FAULTY CONSTRUCTION OF A REINFORCED LATEX ENDOTRACHEAL TUBE

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SUMMARY

This paper describes a fault in the cuff of a reinforced latex endotracheal tube, where the cuff splits into layers. On attempting to deflate the cuff, the pilot balloon subsides but not the cuff itself. The cause of this fault is examined, and, by implication, steps to eliminate it during manufacture suggested. It would appear that not all such available latex endotracheal tubes are potentially liable to this fault.

Reinforced endotracheal tubes have been used in various forms in anaesthesia for many years. At an early stage one such design consisted of a woven silk mesh embedded in gum-elastic. Since then various attempts have been made to incorporate a reinforcing helix of metal, and later nylon, in the wall of a latex tube. In the last few years such tubes have achieved a high degree of reliability.

Still more recently attempts have been made to add an inflatable cuff to these tubes. Results have been uneven, varying from manufacturer to manufacturer.

Figure 1 shows a common fault arising in tubes apparently from a single manufacturer. When deflation is attempted the pilot balloon subsides, but on extubation it would appear that the main cuff is still inflated. Closer examination reveals that the cuff wall is in two layers (fig. 2). When the cuff is inflated air blows up the inner cuff, passes around the end of this and inflates also the covering layer of latex. On deflation the inner cuff subsides, leaving air trapped between it and the covering layer of latex. There is no external or proximal indication that this inflation is present. We consider this fault a dangerous one and it appears sometimes after use on only a few occasions. Tube A (fig. 1) also shows the additional fault of distal extension of the cuff due to the separation of the layers of latex.

These faults appear to arise as follows: Such a tube is built up by repeated dippings of the helix in latex. It would appear that after a few dippings

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FIG. 1

Showing examples of failure of the main cuff to deflate; the pilot balloons have subsided. Tube A also shows distal extension of the cuff due to separation of the layers of latex.
the pilot tube is applied and over its distal end a balloon sleeve is placed to form the basis of the cuff. The whole is then redipped to give final cover and smoothness. Here there appears to be no form of bonding between the balloon sleeve and subsequent layers of latex. Reliance is placed on the last latex dip and its natural “tackiness” and tension to hold the balloon in place. When inflated, air can track between the ends of the balloon sleeve and the covering latex, resulting in the separation of the outside layer of latex from the balloon and from the main shaft of the tube. Chemical bonding of the outside dip to the balloon sleeve and to the main shaft of the tube would obviate both layering of the cuff, and its peripheral extension.

Figure 3 shows in detail the separation described, the photograph of the deflated sleeve being taken through the final layers of latex dip.

Bridger (1962) discussed a parallel problem occurring with a Foley catheter, in which the balloon was left in the bladder on removal of the catheter.

We would wish to record that we have not seen these faults in an endotracheal tube made by Eschmann Bros. and Walsh Ltd. of Shoreham, and we are grateful to this firm for the concern, interest and vigour that they have shown in preventing these troubles in tubes of their manufacture.

ACKNOWLEDGMENTS

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REFERENCE

SOMMAIRE
L'article décrit un défaut de construction dans la manchette d'une canule endo-trachéale renforcée, en latex. La manchette se désagrège en plusieurs couches. Lorsqu'on cherche à dégonfler la manchette, le ballon-pilote se dégonfle bien mais pas la manchette. L'auteur a étudié la cause de ce défaut et il indique implicitement des mesures pour l'éliminer en cours de fabrication. Il semble toutefois que toutes les canules endotrachéales du commerce n'ont pas — même en puissance — la possibilité de produire un jour cet incident.

ZUSAMMENFASSUNG

CORRESPONDENCE
HALOTHANE IN OBSTETRICS
Sir,—Halothane has recently been recommended in your columns as a uterine relaxant (in association with thiopentone anaesthesia) in cases of manual removal of the placenta where a constriction ring or cervical spasm makes it difficult for the obstetrician to pass his hand into the uterus (Crawford, 1962).

Perhaps my experience of a similar technique from the obstetrician's standpoint might be of interest to your readers. Firstly, I must acknowledge that access to the uterine cavity is in fact greatly facilitated by this procedure. On several occasions, however, I have found that the actual separation of the placenta from its uterine attachment has been made a difficult and potentially dangerous undertaking because extreme flaccidity made it almost impossible for the gloved hand to define the uterine wall. This is a difficulty I have not experienced with other forms of anaesthesia, and when to this disadvantage is added the well-recognized increase in the incidence of severe postpartum haemorrhage associated with the use of halothane, it is my opinion that the advantage of easier access to the uterine cavity is considerably outweighed. On balance, therefore, I feel that halothane is best avoided in anaesthesia for manual removal of the placenta.

Incidentally, I think most obstetricians would agree that cervical spasm or constriction ring can always be safely overcome by patient pressure with the "coned" hand associated with judicious deepening of an orthodox anaesthetic.

W. N. ROLLASON, Aberdeen

REFERENCE

HYPERVENTILATION AND CEREBRAL DAMAGE
Sir,—Perhaps I may be allowed to comment on the observation of Drs. Allen and Morris (Brit. J. Anaesth., 34, 828) that deliberate hyperventilation with volume cycled respirators failed to lower the Pco₂ in six of his patients. This is difficult to understand.

Unless these patients had gross errors in gas distribution, or unless ventilation was in marked excess of pulmonary perfusion, then over-ventilation could not have failed to lower the Pco₂, in six of his patients. This is difficult to understand.

I have measured both minute volume and arterial Pco₂ in 867 patients subjected to hyperventilation. Some of these patients were in carbon dioxide narcosis from respiratory failure due to generalized obstructive lung disease and, therefore, had severe ventilation/perfusion defects. In not one of these 867 patients did hyperventilation fail to reduce the Pco₂ below 40 mm Hg, yet Drs. Allen and Morris think such a failure is common!

Professor Gray and Dr. Rees were astonished; I, sir, am incredulous.

JOHN S. ROBINSON, Prescot