LETTERS TO THE EDITOR

SODIUM NITROPRUSSIDE METABOLITES

SIR,

We cannot understand why Du Cailar, *et al.*¹ did not use the t-test for paired data in analyzing the changes in base excess as they had for other results. This should be clarified because if this test were applied a significant difference in base excess would have been apparent post infusion. This would alter their conclusion regarding the lack of effect of clinical doses of sodium nitroprusside on acid base status. It would also be informative to know the individual doses of nitroprusside to see if there were any correspondence with the degree of base deficit.

In this study half the patients suffered a fall in pH to the 7.2 range in the post infusion period. We are surprised the authors did not comment on the hazard of postoperative acidaemia.

David West, M.B.

Frances Smith, M.D., Ph.D.

REFERENCE

1. DU CAILAR, J., MATHIEU-DAUDE, J.C., DESCHODT, J., LAMARCHE, Y., & CASTEL, C. Nitroprusside, its metabolites and red cell function. *Canad. Anaesth. Soc. J.*, 25: (2) (1978).

SIR,

The remarks of D. West and F. Smith are justified. The t-test for paired data was not performed for blood gases, acid-base balance, $\text{Da}\bar{\text{v}}\text{O}_2$, 2-3 DPG and total CN^- levels as was done for carbonic anhydrase, SCN^- , lactate and pyruvate. The reason is that the accumulated data was processed by a different statistician in each case. In fact, in paired studies, post-operative variations of B.E. are significant while preoperative values are not. Thus we must conclude that during surgery, there is some degree of metabolic acidosis. In a previous study (*Ann. Anesth. Franç.* 18: 811-821 (1977), of 25 patients, the mean values of B.E. were as follows: before SNP (T1), -2.13, during SNP (T2), -4.91 and after SNP (T3), -3.60. The change at T2 was significant (difference between means) when compared with what was found at T1; this was not true when values found at T3 were compared to values found at T1. The comparison of our two studies will be more valuable because it will be covering a larger number of patients. But even if the most accurate statistical analysis does show significant variations, it cannot be denied that under SNP hypotension, there is a tendency to metabolic acidosis. West and Smith will probably

TABLEAU I

POSOLOGIE DU NPS EN FONCTION DE LA DUREE DE L'ADMINISTRATION

N°	Age (ans)	Duree Perfusion NPS min	Posologie NPS mg	Posologie NPS $\mu\text{g}/\text{kg} \cdot \text{min}$	BE après NPS
1	50	140	10	1.2	-8
2	58	90	12	1.9	-3
3	18	120	32.5	4.9	-7
4	54	120	20	2.1	-8
5	39	75	22	4	-7.5
6	15	90	27.5	10	-8.5
7	70	150	22.5	3.5	-12
8	75	95	15	2.6	-6.5
9	25	255	65	6.4	+1
10	27	60	25	8.7	-5.5
11	25	155	40	6.5	+2
12	48	100	42	5.8	
13	14	250	35	3.3	0
14	42	125	15	2.4	-7
15	67	80	9	2.5	-8
16	27	80	8	2.3	-10
17	14	100	13.5	2.7	-7.5

FIGURE 1 Correlation entre BE et posologie par kg.min de NPS.

agree with us that during major surgery, there are numerous other causes of metabolic acidosis. As of now, it has been shown in many cases that CN-toxicity is associated with high doses of SNP and therefore, we believe that the problem of metabolic acidosis should be studied with utmost care.

As requested by our colleagues, we are enclosing (table I), doses of SNP used during the study. It will be noted that the patients who received the highest doses of SNP do not necessarily show the lowest negative values of B.E. The absence of correlation between dosage per kg/min and B.E. is more interesting (figure 1). Thus our study does not allow us to conclude that a correlation exists between SNP doses and B.E. We intend to study the matter further.

J. du Cailar, *et al.*

BAIN CIRCUIT

SIR,

I was appalled indeed to read Dr. Roger A. Mullin's letter in the May 1978 issue of the *Journal*. The Bain circuit has been the subject of criticism both warranted and unwarranted. However, to incriminate it where the problem lies not with the equipment but with the anesthetist, is totally unjustified. In all fairness, one must point out that the same sequence of events would have followed had a semi-closed circle absorber system been employed.

The simple expedient of checking the system for leaks prior to the induction of anaesthesia would certainly have precluded the occurrence of these complications. Appropriate intraoperative monitoring with an oesophageal stethoscope or a

precordial stethoscope would have gone far toward making the diagnosis early in the course of the anaesthetic. No matter how heavy the draping or distal the field, the anaesthesiologist always must avail himself of an opportunity to view the operative field and arrange the patient in such a manner that at least an extremity is visible at all times. Alas, a poor mechanic blames his tools.

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SIR,

Drs. Rose, Byrick and Foese in their recent paper (Canad. Anaesth. Soc. J. 25: 353 (1978)) challenge the Bain circuit as a universal breathing circuit. Firstly we wish to commend the authors on their very comprehensive and scientific study. They have clearly shown that a near normal carbon dioxide level can be maintained with a fresh gas inflow of 100 ml·kg·min providing a moderate level of hyper-ventilation is maintained. We have previously shown (Canad. Anaesth. Soc. J. 25: 30 (1978)) that the Bain Circuit with a fresh gas flow of 100 ml·kg·min (RMV 20 per cent higher than the circle absorber system, P_{aCO_2} 40 ± 4 mm Hg) is as efficient as the Magill attachment with a fresh gas flow of 70 ml·kg·min. Thus the authors confirmed our clinical research with their lung model. Our clinical experience has shown that the Mapleson D system can be used safely during spontaneous respiration with a fresh gas flow of 100 ml·kg·min in suitable patients anaesthetized to retain an adequate carbon dioxide response. Clinical selection of patients has presented no greater problems than our previous experience with the circle absorber system. Most patients achieve a 20 per cent increase in respiratory minute volume readily without undue effort. The authors proceed with their study very scientifically and show that there is very little difference in P_{aCO_2} (about 5 torr) between a fresh gas flow of 140 ml·kg·min and spontaneous ventilation without any circuit even at the point of respiratory failure (V_d/V_t ratio of 0.65). However, in their summary they go on to conclude that flows of 200 to 300 ml·kg·min are required even for the average adult patient (V_d/V_t ratio of 0.4) and that, unless this is done, the system will produce potentially dangerous hypercarbia. Why do these authors not accept their own experimental evidence that lower fresh gas flows are acceptable to

maintain a near normal Pa_{CO_2} ? Is it not possible for them to question the preordained dogma?

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We need also to define more clearly what we mean by "the paediatric patient", since, by my own admission, even my own upper limit of ten years (in African children, very few of whom weighed as much as 27 kg at that age) may be too high for well nourished Caucasian children.

I would suggest, therefore, that the title is a misnomer, since Soliman, *et al.* are concerned with analgesia rather than anaesthesia, and have not confined themselves to paediatric patients.

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CAUDAL ANAESTHESIA IN PAEDIATRIC PATIENTS

SIR,

As an anaesthetist with experience of 700–800 caudals in children aged 10 years and under, I read the article "Caudal Anaesthesia in Paediatric Patients" by Soliman, Ansara and Laberge with considerable interest.

They appear to have based their dosage regime on the articles by Schulte-Steinberg and Rahlfs which I criticized fairly severely in a letter to the editor of the British Journal of Anaesthesia on the grounds that the testing technique was inadequate, and that levels of blockade could only be suspect when obtained in anaesthetized children. Soliman, *et al.* said, "Using this formula, we found that lidocaine 1% in a dose of 1 ml/year of age will give a block level within the lumbar region in most cases." I found no evidence in their article to substantiate this statement. Only 66 of their patients received lidocaine, and I would not concur in the idea that an 18-year-old is a paediatric patient.

From my own experience, I suspect that there is a change in predictability of spread of caudal anaesthesia when the body weight exceeds about 27 kg, and that while 16 ml of lidocaine 1% might well produce a block into the lumbar segments in a 16-year-old, I should be very surprised if 1 ml did so in a 1-year-old weighing, let us say, 11 kg.

The only reliable way to test levels of blockade is in a readily rousable patient, and I doubt that Soliman, *et al.* had such children. The degree of blockade required for postoperative analgesia (which is what was tested by Schulte-Steinberg and Rahlfs and by Selima, *et al.*) falls short of that needed for anaesthesia for surgery.

HAZARDS OF ELECTRICAL EQUIPMENT

SIR,

As practicing clinical anaesthetists we are all aware of the hazards of the electrical equipment commonly used in the Operating Room.

For a considerable period of time, "on line" equipment that is drawing power from a cable plugged into a wall socket has had to have Canadian Standards Association approval before being used in the Operating Room. In addition, many hospitals have their bylaws and regulations governing such equipment and insist on the examination by their own bioengineers of such articles before being approved for use on patients.

It appears that the Federal authorities are contemplating a major change in their policies to include all battery powered equipment including such items as nerve stimulators or block monitors and cardiac computers, both battery operated instruments.

There is also talk of introducing a "grandfather clause" excluding from this category all equipment in current use purchased before a certain date.

These suggestions of change of policy raise several obvious issues. For example, equipment of identical design and manufacture being purchased before and after the cut-off date, one may be approved and one may not be approved. It also raises the point of responsibility of manufacturers of equipment. Should these electrical hazards and problems be all cleared up before the instrument or equipment is even advertised, and what are the legal responsibilities of these companies, the hospital, and the staff using such equipment if such regulations have been enforced?

Recently we have had recommendations thrust upon us by the Ontario College of Physicians and Surgeons concerning monitoring and ventilatory alarm devices.

Perhaps the Society should consider making recommendations to the Canadian Standards Association and Bioengineering Departments actu-

ally engaged in assessing instrumentation to be also asked to give input before final legislation is enacted.

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