

TABLE 1. Biochemical Values and Clinical Signs in a Patient with Tetany

	P _{O₂} (torr)	P _{CO₂} (torr)	pH	[Ca ⁺⁺] (mM)	Chvostek's Sign	Trousseau's Sign
During anesthesia	131	29	7.47	0.66	+	+
Awake						
Control (spontaneous ventilation)	98	37	7.44	0.92	—	—
After 30 min of hyperventilation	95	20	7.65	0.77	+	+
Spontaneous ventilation						
5 min	93	28	7.53	0.78	+	+
10 min	96	34	7.47	0.80	+	+
15 min	96	37	7.45	0.86	+	—
30 min	98	39	7.41	0.93	—	—

mental animal, that hypocalcemia and alkalosis in the extracellular fluid acted synergistically in the etiology of tetany. Previous data from this laboratory³ are in agreement with results of these investigators, who pointed out that the magnitude of reduction in [Ca⁺⁺] due to alkalosis *per se* is relatively small, and that alkalosis-induced tetany cannot be explained by the hypocalcemic effect of alkalosis alone. They⁵ also found that hypocalcemia in combination with alkalosis consistently induced tetany, whereas either condition alone did not.

In our patient, arterial blood pressure was well-maintained throughout the hypocalcemic episode. Alterations in both ventricular and peripheral vascular function can occur with hypocalcemia.⁶ When tetany appeared in our patient during anesthesia, calcium chloride was administered, and tetanic manifestations disappeared. In view of the relatively small [Ca⁺⁺] change that occurred with tetany in the awake state, calcium replacement therapy was not considered necessary.

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Cardiorespiratory and Cranial-nerve Sequelae of Surgical Procedures Involving the Posterior Fossa

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Surgical procedures in the region of the posterior fossa are attended by a variety of potential complica-

tions. Certain of these—venous air embolism, for example—are relatively common, and have received investigative attention. Other less frequent complications have received less attention. The following case report illustrates some problems that may attend neurosurgical procedures involving the posterior fossa.

REPORT OF A CASE

An 18-year-old male patient was scheduled for posterior-fossa exploration for a cerebellar mass lesion with associated hydro-

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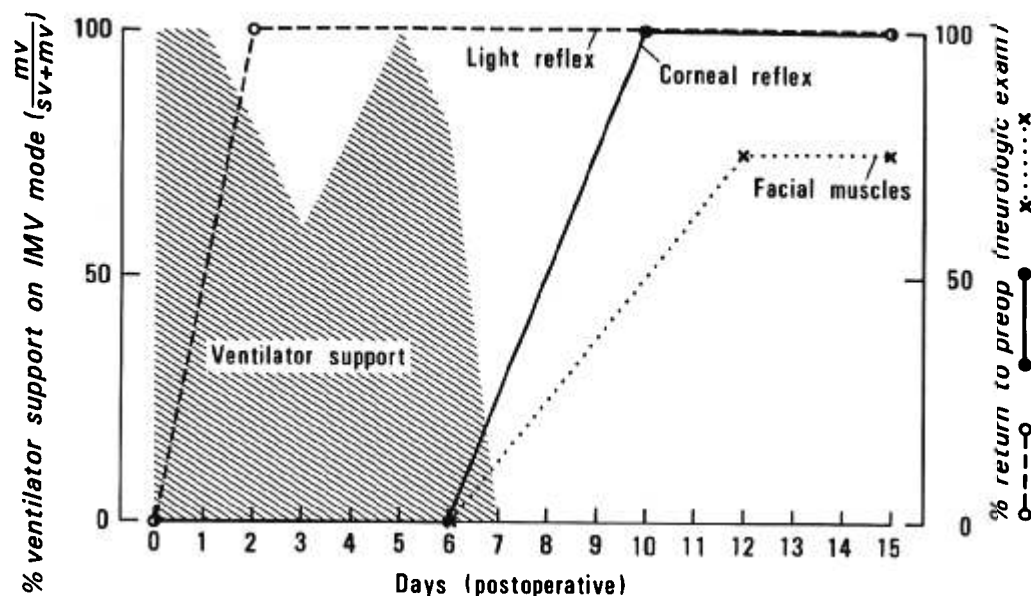


FIG. 1. Resolution of specific cranial nerve deficits, expressed as percentage return to preoperative findings by clinical examination, and the progress made toward independence from ventilatory support. Ventilatory support on IMV mode is expressed as the ratio of mechanical ventilation (MV, number of breaths) to total ventilation (mechanical plus spontaneous, MV + SV, number of breaths).

cephalus. Pertinent portions of his history on admission included complaints of ataxia, right-sided headache, occasional diplopia, nausea with vomiting, and impaired hearing. Preoperatively, results of a detailed neurologic examination were remarkable only for mild ataxia and severe bilateral papilledema (grade 3/4). Preoperatively, the blood pressure range in the hospital was 120/70 to 140/80 torr. Preoperative medication consisted of codeine, 60 mg, diazepam, 10 mg, and atropine, 0.4 mg, administered in two hours prior to operation.

Following preoxygenation, anesthesia was induced with thiopental, 500 mg, iv, in incremental doses, and orotracheal intubation was facilitated by the injection of pancuronium bromide, 10 mg, iv. Manually assisted hyperventilation was begun. Maintenance of anesthesia was accomplished by inhalation of nitrous oxide, 67 per cent, in oxygen, and incremental doses of fentanyl, iv. Ventilation was adjusted to maintain P_{aCO_2} 29–34 torr, with P_{aO_2} 148–168 torr, during the first four hours of the surgical procedure.

Four hours after a stable intraoperative course (total fentanyl dose to this point, 1.1 mg), the surgeon applied traction to the base of the tumor, which extended to the fourth ventricle. There was an immediate precipitous rise in blood pressure from 130 torr to 200 torr systolic with an associated decrease in pulse rate from 80 to 52 beats/min. Enflurane, 1.0 per cent, was added to the inspired gas mixture, and the surgeon was informed of these hemodynamic changes. Surgical traction was discontinued, blood pressure and heart rate returned to normal over several minutes, and enflurane was discontinued. After further dissection, the surgeon again applied traction to the base of the tumor mass. Again, blood pressure rose precipitously to 230 torr systolic, and frequent multifocal ventricular premature contractions appeared. The surgeon was informed of these changes, enflurane, 1.0 per cent, was added to the inspired gas mixture, and 50 mg lidocaine administered iv, with rapid abolition of the ventricular ectopic contractions. Surgical extraction of the mass was completed quickly, whereupon the blood pressure fell to 100 torr systolic, heart rate returned to prestimulation levels, and enflurane was discontinued. The mass was subsequently identified as a grade II astrocytoma of the cerebellar vermis with regional extension.

At the conclusion of a 5½-hour surgical course, nitrous oxide was discontinued. After several minutes of inhalation of 100 per cent oxygen, the patient opened his eyes when his name was called, and demonstrated normal grasp and movement of his

extremities when requested. He made no spontaneous respiratory effort, however, and made only a poor effort when instructed to take a deep breath. Naloxone in incremental doses to a total dose of 1.2 mg was administered to reverse residual narcotic effect. Respirations were shallow, at a normal rate, rather than the slow, deep ventilatory pattern usually associated with the use of narcotics. The patient was transferred to the recovery room with his trachea intubated for assisted ventilation with a Bird ventilator. Although the patient responded to commands regarding grasp and movement of the extremities, an attempt to convert him to spontaneous ventilation was not successful, as evidenced by progressive hypercarbia on serial blood-gas determinations. Therefore, continuous controlled ventilation was instituted. Other specific neurologic deficits found when the patient was in the recovery room included: facial palsy, loss of lateral gaze, loss of corneal reflex, numbness of the upper lip and tongue, absent "doll's" eyes, and absent response to cold caloric stimulation. General protective care with specific attention to the patient's eyes was instituted.

The patient was subsequently transferred to the neurosurgical intensive care unit for continued controlled ventilation and supportive care. For the first 24 hours ventilation was adjusted to maintain P_{aCO_2} 26–29 torr. The patient was then placed on an intermittent mandatory ventilation (IMV) circuit providing sufficient ventilatory support to maintain P_{aCO_2} near 40 torr. Throughout several assessments daily for the following six days, the patient was found capable of sustaining on command a ventilatory pattern of normal tidal volume and rate. Spontaneous ventilation, however, remained inadequate until the seventh postoperative day, whereupon sudden improvement occurred concomitantly with resolution of his other specific neurologic deficits (fig. 1).

DISCUSSION

Certainly air embolus is not the only danger attending posterior-fossa surgery. That surgical procedures involving the posterior fossa can affect respiration has been known for many years.¹ Respiratory deficit as a result of posterior-fossa operations has been described in two recent reports.^{2,3} Cardiovascular sequelae during posterior-fossa operations also have been

reported. Hypertension has been ascribed to trigeminal-nerve stimulation, and bradycardia to vagal stimulation.¹ Finally, specific neurologic deficits following this surgical approach have been reported—hyperesthesia, homonymous hemianopsia, ataxia, and deficits in cranial nerves 3, 6, 7, and 8.⁴

The case presented here involved a critical area of the posterior fossa, the dorsal pons and medulla. This area is the site of respiratory control centers, cranial nerve nuclei and tracts, and vasomotor tracts and centers. The intraoperative events we observed, and the patient's post-operative neurologic condition, are consistent with disruption of these structures (fig. 2).

Wang⁵ has shown that stimulation of the periventricular gray area (floor of the fourth ventricle) and medullary reticular formation (ventral to the periventricular gray matter) produce hypertension via homolateral descending pathways in the cord. De Jong *et al.*⁶ have shown that hypertension also results from interruption of lateral input to the nucleus and tractus solitarius, including input from cranial nerves 9 and 10. Severe hypertension due to cardiovascular center dysfunction (hatched area, fig. 2) may cause, in addition to arrhythmias as observed in this case, intracranial hemorrhage, increased intracranial pressure due to increased cerebral blood volume when autoregulation is impaired, or cerebral edema when the blood-brain barrier is impaired. Additionally, bradycardia may occur as a reflex response to hypertension, or it may occur due to direct stimulation of vagal centers.⁷ Bradycardia sufficient to lower cardiac output may result in cerebral ischemia if cerebral perfusion pressure becomes inadequate.

Berger *et al.*⁸ cite the dorsal respiratory group—DRG (dorsal medulla caudad to the pontomedullary junction)—as the initial intracranial processor for visceral reflexes, and the site for delivery of rhythmic respiratory drive to the ventral respiratory group—VRG—and thence to the intercostals, abdomen, and auxiliary muscles of respiration). This locus may be the site of all respiratory rhythm. The apneustic center—APC (dorsal pons rostral to the pontomedullary junction)—is cited as the normal inspiratory cut-off mechanism. Hypoventilation due to derangements of respiratory centers (bold lettered areas, fig. 2) may, through respiratory acidosis, produce increased intracranial pressure or cerebral edema secondary to cerebral vasodilation and/or metabolic imbalance, even when oxygenation is adequate.

The dorsal pons and medulla are, furthermore, the site of the sensory nucleus of CN5 (sensory tracts from the cornea, mandible, and anterior two thirds of the tongue), motor nucleus of CN6 (abduction of the eyes), motor nucleus and tract of CN7 (facial muscles), and sensory nuclei of CN8 (vestibular component) (slant-

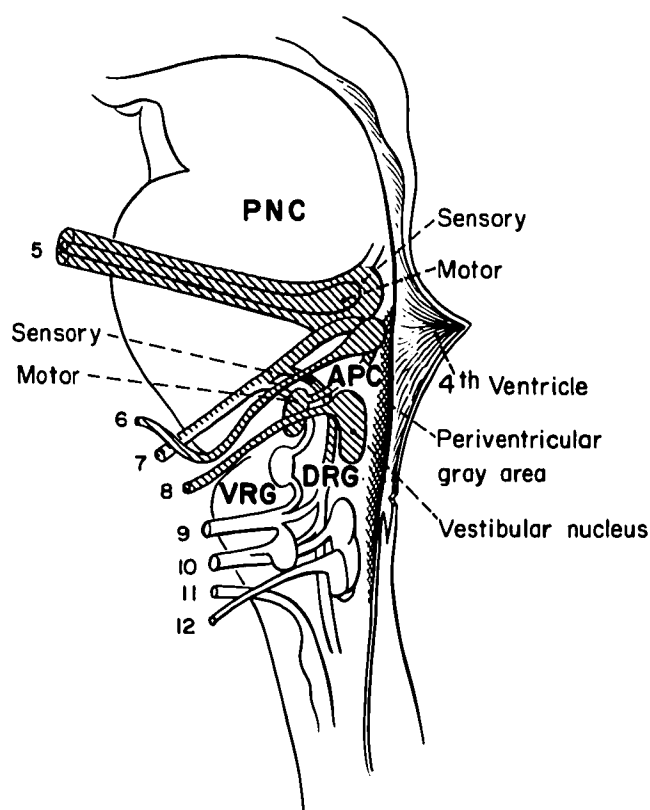


FIG. 2. General locations of cardiovascular centers (hatched area), respiratory centers (bold-lettered areas), and cranial nerves (slant-lined areas) affected in this case. Respiratory centers are designated by the following abbreviations: PNC—pneumotaxic center; APC—apneustic center; DRG—dorsal respiratory group; VRG—ventral respiratory group.

lined areas, fig. 2). All of the aforementioned areas—cardiovascular, respiratory, or cranial nerve components—can be affected in a variety of ways: direct stimulation, direct disruption, compromise as a result of edema, or compromise as a result of vascular events (thromboemboli, stagnant hypoxia, or brain-stem vasomotor reflexes).^{2,9}

The present case illustrates some of the less often appreciated cardiovascular, respiratory and neurologic sequelae attending posterior-fossa surgery. During general anesthesia with mechanical ventilation only cardiovascular changes, as in this case, may be apparent intraoperatively. Such changes should, however, alert the anesthesiologist to expect impaired function in other anatomically related structures. Appropriate therapy should be anticipated prior to the induction of anesthesia. Accurate postoperative assessment of the adequacy of the patient's spontaneous ventilation is necessary, and the means to provide continuous controlled ventilation must be insured. Means to control acute episodes of intraoperative and postoperative hypertension should be at hand. Finally, the need

to provide support and protection when normal reflexes and other physiologic mechanisms are comprised should not be overlooked.

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Medical Gas Outlets—a Hazard from Interchangeable “Quick-connect” Couplers

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The delivery of medical gases from bulk storage facilities through gas pipeline systems is used in hospitals throughout the world for reasons of safety, economy, and convenience. Although such installations offer considerable advantages over the use of small cylinders, there has been a continuing series of accidents due to the “mixing up” of medical gases in pipeline systems. Inadvertent connection of nitrous oxide pipelines to oxygen outlets has been a particularly worrying and dangerous occurrence.¹ This report describes another hazard that may permit the delivery of the wrong gas from a medical gas pipeline system.

REPORT OF AN INCIDENT

A pneumatic tourniquet that is usually inflated with compressed air was found connected to an oxygen piped-gas outlet in the wall of an operating room. Although the male member of the piped-gas connection (or “plug”) was clearly labelled “AIR,” it fitted the oxygen outlet well and the tourniquet was inflated with oxygen. An inspection of all medical gas pipeline outlets and connections in the operating rooms showed that air plugs could be connected to oxygen outlets just as easily as to the appropriate compressed air outlets (fig. 1). Air plugs could also be connected to several of the nitrous oxide outlets, but the plugs for oxygen and nitrous oxide could be connected only to the corresponding outlets for oxygen and nitrous oxide, respectively.

The gas outlets and the “plugs” are standard parts of the Oxecup[†] “quick-connect” system of connections for medical gases.

The plug for each gas has a specific shape fitting a complementary seat retainer bushing within the outlet (fig. 2). Gas flows from the pipeline when the plug protrudes far enough through the bushing to engage a check valve mechanism. The plug may then be retained inside the outlet by a 45 degree clockwise turn that secures two locking pins behind a cam plate. The differing shapes of the plugs and bushings for different gases should prevent misconnection.

The Oxecup plug for air appears to fit through the oxygen bushing further than when an oxygen plug is inserted into an air bushing. This permits the air plug to engage the check valve and open the oxygen pipeline. If the cam plate becomes damaged by wear or misuse, the air plug may be locked and retained in the oxygen outlet. The manufacturer apparently changed the design of the cam plate about five years ago, reducing the possibility of wear and misconnection.

DISCUSSION

The installation and testing of medical gas piping systems are subject to technical standards designed to prevent such mishaps as the inadvertent mixing of medical gases. In the United States, the National Fire Protection Association code 56F “Standard for Non Flammable Medical Gas Systems” requires that quick-connecting station outlets of the type described in this report be designed to avoid the accidental interchange of parts and bodies between outlets for different gases.

At least three hazards may arise from accidental misconnection of Oxecup gas connectors:

1) Connection of an air plug to a nitrous oxide outlet would be especially hazardous if nitrous oxide were erroneously administered to patients. In our institution, service panels with both nitrous oxide and air outlets are restricted to pediatric operating rooms, where the possibilities for such errors are minimal

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