injection of air into cerebral ventricles in the dog, a finding recently confirmed by Stolkarts in man.[3] This effect results from the 36-fold greater solubility in blood of nitrous oxide compared with nitrogen. Nitrous oxide may be carried to the ventricles in volumes far greater than the volumes of nitrogen that may be carried away. For this reason, inhalation of nitrous oxide as anesthesia for pneumoencephalography or soon thereafter may be contraindicated when air is used as the contrast gas, particularly in patients who have pre-existing elevation of intracranial pressure, or decreased intracranial compliance.[1-7] In patients without these conditions the risk may be minimal.[7-9]

The data most commonly quoted by those who suggest a delay before nitrous oxide anesthesia come from Aird's study in 1937.[4] Aird found that 90 per cent of air injected for pneumoencephalography was reabsorbed after two or three days. However, in some patients the air did not completely disappear for six or seven days.

Bergström *et al.*[10] studied two groups of patients undergoing pneumoencephalography during general anesthesia with either halothane or methoxyflurane in nitrous oxide and oxygen. In one group nitrous oxide–oxygen was used for the contrast gas; in the second group, air was used as the contrast gas. The mean duration of retention of perceptible (by x-ray) volumes of nitrous oxide–oxygen contrast gas mixture in intracranial cavities was two days. When air was used as the contrast gas the mean was seven days. The quality of the pneumoencephalography was not compromised by using nitrous oxide–oxygen as the contrast medium.

It seems likely that in the case presented expansion by nitrous oxide of residual intraventricular gas accounted for the "bulging" of the brain observed at operation. This experience suggests that a two- or three-day period is insufficient for reabsorption of intraventricular air in all cases, and that a potentially hazardous increase in intracranial pressure happened to our patient as a result of the use of nitrous oxide for anesthesia. Therefore, when a patient has had an air contrast study within seven days of the proposed operation, two courses seem appropriate: 1) avoid the use of nitrous oxide in the anesthetic gas mixture, or 2) obtain preoperative x-rays to determine whether intracranial air is still present. Should air persist, the operation may be delayed to allow reabsorption, or it may proceed, avoiding nitrous oxide anesthesia.

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Inadvertent Anesthetic Overdose Obscured by Scavenging

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Virtues of scavenging waste anesthetic gases have been extolled[1] and failure to scavenge condemned,[2] although benefits of scavenging remain to be proven.[3] It is generally assumed that elimination of waste gases is a sufficiently benign practice that no justifiable reason exists for failure to scavenge. This may be true, but it should be pointed out that the process of

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scavenging may expose patients to certain hazards. Some, such as pulmonary barotrauma,[4] can probably be avoided by using properly designed and well maintained equipment; others may be unavoidable. We report here a death owing to anesthetic overdose when liquid halothane splashed into the anesthetic circuit. The problem was obscured by the absence of a strong odor of halothane, an unavoidable consequence of scavenging.

Report of a Case

A 30-year-old woman, in coma since an automobile accident three months earlier, was scheduled for ventriculoperitoneal shunt to relieve progressive hydrocephalus. Within 2 minutes after induction of anesthesia, with ventilation assisted as halothane in nitrous oxide and oxygen was given via tracheostomy tube, the blood pressure became inaudible and ventricular tachycardia followed by fibrillation was evident on the electrocardiogram. Anesthetic flowmeters were turned off and the patient's lungs ventilated with oxygen. External cardiac compression was begun, but attempts at resuscitation with repeated electrical defibrillation, intracardiac epinephrine, sodium bicarbonate, lidocaine, phenylephrine and calcium chloride was unsuccessful. The cause of the sudden arrest was not immediately apparent, although acute pulmonary embolism and cerebral herniation were considered.

Soon thereafter, it was noticed that 110 ml of liquid halothane remained in the vaporizer, whereas 190 ml (almost full) had been present before induction of anesthesia. The anesthesia machine was removed from the operating room, the vaporizer filled to the 190-ml mark, and the valve of the oxygen flowmeter serving the halothane vaporizer turned on. The bobbin rose rapidly toward the top of the rotameter tube (as had occurred at the start of the case) and a gurgling sound was heard at the vaporizer. Liquid halothane emerged from the common delivery tube, leaving 130 ml in the vaporizer. A strong odor of halothane was immediately apparent, whereas, owing to effective scavenging, no odor of halothane had been detected during the preceding case. The needle valve of the halothane flowmeter was subsequently found by a service engineer to be defective.

Discussion

Collapse upon induction of anesthesia was initially thought to have been caused by some aspect of the patient's serious illness. Besides having had several episodes of ventricular tachycardia necessitating defibrillation a month before, she had had recurrent pulmonary emboli, and intracranial pressure had become markedly elevated, so that operation was indicated. In contrast, sudden cardiac arrest during induction of anesthesia in an otherwise healthy patient would have strongly suggested anesthetic overdose.

Without scavenging, the strong odor of halothane might have alerted the anesthesiologist to inadvertent administration of an excessive concentration of halothane. The inhaled concentration may have approached 32 per cent‡ with liquid halothane in the anesthesia circuit. In the past, the ability to smell such high concentrations of halothane has assisted in the diagnosis of anesthetic overdose and, by directing attention toward some problem in the vaporizing system, has enabled prompt corrective action.[5] In 1967, Mark described an incident of cardiac arrest occurring shortly after induction of anesthesia. Because the odor of halothane persisted after turning off the halothane and administering what was intended to be pure oxygen, another anesthesia machine was substituted without delay, and the patient was successfully resuscitated. Subsequent examination demonstrated the presence of liquid halothane in the main gas stream of the anesthesia machine.

Kopriva and Lowenstein, in 1969, reported that liquid halothane can be discharged into the anesthetic circuit owing to a faulty needle valve on the halothane flowmeter.[6] They thoroughly discussed the problem, including measures to reduce this hazard. One suggestion was never to fill the vaporizer to more than three-fourths of the "maximum safe level" on the sight glass. Another was to install a limiting orifice in the line delivering oxygen to the vaporizer, thus prohibiting inflow pressures sufficient to force liquid halothane into the delivery tube.

The frequency of undetected anesthetic overdose should decrease with the appearance of inexpensive dependable monitors of delivered anesthetic concentration or the introduction of innovative anesthesia machines that are inherently safer.§ Meanwhile, the elimination of waste anesthetic gases, while potentially beneficial to the health of operating room personnel, prohibits the anesthesiologist from effectively using all five senses in monitoring the anesthetic course. This fact should not be neglected when assessing the cost-benefits ratio for scavenging.

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‡ The vapor pressure of halothane at sea level is 0.32 atm at 20 C.

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