DELAYED LUMEN OBSTRUCTION IN ENDOTRACHEAL TUBES

S. Hoffman and M. Freedman

SUMMARY

Delayed onset of obstruction in the Oxford endotracheal tube during anaesthesia is described in five patients. The effects of intra-cuff volume and pressure on the wall of the tube were investigated and discussed with special reference to the effects of body heat and repeated use on the consistency of the tube. It is concluded that inward collapse of the tube wall is caused by a combination of factors, namely: frequent use, softening of the tube wall by body heat, the gradual increasing of intra-cuff volume and pressure by diffusion of nitrous oxide into the cuff, replacing a damaged cuff by a new one and heat sterilization. Deflation and re-inflation of the cuff to minimal occlusive volume at hourly intervals is suggested as a precautionary measure in the prevention of inward collapse of the tube wall.

One of the causes of upper airway obstruction during anaesthesia is over-distension of an endotracheal tube cuff, with compression of the wall of the tube.

We report five patients in whom cuff compression caused collapse of the endotracheal tube. A common feature was a delay in the onset of airway obstruction by 30–90 min after endotracheal intubation. In each case, an Oxford rubber endotracheal tube had been used.

The possible causes of the compression and collapse of the e.t.t. were investigated and are discussed.

CASE REPORTS

Case 1

A female patient, aged 47 yr, was undergoing surgery for cholelithiasis. Anaesthesia was induced with droperidol and phenoperidine, thiopentone and nitrous oxide in oxygen (5.3 litre/min) and the trachea was intubated following the administration of suxamethonium chloride. A 9-mm Oxford endotracheal tube, which had been used previously, was employed and the latex cuff was inflated with air to the point at which leakage of inspired gases was prevented. In our hospital such tubes are usually cleaned by soaking in and brushing with soap and water. Occasionally, when a patient has a history of sepsis, pulmonary tuberculosis or any other major infection of the respiratory tract, sterilization is by high-pressure autoclave for 10 min.

Anaesthesia was maintained with phenoperidine and nitrous oxide in oxygen (5.3 litre/min). IPPV was given with an Air-Shields Ventimeter Ventilator using pancuronium bromide for neuromuscular blockade. After 30 min of uneventful anaesthesia, a high pitched whistle was heard, during the inspiratory phase of ventilation, in the vicinity of the mouth. Simultaneously, the airway pressure increased by 10 cm H\textsubscript{2}O. An incremental dose of muscle relaxant did not improve ventilation. A 16-gauge suction catheter, passed down the lumen of the endotracheal tube, could not be advanced further than the estimated level of the cuff. On deflation of the cuff, the whistle-sound disappeared and the airway pressure decreased to less than the original value. There was then a free escape of gases around the tube, which resulted in inadequate ventilation of the patient. The cuff was gradually re-inflated to the minimal volume required for occlusion. However, as signs of obstruction returned, the cuff was deflated and the tube withdrawn and replaced by a fresh tube. Thereafter, anaesthesia proceeded without further untoward incident. On examination of the withdrawn tube, the wall at the cuff site was found to be softer and more compressible than it had been before the anaesthetic procedure.

Case 2

In an adult male patient requiring abdominal surgery, anaesthesia was induced and maintained in the manner described in case 1 and the trachea was intubated using a 9-mm Oxford tube which had been used previously. The original integral cuff was inflated with air to the minimum volume for occlusion.
After 1 hour of uneventful ventilation, signs of acute obstruction appeared suddenly. The chest wall was almost immobile, the expiratory tidal volume was reduced to 100 ml as measured by a Wright's spirometer, and the airway pressure increased from 20 to more than 40 cm H$_2$O. The cuff was deflated immediately and then carefully re-inflated to a point where a satisfactory tidal volume was restored. At this point there was a slight leak of inspiratory gases, but by increasing the fresh gas flow, ventilation could be maintained adequately for the duration of the surgical procedure. After removal the tube was examined and found to be unduly softened and easily compressible over the cuff site.

**Cases 3, 4 and 5**

These were similar to the previous cases in nearly all respects. Signs of airway obstruction occurred more or less acutely after 20–60 min. Obstruction was relieved in these patients by deflating the cuff and packing the pharynx with gauze. In patient no. 5, radiographic examination of the endotracheal tube was made in situ with cuff inflation of 4, 5 and 6 ml of air. The constricting effect on the internal diameter of the tube, of increasing volumes of air in the cuff, is shown in figure 1(A, B, C). After cuff inflation with 4 ml of air, there was still a substantial leak of inspiratory gases, but less of a leak after 5 ml. With 6 ml, airway pressure increased to more than 30 cm H$_2$O and obvious severe obstruction was present. After operation, the sections of the endotracheal tubes underlying the cuffs were found, as in the previous cases, to be obviously more compressible than before use.

**INVESTIGATION**

In order to localize the obstruction, one of the defective endotracheal tubes was inserted into a glass tube 17 mm in internal diameter (i.d.). The latex cuff was inflated with 4.5 ml of air (the volume necessary to achieve an air-seal between the cuff and the glass). The tendency to inward collapse of the walls is shown in figure 2(A).

As a control, the original cuff of a new Oxford endotracheal tube was sacrificed and replaced by a new latex rubber cuff and subjected to the same examination. No inward bulging of the wall occurred even when the cuff was filled with 6 ml of air (fig. 2B).

To elucidate the delay in onset of obstruction, a defective tube and the control tube were each inserted into a glass tube of 17 mm i.d. The cuffs were inflated with 4 ml of air and x-rayed at 20 °C and at
OBSTRUCTION OF ENDOTRACHEAL TUBES

37 °C. At room temperature there was no change in the size of the lumen of the control tube. The affected tube, however, showed minimal narrowing of the lumen before inflation and a more pronounced narrowing on introducing 4 ml of air (fig. 3A). The tubes were then immersed in a water-bath at 37 °C. The progressive narrowing of the lumen of the defective tube at its cuff site, after immersion for 20 min and 60 min, is shown in figure 3(B). The lumen of the control tube was unaffected by cuff inflation or by the increase in temperature (fig. 3C).

DISCUSSION

In the cases we report, the following appear to be factors causing obstruction of the endotracheal tube during anaesthesia.

(a) The Oxford rubber endotracheal tube is so manufactured that its wall becomes progressively thinner towards the distal end. Thus the tube is more likely to yield to the compression of over-inflation of the cuff, particularly when it has been used frequently. Sterilization by autoclaving accelerates the deterioration of rubber (Stark and Pask, 1962), and this method is likely to hasten the onset of weakness.

(b) Replacing a leaking cuff by a new and stiffer latex cuff necessitates the use of higher inflation pressures which an old and frequently used tube is less likely to withstand.

(c) We showed that when a defective tube, with the cuff inflated, is immersed in a water-bath at 37 °C, progressive softening occurs, leading to collapse of the tube wall at the cuff site. This process is time-related.

A further possible contributory factor is the diffusion of nitrous oxide into the cuff during anaesthesia.

Tenney, Carpenter and Rahn (1953) and Eger and Saidman (1965) showed that an enclosed gas-filled space in the body will expand if it contains a gas (nitrogen) which is less soluble in blood than the gas respired (nitrous oxide). Stanley, Kawamura and Graves (1974), in their experiments on the diffusion of nitrous oxide into standard air-inflated latex cuffs, showed that nitrous oxide increased the volume of gas in the cuff in a concentration- and time-related fashion. The increases were significant after 1 h of exposure and often dramatic after 4 h. Intra-cuff pressures also increased after the exhibition of nitrous oxide, frequently becoming as great as 70–100 mm Hg. These authors showed that, if the cuff originally contained 5 ml of air and was exposed to 75% of nitrous oxide, after 1 h the cuff volume increased to 10 ml; 50% nitrous oxide increased the volume to 9 ml after 1 h.

Using low-residual volume cuffs, Stanley, Kawamura and Graves (1974) showed experimentally that clinical concentrations of nitrous oxide (25–75%) will result in sufficient diffusion of nitrous oxide in 4 h to increase the cuff pressure by more than 200 mm Hg, with initial cuff volumes of 2–4 ml of air, and to 400 mm Hg with 10 ml of air, or more.

Temperature is another factor which may influence the rate at which nitrous oxide moves across the rubber membrane into the enclosed gas-filled space.

The cases we report appear to illustrate a combination of factors causing delayed endotracheal tube obstruction.

Softening of the tube wall as a result of repeated use, further softening by exposure to body temperature, and an increase of the intra-cuff pressure because of diffusion of nitrous oxide into the cuff will
all, sooner or later, cause an inward collapse of the tube wall. Changing an old cuff for a new latex cuff may contribute to the collapse.

Since the onset of obstruction has been shown to be delayed, it is suggested that the cuff be deflated every hour and then re-inflated to the minimum occlusion volume.

The inherent susceptibility of the Oxford tube to inward collapse of its wall suggests that it is safer to discard a used tube when the cuff has been damaged, especially when there are also signs of undue softening of the tube wall.

REFERENCES

VERZÖGERTE LUMEN-OBSTRUKTION IN ENDOTRACHEALEN RÖHREN
ZUSAMMENFASSUNG

OBSTRUCCION RETARDADA DE LA LUZ EN LAS SONDAS ENDOTRAQUEALES
SUMARIO
Se describe el comienzo de obstrucción retardada en la sonda endotraqueal tipo Oxford durante anestesia en cinco pacientes. Se investigaron los efectos del volumen intramancho y presión sobre la pared de la sonda, comentándose con referencia particular a los efectos del calor corporal y del uso repetido sobre la consistencia de la sonda. Se concluye que el hundimiento interno de la pared de la sonda está causado por una combinación de factores tales como: uso frecuente, reblandecimiento de la pared de la sonda por el calor corporal, el aumento gradual del volumen y presión intramancho por difusión del óxido nitroso al interior del mancho, la substitución de un manguito deteriorado por uno nuevo, y la esterilización por calor. Como medida de precaución para evitar el hundimiento interno de la pared de la sonda se sugiere desinfección y volver a infar el manguito a su volumen oclusivo mínimo a intervalos de una hora.
Minimal effect on respiration  
Unlikely to lower blood pressure in the conscious patient  
Less nausea and vomiting than with pethidine  
Usefully combines with major and minor tranquillisers  
Can be used as a partial antagonist after fentanyl

Fortral injection is supplied in 1 or 2 ml ampoules of pentazocine 30 mg/ml as the lactate.  
Fortral is a registered trade mark.  
Full prescribing information from your hospital pharmacist or on request from Winthrop Laboratories, Surbiton-upon-Thames, Surrey.
The greatest single advance in anaesthesia since

Fluothane
the original halothane – purely from ICI

Product Licence No. 0029/5058

Detailed information is available on request.

Imperial Chemical Industries Limited
Pharmaceuticals Division
Alderley Park, Macclesfield
Cheshire England