

1984

Preclinical Studies of Abdominal Counterpulsation in CPR

Charles F. Babbs

Purdue University, babbs@purdue.edu

Follow this and additional works at: <http://docs.lib.purdue.edu/bmepubs>



Part of the [Biomedical Engineering and Bioengineering Commons](#)

Recommended Citation

Babbs, Charles F., "Preclinical Studies of Abdominal Counterpulsation in CPR" (1984). *Weldon School of Biomedical Engineering Faculty Publications*. Paper 83.
<http://docs.lib.purdue.edu/bmepubs/83>

This document has been made available through Purdue e-Pubs, a service of the Purdue University Libraries. Please contact epubs@purdue.edu for additional information.

Preclinical Studies of Abdominal Counterpulsation in CPR

Charles F. Babbs, M.D., Ph.D.

Biomedical Engineering Center, Purdue University, West Lafayette, Indiana, USA.

[Ann Emerg Med September 1984 (Part 2);13:761-763]

Abstract

Abdominal counterpulsation added to standard cardiopulmonary resuscitation improves blood flow in animal models when compared to chest compressions alone. Similar effects can be demonstrated in analog and digital computer models of the circulation. The technique generates both central aortic and central venous pressure pulses, and successful application of the method depends on maximizing the former and minimizing the latter. Proper technique is important in order to generate the largest possible arteriovenous pressure difference.

Key words: abdominal, adjuncts, cardiopulmonary resuscitation, IAC, interposed abdominal compression

Introduction

The technique of abdominal counterpulsation during cardiopulmonary resuscitation (CPR) consists of applying external pressure to the abdomen during "diastole," the chest relaxation phase. During interposed abdominal compression-CPR (IAC-CPR), abdominal pressure is maintained manually over the mid abdomen for 50% of cycle time, exactly 180 degrees out of phase with chest compressions. A folded blood pressure cuff placed between the hands and the abdomen and connected to an aneroid manometer may be used to monitor the pressure applied. We have obtained good results with negligible trauma in animals using applied abdominal pressures of 120 to 150 mm Hg.

Supported by Research Career Development Award HL-00587 from the National Heart, Lung, and Blood Institute, U.S. Public Health Service, Bethesda, Maryland.

Hemodynamic Effects in the Canine Arrest Model

The hemodynamic effects of abdominal counterpulsation have been discovered independently by several research teams in the last decade. In 1976 Ohomoto and coworkers [1] at Tokyo Women's Medical College described an arrangement of two mechanical pistons, one that compressed the chest and another that compressed the abdomen. They termed 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 appeared to improve carotid flow, mean aortic pressure, and short-term survival in anesthetized dogs with ventricular fibrillation.

Rosborough and coworkers [2] in Houston, while attempting to mimic the physiology of "cough-CPR" in an animal model [3], 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 1981 Ralston, of Purdue University, observed the hemodynamic effects of interposed abdominal compressions as an adjunct to mechanical chest compression in dogs [4]. Interposed abdominal compressions dramatically improved brachial artery blood pressure without a comparable increase in central venous pressure (Figure 1). A subsequent controlled study of IAC-CPR in ten dogs with electrically induced fibrillation showed that the technique significantly improved cardiac output and diastolic arterial pressures [4]. In this study cardiac output was measured with an indicator dilution method specially modified for CPR [5] and constant pressure (20 cm H₂O) ventilation was used.



Fig. 1. Effect of manual abdominal counterpulsation on aortic blood pressure (ABP) and central venous pressure (CVP) in mm Hg during otherwise conventional CPR at compression rate 60/min. Chart speed was increased on right. The subject was an anesthetized dog with ventricular fibrillation.

Voorhees et al. [6] at Purdue have continued this line of research and have shown 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 otherwise standard CPR (Figure 2). In this second study a specially modified spirometer recorded oxygen consumption during CPR. Cardiac output was measured by the Fick technique (O_2 consumption/A-V O_2 difference), and constant volume ventilation, rather than constant pressure ventilation, was applied.

At about the same time Coletti and coworkers in New York were studying the influence of abdominal counterpulsation on cerebral and coronary blood flow in a canine model of cardiogenic shock [7, 8]. They found that when manual abdominal compressions were interposed between heartbeats (as judged from the ECG trace), both carotid and coronary flow, measured with implanted electromagnetic probes, increased.

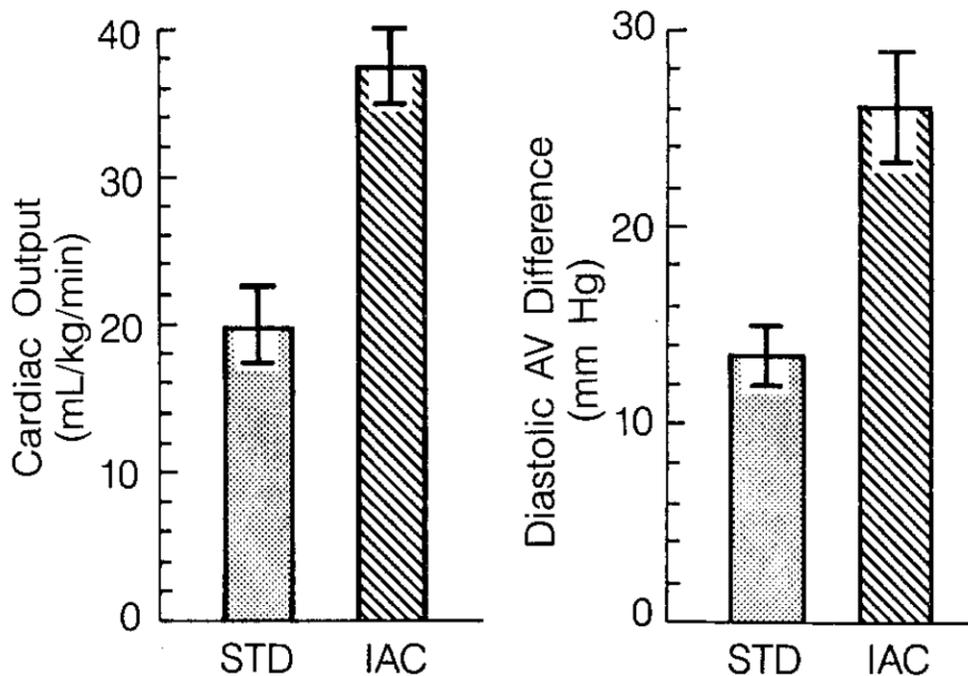


Fig. 2. Cardiac output during alternate trials of standard CPR and IAC-CPR [6].

A study by Walker and coworkers in Detroit [9] has confirmed the earlier animal experiments. These investigators measured perfusion of the cerebral cortex during IAC versus standard CPR in a canine model. They used both manual chest and manual abdominal compression in large supine dogs, in which cardiac arrest was produced by intravenous potassium chloride. The chest compression was done in a side-to-side manner, which is accepted practice in veterinary medicine [10]. They found that cerebral cortical blood flow, measured with a thermal washout method [11] 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. This value obtained with IAC-CPR

was 51% of prearrest cerebral perfusion in the dogs. Voorhees et al, using the radio-labeled microsphere technique to measure regional flow, found cerebral perfusion of 0.28 mL/min/g with IAC-CPR [12], a value significantly greater than that during standard CPR and nearly identical to that reported by Walker et al. [9].

Supportive Data From Electrical Models

The animal studies suggesting the value of IAC as an adjunct to CPR have been confirmed by the author and his coworkers in an electrical model of the circulation [13, 14]. We modeled the heart and blood vessels as resistive-capacitive networks, 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 typically arranged to simulate "thoracic pump" CPR [15], in which blood flow occurs during "pressurization" of all intrathoracic vascular structures. IAC added to chest compression in this model produced flow augmentation according to the expression

$$\text{flow} = \alpha P_c + \beta P_a ,$$

where P_c = peak intrathoracic pressure, P_a = peak abdominal pressure, 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. Simulated IAC-CPR with high peripheral resistance values in this model generated current flow to the head and neck corresponding to a blood flow roughly 70% of normal, the highest value obtained in any simulation. Other theoretical models created by Dinnar at the Tehnion Institute in Haifa, Israel, also demonstrate the value of abdominal counterpulsation during CPR (personal communication, West Lafayette, Indiana, September 7, 1983). Dinnar's model required solution of simultaneous differential equations describing the circulatory system on a digital computer. His preliminary results showed that the greatest flow augmentation occurs when abdominal pulsation is 180 degrees out of phase with chest compression.

Mechanism For Effect and Implications for Regional Flow

There are two probable mechanisms for the beneficial effects of abdominal counterpulsation. The first is similar in some respects to that of the intra-aortic balloon pump: 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 peripheral perfusion, increasing cardiac output. The second mechanism is priming of the intrathoracic pump in a manner analogous to the action of the cardiac atria when the heart is beating normally. Both mechanisms have an effect in the electronic model [13], in that simulated coronary flow is improved when counterpressure is applied either to the abdominal aorta only or to the abdominal veins only.

In order for IAC to augment total flow and coronary perfusion, however, the induced increase in central aortic pressure must be greater than the induced increase in central venous pressure. It is becoming clear from a wide variety of studies that the prime determinant of coronary flow during cardiac arrest and CPR is the central arteriovenous pressure difference during chest recoil or "diastole." Such a diastolic pressure difference is crucial for coronary perfusion during CPR [16] and appears to be the key to restoration of spontaneous circulation when CPR lasts more than two to four minutes [16-18]. Abdominal counterpulsation undoubtedly can induce intrathoracic pressure pulses in both arteries and veins.

The studies just reviewed in animal models and in electronic models suggest that the induced arterial pulses are typically greater than the induced central venous pulses. In occasional animals, however, we have noted that the central venous pulses caused by IAC are greater than the thoracic aortic pulses. These include two of ten animals in Ralston's study [4] and two of nine animals in Voorhees' study [11]. In such animals the augmentation of cardiac output is poor, and myocardial flow is worsened rather than improved by IAC. To optimize IAC for clinical application, therefore, we must come to better understand the factors governing the balance between the induced intrathoracic arterial and venous pressure pulses. Only then can we maximize the diastolic arteriovenous pressure difference generated during IAC-CPR.

Conclusions

There is substantial evidence that abdominal compression can generate true intravascular pressure pulses in both arteries and veins, and that usually the augmentation of thoracic aortic pressure is greater than the augmentation of central venous pressure. In this case antegrade circulation, especially to the heart and brain, can be improved. There remains the possibility, however, that controllable factors, such as suboptimal technique or overloading of central venous capacitance, might abolish a net positive effect or even create venous pressure elevations greater than arterial pressure elevations. As we move from the laboratory into the clinic, therefore, it is important to be alert for these and other possible causes of failure, and to take rational steps to eliminate them. The most fruitful question at this stage is not "Will IAC-CPR work in man?" but rather "How can we make IAC-CPR work in man most effectively?"

References

1. 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.
2. Rosborough JP, Niemann JT, Criley JM, et al Lower abdominal compression with synchronized ventilation -- a CPR modality, abstract. *Circulation* 1981;64:303.
3. Criley JM, Blaufuss AH, Kissel GL: Cough-induced cardiac compression -- a self-administered form of cardiopulmonary resuscitation. *JAMA* 1976;236:1246-1250.
4. Ralston SH, Babbs CE, Niebauer MJ: Cardiopulmonary resuscitation with interposed abdominal compression in dogs. *Anesth Analg* 1982;61:645-651.

5. Geddes LA, Babbs CF: A new technique for the measurement of cardiac output during cardiopulmonary resuscitation. *Crit Care Med* 1980;8:131-133.
6. Voorhees WD, Babbs CF, Niebauer MJ: Improved oxygen delivery during cardiopulmonary resuscitation with interposed abdominal compressions. *Ann Emerg Med* 1983;12:128-135.
7. Coletti RH, Kaskel PS, Cohen SR, et al. Abdominal counterpulsation (AC) -- a new concept in circulatory assistance. *Trans Am Soc Artif Intern Organs* 1982;28:563-566.
8. Coletti RH, Kaskel PS, Bregman D: Abdominal counterpulsation (AC) -- effects on canine coronary and carotid blood flow, abstract. *Circulation* 1982;66:135.
9. Walker JW, Bruestle JC, White BC, et al. Perfusion of the cerebral cortex using abdominal counterpulsation during CPR. *American Journal of Emergency Medicine*, to be published.
10. Palich WE, Gordon AS: Cardiopulmonary resuscitation of dogs: Principles and practice. *J Am Vet Med Assoc* 1967;151:1719-1732.
11. Hoehner PJ, Krause GS, White BC, et al: Determination of cerebral cortical blood flow: A thermal technique. *Ann Emerg Med* 1983;12:2-7.
12. Voorhees WD, Ralston SH, Babbs CF: Regional blood flow during CPR with abdominal counterpulsation in dogs. *American Journal of Emergency Medicine* 1984;2:123-128.
13. Babbs CF, Ralston SH, Geddes LA: Theoretical advantages of abdominal counterpulsation in CPR as demonstrated in a simple electrical model of the circulation. *Ann Emerg Med* 1984;13:660-671.
14. Babbs CF, Weaver JC, Ralston SH, et al: Cardiac, thoracic, and abdominal pump mechanisms in CPR: Studies in an electrical model of the circulation. *American Journal of Emergency Medicine*, in press, 1984.
15. Babbs CF: New versus old theories of blood flow during cardiopulmonary resuscitation. *Crit Care Med* 1980;8:191-195.
16. Ralston SH: *Improved Regional Blood Flow and Resuscitability with Intrapulmonary Epinephrine During Prolonged Cardiopulmonary Resuscitation in Dogs*, thesis. Purdue University, West Lafayette, Indiana, 1982.
17. Ralston SH, Voorhees WD, Babbs CF: Intrapulmonary epinephrine during prolonged cardiopulmonary resuscitation: Improved regional flow and resuscitation in dogs. *Ann Emerg Med* 1984;13:79-86.
18. Redding JS: Abdominal compression in cardiopulmonary resuscitation. *Anesth Analg* 1971;50:668-675.