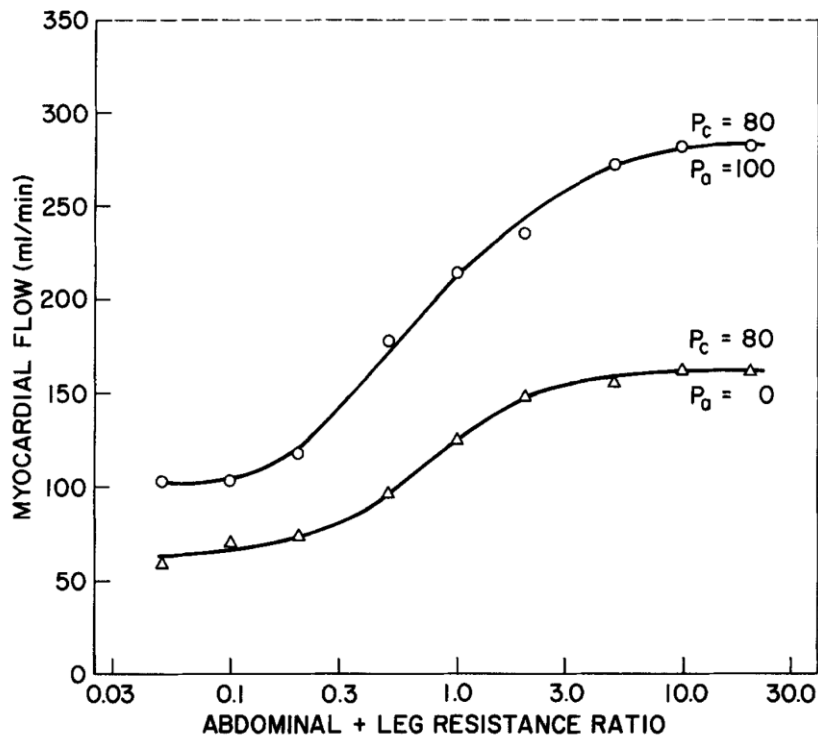
Fig. 3. Simulated effects of an epinephrine-like vasoconstrictor produced by varying vascular resistance of abdominal and lower extremity beds during IAC versus standard CPR in an electrical model of the circulation. Cranial and myocardial resistances were held constant. Data points represent measured values. On the horizontal axis 1.0 (log scale) represents normal peripheral resistance. Smooth curves are hand-drawn trend lines. Coronary flow was nearly doubled by abdominal counterpulsation ($P_a = 100$) in the presence of supranormal peripheral vascular resistance, and flows approaching 50 % of normal resting myocardial perfusion were obtained. Data obtained from reference 41.

Interestingly, both the intraaortic balloon pump-like effect and the thoracic pump priming effect of IAC are mechanically analogous to the chemical effects of epinephrine during CPR, namely the constriction of peripheral arterioles to raise peripheral vascular resistance, and the constriction of peripheral venules to augment right-sided filling pressures. Unlike the effects of high-dose, exogenous epinephrine, however, the effects of IAC can be discontinued immediately after the return of spontaneous circulation, to avoid the deleterious influence of high afterload on an already weakened and diseased heart.

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CLINICAL STUDIES

Two types of clinical studies of IAC-CPR have been reported: smaller scale preliminary studies focusing on hemodynamic mechanisms and safety, and larger scale randomized trials focusing on resuscitation success and survival. This classification of clinical studies is based upon the well-recognized sequence of clinical research in drug development formally described in terms of phase 1, phase 2, and phase 3 clinical trials.[43] Phase 1 studies involve the first drug administration to small numbers of humans to determine biologic activity and potential toxicity, and phase 2 studies determine potential usefulness and dosage ranges in man. It is not until phase 3 that broad, randomized, controlled trials are done in large populations of specified patients to determine efficacy and safety.

Phase 1 and 2 clinical studies

Berryman and Phillips[24] first studied six patients who failed to respond to conventional CPR in the emergency department and found that IAC-CPR raised mean arterial pressure and arteriovenous pressure difference, although the latter was measured in only one patient. In a similar early study in six patients, McDonald[43a] found no effects of IAC. Subsequently, Howard et al.[44] and Howard and Guinness[45] measured coronary perfusion pressure in 14 patients during alternate 2-minute trials of IAC compared with standard CPR. They found that perfusion pressures were approximately doubled by the addition of IAC, as had been shown in previous animal studies.[20, 21] Additionally, three patients had a return of spontaneous circulation during IAC-CPR applied with high compression force after an average of 54 minutes of asystole supported by standard CPR.

Ward and et al.[46] studied end-tidal PCO_2 in 33 adult patients resuscitated with either standard or IAC-CPR in a randomized, cross-over design in which each patient served as his or her own control. Carbon dioxide excretion, an indicator of the rate of venous blood return to the right heart, increased in every patient switched from standard CPR to IAC-CPR and fell in every patient switched from IAC to standard CPR.

Phase 3 prospective randomized studies

The first human survival study in 1984,[47] which showed no significant difference in the outcome of patients treated with IAC-CPR versus standard CPR, temporarily dampened enthusiasm for the technique. In this trial, Mateer et al.[47] conducted a prospective investigation comparing a combination of IAC-CPR and standard CPR, with standard CPR alone being used in patients experiencing out-of-hospital cardiac arrest. The reason for the combined technique in the experimental group was that only two-man ambulance teams were available at the time, one member of which had to drive the ambulance. Thus although IAC-CPR was started in the field, only standard CPR could be applied during transport.

Further, any bystander CPR applied in the field was standard CPR, not IAC-CPR. Of the 145 patients who received field IAC-CPR, 40 patients (28 %) were successfully resuscitated, whereas 45 of 146 patients (31%) treated with standard CPR arrived at the emergency room with a palpable pulse and measurable blood pressure. These resuscitation rates were not statistically different. This initial randomized study, however, was convincing in suggesting the safety of the method (no difference in the incidence of emesis or abdominal trauma). In retrospect, it might today be best interpreted as a phase 2 study focusing on safety because of the practical limitations obviating the use of IAC-CPR continually during cardiac arrest.

Subsequently, however, Sack et a1.[48] demonstrated significant improvement in both the frequency of the initial return of spontaneous circulation and the frequency of long-term survival in patients experiencing in-hospital cardiac arrest treated exclusively either with IAC-CPR or with standard CPR. In this randomized, prospective trial, 103 patients undergoing 135 resuscitation attempts were studied. Immediate resuscitation success was improved from 27 % to 51% with the use of abdominal counterpulsation. At 24 hours more patients were alive in the IAC-CPR group than in the standard CPR group (33 % vs. 13 %) p = 0.02). Additionally, 25 % of patients in the IAC-CPR group survived to hospital discharge versus only 7% in the standard CPR group (p = 0.02, Table II).

Table II. Efficacy of IAC versus standard CPR in man48

End point*	Frequency in IAC-CPR (%)	Frequency in standard CPR (%)
Return of spontaneous circulation	60	25
24-Hour survival	33	13
Survival to hospital discharge	25	7
Neurologically intact survival	17	6

*Forty-eight patients received interposed abdominal compression (IAC) cardiopulmonary resuscitation (CPR) during 71 resuscitation attempts; 55 patients received standard CPR during 64 resuscitation attempts.

In the forgoing clinical study the most striking outcome advantage was seen in those patients whose initial arrest rhythm was asystole or electromechanical dissociation (EMD). Patients experiencing in-hospital cardiac arrest from asystole or EMD have an extremely grave prognosis.[49] Successful restoration of spontaneous circulation in these patients may be more dependent upon the maintenance of an adequate coronary perfusion pressure than restoration of spontaneous circulation in patients with ventricular fibrillation or ventricular tachycardia, which is dependent upon immediate cardioversion. In a follow-up study in a separate and exceedingly difficult to resuscitate population of patients with electrocardiographic findings of asystole or EMD, Sack et a1.[50] demonstrated a significant improvement in immediate resuscitation outcome and 24-hour survival with the addition of interposed abdominal compressions to otherwise standard CPR. In this study, 143 patients experiencing in-hospital cardiac arrest from asystole or

EMD were studied prospectively. Immediate resuscitation rates were improved from 28 % to 48% by the addition of interposed abdominal compressions. Furthermore, a significantly greater number of patients survived 24 hours in the IAC-CPR group compared with the control group (33 % vs 13 % , a 25-fold improvement).

COMPLICATIONS

Early studies of continuous abdominal pressure during CPR led to concerns of abdominal organ damage, specifically hepatic rupture, during simultaneous chest and abdominal compression (Table I). However, recent studies that used interposed abdominal compression techniques have failed to substantiate these concerns. Table III summarizes published reports of complications during IAC-CPR. Of 289 adult human patients receiving interposed abdominal compressions in conjunction with CPR, no clinical evidence of abdominal trauma was seen. Additional necropsy data available on 30 of these patients has failed to reveal any evidence of major abdominal organ damage from the use of IAC-CPR. Currently there is only one reported case[51] of abdominal organ damage involving necropsy-confirmed pancreatic injury in a child receiving interposed abdominal compression during CPR.

Table III. Meta-analysis of complications associated with IAC-CPR

Date	Species	Subjects receiving IAC-CPR	Abdominal trauma (Clinical)	Abdominal trauma (Necropsy)	IAC-CPR vs STD-CPR	Authors
1983	Dog	10	0	0	NR	Voorhees et al.[21]
1984	Human	1	0	1	NR	Waldman et al.[51]
1984	Human	6	0	0 (n = 4)	NR	Berryman and Phillips[24]
1984	Human	7	0	0	p = NS	Howard et al.[44]
1985	Human	145	0	0 (n = 6)	p = NS	Mateer et al.[47]
1986	Dog	10	0	NR	p = NS	Kern et al.[27]
1989	Human	16	0	0 (n = 10)	p = NS	Ward et al.[46]
1992	Human	48	0	0 (n = 5)	p = NS	Sack et al.[48]
1992	Human	67	0	0 (n = 5)	p = NS	Sack et al.[50]
Totals		310	0	1 (3%)		

NR, Not reported; NS, not statistically significant; STD, standard.

Additional concerns regarding the possibility of more frequent emesis during IAC-CPR than during conventional CPR remain unsubstantiated. There are three large-scale human studies[47, 48, 50] that specifically reported the incidence of emesis before and after endotracheal intubation during IAC-CPR compared with standard CPR. All three studies failed to show a difference in the rates of emesis between the IAC and standard CPR groups, and the data of Mateer et a1.[47] suggest a trend toward lower rates of emesis with the addition of IAC. Interestingly, in dogs IAC-CPR produces less gastric insufflation than standard CPR when ventilation pressure is applied to both the trachea and esophagus,[19] probably because the abdominal pressure that is maintained during ventilation tends to prevent gastric insufflation during positive pressure rescue breathing.

IMPROVEMENTS IN TECHNIQUE

Previous investigations of IAC-CPR have varied somewhat in the specific technique of abdominal compression. An analysis of these variations may provide important clues to further optimization of the technique.[34] One easily taught and applied method of IAC-CPR involves left-sided, angled compression of the abdominal aorta against the spine. The rescuer kneels on the victim's left side, with his weight toward his own heels and with arms extended straight to apply IAC. The right hand is positioned so the umbilicus appears in the thumb-index angle, with the thumb pointing toward the feet. The heel of the right hand is thus centered approximately 3 cm to the left of the midline, overlying the abdominal aorta above its bifurcation. This arrangement tends to direct the compression force to maximize contact compression of the abdominal aorta. Although this particular method of abdominal compression can be justified on anatomic grounds,[34] there are as yet no comparative data supporting this particular technique as the optimum means of abdominal compression during CPR.

Along similar lines, Sack et a1.,[50] who utilized a bare-handed abdominal compression technique clinically, speculated that a simple bare-handed approach (Fig. 1, inset) causes better coupling of pressure from the skin surface to the great vessels than the original experimental method, in which the air bladder of a blood pressure cuff was placed between the compressing hands and the abdomen to monitor and limit applied pressure.[20, 21] Quite possibly the previous use of folded manometer cuffs may have contributed adversely to the overall effect of IAC by decreasing the force per unit area directly exerted upon the abdominal aorta, thus reducing the benefit of IAC-CPR. If the risk of causing internal injury is indeed minimal, then the coupling bladder is not needed, and an even simpler and more effective technique of abdominal counterpulsation may be possible.

Another interesting technical difference among published studies of IAC-CPR relates to the overall compression rate. In the original animal studies the compression rate was 60/min.[20, 21] However, in the most successful clinical studies the rate was increased to 80 to 100 compressions per minute.[48, 50] Significant benefits may have accrued by application of IAC-CPR at higher compression rates.[52] In addition, the rhythm provided by 80 to 100 compressions per minute is felt by some rescuers, who have applied the technique in man, to be easier to maintain and to synchronize with chest compression and also less tiring than a slower rhythm requiring more sustained compressions.

Although IAC-CPR seems to be an extremely simple technique, the subtleties of the hemodynamic mechanisms invoked continue to provide an intellectually stimulating area for further research.[53] True optimization of a novel device or method, even a simple one, requires clear and detailed knowledge of the pathophysiology of patients to whom the new method will be applied. In the case of IAC-CPR, evolving ideas about hemodynamic mechanisms during abdominal counterpulsation[25, 41, 42, 52] have led to validation and refinement of the method. This process is still ongoing, and further insights and refinements in the technique for human application are likely as clinical research and application continue.

CONCLUSIONS

IAC-CPR is an easily applied manual technique that represents an evolution, not a revolution, in the treatment of cardiac arrest. Many investigations have demonstrated improved hemodynamics with the use of IAC-CPR during cardiac arrest, specifically increases in coronary and cerebral perfusion pressures, which are theoretically critical in restoring spontaneous circulation and in maintaining long-term neurologically intact survival.

There are two probable mechanisms for the beneficial effects of abdominal counterpulsation. The first is an effect similar to that of the intraaortic balloon pump: contact compression of the abdominal aorta during chest recoil squeezes blood retrograde toward the heart and brain. The resultant augmentation of aortic diastolic pressure encourages greater coronary and peripheral perfusion, in addition to increasing cardiac output. The second is an effect similar to the action of the cardiac atria when the heart is beating normally: hydrostatic compression of intraabdominal veins primes the intrathoracic pump mechanism.

These two effects combine to augment the artificial circulation created by chest compression during CPR. There are now two randomized, prospective human studies in which an improvement in resuscitation outcome in patients suffering from in-hospital cardiac arrest has been observed. The addition of interposed abdominal compression to otherwise standard CPR may be considered an optional adjunct to current standard CPR that can improve hemodynamics and resuscitation success without complications. However, further laboratory studies of the mechanics of abdominal compression as well as larger scale clinical trials are needed before IAC-CPR can be fully optimized and rendered sufficiently practical to replace current basic life support.

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