laryngeal mask group, a successful primary airway was established in 99% of patients on the first attempt, and in 1% of patients insertion failed at the second attempt. In agreement with the authors, the effective airway time was similar for the two devices and the oropharyngeal leak pressure was higher in the Soft Seal laryngeal mask group. The endoscopic score of the larynx was significantly better with the Soft Seal laryngeal mask group than with the Unique LMA group. The changes in cuff pressures and airway morbidity were similar in both groups.

In an observational study, we inserted the Soft Seal laryngeal mask with the cuff inflated at atmospheric pressure [3] in 100 patients and achieved 97% success in the first attempt and 3% in the second attempt. Regarding insertions, 85% were graded as very easy and 12% as easy and were achieved within 20 s. The mean intra-cuff pressure *in vivo* was 40 mmHg (53 cm $H_2O$). The leak pressure was at a mean pressure of 24.8 cm $H_2O$, in agreement with the authors. Only six patients complained of mild-to-moderate sore throat in the first 2 h after operation.

We agree with the authors' conclusion that the LMA Unique and the Soft Seal laryngeal mask are of equal clinical suitability. We believe that inserting the Soft Seal laryngeal mask with the cuff inflated at atmospheric pressure can achieve excellent conditions for insertion and removal.

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# Conditions involving release of pro-inflammatory cytokines predispose to ARDS

doi: 10.1017/S026502150700049X

EDITOR:

Sadis and colleagues [1] investigated risk factors for the development of ARDS in patients receiving multiple transfusions and found that it was not the number of transfusions but thoracic trauma and hypoxia that were associated with the subsequent development of ARDS. Patients who developed ARDS received significantly more fresh frozen plasma. Previous studies showed that septicaemia is an additional predisposing factor for transfusion-related ARDS [2]. Another condition commonly associated with pulmonary oedema during infusion of large amounts of intravenous fluids is diabetic ketoacidosis [3]. All these conditions with their different pathophysiology have in common the release of large amounts of cytokines including tumour necrosis factor (TNF) and interleukin-1 (IL-1). Transfusion of an anti-CD28 monoclonal antibody into human volunteers stimulated T-cells to release large amounts of these two cytokines and led to pulmonary oedema in all subjects of this trial [4]. The mechanism by which these cytokines lead to or predispose to pulmonary oedema has recently been clarified: Alveolar epithelial fluid clearance in pulmonary oedema is dependent on pulmonary epithelial sodium and chloride transport through the apical alveolar epithelial sodium channel and the cystic fibrosis transmembrane conductance

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Accepted for publication 12 March 2007 EJA 4369
First published online 7 June 2007

regulator (CFTR) chloride channel generating the osmotic gradient, which removes water through alveolar aquaporin channels and paracellular pathways from the alveolar air space [5]. TNF is a powerful down-regulator of alveolar epithelial sodium channel expression and was found to induce pulmonary oedema in various animal studies. IL-1 was found to reduce pulmonary alveolar epithelial sodium channel function and expression and sodium uptake in alveolar type II cells. IL-1 can also reduce pulmonary epithelial chloride transport by down-regulation of prostanoid receptors, which leads to a reduction in cyclic adenosine monophosphate (cAMP) and subsequently in cAMP-dependent CFTR function. Pulmonary oedema in meningococcal septicaemia has recently been associated with reduced systemic chloride channel function [6]. The hypoxia found in patients developing ARDS subsequently indicated a reduced fluid clearance associated with a direct cytokine effect preceding the development of ARDS. The association of ARDS with FFP administration may be related to the fact that TNF caused a coagulopathy [7] prompting the administration of FFP. Contributing to the predisposition to ARDS by inflammatory conditions may be the upregulation of P-selectins on vascular endothelial cells, which facilitates the adhesion of neutrophils in the pulmonary circulation and their subsequent migration into the alveolar space. Neutrophil leucocytes are an important source of IL-1 and TNF production.

Future research needs to focus on treatments that can prevent the development of ARDS associated with cytokine-induced reduction of alveolar fluid clearance. The prophylactic application of beta-agonists, which are able to up-regulate alveolar epithelial sodium and chloride transport and were found to reduce lung water in the recent beta agonist lung injury trial, may be able to reduce the risk of this complication.

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# Echocardiographic detection of pooled air in the right upper pulmonary vein after minimal invasive atrial septal defect closure: an alternative approach of de-airing

doi: 10.1017/S026502150700066X

EDITOR:
Cardiac surgery often involves the opening of the heart and great vessels, whereupon these structures inevitably are filled with air. Once the air gets trapped it tends to accumulate at the highest part of the heart, i.e. it is often found in the left ventricular apex, the pulmonary veins, the left atrial appendage and the upper wall of the left atrium. Today de-airing manoeuvres are part of surgical routine and include a multitude of techniques such as venting of the left ventricle and the ascending aorta, manual compression of the heart with the patient alternating between Trendelenburg and reverse-Trendelenburg position, shaking of the heart or venting the left atrium through the right pulmonary vein vent. Unfortunately, manual de-airing techniques are unable to completely and reliably eliminate all retained air [1] and it has been shown that even if

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Accepted for publication 3 April 2007 EJA 4466
First published online 21 June 2007