

Transtacheal Oxygenation*

An Alternative to Endotracheal Intubation During Cardiac Arrest

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Study objectives: Because efforts to secure adequate arterial oxygenation during cardiac resuscitation are more important than efforts to promote CO₂ elimination, we investigated whether continuous transtacheal oxygenation (TTO) could represent a potentially simpler alternative to conventional positive-pressure ventilation with 100% O₂ through an endotracheal tube.

Design: Controlled and randomized.

Setting: Animal laboratory.

Participants: Thirty male Sprague-Dawley rats.

Interventions: The technique for TTO was initially developed and tested in five rats. A model of ventricular fibrillation (VF) was then used to compare the effects of TTO (n = 5) with the effects of O₂ delivery through an endotracheal tube as part of positive-pressure ventilation (n = 5) or through a mask without additional airway intervention (n = 5). VF was induced and left untreated for 4 min, after which chest compression and one of the three oxygenation interventions was started. Defibrillation was attempted after 6 min of chest compression. In a subsequent series, defibrillation was attempted after 10 min of chest compression in rats treated with either TTO (n = 5) or endotracheal intubation (ET; n = 5).

Measurement and results: TTO and ET secured adequate arterial PO₂ during chest compression (213 ± 77 mm Hg and 154 ± 36 mm Hg; not significant), whereas the mask yielded an arterial PO₂ of only 49 ± 38 mm Hg (p < 0.05). Each rat treated with TTO or ET was successfully resuscitated and survived the postresuscitation interval, but none of the rats treated with the mask survived. TTO maintained its efficacy after increased duration of chest compression.

Conclusion: TTO was as effective as conventional positive-pressure ventilation with 100% O₂ for securing oxygenation, resuscitation, and short-term survival and more effective than O₂ delivered through a mask. (CHEST 2001; 120:1663-1670)

Key words: cardiopulmonary resuscitation; intubation, intratracheal; O₂; rats, Sprague-Dawley; trachea; ventricular fibrillation

Abbreviations: ANOVA = analysis of variance; ET = endotracheal intubation; NS = not significant; TTO = transtacheal oxygenation; VF = ventricular fibrillation

It is commonly believed that oxygenation and CO₂ removal are equally important for successful resuscitation from cardiac arrest. This belief has sup-

ported the current practice in which both oxygenation and CO₂ removal are actively pursued during cardiac resuscitation. Typically, this is initially attempted through an unprotected airway using mouth-to-mouth or bag-valve-mask ventilation and subsequently through a protected airway after orotracheal intubation. Implementation of these techniques, however, requires skills that may be difficult to master and retain. Moreover, orotracheal intubation during the crisis situation of cardiac arrest may be technically difficult and increase the risk of complications.^{1,2} Attempts to secure an airway may also disrupt ongoing resuscitation efforts by requiring temporary interruption of chest compression.³

Studies⁴ in animal models of cardiac arrest have shown that large increases in arterial PCO₂ (approx-

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imately 100 mm Hg) are well tolerated and do not compromise resuscitability and survival. In contrast, decreases in arterial O₂ saturation can preclude restoration of cardiac activity.⁵ Thus, development of strategies that could selectively focus on O₂ delivery without actively pursuing CO₂ elimination may prove to be effective and to obviate the need of orotracheal intubation for positive-pressure ventilation.

Previous studies^{6,7} in large animals have shown that continuous tracheal O₂ insufflation can maintain adequate arterial PO₂ for long intervals even in the absence of positive-pressure ventilation or spontaneous breathing. We reasoned that O₂ delivery at constant flow through a small catheter percutaneously advanced through the cricothyroid ligament into the trachea could fulfill the requirements of being simple and effective, and obviate the need for more advanced airway intervention. We devised a method for transtracheal oxygenation (TTO) in a rat model of ventricular fibrillation (VF) and investigated its effects on gas exchange, resuscitability, and short-term survival.

MATERIALS AND METHODS

The studies were approved by our Research and Development Committee and conducted according to institutional guidelines.

Animal Preparation

Sprague-Dawley rats (460 to 558 g) were anesthetized by intraperitoneal injection of sodium pentobarbital, 45 mg/kg, and supplemented with additional doses, 10 mg/kg, at 30-min intervals. Core temperature was monitored with a thermistor (TSD102A; BIOPAC Systems; Santa Barbara, CA) advanced 4 cm into the rectum and maintained between 36.5°C and 37.5°C using an infrared heating lamp.

A lead II ECG was recorded through subcutaneous needles. Polyethylene catheters (PE50; Becton Dickinson; Sparks, MD) were advanced through the left femoral artery into the abdominal aorta, through the right jugular vein into the right atrium, and through the right carotid artery into the left ventricle for pressure measurement and blood sampling. Pressures were measured with reference to the midchest using disposable pressure transducers (Maxxim Medical; Athens, TX). Signals were processed using BIOPAC signal conditioners (BIOPAC Systems), sampled at 250 scans per second, and digitized using a 16-bit data acquisition board (AT-MIO-16XE-50; National Instruments; Austin, TX).

For TTO, the larynx and upper portion of the trachea were surgically exposed through a 2-cm midline incision. The cricothyroid ligament was identified, and a 21-gauge needle was advanced approximately 0.5 cm into the larynx. After aspiration of free air, a guidewire was advanced through the needle lumen into the trachea. A 3F polyurethane pediatric venous catheter (C-PMS-301-J-PED; Cook; Bloomington, IN) was then advanced over the wire and its tip positioned approximately 2 cm from the carina. The catheter was secured in place with 4-0 nylon surgical suture (Fig 1). A transtracheal O₂ flow of 250 mL/min was chosen to approximately match the 15 L/min previously shown⁶ in 24-kg dogs to secure an arterial PO₂ > 100 mm Hg.

Initial studies were conducted to assess whether TTO could

secure arterial oxygenation after suppression of spontaneous breathing using IV pancuronium bromide (0.1 mg/kg) administered at 15-min intervals for a 60-min interval. Subsequent studies were conducted to assess the effects of TTO during cardiac resuscitation. For these studies, a previously developed protocol of VF and closed-chest resuscitation was used.⁸ Animals were prepared as described above, except that the anterior and posterior areas of the chest were shaved to facilitate electrical defibrillation. In addition, the left ventricular catheter was omitted and the right atrial PE50 catheter was advanced through the right femoral vein. Through the right jugular vein, a 3F pediatric radial artery catheter (C-PUM-301-J; Cook) was positioned into the right atrium and a guidewire was advanced through its lumen into the right ventricle for subsequent induction of VF (Fig 2).

Experimental Protocol

Animals were randomized before induction of VF to one of three oxygenation interventions during chest compression. One group represented TTO and had transtracheal catheters placed as described above (Fig 1). A second group represented conventional positive-pressure ventilation via an endotracheal tube and had 5F catheters (Abbocath-T 16-G × 2 inches; Abbott Laboratories; North Chicago, IL) orally advanced into the trachea according to the method described by Stark and coworkers.⁹ Intratracheal placement was verified by CO₂ measurement in the expired gas using an infrared CO₂ analyzer (CO₂SMO model 7100; Novamatrix Medical Systems; Wallingford, CT). Because previous studies¹⁰ have demonstrated adequate oxygenation with chest compression alone, a third group was included in which 100% O₂ was made available during chest compression through a mask loosely positioned around the snout but without additional airway intervention (Fig 2). Animals were allowed to breathe room air before induction of VF and did not receive neuromuscular blocking agents.

VF was induced using a 60-Hz alternating current (range, 1.0 to 7.0 mA) delivered to the right ventricular endocardium. After 4 min of untreated VF, chest compression was started at 200 breaths/min using a pneumatically driven compressor (CJ-80623; CJ Enterprises; Tarzana, CA). The depth of compression was adjusted to achieve a coronary perfusion pressure between 22 mm Hg and 26 mm Hg. These settings had previously been shown^{8,11} to generate approximately 16% of baseline cardiac output and approximately 18% of baseline myocardial blood flow in successfully resuscitated rats. Oxygenation (100% O₂) was started concurrently with chest compression. In the TTO group, O₂ was delivered at 250 mL/min. In the endotracheal intubation (ET) group, O₂ was delivered as part of positive-pressure ventilation using an electronically controlled pneumatic valve (R-481; Clippard Instrument Laboratory; Cincinnati, OH) set to provide one positive-pressure breath with a tidal volume of 3.9 mL/kg animal weight for every two compressions (100 breaths/min). This setting provided a physiologic minute volume of 390 mL/kg/min and matched the postresuscitation minute volume obtained at a respiratory rate of 60 breaths/min. In the third group, O₂ was delivered at a constant flow of 500 mL/min through the mask. This flow rate was set to exceed any potential inspiratory flow during reexpansion of the chest cavity following compression.

After 6 min of chest compressions (10 min of VF), defibrillation was attempted by delivering a maximum of two 2-J direct-current transthoracic electrical shocks (LIFEPAK 9P; Physio-Control Corporation; Redmond, WA). If VF persisted or an organized electrical rhythm with a mean aortic pressure of ≤ 25 mm Hg ensued, chest compression was resumed for an additional 30 s. This sequence of shocks and chest compression was

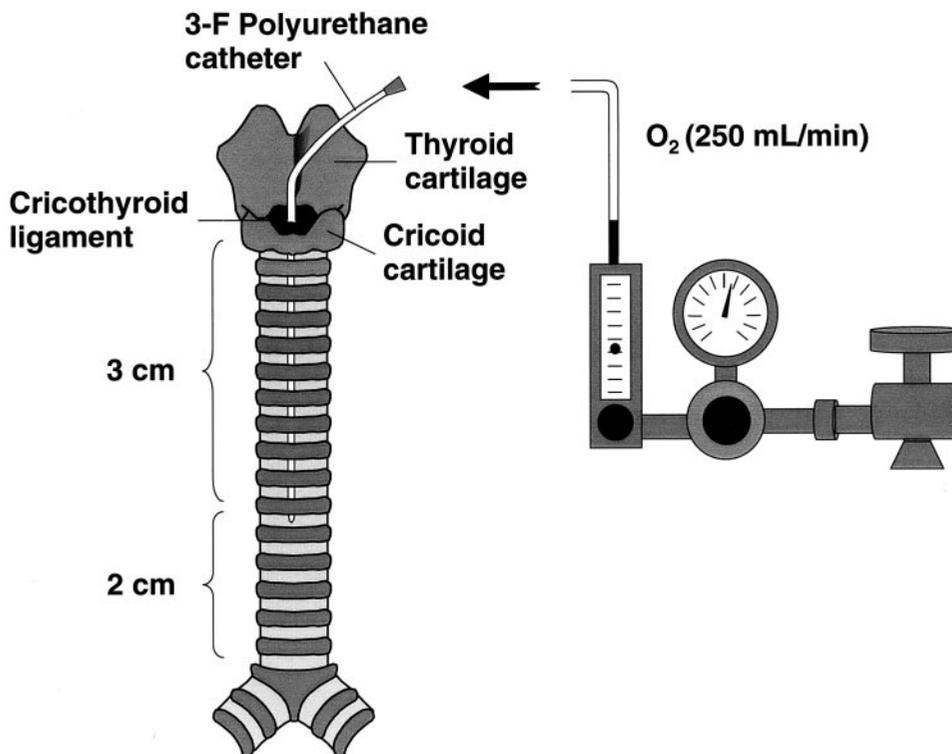


FIGURE 1. Diagram depicting TTO. A 3F polyurethane catheter was advanced through the cricothyroid ligament into the trachea. O₂ was administered at a constant flow of 250 mL/min using an adjustable flowmeter (range, 0 to 1.0 L/min).

repeated—if needed—for a maximum of three cycles, increasing the energy of electrical shocks to 4 J and then to 8 J. Successful resuscitation was defined as a supraventricular rhythm with a mean aortic pressure ≥ 60 mm Hg for ≥ 5 min.

At 5 min postresuscitation, animals that had been randomized to TTO were orally intubated and the transtracheal catheter was removed. Ventilation with 100% O₂ was then continued in both TTO and ET groups using a volume-controlled ventilator (model 683; Harvard Apparatus; South Natick, MA) set at a rate of 60 breaths/min and a tidal volume of 6.5 mL/kg; none of the rats that received O₂ through the mask were successfully resuscitated. At 15 min postresuscitation, the O₂ concentration was reduced to 50%. The animals were monitored for 2 h.

Approximately 400 μ L of blood was sampled from the abdominal aorta at defined intervals for pH, PO₂, and PCO₂ measurements using a blood gas analyzer (Nova Stat Profile 3; Nova Biomedical; Waltham, MA) and for lactate measurements using a lactate analyzer (YSI 2300 STAT; Yellow Spring Instruments; Yellow Springs, OH). An equivalent volume of arterial blood obtained from a donor rat was infused into the right atrium.

Statistical Analysis

Statistical analysis software was used (SigmaStat 1.0 for Windows; Jandel Scientific; San Rafael, CA). Continuous variables were compared using one-way analysis of variance (ANOVA). Changes over time were analyzed using one-way repeated-measures ANOVA. Equivalent nonparametric tests were substituted when tests for normality failed. The data are presented as mean \pm SD. A value of $p < 0.05$ was considered significant.

RESULTS

Efficacy of TTO During Spontaneous Circulation

Apnea prompted progressive increases in arterial PCO₂ from 35 ± 4 to 135 ± 20 mm Hg and decreases in arterial pH from 7.43 ± 0.05 to 6.83 ± 0.04 after 60 min ($n = 5$). The time-coincident arterial PO₂ increased from 66 ± 13 to 324 ± 33 mm Hg. Despite prominent hypercarbic acidosis, indexes of hemodynamic and left ventricular function remained stable throughout the 60-min interval (Table 1).

Effects During Cardiac Resuscitation

Baseline hemodynamic and gas-exchange data were comparable among groups. During chest compression, the average arterial PO₂ was similar in animals treated with TTO ($n = 5$) and ET ($n = 5$; 213 ± 77 mm Hg and 154 ± 36 mm Hg, respectively; not significant [NS]), with essentially identical arterial PCO₂ (27 ± 10 mm Hg and 26 ± 4 mm Hg; NS) and arterial pH (7.23 ± 0.14 and 7.22 ± 0.14 ; NS; Fig 3). In contrast, O₂ delivered through the mask ($n = 5$) yielded an arterial PO₂ of only 49 ± 38 mm Hg, a PCO₂ of 86 ± 21 mm Hg, and a pH of 6.97 ± 0.13 . Prominent and comparable increases in

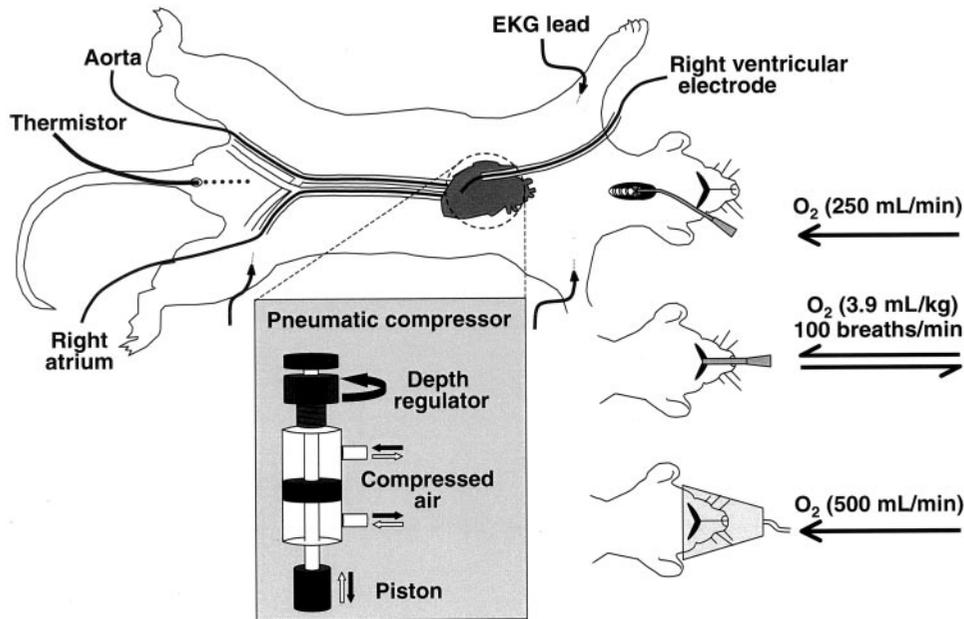


FIGURE 2. Rendition of our rat model of cardiac arrest illustrating the three techniques for oxygenation during chest compression: TTO, ET, and mask. The insert depicts a custom-made, pneumatically powered, electronically controlled piston that was used for chest compression.

arterial lactate occurred in each of the three groups. These were accompanied by decreases in arterial HCO_3^- in the TTO and ET groups (Table 2). In rats treated with the mask, decreases in arterial HCO_3^- were attenuated by increases in arterial PCO_2 .

Each TTO-treated and ET-treated rat was successfully defibrillated and spontaneous circulation was restored. In contrast, defibrillation attempts in rats treated with the mask led to a pulseless electrical activity in each instance. During the postresuscitation interval, TTO-treated and ET-treated rats exhibited comparable time resolution of metabolic and hemodynamic abnormalities (Fig 3, Table 2).

An additional series was conducted to determine whether the efficacy of TTO could deteriorate after increasing the duration of chest compression to 10 min (*ie*, by development of atelectasis). Comparable arterial PO_2 was observed with TTO ($n = 5$) and ET ($n = 5$) at 4 min (171 ± 64 mm Hg vs 184 ± 82 mm Hg; NS) and at 8 min (187 ± 55 mm Hg vs 220 ± 92 mm Hg; NS) of chest compression. The arterial PCO_2 was slightly higher with TTO at 4 min (44 ± 7 mm Hg vs 27 ± 7 mm Hg; $p < 0.05$) but not at 8 min (36 ± 7 mm Hg vs 37 ± 8 mm Hg; NS) of chest compression. Each rat in each group was successfully defibrillated and survived 120 min (Fig 4, Table 3).

Table 1—Effects of TTO in Five Rats During Spontaneous Circulation*

Variables	Baseline	Neuromuscular Blockade		
		15 min	30 min	60 min
pH	7.43 ± 0.05	$7.02 \pm 0.02^\dagger$	$6.93 \pm 0.03^\dagger$	$6.83 \pm 0.04^\dagger$
PCO_2 , mm Hg	35 ± 4	$78 \pm 4^\dagger$	$105 \pm 8^\dagger$	$135 \pm 20^\dagger$
PO_2 , mm Hg	66 ± 13	$337 \pm 20^\dagger$	$351 \pm 25^\dagger$	$324 \pm 33^\dagger$
HR, beats/min	345 ± 29	321 ± 34	$314 \pm 36^\dagger$	320 ± 37
MRAP, mm Hg	2 ± 2	5 ± 6	6 ± 4	5 ± 4
LVSP, mm Hg	154 ± 7	159 ± 8	166 ± 4	$181 \pm 10^\dagger$
LVDP, mm Hg	1 ± 2	1 ± 1	0 ± 2	-2 ± 3
+ dP/dtMAX, mm Hg/s	$3,812 \pm 278$	$3,659 \pm 305$	$3,799 \pm 163$	$4,109 \pm 303$
τ , ms	12 ± 1	12 ± 2	12 ± 2	12 ± 1

*Data are presented as mean \pm SD. MRAP = mean right atrial pressure; LVSP = left ventricular systolic pressure; LVDP = left ventricular end-diastolic pressure; τ = relaxation time constant; + dP/dtMAX = maximal rate of left ventricular pressure rise; HR = heart rate.

$^\dagger p < 0.05$ vs baseline by ANOVA repeated measures.

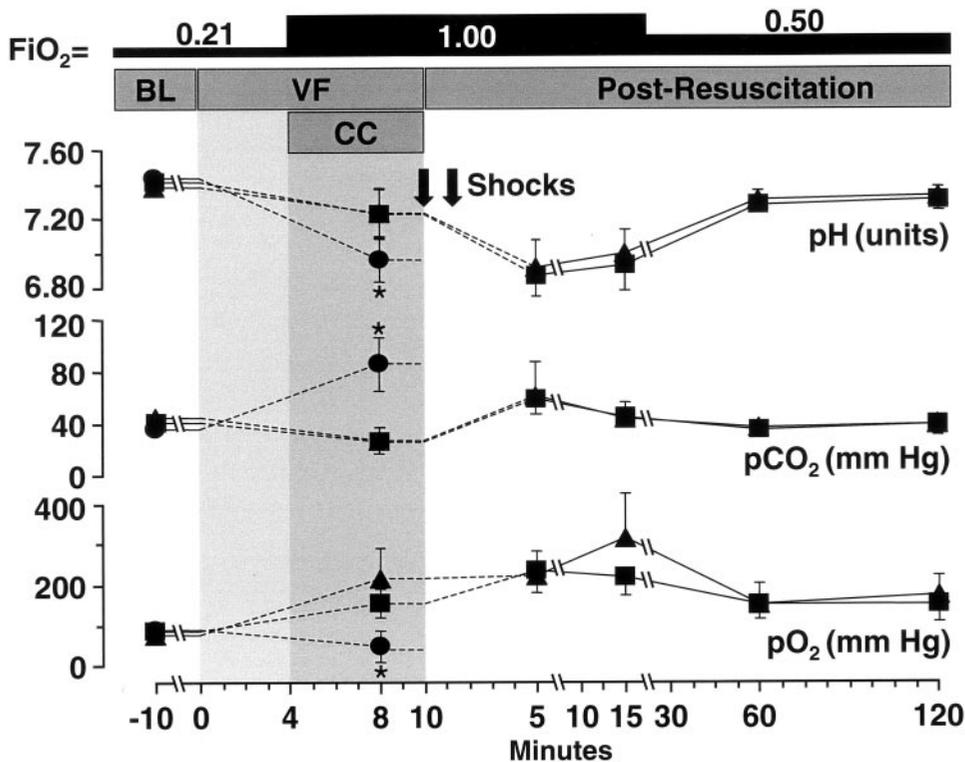


FIGURE 3. Arterial blood gas and pH levels in rats randomized to receive 100% O₂ during chest compression (CC) by TTO (triangles, n = 5), an endotracheal tube during positive-pressure ventilation (squares, n = 5), or a mask (circles, n = 5). The horizontal black bar denotes the fraction of inspired O₂ (FiO₂). BL = baseline. None of the animals that received O₂ through the mask was successfully resuscitated. *p < 0.05 vs TTO and vs endotracheal tube by one-way ANOVA.

DISCUSSION

TTO secured arterial oxygenation and facilitated successful defibrillation with an efficacy comparable to that of positive-pressure ventilation through a “conventional” endotracheal tube. Although not actively pursued, TTO also favored CO₂ elimination, yielding an arterial PCO₂ comparable to that attained with positive-pressure ventilation. The efficacy of TTO persisted despite increased duration of chest compression. In contrast, O₂ delivered through a mask (without attempts to maintain airway patency) failed to secure arterial oxygenation and precluded successful defibrillation.

Oxygenation During Chest Compressions

Adequate oxygenation seems to be essential for successful cardiac resuscitation.⁵ However, the time at which specific interventions for oxygenation become necessary during the resuscitation effort is less clear. Immediate oxygenation is essential in instances of asphyxial arrest,¹² yet several minutes may elapse before O₂ supplementation is required in instances of dysrhythmic arrest.¹⁰ Resuscitation and survival after out-of-hospital cardiac arrest seem to be inde-

pendent of whether mouth-to-mouth ventilation is provided during basic life support.¹³ These observations suggest an O₂ reservoir composed of O₂ bound to hemoglobin and O₂ present in the lungs at the time of arrest. Although such a reservoir may be partially renewed by gasping and chest compression through a patent airway, it is eventually depleted such that O₂ supplementation becomes necessary (*ie*, after 4 min of chest compression).¹⁰

Our studies assessed the effects of TTO at the time when interventions for direct tracheal oxygenation are required. The inadequate oxygenation provided by the mask demonstrated the need for direct tracheal oxygenation during chest compression in our rat model.

Current Approach

Positive-pressure ventilation with 100% O₂ delivered through an endotracheal tube is the “gold standard” for oxygenation during cardiac resuscitation. Additional benefits include airway protection and the option for controlled ventilation. Yet, the crisis situation of cardiac arrest imposes technical and logistic challenges to orotracheal intubation. For example, in adult victims of out-of-hospital cardiac

Table 2—Effects of TTO (n = 5), ET (n = 5), and Mask (n = 5)*

Variables/Groups	Baseline	Chest Compression, 4 min	Postresuscitation		
			15 min	60 min	120 min
MAP, mm Hg					
TTO	130 ± 13	32 ± 3	94 ± 7	91 ± 15	105 ± 19
ET	130 ± 13	33 ± 3	88 ± 13	98 ± 10	91 ± 17
Mask	124 ± 12	33 ± 3			
MRAP, mm Hg					
TTO	2 ± 2	11 ± 3	2 ± 1	3 ± 2	3 ± 1
ET	2 ± 2	11 ± 2	5 ± 3	2 ± 1	2 ± 1
Mask	3 ± 2	11 ± 3			
CPP, mm Hg					
TTO	112 ± 13	23 ± 5	80 ± 8	79 ± 15	94 ± 20
ET	114 ± 15	25 ± 4	74 ± 15	85 ± 9	78 ± 19
Mask	110 ± 14	22 ± 3			
Lactate, mmol/L					
TTO	1.0 ± 0.4	8.7 ± 2.9	9.8 ± 3.5	2.5 ± 0.5	1.7 ± 0.5
ET	0.9 ± 0.4	8.6 ± 3.6	11.0 ± 4.0	2.9 ± 1.7	1.8 ± 1.3
Mask	1.3 ± 0.1	8.3 ± 3.8			
HCO ₃ ⁻ , mmol/L					
TTO	26 ± 2	12 ± 4	11 ± 3	18 ± 2	21 ± 3
ET	26 ± 1	11 ± 3	10 ± 4	16 ± 2	21 ± 6
Mask	24 ± 2	20 ± 5†			

*Data are presented as mean ± SD. Rats were subjected to 4 min of untreated VF and additional 6 min of chest compression before attempting electrical defibrillation. MAP = mean aortic pressure; CPP = coronary perfusion pressure. See Table 1 for expansion of abbreviation. †p < 0.05 vs TTO and vs ET by one-way ANOVA.

arrest, trained personnel failed to properly place an endotracheal tube in 9% of the victims.¹⁴ Similarly, in deeply comatose patients, successful endotracheal tube placement required one attempt in 58% of the victims, two attempts in 26%, and three attempts in 6%.¹⁵ In a recent study¹⁶ involving 305 intubation attempts during out-of-hospital pediatric resuscitation, the trachea was successfully intubated in only 57% of the attempts and was complicated by mainstem intubation in 19%, subsequent endotracheal tube dislodgment in 10%, and inadequate tube size selection in 24%. In addition, there is increased risk of traumatic injury to the oral cavity, pharynx, larynx,¹⁷ and trachea,¹⁸ and inadvertent tube placement into the esophagus.¹⁵ In addition, proper visualization of anatomic structures during orotracheal intubation usually requires interruption of chest compression, which in of itself can compromise resuscitability.³

TTO as an Alternative

The present and other studies^{19–21} challenge the need for positive-pressure ventilation during cardiac resuscitation but emphasize the need for securing adequate oxygenation. For these reasons, a simplified approach that focuses on oxygenation independently of effects on CO₂ elimination is appealing. The efficacy of continuous O₂ delivery during chest compression has been independently confirmed by

other investigators, both in animal models⁷ and in human victims²¹ of cardiac arrest. Thus, TTO could represent a more effective alternative provided deployment is accomplished faster, with higher success rates, and with fewer complications than with orotracheal intubation. In addition, continuous oxygenation impresses as probably less laborious than bag-valve ventilation allowing rescuer efforts to be focused on other more vital resuscitation interventions. However, TTO is not free of complications. Improper technique may cause tracheal or esophageal wall perforation with serious mediastinal emphysema and bleeding. It can also cause cartilage injury with disruption of the voice apparatus.²² Another important consideration is that the airway must be patent above the insertion site to avoid air trapping and barotrauma.

In a recent study, housestaff officers were able to successfully place 12- to 16-gauge catheters through the cricothyroid membrane in 23 of 29 patients after multiple failed attempts to secure oxygenation by ET and bag-valve-mask ventilation.²³ The catheter was used for jet ventilation and promptly reversed hypoxemia. Others²⁴ have reported the successful placement of a transtracheal catheter under similar emergency situations and used it for tracheal ventilation with intermittent O₂.

Implementation of TTO in humans would probably require placement of a 16-gauge catheter ad-

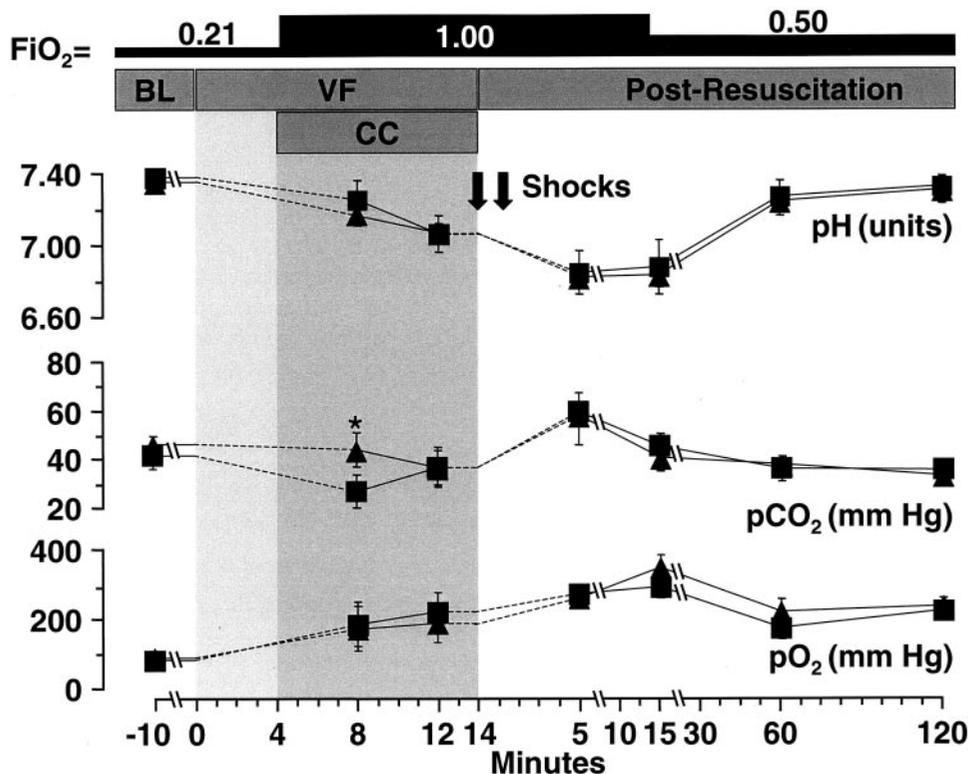


FIGURE 4. Arterial blood gas and pH levels in rats randomized to receive 100% O₂ during chest compression through a transtracheal catheter (triangles, n = 5) or an endotracheal tube during positive-pressure ventilation (squares, n = 5). The horizontal black bar denotes the fraction of inspired O₂. *p < 0.05 vs endotracheal tube by one-way ANOVA. See Figure 3 legend for definition of abbreviations.

vanced through the cricothyroid membrane or the first or second intertracheal ring space.²⁴ This size catheter would allow an O₂ flow of 15 L/min, corresponding to a flow that can secure adequate oxygenation in humans during chest compression.²¹

CO₂ Elimination

Our TTO technique was not purposely designed to remove CO₂. Yet, the arterial PCO₂ during chest compression was practically identical to that in ET-

Table 3—Effects of TTO (n = 5) and ET (n = 5)*

Variables/Groups	Baseline	Chest Compression		Postresuscitation		
		4 min	8 min	15 min	60 min	120 min
MAP, mm Hg						
TTO	135 ± 13	37 ± 2	33 ± 4	94 ± 10	101 ± 10	108 ± 9
ET	141 ± 10	35 ± 3	31 ± 2	83 ± 6	93 ± 8	112 ± 17
MRAP, mm Hg						
TTO	1 ± 1	9 ± 1	11 ± 1	3 ± 1	2 ± 2	1 ± 1
ET	2 ± 3	12 ± 4	14 ± 5	4 ± 3	3 ± 3	1 ± 3
CPP, mm Hg						
TTO	120 ± 15	28 ± 2	24 ± 3	78 ± 9	86 ± 10	96 ± 9
ET	123 ± 9	25 ± 4	21 ± 4	71 ± 7	80 ± 8	98 ± 18
Lactate, mmol/L						
TTO	1.1 ± 0.3	8.7 ± 1.4	11.6 ± 1.6	13.8 ± 1.2	3.8 ± 1.4	2.6 ± 0.9
ET	1.4 ± 0.4	8.6 ± 2.2	11.7 ± 2.2	12.3 ± 3.4	3.8 ± 1.7	1.8 ± 0.5
HCO ₃ ⁻ , mmol/L						
TTO	26 ± 2	16 ± 2†	11 ± 2	7 ± 1	17 ± 3	17 ± 3
ET	24 ± 2	12 ± 2	10 ± 1	9 ± 3	17 ± 3	19 ± 3

*Data are presented as mean ± SD. Rats were subjected to 4 min of untreated VF and additional 10 min of chest compression before attempting electrical defibrillation. See Tables 1 and 2 for expansion of abbreviations.

†p < 0.05 vs ET one-way ANOVA.

treated rats and lower than in mask-treated rats. Similar observations were reported in dogs and attributed to the bellows effect of chest compression.⁷ In humans, the arterial PCO₂ during chest compression was lower with continuous transtracheal O₂ insufflation than with conventional positive-pressure ventilation.²¹ Because CO₂ elimination may already occur during cardiac arrest associated with gasping²⁵ and chest compression,¹⁰ TTO may facilitate these processes by securing airway patency and by promoting CO₂ elimination by convection.

Clinical Implications

The perceived urgency for immediate ventilation and oxygenation propels rescuers to initiate positive-pressure ventilation through an unprotected airway by using mouth-to-mouth or bag-valve-mask ventilation. This approach has been shown to increase the risk of regurgitation with potential for aspiration of gastric contents²⁶ and subsequent development of pneumonia.²⁷

Given the presence of an O₂ reservoir at the onset of dysrhythmic cardiac arrest and a time window before O₂ supplementation is required, ventilatory attempts through an unprotected airway may not be necessary. TTO could be the exclusive airway intervention during chest compression. Once the catheter is secured in place, 100% O₂ (or other gas mixture) could be administered at a constant flow without need of a rescuer to provide positive-pressure ventilation. The tracheal catheter could also be used for drug delivery and—with additional technical development—perhaps incorporate capability for monitoring the expired CO₂ to assess the hemodynamic efficacy of chest compression.

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