

Correspondence

Complications of minitracheotomy

Dear Sir,

Reports of inadvertently placed minitracheotomy tubes appear increasingly in the literature, as do recommendations for preventing them [1, 2]. Since the reproduction of their illustration (Fig. 1) in the paper of Ryan et al. is not very convincing to me, I wish to propose two other anesthesiological safety measurements. Lateral neck radiographs of ICU patients are sometimes difficult to interpret due to the fact that they are taken supine and therefore incomplete. Certainly less expensive and more obvious as routine precautions are direct laryngoscopy and monitoring end-tidal CO₂. A capnograph should be available in every hospital where minitracheotomies are performed and is generally recognised as one of the most reliable ways of avoiding oesophageal intubation.

The use of a Seldinger technique [2] implies the blind insertion of a rather stiff, inflexible introducer a long distance into the trachea, as opposed to the soft, curled and less traumatising end of a guidewire designed for central venous catheterisation. A "peripheral venous catheter" technique which allows only the tip of the introducer penetrating the cricothyroid membrane in order to slide the actual tube sitting on it, gently over that tip, make causing a false route less likely. Insertion of introducer and tube at once, followed by withdrawal leaving the latter in the trachea, should all be done in a single smooth movement.

Finally the point most worthy of note is the indication for elective use of minitracheotomies. Shouldn't it be revised if the patient continues to make satisfactory progress despite an unspotted misplacement of his tube [1]?

Yours faithfully,

P. R. Martens

References

1. Ryan DW, Dark JH, Misra U, Pridie AK (1989) Intraoesophageal placement of minitracheostomy tube. *Intensive Care Med* 15:538–539
2. Allen PW, Thornton M (1989) Oesophageal perforation with minitracheostomy. *Intensive Care Med* 15:543

Dr. P. R. Martens, Department of Anaesthesia and Critical Care, AZ St. Jan Hospital, Ruddershove 10, B-8000 Brugge, Belgium

Response

Dear Sir,

We believe our patient, referred to by Dr. Martens in his letter, had correct initial intra-tracheal placement of the tube, with subsequent migration of the tip through the membranous trachea into the oesophagus as shown by the original slide of the lateral X-ray, which convincingly shows intra-oesophageal placement. None of the techniques suggested by Dr. Martens for verifying initial position would prevent this migration.

We agree that elective minitracheotomies should only be placed in carefully selected patients. In this instance formal tracheostomy, which would otherwise have been essential for wearing, was avoided.

Yours faithfully,

D. W. Ryan and J. H. Dark

Dr. D. W. Ryan, General Intensive Therapy Unit, Freeman Hospital, Freeman Road, High Heaton, Newcastle upon Tyne NE7 7DN, UK

Sir Robert Macintosh and intensive care

Dear Sir,

The death of Sir Robert Macintosh, the first Nuffield Professor of Anaesthetics at the University of Oxford (1937–1965) on 28th August, 1989 at the age of 91 has prompted me to remind your readers of Sir Robert's place in the development of Intensive Care.

Shortly after Professor Macintosh was appointed to the Chair, Lord Nuffield (previously William Morris, the founder of the Morris Motor Company and a great benefactor to medicine) asked him to advise on the provision of apparatus for the support of ventilation in patients suffering from severe paralytic poliomyelitis. Sir Robert Macintosh reviewed all the methods then in vogue and he and Dr. C. L. G. Pratt made a film showing these devices in action. This was shown to Lord Nuffield who was so impressed by the possibilities of the iron lung that he offered to build and donate tank ventilators to any hospital in the British Commonwealth which requested a machine. Nuffield employed both, a young Australian engineer, to design a simple plywood version of the original Drinker machine. Over 800 of these ventilators were built in the Morris workshops at Cowley and distributed to hospitals, the professor and his co-workers provided short courses of instruction for doctors and nurses using the machine.

In 1940, Sir Robert Macintosh recognised that a tank ventilator could be used to provide respiratory support in other circumstances and reported that he had used the Both ventilator to support the ventilation of two patients after a major abdominal operation [1]. In 1944, two of his assistants, Drs. Mushin and Faux [2], reported a trial in which they had nursed 24 patients in a Both ventilator for 24 h after major abdominal operations, in order to try and prevent respiratory complications. They reported that 14 out of the 24 patients had no detectable pulmonary complication whilst the remaining 10 patients had minor respiratory complications only. However, in their article, Mushin and Faux state "It would be incorrect to say that the patients enjoyed their brief spell in the Both respirator. They were not apprehensive but owing to the physical constraint were glad to be taken out at the end of 24 hours". When I later discussed this use of the Both ventilator with Professor Macintosh he said: "I still think the idea is an excellent one but at that time it was badly flawed through no fault of my own. Perhaps I should have realised that in those days only patients helplessly ill were put in iron lungs. The idea of research was unknown and the sound of iron was pretty sinister. It soon went round the City of Oxford that Mr. Blank's patient who went into the Radcliffe to have his gall bladder out, woke up in an iron lung! The surgeon's reputation could not stand for it. Remember that this was over 40 years ago".

Although there is no further record of the use of the Both ventilator for this purpose, Sir Robert Macintosh subsequently provided strong support for the development of a respiratory unit for treating respiratory paralysis. In 1953, Drs. W. Ritchie Russell and J. M. K. Spalding (neurologists) and Alex Crampton Smith (anaesthetist) set up the Respiration Unit in Oxford [3]. Members of this unit contributed greatly to the long-term use of intermittent positive pressure ventilation by designing ventilators, humidifiers and other equipment, defining the basic principles of treatment and elucidating much of the fundamental physiology of this highly successful form of treatment.

Sir Robert Macintosh was a kindly, gentle, modest, unassuming person with an absolute lack of pretentiousness and a complete and searing honesty. He had an enormous impact on the development of anaesthesia, both in the UK and worldwide and many academic departments owe their origins to his persuasive eloquence. It is appropriate that we should also remember his early contribution to the development of intensive care.

Yours sincerely,
R. Trubuhovich

I am grateful to Professor Keith Sykes of University of Oxford, UK, for his valuable assistance.

References

1. Macintosh RR (1940) New use for the Both respirator. *Lancet* II: 745–746
2. Mushin WW, Faux N (1944) Use of the Both respirator to reduce post-operative morbidity. *Lancet* II:685–686
3. Beinart J (1987) A history of the Nuffield Department of Anaesthetics 1937–1987. Oxford University Press, Oxford, pp 43–45

Dr. R. Trubuhovich, Auckland Hospital, Department of Critical Care Medicine, Park Road, Auckland 1, New Zealand

Ethical problems in intensive care medicine and case reporting

Dear Sir,

Jean-Louis Vincent's article [1] provides a fascinating insight into European attitudes on ethical problems in intensive care medicine. While digesting its implications I turned the page to the very next article by Bohrer and colleagues from the University of Heidelberg on the use of clonidine as a sedative adjuvant [2]. The scientific value of the case report is quite clear. However, it does perhaps raise a point of concern.

The patient treated was a 63-year old man who had undergone palliative partial oesophagogastrrectomy for adenocarcinoma. Following an eventful 53-day period of postoperative ventilation, spontaneous ventilation was resumed through the tracheostomy tube. No information was given regarding subsequent progress or discharge from hospital. Although not directly relevant to the case report, an indication of the outcome of hospital admission would have been illuminating. This applies to the majority of case reports.

I believe the justification for protracted intensive therapy must be some expectation of a reasonable recovery. I realise this may be difficult to define and there will be inevitable differences of opinion regarding management. This is clearly illustrated in Vincent's report and the references he cites.

It was the consecutive appearance of these two articles that prompted me to comment.

Yours faithfully,
D.P. Coates

References

1. Vincent J-L (1990) European attitudes towards ethical problems in intensive care medicine: results of an ethical questionnaire. *Intensive Care Medicine* 16:256–264
2. Böhrer H, Bach A, Layer M, Werning P (1990) Clonidine as a sedative adjunct in intensive care. *Intensive Care Med* 16:265–166

D.P. Coates, Consultant in Anaesthesia and Intensive Care, Sir Humphry Davy Department of Anaesthesia, Bristol Royal Infirmary Bristol BS2 8HW, UK

Acute polyneuropathy in critically ill patients

Dear Sir,

Lopez Messa et al. [1] report on 5 patients, who developed signs and symptoms of an acute polyneuropathy during intensive care treatment. Three of them suffered from Acute Respiratory Failure (ARF), 2 of purulent postoperative peritonitis. In all of these patients inadequate nutrition was assumed to be the cause of critically ill polyneuropathy.

During the 1-year-period, January to December 1989, we treated 4 patients similar to those presented by Lopez Messa et al. [1]. They had not shown neurological deficits at the time of admission, but all of them developed flaccid tetraparesis 10–17 days thereafter. Various lifethreatening conditions (polytrauma, acute pancreatitis, status asthmaticus, psoas abscess with septicaemia) necessitated artificial ventilation for a period of more than 10 days. ARF was not present primarily, but 3 patients developed respiratory insufficiency within 24 h after admission, the 4th patient was kept in general anaesthesia postoperatively. Fever between 38° and 40° C occurred in all 4 patients and persisted for more than 2 weeks, a feature described by several authors [1–5]. Cultures of blood, bronchial secretion and urine yielded a great number of different bacteria, *Staph albus* and *Enterococcus* being present in all our patients. These bacteria are not known to produce toxin, which might lead to damage of the peripheral nervous system. Liver function tests were abnormal and white blood count was elevated in all our patients. Two developed hyperosmolality and 2 pathological coagulability. Electrophysiological studies revealed an axonal form of polyneuropathy and in 1 patient there was strong evidence of concomitant autonomic dysfunction with bradycardia, which was treated successfully by a temporary pacemaker.

In contrast to Lopez Messa et al., in our patients nutritional laboratory parameters were within normal limits, e.g. protein, serum albumin and transferrin and there was no evidence of vitamin deficiency.

Three of our patients died, but none of them because of the neurological disorder, a fact which is reported by other authors as well [2, 3, 5]. The surviving patient recovered almost completely within 3 months and only a minimal paresis of both peroneal nerves remained.

In conclusion we agree with Lopez Messa et al. that prolonged infections and respiratory problems may be important factors in developing an axonal polyneuropathy, but we think that at least several other factors such as hyperosmolality [4], pathological coagulability and even drugs, used in intensive care, are equally responsible for axonal polyneuropathy in critically ill patients.

Yours faithfully,

K. Berek, E. Schmutzhard, M. Neumann, H.-P. Haring, B. Pfausler, A. Berek and M. Kofler

References

1. Lopez Messa JB, Garcia A (1990) Acute polyneuropathy in critically ill patients. *Intensive Care Med* 16:159–162
2. Bolton CF, Gilbert JJ, Hahn AF, Sibbald WJ (1984) Polyneuropathy in critically ill patients. *J Neurol Neurosurg Psychiatry* 47:1223–1231
3. Bolton CF, Laverty DA, Brown JD, Witt NJ, Hahn AF, Sibbald WJ (1986) Critically ill polyneuropathy: electrophysiological studies and differentiation from Guillain-Barré syndrome. *J Neurol Neurosurg Psychiatry* 49:563–573
4. Waldhausen E, Keser G, Schulz B, Lippers P (1989) Weaning failure due to acute neuromuscular disease. *Crit Care Med* 17:594–595
5. Zochodne DW, Bolton CF, Wells GA, Gilbert JJ, Hahn AF, Brown JD, Sibbald WA (1987) Critical illness polyneuropathy – a complication of sepsis and multiple organ failure. *Brain* 110:819–842

Dr. K. Berek, Neurointensive Care Unit, Department of Neurology, University Hospital of Innsbruck, Anichstraße 35, A-6020 Innsbruck, Austria