

plotted on the basis of logit analyses of responses in this patient population (fig. 1) revealed that MAC_{EI} is 1.12 per cent; MAC_{EI} is 1.46 per cent. Based on the individual slope equation, MAC_{EI} for 95 per cent of this population equals 1.91 per cent. These investigations were performed at an altitude of approximately 760 m (2,500 ft), while traditional MAC values have been determined at essentially sea level. After appropriate barometric corrections, MAC_{EI} at sea level is calculated to be 1.33 per cent.

DISCUSSION

An inhalation induction often avoids the potential psychologic trauma associated with venipuncture in a child. However, proceeding to attempt tracheal intubation with possibly insufficient anesthesia may result in failure to visualize the cords, laryngospasm, and excessive coughing or "bucking" after successful insertion of the tube. Such clinical signs as fixed and slightly dilated pupils, relaxation of the jaw, and diaphragmatic breathing are usually imprecise endpoints, and hardly apply with modern anesthetic agents. In this study, using methods analogous to those for determining MAC, we have established an estimate of the alveolar halothane concentration necessary to prevent movement completely during tracheal intubation in 50 per cent of children aged 2–6 years (MAC_{EI}). The homogeneity of anesthetic concentrations within each group (table 1) is evidenced by the low standard deviations. The mean ratios of inspired to end-tidal halothane concentrations ranged from 1.10 to 1.18. These ratios agree comfortably with those obtained by Gregory² and Nicodemus³ in previous MAC studies. The proximity of these ratios to unity suggests the absence of significant ventilation-perfusion abnormalities, which would interfere with the accuracy of end-tidal gas determinations. MAC was not determined in this study; however, the aforementioned works by Gregory² and Nicodemus³ suggest MAC values ranging

from 0.91 to 1.07 per cent for this age group. Therefore, at sea level, MAC_{EI} is approximately 1.33 MAC.

MAC_{EI} is of clinical value, since there are a number of surgical situations which would demand a level of anesthesia that not only allows adequate conditions for laryngoscopy, but also prevents subsequent coughing or "bucking." Mechanical stimulation of lower tracheal or carinal reflexes caused by the endotracheal tube or by the sudden, irritative flow of dry gases to these areas probably produces such events. According to the classic guidelines of Guedel, later modified by Gillespie,⁴ abolition of tracheal and carinal reflexes requires a depth of anesthesia greater than that needed for routine surgery. Unlike MAC, therefore, MAC_{EI} is not a *sine qua non* for surgical anesthesia. The extents of reflex suppression elicited by different anesthetics may vary widely, despite the presence of sufficient anesthetic depth for surgical manipulation.

MAC_{EI} has utility as a guideline for performing a specific task with certain anesthetic agents. Based on results obtained with halothane, one might speculate that the MAC_{EI} 's for other agents should be greater than, or at least equal to, MAC. It would be of interest to investigate the constancy of the MAC_{EI} -to-MAC ratio. The determination of MAC_{EI} in addition to MAC would provide another dimension to the analysis of relative drug potencies.

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Ventriculobronchial Fistula Complicating Ventriculoperitoneal Shunt

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Hydrocephalus of varied etiology, such as meningo-myelocoele, aqueductal stenosis, brain tumors, con-

genital anomalies, meningitis, and trauma, necessitates shunting of cerebrospinal fluid (CSF).¹ Since the introduction of the ventriculoperitoneal shunt (VP shunt) by Kausch in 1905,² numerous other methods have been developed to drain the CSF. These methods include ventriculoatrial, ventriculopleural, ventriculogallbladder, ventriculoureteral and ventriculo-fallopian shunts. However, today VP shunt is the pre-

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ferred procedure since it is associated with the fewest complications. This report describes the development of a ventriculobronchial fistula following VP shunt.

REPORT OF A CASE

A 3½-year-old boy was admitted to the hospital for surgical removal of an infected and malfunctioning VP shunt. Eight months earlier the child had been admitted to the hospital for the first time with symptoms of progressing unsteady gait, irritability, mild headaches, and vomiting. Work-up revealed increased intracranial pressure secondary to a large posterior-fossa tumor. A right-sided VP shunt was inserted with the patient under general anesthesia. Ten days later a partial resection of a medulloblastoma was performed via a posterior-fossa craniotomy. The anesthetic course was uneventful. The patient also received a full course of radiation therapy for the tumor. He was discharged after two months of hospitalization.

Three weeks after discharge from the hospital the patient had right-lower-lobe pneumonia, and was treated with antibiotics. He had an intermittent spiking fever, with temperatures as high as 39 C. Chest x-rays revealed persistent right-lower-lobe pneumonia with associated atelectasis and scarring. It was also found that the distal tip of the peritoneal catheter had migrated through the right diaphragm into the atelectatic area (fig. 1). A shuntogram using Conray contrast medium was used to evaluate the function of the VP shunt. The resulting bronchogram of the right lower lung confirmed that the distal tip of the peritoneal catheter had perforated a right-lower-lobe bronchus, establishing a communication between the cerebral ventricle and the bronchus. Laboratory values were normal with the exception of an elevated leukocyte count. The VP shunt was removed with the patient under general endotracheal anesthesia and a Rickham reservoir was inserted. The anesthetic course was uneventful. A chest x-ray was taken in the operating room before removal of the endotracheal tube to rule out pneumothorax. Cultures of ventricular CSF grew *Staphylococcus aureus*. The patient was treated by systemic and intraventricular administration of antibiotics. Postoperatively the patient progressed well and was discharged.

DISCUSSION

The complications associated with VP shunt relate to migration of the whole shunt system,³ or migration of the distal tip of the catheter,^{1,4} and to the mere presence of the catheter in the peritoneal cavity.^{1,10} The reported complications include perforation of hollow abdominal structures (intestines, gallbladder, urinary bladder, and vagina^{1,4,5}) and migration of the peritoneal end of the catheter into the scrotum, hernial sac, and umbilicus.^{1,6} The presence of the catheter in the peritoneal cavity itself has been implicated in small-intestinal obstruction, volvulus of the intestines, inguinal hernias, abdominal pseudocysts, ascites,¹ symptoms simulating appendicitis,⁷ hydrocele,⁸ spontaneous umbilical fistula,⁹ and distant metastasis of cancer.¹⁰

All these complications of VP shunt have minimal implications for anesthetic management. In contrast, the development of a ventriculobronchial fistula, as in our patient, may adversely influence the anesthetic course.

For example, removal of the catheter from the chest

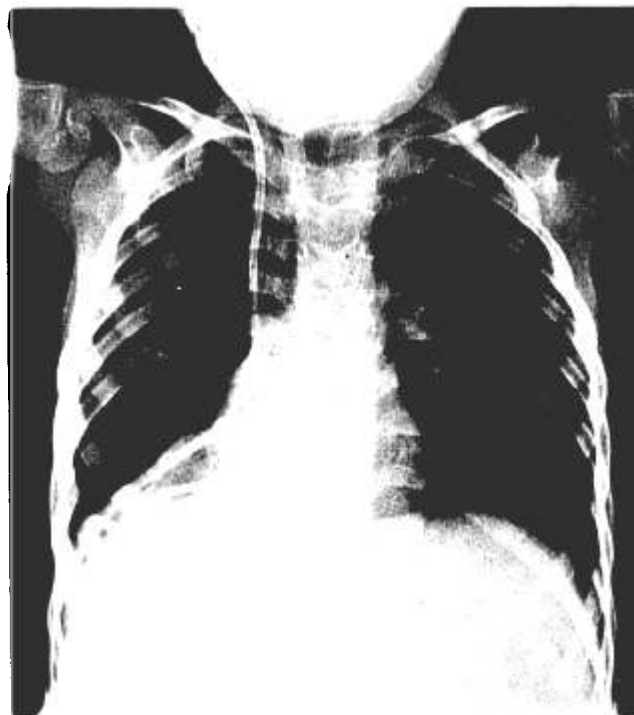


FIG. 1. Anteroposterior view of the chest, showing atelectasis of the right lower lobe. Note the distal tip of the peritoneal catheter of the ventriculoperitoneal shunt in the right lower chest near the atelectatic area of the lung.

can cause pneumothorax, especially during positive-pressure ventilation. The anesthesiologist should bear this possibility in mind in the event of a sudden decrease in pulmonary compliance and/or cardiovascular collapse. In our patient, it is likely that the consolidated atelectatic and chronically inflamed area of the lung sealed off the pleural cavity and prevented the development of pneumothorax.

Another hazard in a patient who has a ventriculobronchial communication is the possibility of an increase in intracranial pressure, especially during positive-pressure ventilation. Kessler *et al.* reported a case in which a patient with a ventriculopleural shunt developed symptoms of increased intracranial pressure and a constant desire to cough.¹¹ Skull x-rays and further investigation revealed a distended ventricular system filled with air secondary to ventriculobronchial fistula. The tip of the pleural catheter had perforated and established a communication with a bronchiole. With the patient under anesthesia in the prone position, these investigators found free flow of CSF from the endotracheal tube.

In our patient, the mechanism of development of a direct communication between the peritoneal catheter and right-lower-lobe bronchus is obscure. Conceivably, the right-lower-lobe pneumonia developing after insertion of the VP shunt could have caused inflammation and adhesions in the right lower chest and initiated the migration of the peritoneal

catheter into the right lower lung. It is also possible that the catheter had first migrated into the right lower chest and set up an inflammatory reaction, atelectasis, and eventually perforation of the bronchus. It seems likely that the persistence of right-lower-lobe pneumonia and atelectasis in our patient were related to the location of the catheter in the right lower chest.

Patients who have VP shunts may be subjected to frequent surgical procedures such as repeated shunt revisions or corrections of other associated congenital anomalies. Since sudden pneumothorax or an increase in intracranial pressure during the course of anesthesia may have catastrophic consequences, it is suggested that a recent chest x-ray be reviewed prior to the induction of general anesthesia in a patient who has a VP shunt.

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Ketamine-induced Hyperthermia—Postictal or Malignant?

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Controversy surrounds the use of ketamine in patients with epilepsy. There is not universal agreement as to the safety of ketamine in those patients judged potentially at risk for malignant hyperthermia either. We report a case where ketamine, administered to an epileptic patient with a myopathy, produced grand mal convulsions. In the postictal phase the axillary temperature rose more than 3 C, mimicking the onset of malignant hyperthermia.

REPORT OF A CASE

A 4-year-old white boy who had Lowe's syndrome (oculocerebrorenal dystrophy) was admitted for evaluation. All the stigmata of the disorder were present: severe mental retardation, marked muscular hypotonia and areflexia, rickets, proteinuria and aminoaciduria, congenital ocular abnormalities, and cryptorchidism. The parents stated the child had been anesthetized twice at the age of 7 months without incident for cataract

surgery. Records of those anesthetics, given at another institution, could not be located.

At 3 years of age, the patient had had febrile convulsions. Since that time the parents had noticed repeated brief "shaking spells" unassociated with tonic-clonic movements, cyanosis, or autonomic dysfunction. Family history revealed another male sibling with Lowe's syndrome and three paternal relatives with neurofibromatosis. There was no family history of any anesthetic catastrophe.

Weight on admission was 15 kg, height 97.8 cm, blood pressure 100/60 torr, pulse rate 92/min, respiratory rate 20/min and axillary temperature 36.4 C. Hemoglobin was 11.5 g/dl and the leukocyte count was 6.9×10^9 cells/l with a normal differential. Serum creatinine, sodium, potassium, chlorides and calcium were all within normal limits. Serum bicarbonate, however, was 17 mmol/l. Serum creatine phosphokinase was 704 IU/l. An electroencephalographic recording made during chloral hydrate sedation demonstrated excessive beta activity, but with intermittent sharp waves and spike-like activity during sleep.

As part of the evaluation, the patient was scheduled for a muscle biopsy. The child was fasted six hours prior to operation and not premedicated. Axillary temperature prior to induction of anesthesia was 36 C, and the ambient room temperature was 24 C. For induction, ketamine, 40 mg, was administered im, and preparation for operation was begun. Five minutes later, the child had generalized tonic-clonic convulsions, lasting approximately two minutes, which terminated spontaneously. In the postictal state the patient appeared to be adequately anesthetized and the decision was made to continue with the biopsy, using local infiltration

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