2013

The Case for Inteposed Abdominal Compression CPR in Hospital Settings

Charles F. Babbs
Purdue University, babbs@purdue.edu

Review

The Case for Interposed Abdominal Compression CPR in Hospital Settings

Charles F. Babbs, MD, PhD

Weldon School of Biomedical Engineering, Purdue University,
West Lafayette, Indiana, USA

(Analg Resusc: Curr Res 3:1, 2013. doi:10.4172/2324-903X.1000109)

Abstract

Interposed abdominal compression (IAC)-CPR includes all steps of standard external CPR with the addition of manual mid-abdominal compressions in counterpoint to the rhythm of chest compressions. IAC-CPR can increase blood flow during CPR about two fold compared to standard CPR without IAC, as shown by multiple studies in computer and animal models. The technique increases the rate return of spontaneous circulation (ROSC) for in-hospital resuscitations from roughly 25% to 50%. Improved survival to discharge is demonstrated in two in-hospital trials. IAC as an adjunct technique is quickly taught and is less complex than most other basic life support maneuvers. A thorough review of published evidence suggests that IAC-CPR, initiated early in the resuscitation protocol, is a safe and effective means to increase organ perfusion and survival, when performed by professionally trained responders in a hospital.

Keywords: Abdomen; Cardiac Arrest; Cardiopulmonary Resuscitation; Meta-analysis; Review

Introduction

Interposed abdominal compression (IAC) – CPR includes all the steps of ordinary standard CPR with the addition of manual, external mid-abdominal compressions by a second or third rescuer.[1] The abdominal compressions are delivered in counterpoint to the rhythm of chest compressions, so that abdominal pressure is maintained whenever the chest is not being compressed. Pulses of central abdominal pressure are applied with overlapping hands between the xyphoid process and the umbilicus. Hand position, depth, rhythm, and rate of abdominal compression are similar to those for chest compression, except for being 180 degrees out of phase and hence "interposed" between chest compressions in time. The force of abdominal compression is similar to that needed to palpate the abdominal aortic pulse. The technique of IAC-CPR achieved some notoriety in the 1990's after the clinical studies of Sack and coworkers, involving several hundred patients, suggested a rough doubling of immediate resuscitation success and neurologically intact, long term survival[1]. One unfortunate typographical error in an illustration in Sack's final paper has led some to conclude that patients were improperly excluded from the analysis; however this is not the case. Sack's statistics and conclusions are exceedingly valid and illustrate the promise of this simple, manual technique to boost the effectiveness of in-hospital resuscitation with no additional equipment, simple and rapid training, and very little added risk to patients.

The following review gives a comprehensive survey of the published studies of IAC-CPR. Relevant full length, peer reviewed publications were identified using evidence evaluation methods described previously[2, 3] and developed specifically for topics in emergency cardiovascular care. Full-length, peer reviewed publications were obtained from MEDLINE searches, the author's files, and reference lists of review articles.

As summarized in Table 1, the search identified a body of work including clinical and preclinical studies documenting hemodynamic and clinical outcomes of IAC-CPR compared to standard CPR. Of the 38 full-length, peer reviewed studies relevant to IAC-CPR, 34 obtained results indicating benefit of IAC, and were termed "positive". Two found no difference between IAC and standard CPR, and two found results worse for IAC than for standard CPR. Only a single case report describes an adverse complication attributable to external abdominal compression—possible traumatic pancreatitis in a child. Even the authors of this report stated that their finding was "not meant to condemn a technique which may ultimately prove superior to conventional CPR[4]".

[1] Standard one or two rescuer CPR is augmented by an additional person applying abdominal pressure whenever chest pressure is released, including any pauses in chest compression for ventilation. In this way gastric insufflation is minimized, as shown in one animal study (reference 40).

Table 1. Broad summary of research on IAC-CPR in 3 classes of experimental models.

Model Class	Subtypes	Results
Mathematical, computer, and mechanical models	Paper and pencil[19]	6 of 6 studies positive (100 percent)
	Analog[14, 16]	
	Digital[15, 17]	
	Digital spreadsheet[18]	
Animal models (dogs and pigs)	Blood flow[11-13, 24-26, 48-50]	19 of 20 studies positive (95 percent)
	Blood pressure[7, 8, 20, 51]	
	Oxygen uptake[23]	
	CO_2 excretion/blood gasses[52]	
	Survival and complications[22, 27, 28, 40, 53]	
Clinical models	Pediatric anesthesia overdose[6]	10 of 12 studies positive (83 percent)
	Acute hemodynamics (BP, $ETCO_2$ ± crossover)[29-31, 43, 54-56]	
	All arrests (mostly VF)[1, 42]	
	Non-VF arrests (EMD, asystole)[32, 33]	

For most end points results for IAC-CPR were between 1.5 and 2.0 times those for comparable standard CPR without abdominal compression.

EMD=electromechanical dissociation, $ETCO_2$=end tidal carbon dioxide concentration, SPICE=simulation program for integrated circuit evaluation, VF=ventricular fibrillation.

Early work on abdominal counterpulsation

Three years before external CPR was described by Kouwenhoven, Jude, and Knickerbocker in 1960[5], Rainer and Bullough in the United Kingdom developed a jackknife maneuver for resuscitation of children from anesthetic overdose, which involved rhythmically compressing first the abdomen and then the chest[6]. The technique required the surgeon to fold the knees of a child as far as possible toward the chin, forcing the thighs against the abdomen. Then "when full flexion is reached, the surgeon compresses the patient's chest with the bent knees, using some of his own weight judiciously to add to the pressure".[6] This method can be regarded as a version of phased abdominal and chest compression and was successful in 8 of 8 reported cases.

In 1972 Molokhia, Norman and coworkers at Boston City Hospital[7] described a similar means of generating an artificial circulation during open chest surgery by alternately compressing the aortic arch and the left ventricle. The addition of interposed aortic compressions augmented coronary perfusion by about 50 percent. In 1976 Ohomoto[8] at Tokyo Women's medical college resuscitated anesthetized dogs with two motor driven pistons, one compressing the abdomen, the other the chest. The addition of phased abdominal compression for 80 percent of cycle time to chest compression for 25 percent of cycle time increased carotid artery blood pressure and improved short-term survival in anesthetized dogs with ventricular fibrillation.

Subsequently Rosborough and coworkers in Houston Texas[9] while attempting to develop an animal model of cough CPR[10] combined simultaneous high-pressure lung inflation with abdominal compression. They found to their surprise that abdominal compression and ventilation alone could maintain carotid flow and aortic pressure during ventricular fibrillation in dogs. They suggested the technique as a new CPR modality. Contemporaneously, Coletti, Bregman, and coworkers in New York were studying a canine model of cardiogenic shock in the laboratory one day, when the mechanical intra-aortic balloon pump failed to work properly[11]. As a substitute they tried manual external abdominal compressions interposed between heartbeats, as judged from the electrocardiogram. Both cerebral and coronary perfusion, measured with electromagnetic flowmeters, increased.

Ralston et al.[12]described the modern idea of alternating compression of the abdomen and the chest for the purpose of external cardiopulmonary resuscitation in the year 1982. The first graphic record demonstrating the phenomenon is reproduced in reference[13]. Subsequently a burst of preclinical studies, followed by clinical studies, appeared. These included a wide variety of model systems and experimental end points (Table 1).

Mechanical and computational studies

A number of investigators with a biomedical engineering background have investigated IAC-CPR by building mechanical, electrical, or computer models[14-19]. A good example of this genre is provided by the electrical model of Ralston and Babbs[14], who appreciated the analogy between the flow of current around an electrical circuit and the circulation of blood through systemic and pulmonary vessels. In their simulations electrical resistance mimicked physiological resistance of vascular beds. Resistive-capacitive networks modeled the heart and blood vessels. Voltages represented intravascular pressures; electric current represented blood flow, inductance modeled blood inertia, and diodes served as the cardiac and venous valves. The application of half-sinusoidal voltage pulses to the model represented the effects of compression of the chest and abdomen during CPR. In this model the addition of IAC (pressurization of the abdominal aorta and inferior vena cava of the model) to standard CPR (pressurization of thoracic structures) produced flow augmentation according to the expression

$$\text{flow} = \alpha P_{th} + \beta P_{abd} ,$$

where P_{th} is peak intrathoracic pressure, P_{abd} is peak intraabdominal pressure, and α and β are constants ($\alpha > \beta$). For typical adult CPR intra-abdominal pressure can be made somewhat greater than intrathoracic pressure ($P_{abd} > P_{th}$), so that chest and abdominal flow components become roughly equal. Thus the addition of IAC doubled flow in this electrical model of the adult human.

Such theoretical models also provided a detailed glimpse into hemodynamic mechanisms of abdominal counterpulsation. Simulated pressure waveforms in a model of a typical adult human circulation[18] during standard and IAC-CPR indicate two mechanisms of hemodynamic benefit. The first is a large and obvious increase in aortic blood pressure during the abdominal compression phase, which is greater than the corresponding rise in central venous pressure. The second is the augmentation of the filling phase of the chest pump during the last part of the compression cycle. Thus external abdominal compression acts like an intra-aortic balloon pump, to squeeze more blood from the aorta into systemic resistance vessels. It also acts to squeeze more venous blood into the chest pump, priming it before the next chest compression. In this way phased abdominal pressure has beneficial effects upon both the arterial and the venous sides of the circulation.

Animal studies

The prediction of such theoretical models that IAC could nearly double blood flow during CPR was confirmed in animal studies—typically involving anesthetized pigs and anesthetized dogs with electrically induced ventricular fibrillation. Generally such models, summarized in Table 1 have found indices of blood flow during CPR to increase by 50 to 100 percent with the addition of IAC to the resuscitation protocol. Examples include increased carotid artery flow[8, 20-22], regional blood flow[23-26], systemic and coronary perfusion pressures[12, 23, 26, 27], cardiac output[23], and oxygen delivery[23].

Animal studies have also tracked resuscitation success and survival. Kern's initial study of groups of 10 animals showed no significant difference in immediate resuscitation success or 24 hour survival[28]. However Lindner et al.[27], using groups of 14 ketamine anesthetized pigs, found return of spontaneous circulation in 0/14 animals with standard CPR vs. 14/14 with IAC-CPR. Tang[22], using the Lifestick device that permitted one rescuer to do alternating chest and abdominal compression, obtained similar positive results in pigs—10/10 vs. 0/10 survival at 48-hours for chest compression plus IAC vs. chest compression alone.

Preliminary clinical studies

Preliminary human studies, short of randomized clinical trials, provided further evidence for hemodynamic effects of external abdominal compressions. Howard et al.[29] instrumented 14 patients in the emergency department for blood pressure measurements during alternate trials of standard versus IAC-CPR. They found that IAC and high force IAC increased diastolic and mean arterial pressures, but not necessarily arteriovenous pressure differences, and that the effects of IAC were greater in non-obese patients.

Adams[30] studied hemodynamics of IAC during human CPR in patients that were unsalvageable by conventional ACLS. There were 13 responders with a mean change in coronary perfusion pressure of 9.5 mmHg and 7 non-responders with a mean change in coronary perfusion pressure of –2.6 mmHg. Berryman and Phillips[31] also studied IAC-CPR in six cardiac arrest patients after unsuccessful ACLS. Mean arterial pressure increased from 26 to 39 mmHg. Villa, Colombo, and coworkers[32] reported one case of a successful 1 hour and 20 min long resuscitation of a patient with pulseless electrical activity using a combination of mechanical chest compression and manual IAC. The patient lived subsequently for 8 hours following cardiac arrest and resuscitation.

Randomized clinical trials

Return of spontaneous circulation and short term survival

Three randomized clinical trials of IAC-CPR for in-hospital cardiac arrest have shown statistically significant improvement of outcome measures[1, 33]. One randomized trial of pre-hospital IAC-CPR, combined, when possible, with standard CPR in the field, showed no difference in outcome or in complications[34]. Pooled analysis of all available data for both pre-hospital and in-hospital resuscitations show improvement in the return of spontaneous circulation with IAC-CPR, compared to standard CPR (Table 2). When only the in-hospital studies are examined, the effect of IAC becomes much greater. Pooled data from the two studies that examined long term, neurologically intact survival following in-hospital resuscitations show a similar relative benefit of IAC-CPR compared with standard CPR.

Table 2. Aggregate human ROSC and survival data for IAC vs. standard CPR

Outcome Measure	Studies	IAC-CPR	Standard CPR
Return of spontaneous circulation (ROSC) in or out-of-hospital	Mateer[34]	40/145 (28%)	45/146 (31%)
	Ward[43]	6/16 (38%)	3/17 (18%)
	Sack #1[1]	29/48 (60%)	14/55 (25%)
	Sack#2[33]	33/67 (49%)	21/76 (28%)
	All 4 studies	108/276 (39%)	83/294 (28%)
Return of spontaneous circulation (ROSC) after in-hospital resuscitation	Ward[43]	6/16 (38%)	3/17 (18%)
	Sack #1[1]	29/48 (60%)	14/55 (25%)
	Sack#2[33]	33/67 (49%)	21/76 (28%)
	All 3 studies	68/131 (52%)	38/148 (26%)
Survival to discharge, neurologically intact after in-hospital resuscitation	Ward[43]	1/16 (6%)	0/17 (0%)
	Sack #1[1]	8/48 (17%)	3/55 (5%)
	Both studies	9/64 (14%)	3/72 (4%)

Complications

The safety of interposed abdominal compressions, reviewed previously[35], has been well documented in 426 humans, 151, dogs and 14 pigs. Only one isolated case report of traumatic pancreatitis in a child[36] describes local trauma from abdominal compression during CPR. These data compare favorably with the well-known and frequent incidence of rib fracture and pulmonary contusion from chest compression during CPR[37-39]. Increased emesis and aspiration from IAC have not been reported, and there is 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[40].

Meta-analysis

A more rigorous meta-analysis of IAC-CPR has recently been published[41] that is limited to human clinical trials in which the end point was either short term or long term survival. These include all available survival data from the three in-hospital trials and one and out-of-hospital trial[1, 33, 42, 43]. That is, hemodynamic data were not considered. Meta-analysis refers to the quantitative synthesis of data from multiple clinical studies in order to minimize both Type I and Type II statistical errors. Inverse variance weighting of outcome data is typically used to account for the relative numbers of patients in the various studies. Results for such an analysis of IAC-CPR trials reported in references are shown in Figure 1. This figure shows cumulative meta-analysis plots that demonstrate historical trends with the publication of each successive clinical study. In each plot the top data point and its 95 percent confidence interval represent the historically first trial, the next a combination of the first two trials, the third a combination of the first three trials, etc. This format is similar to that introduced by Lau and coworkers[44] for cumulative meta-analysis in cardiovascular medicine.

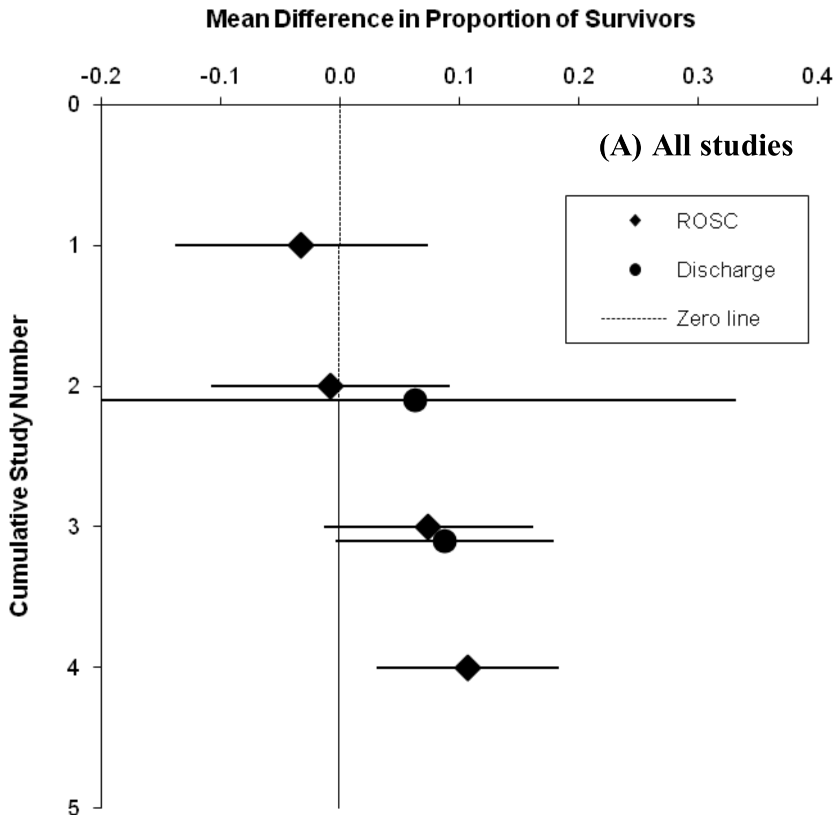
Mean Difference in Proportion of Survivors
-0.2
-0.1
0.0
0.1
0.2
0.3
0.4
0
1
2
3
4
5
Cumulative Study Number
(A) All studies
ROSC
Discharge
Zero line

Figure 1(A)

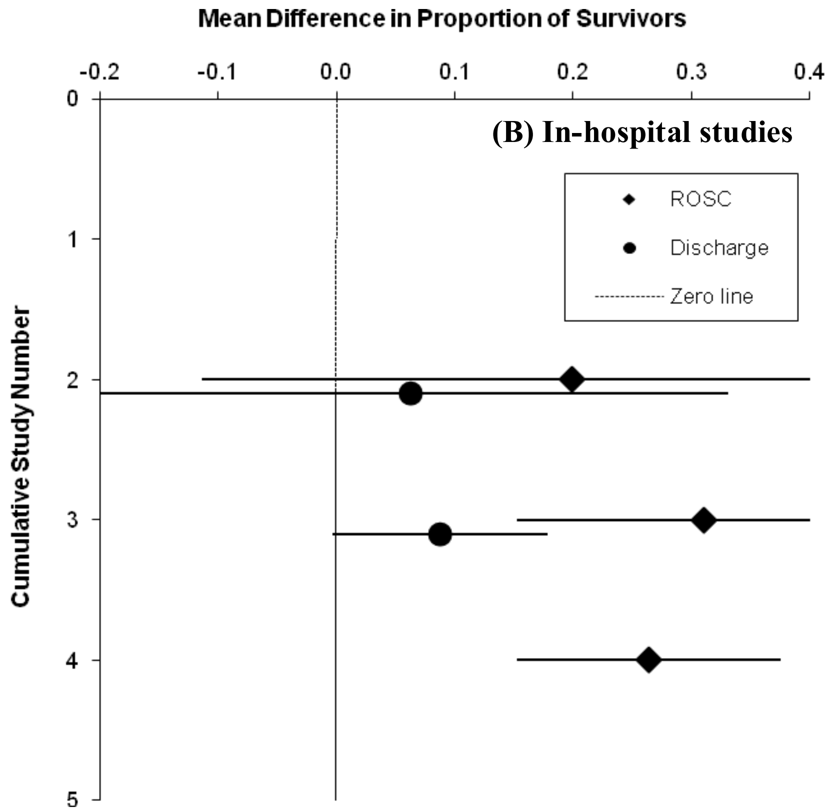

Figure 1(B)

Figure 1. A cumulative meta-analysis for IAC-CPR in terms of the mean difference in proportion of survivors[41]. Successive points from top to bottom represent a separate meta-analysis after the appearance of each study[1, 33, 34, 43] in the series. Solid symbols refer to data for return of spontaneous circulation (ROSC), open symbols to data for neurologically intact discharge survival. Horizontal lines indicate 95 percent confidence intervals. (A) All studies in which survival was measured, including one out-of-hospital and three in-hospital studies. (B) In-hospital studies only[1, 33, 43].

The combined studies of IAC-CPR, including all available in-hospital and out-of-hospital data (Figure 1(A)), showed a significant treatment effect for short-term and probably for long-term survival. The difference in the proportion of survivors, $\overline{\Delta p}$, is 10.7 percent for return of spontaneous circulation and 8.7 percent for hospital discharge. The effect of IAC in the overall meta-analysis is highly significant for return of spontaneous circulation (P = 0.006) and of borderline statistical significance (P = 0.06) for discharge survival. These summary values can be immediately converted into the number-needed-to-treat, $NNT = 1/\overline{\Delta p}$, the number of patients that must be treated to obtain one additional survivor[45]. For return of spontaneous circulation the NNT is 9. For discharge survival the corresponding NNT is 12. When only in-hospital studies are considered (Figure 1(B)) the statistical significance of the meta-analysis for return of spontaneous circulation is even greater (P < 0.0001, NNT = 4).

Practical issues

Practical implementation of a new method requires consideration of issues other than simply efficacy and safety. If a technique is extremely costly, difficult to teach or to learn, or likely only to benefit a small number of patients (with a large number-needed-to-treat) then it may not be suitable for widespread clinical implementation. Since IAC requires no new equipment or drug, its added cost is minimal, as long as adequate personnel are available in the hospital setting. However, as initially discovered by Mateer and coworkers[42], lack of trained personnel in out-of-hospital settings can obviate the use of IAC-CPR.

Benefit vs. cost

Suppose that IAC-CPR were implemented for in-hospital resuscitations only. How many more long term survivors ($\Delta N_{survivors}$) of cardiac arrest might be expected? One simple calculation can be done as follows:

$$\Delta N_{survivors} = N_{arrests} \bullet P_{in\text{-}hosp} \bullet \Delta P_{rosc} \bullet P_{long\ term}\ ,$$

where,

$N_{arrests}$ is the umber of cardiac arrests in nation per year
$P_{in\text{-}hosp}$ is the proportion of in-hospital resuscitations
ΔP_{rosc} is the change in proportion of ROSC attributable to IAC
$P_{long\ term}$ is the proportion of initial saves who walk out of the hospital.

The research reviewed herein indicates that ΔP_{rosc} is 22% for in-hospital studies. Suppose, for example, that $N_{arrests} = 250{,}000$, $P_{in\text{-}hosp} = 0.2$, $\Delta P_{rosc} = 0.2$, and $P_{long\ term} = 0.3$, then
$\Delta N_{survivors} = 3000$ extra survivors

Although IAC-CPR itself is a simple manual technique, the costs of widespread implementation by professional rescuers in any particular hospitals would be modest, since the training procedures (described below) are simple. If upgrades in manikins for training to include abdominal pressure monitors were needed, then these costs would need to be included. However, in any given hospital the needed spare blood pressure cuffs and scrap foam rubber are rather easy to obtain at close to zero cost.

Training

The recommended technique of abdominal compression is adapted from that described by Sack and coworkers[1], who had the most favorable clinical results in their in-hospital study. Condensed instructions for teaching and practicing IAC-CPR are as follows.

Use a two-handed, straight-armed technique similar to that for chest compressions. The abdominal compression point is in the midline, one-half hand width (5 cm) headward of the umbilicus. To ensure proper hand placement on the abdomen, the umbilicus should be visible just below the rescuer's hands as a midline landmark. Apply pressure with heel of the hand straight down at the abdominal compression point, keeping fingers off the abdomen.

The amount of force applied should be sufficient to generate approximately normal mean arterial pressure (95 mmHg) within the abdomen near the abdominal aorta. This value is similar to that reported to give good results in animal studies and in theoretical models. It is also less than that required to produce objectionable discomfort in an awake, conscious volunteer[46]. As has been previously described, this is the same amount of force required for palpation of the abdominal aortic pulse during an ordinary physical examination of the abdomen and is therefore not excessive[46].

It is quite easy to modify a standard whole body mannequin for the teaching and practice of three-rescuer IAC-CPR. One can add extra foam rubber to the lower thoracic compartment of the mannequin and also to the abdominal compartment of the mannequin to simulate soft tissues. The next step is to wrap a standard arm blood pressure cuff around a rolled towel and place it in the abdominal compartment beneath the abdominal compression point (5-cm headward of the umbilicus in the midline). Then bring the tubing, aneroid manometer gauge, and squeeze bulb out at belt-line so they are visible to the trainees. Tape target pressure markers on the manometer dial at 20 and 120 mmHg. Inflate the cuff to a resting pressure of 20 mmHg. During practice coach trainees to hit the target pressure of 120 during IAC and release to a target pressure of 20 during chest compression. Failure to release abdominal pressure can lead to liver injury during the subsequent chest compression.

One can initiate trainees to the rhythm and mechanics of manual abdominal compressions in the following sequence: Let two experienced rescuers perform two-rescuer CPR on the mannequin as usual. The third (abdominal) rescuer, who is the trainee, takes position on opposite side of victim from chest compressor at the level of the abdomen. The chest compressor says, “You press here whenever I release,” pointing to the abdominal compression point. The chest compressor counts “one – AND – two – AND – three – AND …”. The abdominal rescuer applies pressure during “AND”. Abdominal compression is also maintained during mouth-to-mouth or bag-valve-mask rescue breaths to prevent gastric inflation with air[40]. It is helpful to start with slow-motion practice and then gradually to increase toward a normal compression rate. After the rhythm is mastered the trainee can focus on hitting the target pressure on the manometer. If only two rescuers are present, one rescuer does chest compressions and artificial ventilations and the other rescuer does abdominal compressions, keeping pressure on the abdomen whenever chest pressure is released to minimize the risk of gastric inflation with air during mouth to mouth breathing.

Protocols

Outcome can be improved if IAC-CPR is begun as first line therapy in a hospital[1]. It is best not to use IAC-CPR as a last ditch effort on individuals who have already failed conventional resuscitation efforts. IAC is known to be relatively ineffective in the failed ACLS model[47]. Thus the published evidence does not support the use of IAC-CPR as second line therapy. Three rescuer IAC-CPR is not magic and cannot save patients who are already dead. It is a simple extension of conventional two-rescuer CPR, which can improve hemodynamics and outcome when performed by trained professionals in a hospital setting.

Conclusions

Having been independently discovered in Japan, Israel, the United Kingdom, and the United States by diverse researchers primarily interested in other topics, it seems to have been poorly championed and poorly advertised in the scientific community. Mentioned as an alternative adjunct in American Heart Association guidelines since 1992, the method is still rarely utilized. A review of published evidence, however, indicates that IAC-CPR has a sound physiologic basis, does produce improved hemodynamics, and at the very least improves short-term resuscitation success (return of spontaneous circulation) in human beings, when initiated early in the resuscitation protocol in a hospital setting. Going forward training programs need to be developed along with studies to explore the community attitudes and hurdles resulting in underutilization of IAC-CPR.

References

1. Sack JB, Kesselbrenner MB, Bregman D. Survival from in-hospital cardiac arrest with interposed abdominal counterpulsation during cardiopulmonary resuscitation. *JAMA*. 1992;267:379-385
2. Cummins RO, Chamberlain D, Hazinski MF, Nadkarni V, Kloeck W, Kramer E, Becker L, Robertson C, Koster R, Zaritsky A, Ornato JP, Callanan V, Allen M, Steen P, Connolly B, Sanders A, Idris A, Cobbe S. Recommended guidelines for reviewing, reporting, and conducting research on in-hospital resuscitation: The in-hospital "utstein style". *Circulation*. 1997;95:2213-2239
3. Cummins RO. Introduction to the international guidelines 2000 for cpr and ecc. *Circulation*. 2000;102 (suppl I):I-1--I-11
4. Waldman PJ, Walters BL, Grunau CF. Pancreatic injury associated with interposed abdominal compressions in pediatric cardiopulmonary resuscitation. *Am J Emerg Med*. 1984;2:510-512
5. Kouwenhoven WB, Jude JR, Knickerbocker GG. Closed-chest cardiac massage. *JAMA*. 1960;173:1064-1067
6. Rainer EH, Bullough J. Respiratory and cardiac arrest during anaesthesia in children. *British Medical Journal*. 1957:1024-1028
7. Molokhia FA, Ponn RB, Robinson WJ, Asimacopoulos PJ, Norman JC. A method of augmenting coronary perfusion during internal cardiac massage. *Chest*. 1972;62:610-613
8. Ohomoto T, Miura I, Konno S. A new method of external cardiac massage to improve diastolic augmentation and prolong survival time. *Ann Thorac Surg*. 1976;21:284-290
9. Rosborough JP, Niemann JT, Criley JM, O'Bannon W, Rouse D. Lower abdominal compression with synchronized ventilation--a cpr modality. *Circulation*. 1981;64(IV):303
10. Criley JM, Blaufuss AH, Kissel GL. Cough-induced cardiac compression--a self-administered form of cardiopulmonary resuscitation. *JAMA*. 1976;236(11):1246-1250
11. Coletti RH, Kaskel PS, Cohen SR, Bregman D. Abdominal counterpulsation (ac) --a new concept in circulatory assistance. *Trans Am Soc Artif Intern Organs*. 1982;28:563-566
12. Ralston SH, Babbs CF, Niebauer MJ. Cardiopulmonary resuscitation with interposed abdominal compression in dogs. *Anesthesia and Analgesia*. 1982;61:645-651
13. Babbs CF, Blevins WE. Abdominal binding and counterpulsation in cardiopulmonary resuscitation. *Critical Care Clinics*. 1986;2:319-332
14. Babbs CF, Ralston SH, Geddes LA. Theoretical advantages of abdominal counterpulsation in cpr as demonstrated in a simple electrical model of the circulation. *Annals of Emergency Medicine*. 1984;13:660-671
15. Babbs CF, Thelander K. Theoretically optimal duty cycles for chest and abdominal compression during external cardiopulmonary resuscitation. *Acad Emerg Med*. 1995;2:698-707

16. Babbs CF, Weaver JC, Ralston SH, Geddes LA. Cardiac, thoracic, and abdominal pump mechanisms in cpr: Studies in an electrical model of the circulation. *American Journal of Emergency Medicine*. 1984;2:299-308
17. Beyar R, Kishon Y, Sideman S, Dinnar U. Computer studies of systemic and regional blood flow during cardiopulmonary resuscitation. *Medical & Biological Engineering and Computing*. 1984;22:499-506
18. Babbs CF. Cpr techniques that combine chest and abdominal compression and decompression: Hemodynamic insights from a spreadsheet model. *Circulation*. 1999;100:2146-2152
19. Babbs CF. Abdominal counterpulsation in cardiopulmonary resuscitation: Animal models and theoretical considerations. *American Journal of Emergency Medicine*. 1985;3:165-170
20. Beyar R, Kishon Y, Kimmel E, Neufeld H, Dinnar U. Intrathoracic and abdominal pressure variations as an efficient method for cardiopulmonary resuscitation: Studies in dogs compared with computer model results. *Cardiovasc Res*. 1985;19:335-342
21. Einagle V, Bertrand F, Wise RA, Roussos C, Magder S. Interposed abdominal compressions and carotid blood flow during cardiopulmonary resuscitation. Support for a thoracoabdominal unit. *Chest*. 1988;93:1206-1212
22. Tang W, Weil MH, Schock RB, Sato Y, Lucas J, Sun S, Bisera J. Phased chest and abdominal compression-decompression. A new option for cardiopulmonary resuscitation. *Circulation*. 1997;95:1335-1340
23. Voorhees WD, Niebauer MJ, Babbs CF. Improved oxygen delivery during cardiopulmonary resuscitation with interposed abdominal compressions. *Annals of Emergency Medicine*. 1983;12:128-135
24. Walker JW, Bruestle JC, White BC, Evans AT, Indreri R, Bialek H. Perfusion of the cerebral cortex using abdominal counterpulsation during cpr. *Am J Emerg Med*. 1984;2:391-393
25. Hoekstra OS, van Lambalgen AA, Groeneveld AB, van den Bos GC, Thijs LG. Abdominal compressions increase vital organ perfusion during cpr in dogs: Relation with efficacy of thoracic compressions. *Ann Emerg Med*. 1995;25:375-385
26. Wenzel V, Lindner KH, Prengel AW, Strohmenger HU. Effect of phased chest and abdominal compression-decompression cardiopulmonary resuscitation on myocardial and cerebral blood flow in pigs. *Crit Care Med*. 2000;28:1107-1112
27. Lindner KH, Ahnefeld FW, Bowdler IM. Cardiopulmonary resuscitation with interposed abdominal compression after asphyxial or fibrillatory cardiac arrest in pigs. *Anesthesiology*. 1990;72:675-681
28. Kern KB, Carter AB, Showen RL, Voorhees WD, Babbs CF, Tacker WA, Ewy GA. Twenty-four hour survival in a canine model of cardiac arrest comparing three methods of manual cardiopulmonary resuscitation. *J Am Coll Cardiol*. 1986;7:859-867
29. Howard M, Carrubba C, Foss F, Janiak B, Hogan B, Guinness M. Interposed abdominal compression-cpr: Its effects on parameters of coronary perfusion in human subjects. *Ann Emerg Med*. 1987;16:253-259

30. Adams CP, Martin GB, Rivers EP, Ward KR, Smithline HA, Rady MY. Hemodynamics of interposed abdominal compression during human cardiopulmonary resuscitation. *Acad Emerg Med*. 1994;1:498-502
31. Berryman CR, Phillips GM. Interposed abdominal compression-cpr in human subjects. *Annals of Emergency Medicine*. 1984;13:226-229
32. Villa GF, Colombo S, Cabrini L, Scandroglio AM, Torri G. [interposed abdominal compression in mechanical cardiopulmonary resuscitation. Description of a clinical case]. *Minerva Anestesiol*. 1998;64:415-418
33. Sack JB, Kesselbrenner MB, Jarrad A. Interposed abdominal compression-cardiopulmonary resuscitation and resuscitation outcome during asystole and electromechanical dissociation. *Circulation*. 1992;86:1692-1700
34. Mateer JR, Steuven HA, Thompson BM, Aprahamian C, Darin J. Pre-hospital iac-cpr versus standard cpr: Paramedic resuscitation of cardiac arrests. *American Journal of Emergency Medicine*. 1985;3:143-146
35. Sack JB, Kesselbrenner MB. Hemodynamics, survival benefits, and complications of interposed abdominal compression during cardiopulmonary resuscitation. *Acad Emerg Med*. 1994;1:490-497
36. Waldman PJ, Walters BL, Grunau C. Pancreatic injury associated with interposed abdominal compressions in pediatric cardiopulmonary resuscitation. *Am J Emerg Med*. 1984;2:510-512
37. Clark DT. Complications following closed-chest cardiac massage. *JAMA 181(4)*. 1962:337-338
38. Nagel EL, Fine EG, Krischer JP, Davis JH. Complications of cpr. *Critical Care Medicine 9*. 1981:424
39. Silberberg B, Rachmaninoff N. Complications following external cardiac massage. *Surg Gynecol Obstet 119*. 1964:6-10
40. Babbs CF, Schoenlein WE, Lowe MW. Gastric insufflation during iac-cpr and standard cpr in a canine model. *American Journal of Emergency Medicine*. 1985;3:99-103
41. Babbs CF. Simplified meta-analysis of clinical trials in resuscitation. *Resuscitation*. 2003;In Press
42. Mateer JR, Stueven HA, Thompson BM, Aprahamian C, Darin JC. Pre-hospital iac-cpr versus standard cpr: Paramedic resuscitation of cardiac arrests. *Am J Emerg Med*. 1985;3:143-146
43. Ward KR, Sullivan RJ, Zelenak RR, Summer WR. A comparison of interposed abdominal compression cpr and standard cpr by monitoring end-tidal pco2. *Ann Emerg Med*. 1989;18:831-837
44. Lau J, Antman EM, Jimenez-Silva J, Kupelnick B, Mosteller F, Chalmers TC. Cumulative meta-analysis of therapeutic trials for myocardial infarction. *N Engl J Med*. 1992;327:248-254
45. McQuay HJ, Moore RA. Using numerical results from systematic reviews in clinical practice. *Ann Intern Med*. 1997;126:712-720
46. Babbs CF. Iac-cpr: Are we missing the mark in clinical trials? *American heart journal*. 1993;126:1035-1041
47. Ward KR. Possible reasons for the variability of human responses to iac-cpr. *Acad Emerg Med*. 1994;1:482-489

48. Watson JT, Platt MR, Rogers DE, Sugg WL, Willerson JT. Similarities in coronary flow between external counterpulsation and intra-aortic balloon pumping. *American Journal of Physiology*. 1976;230:1616-1621
49. Voorhees WD, Ralston SH, Babbs CF. Regional blood flow during cardiopulmonary resuscitation with abdominal counterpulsation in dogs. *Am J Emerg Med*. 1984;2:123-128
50. Einagle V, Bertrand F, Wise RA, Roussos C, Madger S. Interposed abdominal compressions and carotid blood flow during cardiopulmonary resuscitation: Support for a thoracoabdominal unit. *Chest*. 1988;93:1206-1212
51. Christenson JM, Hamilton DR, Scott-Douglas NW, Tyberg JV, Powell DG. Abdominal compressions during cpr: Hemodynamic effects of altering timing and force. *J Emerg Med*. 1992;10:257-266
52. Lippert AC, Evans AT, White BC, Eyster GE. The effect of resuscitation technique and pre-arrest state of oxygenation on blood-gas values during cardiopulmonary resuscitation in dogs. *Vet Surg*. 1988;17:283-290
53. Kern KB, Carter AB, Showen RL, Voorhees WDd, Babbs CF, Tacker WA, Ewy GA. Cpr-induced trauma: Comparison of three manual methods in an experimental model. *Ann Emerg Med*. 1986;15:674-679
54. Barranco F, Lesmes A, Irles JA, Blasco J, Leal J, Rodriguez J, Leon C. Cardiopulmonary resuscitation with simultaneous chest and abdominal compression: Comparative study in humans. *Resuscitation*. 1990;20:67-77
55. Soroff HS, Ruiz U, Birtwell WC, Many M, Giron F., Deterling RA. Assisted circulation by external pressure variation. *Physiology and Metabolism*. 1969;5:506-513
56. McDonald JL. Effect of interposed abdominal compression during cpr on central arterial and venous pressures. *Am J Emerg Med*. 1985;3:156-159