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Mechanical Ventilation May Not Be Essential for Initial Cardiopulmonary Resuscitation*

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Background: **In a rodent model of cardiac arrest and resuscitation in which the inspired gas mixture was enriched with oxygen, resuscitability and survival were unaffected by positive pressure ventilation. In the present study, in a larger animal model, tidal volumes generated during precordial compression and with spontaneous gasping were quantitated.**

Methods: **Domestic pigs with an average weight of 34 kg were anesthetized with pentobarbital. Ventricular fibrillation (VF) was induced electrically. Precordial compression was begun after 4 min of untreated VF. Each of 22 animals received one of two interventions in conjunction with precordial compression: positive pressure ventilation with oxygen or oxygen supplied at the port of a tracheal tube at ambient pressure. After 8 min of precordial compression, defibrillation was attempted.**

Results: **Only very moderate increases in arterial P_{CO_2} were documented during cardiopulmonary resuscitation in the absence of mechanical ventilation but arterial oxygen tension was consistently in excess of 100 mm Hg. Cardiac resuscitability and 48-h survival were approximately the same in animals maintained on inspired oxygen whether or not they were mechanically ventilated (7/11 or 8/11). In the absence of mechanical ventilation, precordial compression and spontaneous gasping yielded minute volumes that exceeded 5 L.**

Conclusion: **Positive pressure mechanical ventilation did not improve resuscitability or postresuscitation outcome in this porcine model of cardiac arrest.**

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CPP=coronary perfusion pressure; CPR=cardiopulmonary resuscitation; VF=ventricular fibrillation

Key words: cardiopulmonary resuscitation; gasping; oxygen; pig; positive pressure ventilation; precordial compression; ventricular fibrillation

The ABCs of basic life support during cardiopulmonary resuscitation (CPR) currently provide for the establishment of a patent airway ("A") and intermittent positive pressure ventilation, preferably with oxygen enriched inspired air ("B"). These were to be immediately followed by precordial compression ("C").[1-4] However, both experimental and clinical evidence for this ordering of priorities is primarily by consensus rather than on the basis of objective outcome measurements.

Cardiac output and therefore pulmonary blood flow during CPR is typically less than one third of normal.[5-7] Consequently, an optimal ventilation/perfusion ratio would call for correspondingly lesser alveolar ventilation. Chest compression of itself and the resulting recoil of the chest cage also provide for pulmonary ventilation.[2,8,9] More recently, studies from our laboratory have demonstrated that the spontaneous gasping during experimental cardiac arrest of itself augments pulmonary gas exchange[10] and improves the outcome of CPR.[11]

Endotracheal intubation followed by positive pressure ventilation with an FI_{O_2} of 1.0 has been the "gold standard" for cardiac resuscitation by medical and paramedical rescuers. However, there is evidence that in settings of primary cardiac arrest and "sudden death" due to a dysrhythmic event, a delay in defibrillation and chest compression pending completion of tracheal intubation and mechanical ventilation may in fact compromise cardiac resuscitability and postresuscitation survival. This study was therefore undertaken to define the effects of mechanical ventilation on outcome both in terms of resuscitability and postresuscitation survival. Since the airway was intubated in each of the animals, the benefit or detriment attributable to the endotracheal airway, independently of mechanical ventilation, was deferred for separate investigation.

Methods

All animals received humane care in compliance with the Principles of Laboratory Animal Care formulated by the National Society for Medical Research and the Guide for the Care and Use of Laboratory Animals prepared by the Institute of Laboratory Animal Resources and published by the National Institutes of Health (NIH

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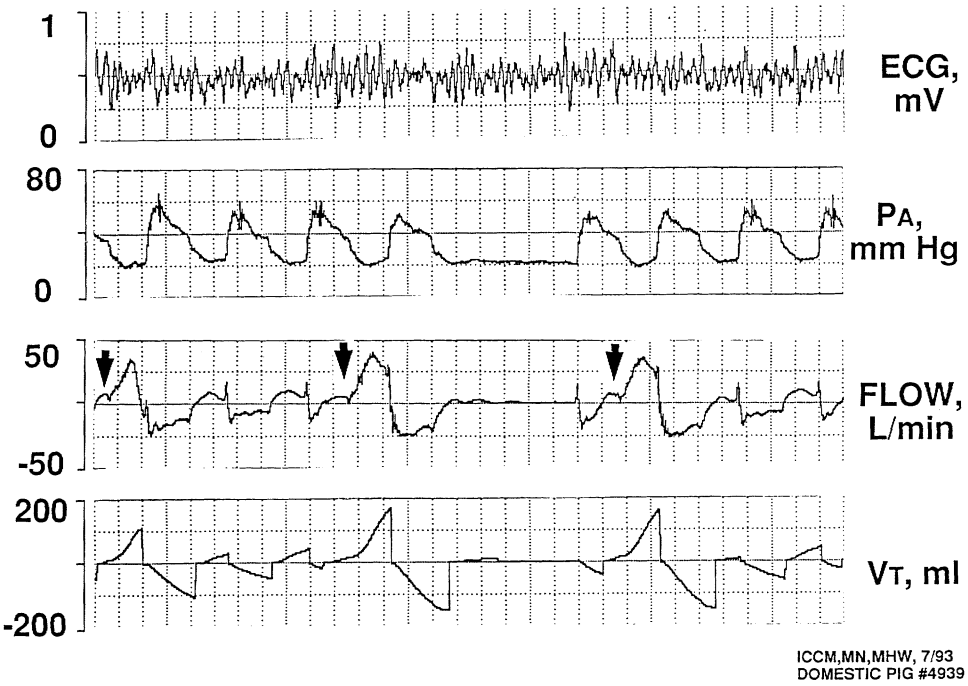

FIGURE 1. A representative tracing of spontaneous gasping during precordial compression. The arrows indicate the start of the gasp. ECG=electrocardiogram; PA=aortic pressure; Flow=airway flow; VT=tidal volume.

publications 86-33, revised 1985).

The porcine model of cardiac arrest utilized for these experiments has been described previously.[12-14] Twenty-two immature domestic pigs of both sexes weighing 29 to 42 kg were fasted overnight except free access to water. Anesthesia was initiated by intramuscular injection of ketamine (20 mg/kg) and completed by ear vein injection of sodium pentobarbital (20 mg/kg). A cuffed endotracheal tube was advanced into the trachea. Animals were then ventilated with a tidal volume of 15 mL/kg, peak flow of 40 L/min, and FIO_2 of 0.21 with the aid of a volume-controlled ventilator (model MA-1; Puritan-Bennett; Carlsbad, Calif). End-tidal PCO_2 was monitored with an infrared analyzer (model 601 POET; Criticare Systems; Milwaukee). Respiratory rate was adjusted to maintain end-tidal PCO_2 between 35 and 40 mm Hg.

A 7F pentalumen thermodilution catheter (model P7110-EH; Abbott Laboratories; North Chicago, Ill) was advanced into a surgically exposed right femoral vein and flow directed into the pulmonary artery. Through the right femoral artery, an 8F angiographic catheter (model 6523; USCI C.R. Bard Inc; Billerica, Mass) was advanced into the descending thoracic aorta. Through the right cephalic vein, a 4F pacing electrode was advanced with electrocardiographic monitoring such that its tip impinged on the right ventricular endocardium. Catheters were advanced with fluoroscopic guidance and position was further confirmed by characteristic pressure pulse morphology. Blood temperature was continuously measured in the pulmonary artery and maintained between 37°C and 38°C utilizing infrared surface heating lamps.

Aortic, pulmonary artery, and right atrial pressures were measured through fluid-filled catheters with the aid of strain gauge pressure transducers (Transpac 42585-01; Abbott Critical Care Systems; North Chicago, Ill). Coronary perfusion pressure was estimated as the arithmetic difference between time-coincident aortic and right atrial diastolic pressure. Cardiac output was measured by thermodilution technique with the aid of a cardiac output computer (model 3300; Abbott Critical Care Systems; North Chicago, Ill) after a right atrial, bolus injection of 5 mL of 5% glucose, which was maintained at a temperature of 4C.

Arterial and mixed venous blood pH, PCO_2, PO_2, and hemoglobin saturation were measured with the aid of an automated pH/blood gas analyzer (model 1306, and Co-Oximeter System model 482; Instrumentation Laboratories; Lexington, Mass). Lactate concentration was measured with an electrode-based lactate analyzer (model 23L; Yellow Springs Instruments; Yellow Springs, Ohio).

For measurements of airway flow, a pneumotachometer (Fleisch model 3500; Hans Rudolph Inc; Kansas City, Mo) was adapted to the endotracheal tube. Gasping was identified from discrete morphologic features of the airway flow recording (Fig 1). Tidal volumes generated by gasping and precordial compression and total minute ventilation were computed by integration of the airway flow signals utilizing software (CODAS; DATAQ Inc; Akron, Ohio). The minute ventilation generated by gasping was estimated as the sum of tidal volumes produced by individual gasps. The minute ventilation attributed to precordial compression represented the difference between total minute volumes and the volumes produced by either gasping or by positive pressure ventilation during each minute of precordial compression.

The quantitative neurologic deficit scores of Bircher and Safar[15] based on observations of activity and on neurologic examinations were utilized. The estimate is computed as a percentage deficit that ranges from 0% (no deficit) to 100% (brain death).

After the animals had partially recovered from anesthesia and spontaneous ventilation had returned, cardiac arrest was induced. A progressive increase in alternating current from 1 to 5 mA delivered to the right ventricular endocardium induced ventricular fibrillation (VF). After 4 min of untreated VF, precordial compression was begun with the aid of a pneumatic piston device (Thumper, model 1000; Michigan Instrument; Grand Rapids, Mich). The compression pad was centered at the junction of the middle and lower third of sternum. It was programmed to provide a compression rate of 80/min and equal compression/relaxation intervals. The compression force was adjusted to decrease anteroposterior diameter within the range of 25 to 30%.

Animals were randomized to two groups coincident with the start of precordial compression. For the first group (A), positive pressure

Table 1—*Immediate Cardiac Resuscitability, Energy Required for Successful Defibrillation, 48-h Survival, and Neurologic Deficit Score (NDS) at 24 and 48 h Following Successful Cardiac Resuscitation

Group	A	B
Resuscitability	7/11	8/11
Defibrillation, J	900±648	1,163±518
48-h survival	7/11	8/11
NDS, % at 48 h	0	0

*A=positive pressure ventilation with oxygen; B=spontaneous breathing with oxygen.

ventilation with the volume-controlled ventilator (model MA-1, Puritan-Bennett) was synchronized to deliver one breath after each five precordial compressions. Tidal volume was adjusted such as to maintain arterial P_{CO_2} between 30 and 40 mm Hg. During precordial compression, F_{IO_2} was maintained at 1.0 by methods previously described.[14] In the second subset of animals (B), a continuous flow of oxygen was delivered through a one-way valve adapted to the endotracheal tube such that this gas flow produced no measurable increase in the airway pressure. A 1-L bag reservoir was incorporated into the inspiratory limb of the system such as to accommodate gasping.

After 12 min of VF, which included 4 min of untreated cardiac arrest ("downtime interval") and 8 min of precordial compression, a maximum of two 300-J direct current countershocks were delivered between the right infraclavicular area and the cardiac apex as previously described.[12-14,16] Failing to reverse VF, the countershocks were repeated after an additional 30-s interval of chest compression. This resuscitation protocol was repeated for a maximum of three cycles. Restoration of spontaneous circulation was defined as the return of supraventricular rhythm with a mean aortic pressure of 60 mm Hg for a minimum of 5 min. Resuscitated animals were observed for an interval of 3 h under critical care conditions during which they were mechanically ventilated with 100% oxygen. Except for positive pressure ventilation with 100% oxygen, no other resuscitative interventions were utilized. After 3 h, the endotracheal tube, vascular catheters, and electrodes were removed. The animals were then returned to their cages and observed for an additional interval of 48 h.

Statistical Analysis

Data are presented as mean±SD unless otherwise stated. For continuous data, differences between the two groups were analyzed by analysis of variance using the Scheffé method for multiple comparison. Categorical variables were analyzed with Fisher's exact test. A p value of less than 0.05 was regarded as significant.

Results

The outcome with respect to cardiac resuscitability and 48-h survival was approximately the same whether or not the animals were mechanically ventilated. Seven of 11 animals receiving positive pressure ventilation and 8 of 11 animals without mechanical ventilatory support were successfully resuscitated (NS). The energy required for successful cardiac defibrillation did not differ significantly between the two groups. All resuscitated animals survived 48 h and there was no neurologic deficit at either 24 or 48 h (Table 1).

Spontaneous gasping began within 1 min after onset of cardiac arrest. During precordial compression in the absence of positive pressure ventilation, gasping significantly increased minute volumes (Table 2). The minute volumes generated by precordial compression in these animals were approximately 3 L during the first minute and subsequently declined to approximately 1 L after 7 min and before defibrillation was attempted. In the animals that were maintained with positive pressure ventilation, the minute volumes that were produced by precordial compression averaged less then 1 L.

Arterial P_{CO_2} remained at less than 40 mm Hg during the first 2 min of precordial compression and no significant differences were detected between the two study groups. Yet, after 8 min of precordial compression, arterial P_{CO_2} averaged 28 mm Hg in animals with positive pressure ventilation and 48 mm Hg in the absence thereof with corresponding differences in arterial pH. Minor reductions in calculated arterial HCO_3^- were associated with increases in blood lactate levels (Tables 3 and 4). With positive pressure ventilation, arterial pH was maintained at 7.40, and arterial HCO_3^- was reduced to 17 mmol/L after 12 min of cardiac arrest. However, in spontaneously breathing animals, pH was reduced to 7.27, and mean arterial HCO_3^- was 20 mmol/L during the same interval (Table 3 and Fig 2). Increases in arterial P_{CO_2} were accompanied by cor-

Table 2—*Ventilation Produced by Gasping, Ventilation Produced by Precordial Compression, and Total Minute Ventilation, During Untreated Ventricular Fibrillation and Precordial Compression

Time	VF1	VF4	PC1	PC4	PC8
Gasping, L/min					
A	4.0±1.1	3.6±1.8	0.7±0.7†	0.1±0.3†	0.1±0.3†
B	4.4±1.7	3.5±2.1	2.2±1.4	1.8±1.1	2.7±1.5
Precordial compression, L/min					
A	—	—	0.8±0.6†	0.4±0.9†	0.4±0.9†
B	—	—	3.0±0.8	1.6±0.8	1.2±0.6
Total, L/min					
A	4.0±1.1	3.6±1.8	6.9±0.8†	6.0±0.9†	5.9±0.8†
B	4.4±1.7	3.5±2.1	5.0±1.2	3.4±1.5	3.9±1.6

*The values represent mean±SD. A=positive pressure ventilation with oxygen; B=spontaneous breathing with oxygen; VF1, VF4=first and fourth minute of untreated ventricular fibrillation; and PC1, PC4, PC8=first, fourth, and eighth minute of precordial compression.
†$p<0.05$ vs B.

Table 3—*Arterial and Pulmonary Artery Blood pH, P_{CO_2}, and P_{O_2} Before and During Cardiac Arrest, and Following Successful Cardiac Resuscitation**

Time	BL	PC2	PC8	PR5	PR60	PR120	PR180
pHa, units							
A	7.48±0.03	7.45±0.05	7.40±0.05†	7.20±0.04	7.41±0.05	7.42±0.04	7.44±0.03
B	7.46±0.04	7.43±0.11	7.27±0.11	7.16±0.08	7.36±0.03	7.40±0.02	7.43±0.02
$PaCO_2$, mm Hg							
A	38±3	33±3	28±3†	51±6	39±4	41±4	41±3
B	39±5	37±9	48±19	57±9	46±4	43±4	42±4
$[HCO_3^-]$a, mmol/L							
A	27±3	22±1†	17±1†	20±2	25±2	26±1	27±1
B	26±2	24±2	21±3	20±2	26±2	26±2	27±2
PaO_2, mm Hg							
A	87±8	165±82	200±106	375±92	503±35	474±33	494±37
B	90±8	215±144	141±81	301±77	442±42‡	447±63	478±55
$pH\bar{v}$, units							
A	7.39±0.03	7.28±0.05	7.23±0.05†	7.15±0.04	7.35±0.04	7.38±0.04	7.39±0.03
B	7.37±0.02	7.26±0.04	7.15±0.06	7.11±0.07	7.32±0.02	7.35±0.02	7.37±0.02
$P\bar{v}CO_2$, mm Hg							
A	46±2	57±6	56±9†	63±5	48±5	49±5	48±5
B	49±2	62±8	71±11	68±8	54±4	51±4	50±4
$P\bar{v}O_2$, mm Hg							
A	56±3	34±4	32±5	67±10	50±8	53±4	51±5
B	59±7	36±3	41±9	64±11	58±8	58±5	55±7
$[HCO_3^-]$, mmol/L							
A	29±1	26±2	23±2	22±1	26±2	28±2	29±2
B	29±3	27±2	24±2	21±2	27±2	28±2	29±2

*The values represent mean±SD. A=positive pressure ventilation with oxygen; B=spontaneous breathing with oxygen; BL=baseline; PC2 and PC8=second and eighth minute of precordial compression; PR5, PR60, PR120, PR180=5, 60, 120, and 180 min following successful cardiac resuscitation.

†$p<0.05$ vs B.

‡$p<0.05$ vs A.

responding increases in mixed venous P_{CO_2} and reciprocal decreases in pH (Fig 3). Accordingly, the venoarterial gradients for pH and P_{CO_2} were approximately the same. Arterial P_{O_2} during precordial compression was maintained in excess of 100 mm Hg whether or not animals were maintained on mechanical ventilation (Fig 2).

After onset of VF, coronary perfusion pressure (CPP) decreased from 94 mm Hg to between 3 and 5 mm Hg. During precordial compression, CPP was initially increased to approximately 17 mm Hg but subsequently declined to average levels of 12 mm Hg during the remaining 7-min interval of precordial compression. There were no significant differences in CPP between the two groups.

Following successful cardiac resuscitation, cardiac output was reduced to 70% of prearrest values. This was associated with increases in the heart rate from an average of 150 to more than 180/min. However, pulmonary artery occlusive pressure remained in the range of 1 to 3 mm Hg. Decreases in cardiac output notwithstanding, mean aortic pressures were approximately the same prior to cardiac arrest and following successful resuscitation in each of the two groups (Table 4). Arterial lactate concentration, which peaked at approximately 6 mmol/L immediately following successful cardiac resuscitation, progressively decreased to less then 2 mmol/L over the 3-h postresuscitation interval (Table 4). Transient increases of arterial P_{CO_2} to 60 mm Hg were observed immediately following successful cardiac resuscitation. Arterial P_{CO_2} then returned to physiologic ranges. Arterial P_{O_2} increased to the range of 300 to 500 mm Hg after successful resuscitation. We therefore observed no differences in the hemodynamic parameters, arterial blood lactate levels, or in arterial blood gas values between the two experimental groups after successful resuscitation.

Discussion

The present study showed that the combination of precordial compression and spontaneous gasping may generate sufficient minute ventilation for successful cardiac resuscitation during the early stages of CPR. Accordingly, adequate gas exchange may be maintained in the absence of positive pressure ventilation. Even though no important increases in $Paco_2$ were observed during the course of these studies, earlier observations had indicated that much greater increases to levels of 100 mm Hg did not compromise cardiac resuscitability in rats[17,18] and in pigs.[18]

Once again, evidence of beneficial effects of spontaneous gasping during cardiac arrest was identified.[18]

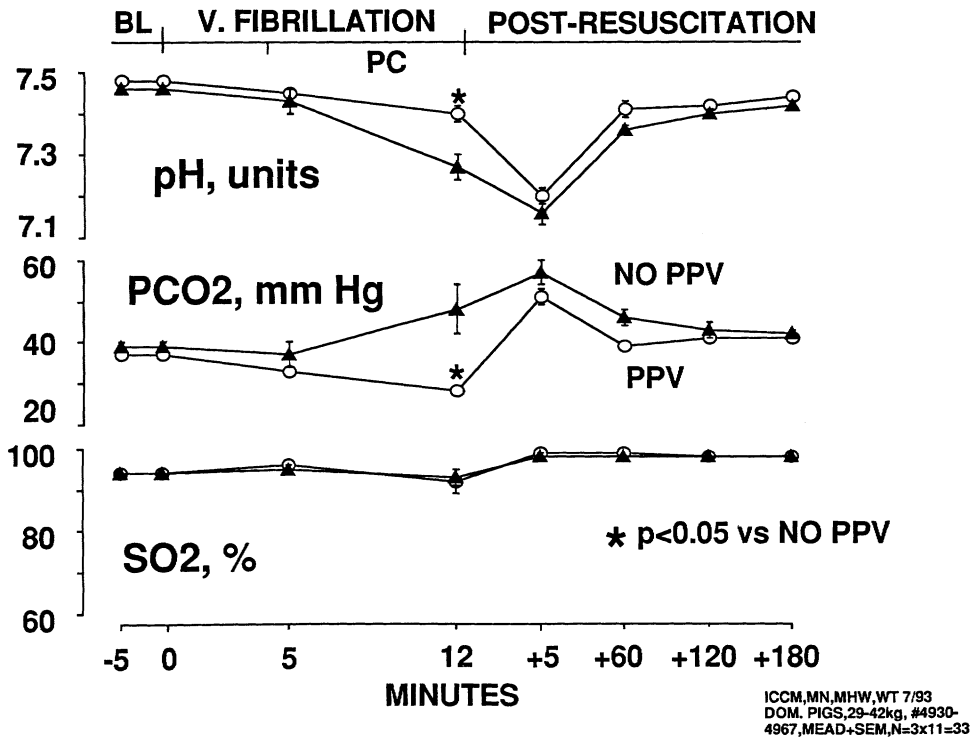

FIGURE 2. Arterial pH, P_{CO_2}, and oxyhemoglobin saturation (SO_2) before and during cardiac arrest, and following successful cardiac resuscitation. Mean±SEM is shown. BL=baseline; VF=ventricular fibrillation; PC=precordial compression; PPV=positive pressure ventilation.

This phenomenon may have escaped detection in earlier studies by our group and by other investigators because gasping is ablated during more profound states of anesthesia and also by positive pressure ventilation independently of the depth of anesthesia. This contrasts with human victims of cardiac arrest in whom gasping is observed quite consistently.[19] The present findings suggest that gasping is useful and that it represents an autoresuscitative response in the absence of external ventilatory support.

In these experimental studies, routine endotracheal intubation secured patency of the airway in compliance with current guidelines.[20,21] As yet undetermined is the specific benefit derived from endotracheal intubation independently of the effects of positive pressure ventilation. Recent studies by Berg and his associates,[9] also

Table 4—*Mean Aortic Pressure (MAP), Cardiac Index (CI), Heart Rate (HR), Pulmonary Capillary Occlusive Pressure (PAOP), and Arterial Blood Lactate Concentration Before Cardiac Arrest and Following Successful Cardiac Resuscitation

Time	BL	PR5	PR60	PR120	PR180
MAP, mm Hg					
A	105±13	99±22	94±17	97±9	92±10
B	103±12	84±14	97±16	100±7	102±7
CI, mL/kg					
A	161±25	155±42	120±32	115±20	113±12
B	170±21	152±52	140±32	135±30	119±29
HR, beats/min					
A	156±24	208±35	186±20	166±22	151±17
B	162±28	180±35	202±34	180±21	168±21
PAOP, mm Hg					
A	2±1	2±1	2±1	2±2	2±1
B	2±1	3±2	3±2	2±2	2±2
Lactate, mmol/L					
A	1.3±0.5	5.8±0.9	2.6±0.9	2.0±1.0	1.3±0.7
B	1.5±0.4	6.0±0.7	2.5±0.8	2.1±1.0	1.4±0.6

*Values represent mean±SD. A=positive pressure ventilation with oxygen; B=spontaneous breathing with oxygen; BL=baseline; PR5, PR60, PR120, PR180=5, 60, 120, and 180 min following successful cardiac resuscitation.

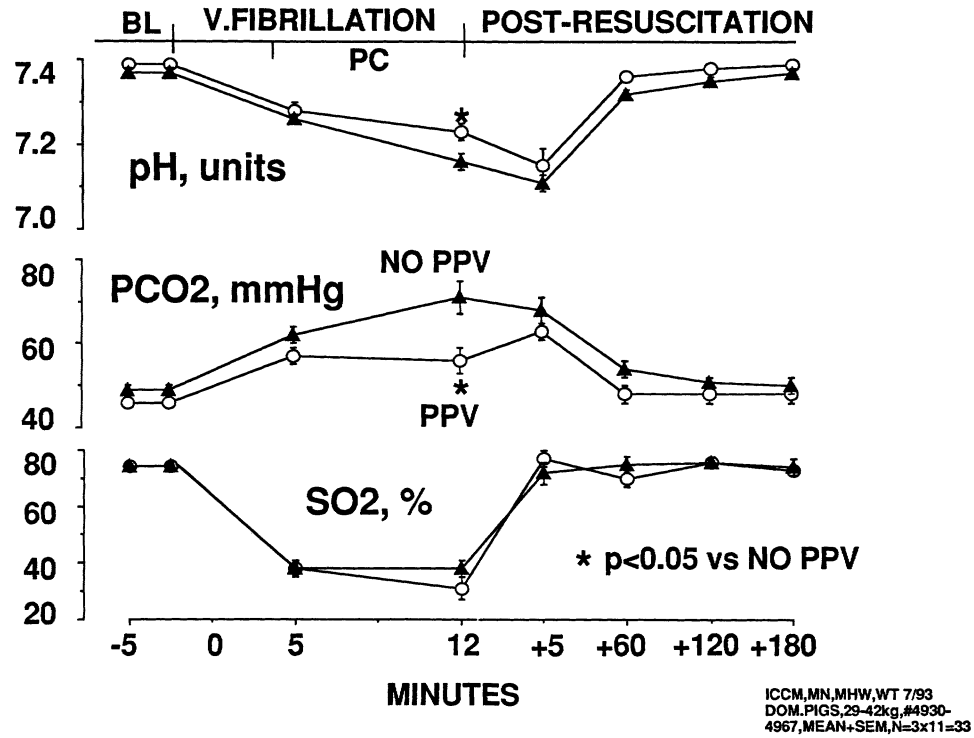

FIGURE 3. Mixed venous pH, P_{CO_2}, and oxyhemoglobin saturation (SO_2) before and during cardiac arrest, and following successful cardiac resuscitation. Mean±SEM is shown. BL=baseline; VF=ventricular fibrillation; PC=precordial compression; PPV=positive pressure ventilation.

on a porcine model, and a more recent study by us[22] on a rat model, demonstrated that neither endotracheal intubation nor mechanical ventilation was a prerequisite to successful cardiac resuscitation. Gasping produces wide swings in translaryngeal pressure with rapid opening of the glottis but intense closing in the intervals between gasps and therefore is likely to maintain patency of the upper airway.[23,24] The present report is fully supportive of those conclusions. We further document that tidal volumes generated by the combination of precordial compression and spontaneous gasping in the absence of mechanical ventilation is sufficient for maintaining arterial blood gas values at essentially normal levels during the initial 8 min of CPR.

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