CPAP Oxygenation during One-lung Ventilation Using a Bain Circuit

To the Editor:—Hypoxemia is a commonly encountered problem during one-lung ventilation. Non-dependent lung continuous positive airway pressure (CPAP) has been shown to significantly improve oxygenation in those patients in which this problem occurs. Multiple devices have been described to deliver CPAP to the non-dependent lung. We suggest the use of a Bain circuit to deliver CPAP to the nonventilated, i.e., non-dependent, lung.

The fresh gas inlet of the Bain circuit (coaxial modification of a Mapleson D circuit) is connected to any oxygen source with a flowmeter (we use an E cylinder oxygen tank) and the oxygen flow is set at 1–2 LPM. The circuit is connected to the non-ventilated lumen of the double-lumen endotracheal tube, and the overflow valve on the Bain circuit is set at the appropriate closure to yield the desired amount of CPAP, which is read from the manometer on the Bain apparatus (fig. 1). Functionally, this behaves exactly as the system described by Thiagarajah et al.

However, the items, a Bain circuit and an E cylinder oxygen tank, are perhaps more readily available and preclude an extensive search of the spare parts box for components necessary to produce CPAP in the non-dependent lung.

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(Accepted for publication December 31, 1986).

Anesthesiology
66:709, 1987

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Volatile Anesthetics and Drug Serum Protein Binding

To the Editor:—Volatile anesthetic agents are potent drugs—as a consequence of both action and interaction. Drug interaction studies in the presence of inhalational anesthetics have mainly been directed toward the pharmacodynamic outcome, and little attention has been paid to what might happen to a drug before it reaches the receptors upon which it acts.

It has recently been reported that inhalational anesthetics significantly change the pharmacokinetics of verapamil.12 The authors demonstrate the necessity of measuring drug concentrations, not only in pharmacokinetic studies, but also when pharmacodynamics are examined. We believe that their findings emphasize the need for more studies on the mechanisms of such interactions, and welcome the contribution of Chelly et al.3

We do, however, feel that attention should be drawn to one of the conclusions from this latter paper, which stated that “Alterations in protein binding cannot be invoked to explain the pharmacokinetic interaction.” This statement may well be true, but is probably exceeding the capacity of the presented data. Apparently, the protein binding experiments have been performed in the absence of the interacting agents. We fail to see that appropriate precautions are described to prevent the disappearance of the inhalation anesthetics from the samples prior to or during equilibrium dialysis. This is, in our opinion, crucial, since inhalational anesthetic agents are fully capable of altering drug binding to serum and tissue proteins by direct action.4–7 The reported absence of an alteration of verapamil binding to plasma proteins is compatible with the absence of the inhalational anesthetics in this otherwise well-performed and interesting study.

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(Accepted for publication January 14, 1987.)