cause seizures which usually require no treatment other than allowing the blood level to fall, but may require and usually respond to valium.) Larry Proano, MD

anesthesia, local; toxicity

Rational management of bronchial asthma. Chodosh S, *Arch Intern Med* **138:**1394-1397, (Sep) 1978.

A general discussion and assessment of the management of bronchial asthma, both inpatient and outpatient, is presented. Often modification of the patient's environment and identification and avoidance of known allergies can provide the basis for success in the patient's management. The mainstay of maintenance pharmacologic treatment is bronchodilator therapy, with the drug of choice being theophylline. The choice of exact agent and route of administration will depend on the individual patient. The second group of bronchodilator agents is the sympathomimetic amines. They may be added to theophylline or used primarily in patients who cannot tolerate theophylline. Terbutaline has the least cardiogenic and most bronchodilator action of the sympathomimetics available for oral use. Ephedrine is still the most commonly used sympathomimetic and when combined with theophylline its action may be comparable to terbutaline. Subcutaneous or intravenous bronchodilator therapy is usually reserved for the acute attack requiring emergency treatment. Cromolyn sodium has decreased the severity of symptoms and the need for other antiasthma medications. Its action may be due to inhibition or release of mast cell mediators. Steroids are usually required in the patient who has persistent symptoms in the face of full therapy. A discussion of dosage requirements for all medications discussed is included. *(Editor's note: Not much new but a useful review of common drugs. A suggestion for when to admit would have been helpful.)* Peter Pons, MD

asthma

Annotation — carbon copy pain of myocardial infarction. Short D, *Am Heart J* **96:**417, (Sep) 1978.

Three hundred and thirty patients with angina were followed until death or until an average of 6½ years had elapsed since the onset of their symptoms. For each individual the site of anginal pain was constant, although different from patient to patient. One hundred and forty-five subsequently developed myocardial infarction. In 136 cases of these (94%), the epicenter of the pain of infarction was similar to that of the previous angina. In the other nine (6%) cases the pain was lower than that of the angina. No patient who developed pain centered above or lateral to the previous anginal pain developed evidence of infarct, regardless of the severity of the pain. *(Editor's note: Pain similar to prior pain is useful in distinguishing cardiac from other chest pain. Unfortunately it will not distinguish angina from infarction pain.)* Brian Allen, MD

angina pectoris; chest pain

Noninvasive diagnosis of deep vein thrombosis by phleborheography. Bynum LJ, Wilson JE III, Crotty CM, et al, *Ann Intern Med* **89:**162-166, (Aug) 1978.

Phleborheography is a noninvasive test for deep vein thrombosis (DVT), a diagnosis difficult to make on clinical signs and symptoms. The technique involves plethysmographic measurements of changes in leg blood volume by means of recording cuffs placed at midthigh, upper, mid, and lower calf. The detection of DVT requires reduction or obliteration of the normal respiratory wave variation of venous return. Also required is that compression of the foot or calf will lead to a baseline elevation of the tracing, reflecting increased leg volume due to the "damming" of blood behind the obstruction. Seventy-five patients with suspected DVT were studied using phleborheography and venography. The study was conducted in a prospective, controlled fashion using blinded interpretation of test results. Acute DVT was accurately diagnosed by phleborheography with no false positive diagnoses. There were 31% false negative diagnoses. Hence, if phleborheography is positive, this diagnosis may be accepted with confidence. If negative, venography may still be required to withhold anticoagulation. *(Editor's note: Although neither test can be conveniently done in the emergency department, the physician can take comfort in the fact that many patients will not have to suffer the morbidity and mortality of venography to confirm the diagnosis of DVT.)* Howard R. Lee, MD

thrombophlebitis, phleborheography

Renal artery embolism. Lessman RK, Johnson SF, Coburn JW, et al, *Ann Intern Med* **89:**477-482, (Oct) 1978.

The clinical features and long-term follow-up of 17 cases of spontaneous renal artery embolism are presented. Cardiac disease, especially atrial fibrillation, was found in 16 of 17 cases. Thirteen patients noted pain in the flank, abdomen, and chest, or lower back, or both. Four patients noted no pain. Nearly half the patients had nausea and vomiting. Abdominal pain or flank tenderness to palpation or percussion was found in 12 patients. The oral temperature was usually elevated. Leukocytosis was present in all patients (mean, 19 900/mm^3). Microscopic or chemical hematuria was the most common finding on urinalysis. Significant pyuria was noted in 10 of 12 cases. Proteinuria was uniformly present. An elevated lactic dehydrogenase (LDH) was present in all cases. Serum creatinine was greater than 4.0 in 65%. In only four cases was the correct diagnosis made on admission. The correct diagnosis was made on the first day of admission in an additional five patients. The diagnosis is made using IVP, retrograde urography, and renal scintiscan or arteriography. Four patients required dialysis. Emboli to other organs caused early death; cardiovascular disease led to later death. A favorable outcome is common with long-term anticoagulants. The role of surgery is unclear. *(Editor's note: In addition to atrial fibrillation, the diagnosis of peripheral embolization should always be high in patients who have recently had a coronary angiogram or aortogram.)* Howard R. Lee, MD

embolism, renal artery

Menopause and coronary heart disease. Gordon T, Kannel WB, Hjortland MC, et al, *Ann Intern Med* **89:**157-161, (Aug) 1978.

A study of 2873 women in the Framingham Study revealed a rise in coronary artery disease incidence after menopause. In addition, the severity of the presenting disease was increased. These effects were present whether menopause was natural or surgical. Removing the uterus alone or removing both the uterus and one or both ovaries increases coronary heart disease risk. No premenopausal woman developed myocardial infarction or died of coronary heart disease. In contrast, such events were common in postmenopausal women. Estrogen use after menopause doubled the risk of coronary heart disease, especially angina pectoris. *(Editor's note: Another in a long series of indictments of the use of exogenous estrogens.)* Howard R. Lee, MD

coronary disease, menopause