LETTERS TO THE EDITOR

SPONTANEOUS BREATHING WITH BAIN CIRCUIT

SIR,

Spoerel, in his article concerning spontaneous respiration during anaesthesia with the Bain circuit (Canad. Anaesth. Soc. J. 25: 30, 1978), attributes the increased respiratory minute volume and low mean P_{aCO_2} values of his patients to light anaesthesia and surgical stimulation. These unpremedicated patients received thiopentone for induction and were then maintained on nitrous oxide/oxygen and one per cent halothane breathing spontaneously with an endotracheal tube. Munson and others,¹ when using halothane alone at 1.1 per cent with a circle system, reported a mean P_{aCO_2} of 60.1 torr in spontaneously breathing unpremedicated healthy patients. With surgical stimulation, the mean P_{aCO_2} decreased approximately 5 torr from the above level.

Grundy and others² have also reported elevated P_{aCO_2} values in premedicated patients undergoing surgery while breathing spontaneously under halothane (concentration not specified) and 50 per cent nitrous oxide/oxygen anaesthesia. Using a Bain circuit, we recently (unpublished) studied a group of unpremedicated healthy patients, many induced with inhalation agents only, who were maintained spontaneously breathing via endotracheal tube on less than 0.7 per cent halothane with 50 per cent nitrous oxide/oxygen mixture. We found it rare during surgery to have a P_{aCO_2} less than 40 torr even at excessive fresh gas flow rates (15,000 ml/m²/min). In spite of our light anaesthesia, some patients still had P_{aCO_2} values in the mid to high 40s.

The mean respiratory minute volume in our patients was less than 6 litres at the high fresh gas flow rates. Guyton states that the average respiratory minute volume in a healthy (unanaesthetized) young adult male averages 6 litres per minute.³ Conway and others⁴ had similar values for awake volunteers breathing spontaneously on the Bain circuit. Munson and others¹ state that several investigators have demonstrated a diminished ventilatory response in man during halothane anaesthesia and, therefore, assisted ventilation during light levels of halothane anaesthesia is necessary if a normal level of P_{aCO_2} is to be obtained.

I am curious to know if Dr Spoerel has any

thoughts as to why the respiratory minute volume and P_{aCO_2} values in his patients differ from the data cited above.

Robert L. Rayburn, M.D.
Major, M.C.
Anesthesiology & Operative Service
(AFZG-MDS-A)
Brooke Army Medical Center
Fort Sam Houston, Texas, 78234

REFERENCES

1. MUNSON, E.S., LARSEN, C.P., BABAD, A.A., REGAN, M.J., BUECHEL, D.R., & EGER, E.I. The effects of halothane, fluoroene, and cyclopropane on ventilation: a comparative study in man. *Anesthesiology* 27: 716 (1966).
2. GRUNDY, E.M., SHIN, M.Y., BENNETT, E.J., & PATEL, K.P. The effect of assisted ventilation on arterial carbon dioxide. *Canad. Anaesth. Soc. J.* 24: 390 (1977).
3. GUYTON, A.C. *Textbook of Medical Physiology*. Third Edition. Philadelphia. Saunders, 552 (1966).
4. CONWAY, C.M., SEELEY, H.F., & BARNES, P.K. Spontaneous ventilation with the Bain anaesthetic system. *Brit. J. Anaesth.* 49: 1245 (1977).

SIR,

We appreciate the opportunity to reply to the letter by Major Rayburn, who is concerned about the CO_2 levels reported in our patients in comparison to other studies. Munson and co-workers¹ observed a progressive depression of the CO_2 response with increasing depth of anaesthesia and stated that with approximately equal anaesthetic levels the degree of depression varied from agent to agent; their study was done on unstimulated patients anaesthetized with one anaesthetic agent only and they monitored the alveolar concentration of the agent used. The inspired halothane concentration of one per cent, which we reported, would result in an alveolar concentration of 0.7–0.8 per cent, a range for which Munson, *et al.* reported a P_{aCO_2} of 48 mm Hg. Grundy and others² found a mean P_{aCO_2} of approximately 50 ± 10 mm Hg in eighteen premedicated children with a mean age of four years, breathing spontaneously under anaesthesia with an unspecified halothane concentration. In our opinion, neither of the two papers contain data recorded under conditions which permit a direct

comparison of absolute values with ours. However, the finding by Munson and others of a reduction of the P_{aCO_2} with surgery would support our assumption that patients undergoing painful stimulation have a better CO_2 response.

In conducting this study with young patients between 18 and 30 years of age, uniformly anaesthetized for the removal of two to four impacted wisdom teeth (a procedure which should produce a similar intensity of pain in each patient), we felt that we had a fairly controlled situation that would be reproducible. In addition, we could expect the highest CO_2 production in adult patients in this age group and it seemed most likely that here a state of hypercapnia would occur if the inadequacy of the breathing circuit forced the patient to retain CO_2 with the volumes of fresh gas supplied. Our concern was whether spontaneously breathing patients could compensate for the limitations imposed by the restricted gas flow. The observations by Conway and others³ on conscious volunteers are not really pertinent; they determined the lowest fresh gas flow which would prevent rebreathing completely. On the other hand Willis, Pender and Mapleson⁴ showed that in conscious volunteers a reduction of the fresh gas flow to 1.5 times the respiratory minute volume induced only a 10 to 20 per cent increase in ventilation; this fresh gas flow was well below the level where Conway³ and others observed the onset of rebreathing. Even a reduction of fresh gas flow to equal the respiratory minute volume could be compensated by an increase of ventilation by 50–60 per cent. It was interesting to note that under our conditions patients were able to produce a compensatory effort of this magnitude under nitrous oxide-halothane anaesthesia.

With the Magill Circuit which is recommended for spontaneous respiration and fresh gas flows in the magnitude of those used by Kain and Nunn,⁵ we obtained the same P_{aCO_2} and the same minute ventilation as with the Bain Circuit with a fresh gas flow of 100 ml/kg/min; we assumed on this basis that suitably anaesthetized patients could be allowed to breathe spontaneously with the Bain Breathing Circuit and a fresh gas flow of 100 ml/kg/min. This statement does not contradict the conclusions by Munson *et al.*, that assisted ventilation is necessary under anaesthesia if a normal level of P_{aCO_2} is to be maintained consistently. Our findings only indicate that under these conditions an elevated P_{aCO_2} is likely not due to the circuit as such. Underventilation raises the alveolar CO_2 level with any breathing circuit; if

the respiratory minute volume is smaller than the fresh gas flow per minute then a further increase in fresh gas flow alone will only remove more CO_2 from the circuit but not from the lungs and therefore not change the alveolar CO_2 tension. Under these conditions excessive fresh gas flows, as mentioned by Major Rayburn, cannot compensate for the depressed respiration and would have no effect on the patient's P_{aCO_2} unless the volume of ventilation is increased.

J.A. Bain, M.D.

W.E. Spoerel, M.D.

REFERENCES

1. MUNSON, E.S., LARSEN, C.P., BABAD, A.A., REGAN, M.J., BUECHEL, D.R., & EGER, E.I. The effects of halothane, fluroxene, and cyclopropane on ventilation: a comparative study in man. *Anesthesiology* 27: 716 (1966).
2. GRUNDY, E.M., SHIN, M.Y., BENNETT, E.J., & PATEL, K.P. The effect of assisted ventilation on arterial carbon dioxide. *Can. Anaesth. Soc. J.* 24: 390 (1977).
3. CONWAY, C.M., SEELEY, H.F., & BARNES, P.K. Spontaneous ventilation with the Bain anaesthetic system. *Brit. J. Anaesth.* 49: 1245 (1977).
4. WILLIS, B.A., PENDER, J.W., & MAPLESON, W.W. Rebreathing in a T-piece; volunteer and theoretical studies of the Jackson Rees modification of Ayre's T-piece during spontaneous respiration. *Brit. J. Anaesth.* 47: 1239 (1975).
5. KAIN, M.L. & NUNN, J.F. Fresh gas economics of the Magill Circuit. *Anesthesiology* 29: 964 (1968).

EMPTY TANKS AND BAIN CIRCUITS

SIR,

I recently visited a hospital using Bain Circuits exclusively, with pipeline oxygen and nitrous oxide tanks mounted on the gas-machines. A nitrous oxide tank emptied, with some delay in detecting this, and turning on the second tank. In this event, fresh gas inflow falls by about two-thirds, with instant re-breathing of CO_2 , plus rapid loss of analgesia. There is no alarm for this occurrence, except a high-limit oxygen analyser, but the incidence can be reduced to near zero by the use of large tanks for nitrous oxide, chained in a safe position, with a proper pin-indexed hose to one of the nitrous oxide hangers.

A.J. Dunn, M.D., F.R.C.P.(C)

Department of Anaesthesia

St. Michael's Hospital

30 Bond Street, Toronto, M5B 1W8