CORRESPONDENCE

OCCLUSION OF DORSALIS PEDIS ARtery

Sir,—I was interested to read the paper by Husum, Palm and Eriksen (1979) on the effects of occlusion of the dorsalis pedis artery, but dismayed that they see fit to condemn its routine cannulation.

For the past 7 years I have, if possible, used a cannula in the dorsalis pedis artery to monitor arterial pressure and gases in patients undergoing neurosurgical operations. During that time I have cannulated this artery in more than 500 patients ranging in age from 18 months to 80 years. In some of these I have used the same artery on more than one occasion and have not seen any clinical evidence of compromise to the circulation.

It is right and proper that the effects of the techniques we use should be investigated and the results published. All the choices we make are compromises reached by weighing up the relative advantages and disadvantages of the alternatives. I hope that, if the occasion arises, my anaesthetist will use my dorsalis pedis artery in preference to the alternatives. I hope that, if the occasion arises, my anaesthetist will use my dorsalis pedis artery in preference to the alternatives.

All the choices we make are compromises reached by weighing up the relative advantages and disadvantages of the alternatives. I hope that, if the occasion arises, my anaesthetist will use my dorsalis pedis artery in preference to the alternatives.

We fully agree that the reinforced tubing suggested by Dr Inglis would have inherent safety advantages and we will endeavour to find suitable tubing. Dr Inglis was kind enough to send us a sample of p.v.c. compressed air hose to illustrate his proposal, but unfortunately this tubing would not itself be suitable as it is not antistatic and we know from experience that the plasticized p.v.c. material from which it is made can become brittle after prolonged exposure to halothane.

We feel it necessary to point out that the outer polythene tube does not have an indefinite life. Small cracks are liable to develop in this tubing, which may result in undesirable leaks although these are clearly less critical than the cutting off of fresh gas supply by the collapse of the inner tube. It is therefore important that users recognize that neither the inner or outer tube of the coaxial circuit is to be considered permanent and that they require frequent inspection.

It would be useful to collate and analyse the experience of anaesthetists concerning the safe life of these assemblies, and if users would care to send details of their experience to the undersigned, we would be pleased to see if these could lead to recommendations concerning a safe working life which could be submitted for publication in these columns.

DAVID HURTER
Kent

REFERENCE


TORSION OF THE INNER TUBE

Sir,—I wish to report a hazard discovered during the use of a Bain type coaxial anaesthetic circuit, manufactured by Messrs Penlon Limited (Bain and Spoerel, 1972). This fault is, I believe, hitherto unreported in the literature.

Following connection of the system to the fresh gas outlet of an anaesthetic machine, it was noted that no fresh gas was being delivered at the patient end connector. On inspection of the valve and tubing it was discovered that the inner fresh gas supply tube of the coaxial system had become twisted (approximately eight times) along its longitudinal axis, resulting in occlusion of the inner tube.

The fault was easily corrected by dismantling the system and untwisting the tube. I believe that this failure was caused by the usual "twist and push" action used to fit the patient end connector to the mask or catheter mount, the outer tube rotating slightly on the end connector each time, allowing the torsion of the inner tube to increase, leading eventually to occlusion as described.

I have discussed this fault with the manufacturers, who recognize this potential hazard, and recommend that in addition to confirming gas flow and inspecting the inner tube, the coaxial tubing assembly should be replaced at regular intervals, as continued exposure to anaesthetic agents softens the material of the inner tube, rendering it more susceptible to occlusion as a result of torsion.

I would suggest that instead of incurring repeated expense in such regular total replacement of both tubes, the inner fresh gas tube should be manufactured from a textile-reinforced material, which, by its nature, would resist torsion and occlusion, even after softening as a result of exposure to anaesthetic agents.

MICHAEL S. INGLIS
Poole, Dorset

REFERENCE


Sir,—The hazard caused by excessive twisting of the inner tube is specifically referred to in the "pre-use safety checks" included in our Data sheet CC178PS which is included with all coaxial circuits when supplied.

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We feel it necessary to point out that the outer polythene tube does not have an indefinite life. Small cracks are liable to develop in this tubing, which may result in undesirable leaks although these are clearly less critical than the cutting off of fresh gas supply by the collapse of the inner tube. It is therefore important that users recognize that neither the inner or outer tube of the coaxial circuit is to be considered permanent and that they require frequent inspection.

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B. R. SUGG
Technical Director,
Penlon Ltd, Abingdon

APPARATUS FAILURE—CAUSE FOR CONCERN

Sir,—An issue of your journal was devoted to the hazards of anaesthesia and in that Editorial, Hull and Norman (1978) drew particular attention to the danger of any change in the usual practice of anaesthesia. Since the publication of that issue we have experienced three serious apparatus faults and these problems occurred when changes were made in our usual practice of anaesthesia.

In the first incident a new Lact co-axial breathing circuit (Lack, 1976a, b) was brought into use after examination in accordance with the accompanying leaflet. During the course of a discussion on the characteristics of the circuit, we realized that it was wrongly manufactured, although it appeared to function normally. The faulty circuit was so constructed that the inner expiratory tube, which should have been connected to the expiratory (exhaust) valve, was in fact connected to the reservoir bag. This arrangement
placed the whole of the circuit tubing (1.5 m long $\times$ 28 m$^{-3}$ diameter) and the reservoir bag on the patient's side of the expiratory valve and converted it to deadspace. The exhaust outlet assembly is a robust metal construction, an integral part of which is the right-angled connection linking the inner expiratory tube to the expiratory valve. In the faulty circuit, this right-angled connection turned down to the reservoir bag instead of up to the exhaust valve, but this was only visible when looking down the fresh gas inlet taper of the circuit. A test of the integrity of the circuit is to disconnect the inner and outer tubes at the patient end, and exhale down the inner expiratory tube with the expiratory valve open. The properly assembled system directs the expired air to atmosphere via the valve. In the faulty system expiration distends the bag. Our recognition of this fault led to the issue of a recent hazard warning (Ref. DHSS 79/18).

In the second incident, the cuff of a nasotracheal tube collapsed inward and obstructed a patient's airway. A change in the usual routine of preparing these tubes had been instituted, unknown to the anaesthetist. They were being packed for autoclaving with the non-return inflating valve in place on the end of the pilot tube, although the cuff was not inflated. It seemed that the small amount of air trapped after insertion of the valve, expanded by heat and aided by the negative pressure in the autoclaving cycle was enough to weaken the wall of the tube. On cuff inflation the weakened tube wall tends to collapse inward and occlude the lumen, particularly when there is a restriction to the outward expansion of the cuff when the tube is in the trachea. Examination of several similar tubes revealed the same fault to a varying degree (Warne Surgical Products Ltd, personal communication). Nevertheless it would seem wise not to autoclave endotracheal tubes with non-return inflating valves in situ.

In the third incident, the oxygen supply failed during the administration of a general anaesthetic. A Boyle's machine supplied by a flexible pipeline from a wall-mounted connection was in use. The pipeline was suspended over a connection point on the back of the machine. In order to avoid acute angulation, there should always be an adequate length of pipeline, never at full stretch, which should curve naturally away from the permanent connection point. Consideration should perhaps be given to strengthening pipelines at this point.

Fortunately, none of these incidents resulted in harm to patients.

J. Muir
R. Davidson-Lamb
Aberdeen

REFERENCES

MINAXOLONE AND MALIGNANT HYPERThERMIA

Sir,—Althesin has been shown to block initiation of the malignant hyperthermia syndrome by halothane in MHS swine (Hall, Trim and Woolf, 1972; Harrison, 1973), and recommended for use in human subjects susceptible to the condition (Honda et al., 1977).

It was of interest, therefore, to ascertain whether minaxolone, the water-soluble steroid anaesthetic (Aveling et al., 1979; McNeill, Clarke and Dundee, 1979) possessed similar properties.

We report here the results of our investigation in four MHS swine of the effects of continuous infusion of minaxolone on the initiation of the MH syndrome by halothane. The experimental programme used was similar to that described previously (Harrison, 1973) with modification which included:

(1) Direct arterial pressure monitoring via femoral artery cannulation.
(2) Use of a cerebral function monitor.
(3) GLC assay of blood concentrations of minaxolone during the experiment.

Dosage of minaxolone was as follows: induction—1.4—2 mg kg$^{-1}$; maintenance infusion—40-100 $\mu$g kg$^{-1}$ min$^{-1}$; total dose (45-60 min)—4-6 mg kg$^{-1}$.

Intravenous concentrations of minaxolone varied from 0.66 to 1.55 $\mu$g ml$^{-1}$ in most, and 2.84 $\mu$g ml$^{-1}$ in one pig. Where the infusion rate was greatly increased in an attempt to abort the onset of MH.

Although minaxolone itself did not provoke the onset of the syndrome, in all four experiments it failed to block initiation of the MH syndrome in response to the administration of halothane, the syndrome being well established within 20 min of exposure in each case.

We cannot as yet define the role, if any, of cromophor (the solvent for Althesin) in these contrasting actions of Althesin and minaxolone.

G. G. Harrison
D. F. Morrell
Cape Town, South Africa

REFERENCES