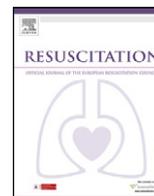




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Resuscitation

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Experimental paper

Impairment of carotid artery blood flow by supraglottic airway use in a swine model of cardiac arrest[☆]

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ARTICLE INFO

Article history:

Received 27 September 2011

Received in revised form 24 February 2012

Accepted 20 March 2012

Available online xxx

Keywords:

Oropharynx

Pharynx

Intubation

Laryngeal masks

Intubation

Cardiopulmonary resuscitation

Supraglottic airway

ABSTRACT

Objective: Supraglottic airway devices (SGDs) are often used as an alternative to endotracheal tube (ETT) during cardiopulmonary resuscitation (CPR). SGDs can be inserted 'blindly' and rapidly, without stopping compressions. These devices utilize pressurized balloons to direct air to the trachea and prevent esophagus insufflation. We hypothesize that the use of a SGD will compress the carotid artery and decrease carotid blood flow (CBF) during CPR in pigs.

Methods: Ventricular fibrillation (VF) was induced in 9 female pigs (32 ± 1 kg) followed by 4 min without compressions. CPR was then performed continuously for 3–6-min intervals. During each interval, an ETT was used for the first 3 min, followed by 3 min of each SGD (King LTS-D™, LMA Flexible™, Combitube™) in a random order. The primary endpoint was mean CBF (ml/min). Statistical comparisons among the 4 airway devices were performed by Wilcoxon Rank test. Post mortem carotid arteriographies were performed with SGDs in place.

Results: CBF (median ml/min; 25/75 percentile) was significantly lower with each SGD [King (10; 6/41), LMA (10; 4/39), and Combitube (5; –0.4/15)] versus ETT (21; 14/46) ($p < 0.05$ for each SGD compared with ETT). Arteriograms showed that with each SGD there was compression of the internal and external carotid vessels.

Conclusion: The use of 3 different SGDs during CPR significantly decreased CBF in a porcine model of cardiac arrest. While the current study is limited to pigs, the findings suggest that further research on the effects of SGD use in humans and the effects on carotid artery blood flow is warranted.

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1. Introduction

For many years endotracheal intubation (ETI) has been the gold standard for ventilation by advanced life support personnel during cardiopulmonary resuscitation (CPR).¹ Recently, supraglot-

tic airway devices (SGDs) have been promoted as an alternative to endotracheal intubation for patients undergoing CPR.¹ The supraglottic airway devices can be placed 'blindly' and rapidly, without the need to stop chest compressions.^{2,3} The benefits of not stopping chest compressions during CPR have been well recognized, and consequently, supraglottic airway devices have become popular, especially for patients with out-of-hospital cardiac arrest.⁴

Despite this recent shift in airway management practice, little is known about the potential effects of supraglottic airways on cardiovascular hemodynamics.⁵ Anatomically, the carotid arteries track inside a relatively non-distensible sheath in the median portion of the neck, adjacent to the laryngeal and pharyngeal space. As such, any increase in pressure in the retropharyngeal space could be theoretically transmitted to the carotid arteries and reduce carotid blood flow. In this study we examined the hypothesis that use of a supraglottic airway adjuncts in pigs undergoing CPR will result

[☆] A Spanish translated version of the abstract of this article appears as Appendix in the final online version at <http://dx.doi.org/10.1016/j.resuscitation.2012.03.025>.

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in compression of the carotid arteries, thereby reducing cerebral blood flow.

2. Methods

The study was approved by the Institutional Animal Care Committee of the Minneapolis Medical Research Foundation of Hennepin County Medical Center.

2.1. Surgical preparation

Nine female pigs (32 ± 1 kg) were used for the hemodynamic study. The anesthesia, surgical preparation, data monitoring, and recording procedures used in this study have been previously described.⁶ The initial sedation was achieved with intramuscular ketamine (7 ml of 100 mg/ml; Ketaset; Fort Dodge Animal Health, Fort Dodge, IA) followed by isoflurane and propofol infused at 160 μ g/kg/min. While spontaneously breathing but sedated, each pig was intubated with a size 7.5 endotracheal tube. Once apneic, they were treated with positive pressure ventilation with room air (21% of FiO_2). While sedated and mechanically ventilated, a burr hole was created in the cranium halfway between the left eyebrow and the posterior bony prominence of the skull. An intracranial pressure transducer (Camino, IntraLife Sciences, Plainsboro, NJ) was placed through the burr hole. Real-time intracranial pressures were recorded continuously. The left common carotid artery was then surgically exposed through a 2 cm incision and a Doppler flow probe (Transonic 420 series multichannel; Transonic Systems, Ithaca, NY) was placed as distal as possible to quantify common carotid blood flow. Femoral artery cannulation was performed and aortic pressures were measured at the level of the diaphragm with a pressure transducer (Mikro-Tip Transducer; Millar Instruments, Houston, TX). Central venous pressure was similarly measured in the right atrium. Animals received an intravenous heparin bolus (100 U/kg). The animals were then ventilated with $\text{FiO}_2 = 0.21$ using a volume-control ventilator (Narkomed 2A; Dräger Medical, Telford, PA) with a tidal volume of 10 ml/kg and a respiratory rate adjusted to maintain end tidal CO_2 (ETCO_2) between 35 and 42 mmHg. Airway pressure was measured continuously, as a surrogate for intrathoracic pressure, with a micromanometer-tipped catheter positioned at the junction of the anesthesia circuit and either the endotracheal tube or supraglottic airway. Nostril plugs were inserted in the nose. Surface electrocardiographic recordings were also acquired continuously. All data were recorded with a digital recording system (BIOPAC MP 150, BIOPAC Systems, Inc., CA, USA). ETCO_2 , tidal volume, minute ventilation, and blood oxygen saturation were continuously measured with a respiratory monitor (CO_2 SMO Plus; Novamatrix Medical Systems, Wallingford, CT).

2.2. Measurements and recording

All the variables (aortic, right atrial, airway, intracranial, coronary perfusion pressures, and common carotid blood flow) were measured continuously but analyzed during the last minute of each intervention (for every airway tube) described in the protocol below. Coronary perfusion pressure during CPR was calculated as described previously.⁶ Four consecutive decompression measurements before the delivery of positive pressure ventilation were averaged. These measurements were repeated three times within each minute studied, and the average of the three mean values was reported as the mean coronary perfusion pressure during this minute. Common carotid blood flow was calculated by numerically integrating values for the antegrade minus the retrograde flow recorded over 1 min.

2.3. Experimental protocol

The experimental protocol was designed to compare the effect of three different supraglottic airway devices versus an endotracheal tube. Upon completion of the surgical preparation, when oxygen saturation was $>95\%$ and ETCO_2 was stable between 35 and 42 mmHg for 15 min, ventricular fibrillation was induced by delivering direct current through a temporary right ventricular pacing wire. Ventilation and propofol administration were discontinued. After 4 min of untreated ventricular fibrillation, closed-chest standard CPR was performed 100 compressions/min with a 50% duty cycle and a compression depth 25% with a pneumatically driven automatic piston device (Pneumatic Compression Controller; Ambu International, Glostrup, Denmark), as previously described.⁷ The anterior chest wall was allowed to recoil passively but completely. During CPR, ventilation was performed with a resuscitator bag at $\text{FiO}_2 = 1$ with a tidal volume of approximately 10 ml/kg at a rate of one positive pressure ventilation every 10 compressions. An impedance threshold device ITD (ResQPOD 10 cm H_2O , Advanced Circulatory Systems, Roseville, MN) was used to assure that there was an adequate seal between each of the airway adjuncts studied and the trachea or supraglottic tissues.⁸ The seal was assessed by measuring the decompression phase airway pressure and in all studies an impedance threshold device (ITD) was used for seal validation. Only studies that demonstrated an intact seal during the decompression phase of CPR were used for analysis.

The protocol compared the physiological effects of the three different supraglottic airway devices versus an endotracheal tube and an open airway. Ventricular fibrillation was induced when an endotracheal tube was in place and 4 min later CPR was performed with the endotracheal tube in place. After 4 min of closed-chest compressions, the endotracheal tube balloon was deflated, and rapidly removed. Using a computer-generated randomization sequence, one of three supraglottic airway adjuncts (King LTS-DTM, LMA FlexibleTM, Combitube 41 FTM) was rapidly inserted and inflated per the manufacturer's instructions for use. Specifically, a King LTS-D Size 4 (King Systems, Noblesville, IN, USA) was introduced into the esophagus and the balloons were inflated to a pressure of 43–50 mmHg. The single-use LMA Flexible Size 4 (Laryngeal Mask Company Ltd., Jersey, Channel Islands, UK) was introduced into the larynx and inflated with 20 ml before insertion as recommended in some studies.^{9,10} The esophageal-tracheal Combitube 41 F (Tyco Healthcare Group LP, Mansfield, MA, USA) was inserted into the trachea and the proximal balloon was inflated with 100 ml and then the distal balloon was inflated with 15 ml per manufacturers' recommendation. Correct placement of all devices was confirmed by pulmonary auscultation and with ETCO_2 . The airway pressure manometer was connected to the junction of each airway device and the resuscitator bag. CPR was performed in 3 min intervals, first with an endotracheal tube and then with a supraglottic airway, per the randomization protocol. After each supraglottic airway intervention, the endotracheal tube was inserted again for 3 min to obtain new baseline measurements since the primary comparison was always between the parameters measured with the endotracheal tube in place and the measurements recorded immediately thereafter with the next supraglottic airway device. The volume used to inflate the endotracheal tube was 10 ml. Thus, each paired intervention included 3 min of CPR with an endotracheal tube and 3 min of CPR with a supraglottic airway. The measurements were made in the last minute of each intervention.

Each time one of the four different devices was removed, the airway was unprotected for 5 s before starting the introduction of the next device. The airway was always removed following the same procedure: the ITD was removed, the balloon of the airway device was deflated, the airway device was removed, airway was left unprotected for 5 s, the next airway device was introduced, its'

Table 1

Values are shown as median; 25/75 percentile. CBF: carotid artery blood flow (ml/min) with an ET tube and three supraglottic devices during CPR in pigs.

	Systolic CBF	Diastolic CBF	Mean CBF
ET tube	141; 78/356	-55; -148/-37	21; 14/46
Combitube	40; 7/182*	-12; -57/-3*	5; -0.4/15*
King tube	146; 55/290*	-58; -162/-30	10; 6/41*
LMA	73; 43/187*	-34; -85/-20	10; 4/39*
Open airway	141; 76/289	-50; -146/-36	24; 12/41

* $p < 0.05$.

balloon was inflated, the ITD was set in position, and ventilation was restarted. Carotid blood flow was assessed for each device in the last minute of the 3 min interval and then it was compared to the carotid blood flow measured in the 5 s period immediately after its removal and before the insertion of the next device. Measurements of carotid blood flow were made during the 5 s period of time as a way to assess the key physiological parameters with an open airway. The supraglottic airway balloon pressures were measured in all airway devices while balloons were inflated. When the protocol was completed, the pigs were sacrificed with a 10 ml injection of 10 M potassium chloride.

2.4. Radiological imaging

Twelve euthanized female pigs (31 ± 1 kg) were used to assess the anatomical effects of the supraglottic airway devices by post mortem carotid arteriography. Bilateral carotid arteriography was performed within 5 min of sacrificing the pig. While still anticoagulated from the heparin, sheaths were placed in the proximal common carotid artery at the point of exit from the thoracic cage. Radiographic images were obtained in each pig first with the endotracheal tube in place and then with one of the three supraglottic airway devices using 4 ml of contrast (Optiray 350, Tyco Healthcare Mallinckrodt, Gosport, Hampshire, UK) injected simultaneously into each carotid artery. The supraglottic airway devices were inflated as they had been in the living pigs as described above. Standard skull film (Fuji IP Cassette Type CC, Fuji Photo Film Co., Ltd., Tokyo, Japan) X-ray settings were used (62 mV and 2.5 mA) and image was processed digitally (Fujifilm FCR XG 5000). A total of 4 animals per supraglottic device were studied post mortem.

2.5. Statistical analysis

All values with a normal distribution are expressed as mean \pm SD, values with a non-normal distribution are expressed as median; 25/75 percentile. The primary end point, determined *a priori*, was common carotid blood flow. Each parameter measured with the supraglottic airway in place was compared with data recorded from the immediately preceding interval using an endotracheal tube. The Wilcoxon Rank test was used for comparison since the data did not pass the normality test. A p value < 0.05 was considered statistically significant. Statistical analyses were performed with SPSS® Statistics 17.0.

3. Results

The goal of our study was to compare carotid blood flow during CPR with an endotracheal tube and three different supraglottic airways. There were statistically significant reductions in carotid blood flow with all supraglottic airway adjuncts when compared with an endotracheal tube, as shown in Table 1 and Figs. 1 and 2. As shown in the representative tracing from one study in Fig. 1, the changes in carotid blood flow occurred instantaneously with placement and removal of each airway adjunct. A 3 min duration was selected for each supraglottic airway application such that

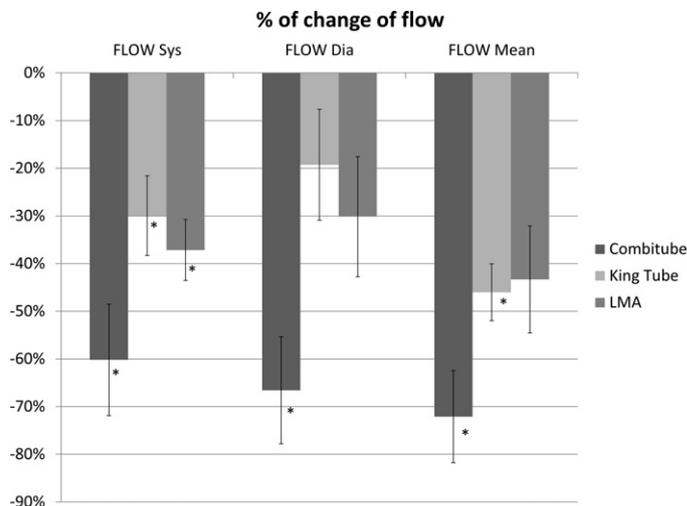


Fig. 1. Percentage of change of carotid blood flow compared to an ET tube with each of the supraglottic devices during CPR, * $p < 0.05$.

all four devices could be used in each animal. This decision was based in part on pilot studies showing that the effect of each of the three supraglottic airway devices and the endotracheal tube on carotid blood flow remained constant from as soon as they were inflated for up to 20 min of CPR. Carotid blood flow measured during CPR in the absence of any airway device was not different than the carotid flow measured with an endotracheal tube in place (Table 1). However, as with the endotracheal tube data shown in Table 1, each time the supraglottic airway device was removed and prior to placement of an endotracheal tube, carotid blood flows instantaneously increased to the same levels observed prior to placement of the supraglottic airway devices, and equal to that seen with the endotracheal tube in place (Fig. 3). No significant differences were observed between the endotracheal tube and the supraglottic airways devices when comparing airway pressures (Table 2), aortic, intracranial, coronary perfusion pressures, or $ETCO_2$.

All airway adjuncts were used following the instructions for use. The Combitube pharyngeal balloon was inflated, for example, with 100 ml of air. When inside the supraglottic space, the Combitube balloon pressure was found to be > 86 mmHg. A similar approach was used for the other supraglottic airway devices. The King LTS-D pressure was 43 ± 2 mmHg, and the LMA pressure was 35 ± 2 mmHg.

Radiographic images obtained when performing bilateral carotid arteriograms are shown in Fig. 4. A qualitative assessment of the X-rays showed that with insertion and inflation of each of the three supraglottic airway devices, there was direct anatomical compression of the internal and external carotid vessels.

4. Discussion

Results from this study in pigs suggest that there may be unanticipated consequences of using supraglottic airway devices in the management of patients in low-flow states such as cardiac arrest. The results support the hypothesis that use of supraglottic devices has the potential, at least in pigs, to impair carotid blood flow during CPR when there is already a decrease in vital organ perfusion. The mechanism appears to be secondary to an increase in supraglottic pressure with compression of the carotid arteries. A marked decrease in carotid blood flow during CPR in pigs was observed with three different supraglottic airway devices when compared with an endotracheal tube or an open, unprotected airway. Additional confirmation of the negative effects of the supraglottic airway devices

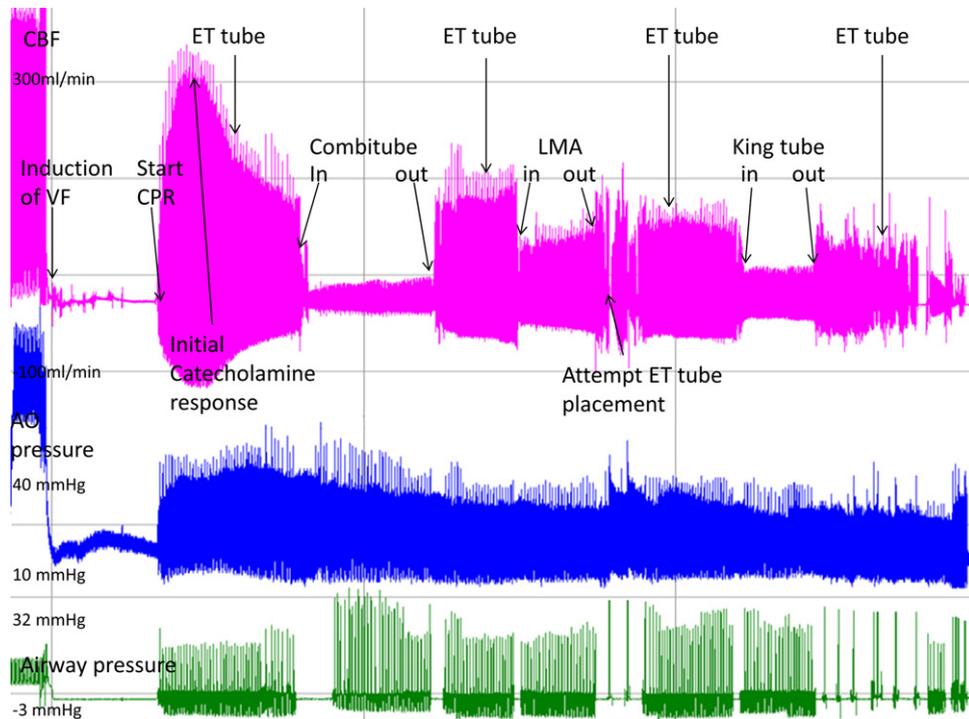


Fig. 2. Real time tracing during one experiment. Pink: carotid blood flow (CBF); blue: thoracic aortic pressure (AO pressure); green: airway pressure. The figure shows that every time a supraglottic airway device is used carotid blood flow significantly decreases without any differences in aortic pressure. Ventricular fibrillation (VF), cardiopulmonary resuscitation (CPR). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of the article.)

in pigs was demonstrated by carotid angiography post mortem, demonstrating internal compression and obstruction of contrast iodine flow only in the presence of the supraglottic airways. The consistency of the aortic pressure associated with an immediate

rise of carotid blood flow after each of the supraglottic devices were removed demonstrated that the reduction of carotid artery flow was not caused by a change in chest compressions. While these findings are provocative they should not be used as a justification

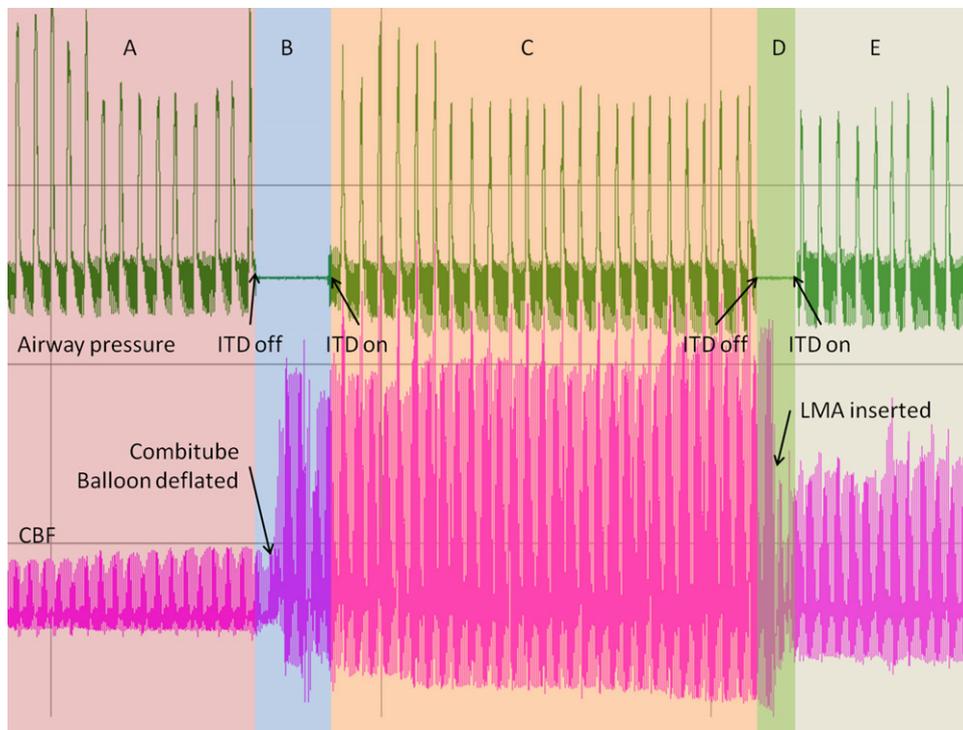


Fig. 3. Real time tracing of the transition from a supraglottic device to an endotracheal (ET) tube and from an ET tube to a supraglottic device. A: Combitube; B: transition to an ET tube; C: ET tube; D: transition to a supraglottic device; E: LMA. Pink: carotid blood flow (CBF); green: airway pressure (AP). The figure shows that when the balloon of a supraglottic device is deflated the carotid blood flow immediately increases while the airway is left in its physiological state and before insertion of an ET tube (B); when an ET tube is removed carotid blood flow does not change (C); and when a supraglottic device is inserted and its' balloon is inflated carotid blood flow decreases (D). Impedance threshold device (ITD).

Table 2

Hemodynamic parameters measured during CPR. Values are shown as mean ± SD and in mmHg. AP: airway pressure; Ao Sys: systolic aortic pressure; Ao Dia: diastolic aortic pressure; Ao Mean: mean aortic pressure; RA Dia: diastolic right atrial pressure; ICP max: maximum intracranial pressure; ICP min: minimum intracranial pressure; ETCO2 mean: mean end tidal CO₂; CoPP: coronary perfusion pressure.

	AP max	AP min	Ao Sys	Ao Dia	Ao Mean	RA Dia	ICP max	ICP min	ICP Mean	ETCO2 Mean	CoPP
ET tube	1.7 ± 0.5	-6.1 ± 1.4	42 ± 21	13 ± 8	24 ± 53	-1 ± 2.4	28 ± 15	10 ± 9	18 ± 6	17 ± 8	9 ± 10
Combitube	2.0 ± 0.7	-5.3 ± 2.1	48 ± 23	15 ± 9	27 ± 13	-2 ± 4	31 ± 14	13 ± 3	19 ± 6	19 ± 9	10 ± 12
King tube	1.9 ± 0.7	-5.2 ± 2.1	40 ± 13	15 ± 4	23 ± 8	-0.8 ± 1.5	30 ± 8	13 ± 4	19 ± 5	18 ± 5	10 ± 4
LMA	2.2 ± 0.5	-5.2 ± 1.5	40 ± 28	16 ± 10	24 ± 16	1 ± 0.7	21 ± 7	10 ± 2	16 ± 3	14 ± 5	9 ± 10

to change current patient care protocols but rather to encourage further consideration and research.

Prior studies in humans provide additional data supportive of the hypothesis that the supraglottic airways compress the carotid arteries during CPR, even when used properly. Colbert et al. showed that even during routine anesthesia, carotid blood flow is decreased with a LMA.¹¹ This effect was most pronounced in elderly patients where forward flow was decreased by 11% when compared with deflation of the cuff in the same patient. Both the carotid artery cross sectional area and flow through the vessel were significantly reduced in that study, when comparing these parameters during cuff inflation with post deflation values. However, an echographic study by Nandwani et al. found no compression of the carotid artery or internal jugular vein in humans when LMA was used.¹² We speculate that in the setting of low flow, which is universally the case during CPR, the reduction in carotid diameter and flow, may be clinically important. In addition, studies in human cadavers have examined the force required to dislodge different airway devices: forces (median, IQR) required to dislodge

the various airway adjuncts varied from 12.5 to 28.3 lbs.¹³ Another radiographic study in human cadavers demonstrated that LMA inflation resulted in an anterior shift of the thyroid, arytenoids, and cricoid cartilages.¹⁴ Thus, the outward forces are substantial and we speculate may also result in compression of the more compliant vasculature that is also contiguous to the LMA cuff. One study showed the outward pressure generated by the Combitube was 188 ± 118 mmHg.¹⁵ In the setting of cardiac arrest and CPR, the magnitude of these pressures may well have significant impact on carotid blood flow.¹⁵⁻¹⁷

There are several important limitations of this study. First and most importantly, human and porcine anatomies are different, the supraglottic airway devices were designed for humans not pigs, and there are not any inflation parameters for the supraglottic devices for the pigs. In this study we chose to use the flexible LMA due to the anatomy of the pig. While human airways feature some curvature, swine airways are linear. Using an alternative LMA device would have limited our ability to facilitate insertion as compared to the flexible LMA due to the rigidity of the device's

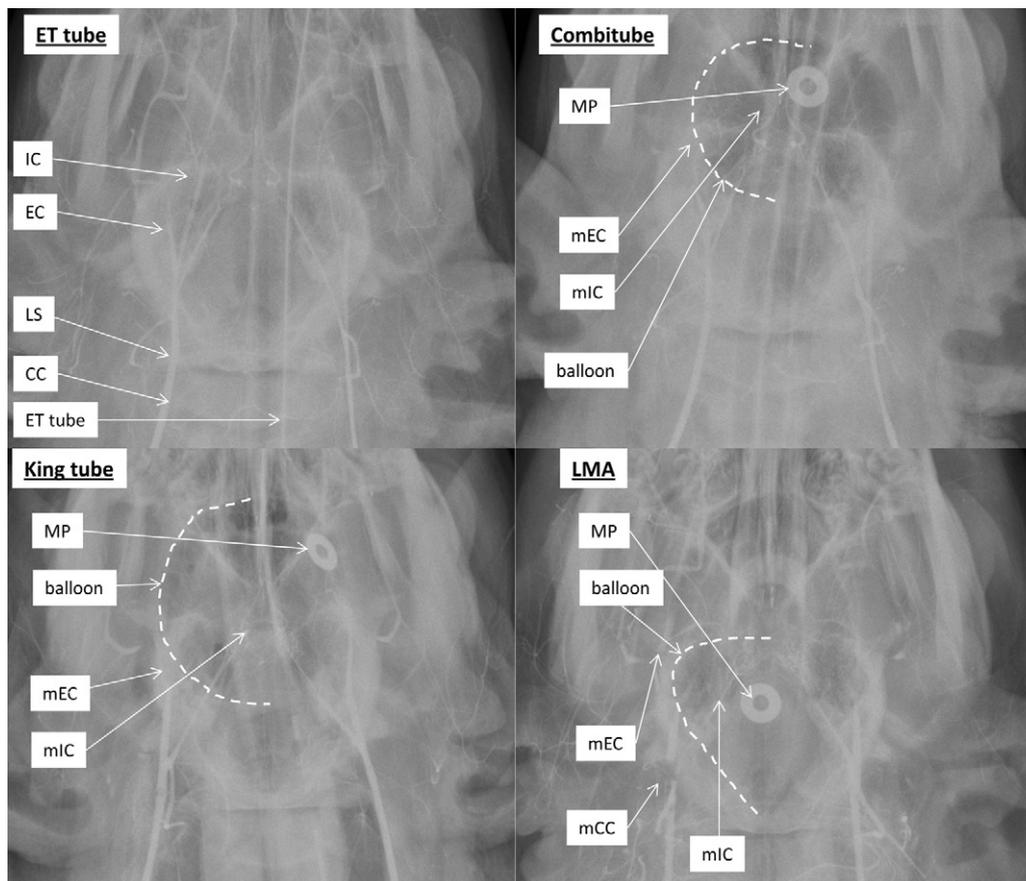


Fig. 4. The X-rays show the three supraglottic airway devices are responsible for a direct anatomical compression of the internal and external carotid vessels. IC: internal carotid artery; EC: external carotid artery; CC: common carotid and sheath; LS: limit of the sheath inside the common carotid artery; ET tube: endotracheal tube, balloon not visible because it is farther in the trachea; MP: metallic plunger glue on the balloon of the Combitube, King tube and LMA; balloon: white dash line, right limits of the balloon of the Combitube, King tube and LMA; mCC: missing right common carotid artery; mEC: missing right external carotid artery; mIC: missing right internal carotid artery.

design. The compression cuff is the same for all LMA models. However, the primary purpose of the current study was to determine whether it was theoretically possible to reduce carotid blood flow during CPR by outward compression from a supraglottic airway device. The data are clear; at least in pigs inflation of one of several different kinds of supraglottic airway device can substantially reduce carotid blood flow. Whether this phenomenon occurs in humans is unknown but our data suggest that further human studies on the potential impact of this family of devices on blood flow to and from the brain during CPR is warranted. Importantly, our intent was not to evaluate which supraglottic airway device may cause the greatest reduction of carotid blood flow in pigs, as they were designed for human use, but rather to determine if supraglottic airway adjuncts, as a class of technologies, could alter carotid blood flow during CPR. Furthermore those results may not apply to other supraglottic airway devices. Second, we did not measure the effect of the supraglottic airway adjuncts on jugular venous pressures. Knowing that venous structures are more likely to become compressed from external pressure than arterial structures, it is reasonable to postulate that supraglottic devices may cause jugular venous compression thereby further reducing cerebral perfusion by causing cerebral outflow obstruction. Third, we did not measure cerebral perfusion pressure, which would have necessitated placement of a carotid artery pressure transducer probe distal to the point of potential compression by the supraglottic airway devices and the transducer itself may have altered blood flow and thus cerebral arterial pressure. Further, no direct blood flow measurements with microspheres to the brain were performed. Future studies are needed in this regard. Fourth, because in most cases, the resuscitation team does not control the cuff pressure during the first minutes of cardiopulmonary resuscitation we choose to inflate the balloons per the manufacturer's instructions for use without further adjustment based upon the cuff pressure.

The data from this study that further study on the potential hemodynamic impact of supraglottic airway devices in humans undergoing CPR is warranted. The authors recognize that supraglottic airway devices have the advantages of rapid advanced airway management that allows for uninterrupted chest compressions per American Heart Association standards, asynchronous ventilations, and some protection against aspiration. However, the current study demonstrates that at least in a pig model of cardiac arrest, the advantages of supraglottic airways that were designed for humans comes at the price of a significant reduction in carotid blood flow.

5. Conclusion

In pigs undergoing CPR, use of supraglottic airway device originally designed for human use was associated with a significant reduction in carotid blood flow when compared with an endotracheal tube. These new findings in pigs suggest that further research on the effects of supraglottic airway device use in humans and their potential effects on carotid artery blood flow is warranted.

Conflict of interest statement

None.

Contributors

All authors have participated to the conception, design and writing of this manuscript.

Funding source

The study was funded by an Institutional, Division of Cardiology grant at the University of Minnesota to Dr. Yannopoulos.

Ethical approval

The study was approved by the Institutional Animal Care Committee of the Minneapolis Medical Research Foundation of Hennepin County Medical Center, and all animals received treatment in compliance with the National Research Council's 1996 Guide for the Care and Use of Laboratory Animals.

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