systemic hypertension develops as well as pulmonary hypertension, and there are significant cardiac arrhythmias, including ventricular tachycardia, during the apnea. Apnea occurs during inspiration and a salient clinical sign in these patients is the occurrence of loud snoring. A simple screening procedure consists of recording the sound of the patient's respirations during early sleep by means of a cassette tape recorder at the patient's bedside at home or in the hospital. If findings are suspicious, a polygraphic study of nocturnal sleep is indicated. At present no effective drug therapy has been found and treatment consists of tracheostomy, so prompt otolaryngologic consultation is indicated. Symptomatology is totally reversed by this procedure. In the other form of sleep apnea (so-called central apnea), no obstruction is encountered and respiratory effort ceases. In this form of the disorder, tracheostomy is less helpful.

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Lithium Toxicity in the Central Nervous System

LITHIUM CARBONATE is the accepted treatment of acute hypomania and mania. The mild side effects commonly experienced include nausea, vomiting, diarrhea, fatigue and weakness. Polydipsia and polyuria commonly occur and may be due to inhibition of the action of antidiuretic hormone. There may be a fine, mild resting tremor of the hands which is responsive to treatment with propanolol. The serious signs and symptoms of central nervous toxicity of lithium usually appear with serum concentrations greater than 1.5 mEq per liter and include lethargy, lightheadedness and confusion, accompanied by ataxia, drowsiness, slurred speech, tinnitus, blurred vision and profound weakness. The features of severe intoxication are striking and usually appear either over several days of lithium therapy or following an acute overdose. A patient with severe, acute lithium intoxication presents with tremor, increased neuromuscular irritability, increased deep tendon reflexes, nystagmus, confusion and even convulsions. Nonneurologic signs of intoxication include vomiting and acute diarrhea. All of these signs may progress to stupor, coma and eventual death. It is generally thought that life-threatening lithium intoxication does not occur below serum levels of 3.0 mEq per liter. However, the threshold for intoxication may be lowered in elderly patients and in patients taking other medications including the anticholinesterases and haloperidol. There have been some patients with toxic neurologic manifestations in whom serum lithium levels were within the accepted therapeutic range. Alterations in lithium tolerance may be produced by trauma, infection and sodium restricted diets. Severe lithium toxicity has been reported in a woman with a viral infection causing high fever and a serum lithium level of 1.0 mEq per liter.

An electroencephalogram in a patient who is lithium intoxicated may show a variety of changes. Status epilepticus has been reported in previously nonepileptic patients with lithium concentrations in the therapeutic range. The relationship of lithium to seizures was studied in 16 epileptics in whom only one instance of increased clinical seizure activity was reported. In ten of the 16 patients there was a clear reduction in seizure frequency during lithium therapy. The continuous use of lithium for more than eight months has been reported to be associated with increased muscle tone. The development of the altered muscle tone appears to be directly related to the duration of lithium maintenance therapy and is not relieved by benztropine, an antiparkinson drug. Rarely, patients taking lithium can develop choreoathetosis. Patients exposed to elevated lithium levels for prolonged periods have survived with permanent damage to the cerebellum and basal ganglia.

In assessing lithium toxicity, the clinical signs are the most important means of detecting intoxication. Serum levels may be used for confirmation, but it should always be kept in mind that toxicity may occur with serum levels in the therapeutic range. When signs of serious intoxication are noted, lithium administration should be stopped immedately. As a pharmacologically active cation, lithium is not metabolized in the body, but is excreted by the kidneys. The serum half life of lithium is usually about 16 hours. Hemodialysis, or, if hemodialysis is not available, peritoneal dialysis may be used effectively to lower serum lithium levels. Long-duration hemo-

dialysis for 12 to 16 hours will avoid a rebound effect due to the slow movement of lithium between intracellular and extracellular compartments. The usual cause of death in cases of acute overdose is pneumonia or shock. With appropriate attention to cardiac, renal and pulmonary function, and to the maintenance of fluid and electrolyte balance, the recovery of patients with signs and symptoms of acute lithium toxicity will be optimal.

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Syndrome of Infant Botulism

SINCE THE RECOGNITION of infant botulism about a year ago, at least 20 cases have been reported from various parts of the United States. The incidence of the disease process is, therefore, more common than originally anticipated. Both boys and girls who have been previously healthy are affected in early infancy, and over a period of one to two days the ability to suck and swallow is lost, head control is greatly diminished to absent, and the cry is weak. They may be constipated. Cranial nerve abnormalities include ptosis, sluggish pupillary light reflexes, decreased extraocular motility, a paucity of facial mobility and a decreased gag reflex. The muscle tone and stretch reflexes are reduced. Careful electrophysiologic investigation shows small, short motor unit potentials or small compound muscle action potentials (CMAP) amplitude with an increase in CMAP amplitude with repetitive stimulation as has been described earlier in patients with botulism.

The botulinal toxin may not be identified in the serum but the toxin, as well as Clostridium botulism organisms, have been identified in stool specimens. No botulinal toxin has been identified from the stool of clinically normal humans.

The question, of course, is how do infants get botulism? Some of the infant patients were breast-fed and others received commercial formula, in addition to a variety of infant foods. The C. botulinum spore is ubiquitous, but after intensive epidemiological investigations, no source of the botulinal toxin has been identified. It has been suggested, therefore, that there may be an *in vivo* production of the botulinal toxin, as shown to occur in other forms of animal life.

Although three known patients have had respiratory arrests, all patients with infant botulism have recovered, receiving general supportive care. None of the patients from California have received antitoxin. There has been no evidence that infant botulism is related to the sudden infant death syndrome.

Botulism should be considered in infant patients in whom there has been rapid onset of weakness, with or without abnormalities of cranial nerve function. Careful electrophysiological investigation should be carried out, and specimens of blood and stool should be sent to laboratories equipped for identification of the botulinal toxin.

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Treatment of Multiple Sclerosis with Adrenal Cortical Steroids

THE CAUSE AND CURE of multiple sclerosis (MS) still elude us and many therapies are used to control the symptoms or slow the recurrent inflammatory-degenerative process. The possible immunological basis of the demyelination has been treated without success by transfer factor, antithymocyte globulin or azathioprine (Imuran®). For several years, adrenal corticosteroids or ACTH have been tried, and it seems appropriate to review the current status of this controversial therapy.

When a patient is in an acute relapse and fails to respond to bed rest and simple supportive