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ABSTRACT

To test the hypothesis that fluctuations in global intrathoracic pressure are the dominant cause of blood flow during external cardiopulmonary resuscitation (CPR) the authors studied the effects of open pneumothorax on experimental CPR in 7 domestic pigs and 12 mongrel dogs. Similar studies were conducted independently at three laboratories and are reported jointly. All studies were conducted during electrically induced ventricular fibrillation and with standard CPR technique, including ventral-dorsal chest compression at 60/min, 0.5 sec compression duration, 1:5ventilation:compression ratio. During alternate periods of CPR, intrathoracic pressure was vented through bilateral chest tubes, placed to create open pneumothorax and partial collapse of the lungs. During this maneuver, global intrathoracic pressure fluctuations were greatly attenuated, but direct but direct cardiac compression and adequate ventilation continued.

In the three laboratories, systolic/diastolic arterial pressures during CPR with thoracic venting (± SE) averaged 68 ± 4.2/28 ± 3.3, 60 ± 10/18 ± 4.5, and 66 ± 6.3/23 ± 1.5 mm Hg. These values are compared to 68 ± 4.4/27 ± 3.0, 67 ± 12/17 ± 6.1, and 56± 6.2/22 ± 1.9 mm Hg with the thorax intact. Carotid artery mean flow, measured with an in-line flowmeter, was 13.0 ± 2.2 ml/min vented vs. 13.4 ± 2.6 intact in 7 pigs; 11.4 ± 3.8 ml/min vented vs. 11.2 ± 3.7 intact in 5 dogs. Cardiac output, determined by indicator dilution, was 25 ± 4.3 ml/min/kg vented vs. 20 ± 4.3 intact in 7 dogs. Thoracic venting did not decrease blood pressures and flows during CPR, as would be predicted from the hypothesis that generalized intrathoracic pressure fluctuations are the dominant hemodynamic mechanism. The results are consistent with the classical notion that CPR works by compression of the heart between the sternum and the spine. This mechanism should not be discounted in future attempts to improve CPR.

Key words: blood flow cardiac pump, hemodynamic, intrathoracic pressure, mechanism, thoracic pump, vest CPR

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INTRODUCTION

Recently, mechanisms of blood flow during CPR have become the subject of considerable discussion [1-4]. Traditionally, it has been taught that external chest compression produces artificial circulation because the heart is squeezed between the sternum and the spine [5, 6]. However, the discovery of cough CPR [2, 7] and several techniques of flow augmentation by manipulation of intrathoracic pressure [8, 9] require that one consider at least two possible mechanisms for the generation of artificial circulation by external chest compression. The first is the "cardiac pump" mechanism in which the heart acts as a pump because it is compressed between the sternum and the spine. The second is the "thoracic pump" mechanism, first suggested by Waters [10] and Thompson et al. [11, 12], and elaborated in several recent articles [1-3]. According to the thoracic pump mechanism, global elevations of intrathoracic pressure, produced by cough or thoracic compression, are vented by arterial outflow to peripheral tissues. Direct compression of the heart is irrelevant for generating blood flow. Instead, intrathoracic pressure squeezes blood from the pulmonary vascular bed, through the heart, and into peripheral vascular beds--the heart acting only as a "passive conduit" [2]. The theoretical rationale for both mechanisms has been discussed in detail by one of the authors [3]. Which is the predominant mechanism of blood flow during standard, external CPR is a question of importance for the rational optimization of this life-saving clinical procedure.

In the spring and fall of 1979, by remarkable coincidence, three groups conducted the same study in experimental CPR independently and unbeknownst to each other. In subsequent informal discussions, the authors became aware of this fact and have decided to publish jointly. The separate groups at the University of Southern Maine, the University of Pittsburgh, and Purdue University were inspired in an identical fashion to investigate the effects of venting intrathoracic pressure through bilateral chest tubes during experimental CPR. If global elevations of intrathoracic pressure are the actual cause of blood flow, then venting of the pleural space via chest tubes, in the presence of partial atelectasis, should reduce markedly the arterial pressures and flows generated by chest compressions. On the other hand, if cardiac compression is responsible for blood flow, then venting of thoracic pressure during chest compressions should not have a profound effect. In each of these laboratories, therefore, the authors conducted quite similar experiments in which the blood pressures and flows generated during CPR in animals were measured during alternate periods, with and without venting of the expanded pleural space.

METHODS

Animal Preparation

Nineteen animals, including 7 domestic pigs (18-26 kg) anesthetized with sodium thiamylal (22 mg/kg ip); 5 mongrel dogs (10-22 kg) anesthetized with ketamine (titrated to effect); and 7 mongrel dogs anesthetized with sodium pentobarbital (30 mg/kg iv) served as subjects. The thoracic diameters of the pigs at the level of the sternum were: dorsal-ventral, 19-21 cm, left-right, 17-19 cm. The thoracic diameters of the dogs were dorsal-ventral, 14-21 cm, left-right, 11-13 cm. The ratios of dorsal-ventral to left-right chest diameters averaged 1.4. Endotracheal

intubation and monitoring of the ECG, arterial blood pressure, and central venous pressure were carried out in all animals. Arterial pressure was measured in the thoracic aorta (Maine and Pittsburgh) or in the axillary artery (Purdue). Mean blood pressures were derived electronically. In both pigs (Maine) and some of the dogs (Purdue), pleural pressure was recorded through a percutaneous, fluid filled catheter in a manner similar to that described by Downs [13]. Carotid artery blood flow was measured in both pigs and dogs, using either a Medicon model M-4001 in-line electromagnetic flowmeter (Maine) or a bubble flowmeter of the type described by Redding and Cozine [14] (Pittsburgh). In the other dogs, cardiac output was determined by the saline dilution method, specially adapted for the low flow conditions of CPR [15].

Experimental CPR

In all three groups of animals, ventricular fibrillation was induced by 60 Hz electrical stimulation of the heart. With the aid of a mechanical chest compressor and ventilator (Thumper®, Michigan Instruments, Grand Rapids, Ml), experimental CPR was begun either immediately or after a 2-min delay (Pittsburgh), according to standard American Heart Association Guidelines [16]: 60 ventral-dorsal chest compressions/min, 12 ventilations/min, with compression force sufficient to produce 3-6 cm depression of the sternum. The duration of compression was 50% of cycle time and the tidal volume was 10-15 ml/kg. In the Maine and Pittsburgh studies, ventilation was interposed after every 5th chest compression. In the Purdue study, ventilation was not synchronized with the compression cycle and was delivered randomly with respect to chest compression. In all studies, the force of chest compression was maintained at a constant value throughout the experiment.

Thoracic venting was accomplished by bilateral 5 mm internal diameter endotracheal-type tubes placed in the 6th interspace bilaterally (Maine); 3-5 mm internal diameter pediatric chest tubes, 2 on each side (Pittsburgh); or 6 mm internal diameter polyethylene tubes with multiple side-holes inserted through the 3rd and 6th inter-spaces bilaterally (Purdue). In all animals, the chest tubes were secured with purse string sutures to form a visually air tight seal. Venting of the thorax through these tubes allowed several hundred ml of air to enter the pleural space, producing partial atelectasis. However, adequate ventilation was maintained, despite the enlarged pleural space, as could be verified by measurements of tidal volume. Venting of the thorax was verified by exit of air and some fluid from the chest tubes during ventilation or chest compression and by decreasing pleural pressure fluctuations. Thoracic venting was reversed by clamping the chest tubes after hyperinflation of the lungs or by connection of the tubes to low suction (Pittsburgh).

In the Maine study, CPR was performed on pigs under each of two test conditions (with and without thoracic venting) for alternate 2-min periods, lasting a total of 4-6 min. In the Pittsburgh study, the test conditions were evaluated for alternate 10-min periods, lasting up to 2 h. In the Purdue study, repeated trials of fibrillation and CPR for 2 min, followed by defibrillation and recovery, were performed with thoracic venting and compared to both prior and subsequent control trials without thoracic venting.

RESULTS

An original graphic record from the Maine study (Fig. 1) typifies the results for individual animals in all three laboratories. Thoracic venting eliminated or greatly attenuated pulsatile changes in intrathoracic pressure without producing profound changes in arterial blood pressure or blood flow. Arterial blood pressure was much greater than pleural pressure during artificial systole prior to thoracic venting. Without venting pleural pressure during compression averaged 7 ± 2 mm Hg in seven trials in 5 pigs and 7 ± 1 mm Hg in 19 trials in dogs, much less than systolic arterial pressures during CPR. When the chest was vented, pleural pressure fluctuated around zero and the fluctuations were not synchronous with chest compressions.

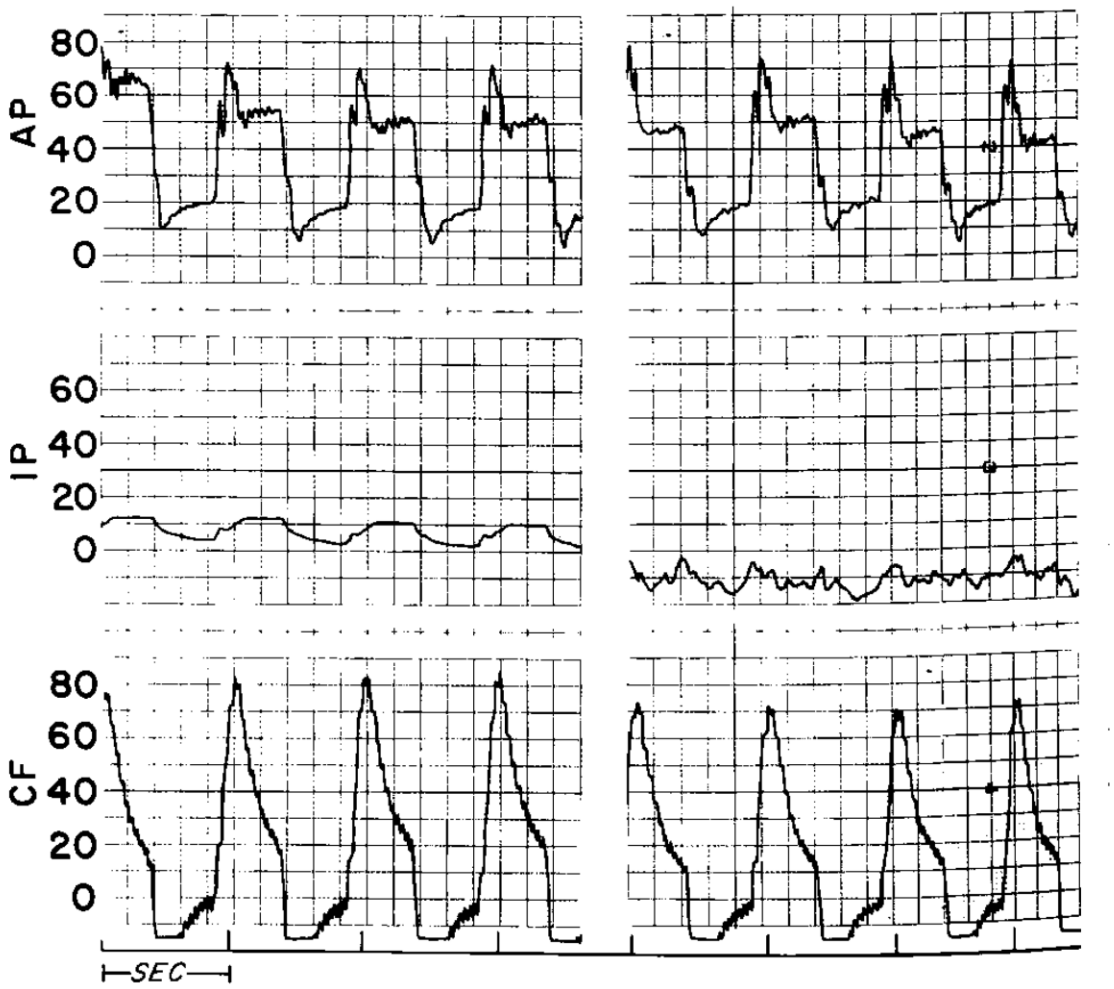

FIG. 1. Pulsatile arterial pressure (AP, mm Hg), intrapleural pressure (IP, mm Hg), and carotid artery blood flow (CF, ml/min) during experimental CPR in a 25.8 kg pig. Data recorded 30 sec before thoracic venting (left) and 30 sec after thoracic venting (right) are compared. Zero level for IP was established before CPR with thorax resealed after initial placement of chest tubes.

Figure 2 presents the arterial pressure data from all three laboratories. The vertically aligned points represent systolic and diastolic pressures. No significant effect of thoracic venting was observed in any of the experiments. In the dogs, the arteriovenous pressure difference (± SE) between the thoracic aorta and superior vena cava during thoracic venting averaged 16 ± 7 mm Hg (Pittsburgh) and 11 ± 3 mm Hg (Purdue), as compared to 16 ± 5 mm Hg (Pittsburgh) and 9 ± 3 mm Hg (Purdue) without thoracic venting. In the pigs, the arteriovenous pressure difference averaged 17 ± 3 mm Hg during venting, as compared to 19 ± 3 mm Hg when the thorax was intact. In all three studies, neither the arterial blood pressure nor the systemic perfusion pressure was affected by thoracic venting.

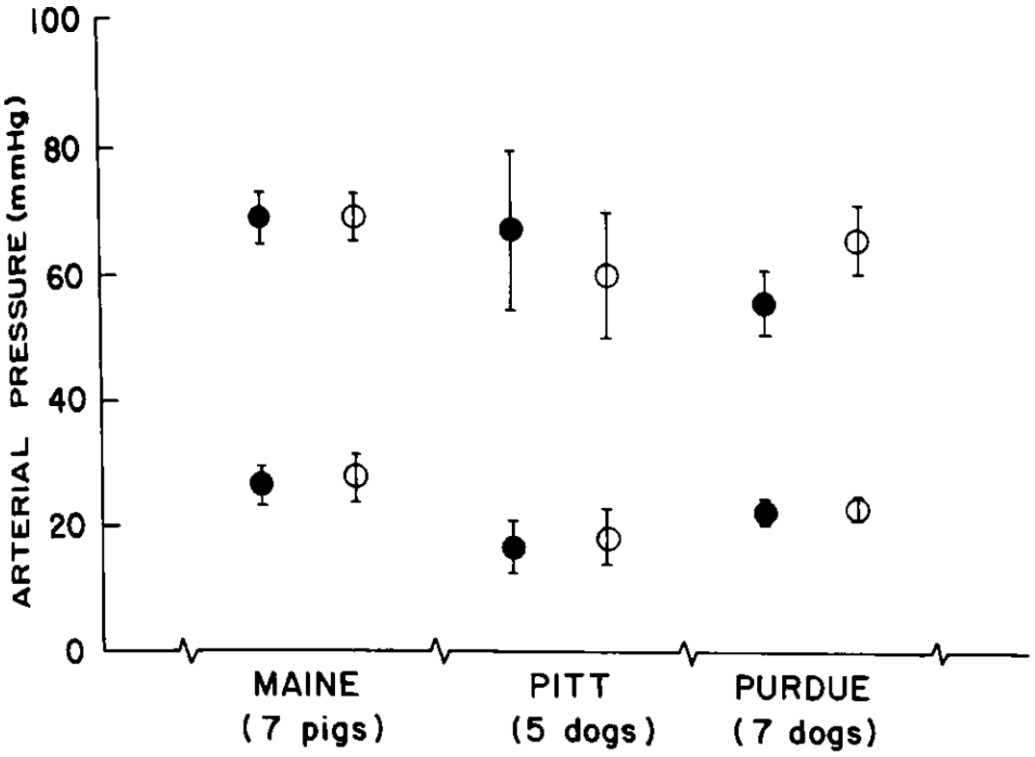

FIG. 2. Effect of thorax venting upon arterial pressures during experimental CPR. Solid symbols = pleural space evacuated; open symbols = pleural space vented. Average systolic and diastolic arterial pressures (± SE) for the animals at each laboratory are plotted.

Blood flow data for CPR with the thorax intact vs. vented are compared in Figure 3. Contrary to expectations based on the thoracic pump mechanism, creation of open pneumothorax did not cause blood flow during CPR to decrease.

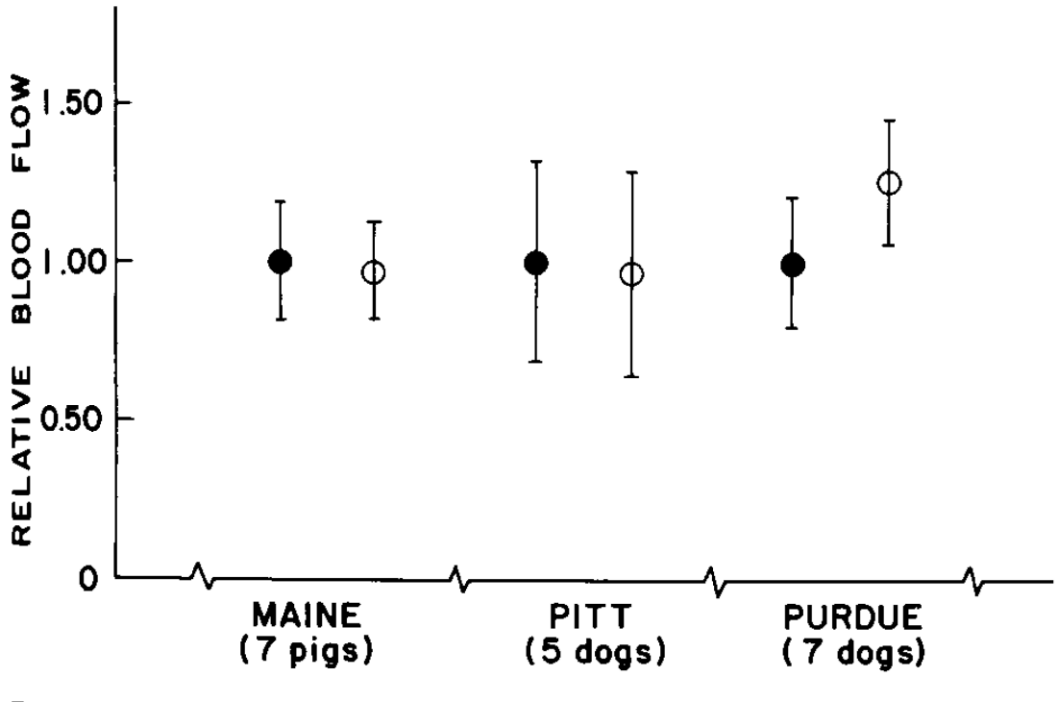

FIG. 3. Relative blood flow with intact thoracic wall and pleural space evacuated (solid symbols) compared to otherwise identical test conditions with thoracic venting (open symbols) in three similar experiments. Absolute values corresponding to 1.00 on the ordinate are 13.4 ml/ min, 36% of prefibrillation value for the Maine study; 11.2 ml/min, 23% of prefibrillation value for the Pittsburgh study; and 19.7 ml/min/kg, 18% of the prefibrillation value for the Purdue study. Mean relative flow values (± SE) are plotted.

DISCUSSION

Two groups have recently concluded from their studies that the thoracic pump mechanism is responsible for the majority of blood flow during standard CPR and that compressions of the heart is irrelevant in many cases [1, 2]. In support of this thesis, Wilder et al. [17], Harris et al. [18], and more recently Chandra et al. [8] have shown that arterial pressure and/or carotid artery blood flow is augmented when chest compression and ventilation are simultaneously applied. However, if intrathoracic pressure fluctuations are, indeed, the principal driving force propelling blood from the lungs, through the heart, and into the periphery, then abolition of those fluctuations should reduce markedly vascular pressures and flows.

We have performed this critical experiment independently at three laboratories, in two species, using a variety of techniques to measure blood flow. The results appear entirely consistent and definitive. Open pneumothorax did not cause reduction of blood flow during CPR in the animals studied. It is difficult to reconcile these observations with the hypothesis that fluctuations of global intrathoracic pressure are the principal cause of blood flow during CPR.

This is not to say, however, that the thoracic pump mechanism is fictitious or unimportant. During cough CPR [2, 7] and modified CPR with simultaneous compression and ventilation [8], the airway is closed or pressurized during chest compression and air cannot escape. High generalized intrathoracic pressures are then possible, and it is reasonable to expect significant venting of the thoracic pressure by outflow of blood to the periphery. However, during standard CPR in which the airway is open during chest compression, venting of intrathoracic pressure via the trachea may greatly attenuate the effectiveness of the thoracic pump mechanism.

The authors' own studies of standard CPR indicate a minimal degree of thoracic pumping, because relief of intrathoracic pressure caused little diminution in the artificial circulation developed. However, these results are not inconsistent with the classical concept that the heart is effectively compressed and functions as a pump during CPR. Action of the heart as a pump was confirmed in these studies by measurement of positive blood pressure differences across the heart, i.e., between the intrathoracic portions of the aorta and vena cavae, and by direct observations of the mediastinum through a wide-open thoracostomy at the end of several of the dog experiments (Pittsburgh). Previously, one of the authors has visualized compression of the heart in a similar animal model of CPR, by angiographic techniques [19].

In an effort to reconcile the apparently conflicting results of this study with those of Rudikoff et al.[1], who have marshaled evidence that direct cardiac compressions is unlikely, the authors would speculate that there is a significant difference in the size of the animal models. In the large (20-45 kg) deep-chested subjects, such as German shepherd dogs used by the Hopkins group [1], the heart is probably not compressed, and thoracic pumping is likely to be the dominant hemodynamic mechanism. These animals have thoracic geometries different from the animals used in the present studies. The ventral-dorsal chest geometry of such large dogs models that of more barrel-chested human victims. In the present studies the experimental animals had ventral-dorsal chest diameters less than most 20-45 kg dogs. The ventral-dorsal chest geometry of small dogs and small pigs models that of adolescents and thinner chested human adults. The authors believe direct cardiac compression occurs in such narrower chested victims. Indeed, Rudikoff et al.[1] have found indirect evidence of cardiac compression in 11 of 37 patients in whom radial artery pressure during resuscitation was recorded, as well as in one 12-kg dog studied in their laboratory.

Taken together, the recent reports of Nieman et al. [2], Rudikoff et al. [1], and the authors' own observations reported here indicate two plausible mechanisms for blood flow during CPR. One or the other mechanism may predominate in different experimental situations or in different individual victims of cardiopulmonary arrest. Which is the predominant mechanism in a given subject probably depends upon several factors, including the ventral-dorsal chest diameter, the presence or absence of cardiac enlargement, the stiffness or resiliency of the anterior chest, and

whether or not ventilations are applied simultaneously with chest compression. Accordingly, the authors believe future improvements in CPR technique should be applicable to both cardiac pumping and thoracic pumping. These may include, among others, fluid loading, venoconstrictors, negative diastolic airway pressure, abdominal binding, and leg elevation [3]. Such interventions, which theoretically improve the blood flow generated by both mechanisms, are the most fruitful objects of further research.

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REFERENCES

l. Rudikoff MT, Maughan WL, Effron M, et al: Mechanisms of blood flow during cardiopulmonary resuscitation. Circulation 1980; 61:345

2. Niemann JT, Rosborough JP, Hausknecht M, et al: Cough CPR--documentation of systemic perfusion in man and in experimental model--a "window" to the mechanism of blood flow in external CPR. Crit Care Med 1980; 8:141

3. Babbs CF: New versus old theories of blood flow during cardiopulmonary resuscitation. Crit Care Med 1980; 8: 191

4. Weisfeldt ML, Rudikoff MT, Cohen J, et al: Movement of blood through manipulation of intrathoracic pressure. Med Instrument (Abstr). 1980; 14:59

5. Kouwenhoven WB, Jude JR, Knickerbocker GG: Closed-chest cardiac massage. JAMA 1960; 173:1064

6. Council National Academy of Sciences-National Research Cardiopulmonary Resuscitation-Conference Proceedings ISBN #0-309-01494-8 Division of Medical Sciences, National Academy of Sciences, Washington, DC, 1967

7. Criley JM, Blaufuss AH, Kissel GL: Cough-induced cardiac compression--a self-administered form of cardiopulmonary resuscitation. JAMA 1976; 236:1246

8. Chandra N, Rudikoff M, Weisfeldt ML: Simultaneous chest compression and ventilation at high airway pressure during cardiopulmonary resuscitation (CPR) in man. Circulation 1978; 58: 203

9. Chandra N, Cohen JM, Tsitlik J, et al: Negative airway pressure between compression augments carotid flow during CPR. Circulation 1979; 60: 169

10. Waters RM: Resuscitation-artificial circulation by means of intermittent high pressure chest inflation with oxygen (a preliminary report). Anesth Analg Bull 1921; 15:10

11. Thompson SA, Quimby EM, Smith BC: The effect of pulmonary resuscitative procedures upon the circulation as demonstrated by the use of radioactive sodium. Surg Gynecol Obstet 1946; 83:387

12. Thompson SA, Rockey EE: The effect of mechanical artificial respiration upon the maintenance of the circulation. Surg Gynecol Obstet 1947; 84:1059

13. Downs JB: A technique for direct measurement of intrapleural pressure. Crit Care Med 1976; 4:207

14. Redding JS, Cozine RA: A comparison of open chest and closed chest cardiac massage in dogs. Anesthesiology 1961; 22:280

15. Geddes LA, CF Babbs: A new technique for the measurement of cardiac output during cardiopulmonary resuscitation. Crit Care Med 1980; 8:131

16. Gordon A: Standards for Cardiopulmonary Resuscitation (CPR) and Emergency Cardiac Care (ECC). JAMA 1974; 227(suppl):833

17. Wilder RJ, Weir D, Rush BF, et al: Methods of coordinating ventilation and closed chest cardiac massage in the dog. Surgery 1963; 53:186

18. Harris LC, Kirimli B, Safar P: Augmentation of artificial circulation during cardiopulmonary resuscitation. Anesthesiology 1967; 28:730

19. Babbs CF: Cardiac angiography during CPR. Crit Care Med 1980; 8:189