Effects of Tracheal Insufflation of Oxygen (TRIO) on Blood Gases during External Cardiac Compressions in Dogs under Ventricular Fibrillation

Kazufumi OKAMOTO, Kenji URATA, Hirotada KATSUYA* and Tohru MORIOKA

Tracheal insufflation of oxygen (TRIO) is a form of constant-flow ventilation. We studied the effect of TRIO at a flow rate of 2 L/kg/min on arterial blood gases during external cardiac compressions in dogs with ventricular fibrillation. During the combined application of TRIO and external cardiac compressions, all animals were adequately oxygenated and hyperventilated except in cases where lung edema developed in the course of cardiopulmonary resuscitation (CPR). No pulmonary barotrauma was observed. The findings suggest that TRIO might be used as a temporary measure for emergency ventilation when CPR is performed in certain situations such as upper airway abnormalities or cardiac arrest outside the hospital setting, where intermittent positive pressure ventilation is not feasible.
(Key words: apnea, constant-flow ventilation, cardiac arrest, external cardiac compressions, tracheal insufflation)

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The standard management of cardiac arrest consists of intermittent positive pressure ventilation (IPPV) combined with external cardiac compressions[1]. However, in certain cases, such as cases of upper airway abnormalities or cardiac arrest outside the hospital setting, conventional IPPV may be impossible. To manage difficult situations such as these, an alternative rapidly applicable technique is required.

Tracheal insufflation of oxygen (TRIO) is a technique in which oxygen is introduced into the trachea at a constant flow rate via a catheter advanced to the level of the carina[2]. This technique has been found to provide adequate oxygenation without preoxygenation[2] and to produce normocarbia in apneic dogs when the flow rate is increased[3]. One advantage of this technique is its simplicity. No special equipment is required beyond a source of oxygen and a small caliber catheter. The present study was designed to see whether TRIO could maintain an adequate pulmonary gas exchange during cardiopulmonary resuscitation (CPR) in experimental animals.

Methods

Animal preparation

Six adult dogs of mixed breeds, weighing between 7.0 and 12.0 kg, were anesthetized with sodium pentobarbital (25 mg/kg iv). The animals were restrained in the supine position on a V-shaped board and tra-

*Department of Anesthesiology, *Division of Intensive Care Medicine, Kumamoto University Medical School, Kumamoto, Japan*

Address reprint requests to Dr. Okamoto: Department of Anesthesiology, Kumamoto University Medical School, 1-1-1 Honjo, Kumamoto, 860 Japan

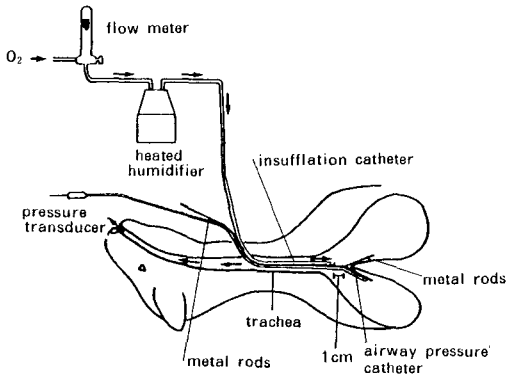

Fig. 1. Schematic diagram of the experimental design of TRIO.

cheostomized. Catheters were placed in a femoral vein for intravenous drug administration and in an artery for monitoring blood pressure and blood sampling. The arterial line was connected to a pressure transducer (P23ID, Gould Statham) and systemic arterial pressure was continuously recorded on a polygraph (RM-6200, Nihon Kohden). Heparin (1 mg/kg iv) was given to prevent clot formation in the catheters and to reduce intravascular coagulation during circulatory arrest. The lead II of ECG was monitored continuously. Body temperature was monitored with a rectal thermometer (MGA-III, Nihon Kohden) and maintained within normal range using a fluid-filled heating pad.

A single-lumen catheter (2.0 mm ID and 2.7 mm OD) was used for insufflaltion of oxygen. A smaller catheter (1.5 mm ID and 2.0 mm OD) was used to measure airway pressure. Both catheters had previously been fixed to two parallel metal rods with a bifurcation at the tip, as shown in figure 1. This device was inserted into the trachea through the tracheostoma until the bifurcation of the metal rods had reached the carina. Thus the tip of the insufflation catheter was placed 1 cm above the carina and the tip of the catheter for monitoring airway pressure was placed 2 cm beyond the carina. Metal rods were used to facilitate the insertion of the catheters and to keep them in a fixed position. A bronchoscope was used to check whether the tips of the two catheters had been appropriately positioned. The insufflation catheter was then connected to a tube from a gas delivery system, consisting of an oxygen flowmeter (The British Oxygen CO.) and a heated humidifier (MR 310, Fisher-Paykel Medical, Inc.). The airway pressure monitoring catheter was connected to a transducer (TP-200T, Nihon Koden) for continuous recording on a polygraph.

A tracheostomy tube with an inflatable cuff was then inserted and connected to a ventilator (CV-2000, McGaw Respiratory Therapy Co.). The animals were mechaically ventilated with room air through this tracheostomy tube with a tidal volume of 10–15 ml/kg at a rate sufficient to maintain Pa_{CO_2} at 30 to 45 mmHg.

Experimental protocol

After arterial blood samples had been drawn for control values, ventricular fibrillation (VF) was induced by an intravenous injection of potassium chloride (1 mEq/kg). Mechanical ventilation (IPPV) was interrupted as soon as VF was confirmed. The tracheostomy tube was withdrawn. The insufflation catheter and the airway pressure monitoring catheter were left in place. The tracheostoma was closed with sutures. The animals remained without any therapy for 1 min. Then, manual external cardiac compressions at a rate of 60–80/min and TRIO at a flow rate of 2 L/kg/min were initiated. For the cardiac compressions, the sternum was depressed about 3 to 5 cm. Intravenous epinephrine (0.5 mg) was given every five minutes to maintain the systolic blood pressure above 100 mmHg. Sodium bicarbonate and calcium chloride were not administered at any time during this study. Thirty minutes after the initiation of VF, resuscitation with both TRIO and external cardiac compressions was replaced by conventional CPR (IPPV with 100% oxygen and external cardiac compressions) requiring the re-insertion of a tracheostomy tube. Conventional CPR was applied for 5 min.

Arterial blood samples for blood gas analysis, pH and potassium determinations were obtained 1, 3, 5, 10, 15, 20, 25, 30 and

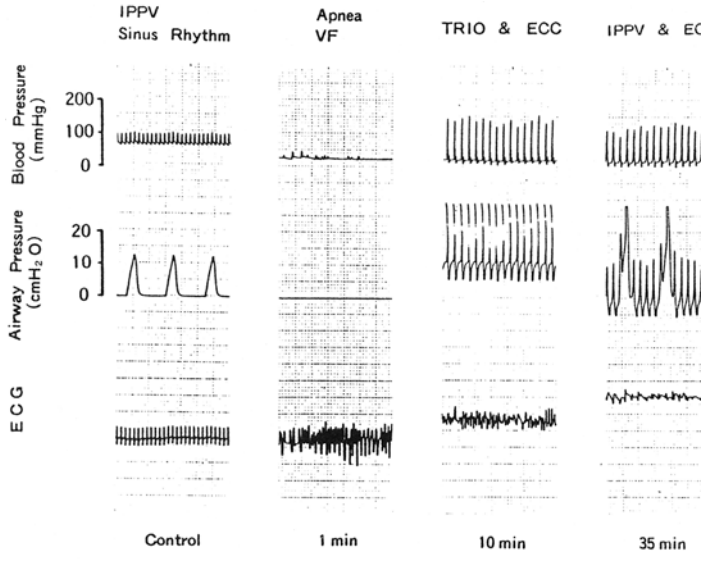

Fig. 2. Arterial blood pressure, endobronchial airway pressure and ECG using four different ventilation patterns. Note the airway pressure recordings during external cardiac compressions (ECC). The minimum airway pressure value is kept positive during the combination of TRIO and ECC. On the other hand, negative pressure in the airway synchronous with ECC is observed during the combination of intermittent positive pressure ventilation (IPPV) and ECC. VF = ventricular fibrillation.

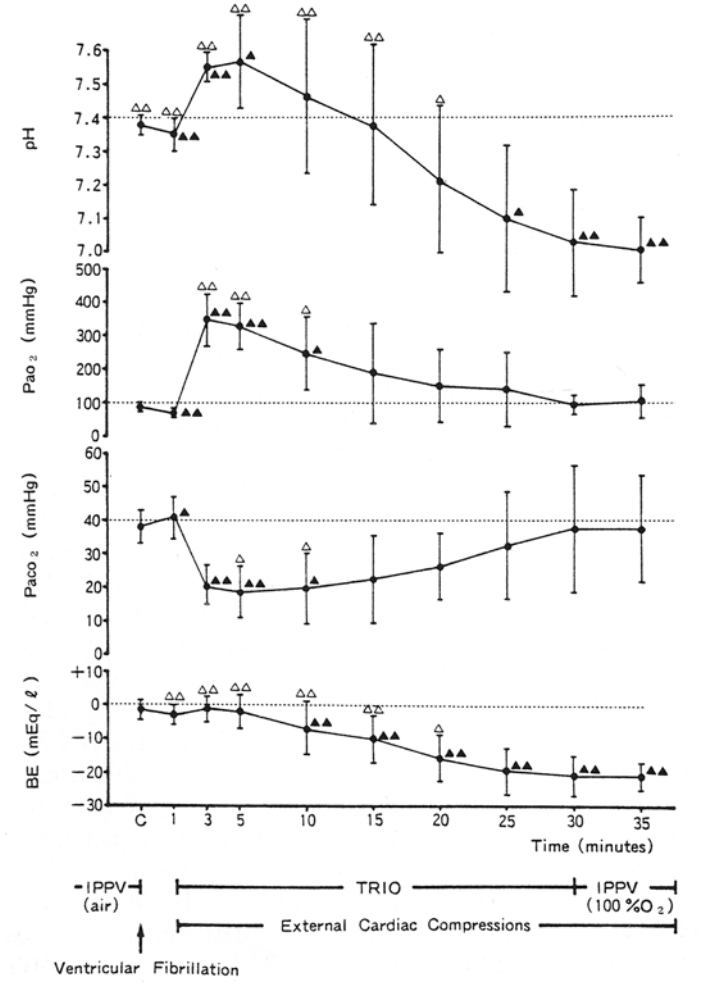

Fig. 3. pH, Pa_{O_2}, Pa_{CO_2}, and base excess (BE) before ventricular fibrillation and during cardiopulmonary resuscitation with TRIO or intermittent positive pressure ventilation (IPPV).

C = control (before ventricular fibrillation). Significantly different from control, ▲ $P < 0.05$; ▲▲ $P < 0.01$. Significantly different from the value of the combination of IPPV and external cardiac compressions (35 min after ventricular fibrillation), △ $P < 0.05$; △△ $P < 0.01$.

Table 1. Airway pressure, arterial blood pressure, body temperature and potassium concentrations before and during cardiopulmonary resuscitation

	IPPV	Apnea	TRIO							IPPV
			External Cardiac Compression							
	Control		Minutes after Ventricular Fibrillation							
		1	3	5	10	15	20	25	30	35
PAW	13	0	25[a]	26[a]	26[b]	27[b]	30[b]	39[b]	39[a]	36
(cmH_2O)	±3		±8	±7	±8	±5	±8	±24	±25	±24
MAW	0	0	3[a,c]	3[a,c]	4[a,c]	4[a,c]	4[a,c]	5[c]	2	−3[a]
(cmH_2O)			±3	±2	±3	±3	±3	±6	±4	±2
SAP	158[c]	17[b,d]	117[a]	129	148	125	133	149	136	113[a]
(mmHg)	±39	±2	±19	±19	±62	±17	±29	±42	±26	±7
MAP	109[d]	17[b]	44[b]	33[b]	34[b]	25[b]	27[b]	37[b]	36[b]	29[b]
(mmHg)	±25	±2	±31	±12	±22	±8	±16	±21	±18	±17
DAP	91[d]	17[b]	13[b]	11[b]	15[b]	6[b]	12[b]	13[b]	10[b]	11[b]
(mmHg)	±20	±2	±11	±12	±19	±6	±15	±23	±18	±12
BT	38.8[c]	38.8[c]	38.8	38.8[c]	38.7[c]	38.5	38.3	38.3	38.3[a]	38.2[a]
(°C)	±0.9	±0.9	±0.9	±0.9	±1.0	±1.1	±1.2	±1.3	±1.3	±1.3
K	3.7	25.6[a]	14.5[b]	13.4[b]	11.4[b]	12.0[b]	12.0[b]	11.5[b]	11.8[b]	12.3[b]
(mEq/L)	±0.3	±7.5	±2.7	±3.9	±2.7	±2.6	±2.7	±2.6	±2.3	±1.1

Values are expressed as mean±SD. PAW = peak airway pressure; MAW = minimum airway pressure; SAP = systolic arterial pressure; MAP = mean arterial pressure; DAP = diastolic arterial pressure; BT = body temperature; K = potassium concentration. Significantly different from control, a <0.05; b <0.01. Significantly different from the value of the combination of IPPV and external cardiac compressions (35 min after ventricular fibrillation), c <0.05; d <0.01.

35 min after the initiation of VF. Arterial blood gas tensions and pH were measured with a Corning 168 pH/blood gas analyzer. Potassium was measured with a Nova 5 electrolyte analyzer. The paired Student's t-test was used for statistical analysis. $P<0.05$ was considered significant.

Results

Figure 2 shows representative portions of sequential recordings of arterial blood pressure, endobronchial airway pressure, and ECG in one dog before and after the initiation of TRIO and external cardiac compressions. During CPR, changes in airway pressure synchronous with external cardiac compressions were observed in all dogs.

Figure 3 shows changes in arterial blood gases before and during CPR. Pa_{O_2} increased significantly from the control value of 88.7 ± 11.1 to 345.0 ± 82.0 mmHg after the initiation of both TRIO and external cardiac compressions (3 min after VF), and decreased gradually over the period of CPR. Adequate exygenation was maintained in all dogs throughout the entire course of CPR except for one dog. Of the six dogs studied, two dogs developed lung edema with frothy pink fluid in the trachea over the course of CPR. One of them became hypoxemic (Pa_{O_2} <50 mmHg) 20 min after VF. Thirty minutes after VF under TRIO, the mean Pa_{O_2} was 95.3 ± 31.3 mmHg. Thirty-five minutes after VF under IPPV, the mean Pa_{O_2} was 108.2 ± 45.3 mmHg. Pa_{CO_2} decreased significantly from the control value of 37.8 ± 4.9 to 20.2 ± 6.8 mmHg after the initiation of both TRIO and external cardiac compressions (3 min after VF), and increased gradually over the period of CPR. However, Pa_{CO_2} remained lower than the control value throughout the entire course of CPR except for the two

dogs that developed lung edema. Although the two dogs were hypocarbic for 10 to 20 min, hypercarbia developed 25 min after VF. Thirty minutes after VF under TRIO, the mean Pa_{CO_2} was 37.7 ± 19.6 mmHg. Thirty-five minutes after VF under IPPV, the mean Pa_{CO_2} was 37.5 ± 16.4 mmHg.

Changes in endobronchial airway pressures (peak and minimum), arterial pressures (systolic, mean, and diastolic), body temperature and potassium concentrations measured throughout the study are shown in table 1. The mean peak airway pressure increased significantly from the control value after the initiation of TRIO and external cardiac compressions (3 min after VF). It then gradually increased over the period of CPR. No pulmonary barotrauma was observed during CPR. The mean minimum airway pressure values were kept at a positive level of 2 to 5 cmH_2O during combined administration of TRIO and external cardiac compressions. The application of TRIO (2 L/kg/min) seemed to induce an "auto-PEEP" effect during CPR, while the mean minimum airway pressure value was −3 cmH_2O during the combination of IPPV and external cardiac compressions.

Discussion

The technique of ventilating with air or oxygen introduced into the tracheobronchial tree at a constant flow rate, namely constant-flow ventilation (CFV) is not new[4,5]. TRIO used in this study is a form of CFV[2].

In 1985, Slutsky et al.[2] reevaluated the technique of CFV using modern technology and proposed the new acronym "TRIO". They could sustain the life of apneic dogs for several hours with TRIO. Recently, we have shown that CO_2 elimination is improved by increasing the flow rate of TRIO[3]. For example, TRIO at a flow rate of about 3 L/kg/min could sustain normocarbia in apneic dogs, keeping them alive for over 4 hours.

The present study shows that TRIO sustains adequate pulmonary gas exchange in dogs during CPR except in cases where lung edema has developed. In light of our previous data[3] and the data of Slutsky et al.[2], TRIO differs substantially from the apneic oxygenation[6] since TRIO can eliminate CO_2 and maintain adequate oxygenation without preoxygenation. In addition, TRIO differs from the conventional transtracheal oxygenation techniques[7–10]. The conventional transtracheal oxygenation technique generally uses a short needle or catheter for oxygen insufflation into the trachea, and cannot sustain sufficient gas exchange to support life for a prolonged period.

The true mechanisms of pulmonary gas exchange during TRIO have not been clarified yet. According to the traditional theory of gas transport in the lung, adequate gas exchange would not be obtained without a larger tidal volume than the deal space volume. In our previous study[3], however, we have shown that normocarbia can be maintained by simply insufflating oxygen at a constant flow rate into the trachea in apneic dogs, provided the flow rate is sufficient. Burwen et al.[11] have postulated that gas transport during TRIO occurs in two distinct zones. Zone I is the region just distal to the catheter where gas is transported dominantly by a jet flow. Zone II is the region free from any influence of the jet flow. The latter includes the peripheral regions of the lung where gas transport is dominated by molecular diffusion enhanced by cardiogenic oscillations. Burwen et al.[11] have shown that cardiogenic oscillations increase gas transport about fourfold in dog lungs during TRIO. This value is in close agreement with previous reports[12,13]. In contrast, Brampton et al.[14] have recently shown that gas transport is enhanced about 5–10 times by pulsatile pulmonary blood flow, rather than cardiogenic oscillations. Their finding is consistent with that of Terasaki et al.[15].

In the absence of cardiac activity, both ordinary pulsatile pulmonary blood flow and intratracheal cardiogenic gas oscillations are lost. Gas transport by TRIO may be markedly impaired in dogs under VF. In the present study, however, we have shown that TRIO can produce adequate gas transport in

dogs under VF provided TRIO is combined with external cardiac compressions. We observed major airway pressure changes due to external cardiac compressions in all the dogs during CPR. These rhythmic airway pressure changes are similar to the pressure changes caused by the beating of the heart. Though each cardiac compression-induced gas movement may be too small to provide ventilation of the lungs, these back-and-forth gas movements may accelerate gas mixing in the anatomical dead space and the alveoli. When TRIO is combined with gas movements in the peripheral airways produced by external cardiac compressions, insufflated oxygen may be efficiently transported to the alveoli and alveolar CO_2 may be quickly carried away. Thus, pulmonary ventilation may be maintained by combining external cardiac compressions wit TRIO, without any need for IPPV.

Mackenzie et al.[16] and Webster et al.[17] suggested that ventilation through Cohn's collateral channels may be an important mechanism of gas transport during TRIO. When lung edema develops, fluid in the peripheral airways may prevent collateral channels from effectively maintaining gas transport to the obstructed lung units[18]. Their suggestion may be able to account for our findings here. In the present study, TRIO was not able to maintain adequate gas exchange when lung edema developed in the course of CPR. TRIO should be contraindicated for patients with lung edema. Patients with chronic obstructive lung diseases have extensive collateral channels but these channels are not found in neonates. According to the hypothesis of Mackenzie et al.[16] and Webster et al.[17], TRIO may produce efficient gas exchange during CPR in patients with chronic obstructive lung diseases, but this may not be applicable to neonates.

A flow rate of 2 L/kg/min was used for TRIO in the present study. Despite this high flow rate, there was no significant difference between TRIO and IPPV in peak airway pressure values. Although pulmonary barotrauma was observed in no dogs during this experiment, a high flow TRIO has an inherent risk of barotrauma. Avoiding excessive elevations of airway pressure is a key factor in preventing pulmonary barotrauma[19,20].

Since compliance of the human chest wall is less than that of dogs[21], the peak airway pressure may reach dangerous levels if the same high flow rate is used in humans. The potential risk of barotrauma can be minimized by reducing the flow rate to the lowest required level during TRIO. In the present study, TRIO at a flow rate of 2 L/kg/min combined with external cardiac compressions at a rate of 60–80/min, resulted in hypocarbia. Since CO_2 elimination increases with an increasing flow rate of TRIO[3], a lower flow rate than 2 L/kg/min may suffice for sustaining normocarbia during CPR. The new standards and guidelines for CPR in the U.S.A.[1] recommend that the external cardiac compression rate should be increased to 80–100/min. The higher the compression rate, the more CO_2 can be eliminated. Therefore, it may be that the flow rate of TRIO can be further reduced when the compression rate is increased to 80–100/min.

The major objective of CPR is to provide oxygen to the brain and other vital organs until satisfactory cardiopulmonary function is restored[1]. To prevent irreversible cerebral damage, CPR must be started immediately. However, in cases where IPPV is not feasible, whether because of upper airway abnormalities or a lack of ventilator equipment, a substitute for IPPV is required. Since TRIO is very simple and requires no special equipment except for a source of oxygen and a small caliber catheter, we believe TRIO will prove to be a promising substitute for IPPV in various emergency situations.

In conclusion, we have shown that TRIO at a flow rate of 2 L/kg/min can maintain adequate arterial oxygenation and hypocarbia during external cardiac compressions in dogs under VF. No pulmonary barotrauma was observed. We postulate that this efficient gas transport may be attributed to the effects of combining TRIO with external cardiac compressions. When IPPV is not feasible, TRIO combined with external cardiac compressions should prove a promising

substitute for IPPV during CPR.

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