anesthetists consider acceptable. Inspired P_{CO_2} was determined by averaging the *highest* inspired tensions (their italics). Humphrey[2] has taken inspired P_{CO_2} to be the *minimum* inspired CO_2 tension and, furthermore, regarded a rise from zero to 2 mmHg (0.3%) as the end-point. The reason for this difference may be explained by illustrating the changes of carbon dioxide tension during inspiration with a Mapleson D system. Figure 1, after Nott, Walters and Norman,[3] shows breath-by-breath levels of CO_2 during spontaneous breathing with the Bain circuit in a 70-kg adult. The highest inspired CO_2 tension is indicated at (a) and the minimum at (b) for one breath. The pattern is not constant and varies with the proportion of fresh gas from the supply that is added to expired alveolar gas from the previous breath. These changing CO_2 levels are mirrored by the oxygen concentrations.

Dean and Keenan conclude that a fresh gas flow of twice the minute volume, or $\dot{V}_F/\dot{V}_E$ greater than 1.89, probably will avoid rebreathing but in as many as 13% of patients this will not be so. Their initial flow rates were high enough for an inspired CO_2 tension of 0%. Can the authors review the records and compare here the $\dot{V}_F/\dot{V}_E$ ratio if rebreathing is taken to be a rise of 2 mmHg in the mean lowest inspired CO_2 tension?

Lastly, we suggest that a circuit which requires such high flows in spontaneous breathing is indeed wasteful and that a Mapleson A system will be more appropriate!

M. R. NOTT, F.F.A.R.C.S.
Consultant, Department of Anaesthetics, Royal West Sussex Hospitals, St. Richard's, Chichester, England

J. NORMAN, PH.D.
Professor of Anaesthetics, University of Southampton and The General Hospital, Southampton, England

REFERENCES

1. Dean SE, Keenan RL: Spontaneous breathing with a T-piece circuit. ANESTHESIOLOGY 56:449–452, 1982
2. Humphrey D: The Lack, Magill and Bain anaesthetic breathing systems. J R Soc Med 75:513–524, 1982
3. Nott MR, Walters FJM, Norman J: The Lack and Bain systems in spontaneous respiration. Anaesth Intensive Care 10:333–339, 1982

(Accepted for publication October 20, 1982.)

Anesthesiology
58:393–394, 1983

In reply:—We thank Drs. Nott and Norman for their interesting comments. Ideally, one should use *mean* inspired P_{CO_2} as a measure of rebreathing, but the methodology available to most of us does not allow it. One therefore is forced to use the *minimum* inspired P_{CO_2} (as did Nott *et al.*[1] and Humphrey[2]), or the *highest* inspired P_{CO_2}, as we did. Because the former will be lower and the latter higher than the *mean* inspired P_{CO_2}, we chose the latter since it would tend to overestimate rebreathing. We chose a value of between 5 and 10 mmHg because it was readily identifiable and, as we noted in our paper,[3] we observed that it was not associated with any increase in either end-tidal P_{CO_2} or minute volume ventilation. Thus, we are confident that significant rebreathing at the alveolar level did not occur in our study.

We agree that a *mean* inspired P_{CO_2} of 10 mmHg (1.4%) would represent significant rebreathing, as Nott and Norman suggest. But the mean inspired P_{CO_2} was no doubt well below that figure in our study. Most of our subjects produced a respiratory CO_2 waveform similar to that seen in the far left panel of the figure furnished by Nott and Norman. The highest inspired P_{CO_2} tended to occur late in inspiration, when respiratory flow was slow and probably contributed little to alveolar ventilation. Interestingly, in that same waveform referred to above, the minimum inspired P_{CO_2} appears to be 4 mmHg (which would represent mild rebreathing to Nott and Norman) while the highest inspired P_{CO_2} is 11 mmHg, which would represent a mild degree of rebreathing to us. Thus, we might have different end-points but they seem to be compatible. We also note that, in that same figure, the ratio of $\dot{V}_F$ (143 $ml \cdot kg^{-1} \cdot min^{-1}$) to $\dot{V}_E$ (97 $ml \cdot kg^{-1} \cdot min^{-1}$) is 1.47. This too is entirely compatible with our finding that a slightly higher value of $\dot{V}_F/\dot{V}_E$ (1.87 ± 0.27) is needed to prevent rebreathing.

We cannot answer the question regarding the $\dot{V}_F/\dot{V}_E$ ratio at which a rise of 2 mmHg in the lowest inspired P_{CO_2} was seen, because the observed value of minimum inspired P_{CO_2} was zero in many of our 30 subjects when the highest inspired P_{CO_2} was between 5 and 10 mmHg.

Whether a Mapleson A is preferable to a Mapleson D system is a matter of opinion. We like the simplicity and versatility of the Mapleson D system and are unpersuaded that high flows are "wasteful" in any meaningful way.

STEVEN E. DEAN, M.S.
RICHARD L. KEENAN, M.D.
Professor and Chairman
Department of Anesthesiology
Medical College of Virginia
Richmond, Virginia 23298

REFERENCES

1. Nott MR, Walters FJM, Norman J: The Lack and Bain systems in spontaneous respiration. Anaesth Intensive Care: 10:333–339, 1982.
2. Humphrey D: The Lack, Magill and Bain anaesthetic breathing systems. J R Soc Med 75:513–524, 1982
3. Dean SE, Keenan RL: Spontaneous breathing with a T-piece circuit. ANESTHESIOLOGY 56:449–452, 1982

(Accepted for publication October 20, 1982.)

Anesthesiology
58:394, 1983

A Double-lumen Right Atrial Catheter for Open Heart Surgery

To the Editor:—Monitoring of right atrial pressure (RAP) using a central venous catheter (CVC) is desirable in patients undergoing open heart surgery. The need to use a CVC simultaneously for pressure monitoring and drug or fluid infusion frequently arises. Since the insertion of a second CVC can be time-consuming and carries with it increased risks,[1–5] we developed a double-lumen catheter that by itself will allow continuous pressure monitoring with simultaneous administration of drug infusion or blood sampling.

A tapered, radiopaque 7F double-lumen catheter was developed that is 20 cm long with a 5-cm proximal port.* The distal lumen may be used for blood sampling or for drug infusion while the smaller proximal lumen may be used for pressure monitoring. The catheter is inserted by Seldinger technique, using a standard 0.035-inch guide wire inserted through the larger distal lumen. The catheter serves as its own dilator and no sheath introducer system is necessary for its insertion. Both lumens are filled with a heparinized saline solution in order to prevent clotting.

The catheter has been employed without complications in ten patients undergoing open heart surgery. The right internal jugular vein has been used in all instances. We have been able to monitor RAP accurately and continuously without interference even during blood sampling or drug infusion. It is felt that this method is reliable, convenient, and useful when RAP monitoring and simultaneous drug infusion or blood sampling are required.

JOSE MARQUEZ, M.D.
JOHN J. SASSANO, M.D.
PETER M. WINTER, M.D.
PETER M. WATERMAN, M.D.
Department of Anesthesiology
Presbyterian-University Hospital of Pittsburgh
DeSoto at O'Hara Streets
Pittsburgh, Pennsylvania 15213

* Edwards Laboratory.

REFERENCES

1. Knoblanch GE: Respiratory obstruction due to haematoma following internal jugular vein cannulation. Anaesth Intensive Care 7:286, 1979
2. Brown CS, Wallace CT: Chronic hematoma—A complication of percutaneous catheterization of the internal jugular vein. ANESTHESIOLOGY 45:368–369, 1976
3. Wisheart JD, Hassan MA, Jackson JW: A complication of percutaneous cannulation of the internal jugular vein. Thorax 27:496–499, 1972
4. James RM, Bevis A, Myers RT: Experiences with central venous and pulmonary artery pressure in a series of 3,500 patients. South Med J 65:1299–1307, 1972
5. Dailey PO, Griepp RB, Shumway NE: Percutaneous internal jugular vein cannulation. Arch Surg 101:534–536, 1970

(Accepted for publication October 20, 1982.)

Anesthesiology
58:394–395, 1983

Thiobarbiturates Induce Arrhythmias in Dogs

To the Editor:—We would like to comment on the recent paper by Smith and Dresel[1] in which they describe the possible origin of epinephrine-induced arrhythmias in halothane-anesthetized dogs. It is well-recognized that thiobarbiturates often induce ventricular bigeminy which is coupled to the preceding sinus beat in dogs.[2] While it has been reported that thiamylal sodium causes these arrhythmias more frequently than thiopental sodium, both agents will cause ventricular bigeminy when given at anesthetic dosage.[2] The clinical importance of this arrhythmia in the dog is in dispute at the present time. The cause has been related to increased arterial pressure, the concentration, and dose of thiobarbiturate administered.[2,3] In addition, the