LETTERS TO THE EDITOR

Scavenging Valve

Dear Sir,

We would like to respond to an article which appeared in the September 1981 journal authored by Dr. Richard M. Flowerdew. This article was titled "A Hazard of Scavenger Port Design."

Dr. Flowerdew, in his article, called attention to several points concerning the Ohio Medical Products' scavenger valve. A recommendation was made that the scavenger valve should be locked in a position so that the exhaust port of the valve exits horizontally, but in the opposite direction of the inhalation and exhalation breathing valve ports. The Ohio scavenger valve incorporates three set screws in the base of the valve located on the face of the hex nut. The purpose of these screws is to lock the valve into the desired position. We recommend to our customers that the valve be positioned so that the adjusting control knob faces the user, then secure the three locking set screws.

The Ohio Medical Products' scavenging valve was originally introduced on the market, having a 22 mm exhaust port. In light of the pending standards for gas scavenging, an adapter kit has been made available for modifying the 22 mm exhaust port to a 19 mm/30 mm exhaust port. This adapter can be simply epoxied into the existing scavenging valve by the user. To order an adapter kit, part number 216-6726-800, contact your local Ohio Medical Products' dealer/sales representative. This kit is designed to be installed by hospital personnel. All scavenging valves currently being sold by Ohio Medical Products have the adapter installed.

The article continues on, recommending the use of color identification to identify the difference between the scavenging tube and the breathing tube. Ohio Medical Products incorporates a yellow colored band at the ends of the 19 mm scavenging tube to aid in visual identification.

We, however, agree wholeheartedly with the writer that "There is still no substitute for a meticulous check of the anaesthetic equipment before use."

Sincerely,
Ronald J. Luich, P.Eng.
Manager, Product Safety,
Ohio Medical Products,
P.O. Box 7550,
Madison, Wisconsin 53707, U.S.A.

Masseter Spasm Induced by Succinylcholine in Children

Sir,

With reference to the article of Eugene H. Flewellen and Thomas E. Nelson[1] recently published in the Canadian Anaesthetists' Society Journal I wish to relate the following incident.

A 16-year-old Nigerian boy with *Nephrotic Syndrome* presented for inguinal hernia surgery. He did not receive any narcotic premedication but was given atropine 0.6 mg intravenously at induction. Anaesthesia was induced with alfathesin 0.1 $ml \cdot kg^{-1}$ and maintained with a 2:1 mixture of nitrous oxide and oxygen with halothane, using a face mask. However, because the airway was not satisfactorily maintained, it was decided to intubate the trachea after 10 minutes of general anaesthesia. Suxamethonium bromide 40 mg was given intravenously to facilitate orotracheal intubation. Marked fasciculations were observed, followed by masseter spasm which prevented attempts to open the mouth and intubate the patient. It was noted that before injection of suxamethonium the jaw muscles were sufficiently relaxed to allow easy insertion of an oropharynegeal airway.

The patient was ventilated with 100 per cent oxygen and halothane by face mask until about five minutes later when it became possible with some difficulty to intubate the trachea. The patient resumed spontaneous respiration shortly after, and the rest of the anaesthetic with nitrous oxide, oxygen and halothane was uneventful. Temperature was monitored and there was no hyperthermia. Malignant Hyperthermia has not been reported in a Nigerian.

Post-operative investigation included electrolytes and urea, calcium and phosphate, haemogram, blood sugar, and liver function tests. The results were all normal. Lactic dehydrogenase (LDH), aldose and creatinine phosphokinase (CPK) were markedly elevated. Facilities for caffeine-induced contracture test and serum pyrophosphate levels were not available.

The patient had none of the neurological or neuromuscular diseases known to be associated with muscle rigidity following suxamethonium. No other member of his family has had an anaesthetic before.

Two main alternatives were considered in this patient. He may be susceptible to malignant hyperthermia syndrome, and the althesin induc-

tion may have aborted a full clinical picture.[2,3] The other is the possible association with nephrotic syndrome in this patient. I am not aware however of any such association reported in the literature.

It will be good to know if others have experienced any similar event with nephrotic syndrome patients.

Yours faithfully,
Department of Anaesthesia,
College of Medicine,
C.E. Famewo, F.R.C.P.(C),
University College Hospital,
Ibadan, Nigeria.

REFERENCES

1. FLEWELLEN, E.H. & NELSON, T.E. Masseter Spasm induced by succinylcholine in children. Can. Anaesth. Soc. Jour. *29*: 42 (1982).
2. JUDELMAN, H. & Pirie, D.H. Anaesthesia with althesin in a patient with previous malignant hyperpyrexia. Brit. J. Anaesth. *45*: 519 (1974).
3. HARRISON, G.C. Althesin and malignant hyperthermia. Brit. J. Anaesth. *45*: 1019 (1975).

DEFECTIVE TRACHEAL TUBE CONNECTOR

DEAR SIR,

Re: W.D. Lahay: Defective Tracheal Tube Connector, Can. Anaesth. Soc. J. *29*: 80 (January 1982).

Dr. Lahay's letter has prompted me to report another case in which a defective tracheal tube connector could have caused serious complications.

Shortly after the intubation of a patient who was being prepared for a radical mastoïdectomy, I noticed that there was a leak in the anaesthetic circuit. All the connections were checked and it was discovered that the leak originated from the plastic tracheal tube connector (National Catheter No 86050).

The leak was caused by a flap-like break in the portion of the connector which is inserted in the tracheal tube. The connector was carefully removed for replacement. A close examination showed that the flap-like piece of plastic, which measured approximately 5 mm × 20 mm, was almost completely separated from the connector. In fact, it was easily detached by light finger pressure during examination.

It seems that the connector had been crushed at some time before being used in the operating room because the extremity was flattened and the plastic showed whitish discoloration plaques compatible with the stress of deformation.

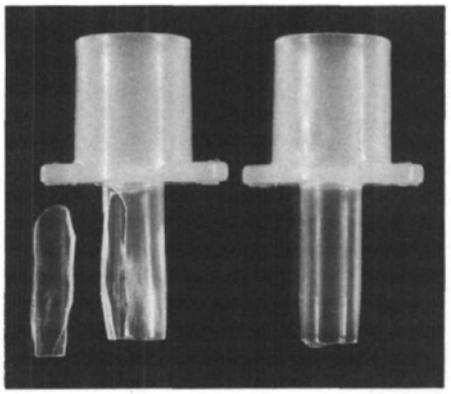

FIGURE 1 The defective tracheal tube connector compared with a normal connector. The detached piece is clearly shown.

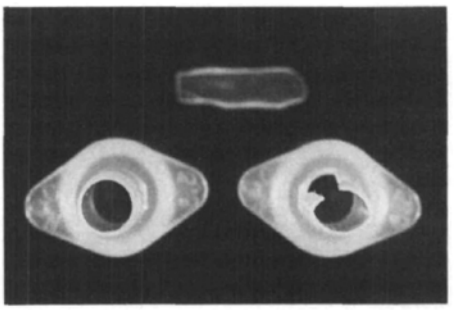

FIGURE 2 Top view of the defective connector compared with a normal connector. The deformation of the extremity of the defective connector suggesting crushing is evident.

If this defective tracheal tube connector had not been noticed, the piece of plastic could have been aspirated and the patient would have undoubtedly suffered serious consequences.

By reporting this experience, I want to stress Dr. Lahay's recommendation that all tracheal tube connectors should be carefully checked before use.

Henri Desmeules, M.D., F.R.C.P.(C),
Département d'Anesthésie-Réanimation,
Centre Hospitalier de l'Université Laval,
Sainte-Foy, Québec

DEFECTIVE PRESSURE/FLOW ALARM

DEAR SIR,

I would like to draw to the attention of your readers a potential hazard in the Drager Narkomed II anaesthetic machine system incorporating

the integral Model DPM-S Drager pressure monitor. We have found an intermittently evident defect in the audio-visual alarm system. We believe that the problem may lie in the OFF-ON switch which activates both the anaesthetic system and the alarm systems monitoring pressure and oxygen flow ratios. There are two situations which we have found on several occasions with defective alarms: (1) All of the pressure-flow alarms can be audible and visible for no detectable reason, and can be silenced by pushing IN the OFF-ON button, but the defective alarm resumes as soon as this IN pressure is released. (2) As a check on the visible and audible elements of the alarm, they can be tested and activated by pushing IN on the OFF-ON button.

Thus the OFF-ON switch, when pushed IN, can either activate or deactivate the audible and visual alarm indicators. We do not believe that this was intended in the design. The Drager service representative agrees that the switch may require adjustments from time to time. Our Department of Biomedical Engineering is critically examining the design, durability and mounting supports of the switch and electronic circuitry. The cause of the malfunction is incompletely assessed at present. Our chief concern with the problem has been that it occurs intermittently and unpredictably. The machine alarms may check out 100 per cent before use, then fail either way as indicated above once a case has started. Such machines are immediately removed from service and replaced. Until the problem is more precisely identified and corrected we consider that an alert to anaesthetists is warranted.

Yours sincerely,

W. Douglas Lahay, M.D., F.R.C.P.(C),
Department of Anaesthesia,
Health Sciences Centre Hospital,
University of British Columbia,
Vancouver, B.C.

Epidural Narcotics and Control of Arterial Pressure in a Pre-eclamptic Patient

Sir,

Since epidural morphine[1] and morphine with fentanyl[2] for pain relief were first described, many other favourable reports have been published. However, it seems that epidural narcotics have not yet been used for the management of hypertension due to toxaemia of pregnancy.

A 25 year old primipara in the 39th week of gestation was admitted for rupture of membranes. The blood pressure was 230/160 mmHg and heart rate 96 beats/minute at rest; there were other stigmata of pre-eclampsia, such as agitation, headache, hyperactive deep tendon reflexes, peripheral oedema and slight proteinuria. Without premedication an epidural catheter was inserted through the T_{12}/L_1 interspace and progress of labour was followed by internal-external foetal monitoring.

Fentanyl 0.1 mg and morphine 2 mg were injected through the catheter with the patient in the supine position, as described in a previous report.[3] Five minutes later the blood pressure had decreased from 205/130 to 160/110 mmHG, the headache had disappeared, and the patient began to relax; the heart rate did not change. At that point it was decided to inject successive doses of fentanyl to control both labour pains and blood pressure, and fentanyl 0.1 mg in normal saline 6 ml was injected 35 minutes later since the blood pressure had begun to rise again and had reached 180/130 mmHg. Despite this a vague feeling of malaise persisted and therefore the patient was turned on her side twenty minutes later. The blood pressure promptly fell to 140/90 mmHg and the malaise disappeared. The lateral position was then maintained until delivery. Figure 1 depicts blood pressure and times and administration of narcotics throughout labour.

Seven hours and 15 minutes after the first epidural injection, naloxone 0.4 mg was administered intravenously: agitation, a strong headache and suprapubic pain promptly recurred, but blood pressure and heart rate did not change and remained at 150/105 mmHg and 98 beats/min respectively. Because of this reaction two further doses of fentanyl were administered within 12 minutes from one another to restore the

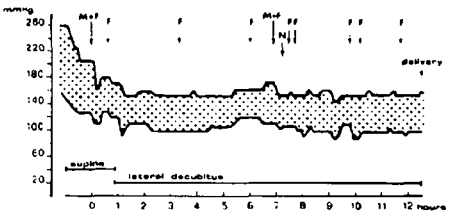

FIGURE 1 Blood pressure recordings. (M) is morphine, (F) fentanyl, (N) naloxone.

patient to the condition which had prevailed before naloxone. During the remaining labour the blood pressure did not rise above 160/105 mmHg and did not fall below 140/90 mmHg. She delivered approximately 12 hours and 30 minutes after the first epidural injection; the infant had an Apgar score of 6 and 10 at 1 and 5 minutes respectively.

Our observations corroborate the hypothesis that the anti-hypertensive effects of epidural opiates can be ascribed to a strong direct action on spinal opiate receptors. We believe that concurrent sedation was the result of rapid vascular absorption of the narcotics from the epidural space and consequent interaction with opiate receptors in the brain. It has been established that morphine concentrations in the blood after epidural injection are similar to those after intramuscular administration of the same dose.[4] The lack of peripheral vascular response to intravenous naloxone after epidural narcotics is consistent with the hypothesis of spinal receptor involvement which is less easily counteracted than that which occurs at the level of the central receptors.

Ezio Vincenti,
Bruno Tambuscio,
Istituto di Anestesiologia,
Università di Padova,
35100 Padova (Italia).

REFERENCES

1. BEHAR, M., MAGORA, F., OLSHWANG, D. & DAVIDSON, J.T. Epidural Morphine in Treatment of Pain. Lancet *i*: 527–529 (1979).
2. DE CASTRO, J., D'INVERNO, E., LECRON, L., LÉVY, D. & TOPPET-BALATONI, E. Perspectives d'utilisation de morphinoides en anesthésie loco-régionale: justification, premiers résultats.
3. VINCENTI, E. Possibili alternative all'impiego degli anestetici locali. In: "Il blocco peridurale continuo in travaglio di parto: realtà e prospettive", SEMES Ed., Padvoa, 32–34 (1981).
4. CHAUVIN, M., SAMII, K., SCHERMANN, J.M., SANDOUK, P., BOURDON, R. & VIARS, P. Plasma concentration of morphine after i.m., extradural and intrathecal administration.

INFECTION RISK OF LONG TERM INDWELLING EPIDURAL CATHETERS

SIR,

It was of great interest to read about long-term indwelling epidural catheters in the treatment of cancer pain.[1] It seems that epidural morphine has a much better analgesic effect than all other known pain treatments.

One year ago we started epidural morphine treatment in cancer pain. A filter is always used in line (Millex with 0,22 my-m pore size). The use of a millipore filter was described by Desmond in 1972.[2]

The risk of infection by long-term indwelling epidural catheters appears to be minimal, when one reads the literature on the subject.[3–5] A case history will be presented to illustrate this problem:

A woman of 48 years with multiple cancer metastases was treated with epidural morphine for 76 days in which time 201 injections were given through the catheter. The catheter was established in the epidural space between lumbar vertebrae 2 and 3. At the post-mortem the lumbar vertebral column was dissected and every

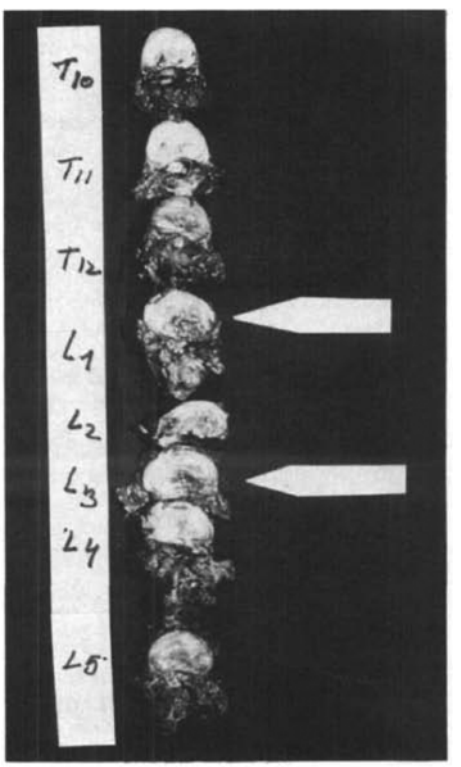

FIGURE 1 The single vertebrae from the lumbar and thoracic column. L = lumbar vertebra. T = thoracic vertebra. Between the arrows the epidural catheter was placed.

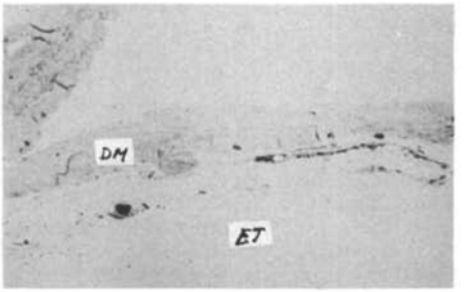

FIGURE 2A

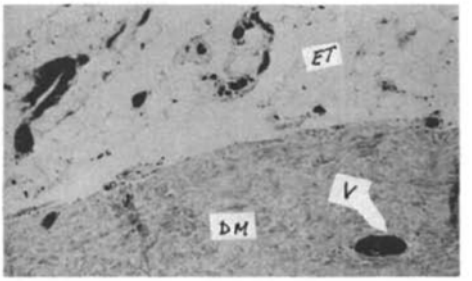

FIGURE 2B

FIGURE 2A & B Biopsy from dura mater and the epidural tissue where the catheter was. DM = dura mater; ET = epidural tissue; V = vessel. A: Magnitude 250 ×. No sign of infection. B: Magnitude 400 ×. Very little reaction is seen in the epidural tissue (ET). Perivascular cell infiltration and some slightly increased vascularisation

single vertebra uncovered (Figure 1). No macroscopic infection was seen. Biopsies from the dura mater and the epidural tissue were also without any sign of infection (Figure 2).

The low risk of infection by long-term indwelling epidural catheters is probably due to several factors. First, the epidural space is very vascular, so that bacteria would be killed very quickly by leucocytes. Second, the filter in line will retain nearly all microbes. Finally, it is possible that drugs are bacteriostatic.[6]

The conclusion must be that the method is very safe, but further investigation must be done to elucidate this problem.

Finn Redke Christensen, M.D.,
Lars Willy Andersen, M.D.,
Department of Anaesthesia,
Frederiksborg County Hospital,
Hillerød, DK-3400,
Denmark.

REFERENCES

1. ZENZ, M., SCHAPPLER-SCHEELE, B., NEUHAUS, R., PIEPENBROCK, S. & HILFRICH, J. Long-term peridural morphine analgesia in cancer pain. Lancet *i*: 91 (1981).
2. DESMOND, J. The use of micropore filters in continuous epidural anaesthesia. Can. Anaesth. Soc. J. *19*: 97–100 (1972).
3. ABOULEISH, E. Spinal infections by epidural catheters. Anesthesiology, suppl. 372 (1979).
4. BAKER, A.S., OJEMANN, R.G., SWARTZ, M. & RICHARDSON, E.P. Spinal epidural abscess. New. Engl. J. Med. *293*: 463–468 (1975).
5. BARRETO, R.S. Bacteriological culture of indwelling epidural catheters. Anesthesiology *23*: 643–646 (1962).
6. JOHNSON, B.H. & EGER, E.I. Bactericidal effects of anesthetics. Anesth. Analg. *58*. 136–138 (1979)