1994

The Evolution of Abdominal Compression in Cardiopulmonary Resuscitation

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SPECIAL CONTRIBUTION

Proceedings of the First Purdue Conference on IAC-CPR

The Evolution of Abdominal Compression in Cardiopulmonary Resuscitation

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ABSTRACT

Objective: To review the history of external abdominal compression as an adjunct to cardiopulmonary resuscitation (CPR), tracking the development of five major themes over the course of the 20th century: 1) augmentation of peripheral resistance by physical means, 2) risk of hepatic injury with abdominal compression, 3) counterpulsation vs. sustained compression, 4) the abdominal pump mechanism, and 5) contact compression techniques.

Methods: Literature retrieved from successive MEDLINE English-language searches was reviewed with a special emphasis on work and concepts highlighted by participants at the First Purdue Conference on Interposed Abdominal Compression-CPR, September 1992.

Results: External abdominal compression of one form or another has been studied as a means of resuscitation by many investigators throughout the 20th century. Experimental and clinical studies have shown generally consistent evidence of hemodynamic augmentation by abdominal compression during various forms of CPR. Recent advances include a modified theoretical understanding of hemodynamic mechanisms and demonstration of clinical potential in humans. Inconsistencies in published results may be due to differences in mechanical techniques of abdominal compression. Based on these studies, a modified manual technique for "contact compression" of the abdominal aorta is recommended.

Conclusions: A technique for left-of-center, angled compression of the abdominal aorta against the crest of the spine is recommended. Further well-supervised and controlled clinical trials using this standardized technique are warranted as a prelude to more widespread clinical application of abdominal compression in CPR.

Key words: abdomen, adjunct, animal studies, counterpulsation, history, IAC-CPR, interposed, physiology, preclinical studies. review

Acad. Emerg. Med. 1994; 1:469-477

Supported in part by Grant HL-42015 from the National Heart, Lung, and Blood Institute, U. S. Public Health Service, Bethesda, Maryland, USA.

Five unifying concepts have emerged during the evolution of abdominal compression as an adjunct to resuscitation from cardiac arrest: 1) the desirability of increasing peripheral resistance to nonvital organs by physical as well as by pharmacologic means, so that available blood flow can be directed toward the heart and the brain; 2) the danger of complications, including aspiration and hepatic trauma from abdominal compression; 3) the recognition that the benefits of abdominal compression can be achieved, and complications avoided, when abdominal compression is applied in a phasic manner, typically during the release phase of chest compression; 4) the demonstration that phasic external compression of the abdominal aorta can invoke an independent blood-pumping mechanism, in which blood is squeezed from the aorta into the periphery, which is complementary to the mechanisms impelling forward flow in response to chest compression; and 5) the finding that the most direct route to external abdominal aortic pumping is by left-of-midline manual compression of the aorta against the crest of the spine in a target zone just craniad to the umbilicus.

The objectives of the present paper are to review the evolution of abdominal compression in resuscitation and to suggest specific refinements in manual technique for future clinical trials. To this end, articles addressing abdominal compression were retrieved from successive MEDLINE English-language searches and were reviewed with a special emphasis on work and concepts highlighted by participants at the First Purdue Conference on Interposed Abdominal Compression-Cardiopulmonary Resuscitation (IAC-CPR), September 1992.

SUSTAINED ABDOMINAL COMPRESSION DURING CPR

In 1906 Crile and Dolley noted the importance in CPR of obtaining a coronary artery perfusion pressure of at least 30-40 torr, and advocated use of a pneumatic pressure suit placed over the legs and abdomen, as well as epinephrine, to increase peripheral resistance and to prevent peripheral pooling of venous blood.[1] In 1914 Crile wrote in his book Anemia and Resuscitation,

> The author was able, personally, to produce on a recently deceased person, a total circulation, which caused a pulse in the radial artery and bleeding of the peripheral vessels. Even a measurable blood pressure (registered by a sphygmomanometer) could be recorded through the combined effect of a tightly blown up rubber suit, which covered the lower extremities and the abdomen, and the strong, rhythmical pressure through the hands, placed broadly on both sides of the thorax.[2]

Shortly after the rediscovery of closed-chest cardiac massage by Kouwenhoven, Jude, and Knickerbocker in the late 1950s,[3] Birch and coworkers[4] studied dogs and baboons as animal models for CPR. They produced ventricular fibrillation with transchest electric shock and performed mechanical CPR with a pneumatic piston device. In one of the animals they exerted sustained abdominal pressure manually during chest compression to "augment venous return." Necropsy of this animal showed rupture of the liver. A similar result was obtained in a second animal. In 1967 Harris, Kirimli, and Safar[5] studied a variety of interventions to improve closed-chest CPR, including continuous manual compression of the upper abdomen during CPR, in dogs with electrically induced ventricular fibrillation. These studies were conducted with a Beck-

Rand sternal compressor and fixed tidal volume ventilation (15 mL/kg) interposed after every fourth sternal compression. In six dogs, carotid flows consistently increased by a factor of 1.5 to 2 when sustained, upper abdominal compression was added to otherwise conventional external CPR. However, these investigators also found liver lacerations at necropsy in two of six dogs and, therefore, did not recommend sustained manual compression of the upper abdomen as an adjunct to CPR in clinical practice.

In 1971 Redding, who was well aware of the benefits of raised peripheral vascular resistance in CPR,[6] reinvestigated the benefit-risk of static abdominal binding in a study involving 145 dogs. He demonstrated improved carotid arterial flow and survival in experimental CPR using continuous abdominal compression, produced by a blood pressure cuff secured around the mid-abdomen and inflated to 150-200 torr.[7] This intervention provided a physical means of raising peripheral vascular resistance that was directly comparable to chemical means, namely vasoconstrictor drugs. Redding's model of cardiac arrest was produced by occlusion of the endotracheal tube for 6-9 minutes until pulsatile blood pressure ceased. Standard or experimental CPR was performed until pulsatile blood pressure returned or until 20 minutes had elapsed. Surviving animals were observed for 24 hours.

Redding consistently observed that whenever aortic diastolic pressure during CPR could be raised above 40 torr, pulsatile blood pressure could be restored. Standard CPR was effective in producing aortic diastolic pressure of 40 torr or more in only a few dogs, and in these animals spontaneous circulation returned. On the other hand, CPR augmented either by methoxamine (a vasoconstrictor drug) or by abdominal binding usually led to aortic diastolic pressures above 40 torr, with return of pulsatile blood pressure. Continuous abdominal binding in Redding's studies produced, on the average, 34 torr higher aortic diastolic pressure than did standard CPR. Redding observed no greater incidence of liver damage to animals during CPR with continuous abdominal binding than in similarly resuscitated animals without abdominal binding.

Redding's work is particularly important because the endpoint measured—survival—is the one of paramount clinical interest. Three of 15 dogs receiving standard CPR for 20 minutes or less after asphyxial cardiac arrest resumed spontaneous circulation, and one of the 15 survived 24 hours. However, 14 of 15 dogs receiving CPR with abdominal binding for 20 minutes or less after asphyxial cardiac arrest resumed spontaneous circulation, and nine survived 24 hours. Subsequent studies with abdominal compression at 100, 150, and 200 torr and with the combination of abdominal compression and methoxamine in 40 additional animals had outcomes consistent with these favorable results.

A decade later Bircher, Safar, and Stewart reported a preliminary study of mechanical CPR using a Thumper Cardiopulmonary Resuscitator (Michigan Instruments, Grand Rapids, MI) in broad-chested dogs emerging from halothane anesthesia with electrically induced ventricular fibrillation. These animals were resuscitated with a pediatric pneumatic antishock garment continuously inflated about the legs and abdomen for ten-minute periods, alternating with ten-minute control periods.[8] "No major complications attributable to the . . . [pneumatic antishock garment], ... particularly no major lacerations of the liver" were found in 12 dogs receiving this treatment. Diastolic arterial pressure and carotid arterial flow increased nearly twofold.

The authors subsequently repeated CPR experiments with the pneumatic antishock garment and the Thumper device in another six broad-chested, ketamine-anesthetized dogs with electrically induced ventricular fibrillation in order to observe the ease of restoration of spontaneous circulation and neurologic recovery thereafter.[9] After five minutes of standard CPR, a pediatric pneumatic antishock garment was inflated to 90 torr, and CPR continued for 30 minutes. There was a doubling of carotid arterial blood flow. However, this group also had significantly more arterial hypoxemia and hypercarbia compared with control animals receiving standard CPR, indicating gross intrapulmonic shunting in the pneumatic antishock garment-CPR group. The authors attributed the blood-gas deterioration to restriction of ventilation by the garment, which was used in conjunction with the constant pressure ventilation system of the Thumper device. Strikingly, all dogs in the pneumatic antishock garment CPR group had ruptured livers with hemorrhage into the peritoneal cavity, whereas there was no such injury in seven dogs in the standard CPR group. In comparing their two studies, Bircher and coworkers concluded that "the mechanism of hepatic damage and hypoventilation may be the splinting of the liver immediately beneath the lower sternum." Hepatic injury was found only in the second study, which used longer periods of continuous abdominal compression.

In contrast, Chandra, Rudikoff, Weisfeldt, and their coworkers at Johns Hopkins reported several CPR studies using sustained abdominal binding in animals and in man that suggest elevated blood pressures and flows can be achieved without liver damage.[10,11] In a series of 15 dogs they found that tightly binding the abdomen over an inflatable bladder with adhesive tape from the xyphoid to the iliac crest increased aortic systolic pressure from 28 to 57 torr and carotid blood flow from 14 to 32 mL/min.[11] Carotid flows increased in 13 of the 15 dogs after abdominal binding, and there was no evidence of liver injury with mechanically controlled chest compression and abdominal binding in any of the 15 animals.[11]

In Chandra et al.'s human studies, radial-artery blood pressure was measured during 30-60 second periods of abdominal binding in ten patients experiencing cardiac arrest, and standard CPR was applied with a mechanical device. The abdomen was bound with an inflatable 30-cm square bladder that was positioned anteriorly on the patient's abdomen, extending from the lower ribs to the anterior superior iliac spine, held in place by a large strap and inflated to approximately 100 torr. Abdominal binding increased diastolic radial-artery pressure from 35 to 50 torr on the average. In six patients who underwent abdominal binding for periods in excess of four minutes, higher arterial pressure persisted for the duration of abdominal binding. One of these six patients was successfully resuscitated, but died 24 hours later. None of the six patients showed evidence of abdominal visceral injury at autopsy. Specifically, no liver rupture was seen.

The issue of liver laceration is obviously crucial in any discussion of the overall benefit-risk of static abdominal compression in CPR. Based upon these studies, the risk of liver damage and intraperitoneal hemorrhage in patients who undergo abdominal compression during CPR is difficult to estimate. Variability of this complication among the earlier studies suggests that the risk is technique-dependent. Although liver rupture was avoided in a large majority of experimental subjects and survival benefits were found with abdominal binding,[7] the potential for hepatic injury strongly dampened initial enthusiasm for the technique.

ABDOMINAL COUNTERPULSATION DURING CPR

Abdominal counterpulsation refers to the addition of interposed abdominal compressions (IAC) to otherwise standard CPR. During IAC-CPR, as opposed to CPR using continuous abdominal binding, the abdomen is compressed alternately or reciprocally as chest compression is released. With IAC-CPR, little or no pressure is applied to the abdomen during chest compression, so the liver is not splinted and trapped beneath the rib cage, but rather is free to move in response to transidaphragmatic pressure gradients. The technique was discovered independently by a variety of research teams in the 1970s and 80s. Phasic manual compression of the thoracic aorta, alternating with internal cardiac massage, in an open-chest, anesthetized dog was first described by Molokhia and coworkers in 1972.[12] They found that the addition of interposed, digital aortic compression during internal cardiac massage significantly improved coronary sinus blood flow. In 1976 Ohomoto and co-workers[13] at the Tokyo Women's Medical College described an arrangement of two mechanical pistons for external CPR, one that compressed the chest and another that compressed the abdomen. They called the technique "counter-massage" and found in preliminary studies that phased abdominal compression for 80% of cycle time and chest compression for 25% of cycle time improved carotid flow, mean aortic pressure, and short-term survival in anesthetized dogs having ventricular fibrillation. Independently, in Houston, Texas, while attempting to develop an animal model of cough-CPR, Rosborough and coworkers[14] combined simultaneous high-pressure lung inflation with abdominal compression. They found that abdominal compression and ventilation alone could maintain carotid flow and aortic blood pressure during ventricular fibrillation in dogs, and they suggested the technique as a new CPR modality.

In our laboratory the method was first demonstrated serendipitously by Ralston during a difficult resuscitation of an anesthetized dog. In a subsequent controlled study, ventricular fibrillation was induced electrically in pentobarbital-anesthetized dogs. Cardiac output during alternate three-minute trials of the two techniques was measured using a modified indicator dilution technique adapted to the low-flow conditions of CPR,[16,17] and central arterial and venous blood pressures were also measured. Ventricular fibrillation was induced electrically in ten anesthetized dogs, and either IAC-CPR or standard CPR was initiated, while arterial and venous blood pressures and cardiac output were monitored. Ventilation was provided via the constant-pressure ventilator of the Thumper device. The two CPR methods were alternated every three minutes over a period of 30 minutes.

IAC-CPR was performed by manual compression of the mid abdomen with a standard 12-cm-wide blood pressure cuff, folded to rectangular dimensions of 12 x 15 cm and inflated with air to a thickness of 3 cm. The bladder of the cuff was attached to a transducer in order to monitor pressure applied to the abdomen. During the release phase of chest compression, manual pressure was applied to the abdomen, generating pressure pulses of 120-150 torr. The over-and-under hand position for abdominal compression was similar to that used in basic CPR for manual chest compression, except that the fingers of the bottom hand were spread to provide a surface area of compression approximately equal to that of the flattened blood pressure cuff. The duty cycle of abdominal compression was exactly complementary to that of chest compression (i.e., 50% of cycle time). Results of this study showed that the addition of IAC to standard CPR

essentially doubled coronary perfusion pressure and cardiac output. Other animal studies confirmed this effect when constant volume ventilation, rather than constant pressure ventilation, was applied and also demonstrated improved oxygen delivery to peripheral tissues.[18]

At about the same time, Coletti and coworkers[19,20] in New York were studying the influence of abdominal counterpulsation upon cerebral and coronary blood flow in a canine model of cardiogenic shock. They found that when manual abdominal compressions were interposed between heartbeats, as judged from the ECG trace, both cerebral and coronary perfusion increased. Using a side-to-side chest compression technique, as is accepted practice in veterinary medicine, [21] Hoehner and coworkers[22] in Detroit studied perfusion of the cerebral cortex during IAC versus standard CPR in a canine model. In their study, using both manual chest and manual abdominal compressions in large supine dogs, cerebral cortical blood flow, measured with a thermal washout method immediately after beginning CPR, averaged 0.06 mL/min/g in animals receiving chest compression alone and 0.27 mL/min/g in animals receiving chest compressions with IAC-CPR. Independently, Voorhees and coworkers,[23] using the radioactive-microsphere technique in dogs, obtained a cerebral perfusion of 0.28 mL/min/g with IAC-CPR. This value was significantly greater than that during standard CPR and nearly identical to that reported by Hoehner and co-workers. These values obtained with IAC-CPR represent approximately 30-50% of prearrest cerebral perfusion in dogs under anesthesia.

HEMODYNAMIC MECHANISMS OF IAC-CPR

In addition to the animal studies just described, several theoretical papers set the stage for subsequent clinical investigations of IAC-CPR. In our laboratory we developed an electrical model of the circulation to study relevant hemodynamic mechanisms.[24, 25] The heart and blood vessels were modeled as resistive-capacitive net- works, pressures as voltages, blood flow as electric current, blood inertia as inductance, and the cardiac and venous valves as diodes. Pressurization of the chest and abdomen, as would occur in CPR, was simulated by application of half-sinusoidal voltage pulses between vascular capacitances and ground. The model was implemented in an analog electrical circuit and typically was arranged to simulate thoracic pump CPR,[26] in which blood is impelled by compression of all intrathoracic vascular structures. Instantaneous pressures and flows at any chosen point in the circulation could be displayed on an oscilloscope chest compression in this model, produced flow augmentation according to the expression

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

where P_{th} = peak intrathoracic pressure, P_a = peak abdominal pressure or, more precisely, peak perivascular pressure exerted on the aorta or on the inferior vena cava, and α and β are constants ($\alpha > \beta$). IAC enhanced simulated blood flow to the heart and brain as well as total flow in the electrical model. The degree of flow augmentation depended on both the abdominal pressure applied and the peripheral vascular resistance. For standard conditions, flow during IAC-CPR was about twice that without IAC, duplicating results in animal experiments and confirming that effects seen in the canine model were not caused by anatomic peculiarities of the dog. Further,

simulated IAC-CPR with high peripheral resistance values in this model (as would be created in vivo by epinephrine administration) generated current flow to the head and neck corresponding to a blood flow roughly 70% of normal, the highest value obtained in any simulation.

In a companion mathematical model of the most important resistive capacitive elements of the circulation involved in IAC, the mechanism for the generally positive augmentation of coronary perfusion pressures by IAC was explained on the basis of differences in the rise times of the intra- thoracic arterial and central venous pressure pulses induced in response to external abdominal compression.27 The same equations also anticipated future developments nearly a decade later, including the possibility that more direct compression of the abdominal aorta itself would be more effective than generalized abdominal compression and the possibility that maximal artificial cardiac output dur- ing IAC-CPR might be achieved at a faster overall compression rate (short- er cycle time) than maximal artificial cardiac output during chest compression alone.

CLINICAL STUDIES

Clinical research in IAC-CPR began with preliminary studies, focusing on hemodynamic mechanisms and safety. Berryman and coworkers[28] were the first to try the technique in six patients who failed to respond to conventional CPR in the emergency department. They found that IAC-CPR raised mean arterial pressure, and in one patient in whom it was measured, IAC-CPR increased the arteriovenous pressure difference. Subsequently, mixed results with the technique were reported. These included a small series of patients studied by McDonald, who compared blood pressures generated by IAC-CPR versus standard CPR.[29] A larger prehospital clinical trial by Mateer and coworkers[30] compared survival in a control group of patients resuscitated by conventional means with survival in an experimental group of patients resuscitated by a combination of IAC-CPR and standard CPR. IAC-CPR was administered before and after transport whenever it was practically possible to do so. Both of these studies found no difference in IAC-CPR versus standard CPR, either in terms of efficacy or in terms of safety. Importantly, Mateer's initial randomized study found no difference in the incidence of emesis or abdominal trauma.

Meanwhile, Howard and Guinness et al.[31-33] were making measurements of coronary perfusion pressure in 14 patients during alternate two-minute trials of IAC-CPR 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.[16, 18] Additionally, three patients had 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 coworkers[34] studied end-tidal CO_2 in 33 adult patients resuscitated with either standard CPR or IAC-CPR in a randomized, crossover design, in which each patient served as his or her own control. CO_2 excretion, an indicator of the rate of venous blood return to the right heart, increased in every patient who was switched from standard to IAC- CPR and fell in every patient who was switched from IAC to standard CPR.

The most definitive and recent studies of outcome in well-defined clinical populations have been reported by Sack and coworkers.[35] They prospectively studied 103 patients undergoing 135 resuscitation attempts. Patients experiencing in-hospital cardiac arrest were treated exclusively either with IAC-CPR or with standard CPR. End-points included both the frequency of initial return of spontaneous circulation and the frequency of long-term survival. The frequency of return of spontaneous circulation 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% versus 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 ($p = 0.02$).

In the forgoing clinical study, the most striking outcome advantage was seen in those patients in whom the initial arrest rhythm was asystole or electromechanical dissociation (EMD). Such an individual has an extremely grave prognosis,[36] and successful restoration of spontaneous circulation may be more dependent on the maintenance of adequate coronary perfusion pressure than on restoration of spontaneous circulation in patients who have ventricular fibrillation or ventricular tachycardia. In a subsequent prospective study of 143 patients with in-hospital cardiac arrest from either asystole or EMD, Sack and coworkers[37] demonstrated a statistically significant improvement in immediate resuscitation outcome and 24-hour survival with the addition of interposed abdominal compressions to otherwise standard CPR.[37] Immediate resuscitation rates (28% versus 48%) and 24 hour survival rates (13% versus 33%) were better with IAC-CPR than with standard CPR.

Complications attributable to IAC-CPR in all clinical series have been minimal.[30,35,37] Currently there is only one reported case of IAC-related abdominal organ damage in approximately 300 resuscitated patients described in the literature. This case of necropsy-confirmed pancreatic injury occurred in a child receiving interposed abdominal compression during CPR.[38] Initial concerns regarding the possibility of more frequent emesis during IAC-CPR than during conventional CPR have remained unsubstantiated. All three randomized clinical trials previously cited failed to show a difference in rates of emesis between the IAC and standard CPR groups. Interestingly, in anesthetized dogs, IAC-CPR produces less gastric insufflation than standard CPR when ventilation pressure is applied to both the trachea and the esophagus,[39] probably because the abdominal pressure that is maintained during ventilation tends to prevent gastric insufflation during positive pressure rescue breathing.

FURTHER REFINEMENTS IN TECHNIQUE

Most published research on the subject suggests that the addition of manual IAC to otherwise standard CPR is likely to improve hemodynamics during resuscitation. There is exceedingly little evidence that IAC-CPR is harmful or less beneficial than standard CPR. However, there are occasional inconsistencies in the results obtained at different institutions.

During the First Purdue Conference on IAC-CPR in October 1992, currently active investigators studying IAC-CPR not only presented summaries of their work, but also gave practical demonstrations of the various techniques of phasic abdominal compression under investigation in current clinical studies. The practical sessions revealed remarkable differences in the precise

mechanics by which external abdominal compressions were being applied at various institutions. The alternative methods demonstrated at the conference included 1) central abdominal compression centered over the umbilicus, using over-and-under hand position similar to that used for chest compressions in standard CPR; 2) epigastric compression, using over-and-under hand compression; 3) left paramedian compression, using side-by-side hand position, with one hand caudad and one hand craniad to the umbilicus; 4) broad abdominal compression, centered over the umbilicus, using approximated open hands with thumbs together and fingers widely spread; 5) broad, central abdominal compression, using a hardcover book to distribute the force uniformly over the anterior abdominal wall.

These sessions prompted analyses of some neglected anatomic and mechanical aspects of abdominal compression, leading to guidelines for more consistent, effective, and safe IAC in future trials.[40] A key concept in understanding the mechanical effects of IAC was contributed by my colleague, Jack Debes, at Purdue University, who pointed out the distinction between contact compression and hydrostatic compression of structures within a gas and fluid filled space such as the human abdomen. In IAC-CPR, contact compression occurs to the extent that the localized external force applied to the abdominal wall is directly transmitted through intervening tissues to underlying structures such as the aorta and great veins. Hydrostatic compression occurs to the extent that a generalized rise in intra-abdominal pressure is created and transmitted uniformly to all sides of various intra-abdominal structures. If only generalized hydrostatic compression of the abdominal vessels occurred during cardiac arrest, the stiff wall of even a non-diseased aorta would resist such compression more than the compliant walls of intra-abdominal veins, and central venous pressure would tend to rise during abdominal manipulation even more than thoracic aortic pressure. Maximal augmentation of coronary perfusion pressure, therefore, must require selective contact compression of the abdominal aorta.

Assuming that the goal of IAC-CPR is to maximally compress the abdominal aorta with minimum risk to other vital viscera, a simple review of the relevant human anatomy,[41,42] suggests one particularly safe and direct route for external abdominal counterpulsation, which is different from methods previously reported. This route can be described in terms of the craniad-caudad and right-left coordinates of the skin-surface contact point for external abdominal compression and also by the angle or vector of the requisite compression force.

Because the abdominal aorta bifurcates at the level of the umbilicus, IAC techniques that center on the umbilicus, especially those involving large areas below the umbilicus, are likely to be only partially effective in achieving contact compression of the abdominal aorta itself. To achieve direct contact compression of the abdominal aorta, the best site for IAC must be headward of the umbilicus--no farther than the tip of the xiphoid process. Because the rectus and oblique muscles are tethered to the tapering costal margins superiorly, where they tend to impede effective IAC applied in the immediate subcostal third of the sterno-umbilical line, it is natural in practice to constrain IAC to a level corresponding to the lower two-thirds of the sterno-umbilical line. Avoidance of the immediate subcostal region also minimizes the chances of traumatizing the left lobe of the liver and the head of the pancreas. In an adult this target level includes a span of about 8-9 cm cranial to the umbilicus, just enough to accommodate the width of one hand.

Abdominal cross-sectional anatomy and computed tomography scans suggest similarly tight constraints on the right-left coordinate and angle of applied force for optimal contact compression of the abdominal aorta against the bony pedestal of the lumbar spine. Anatomically, the most obvious and direct means of contact compression is by application of a force vector, which if extended through the body would traverse the central axis of the aorta at an angle perpendicular to the surface of the vertebral column (Fig. 1). Such a force at a mean angle about 11 degrees from the vertical[40] would most effectively tend to flatten the aorta against the spine, which is tethered in position by retroperitoneal fascia and the emerging segmental arteries. At this site, the aorta is more susceptible to direct compression through the anterior body wall than more lateral structures. The only intervening structures in the direct path of the compressive force are overlying loops of bowel, which are free to move in response to applied manual compression and thus are likely to avoid repeated entrapment against the spine.

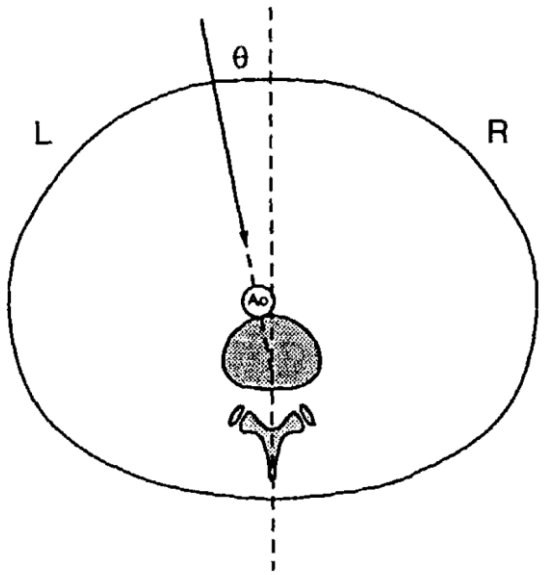

FIGURE 1. Sketch of human cross-sectional anatomy showing relations of the aorta, spine, and body wall two fingerbreadths above the intercristal plane. The abdominal aorta rests near the crest of the spine with its longitudinal axis one-fourth to one-half of the aortic diameter to the left of the midline. The angle, θ, of the force vector most likely on anatomic grounds to compress the aorta directly against the spine is shown with respect to the median sagittal plane. Redrawn from Carter et al.[41] This angle averages 11 degrees.

Thus, if one's goal is to maximize aortic contact compression, anatomic considerations severely constrain the site and direction of the required manual force vector in three dimensions. The craniad-caudad coordinate is one-third of the distance between the level of the umbilicus, or intercristal plane, and the level of the xiphisternal junction. The right-left coordinate is one-fifth the distance from the umbilicus to the lateral abdominal margin or 3 cm to the left of midline; and the direction of force is perpendicular to the long axis of the aorta in the sagittal plane and about 11 degrees from the vertical in the transverse plane.[40] (For obese subjects the right-left coordinate of the compression point on the skin surface must be a further absolute distance from the midline to maintain the nominal 11 degree angle.)

The validity of these spatial coordinates for the most direct vector from the skin surface to the abdominal aorta can be tested by any reader through self-examination or during routine physical examination of the abdomen in patients. Self-examination may be done either in the sitting position, or preferably in the supine position. Examination just before, rather than just after, a meal is recommended, since food in the pyloric antrum can somewhat obscure the palpation of

the aortic pulse. With about 5 cm of painless compression using the opposing fingertips, the aortic pulse of a lean individual can be readily appreciated. With one additional centimeter of compression the pulse becomes very strong, and with deep pressure one can appreciate a thrill over it, providing evidence of turbulent flow consistent with compression of the abdominal aorta by an external, manual technique.

A natural position of rescuer and victim to achieve such contact compression of the abdominal aorta is one in which the rescuer kneels on the victim's left side (Fig. 2, top). If the rescuer's knees touch the side of the victim's abdomen and the rescuer's arms extend straight toward the abdominal surface, the extended arms will make an angle with the vertical of approximately 11 degrees, as the rescuer rocks forward to compress the abdomen with the hands positioned slightly to the left of midline.

The exact hand position for reasonably direct contact compression of the abdominal aorta may be given by a "rule of thumb," as shown in Figure 2, bottom. In non-obese subjects the right hand is placed on the abdomen so that the umbilicus appears in the thumb-index angle, the thumb pointing toward the feet. The heel of the right hand is centered approximately 3 cm to the left of midline, and the fingers are slightly curved to minimize contact compression of the inferior vena cava on the right side of the spine. The fingers curl convex, upward slightly, and the fingertips rest gently on the abdomen, not to apply force, but only to steady the position of the hands. In this way contact is made through the hypothenar and thenar pads of the right hand, along a craniad-caudad line overlying the abdominal aorta. This position is easily practiced on a normal volunteer, in whom the aortic pulse can be well appreciated as the aorta is minimally compressed against the spine without significant discomfort to the volunteer.

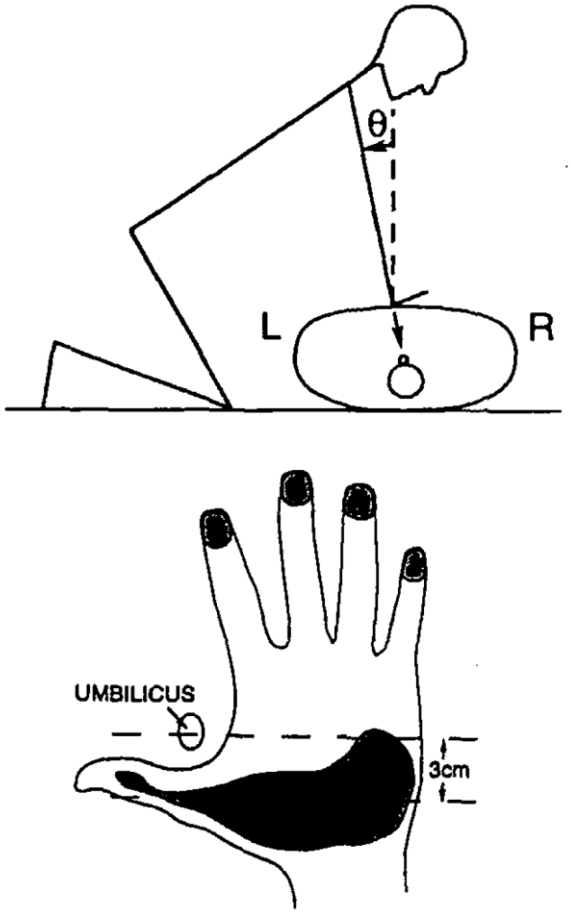

FIGURE 2. Sketches of recommended technique for application of interposed abdominal compressions during CPR. *Top,* natural position of rescuer and victim for proposed anatomically optimal, transcutaneous compression of the abdominal aorta: the rescuer kneels on the victim's left side with weight toward the rescuer's heels and straight arms extended to apply IAC. When the chin is over the wrists, the angle of compression is approximately 11 degrees from the vertical. *Bottom,* "rule of thumb" for locating the best compression point on the abdominal wall by a rescuer kneeling at the victim's left side. In non-obese subjects the right hand is positioned so that the umbilicus appears in the thumb-index angle, thumb pointing toward the feet. The heel of the right hand is centered approximately 3 cm to the left of the midline, overlying the abdominal aorta above its bifurcation. Shaded areas indicate direct contact between the right hand and the abdominal wall when the fingers are slightly curved to minimize contact compression of the inferior vena cava on the victim's right. For obese subjects the craniad-caudad level is judged with respect to the bony landmark of the left iliac crest, and the heel of the right hand is moved laterally so that the umbilicus lies opposite the knuckles.

The amount of force required also can be practiced in this exercise, because the external compression force needed to obtain a maximal palpable aortic pulse in a normal, healthy subject[40] is the same force that generates an internal periaortic counterpressure equal to normal mean aortic pressure, or about 95 torr. When periaortic pressure is substantially less than intraluminal pressure, the aorta remains fully open. When periaortic pressure equals mean intraluminal pressure, the aorta intermittently collapses, and the local palpable pulse, caused by radial expansion of the aorta, is maximal. When periaortic pressure is substantially greater than intraluminal pressure, the aorta fully collapses, and the local pulse is attenuated. Thus an aortic wall pressure, approximating mean arterial pressure, is a reasonable value for circulatory augmentation during CPR. The required external force stops just short of producing painful abdominal compression in awake, normal volunteers or in patients undergoing routine physical examination of the abdomen. The forgoing recommendations for site, angle, and force of external abdominal compression may well provide a basis for standardized, uniform technique in future multicenter clinical trials.

CONCLUSION

During the past century a wide variety of investigators have demonstrated, both in animals and in man, improved blood pressure and blood flow above the diaphragm when some type of external compression is applied below the diaphragm in addition to conventional resuscitative measures. Rhythmically interposed abdominal compressions seem to provide even greater hemodynamic benefit and substantially less risk than does continuous abdominal binding. Both total flow and the distribution of flow to vital organs above the diaphragm are improved, while the chances of liver entrapment and damage during chest compression are reduced. The technique of IAC-CPR can be performed with the bare hands of a second or third rescuer. It requires no special equipment and in the future could be incorporated into training programs for basic rescuers. In this sense IAC-CPR may constitute a logical evolution in basic life support. Future research involving multiple institutions will require standardization of manual techniques for the application of IAC-CPR. The left-sided, angled technique for selective aortic compression may represent a further refinement of abdominal manipulation in resuscitation.

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