hospital day the size of the mediastinal tumor had decreased markedly as a result of radiation and chemotherapy. Repeat bronchoscopy showed inflammation of the vocal cords and subglottic larynx. The trachea was patent, but the lower half was inflamed and narrowed. The left main-stem bronchus was 50 per cent open. This represented a dramatic improvement in airway patency. Both endotracheal tubes could now be removed. A #4 Aberdeen tracheostomy tube was placed, with adequate spontaneous ventilation maintained.

Over the next four hospital days the clinical course was complicated by an abdominal mass in the right lower quadrant and sepsis. The patient's condition gradually deteriorated, and she died on the fifteenth hospital day in septicemic shock. Permission for postmortem examination was denied.

Discussion

This case demonstrates that treatment of critical airway obstruction of the distal trachea and a main-stem bronchus by intubation of the trachea or tracheostomy may fail. A system that will independently ventilate each lung beyond the sites of obstruction is then needed. Double-lumen tubes (*e.g.*, Carlens tube) are not made in sizes appropriate for small children. In addition, endobronchial intubation has to be individualized according to the extent and site of obstruction. Anatomically the size of the glottic opening and the cricoid diameter preclude both tubes passing via this route. Thus, one tube is passed via a tracheostomy, which allows bronchoscopy at the same time to guide the tube into the main-stem bronchus. Following positioning of this tube, the endotracheal tube can then be passed through the glottis in the conventional manner to the carina or into the opposite bronchus distal to the obstruction. Positions of the tubes are confirmed radiographically.

This approach to management of critical airway obstruction involving the trachea and main-stem bronchus may be life-saving. It will maintain the airway until further therapy decompresses the obstructing tumor.

Inflowing Gas Leak, A Potential Source of Hypoxia

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Prevention of delivery of hypoxic gas mixtures to patients requires: 1) that the oxygen concentration in the inflow (*i.e.*, the flow from the machine to the anesthetic circuit) gases be adequate; 2) that the total flow of oxygen be large enough to replace the oxygen used by the patient. Considerable attention has been given to the first requirement: we have master–slave regulators that insure that we cannot use nitrous oxide if oxygen pressure is lacking,[1] proportioning devices prevent the delivery of an oxygen concentration lower than some preset value,[2] and we now arrange our flowmeter sequence so that a leak in an individual flow tube will not result in the preferential ejection of oxygen.[3]

We recently experienced several machine failures that might have produced hypoxia by causing the total flow of oxygen to be less than sufficient to replace the oxygen used by the patient. Surprisingly, this defect was found in two machines used sequentially in one case. It also was found in the case of a second patient who received anesthetic from a third machine.

Report of Two Cases

Case 1. A healthy 15-year-old boy was anesthetized with nitrous oxide, oxygen and meperidine and mechanically ventilated via an endotracheal tube (rate 12/min, tidal volume 650 ml, peak pressure 25 cm H_2O). The gases were delivered from an Ohio anesthetic machine with a "side-arm" Vernitrol® vaporizer (all the defective machines were of this type).

We attempted to reduce the total inflow rate, but 1,000 ml nitrous oxide and 600 ml oxygen barely kept the ventilator bellows full, and the oxygen concentration in the circle system (as measured

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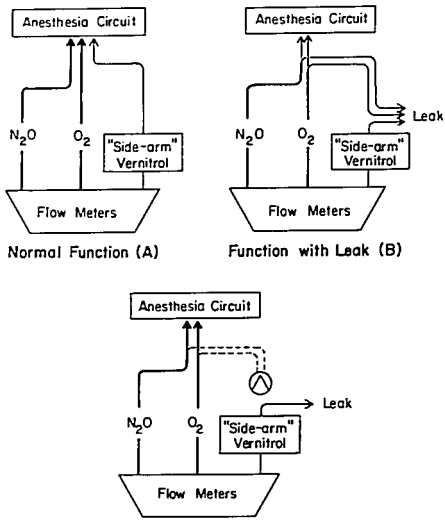

FIG. 1. Inflowing nitrous oxide and oxygen (left and middle lines) normally would be delivered undiminished to the anesthesia circuit (*A*). However, a leak indicated at the on–off mechanism for the "side-arm" Vernitrol® would permit a portion or all of the nitrous oxide–oxygen mixture to escape (*B*). A check valve will eliminate the possibility of a significant back-leak although oxygen directed through the "side-arm" Vernitrol® still would be lost (*C*). This check valve may be added to older machines.

by a polarographic analyzer sensing in the inspiratory limb) fell below 30 per cent. Examination failed to demonstrate a leak in the circle system or ventilator. The patient manifested no evidence of abnormal oxygen consumption (*e.g.*, no increased temperature or muscle rigidity), but to maintain the inspired oxygen concentration at 30 per cent and keep the bellows full required flowmeter settings of 1 l/min each for nitrous oxide and oxygen. In the absence of the pressure generated by the ventilator, these flow rates did indeed deliver 50 per cent oxygen rather than the 30 per cent recorded. The above findings were compatible with a reduced inflow secondary to a leak within the machine. The original machine therefore was replaced with a second machine, also with a "side-arm" Vernitrol®. However, the same sequence of events transpired with the second machine. Tests again indicated an internal leak in the second machine, and it was replaced with a third anesthetic machine manufactured by a different company. With the third machine we could reduce the inflow to 200 ml/min oxygen and 200–300 ml/min nitrous oxide. This maintained a steady volume in the circle system with an inspired oxygen concentration of 30 per cent. The operation was completed and the patient recovered uneventfully.

Case 2. A 30-year-old woman was anesthetized and mechanically ventilated via an endotracheal tube (tidal volume 550 ml, frequency 12/min). Enflurane was delivered from the "side-arm" Vernitrol® with 2 l nitrous oxide and 1.2 l oxygen. Under these circumstances an inspired oxygen concentration of 25 per cent was found. Shortly thereafter systolic pressure decreased from 100 to 70 torr, pulse rate decreased from 100 to 80 beats/min, and the skin appeared dusky. The inflow was changed to 8 l/min oxygen, systolic pressure immediately returned to 110 torr, and the pulse rate gradually increased to 100 beats/min. With 1 per cent enflurane in 1.5 l nitrous oxide and 1.6 l oxygen the inspired oxygen concentration decreased to 38 per cent. With the overflow (pop-off) valve closed, an inflow of 1 l/min failed to maintain the ventilator bellows full. With an inflow of 3 l/min nitrous oxide, plus 3.2 l/min oxygen, the bellows remained full and the inspired oxygen concentration was stable at 50 per cent. The remainder of the case was uneventful.

DISCUSSION

Examination of the defective machines revealed the same defect in each: a broken "O" ring in the on–off mechanism at the base of the "side-arm" Vernitrol®. A review of the machine's circuitry (fig. 1) indicates that the mechanism is not protected from

back pressure in the outlet line and hence is particularly apt to serve as a leak source in the presence of positive-pressure ventilation.

We believe the loss of inflow gases accounts for our findings and suggests a potentially life-threatening hazard. Obviously we did not deliver the flow rate indicated by our flowmeters. We estimate that an inflow of 2 l/min indicated on the meters actually represented an inflow of only 500 ml/min. At this flow rate and a delivered oxygen concentration of 30 to 40 per cent, the total quantity of oxygen could be inadequate to replace the amount consumed by the patient. Had the use of the oxygen analyzer not informed us of diminishing oxygen levels and the disparity between the flowmeter concentrations and the actual circuit concentrations, we might have persisted in delivering what appeared from the flowmeter settings to be 50 per cent oxygen. Hypoxia could, and in Case 2, probably did, ensue.

A check valve to prevent the above-described loss of inflow gas has been developed by Ohio Medical Products and will be furnished as a standard feature on all new "side-arm" vaporizers (fig. 1). This check valve also will be available (stock number 207-8027-800) to all current users of the "side-arm" vaporizers at a nominal charge. A similar check valve (stock number 207-8028-800) will be available for Unitrol® systems. These valves are available only through Ohio service representatives, who are responsible for the proper installation of the valve system.

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Pain Therapy

ELECTRICAL STIMULATION AND NALOXONE Focal electrical stimulation of the brain is known to produce analgesia in several species. It is interesting that stimulus-elicited analgesia has much in common with analgesia produced by narcotic administration. Thus, electrical stimulation and narcotics appear to exert their effects at the same anatomic site. Drugs affecting transmission in central monoamine pathways alter both narcotic and electrically induced analgesia. Tolerance to stimulation of the brain develops, and cross-tolerance between electrical stimulation and narcotics has been observed. The authors now report the exciting observation that stimulus-produced analgesia can be partially antagonized by the narcotic antagonist naloxone. Analgesia was measured by examining the response to radiant heat in 41 male Sprague-Dawley rats. In one group, intense analgesia was produced by electrical stimulation. Administration of naloxone (1 mg/kg) reduced analgesia by 38 per cent. In a second group in which the stimulating current was adjusted to produce intermediate analgesia, naloxone again reduced analgesia by 38 per cent. Higher doses of naloxone (2 and 4 mg/kg) had no further effect. The data suggest that "there is a neural system in the brain which uses an endogenous, morphine-like substance to produce analgesia," and "that activation of this system can be brought about either pharmacologically by direct receptor stimulation, or electrically by release of the endogenous substance." *(Akil H, Mayer DJ, Liebeskind JC: Antagonism of stimulation-produced analgesia by naloxone, a narcotic antagonist. Science 191:961–962, 1976.)*