

## Reports of Scientific Meetings

Ellis N. Cohen, M.D., Editor

### Low-flow and Closed-system Anesthesia

A recent symposium on low-flow and closed-system anesthesia held at the University of Colorado School of Medicine, Denver, Colorado, April 1978, demonstrated a renaissance of interest in low-flow anesthetic systems. This has resulted from concern over anesthetic pollution and the recognition that closed-system anesthesia offers a sensitive and often quantitative, noninvasive monitor of changes in metabolic status, cardiac output, and other dose-response effects in the course of anesthesia.

Oxygen consumption ( $\dot{V}_{O_2}$ ) in a closed system has been used since 1868 to measure metabolic activity. By keeping the monitored oxygen concentration constant, the oxygen flowmeter provides quantitative information about the patient's metabolic status. Oxygen utilization decreases in hypothermia, hypothyroidism, cardiac arrest, and pulmonary shunting, and with use of an extremity tourniquet. Its decrease is also a very early sign of pending hypotension, sometimes detectable 10 to 20 minutes before changes in blood pressure occur and with volume losses as small as 10 per cent.  $\dot{V}_{O_2}$  is increased by anxiety, fever, light anesthesia, and release of an extremity tourniquet. It is also a measure of the adequacy of resuscitation, as base deficit is corrected when the increased oxygen uptake satisfies the oxygen debt generated by hypoxia or ischemia, *i.e.*, when  $\dot{V}_{O_2}$  returns to normal.

Indirect measurement of arteriovenous oxygen difference by use of inspired and expired breath analysis and knowledge of  $\dot{V}_{O_2}$  from the flowmeter settings permit the use of the Fick equation for the calculation of cardiac output in the absence of hypoxic states.  $CO_2$  or  $N_2O$  could be used in a similar manner.

For a closed-system anesthetic,  $\dot{V}_{O_2}$  may be estimated from ten times the patient's metabolically active mass, *i.e.*, the patient's lean weight (kg) raised to the three-fourths power (Brody). A 70-kg patient's  $\dot{V}_{O_2}$  is 242 ml/min ( $70^{3/4} = 24.2$ ). Metabolically active mass relates to many useful physiologic modalities.:

Minute alveolar ventilation (ml/min) (to maintain  $P_{ACO_2} = 40$  torr) is  $160 \times kg^{3/4}$

$$CO_2 \text{ production (ml/min)} = 8 \times kg^{3/4}$$

$$\text{Basal water requirement (ml/hr)} = 5 \times kg^{3/4}$$

$$\text{Cardiac output (100 ml/min)} = 2 \times kg^{3/4}$$

$$\text{Basal glucose utilization (g/hr)} = 0.75 \times kg^{3/4}$$

Measurement of  $\dot{V}_{O_2}$  also permits precise control of alveolar carbon dioxide concentration from:

$$V_t = \frac{\dot{V}_{CO_2}}{f \cdot F_{ACO_2}} + V_D + PC_{equipment}$$

where

$V_t$  = tidal volume (ml)

$\dot{V}_{CO_2}$  = carbon dioxide production (ml/min)

$f$  = desired frequency of ventilation

$F_{ACO_2}$  = desired alveolar concentration of  $CO_2$  ( $= P_{ACO_2} / (P_B - P_{H_2O})$ ) *e.g.* 0.056 for  $P_{ACO_2} = 40$  torr

$P$  = circuit pressure (cm  $H_2O$ )

$V_D$  = 1 ml/kg in a patient whose trachea is intubated

$C_{equipment}$  = equipment compliance (10 ml/cm  $H_2O$  of circuit pressure with rubber hoses and 5 ml/cm  $H_2O$  with plastic hoses)

This calculation assumes that the respiratory quotient is 0.8 and that alveolar  $CO_2$  approximates arterial.

While closed-system anesthesia is not new, its use as a monitor and its quantitative delivery are innovative. Dr. Harry Lowe deserves much credit for making this approach accessible. Whole-body anesthetic uptake is constant for the intervals between consecutive square numbers in minutes since commencing the anesthesia, *e.g.*, the same dose is needed for the interval between 1 and 4 minutes as is needed for the interval between 81 and 100 minutes. This relationship is an approximation rather than a logical process implicit in drug distribution. The dose may be given by a vernier control vaporizer, a syringe pump regulated by a computer, or a liquid bolus from a syringe for each period, or several interval doses may be averaged and given in fractions at regular intervals (*e.g.*, during induction). The dose required for an interval between squares is given from:

$$\text{Dose} = 2 \times AD_{95} \times \lambda_B \times \dot{Q}_c$$

where

$AD_{95}$  = 1.3  $\times$  minimum alveolar concentration, *i.e.*, the dose to anesthetize 95 per cent of patients

$\lambda_B$  = blood-gas solubility coefficient

$\dot{Q}_c$  = cardiac output (dl/min): approximately 0.7 dl/kg or  $2 \times kg^{3/4}$

A 70-kg patient receiving halothane- $O_2$  anesthesia would need 222 ml of halothane vapor or 1 ml of liquid for the period between time squares. If the patient were receiving  $N_2O$ - $O_2$ -halothane anesthesia at 0.65 MAC (*i.e.*,  $AD_{95}/2$ ) of  $N_2O$ , the halothane dose would be 111 ml, as MAC's are additive. Doses required clinically may be slightly higher, as fresh soda lime and rubber hoses also compete for the patient's anesthetic by absorption.

Depth of anesthesia is best evaluated in the customary manner, beautiful mathematics notwithstanding! As with a semiclosed system, hypotension during induction is the

result of cardiac depression, vasodilation, or inadequate stimulation. Only the dynamics of administration are changed; not those of uptake and distribution. The administration of a fixed mass of drug permits the use of a smaller amount of total anesthetic and a faster, predictable emergence resulting from minimal body storage of anesthetic agent.

Both the advantages and the hazards of closed-system anesthesia are vastly overstated. Waters developed this technique to minimize saturation of his own tissues with the aroma of ether, to reduce the hazard of explosion with flammable agents, and for economy. Subsequently, it has been espoused to maintain pulmonary humidity and body heat, and to prevent pollution. In American operating rooms the savings are approximately \$2.50 per case in comparing N<sub>2</sub>O-O<sub>2</sub>-halothane anesthetics at 5-l flow with the closed system. Were the latter used in every case, it would save \$20,000-30,000 annually in a busy ten-room operating suite, and perhaps more in terms of global pollution effects. Only 0.5 per cent of atmospheric halogen pollution can be attributed to anesthetics. The implication this small amount may have for the ozone layer is uncertain. Closed-system anesthesia does not eliminate the need for pollution monitoring in the operating room, as leaks from high-pressure wall lines may occur. In addition, one often desires to use high flows even in conjunction with a closed system, *e.g.*, to speed induction or emergence. Therefore, scavenging of waste gases is relevant to permit flexibility in the choice of anesthetic technique.

A low-flow system with flows greater than for a closed system, but no greater than 1 liter total flow, provides the convenience of dispensing with the mathematics of a closed system, but maintains economy, minimizes pollution, and provides a sensitive ventilatory measure of depth of anesthesia or relaxation. Small changes in ventilation are nearly as obvious here as with a closed system, whereas they would be obscured by a routine 5-l flow.

Of the hazards, the most serious is overdose. This commonly results from miscalculation of the dosage on the basis of the patient's gross weight. In an obese patient, the dose intended for the fat compartment would go to the vessel-rich compartment if given early in the course of anesthesia, although the calculated dose would eventually be required by the fat. This may be corrected by dosing initially on the basis of the lean mass:

$$\text{Per cent body fat} = 90 - 2(H - G)$$

where

H = height (inches)

G = girth (inches)

$$\text{Lean mass} = \text{weight} - (\text{weight})(\text{per cent fat})$$

When halothane was introduced, several catastrophes resulted from its use in closed systems with in-line vaporizers, as the potency of the agent was not appreciated. Deaths also resulted from vernier-controlled vaporizers in closed systems because their efficiency was not recognized. Bolus doses of 1 ml liquid halothane may

appear to pose a similar hazard at first glance. Overdosage is perhaps less of a problem than in a high-flow system. The dose is administered in the expiratory limb so that mixing may occur in the CO<sub>2</sub> absorber. A 1-ml bolus provides only 200 ml vapor, which in an 8-l circuit would increase the inspired concentration by no more than 2.5 per cent after an ephemeral period of equilibration. The increase would be transient because the absolute quantity is limited and the inspired concentration is altered by tissue uptake. A further safety feature is that accumulation of unused agent in the circle is slow. As with a semi-closed system, overdose may be managed effectively by increasing the gas flow, and by using dose volumes guided by clinical signs and an anesthetic gas monitor rather than extrapolations for the average patient.

The availability of oximeters has been an essential boon to low-flow anesthetic systems by permitting the use of N<sub>2</sub>O in a safe manner at total flows less than 1 l. Prior to the use of these devices there was the hazard that an oxygen demand in excess of predicted need would not be detected if the reservoir bag were filled with N<sub>2</sub>O. Oximeters also permit the safe administration of large volumes of N<sub>2</sub>O relative to O<sub>2</sub> early in the course of a closed-system technique when the former's uptake is quite rapid. Unfortunately, oximetry permits estimation of N<sub>2</sub>O concentration only by subtraction, and therefore is not sensitive to N<sub>2</sub> accumulation from tissue stores (tissue N<sub>2</sub> = 14 ml/kg). A more serious source of N<sub>2</sub> accumulation is an air leak in a mechanical ventilator. This problem is clinically minimized by adequate preoxygenation, periodic venting of the anesthetic circle, or the use of a 100 per cent O<sub>2</sub>-halogenated anesthetic technique. Direct N<sub>2</sub> measurement, *e.g.*, by mass spectrometry, would obviate this minor problem.

Carbon monoxide accumulation from endogenous production has been suggested as a hazard of closed-system anesthesia. Dr. P. V. Cole *et al.* presented data showing that although CO accumulates in the anesthetic system, carboxyhemoglobin concentrations actually decreased due to dissociation caused by exposure to the high FI<sub>O<sub>2</sub> used during anesthesia.</sub>

High-flow systems offer much in terms of easy control of alveolar gas composition, and are especially attractive for use during induction, when anesthetic uptake is great and is rapidly changing. However, after an initial 9-16-min period, a closed system is quite practical.

In concluding the conference, Dr. Lowe noted that "high-flow systems cover the patient with a blanket of complacency, ignorance, and mental stagnation." The mathematical convolutions of closed-system anesthesia are at once fascinating and forbidding. They perhaps limit its use to long, tedious cases. Once mastered, however, the technique is addicting and enlightening, because of its marked sensitivity to the elements involved in vigilant anesthetic management.

BERNARD S. MILLMAN, M.D.  
*Assistant Professor of Anesthesia  
Stanford University Medical Center  
Stanford, California 94305*