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Rescue Ventilation Through a Small-Bore Transtracheal Cannula in Severe Hypoxic Pigs Using Expiratory Ventilation Assistance

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BACKGROUND: Suction-generated expiratory ventilation assistance (EVA) has been proposed as a way to facilitate bidirectional ventilation through a small-bore transtracheal cannula (TC). In this study, we investigated the efficiency of ventilation with EVA for restoring oxygenation and ventilation in a pig model of acute hypoxia.

METHODS: Six pigs (61–76 kg) were anesthetized and ventilated (intermittent positive pressure ventilation) via a cuffed endotracheal tube (ETT). Monitoring lines were placed, and a 75-mm long, 2-mm inner diameter TC was inserted. After the baseline recordings, the ventilator was disconnected. After 2 minutes of apnea, reoxygenation with EVA was initiated through the TC and continued for 15 minutes with the ETT occluded. In the second part of the study, the experiment was repeated with the ETT either partially obstructed or left open. Airway pressures and hemodynamic data were recorded, and arterial blood gases were measured. Descriptive statistical analysis was performed.

RESULTS: With a completely or partially obstructed upper airway, ventilation with EVA restored oxygenation to baseline levels in all animals within 20 seconds. In a completely obstructed airway, $Paco_2$ remained stable for 15 minutes. At lesser degrees of airway obstruction, the time to reoxygenation was delayed. Efficacy probably was limited when the airway was completely unobstructed, with 2 of 6 animals having a Pao_2 <85 mm Hg even after 15 minutes of ventilation with EVA and a mean $Paco_2$ increased up to 90 mm Hg.

CONCLUSIONS: In severe hypoxic pigs, ventilation with EVA restored oxygenation quickly in case of a completely or partially obstructed upper airway. Reoxygenation and ventilation were less efficient when the upper airway was completely unobstructed. (Anesth Analg 2015;120:890–4)

In a "cannot intubate, cannot oxygenate" (CICO) situation, a percutaneous cricothyroidotomy should be performed rapidly to restore oxygenation and avoid brain damage or death.[1,2] This life-saving procedure can be performed with either a small-bore (inner diameter [ID] ≤2 mm) or a (cuffed) wide-bore cannula (ID = 4 mm or larger).[3] Survey studies suggest that a majority of anesthesiologists prefer the use of a small-bore cannula technique when performing an emergency cricothyroidotomy[4,5] because insertion might be easier and less traumatic. The insertion of a small-bore catheter through the cricothyroid membrane can be performed quickly.[6] Providing effective reoxygenation and ventilation, however, through such a narrow cannula may be difficult[7,8] and potentially risky. Because resistance to gas flow is inversely related to the ID, a high-pressure oxygen source is needed to generate adequate flow through a small-bore cannula.[9] In addition, passive expiratory outflow through a small-bore airway cannula is limited,[10] so egress of gas must take place through the upper airway. Obstruction of the upper airway (e.g., because of edema, laryngospasm, or tumor) or insufficient expiratory time can result in air trapping. Several reports of barotrauma and circulatory collapse resulting from high-pressure oxygen source ventilation can be found in the literature.[11–14]

Techniques proposed to facilitate the egress of gas through a small-bore cannula include applying thoracic and abdominal compression, inserting an additional cannula,[15,16] or applying suction to the airway cannula during the expiratory phase.[17,18] We have previously described a manually operated device (DE 5) that uses high-velocity gas flow to create suction by the Bernoulli effect during expiration. The DE 5 establishes not only flow-controlled inspiration but is also capable of actively supporting expiration (expiratory ventilation assistance [EVA]) (Fig. 1, A and B).[19] In an artificial lung model, this emergency ventilation device achieved up to 8.3 $L \cdot min^{-1}$ of minute volume through a 2-mm ID transtracheal cannula (TC) with complete outflow obstruction.[20] Although EVA in vitro appears promising, evaluation in vivo is mandatory. The primary aim of the present explorative study was to determine the efficiency of EVA regarding reoxygenation in an acute hypoxic pig model with an obstructed upper airway. In addition, we studied the influence of upper airway patency on reoxygenation and ventilation with EVA.

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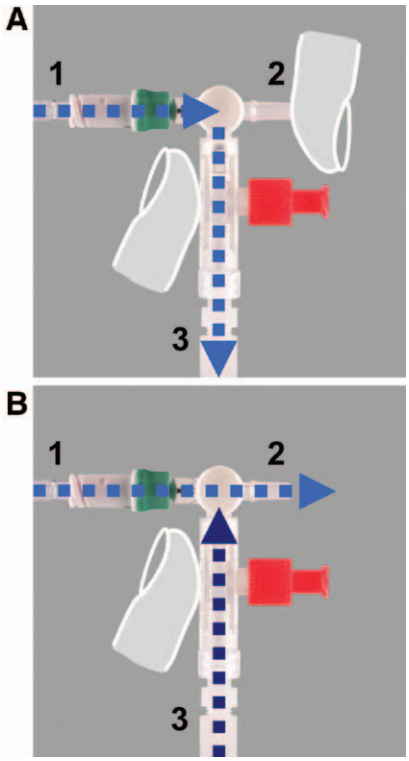

Figure 1. The two functional modes of the DE 5. A, Device activated, insufflation: oxygen flows from the inlet (1) to the connecting tubing (3). The outlet (2) is occluded by finger. B, Device activated, expiratory ventilation assistance: oxygen flows from the inlet (1) to the outlet (2) entraining gas from the connecting tubing (3) by the Bernoulli effect.

METHODS

Experimental Set-Up

The study was approved by the local Animal Welfare Committee (DEC number 2009–070). After overnight fasting with free access to water, 6 pigs (61–76 kg) were premedicated with IM tiletamine/zolazepam (6 mg·kg^{-1}) and atropine (0.05 mg·kg^{-1}). Anesthesia was induced with 4 to 8 mg·kg^{-1} propofol and 1 μg·kg^{-1} sufentanil via an IV catheter inserted in a vein in the ear of the animal. The trachea was intubated with an 9.0-mm ID cuffed endotracheal tube (ETT). The lungs of the pigs were mechanically ventilated via intermittent positive pressure ventilation (IPPV) with an fraction of inspired oxygen (F_{IO_2}) of 0.4, a tidal volume of 10 mL·kg^{-1}, and a respiratory rate adjusted to establish an end-tidal P_{aCO_2} of approximately 40 mm Hg. Anesthesia was maintained by continuous infusions of sufentanil (8 μg·kg^{-1}·h^{-1}), propofol (9 mg·kg^{-1}·h^{-1}), and pancuronium (0.3 mg·kg^{-1}·h^{-1}). A 9 Fr sheath (Arrow, Reading, PA) was inserted in the right jugular vein, and a pulmonary artery catheter (Edwards Lifesciences Corporation, Irvine, CA) was positioned. Both femoral arteries were cannulated with an 18 G arterial catheter (Arrow) for continuous arterial blood pressure monitoring and arterial blood sampling. The trachea was exposed surgically with a midline incision, and a 75-mm long, 2-mm ID TC (Emergency Transtracheal Airway Catheter; Cook Medical, Bloomington, IN) was inserted just below the tip of the ETT into the trachea between the third and fourth cartilage ring under bronchoscopic guidance. Intratracheal pressure was measured continuously via an epidural catheter flushed with saline, inserted via the tracheal tube, and positioned 2 cm above the carina with bronchoscopic guidance. All pressure catheters were connected via pressure transducers to a multichannel recorder and a digital data acquisition system (IDEEQ-system; University Maastricht, Maastricht, The Netherlands) and were recorded continuously throughout the experiment.

Part 1: Completely Obstructed Upper Airway

After stable baseline recordings and arterial blood gases for at least 30 minutes, the ventilator was disconnected, leaving the tracheal tube open to room air. After 2 minutes of apnea, the ETT was occluded by a plug to simulate a completely obstructed upper airway, and reoxygenation with EVA was initiated through the TC. EVA was applied with a manual ventilation ejector (DE 5; Fig. 1, A and B), connected to a pressure-compensated oxygen flowmeter set at 15 L·min^{-1}. By using a 30·min^{-1} rate, an initial inspiration/expiration (I/E) ratio of 1:1 was adjusted to keep the end-expiratory intratracheal pressure between 0 and 10 cm H_2O. Arterial blood samples were collected at baseline (before the apnea period of 2 minutes), at the start of ventilation with EVA (0), and after 10, 20, 30, 60, 180, 300, 600, and 900 seconds. After 15 minutes EVA was stopped, and the lungs of the pigs were mechanically ventilated through the ETT. After completion of the experiment, a bronchoscopic evaluation of the airway was performed.

Part 2: Increased Upper Airway Patency

In the second part of the study, the experiment was repeated with varying degrees of tracheal tube obstruction simulating different levels of upper airway patency. After stable baseline recordings for 30 minutes, the mechanical ventilator was again disconnected, and after a 2-minute apneic period, ventilation with EVA was initiated in random order with different degrees of ETT obstruction: left open to room air, partially obstructed via a capping device with either a 3-mm orifice or a 50-mm long, 2-mm ID tubing, or completely patent. EVA was applied with the DE 5 at a rate of 30·min^{-1} with an I/E ratio of 1:1. Arterial blood samples were collected at baseline (before the apneic period), at the start of ventilation with EVA (0), and after 10, 20, 30, 60, 180, 300, 600, and 900 seconds. The lungs of the animals were ventilated mechanically, and all monitored pressures and blood gas values were allowed to return to normal between experimental runs. In addition, between each trial, a bronchoscopic evaluation of the trachea was performed. Upon completion of the experiments, the pigs were euthanized with pentobarbital (150 mg·kg^{-1}), and 4 pigs underwent sternotomy to allow macroscopic examination of the lungs.

Statistical Analysis

Our study was designed as a pilot trial for EVA, a new concept of ventilatory support. On the basis of the absence of previous comparable in vivo reoxygenation trial(s), the animal welfare committee provided permission for the use of 6 animals.

Descriptive statistical analysis was performed, and the data are presented as median [range]. A 99% confidence limit for the binomial probability was calculated according to the Clopper-Pearson method. This method is exact for 6/6 animals.[21]

RESULTS

At baseline, the minute volume during IPPV to achieve normocapnia was 9.9 [9.1–12.0] L·min^{-1}. The mean compliance at baseline was 30.5 [28.1–33.8] mL·cm H_2O^{-1}.

Part 1: Completely Obstructed Upper Airway

After 2 minutes of apnea, the Pao_2 decreased to a median of 25 [20–31] mm Hg, and the oxygen saturation was 45 [33–60] % (Table 1). Within 20 seconds after the initiation of EVA, hypoxemia was corrected (arterial oxygen saturation >95%) in all 6 animals (lower 99% confidence limit = 46%). During the apnea period, $Paco_2$ increased to a median of 54 [46–56] mm Hg, and did not change significantly during ventilation with EVA.

Part 2: Increased Upper Airway Patency

With the ETT, partially obstructed EVA also restored oxygenation within 20 seconds (arterial oxygen saturation >95%). When the airway was left completely open, however, 2 of 6 animals had a Pao_2 <85 mm Hg after 15 minutes. The efficacy of EVA decreased as the ETT was less obstructed, resulting in protracted reoxygenation and severe hypercarbia with a completely open airway (Fig. 2, A–C).

Macroscopic Examination

Hyperemia of the posterior tracheal wall at the level of TC insertion was seen in all animals by tracheobronchoscopy. There were no mucosal lacerations, macroscopically evident edema, or endobronchial hemorrhagic secretions. Postmortem examination revealed no pneumothorax or subcutaneous emphysema.

DISCUSSION

EVA, applied by the DE 5, restored oxygenation through a small-bore TC in severely hypoxic pigs within 20 seconds with a completely as well as partially obstructed upper airway.

The exact amount and flow of oxygen necessary to rapidly reoxygenate an adult patient are unknown. Flack and Hardman[22] calculated in a mathematical model that an increase in tidal volume results in faster reoxygenation. The Difficult Airway Society guidelines recommend the use of a high-pressure device capable of delivering a high minute volume to reoxygenate a patient in a CICO situation after small-bore cricothyroidotomy.[23] In a previous in vitro study, the DE 5, driven by an oxygen flow of 15 L·min^{-1}, achieved a calculated minute volume of 7.1 L·min^{-1} through a TC in a lung model at a compliance of 30 mL·cm H_2O^{-1} (similar to that of the pigs in our study).[19] In an adult, this minute volume would not only be enough for swift reoxygenation but would also provide adequate ventilation. Although in our study hypercarbia could be limited during a period of 15 minutes in the animals with an obstructed upper airway, $Paco_2$ did not return to baseline levels during ventilation with EVA. A plausible explanation is that during IPPV the pigs required a minute volume of 9.9 L·min^{-1} for normoventilation (Table 1), which is a value far above that achievable with the DE 5.

In case of a completely obstructed upper airway and the application of EVA there is not only the risk of air trapping by overvigorous insufflation and inadequate expiration, but also the possibility of developing subatmospheric intratracheal pressure by prolonged active gas evacuation. In the present study the I/E ratio was slightly varied to keep intratracheal pressures between 0 and 10 cm H_2O. In clinical

Table 1. Pao_2 and $Paco_2$ at Complete Upper Airway Obstruction Before (−120 Seconds) and After 2 Minutes of Apnea (0) and Subsequent Ventilation with Expiratory Ventilation Assistance During 15 Minutes (Part 1 of the Study)

	−120 seconds	0 second	10 seconds	20 seconds	60 seconds	180 seconds	900 seconds
Pao_2 (mm Hg)	171 [156–200]	25 [20–31]	84 [47–208]	254 [178–310]	404 [381–416]	539 [494–562]	537 [490–565]
Sao_2 (%)	100	45 [33–60]	96 [82–100]	100	100	100	100
$Paco_2$ (mm Hg)	39 [37–40]	54 [46–56]	55 [48–60]	55 [50–62]	56 [49–59]	55 [51–60]	57 [56–75]

Data presented as mean [range].
Sao_2 = arterial oxygen saturation.

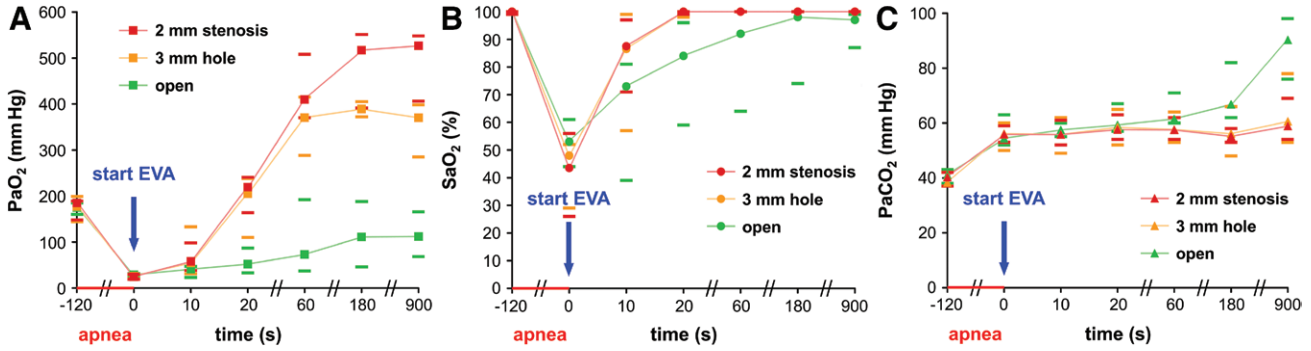

Figure 2. A, Course of Pao_2 at different upper airway patency before (−120 seconds) and after 2 minutes of apnea (0) and subsequent ventilation with expiratory ventilation assistance (EVA) during 15 minutes (part 2 of the study). The endotracheal tube (ETT) either fully open or obstructed with a 3-mm hole or a 50-mm long, 2-mm stenosis. Data presented as mean and range. B, Course of arterial oxygen saturation (Sao_2) at different upper airway patency before (−120 seconds) and after 2 minutes of apnea (0) and subsequent ventilation with EVA during 15 minutes (part 2 of the study). The ETT either fully open or obstructed with a 3-mm hole or a 50-mm long, 2-mm stenosis. Data presented as mean and range. C, Course of $Paco_2$ at different upper airway patency before (−120 seconds) and after 2 minutes of apnea (0) and subsequent ventilation with EVA during 15 minutes (part 2 of the study). The ETT either fully open or obstructed with a 3-mm hole or a 50-mm long, 2-mm stenosis. Data presented as mean and range.

resuscitation such intratracheal pressures are unlikely to be monitored. Attention to chest wall excursion and relaxation in the inspiratory and expiratory phases, respectively, may be the only monitor. However, in critical situations, modest subatmospheric end-expiratory pressure may be desirable and improve venous return and be beneficial for both cardiac and cerebral perfusion.[24] Finally, small-bore transtracheal catheters may become dislodged, leading to rapid development of subcutaneous emphysema and ineffective ventilation or oxygenation.

We found ventilation with EVA less effective in normalizing blood gases, when the ETT was completely patent. To achieve sufficient ventilation in such conditions, a high driving pressure is mandatory.[25] The driving pressure necessary for "classic" jet ventilation in an adult patient ranges between 1.5 and 3.0 bar (approximately 22 and 44 psi). Because the inspiratory pressure of the DE 5 measured in front of the TC is just approximately 110 mbar (approximately 1.6 psi) at 15 $L \cdot min^{-1}$,[19] we note that EVA is not just a "modified" type of jet ventilation. Flow-controlled EVA for ventilation through small-bore cannulas or catheters should instead be considered a "hybrid" mode filling in the gap between classic jet ventilation through a small-bore catheter using high pressures in an open airway and conventional low-pressure ventilation (e.g., IPPV) through a large-bore tube in an airway sealed by a cuff.

One of the limitations of our study design is that the insertion of the TC was performed under stable conditions after surgical exposure of the trachea. This study does not address the difficulty of successfully performing a cricothyroidotomy in a CICO situation, a challenge clearly described in the Fourth National Audit Project in the United Kingdom.[26] Furthermore, we continued ventilation with EVA for only 15 minutes, which simulated the time required to reoxygenate and stabilize a hypoxic patient after obtaining access to the airway by needle cricothyroidotomy. Possible effects of prolonged ventilation with EVA, however, such as damage to the respiratory mucosa because of cold, dry gas, have not been studied. The degree of upper airway patency was modeled with the use of fixed diameters, which is a simplification of clinical reality. Clinically, the diameter of the upper airway is dynamically variable and is likely influenced by the intratracheal pressures. This study, however, did not determine the influence of EVA on patency of the upper airway.

In summary, ventilation with EVA restored oxygenation quickly in severely hypoxic animals with a partially or completely obstructed upper airway. The efficacy of EVA decreased in an open airway.

DISCLOSURES

Name: Ankie E. Hamaekers, MD.
Contribution: This author helped to design and conduct the study, collect and analyze the data, and prepare the manuscript.
Attestation: Ankie E. Hamaekers approved the final manuscript, attests to the integrity of the original data and the analysis reported in this manuscript, and is the archival author.
Conflicts of Interest: Ankie Hamaekers is an unpaid consultant for Ambu and has received free samples of airway equipment for teaching and clinical evaluation from several companies. She has no financial interest in any company.

Name: Tim van der Beek, MD.
Contribution: This author helped to collect and analyze the data, and prepare the manuscript.
Attestation: Tim van der Beek approved the final manuscript, and attests to the integrity of the original data and the analysis reported in this manuscript.
Conflicts of Interest: This author has no conflicts of interest to declare.
Name: Maurice Theunissen, MSc.
Contribution: This author helped to analyze the data, and prepare the manuscript.
Attestation: Maurice Theunissen approved the final manuscript.
Conflicts of Interest: This author has no conflicts of interest to declare.
Name: Dietmar Enk, MD, PhD.
Contribution: This author helped to design and conduct the study, collect and analyze the data, and prepare the manuscript.
Attestation: Dietmar Enk approved the final manuscript, and attests to the integrity of the original data and the analysis reported in this manuscript.
Conflicts of Interest: Dietmar Enk is the inventor of the DE 5 and receives royalty payments from Dolphys Medical. The Maastricht University Medical Center also receives royalty payments from Dolphys Medical.
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