I have found the Polio blade helpful when endotracheal intubation must be accomplished after the patient has been prepped and draped, the anesthesia screen becoming a factor limiting access to the airway. In this situation, endotracheal intubation may be difficult with conventional laryngoscopes that have handle-to-blade angles of 72 or 90 degrees. The 170-degree angle of the modified lock of the Polio blade avoids the mechanical disadvantage of a handle that encroaches upon the anesthesia screen enclosure (fig. 2).

I have used the Polio blade numerous times in the last five years for this purpose and have found it completely satisfactory.

REFERENCES


Extent and Duration of the Nitrous Oxide Second-gas Effect on Oxygen

LOUIS BOJRAB, M.D.,* AND ROBERT K. STOEITING, M.D.†

Rackow et al. predicted that the reverse of diffusion anoxia (alveolar hyperoxigenation) would occur during anesthetic induction with nitrous oxide. Indeed, arterial oxygen partial pressures increased in two patients when nitrous oxide was administered despite unchanged inspired oxygen concentrations. The increased PaO₂'s resulted from large-volume nitrous oxide uptake into pulmonary capillary blood and represented the second-gas effect of nitrous oxide on oxygen. Since nitrous oxide uptake may remain greater than 100 ml/min even after 90 minutes, we speculated that increased PaO₂'s might persist for several minutes. This study reports the extent and duration of the nitrous oxide second-gas effect on oxygen during anesthesia and operation in man.

METHODS

Six adults without known pulmonary or cardiovascular abnormality were studied during elective operations for excision of intracranial tumors. All patients received thiopental for induction, followed by succinylcholine to facilitate endotracheal intubation. Anesthesia was maintained with halothane, 1 per cent, in 30 per cent oxygen in nitrogen, using a nonrebreathing system (Fink valve). Subsequently the patients were paralyzed with dimethylthubocurarine and respirations were controlled with a volume ventilator. Inspired ventilatory volume was about 15 ml/kg and respiratory rate, 8 to 10 breaths/min. Inspired oxygen was maintained at 30 per cent as confirmed by constant monitoring with a Biomarine oxygen analyzer.

After about two hours of anesthesia and operation, serial arterial blood samples were drawn every 10 minutes from an indwelling radial-artery catheter. At this time the tumor was being excised and the painful stimulus was judged to be minimal and unchanging. When PaO₂'s did not vary more than 5 torr in three consecutive samples, 70 per cent nitrous oxide was rapidly substituted for nitrogen. PaO₂ and PaCO₂ values were measured for the next 60 minutes.

RESULTS

The maximum increases in PaO₂ averaged 14 per cent above control (P < 0.05) and occurred one minute after the start of nitrous oxide administration (fig. 1). This represented an absolute increase in PaO₂ from 121 to 145 torr. PaO₂'s remained 9 per cent above
Nitrous oxide (70 per cent) was substituted for nitrogen at zero time, keeping the inspired oxygen concentration 30 per cent. Data are plotted as per cent changes from control (mean ± SE) during the 60 minutes of nitrous oxide administration.

control after 30 minutes ($P < 0.05$). Values after this time were less than 5 torr different from control.

$P_{a\text{CO}_2}$'s changed less than 1 torr from control (24.3 ± 2.0 torr, mean ± SE) during nitrous oxide administration.

**Discussion**

Large-volume nitrous oxide uptake into the pulmonary capillary blood was associated with increased $P_{a\text{O}_2}$. Nitrous oxide uptake may be 1,000 ml/min during the first minute of administration, and this corresponded to the period of maximum increase in arterial oxygenation. As nitrous oxide uptake decreased with time, $P_{a\text{O}_2}$'s declined towards control. We predicted that continued large-volume nitrous oxide uptake would maintain $P_{a\text{O}_2}$'s above normal for several minutes. These data suggest that the second-gas effect of nitrous oxide on oxygen may last about 30 minutes.

The second-gas effect may result from increased tracheal inflow or concentration of the second gas in a smaller lung volume. When total-body equilibration is present, the only possible explanation for the second-gas effect is the concentrating effect. Continued oxygen uptake precludes equilibration and, therefore, both mechanisms are partially responsible for the second-gas effect of nitrous oxide on oxygen.

Changes in inspired oxygen concentration alter $P_{a\text{O}_2}$. Continuous monitoring of inspired oxygen concentration confirmed that it remained unchanged. Changes in cardiac output may also influence $P_{a\text{O}_2}$ For example, if venous admixture and oxygen consumption did not change, an increased cardiac output would elevate mixed venous $P_{a\text{O}_2}$, resulting in increased arterial oxygenation. In like manner, decreased cardiac output would lower $P_{a\text{O}_2}$. Although we did not measure cardiac output, it would seem an unlikely explanation for our results. Surgical stimulation was minimal and measurements were made only after at least two hours of anesthesia and positive-pressure ventilation. Nitrous oxide administered to volunteers anesthetized with halothane produced signs of peripheral sympathomimetic stimulation but did not change cardiac output. Addition of nitrous oxide probably increased the halothane concentration in our study by the same mechanism described for oxygen. This could conceivably decrease cardiac output and attenuate the maximum increase in $P_{a\text{O}_2}$.

Kitahata *et al.* demonstrated an increased alveolar carbon dioxide partial pressure following nitrous oxide administration. This phenomenon was due to the concentration effect of nitrous oxide uptake on carbon dioxide. Breathing 60 per cent nitrous oxide increased $P_{a\text{CO}_2}$'s less than 1 torr when the control end-tidal carbon dioxide partial pressure was 30 torr. Control $P_{a\text{CO}_2}$'s in our study averaged 24 torr, which explains the lack of significant change when nitrous oxide was administered.

We conclude that $P_{a\text{O}_2}$ will be greater during inhalation of 30 per cent oxygen and 70 per cent nitrous oxide than during inhalation of 30 per cent oxygen in nitrogen. This increased oxygenation is present for about the first 30 minutes of nitrous oxide administration and is the result of large-volume nitrous oxide uptake which increases tracheal inflow of oxygen-containing gases and also concentrates the oxygen in a smaller lung volume. However, this second-gas effect of nitrous
oxide on oxygen cannot be relied upon to produce a clinically significant increase in arterial oxygenation.

REFERENCES

Possible Nephrotoxicity from Enflurane in a Patient with Severe Renal Disease

ROBERT W. LOHNING, M.D., PH.D.,* AND RICHARD I. MAZZI, M.D.†

Enflurane (Ethane, 2-chloro-1,1,2-trifluoroethyl difluoromethyl ether) is metabolized to inorganic fluoride, but to a lesser extent than methoxyflurane. Renal dysfunction associated with its administration has not been reported in man. This report describes deterioration of renal function following enflurane anesthesia in a patient who several years earlier had received a kidney transplant.

REPORT OF A CASE

A 24-year-old woman was admitted to the University of Oregon Hospital for insertion of a saphenous-vein arteriovenous shunt in an arm in preparation for nephrectomy and a new allograft transplant. History revealed chronic glomerulonephritis, initially diagnosed at the age of 14 years, leading to transplantation of a cadaver kidney at the age of 22 years. Following transplantation the patient had been admitted to the hospital three times for episodes of rejection. Six months prior to the present admission she had received an Austin Moore hip prosthesis because of aseptic necrosis of a femoral head. Halothane anesthesia had been administered on that occasion without complication, BUN varying between 23 and 33 mg/100 ml during the postoperative period. In the following months renal function had stabilized at levels which indicated minimal reserve. At the time of the present admission, laboratory values included BUN 54 mg/100 ml, serum creatinine 3.6 mg/100 ml, and creatinine clearance 15.3–17.3 ml/min. Urinary volumes ranged from 1,300 ml/day to as much as 2,600 ml/day following treatment with furosemide. Drug treatment included azathioprine, prednisone, furosemide, chloral hydrate, meperidine, and propanidone.

On December 4, 1972 following premedication with meperidine and atropine, anesthesia was induced with thiopental and 3.0 per cent enflurane with oxygen and nitrous oxide. Enflurane concentrations during the procedure, which lasted 3 hours and 45 minutes, ranged from 0.5 to 0.8 per cent. Both operation and anesthesiia were uncomplicated. During the first two postoperative days, urinary output averaged 1,900 ml/day, exceeding fluid intake by 800 ml/day. This resulted in a fluid deficit of approximately 1,500 ml/day, including insensible losses. BUN increased to 105

* Associate Professor of Anesthesia, Department of Anesthesiology, University of Oregon, Portland, Oregon
† Associate Professor of Anesthesia, Department of Anesthesiology, Stanford University School of Medicine, Stanford University, Stanford, California; Chief, Anesthesiology Service, Veterans Administration Hospital, Palo Alto, California 94304.

Accepted for publication August 3, 1973. Supported in part by Public Health Service Grant GM-18514 and MHR #5051-01, VA Hospital, Palo Alto, California.

Address reprint requests to Dr. Mazzi.