options, such as nitroglycerin and calcium channel blockers, for treatment of cocaine-associated MI.

Central nervous system effects include seizures and strokes. Seizures are most commonly tonic-clonic. Some investigators speculate that cocaine causes a decline in the seizure threshold. However, hyperpyrexia and acidemia may exacerbate the situation. Hemorrhagic and ischemic strokes have been described after cocaine use, regardless of the route of administration. Hypertension may predispose cocaine users to cerebrovascular accidents as a result of an acute rise in blood pressure. Other less common effects include migraine headache, acute dystonic reactions, and cerebral vasculitis (one case).

Cocaine hepatotoxicity has been documented in various animal models, but its existence in humans is speculative. This entity appears pathologically similar to acetaminophen toxicity, with zones of well-demarcated necrosis, mild hepatitis, and steatosis in surviving hepatocytes. The mechanism of its development is unknown, but is thought to be related to the production of a toxic free radical (norcocaine nitroxide).

Intestinal ischemia, or the "body packer syndrome," is a consequence of a smuggling tactic. Cocaine dealers wrap 5 to 7 grams of cocaine in individual latex packets. These are then ingested—as many as 180 at one time. If the packets rupture, a large bolus of cocaine is delivered to the gastrointestinal mucosa and causes vasospasm and ischemia. Deaths have been reported with this syndrome, and many believe that surgical removal is the only appropriate therapy.

The pulmonary effects of freebase cocaine smoking include pulmonary edema, pulmonary barotrauma from deep inspiration and prolonged Valsalva maneuver, pulmonary alveolar hemorrhage, hypersensitivity pneumonitis, and bronchiolitis obliterans. The mechanism of pulmonary edema is postulated to be a direct cocaine-mediated increase in pulmonary vascular permeability.

Among drug users with endocarditis, the frequency of cocaine use is 78%. The reasons for the relationship between cocaine and infective endocarditis are hypothetical and include frequency of use, methods of administration, differences in bacterial flora, and a direct effect of cocaine on the immune system. HIV infection and cocaine abuse has been well described in San Francisco, with an HIV seroprevalence of 35% in daily intravenous cocaine users.

One case of renal infarction has been reported in a 32-year-old man after intravenous injection of cocaine. A recent study identified 39 patients in whom acute rhabdomyolysis developed after cocaine use. Acute renal failure developed in one third of the group. This subset had a higher peak serum creatine kinase level. Severe hepatic dysfunction developed in most patients with renal failure.

Obstetrical complications of cocaine use have been well described and include preterm labor, spontaneous abortion, and abruptio placentae. Infants born to cocaine-using mothers are more likely to have intrauterine growth retardation, lower APGAR scores, congenital heart and genitourinary malformations, and neurobehavioral impairment.

Psychiatric manifestations of cocaine abuse vary depending on the amount and extent of use. Acute intoxication results in impaired judgment, impulsiveness, hypersexuality, and psychomotor agitation. A prolonged binge causes anxiety, irritability, panic, fear of impending death, paranoia, and delusions. With abstention, there is extreme exhaustion, intense depression, and hyperphagia. Withdrawal from cocaine is intense and requires a coordinated approach by trained physicians, counselors, and paraprofessionals.

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KEY STEPS IN EVALUATING THE DIZZY PATIENT

Because "dizziness" has different meanings to different people, a careful history is essential to accurately differentiate other forms of dizziness from vertigo, and to diagnose the cause. When a patient complains of dizziness, it is important to ask what he or she means by the symptom and then wait for the patient's answer, without suggesting any explanation. As a group, patients with dizziness tend to be suggestible; leading questions will produce a useless history. The patient who complains of

"vertigo" should be assessed as though the complaint were "dizziness." Few nonphysicians use the word "vertigo" properly.

VERTIGO V DIZZINESS

Given time to frame an answer, the patient's description will fall into one of four categories.

An illusion of motion. The feeling that something is moving when it is not describes vertigo and is a vestibular system dysfunction. Regardless of whether the patient reports a sensation of spinning, rocking, or tilting, it is vertigo. The challenge is to decide whether the problem originates in the central or peripheral nervous system.

Near-syncope, or sensation of impending faint. This is a manifestation of decreased cerebral blood flow. The prime suspect is drug therapy, particularly effective antihypertensives, but the patient also should be queried about the use of over-the-counter drugs and alcohol intake. Cardiac arrhythmias rarely cause dizziness. "Swooning" or fainting is a normal phenomenon that tends to run in families. The cause is a failure of the sympathetic pulse in the upright posture, or inappropriate vasodilation. It is best treated by educating the patient to sit or lie down, or put the head down.

Syncope is distinctly different, characterized by a sudden loss of consciousness with no warning. The cause is usually cardiovascular.

Disequilibrium, or sensation of losing balance. It is informative to watch the patient walk. Patients with Parkinsonism (who may say they feel as though they are falling backwards), cervical spondylosis, or spasticity all have characteristic gaits.

In the elderly individual, arthritis, trauma, or alcohol may lead to a gait disorder that the patient calls dizziness. The underlying cause is proprioceptive dysfunction in the legs. Drugs will not relieve this problem and are best avoided. Instead, the patient should be instructed never to get up and use the bathroom at night without first turning on the light and sitting on the edge of the bed for at least 1 minute.

Ill-defined light-headedness. Ill-defined dizziness that is not vertigo, near-syncope, or disequilibrium is generally a psychiatric problem. The dizziness is usually being used as a metaphor for depression, anxiety, or both.

Since each of these four types of responses accounts for 25% of patients who present with dizziness, 75% of patients who present with dizziness will not respond to anti-vertigo medication. Indeed, their symptoms of dizziness will worsen with the superimposition of side effects of vertigo medications.

For patients whose presenting symptom is clearly vertigo, the diagnostic workup must differentiate whether the causative disorder is in the central (brainstem or parts of the cerebral cortex, particularly the temporal lobe) or peripheral (end organ or peripheral nerve) nervous system.

DIFFERENTIAL DIAGNOSIS OF VERTIGO

The physical examination must include a complete examination of both the vestibular and cochlear eighth nerves. Because of the close proximity of these two nerves and of their end organs, disease of one may affect the other.

Cochlear nerve evaluation

Examination of the cochlear system requires a test for pure tone hearing loss. If this is positive, then it must be determined whether the loss is sensorineural or conductive. These tests can be carried out in the office, using the Weber test (a vibrating tuning fork placed at the midline of the skull) and the Rinne test (placing the tuning fork first over the mastoid bone and then in front of the ear.

There are practical problems with both the Weber and Rinne tests. For example, in the Weber test, the patient is asked to indicate on which side the sound can be heard, and tends to point directly to the tuning fork, rather than to one side or the other. The best way to perform the Rinne test is to put the tuning fork first over the mastoid process and then in front of the ear and ask the patient which is louder. If the patient with hearing loss indicates that it was louder when in front of the ear, then there is no doubt about the presence of a sensorineural problem.

If there is sensorineural hearing loss, one needs to discriminate between cochlear (end organ disease) and retrocochlear (peripheral or central neural disease). An adequate office screening test involves whispering words into one affected ear while making enough noise in the other ear so that the patient cannot hear through it. A patient with cochlear-type hearing loss, as occurs in Meniere's disease, should be able to distinguish 70% or more of the words heard; in retrocochlear hearing loss, the patient may understand only 2 of 10 spoken words.

Vestibular nerve evaluation

The objective of the vestibular nerve examination is to elicit nystagmus and stress the vestibular apparatus to reproduce vertigo. Nystagmus can be observed by asking the patient to look about 45° to the left and then to the right and, if nystagmus develops, recording the fast-phase

and slow-phase direction and the position in which they occur. The Nylen-Barany maneuver will indicate whether the vertigo is positional only.

Electronystagmography can be carried out, but it is an uncomfortable procedure and most patients can be diagnosed without it.

As a rule, if the patient has slow phase nystagmus toward the lesion and fast phase nystagmus away from the lesion, or says the environment is spinning away, the lesion is peripheral, involving the nerve or end organ. Cochlear criteria can be used to localize the lesion to one of three categories: peripheral-cochlear, peripheral retrocochlear, or central.

The common causes of peripheral cochlear disease are vestibular neuronitis, labyrinthitis, and benign positional vertigo. Retrocochlear disease is caused by acoustic schwannoma, meningioma, or cerebellopontine angle tumors. A central disorder is caused by vertebrobasilar insufficiency, demyelinating disease, or drugs that intoxicate the vestibular system.

TREATMENT OF VERTIGO

Although some disorders that cause vertigo require specific treatment, many are self-limiting and the patient can be treated symptomatically with one of three classes of anti-vertigo drugs. (1) The anticholinergic antihistamines work by suppressing the central pathways and are all equally useful. Drugs in this class include buclizine, cyclizine, dimenhydrinate, diphenhydramine, diphenidol, and meclizine. (2) Anticholinergic phenothiazines, such as promethazine, help to relieve the nausea associated with vertigo. These drugs are often given parenterally in the emergency room. (3) Scopolamine is the prototype anticholinergic drug, with psychiatric and cardiac effects.

It is acceptable to use benzodiazepines in the emergency room to relieve anxiety, but they cause nystagmus and vertigo, and should not be used to the treat the vertigo per se.

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