Correspondence

Experience with parents' presence during anaesthesia induction in children

To the Editor:

We were interested to read the paper by Hannallah and Roasales (Can Anaesth Soc J 1983, 30: 286-9) and wish to make further observations on the importance of the parents' presence during anaesthesia induction.

For many years it has been our regular practice to interview the child together with the parents pre-operatively. Every effort is made to gain their confidence and to give adequate explanations about the prospective anaesthetic.¹

The reluctance of some anaesthetists (to allow parents to be present) is overcome by using a "simpler" method of induction. Our personal preference is i.m. Ketamine and scopolamine administered whilst the child is held in a parent's arms in the waiting area.²

Similarly we also encourage the early admission of a parent into the recovery room - thus leaving the overall impression with the child that the parent has been constantly present and also relieving pressure on the recovery room nurses.

We suggest that the two weeks' follow up after surgery is unlikely to be sufficient to reveal the long-term beneficial effects of the intervention for the experimental group or the detrimental effects for the control group.

A most interesting finding in the study, and one that the authors did not discuss, is displayed in Table VII: It reveals a high percentage of children in both groups with emotional difficulties after surgery, indicating the need for a baseline of behaviour prior to surgery for the experimental and control groups.

A need exists for additional intervention such as information, anticipatory guidance, support of care-

givers etc. for the parents and their children, in addition to the presence of parents during induction of anaesthesia.

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Rebreathing and the Bain circuit

To the Editor:

Once again Spoerel (Can Anaesth Soc J 1983; 30: 148-54), in attempting to justify the unjustifiable ($100 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ flow in spontaneous breathing patients) has contributed to the misunderstanding of the mechanics of the Mapleson D breathing system.

While finally agreeing with Mapleson¹ that this system requires a $\dot{V}_E/\dot{V}_F$ ratio of at least 0.5, he also states that FiCO_2 is "passive" and does not contribute to CO_2 homeostasis. I do not think that Spoerel has clearly supported this dangerous concept, which, if carried to its logical conclusion, may be interpreted as inferring that rebreathing is not a significant contributor to high PaCO_2 under anaesthesia!

Really though, it's about time that this spontaneous versus controlled respiration concept was finally discarded.

This and other papers^{2,3} on similar aspects of

Mapleson D circuit mechanics confirm the need to differentiate between the patient and the breathing system when considering CO₂ elimination.

Alveolar ventilation remains the only way to remove CO₂ from the patient; whether or not CO₂ is removed from the Bain or any other version of the Mapleson D system depends upon the relationship between fresh gas flow and the duration of the expiratory phase. Whether the ventilation is spontaneous or controlled is irrelevant.

If this flow/time relationship is appropriate, V_F may approach V_E but, if either expiratory time or fresh gas flow is too low, rebreathing will occur and *must* hinder CO₂ washout. Spoerel may regard this as a passive phenomenon if he wishes, it certainly is inevitable. It can equally certainly be regarded as a CO₂ load.

Spoerel's own data confirm this view as Figures 4 and 6 (p. 152-153) demonstrate:

A Pre-naloxone $f = c 10 \cdot \text{min}^{-1}$, $\dot{V}_E = 70 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, $F_{ET}\text{CO}_2 = 7.5$, $F_{i}\text{CO}_2 = 0.6$, $\dot{V}_F = 100 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, $t_{Exp} c 3.5 \text{ sec}$.

B Post-naloxone $f = 26 \cdot \text{min}^{-1}$, $\dot{V}_E = 133 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, $F_{ET}\text{CO}_2 = 6.75$, $F_{i}\text{CO}_2 = 3.5$, $\dot{V}_F = 100 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, $t_{Exp} < 1 \text{ sec}$.

A Alveolar hypoventilation - $\dot{V}_A c 50 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, $F_{ET}\text{CO}_2 7.5$, but at $\dot{V}_F 100 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ and $t_{Exp} 3.5 \text{ sec}$, adequate CO₂ washout of the circuit $F_{i}\text{CO}_2 0.6$.

B Alveolar ventilation - apparently adequate $\dot{V}_A 91 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ (approximately $2 \times A$) but at $\dot{V}_F 100 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ and $t_{Exp} < 1 \text{ sec}$, inadequate CO₂ washout. $F_{ET}\text{CO}_2 6.75$ (despite 80 per cent increase in $\dot{V}_A$).

Is it really possible to say that a $\dot{V}_F$ of $100 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ is adequate for CO₂ elimination here or that the $F_{i}\text{CO}_2$ of 3.5 per cent does not contribute to CO₂ retention ($F_{ET}\text{CO}_2 6.75$) and hence is not a CO₂ load?

The Mapleson D system as originally designed by Philip Ayre⁴ was simple in construction and, being valveless, had minimal expiratory resistance, both desirable features for the paediatric and neurosurgical applications he intended. It was never expected to be either economical or to be used with "fixed" fresh gas flow rates.

Mapleson recognized almost 20 years ago that non-rebreathing in this system required correlation with the patient's minute ventilation ($>2 \times$). Since his own data, as well as that of many others,

confirms this, perhaps Dr. Spoerel will recognize it too!

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- 2 Byrick RJ. Can Anaesth Soc J 1980; 27: 96.
- 3 Humphrey D. The Lack, Magill and Bain anaesthetic breathing system: a direct comparison in spontaneously breathing anesthetized adults. Journal of the Royal Society of Medicine 1982; 75: 513-24.
- 4 Ayre P. Lancet 1937; 1: 561.

REPLY

Thank you for the opportunity to reply to Dr. Imrie's letter. I am distressed by the thought that I may have repeatedly contributed to the misunderstandings of the Mapleson D breathing system. It was all done with the good intention to make observations and to report data which should eventually lead to an end of the confusion about which Dr. Imrie complains in his letter. Why is this unjustifiable?

I am not aware that I ever disagreed with Mapleson that a $\dot{V}_E:\dot{V}_F$ ratio of about 0.5 was required to avoid rebreathing, as indicated by the presence of CO₂ during inspiration ($F_{i}\text{CO}_2$). But a ratio has two sides; I showed in my paper that the same ratio (i.e., fresh gas flow [$\dot{V}_F$] equals two times minute ventilation [$\dot{V}_E$]) can also be achieved and rebreathing be avoided by a suitable depression of $\dot{V}_E$ with a narcotic.

Furthermore, since the tidal volume varies from breath to breath in spontaneous respiration, the $\dot{V}_E:\dot{V}_F$ ratio and also $F_{i}\text{CO}_2$ change with each breath. This can be readily demonstrated in the capnogram of a conscious person breathing through a Bain system with a $\dot{V}_F$ of $100 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ and voluntarily alternating slow normal breathing with short periods of marked hyperventilation (see Figure); the $F_{i}\text{CO}_2$ changes with the breathing pattern but does not alter the end-tidal CO₂.

The association of a raised $F_{i}\text{CO}_2$ with a near normal PaCO_2 in spontaneous breathing under anaesthesia which I reported, was also seen by Byrick¹ who found no correlation between PaCO_2 and a calculated inspiratory CO₂ load. Meakin and Coates² evaluated spontaneous breathing with the Bain system and remarked: "paradoxically, those patients in whom the degree of rebreathing was greatest had the lowest end-tidal CO₂." If rebreathing in the Bain system would actively contribute to hypercarbia, as assumed by Dr. Imrie, it has yet to be documented; the presently available evidence supports the "dangerous" concept that the $F_{i}\text{CO}_2$ does not affect CO₂ homeostasis.

The assumed danger of rebreathing led to the demand