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function in an apparently normal manner. The severity of the dementia can be quite variable even from day to day. Apathy and inattentiveness are usually the first symptoms to appear. Gait disturbances generally occur later than the dementia, often several months after the onset of mental symptoms. Commonly, a spastic gait with increased tendon reflexes and extensor plantar signs are present. At times the gait is shuffling or may mimic parkinsonism. The patients frequently fall, and in advanced cases walking, sitting, and standing become increasingly difficult. Urinary incontinence completes the triad of symptoms and may not be noticed by the patient. Often a detailed history from both the patient and family members is necessary to arrive at the correct diagnosis.[2-5]

A diagnosis of hydrocephalus in the elderly patient is difficult to obtain by clinical criteria. Dementia may be produced by drug toxicity, electrolyte disturbance, brain tumor, and metabolic or endocrinologic disorders. Separating patients into etiologic groups may require a comprehensive battery of tests. CT scanning is a safe, noninvasive technique that has greatly facilitated the diagnosis of hydrocephalus and eliminated the need for other invasive procedures.

The prognosis of hydrocephalus is poor if left untreated. Approximately one half of the patients die within the first eight to ten years after diagnosis.[6] The response of patients to cerebrospinal fluid diversion is variable. Those with symptoms of less than six months' duration, without structural etiology or cortical atrophy, whose symptoms improve after cerebrospinal fluid pressure is lowered and who have abnormal saline infusion and radionuclear cisternography have the best prognosis. Sixty percent of patients improve after shunting, with an operative mortality of five to ten percent.[7,8]

In summary, occult hydrocephalus in the elderly can present with a varied clinical format and may suggest diseases of a vascular or degenerative basis. The triad of dementia, gait disturbances, and urinary incontinence should lead one to suspect this disorder. However, it is important to realize that the clinical manifestations may be subtle; therefore, patients who show disordered mental function should receive a thorough workup to explain their dementia.

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Carbon Monoxide Poisoning

Patrick F. Caulfield, MD, and David N. Little, MD
Burlington, Vermont

Carbon monoxide, a hazard in many industries, manufacturing processes, and places of work, is considered the most common cause of industrial poisoning in man. It is odorless, tasteless, colorless, nonirritating, and gives no warning of its presence. In addition, the symptoms associated with its toxicity may be mild and nonspecific.[1] As it has about the same density as air, it mixes readily without stratification.[2] It is produced from the incomplete combustion of organic materials and is a prominent constituent of exhaust fumes from automobile and other engines and from furnaces. Some industries with a greater risk of carbon monoxide exposure include iron and steel foundries, pulp paper mills, petroleum refineries, sintering mills, and plants manufacturing formaldehyde and coke.[1] Carbon monoxide's ubiquity and its avidity for combining with hemoglobin at the expense of oxygen make it a major environmental

From the Department of Family Practice, College of Medicine, University of Vermont, Burlington, Vermont. Requests for reprints should be addressed to Dr. David N. Little, University of Vermont, A-111 Given Building, Burlington, VT 05405.

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health hazard.[3] A concentration of less than 0.1 percent in the inspired air can be fatal.

Case Report

A 21-year-old, married, white man presented to the family practice office with a chief complaint of "blacking out." While at work in a small printing factory, he noted the onset of progressive weakness, severe headache, nausea, and dizziness. His co-workers had assisted him outside the building, where he was noted to lose consciousness for a period of several seconds. When he regained consciousness, the patient was not disoriented or incontinent. He was brought to the office by his co-workers.

Further questioning revealed that the patient's job involved unloading cargo from a propane powered forklift, operated within a confined space. Approximately three months before the above episode and soon after the company purchased the forklift, he and several of his co-workers had noted the onset of headache. The patient's headache was generalized and most noticeable toward evening. There were associated symptoms of fatigue, anorexia, and episodic dizziness. In retrospect, the patient recalled that his headache was generally worse on Fridays and would subside over the weekend when he was not at work. There was no prior history of significant medical illness; review of symptoms was noncontributory with respect to the above episode. The patient's habits included smoking ten cigarettes a day.

On physical examination the patient was alert and oriented. The blood pressure was 120/72 mmHg; pulse, 88 beats per minute and regular; oral temperature, 36.8 C; and respiration, regular at 20 per minute. The chest was clear on auscultation; cardiac rate was regular; and no abnormal sounds were heard. Neurologic examination was within normal limits. Except for generalized pallor, no skin changes were present.

Laboratory data showed a total white blood count of 8,700/cu mm, with a normal differential count. Hemoglobin was 15.7 gm/100 ml, and hematocrit 45 percent with normal red blood cell indices. The serum glucose was 92 mg/100 ml. Serum potassium was 4.2 mEq/liter. Serum creatinine was 1.1 mg/100 ml. Serum calcium was 9.3 mg/100 ml.

Because of the suspicion that the forklift exhaust was responsible for the patient's symptoms, a determination of carboxyhemoglobin was made. On a blood specimen drawn approximately one hour after leaving the place of work, carboxyhemoglobin was reported at 31 percent, with a normal value of 0.2 percent and up to 8 percent in smokers. Subsequent determinations of carboxyhemoglobin level from the patient's co-workers with all samples drawn at the end of a working day ranged from a high of 21 percent to a low of 11 percent.

Consultation was obtained from the Division of Occupational Health of the state of Vermont; the involved plant was investigated by an industrial hygiene engineer. Carbon monoxide analyzers were used to determine levels of carbon monoxide produced by the forklift truck. At a distance of three feet from the exhaust pipe, with the truck engine in the idle mode, a reading of 110 ppm of carbon monoxide was recorded. A reading of 300 ppm was recorded at two feet. Background carbon monoxide readings obtained with the large loading dock door open were recorded as 0 to 4 ppm. The maximum allowable concentration permitted in industrial installations by the National Institute of Occupational Safety and Health Administration is 50 ppm, but some authorities consider even this figure to be too high for prolonged exposure.[4]

Comment

A propane powered forklift truck, particularly with an improperly tuned engine, may represent an industrial hazard when used in a confined space. As the above case illustrates, symptoms may be recognized by workers but not reported for medical attention until significant toxic effects have occurred.

The most commonly associated symptoms of carbon monoxide poisoning are dose related (Table 1). Initial symptoms involve the central nervous system and include headache, dizziness, and nausea progressing to vomiting. Hyperreflexia and paresthesia are reported less frequently. Hypertension may be found in two thirds of the patients, and tachycardia in less than 10 percent. Muscular weakness and complaint of inability to continue work are common (greater than 50 percent). Respiratory complaints of shortness of breath and chest tightness may be found in 2 to 10 percent of patients.[3,4]

Progression into collapse, stupor, coma, and death may ensue unless the patient is removed

Table 1. Carbon Monoxide Poisoning

Percent Carboxyhemoglobin Saturation in Blood	Signs and Symptoms
0-10	None
10-15	Headache
25	Severe headache, nausea, fatigue, poor exercise tolerance
35	Severe headache, weakness, vomiting, dizziness, visual disturbance
45	Collapse, vomiting, tachypnea, tachycardia
55	Stupor, convulsions, Cheyne-Stokes respiration, may be fatal
65	Coma, convulsions, cardiovascular, and respiratory depression, often fatal
>70	Nearly always fatal

from the carbon monoxide exposure. Aspiration pneumonia, adult respiratory distress syndrome, cerebral hemisphere demyelination, peripheral neuropathies, rhabdomyonecrosis, myocardial damage, and renal insufficiency may all occur as complications of severe carbon monoxide poisoning.

Detection is dependent on history and a high index of suspicion. Since biochemical repair begins as soon as the patient is exposed to fresh air, a history of exacerbation and remission of symptoms may be obtained. The possibility of carbon monoxide poisoning should be considered in patients without other explanation for a syndrome of headache, dizziness, weakness, and nausea. Investigation of industrial or private exposure to exhaust fumes from internal combustion engines or furnaces may be indicated in the workup of this problem.

The administration of oxygen is the most important element in therapy. For a resting adult, it takes four hours to eliminate one half the carboxyhemoglobin breathing room air, but only 40 minutes if breathing pure oxygen. When respiration is absent or inadequate, artificial respiration may be necessary; in severe cases, hyperbaric oxygen has been life saving.[5]

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Personality and Values in Family Medicine Residents

Gordon J. DiRenzo, PhD
Wilmington, Delaware

It seems to be a prevalent, and somewhat optimistic, assumption that people entering medicine nowadays, and family medicine in particular, are by and large appreciably different in personality from their counterparts of a decade or two ago. Chiefly indicative of this situation are the alleged differences in values that are typical of today's physicians. Evidence to support this assumption, however, is not conclusive. Efforts have been directed at an exploratory analysis of this question through a number of conceptual and methodological dimensions that have not been applied in the

From the Department of Sociology, University of Delaware, and the Family Practice Residency Program, St. Francis Hospital, Wilmington, Delaware. Requests for reprints should be addressed to Dr. Gordon J. DiRenzo, Family Practice Residency Program, St Francis Hospital, Seventh and Clayton Streets, Wilmington, DE 19805.

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