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Sandra H. Ralston

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

Mark J. Niebauer

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Cardiopulmonary Resuscitation with Interposed Abdominal Compression in Dogs

Sandra H. Ralston, RN, BS*, Charles F. Babbs, MD, PhD#, and Mark J. Niebauer MS*

From the

Biomedical Engineering Center, and the

Department of Veterinary Physiology and Pharmacology,

Purdue University,

West Lafayette, Indiana 47907

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Address for reprints, correspondence, and proofs:

Charles F. Babbs MD, PhD Biomedical Engineering Center, Potter Building, Purdue University, West Lafayette, IN 47907

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* Graduate student in physiology and pharmacology

Associate Research Scholar, Biomedical Engineering Center

ABSTRACT

This study was conducted to evaluate the hemodynamic effectiveness of a new modification of cardiopulmonary resuscitation (CPR), termed interposed abdominal compression - CPR (IAC-CPR). IAC-CPR utilizes all the steps of standard CPR with the addition of abdominal compressions interposed during the release phase of chest compression. Ventricular fibrillation was induced electrically in 10 anesthetized dogs, and either IAC-CPR or standard CPR was initiated while arterial and venous blood pressures and cardiac output were monitored. The two CPR methods were alternated every three minutes over a period of thirty minutes. The addition of interposed abdominal compressions to standard CPR improved arterial pressures and perfusion in 10/10 dogs. Brachial arterial blood pressure averaged 87/32 mmHg during IAC-CPR vs. 58/16 mmHg during standard CPR. Cardiac output (±S.E.) averaged 24.2 ±5.7 ml/min/kg during IAC-CPR vs. 13.8 ±2.6 ml/min/kg during standard CPR. IAC-CPR requires no extra mechanical equipment, and, if proven effective in human trials, may improve resuscitation success in the field and in the hospital.

Key (indexing) terms: CPR, resuscitation, sudden death, ventricular fibrillation, emergency cardiac care.

INTRODUCTION

Recently several modifications of cardiopulmonary resuscitation (CPR) that generate improved blood flow compared to standard CPR[1], have been discovered in the laboratory and tested on a limited basis in the clinic. Improvements in blood pressures and blood flows during experimental CPR have, for example, been reported with increased duration of chest compression[2,3], with simultaneous chest compression and ventilation at high airway pressure[4,5,6], with negative diastolic airway pressure[7], and with abdominal binding[8]. These studies leave little doubt that improved blood flow is possible during CPR, and they provide valuable insights into mechanisms that generate blood flow during CPR[9]. However, because special mechanical equipment is necessary, techniques such as simultaneous chest compression and ventilation at high airway pressure or application of negative diastolic airway pressure constitute advanced life support techniques, not applicable to field resuscitation by basic rescuers or to initial attempts at resuscitation in the hospital. Manual versions of CPR with simultaneous compression and ventilation have been developed and tested by Redding[10] and by Gordon[11] and their coworkers but were not recommended as significantly better than standard CPR.

This report describes animal studies of a new form of modified CPR which seems applicable to basic life support. It can be performed by two or three rescuers with no equipment other than

their bare hands. It includes all the procedures of standard CPR and so constitutes an evolution rather than a revolution in technique. We have termed this modification IAC-CPR to denote interposed abdominal compression.

IAC-CPR: This technique involves standard ventilation and chest compression with the addition of abdominal compressions interposed between chest compressions. The method was discovered by one of us (SHR) serendipitously during a difficult resuscitation in the animal laboratory. CPR is performed exactly as recommended in current American Heart Association standards[1], and in addition the abdomen is compressed alternately or reciprocally as chest compression is released. This technique of abdominal counterpulsation necessarily would require two or three rescuers, as illustrated in Figure 1.

Having observed significantly improved arterial blood pressure during the chance discovery of IAC-CPR as compared to standard CPR, we conducted the following research to determine if arterial pressures and cardiac output were consistently improved by the addition of alternate abdominal compressions to the mechanics of standard CPR.

METHODS

To compare blood flow generated by IAC-CPR with that generated by standard CPR, we measured cardiac output during alternate three-minute trials of the two techniques in animals during electrically induced ventricular fibrillation, using a modified indicator dilution modified technique adapted to the low flow conditions of CPR. We compared IAC-CPR and standard CPR in both large and small mongrel dogs, since the size of animals studied may substantially influence the outcome of CPR experiments[6].

Ten mongrel dogs were selected for the study. The five large dogs weighed 15-26 kg (mean 18.9 kg), had dorsal-ventral chest diameters ranging from 21-25 cm (mean 23 cm) at the level of the heart, and chest circumferences of 57-61 cm (mean 60 cm). The five small dogs weighed 9-13 kg (mean 11.6 kg), had dorsal-ventral chest diameters ranging from 16-21 cm (mean 19 cm) at the level of the heart, and chest circumferences of 45-53 cm (mean 49 cm).

The dogs had free access to food and water prior to anesthesia. Each animal was anesthetized with pentobarbital sodium (30 mg/kg i.v.). The trachea was intubated with the largest possible cuffed tracheal tube. The following catheters were inserted: (1) a pigtail catheter was advanced into the left ventricle via a femoral artery for injection of indicator to measure cardiac output; (2) a 40 cm long, 0.1 cm internal diameter

catheter was advanced to the thoracic aorta and attached to a motor-driven syringe for withdrawal of blood during inscription of dilution curves; (3) a catheter to monitor arterial pressure was advanced 5-10 cm into the right brachial artery; (4) a catheter to monitor central venous pressure was advanced via the left femoral vein into the right atrium. The catheters employed for arterial and venous pressure monitoring were connected to matched Statham pressure transducers. Heparin (1 mg/kg i.v.) was given to retard clot formation in the catheters, to permit reinfusion of blood withdrawn during inscription of dilution curves, and to diminish intravascular coagulation during CPR.

The animal was placed in dorsal recumbency on a V-shaped board with the limbs securely tied to the board to prevent lateral motion of the chest during CPR. A Thumper mechanical resuscitator (Michigan Instruments, Inc., Grand Rapids, Michigan) was used for chest compression and ventilation. Subcutaneous electrodes for recording the electrocardiogram (Lead II) were secured in place, and wire mesh electrodes for sternal-to-back defibrillation were applied to the shaved skin of these regions with electrolytic gel. The V-shaped, 20 x 20 cm back electrode for defibrillation conformed to the animal board, and the wire mesh of the sternal electrode was molded to the chest compression pad of the Thumper. The chest compression pad was rectangular in shape and 6 X 10 cm in dimensions.

The pad used for abdominal compression was a standard 12 cm width blood pressure cuff folded to rectangular dimensions of 12 X 15 cm and inflated with of air to a thickness of 3 cm. The bladder of the cuff was attached via the filling hose to an aneroid manometer and to a linear core pressure transducer in order to monitor pressure applied to the abdomen. IAC-CPR was performed by manual compression of the mid abdomen with this inflated pad in a way to generate pressure pulses of 120-150 mmHg. The duty cycle of abdominal compression was complimentary to that of chest compression i.e., 50% of cycle time (0.5 sec abdominal compression duration). The position of the hands for abdominal compression was similar to that used in basic CPR for manual chest compression except that the fingers were spread to provide a larger surface area of compression approximately equal to that of the flattened blood pressure cuff.

Physiologic Monitoring

A five-channel graphic record was inscribed using a Physiograph direct-inking recorder (Narco Bio-Systems, Houston, Texas). Channels 1, 2, 3, and 4, displayed the electrocardiogram, arterial blood pressure, venous blood pressure, and abdominal compression pressure, respectively. Pressure channels were calibrated and their linearity confirmed using a mercury manometer.

Channel 5 of the graphic record displayed indicator dilution curves for measurement of cardiac output by the saline-

conductivity method[12], specially modified for the low flow conditions of CPR[13]. This method employs 5% NaCl solution as the indicator and a calibrated, flow-through conductivity cell as the detector. Its accuracy has been confirmed by comparison with the direct Fick method under conditions of CPR[14]. Two-ml aliquots of 5% saline indicator were injected forcibly into the left ventricle and blood samples were withdrawn through the detector via the catheter placed in the thoracic aorta. This injection--sampling configuration permits mixing of indicator in blood during CPR adequate for accurate measurements of cardiac output[13].

Experimental CPR

After control measurements of blood pressures and cardiac output were obtained, a single episode of ventricular fibrillation was produced by 60 Hz electrical stimulation of the left ventricular endocardium. A fine, 0.1 mm, stainless steel wire threaded through the lumen of the left ventricular catheter carried electric current to the heart for this purpose. Immediately after electrocardiographic confirmation of fibrillation, ventilation and chest compression were initiated using the Thumper driven with 100% oxygen at 60 psi. This device provided standard CPR continuously throughout the experiment.

The technique of abdominal compression was added to the CPR provided by the Thumper during alternate 3 min intervals. Five

3-min trials of IAC-CPR and five 3-min trials of standard CPR were evaluated alternately in the same animal during one continuous episode of ventricular fibrillation. In half of the dogs IAC-CPR was begun first and in half of the dogs standard CPR was begun first. After a 30 sec recording of pulsatile blood pressures for a given mode of CPR, dilution curves were obtained. Then the mode of CPR was changed and the process repeated. In this sense, each animal served as its own control.

During both standard CPR and IAC-CPR, the ventilation pressure was 20 cm of water, the ventilation duration was 0.5 sec, and ventilations were interposed after every 5th chest compression. The chest compression force, 40-80 lbs for small dogs and 60-120 lbs for large dogs, was selected to produce approximately equal sternal displacement as a percentage of dorsal-ventral chest diameter (mean 25%) in the two groups of dogs. In each dog the force of chest compressions was maintained the same for both standard and IAC-CPR. In both standard and IAC-CPR the compression rate was 60/min, and the duty cycle of compression was 50 percent of cycle time (compression duration = 0.5 sec).

Post-resuscitation protocol

After the 10 consecutive trials of standard and experimental CPR electrical shock was applied to defibrillate the ventricles. If necessary, intracardiac epinephrine was given via the left ventricular catheter to promote recovery of the circulation.

After recovery of the circulation the animal was monitored for 30 min to determine if any lethal complications of the experiment had occurred. Then the animal was sacrificed by ventricular fibrillation without resuscitative measures and a thorough gross post-mortem examination performed. Special attention was given to identification of possible trauma to the abdominal viscera as a result of IAC-CPR.

Data analysis

To compare effects of experimental CPR mean cardiac output during the 5 trials of standard CPR and the 5 trials of IAC-CPR was calculated for each animal. Student's t test for paired data was used to test to test the null hypothesis that these mean values of cardiac output per kilogram were the same during IAC-CPR and standard CPR in the population of 10 dogs. A similar analysis was performed for measurements of brachial arterial and and venous blood pressures and of the arteriovenous pressure difference. If necessary, a square root transformation was performed on the data before calculation of Student's t statistics, to satisfy the assumption of approximate normality of the sampling distribution required for the t-test[15,16].

RESULTS

Cardiac Output

Cardiac output generated by IAC-CPR was greater than that generated by standard CPR in every animal (Figure 2). In Figure 2 each data point represents the mean of 5 measurements in a single dog and each symbol type represents a given animal. The paired differences in mean cardiac output for the 10 dogs are significantly different from zero ($p<0.005$) $t=4.79$ $df=9$). If the mean cardiac output during standard CPR in each dog is assigned a value of 100 percent, the corresponding values during IAC-CPR ranged from 122 to 372 percent. Within a given animal the coefficient of variation (S.D./mean) of the five cardiac output measurements ranged from 11 to 33 percent (mean 21 percent) during standard CPR and from 4 to 34 percent (mean 18 percent) during IAC-CPR.

Blood Pressures

Maximal (systolic) and minimal (diastolic) arterial pressures were higher during IAC-CPR than during standard CPR in all 10 dogs (Figures 3 and 4). The central diastolic arteriovenous pressure gradient, which may be critical for coronary perfusion, was improved during IAC-CPR in 8 of 10 dogs (Figure 5). The sets of paired differences in systolic and diastolic arterial pressure and in the arteriovenous pressure difference for the 10 dogs are

each significantly greater than zero ($p < .01$).

Other observations

Abdominal counterpulsation did not cause obvious regurgitation of gastric contents in any of the 10 dogs, even though the animals had not fasted prior to the experiment. After defibrillation 7 of the 10 dogs survived for 30 min. No significant gross trauma to intraabdominal organs was seen at post-mortem examination. Serosanguinous abdominal fluid was observed in three of the ten animals and intramesenteric hemorrhages were observed in one animal, findings not considered serious in these heparinized animals. Liver laceration never occurred.

DISCUSSION

The addition of interposed abdominal compression to standard CPR greatly improves blood pressure and blood flow in both the large dog and the small dog model of cardiopulmonary arrest. We have speculated that the thoracic pump mechanism for generating blood flow is more important in large animals, while the traditional cardiac pump mechanism is more important in smaller animals[9,6]. If so, one can conclude that IAC-CPR is effective in improving hemodynamics caused by either mechanism. The increase in arterial pressure during IAC-CPR is clearly not an artifactual transmission of pressure from the abdomen to the thorax, since since both the diastolic arteriovenous pressure difference and the total blood flow improve.

The improvement in arterial pressure during the diastolic phase (release of chest compression) and in central arteriovenous pressure difference during IAC-CPR is significant in that it is likely to enhance coronary perfusion. Coronary flow during standard CPR is reduced at least in proportion to cardiac output[17] and perhaps even more[18,19] but is essential for return of cardiac function and survival[20]. Quite possibly IAC-CPR offers an especially effective means of increasing coronary flow both, by improving total flow and by favorably altering the distribution of aortic run-off during chest recoil.

We hypothesize that the hemodynamic effects of interposed abdominal compression include pump priming and aortic counterpulsation. Abdominal compression, like atrial contraction in the normally beating heart, may encourage blood into the main pumping chamber, which during CPR may include the thorax as a whole, the cardiac ventricles, or both[9]. Moreover, diastolic abdominal pressurization must, to some degree, improve the distribution of blood flow, favoring the brain and the heart as compared to kidneys, intestines, and lower extremities. To the extent that aortic counterpressure occurs, the effect is similar to surgical cross-clamping of the aorta in an extreme hemodynamic emergency. However, total cardiac output is dramatically increased by alternate abdominal compression, suggesting an equally important effect of IAC-CPR on the abdominal venous vasculature.

Previously, Harris and associates[21] found that continuous manual compression of the abdomen increased carotid flow by 2/3, a degree of flow augmentation similar to that in the present study. However, these authors did not recommend manual compression of the upper abdomen during CPR because lacerations of the liver were noted in 2 of 6 dogs. In 1971 Redding demonstrated improved carotid arterial flow and survival in experimental CPR with continuous abdominal compression by a blood pressure cuff secured around the mid-abdomen[22], while observing no greater incidence of liver damage during CPR with continuous abdominal binding than in similarly resuscitated animals without abdominal binding. Recently Bircher, Safar, and Stewart reported a study

of experimental CPR in dogs in which a pressure suit was continuously inflated around the legs and abdomen[23]. They found "no major lacerations of the liver" in 12 dogs receiving this treatment, which did increase arterial pressure and carotid flow at least transiently. Rosborough and coworkers have reported that synchronous abdominal compression and lung inflation can produce effective artificial cough-CPR in dogs with no evidence of visceral trauma[24].

We suggest that the small but significant incidence of of liver laceration with continuous abdominal binding is due to entrapment of the liver by the rib cage as the chest in compressed. However, during interposed as opposed to continuous abdominal compression, the liver is allowed to recede at the time the chest is compressed, so that entrapment and laceration of the liver is less likely. We have observed such back-and-forth motion of the liver and diaphragm fluoroscopically during IAC-CPR in two dogs, using techniques we have previously described[25]. Although it is certainly possible that excessively rough or vigorous abdominal compression could traumatize the liver or spleen, we believe that central abdominal compression over a large area with 120 - 150 mmHg pressure, which is adequate to augment perfusion, is much less than that required to produce blunt trauma.

Abdominal counterpressure during CPR did not cause regurgitation in the animals in this study, but it is fitting to mention

the possibility of provoking regurgitation and aspiration by IAC-CPR. In our animals a tracheal tube was securely in place, and gastric insufflation did not occur. Gastric distension is a common sequela of mouth-to-mouth ventilation in humans[26], and abdominal pressure may induce vomiting after the stomach is distended with air[1]. However, one may speculate that if the IAC technique were used consistently from the beginning of resuscitation, gastric distension might be entirely prevented by the abdominal counterpressure. In the technique described in this study, abdominal pressure was applied and maintained throughout ventilation, in exact counterpoint to the rhythm of chest compression. Quite likely this technique would prevent passage of air into the stomach during mouth-to-mouth rescue breathing in man. The most probable situation in which interposed abdominal compressions might induce regurgitation would be if the technique were added after a period of conventional CPR - as might occur after others come to the aid of a lone rescuer. Since there are no good animal models for mouth-to-mouth ventilation, this issue will have to be settled by clinical experience.

In summary, the addition of intermittent abdominal compression to standard CPR appears to be a simple, safe, and effective means of improving perfusion during initial resuscitative efforts. The technique appears to be applicable to field CPR by basic rescuers and emergency medical personnel. It requires no extra mechanical equipment, and, if proven effective in human trials, could be easily incorporated into existing training

programs for lay rescuers and hospital personnel.

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Figure legends

Figure 1. Artist's conception of basic rescuers performing IAC-CPR. For clarity, both rescuers are shown on the same side of the victim. (A) With two rescuers the first compresses the chest and ventilates while the second compresses the abdomen. (B) With three rescuers ventilation, chest compression, and abdominal compression are each performed by a single individual.

Figure 2. Paired observations of cardiac output during standard CPR (STD) and CPR with interposed abdominal compressions (IAC) in 10 dogs. Each data point represents the mean of 5 trials in the same animal.

Figure 3. Paired observations of systolic brachial arterial pressure during standard CPR (STD) and CPR with interposed abdominal compressions (IAC) in 10 dogs. Each data point represents the mean of 5 trials in the same animal.

Figure 4. Paired observations of diastolic brachial arterial pressure during standard CPR (STD) and CPR with interposed abdominal compressions (IAC) in 10 dogs. Each data point represents the mean of 5 trials in the same animal.

Figure 5. Paired observations of central arteriovenous pressure difference during standard CPR (STD) and CPR with interposed

abdominal compressions (IAC) in 10 dogs. Each data point represents the mean of 5 trials in the same animal.

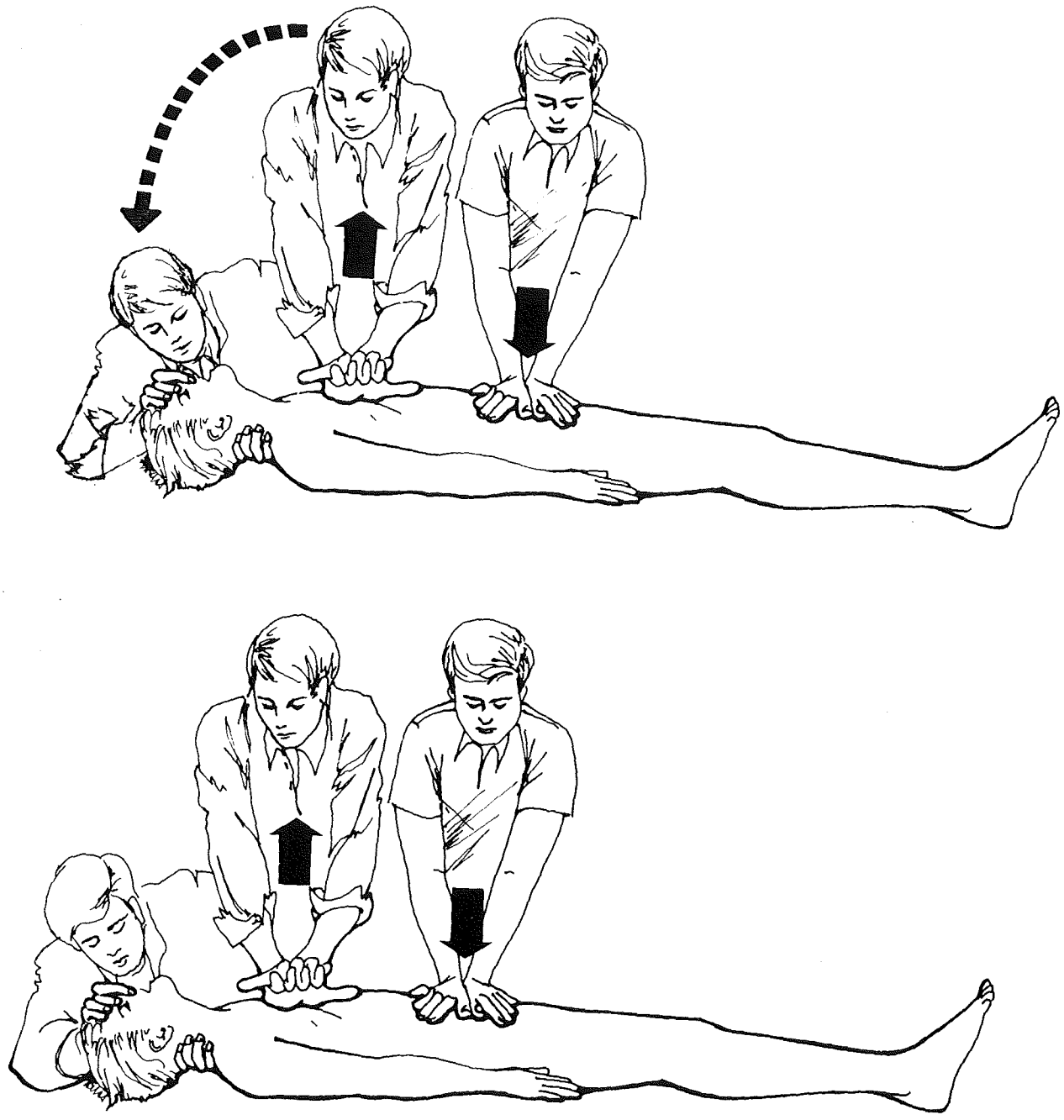

Fig. 1

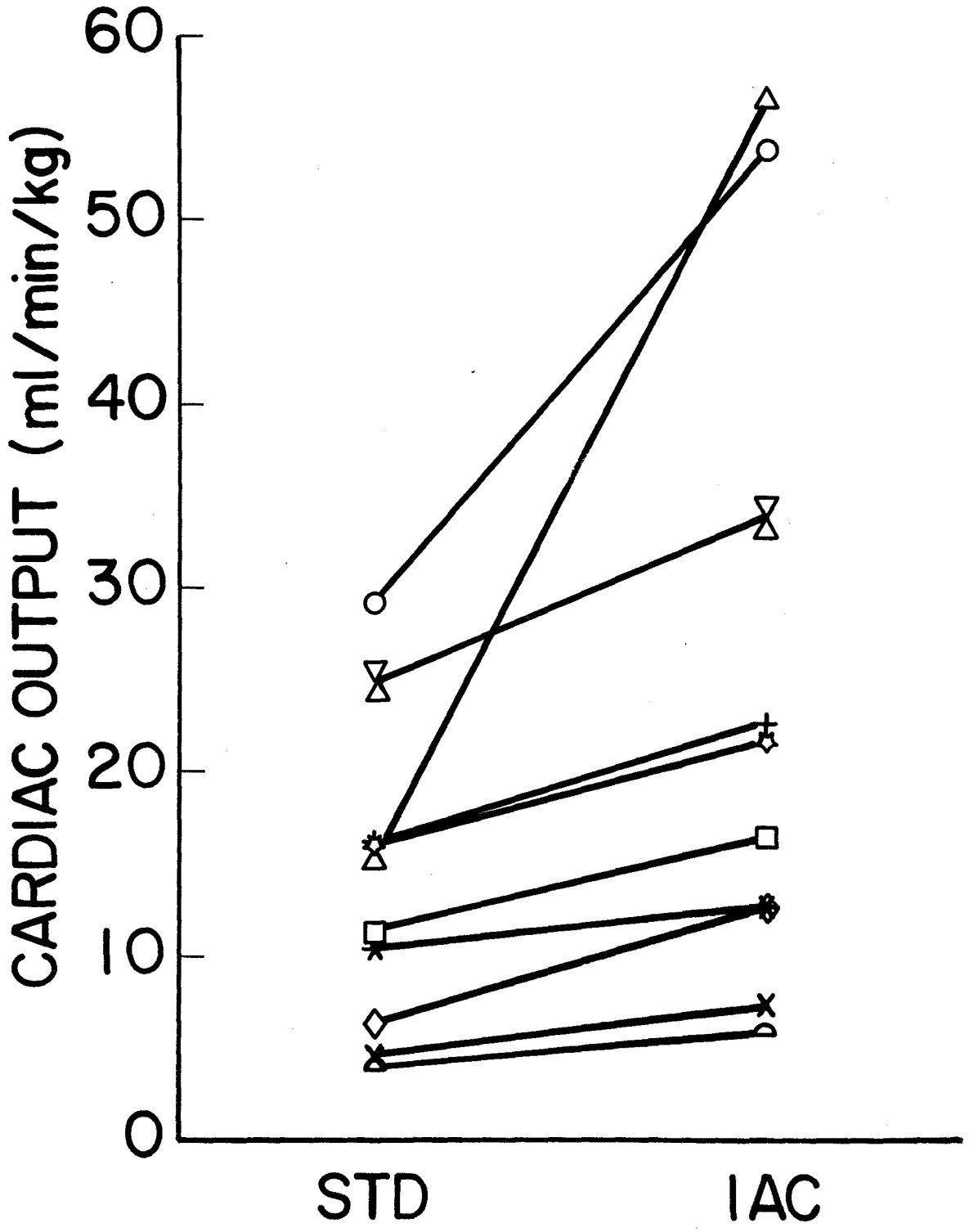

Fig. 2

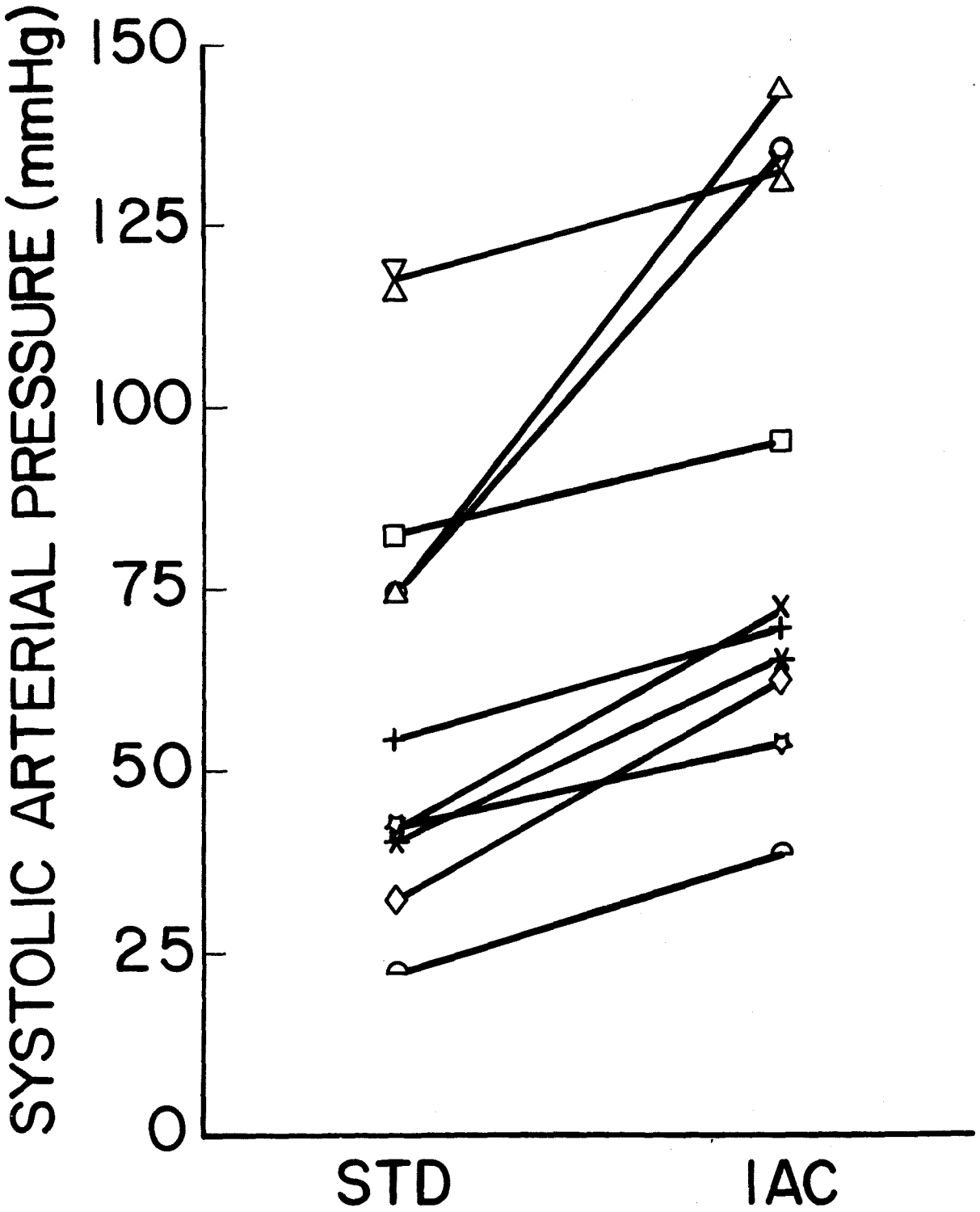
SYSTOLIC ARTERIAL PRESSURE (mmHg)
150
125
100
75
50
25
0
STD
IAC

Fig. 3

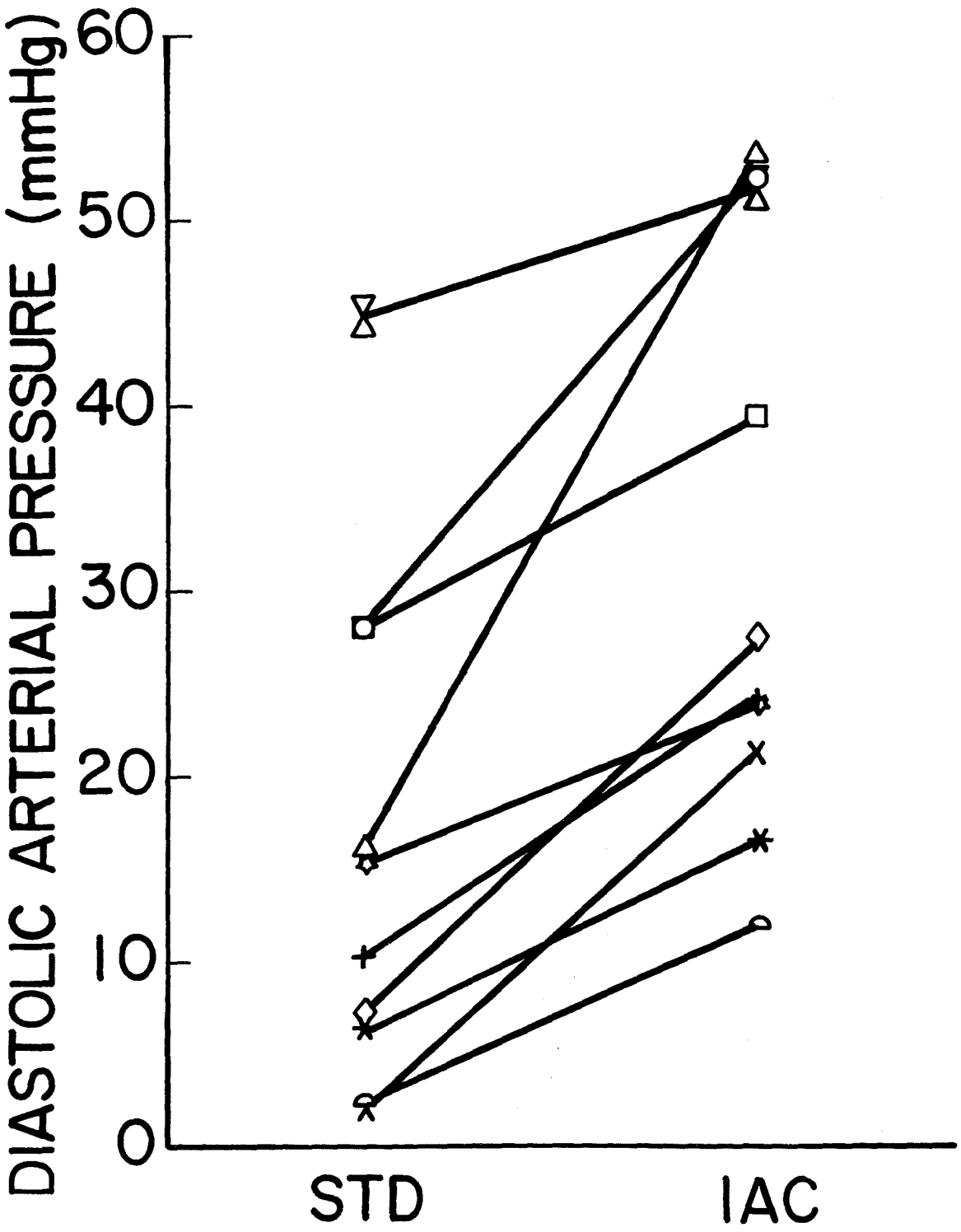

Fig. 4

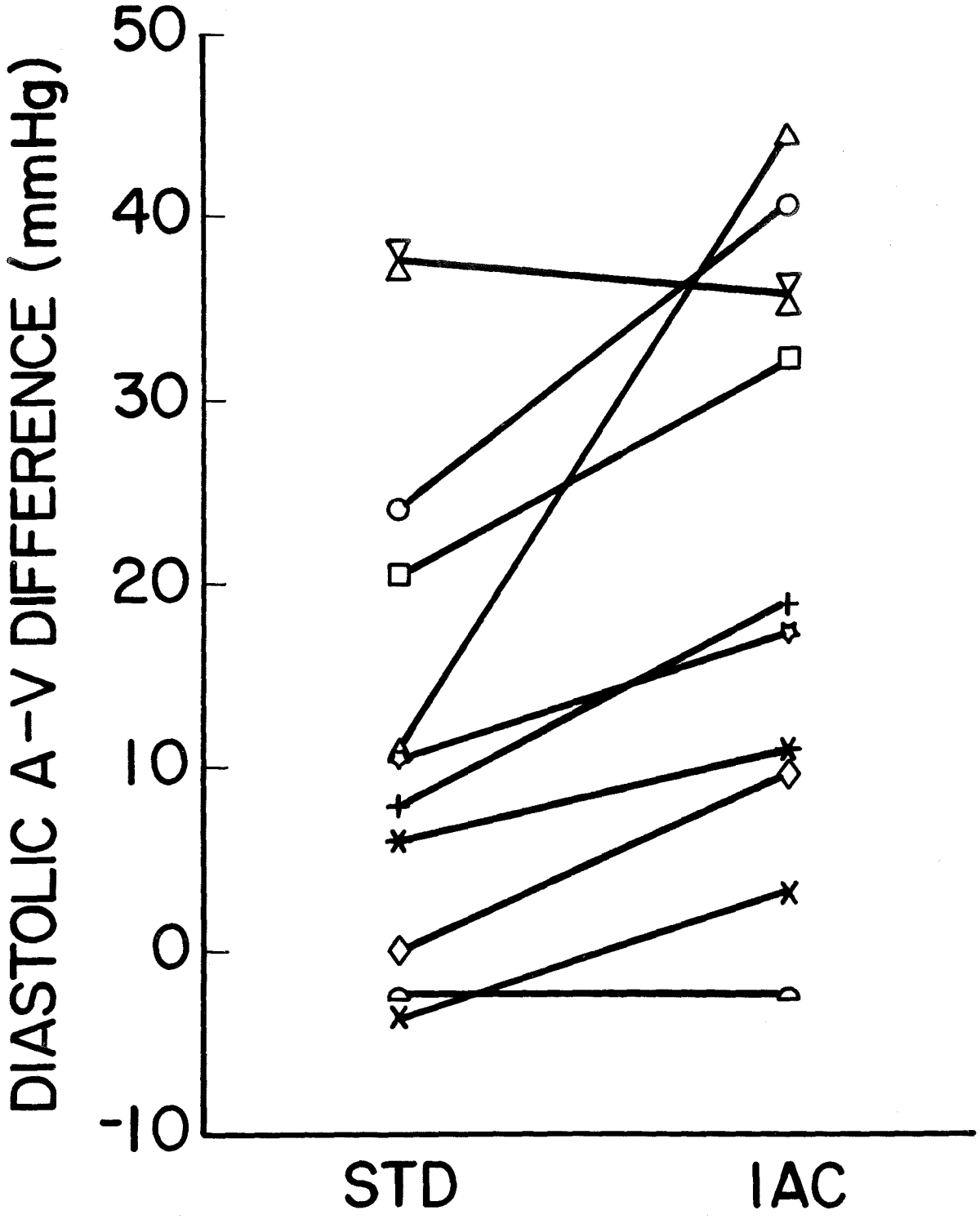

Fig. 5