LETTERS TO THE EDITOR

ANAESTHETIC MACHINE STANDARD

SIR:

When a new standard for improved safety is introduced which quickly results in a disaster of its own, I feel that criticism is warranted. The new downstream location of the O₂ on anaesthetic machines is bound to result in further mishaps despite the modifications, already found to be necessary, to correct the new hazard. It is the equivalent of the automobile manufacturers deciding to reverse the locations of the accelerator and brake pedals. (As good a case could be made for this change.)

Although only one major accident has been widely publicized, I believe there is at least one other unreported to now. How many near misses have there been? Shortly after acquiring a machine with the new safety feature, we experienced two instances where N₂O was given instead of O₂. Since then the machine has only been used as a spare. I am sure that many similar occurrences have been experienced in hospitals across the country.

I venture to suggest that the cure will be worse than the disease. The C.S.A. has recommended that all machines should be converted by July 1978 and Dr. Wyant in his editorial suggests that "conversion is relatively inexpensive and easily carried out by manufacturers". It is certainly not inexpensive when one considers the number involved throughout the country and even worse it would appear that the manufacturers can certainly not handle this volume in a hurry. In the meantime hospitals are going to be in possession of machines with two different O₂ locations, and the resultant hazards are too awful to contemplate. The only solution is to ban the use of nitrous oxide on machines that have not been converted, otherwise this ill-conceived scheme of conversion will have little that is SAFE about it.

P.M.F. McGarry, M.B., F.R.C.P.(C)
Anaesthetic Department
Grace General Hospital, Winnipeg

SIR:

As author of the Editorial on "Some Dangers in Anaesthesia" in the March issue of the Journal

and as Chairman of the Committee on Anaesthetic Equipment and Respiratory Technology of the Canadian Standards Association, I wish to thank you for the opportunity to reply to the letter from Dr. P.M.F. McGarry.

Our committee, which is composed of practicing anaesthetists from across the country and representatives of industry, has wrestled for the last several years with the problem of writing a standard for anaesthesia machines and specifically with the problem of the location of the oxygen flowmeter, which is the item with which Dr. McGarry takes issue. Before incorporating this suggestion into the standard both it and, indeed, the entire draft standard, has been scrutinized by the Committee on Standards of Anaesthetic Practice of the Canadian Anaesthetists' Society, which in turn reports to Council.

To judge from the last sentence of Dr. McGarry's letter, it would appear that his apprehension is based on the assumption that at the present time all machines have the oxygen on the left. Let me assure him that this is far from being the case. In this country we are in the unfortunate position of having machines with the oxygen on the right, on the left, and even some with oxygen in the center of the flowmeter assembly, some of these different positions being found in the same department. Consequently, I respectfully submit that the comparison with controls on a motor car is not appropriate, since I am not aware of any car manufactured in any country which does not have a standard relationship of accelerator to brake pedal. There is obviously a major hazard in this very great variation in position of the control for vital oxygen and our committee was faced with three possible solutions: (1) to do nothing and let the situation continue in perpetuity; (2) to specify oxygen on the left or; (3) oxygen on the right. Whichever of these two positions were chosen, a considerable proportion of anaesthetists would indeed be faced with a re-learning situation. In the final analysis we chose the position on the right because of the greater safety this position confers in case of a leakage from an upstream flowmeter tube, with consequent loss of oxygen. Part of the proposed standard is the requirement for a distinctive shape of the oxygen control knob and this should do much to simplify

the transition. Whatever the difficulty is during this transition period, at least the confusion is not allowed to go on indefinitely.

As far as the remarks about the cost of conversion are concerned, our committee was assured by the manufacturers that the cost per machine was not prohibitive and, while I agree that it may add up to a few thousand dollars to any one department, this is a one-time expenditure and not very much, given the total budget of any hospital or even an anaesthesia department. Moreover, what price-tag does one attach to safety?

I hope that this explanation will assist in allaying Dr. McGarry's anxieties. I am sure that the standardization of the oxygen controls is a great step forward in providing safety for our patients.

Gordon M. Wyant, C.D.,
M.D., F.F.A.R.C.S.

CYANIDE TOXICITY FOLLOWING NITROPRUSSIDE

SIR:

Comparison of the blood cyanide levels in patients infused with sodium nitroprusside (SNP) reported by Aitken, *et al.*¹ with our results² reveals certain major differences which require explanation:

(1) Our patients had a mean total blood cyanide similar to those of Aitken (25 $\mu\text{mol/l}$ and 24 $\mu\text{mol/l}$ respectively) although they received almost twice the mean dose of SNP (0.42 mg/kg compared with 0.22 mg/kg). Similarly the highest blood cyanide of 76 $\mu\text{mol/l}$ in a patient receiving 0.74 mg SNP/kg compares with 107 $\mu\text{mol/l}$ in one of our patients receiving 1.9 mg/kg.

(2) These authors further suggested that cyanide levels reached a maximum within 45 minutes after infusion. We found that 60 minutes after the end of infusion mean blood cyanide levels were down by 50 per cent compared with a 20 per cent reduction in their levels.

One explanation for these differences may be the shorter mean infusion time used by Aitken (36 minutes compared with 88 minutes for our patients). However, since cyanide levels appear to be related more to total dose than to dose rate,^{1,2} this may not provide the entire answer.

A difference in analytical technique will certainly have contributed to the disparity in findings. Aitken and colleagues determined

cyanide levels on whole blood samples following storage in deep freeze. Our determinations were made *separately* on fresh plasma and saline washed RBC. Artifactual formation of cyanide from plasma thiocyanate occurs on assay of whole blood³ and this is probably increased by freezing and thawing.⁴

Aitken, *et al.* also stated that increasing degrees of metabolic acidosis are associated with blood levels above 53 $\mu\text{g}\%$ (20 $\mu\text{mol/l}$) and four out of 13 of their patients showed signs of this 45–180 minutes after peak cyanide levels were attained. One of these patients, who was most obviously affected had a blood cyanide level of 205 $\mu\text{g}\%$ (76 $\mu\text{mol/l}$). As a consequence they recommend that for short-term infusions the dose of SNP should be no higher than 0.5 mg/kg. Following SNP infusion over 98 per cent of the blood cyanide is located in the erythrocytes and plasma levels determine toxicity.² So ignoring the contribution of artifactually produced cyanide, the above blood HCN values would be synonymous with plasma levels of 0.3 and 1.2 $\mu\text{mol/l}$ respectively. These figures are placed in perspective by comparison with the value of 9.3 $\mu\text{mol HCN/l}$ plasma calculated² for the patient of MacRae and Owen who developed severe but reversible metabolic acidosis (one out of a thousand patients⁵) and by the fact that six out of 26 of our patients had plasma cyanide levels above 1.2 $\mu\text{mol/l}$ without obvious toxicity.

The delay in development of acidosis in Aitken's patients is also puzzling. Their findings contrast with those in our dogs (soon to be published) infused with 1.5 mg SNP/kg for one hour where lactate and base deficit rose and fell in parallel with plasma cyanide levels. A mean rise in lactate and base deficit of 2.38 mmol/l and 3.8 mmol/l respectively coincided with a maximum increase of 1.36 $\mu\text{mol HCN/l}$ plasma at the end of infusion. These changes in base and lactate were small and reversed spontaneously.

Three out of four of their patients who were said to show evidence of tissue hypoxia had a base deficit at the start of SNP infusion and their reported lactate levels following nitroprusside were not very different from the mean intraoperative lactate levels for all their patients. In addition, Aitken, *et al.* state that for three of their patients with the greatest increase in base deficit the appearance of delayed acidosis was exacerbated in the postoperative period when ventilation support was withdrawn.

A recommendation which proposes to drastically revise an accepted maximum dose of a valuable agent like SNP requires much firmer evidence than that provided by these authors.

C.J. Vesey, M.Sc.
P.V. Cole, F.F.A., R.C.S.,
Anaesthetic Laboratory,
St. Bartholomew's Hospital,
West Smithfield,
London E.C.1.

REFERENCES

1. AITKEN, D., WEST, D., SMITH, F., POZNANSKI, W., COWAN, J., HURTIG, J., PETERSON, E., & BENOIT, B. Cyanide toxicity following nitroprusside induced hypotension. *Canadian Anaesthetists' Society Journal* 24: 651-660 (1977).
2. VESEY, C.J., COLE, P.V., & SIMPSON, P.J. Cyanide and thiocyanate concentrations following sodium nitroprusside infusion in man. *British Journal of Anaesthesia* 48: 651-660 (1976).
3. VESEY, C.J. & WILSON, J. Red cell cyanide. *Journal of Pharmacy and Pharmacology* 30: 20-26 (1978).
4. BALLANTYNE, B. *In vitro* production of cyanide in normal human blood and the influence of thiocyanate and storage temperature. *Clinical Toxicology* 11: 173-193 (1977).
5. MACRAE, W.R. Nitroprusside-induced metabolic acidosis. *Anesthesiology* 45: 578-579 (1976).

SIR:

We agree that the maximum cyanide levels in our study are equal to those reported by Vesey, *et al.*¹ in a situation where twice the dose of nitroprusside was administered. In a recently published study, Behnia, *et al.*² showed that in the presence of halothane anaesthesia blood cyanide levels following nitroprusside infusions were lower. In our study 5/13 patients did not receive halothane and the others received minimal quantities because of the use of morphine and relaxants and the nature of the operative procedure. Differences in anaesthetic technique may have contributed to the variation in cyanide level under discussion. Another point for consideration is that the relationship between the measured maximum blood cyanide and the actual peak blood cyanide was not established in either study. We based our conclusion that the peak level occurred within 45 minutes following the end of the infusion on samples collected during the infusion and half hourly thereafter. The mean time of the maximum cyanide level in our study was 15 minutes post-infusion. The sampling pattern in the study of Vesey, *et al.*¹ is too infrequent to permit

us to decide when the peak cyanide level occurred. We can agree that cyanide appeared to be cleared from the blood more quickly in their study than in ours – a further factor which would influence the height of the peak. The use of a semi-closed circuit in our study may have reduced the pulmonary clearance of cyanide.

We agree that *in vitro* cyanide formation from thiocyanate can occur. Because of this the true cyanide values in our patients may have been 5-10 per cent lower than our measured values. This estimate is based on our observed pre-infusion cyanide levels, the absence of a significant change in thiocyanate during our study period and the cyanide levels observed in our control group. It would be in accord with the amount of *in vitro* cyanide formation detected by Vesey and Wilson³ at this plasma cyanide level.

We are unable to comment on the absence of obvious toxicity in the study of Vesey, *et al.*¹ or MacRae⁴ as no indices of tissue hypoxia were presented in their papers. We will refrain from commenting on the unpublished dog study. Our reference to the exacerbation of acidosis when support of ventilation was withdrawn was intended to illustrate how mild degrees of metabolic acidosis can assume clinical significance in post-operative management.

Frances Smith, M.D., Ph.D.
David West, M.B., F.R.C.P.(C).
Department of Laboratory Medicine,
Ottawa Civic Hospital,
1053 Carling Avenue,
Ottawa, Ontario, Canada, K1Y 4E9.

1. VESEY, C.J., *et al.* Cyanide and thiocyanate concentrations following sodium nitroprusside infusion in man. *British Journal of Anaesthesia* 48: 651-660 (1976).
2. BEHNIA, R., *et al.* Metabolism of sodium nitroprusside in dogs awake and anesthetized with halothane. *Anesthesiology* 48: 260-262 (1978).
3. VESEY, C.J. & WILSON, J. Red cell cyanide. *J. Pharm. Pharmacol.* 30: 20-26 (1978).
4. MACRAE, W.R. Nitroprusside-induced metabolic acidosis. *Anesthesiology* 45: 578-579 (1976).

ACUTE EPIGLOTTITIS AND NASOTRACHEAL INTUBATION

SIR:

Acute epiglottitis and nasotracheal intubation still deserves some comments and corrections. In March 1978 (vol. 25, No. 2) this journal dedicated one editorial (1) and one article (2) to this major therapeutic problem.

Doctor A.W. Conn in such an editorial¹ seems to state that the Montreal Sainte-Justine's group "recommends the use of intravenous agents and relaxants as a safe method". I would like to remember the following periods of our article (January 1977) on this subject.³

"Inhalational anaesthesia (halothane) as described by some Australian and European authors is the most convenient and safe technique (references 15-18). Halothane, oxygen and a depolarizing relaxant is another proposed choice (reference 26). Intravenous anaesthesia (thiopentone, diazepam) and succinylcholine after an oxygenation period of three to five minutes, is a rapid technique of anaesthesia (references 21 and 22) which can be associated with the Sellick manoeuvre (reference 32). However, we must stress a warning: less experienced anaesthetists or those not experienced in paediatric anaesthesia should avoid the use of intravenous induction and relaxants in this disease."

That is, we very clearly recommend inhalational anaesthesia (halothane) and we discourage the use of intravenous induction and relaxants in the common management of these patients, in spite of the fact that some experienced members of our staff have employed thiopentone or diazepam and succinylcholine in very safe and convenient conditions. Moreover, a clean cut critic was made against the use of Ketamine.

These corrections being pointed out, I would like to address some comments to Doctor Conn: (1) I have difficulty in justifying two successive tracheal intubations (oral, followed by nasal) in the same patient, as Doctor Conn recommends in his editorial. After all, the great majority of the patients with acute epiglottitis are very conveniently ventilated with a gentle assistance by mask^{2,3,4} and this induces a sufficiently deep stage of anaesthesia to allow the insertion of a nasotracheal tube. (2) "Air-leak" around the tube is not a criterion for extubation, because, unlike laryngitis, small-sized tubes most often allow a continuous leak in epiglottitis cases.²

The best criteria we found for extubation are: disappearance of general manifestations of toxæmia and regression of epiglottic swelling (12 to 72 hours).

As far as the article of Doctors Hannallah and Rosales² is concerned, I would like to give them my best congratulations for the nice contribution they bring in this field, even if in their "review" they decided to forget Sainte-Justine's first Canadian publications on the subject.^{5,3}

In certain cases of epiglottitis with mild respiratory obstruction (grade 1 or 2 of the Hannallah and Rosales grading classification) and with full stomach, I would be much less categorical than they are when stating that: "There is no place for intravenous induction agents or the use of muscle relaxants in these patients."

In any case, what is important is that we agree on several substantial points and, altogether, we recommend inhalational anaesthesia as the most convenient and safe technique in the common management of acute epiglottitis.

Victor Faria Blanc, M.D., F.R.C.P.(C)
Director, Department of Anaesthesia,
Hôpital Sainte-Justine,
Associate Professor, Montreal University,
Montreal (Quebec), Canada.

REFERENCES

1. CONN, A.W. Epiglottitis - editorial - *Canad. Anaesth. Soc. J.* 25(2): 73 (1978).
2. HANNALLAH, R. & ROSALES, J.K. Acute epiglottitis: current management and review. *Canad. Anaesth. Soc. J.* 25(2): 84 (1978).
3. BLANC, V.F., WEBER, M.L., LEDUC, C., LABERGE, R., DESJARDINS, R., & PERRAULT, G. Acute epiglottitis in children: management of 27 consecutive cases with nasotracheal intubation, with special emphasis on anaesthetic considerations. *Canad. Anaesth. Soc. J.* 24(1): 1 (1977).
4. ADAIR, J.C. & RING, W.H. Management of epiglottitis in children. *Anesth. & Anal.* 54: 622 (1975).
5. WEBER, M.L., DESJARDINS, R., PERRAULT, G., RIVARD, G., & TURMEL, Y. Acute epiglottitis in children - treatment with nasotracheal intubation: report of 14 consecutive cases. *Pediatrics* 57: 152 (1976).

SIR:

I wish to thank Dr. Victor Blanc for his comments and criticisms.

His paper does give preference to inhalation anaesthesia with halothane, but some members of his group obviously prefer an intravenous technique. Therefore, a correction should be inserted in the third paragraph of my Editorial, to read "Some members of another group. ..." There is universal agreement that intravenous methods are inappropriate in epiglottitis.

In regard to Dr. Blanc's two comments, my answers are based on discussion of his paper with Dr. Sweeney (Adelaide) and Dr. Barker (Melbourne), who have combined experience with several hundred cases. A preference was expressed for a preliminary oral intubation to establish airway control as early as possible and using the easiest route. The use of a stilette was possible orally in case of difficulty, and the oral tube reduced laryngeal oedema temporarily during deepening of the anaesthesia. Thus, nasal intubation was simplified and safer.

In regard to the presence of an air leak prior to extubation, our combined experience suggests that an air leak may be absent in a proportion of cases following intubation. The subsequent appearance of such a leak confirms a reduction in swelling.

There is no disagreement with Dr. Blanc's emphasis on reduction of toxæmia and epiglottic swelling as important conditions prior to extubation.

I wish to thank Dr. Blanc for his interest and comments.

A.W. Conn, M.D., F.R.C.P.(C)