Pulmonary oedema after high infusion rate of sulprostone

Editor—The use of sulprostone [prostaglandin (PG) E₂] for atonic uterine haemorrhage is increasing. Despite the potential of the drug to cause pulmonary oedema and coronary artery spasm, few clinical reports of side-effects have been published. We present a case of a 35-yr-old healthy Jehova’s Witness requiring emergency surgery for a postpartum haemorrhage of at least 1 litre of blood. Rapid sequence induction and intubation were uneventful. Bleeding was controlled by manual compression and insertion of a uterine balloon device. Sulprostone was infused at a rate varying from 2.1 to 8.2 μg min⁻¹. During the operation, which lasted ~2 h, estimated total blood loss reached 3 litre, and 1 litre of lactated Ringer’s solution and 3 litre of 6% hydroxyethylstarch (HES) (Voluven®) were administered. Blood was withheld in accordance with the patient’s expressed wish. Adequate haemostasis was achieved, facilitated by the use of tranexamic acid 2 g, calcium 2.2 mmol, and desmopressine 24 μg. The patient remained haemodynamically stable throughout the procedure with a final core temperature of 37.2°C. We therefore decided to extubate her trachea in the operating theatre. At this stage, ~600 μg of sulprostone had been infused. Just before extubation, the patient showed signs of agitation, such that pulse oximetry readings became unreliable. After extubation, the patient remained restless and did not respond to verbal commands. Owing to persistent cyanosis, despite oxygen 100% by face mask, we decided to re-intubate her trachea. This proved very difficult due to flooding of the laryngeal inlet with colourless fluid. Saturation was restored to 100% with 20 cm H₂O positive end-expiratory pressure (PEEP) and an F₁O₂ of 0.6. Furosemide 60 mg was given. In intensive care, she was successfully weaned from the ventilator the next day and subsequently discharged to the ward. No blood or blood products were administered and the lowest haemoglobin concentration measured was 3.8 mmol litre⁻¹.

We have learned three lessons from this experience. First, we suspect that the rate of infusion of sulprostone and not only the total dose might be a determinant of the development for pulmonary oedema. Secondly, restlessness at emergence after administration of sulprostone should alert the anaesthetist to the possibility of pulmonary oedema, even if no fluid is visible in the tube as was the case with our patient, and that re-intubation might be difficult. Thirdly, since the mechanism of pulmonary oedema after sulprostone infusion is not completely understood, mainstay of therapy remains supportive including intubation and ventilation with PEEP.

PGE₂ in animal models can increase the hydrostatic pressure and vascular permeability of the pulmonary vascular bed, which can lead to pulmonary oedema.
However, the mechanism of pulmonary oedema in humans is, to the best of our knowledge, not known. If increased hydrostatic pressure is important, then the use of furosemide can be justified. Therapy directly aimed at neutralizing PGE₂ is currently experimental.⁵ Although estimation of blood loss during major bleeding may be difficult, we do not think that our patient developed pulmonary oedema due to overtransfusion as we are confident that we did not infuse a greater volume of i.v. fluid than the amount of blood lost, and she had a normal cardiac function. We are not aware of any report suggesting that pulmonary oedema might be directly caused by HES solutions.

M. Hagenaars*
J. T. A. Knape
E. M. J. M. Backus
Beverwijk, Utrecht and Enschede, The Netherlands
*E-mail: hagenaas7@xs4all.nl

Pathway of the tracheal tube and complications of nasal intubation

Editor—We read with interest the paper of Ahmed-Nusraoth and colleagues¹ who compared the frequency with which the preformed, reinforced, and thermosoftened preformed tubes pass through upper and lower pathways in nasal intubation. They concluded that the tracheal tubes, particularly preformed tubes, frequently took the less favourable pathway, in spite of specific attempts to avoid this. However, several issues of study design merit further comment. The Mallickrod™ preformed nasal tube is a cuffed PVC tube with a stiff texture and requires the thermosoftening treatment before intubation to decrease the incidence of epistaxis and nasal damage.² However, it must be emphasized that the tube will cool to the ambient temperature of the room very quickly and consequently, become stiffer. Accordingly, if the intubation is to be delayed for a long time because of a difficult manipulation, the positive effects of the thermosoftening tube will be negated by the time taken to attempt reintubation.³ This may be a reason for no significant differences in all observed variables between the preformed and thermosoftened preformed tubes.

We are also very interested in the nasal tube size selection methods, in which sizes 7 and 6 mm tubes were used for male and female patients, respectively. They seemed to have selected smaller tube sizes for the adult patients with a mean weight of >70 kg compared with those used in other previous studies.³–⁷ In the vast majority of adults, sizes 7–7.5 mm nasal tube can smoothly pass the nares.

On the basis of the assumption that a 50% reduction in the tubes passing through the upper nasal pathway would be a clinically important difference between the groups, a sample size of 30 patients each group was selected to detect this difference with a power of 80% and a P-value of 0.05. However, the power of the study is not sufficient to detect a statistically significant in the incidence of epistaxis between the groups, although the number of epistaxes caused by passing tubes was apparently greater in the use of the preformed tubes than in the use of the reinforced and thermosoftened preformed tubes. The small sample size may have prevented authors from excluding a type II error when comparing the incidences of epistaxis between left and right nostril intubations, and evaluating the proportions of the preformed and thermosoftened preformed tubes causing epistaxis between upper and lower nasal pathways. Therefore, we do not agree the conclusion of this study that the two nostrils have a similar incidence of epistaxis, when the tube is passed through either nostril with the bevel facing to the left.

F. S. Xue*
X. Liao
Y. M. Zhang
Beijing, People’s Republic of China
*E-mail: fruitxue@yahoo.com.cn

Editor—We thank Professor Xue and colleagues for their interest in our article.¹ The purpose of our investigation was to determine the frequency with which tracheal tubes traversed the upper nasal pathway, because when they traverse this pathway, there is a far greater chance of damage to the middle turbinate,⁴ ⁸ than if they traverse the lower nasal pathway. The observation that intubation of the upper nasal pathway caused significantly more epistaxes was an unanticipated bonus, but it was not the reason for our study. We reported this significant event in our paper and added the other non-significant epistaxis data for completeness. We did not claim to have proved or to have