The detection method for this PCR product used submarine gel electrophoresis. Culture medium and culture transport solution were also analyzed by direct DNA fluorescent assay. Sensitivity, specificity, and positive and negative predictive values were calculated for culture and Amplicor PCR assay. McNemar chi square analysis was used to evaluate the hypothesis that tests were not statistically different.

During the first 5 weeks of the study period, 337 individuals provided endocervical samples. Prevalence of *C. trachomatis* was 17 of 337 (5.0%). McCoy cell culture was 71% sensitive and 100% specific with a positive predictive value (PPV) of 100% and a negative predictive value (NPV) of 98.5%. The Amplicor PCR-based assay was 94% sensitive and 99.7% specific with a PPV of 94% and a NPV of 99.7%. Only 1 discrepant sample gave a positive culture with a negative Amplicor PCR-assay. Follow-up of residual media was not possible, but the sample was considered to be a true positive due to the production of inclusion bodies in culture. Six samples gave a positive Amplicor PCR-assay and a negative culture. Five of these discrepant samples tested positive in a second PCR assay and were considered to be true positive. One tested negative and was considered to be a false positive. Preliminary analysis with McNemar chi square indicated no significant difference between the two assays. However, if results for the next 663 samples follow the current trend, the *P* value is estimated to be .044.

In preliminary results of a study in progress, the Amplicor PCR-based assay for *C. trachomatis* compared favorably with standard culture methods. If results follow the trend, 1,000 samples should allow detection of significant differences in assay methods.

**COMBINED MUSCLE-STROMAL TUMOR (STROMALOMA) OF UTERUS.** Hussain E. Rawji, MD, Max E. Blue, MD, Stephen P. Snow, MD, and Orlando R. Gonzalez, MD. Departments of Obstetrics and Gynecology and Pathology, Orlando Regional Healthcare System, Fla.

A 47-year-old white woman came to the emergency room with sudden onset of abdominal pain. Examination showed a huge rock-hard mass from the symphysis pubis to the xiphoid process. Computed tomography scan revealed a mass consistent with a uterine fibroid with two necrotic centers. At surgery the uterus was found to be greatly enlarged, weighing 6,150 g and measuring 30 x 20 x 20 cm. The myometrium contained a 23 cm circumscribed solid yellow-white nodule that bulged above the cut surfaces. Two areas (each 2 cm in diameter) of yellow necrosis were present. A small (1 cm) firm white leiomyoma was present elsewhere. Microscopically the tumor consisted mainly of cells intermediate between smooth muscle and endometrial stromal cell type. Each low power field contained areas resembling endometrial stroma.

Neoplasms composed of both smooth muscle and endometrial stroma (stromaloma) are very rare, and little is known about them. Most are benign, but the presence of endometrial stroma, as in a low grade stromal tumor, casts uncertainty over the behavior, as most endometrial stromal tumors are malignant.

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**Medicine**

THE VALUE OF MULTICHANNEL BIOCHEMICAL SCREENING: A PROSPECTIVE STUDY IN A BALTIMORE CITY HOSPITAL WARD SERVICE. Joseph Puthumana, MD, Robert P. Ferguson, MD, Frank Kohler, MD, Sohail Zaidi, MD, Jose Chavez, MD, and Huma Shakil, MD. Union Memorial Hospital, Baltimore, Md.

Previous studies have suggested that routine biochemical testing is not cost effective and can have misleading results. However, most of these studies are dated and/or deal with different patient populations than are the current reality of city hospital ward services.

To assess the clinical utility of multichannel screening, we are studying 300 randomly selected patients admitted to Union Memorial Hospital medical staff service. Patients automatically receive an SMA 24. Admission history and physical examination are prospectively reviewed by a five-member residency/faculty panel, and working diagnoses are established. For each patient, the 24 components of the SMA, by group consensus, were divided into tests that were acutely indicated, chronically indicated, or not indicated by any current or known medical conditions (screening). Abnormal results were divided, based on predetermined criteria, into expected and unexpected; unexpected was further divided into important or clinically unimportant. Abnormalities judged important by clinical criteria or by degree of variance from the reference range resulted in a prescribed course of action. Hospital testing costs were computed.

Of the first 58 patients studied in November and December 1993, the mean age was 48 years (range 18 to 85). Patients were 50% male and 50% female, 75% black and 23% white. Of 1,392 biochemical tests, 397 (28%) yielded abnormal results. Of 346 acute tests, 115 (33%) yielded abnormal results. Of 74 chronic tests, 37 (50%) yielded abnormal results. Of 978 screening tests, 245 (25%) yielded abnormal results. Of the 245 screening tests with results outside the reference range, 20 results were expected and 48 were judged unimportant. The
remaining 177 (18%) screening abnormalities were unexpected and important. By consensus, actions recommended were the following: immediate treatment (15), repeat tests (31), workup (122), outpatient treatment (1), no workup (8). Of the 58 patients, only 5 had a negative screening profile. The most common screening abnormalities (more than 25% of patients) were lactate dehydrogenase, total iron, cholesterol, magnesium, uric acid, and amylase. The true costs were $10 per panel ($580).

Preliminary results indicate that multichannel biochemical screening in this population has a high yield with significant clinical implications at minimum cost. We believe that if these preliminary trends continue, the study will strongly support the use of multichannel biochemical screening in similar patient populations on a routine basis.

EXTREME HYPERNATREMIA WITHOUT NEUROLOGIC INJURY: POSSIBLE PROTECTION BY HYDROCEPHALY. Julia Dannelly, MD, and R. Michael Culpepper, MD. University of South Alabama, Department of Medicine, Mobile.

Extreme hypernatremia ($S_{nr} > 180$ mEq/L) is associated with high morbidity and mortality rates. Morbidity and death are presumed to be due to intracerebral hemorrhage after osmotic shrinkage of brain cells. An increase in cellular osmoles protects against cellular osmotic shrinkage. Hydrocephalus may provide another buffer to decrease injury from changes in central nervous system cell volume.

A 27-year-old woman with growth and mental retardation, muscle spasticity, and seizures since age 1 month had a 48-hour history of muscle laxity, decreased response to family, and poor oral intake. A seizure was noted on presentation.

In the hospital (days 0 to 3), normal saline (0.9%) was infused to replenish the extracellular fluid volume; 1/2 normal saline was then given orally with water to replenish body water. The patient’s baseline mental function returned at a serum sodium level = 180 mEq/L. On both outpatient visits, neurologic function was unchanged.

This infantile patient has survived extreme hypernatremia that was associated with a 15% to 20% body water loss. She maintains an intense diuresis that likely reflects her inability to obtain water normally. The "cushioning" effect of a high intraventricular volume may help protect against shifts in central nervous system cell volume.

TREATMENT OF HYPMAGNESEMIA WITH THE POTASSIUM-SPARING DIURETIC AMILORIDE. Joel T. Bundy, MD, David J. Connito, MD, Michael D. Mahoney, MD, and Paul J. Pontier, MD, PhD. Naval Medical Center, Portsmouth, Va.

Hypomagnesemia is a common electrolyte abnormality. However, only rarely has it been attributed to idio-

pathic renal magnesium wasting. We describe a case of idiopathic renal magnesium wasting and a unique approach to treatment.

An 18-year-old black woman came to us complaining of pedal spasm for three days. She had no other constitutional symptoms, no significant past medical history, was taking no medications, and had normal results on physical examination. Laboratory assessment revealed a serum magnesium level of 1.0 mg/dL with a urinary magnesium level of 48 mg/day. All other laboratory values were within normal limits, including urine calcium, urine aldosterone, plasma renin activity, plasma aldosterone, parathyroid hormone, vitamin D, and thyroid-stimulating hormone. Urine screen for diuresis also yielded normal results, and neither parent had a depressed magnesium level. Treatment with intravenous and oral magnesium only partially corrected the serum magnesium level because there was also a marked increase in the urine magnesium excretion. Treatment with amiloride was begun, and a further rise in the serum magnesium level was seen along with a concomitant decrease in the fractional excretion of magnesium and improvement in symptoms.

This case illustrates the potential benefit of amiloride in treatment of hypomagnesemia due to renal magnesium wasting. Magnesium reabsorption occurs in the distal convoluted tubule, the site where amiloride also exerts its effect. The mechanisms of magnesium sparing with amiloride include lowering of the transepithelial electrical potential, thus favoring magnesium reabsorption; alkanization of the urine, which may decrease magnesium excretion; and inhibition of magnesium secretion. In light of these findings, amiloride should be considered in the treatment of idiopathic renal magnesium wasting disorders.

SPONTANEOUS HYPOGLYCEMIA IN CONGESTIVE HEART FAILURE DUE TO HYPERINSULINISM. Michelle Wilson, DO, Timothy Daugherty, MD, and Kaveh Ehsanipoor, MD. Memorial Medical Center, Department of Internal Medicine, Savannah, Ga.

Spontaneous hypoglycemia associated with congestive heart failure (CHF) is rarely reported in adults, although more cases have been reported in neonates and infants. The mechanism of hypoglycemia in CHF has not been clearly defined. In 1 reported case, the serum insulin level was measured and was found to be appropriately suppressed.

A 72-year-old man with a known history of hypertensive dilated cardiomyopathy came for treatment with exacerbation of CHF. He had no history of diabetes and had not ingested alcohol or an oral hypoglycemic agent. On hospital day 2, profound hypoglycemia developed and required aggressive intravenous glucose infusion. Laboratory workup showed the following values: blood urea nitrogen, 47 mg/dL; creatinine, 1.8 mg/dL; and cortisol, 34.1 ng/dL. The serum T4 level was normal. Serum glu-
cose, insulin, and C peptide levels before and after therapy were as follows: before—glucose 21 μU/mL, insulin 33 ng/mL, and C peptide 27 μg/dL; after—glucose 51 μU/mL, insulin, 4 ng/mL, and C peptide 7.4 μg/dL.

As noted, an inverse relationship exists between insulin and serum glucose levels, indicating hyperinsulinemia as the cause of hypoglycemia. Serum glipizide and glyburide were not detectable. Computed tomography of the abdomen yielded negative results.

Hypoglycemia in CHF is due to hyperinsulinemia, which is probably caused by impaired insulin degradation and shunting of portal blood into the systemic circulation. Coexisting renal insufficiency could be another contributing factor.

CORTICOSTEROID-BINDING GLOBULIN DEFICIENCY MASQUERADING AS ADRENOCORTICAL INSUFFICIENCY. Edwin M. Purvis, MD, Thomas Peter, MD, and Stephen A. Breetzke, MD. 81st Medical Group, Department of Medicine, Keesler AFB, Biloxi, Miss.

A 71-year-old white man with underlying cirrhosis due to hepatitis B and C had evaluation of symptoms of orthostatic faintness and documented orthostatic hypotension. Rapid cosyntropin stimulation test revealed subnormal serum cortisol of <5 μg/dL at baseline and 60 minutes after administration of cosyntropin; plasma corticotropin level was within normal limits, and magnetic resonance imaging of the sella and suprasellar region yielded normal results. Based on these findings, a tentative diagnosis of idiopathic secondary adrenocortical insufficiency was made, and the patient was treated with a physiologic replacement dose of hydrocortisone (20 mg orally in the morning and 10 mg orally in the evening). Despite this therapy, his condition remained subjectively and objectively unimproved with regard to orthostatic symptoms and signs.

Subsequent endocrine evaluation revealed a supranormal production rate of cortisol, as reflected by 24-hour urinary free cortisol value >300 μg on two different occasions. Deficiency of corticosteroid binding globulin (CBG) was suspected and confirmed by direct serum assay. Subsequently, hydrocortisone replacement therapy was discontinued without clinical sequelae.

Discordant measurements of increased cortisol production (evidenced by 24-hour urinary free cortisol value) and decreased serum cortisol value (reflecting both free and CBG-bound cortisol) should cause suspicion of CBG deficiency, a diagnosis that can be confirmed by direct measurement of CBG. This innocuous biochemical anomaly can be a feature of advanced hepatic failure and must be distinguished from true adrenocortical insufficiency.

PSEUDOTUMOR CEREBRI: AN UNUSUAL PRESENTATION OF SARCIOIDOSIS. Anthony Stampaia, MD. Presbyterian/St. Luke's Medical Center, Denver, Colo.

A 39-year-old black man with diabetes mellitus and hypertension was admitted to the hospital with a 3-week history of headache and blurred vision but normal blood pressure. Best corrected visual acuity was 20/50 bilaterally. Slit lamp examination of anterior segment showed no signs of inflammation. Direct and indirect ophthalmoscopy revealed massive papilledema in each eye, with protein around the disk and macular exudate. Opening pressure at lumbar puncture was 290 mm H2O; there were no inflammatory cells. Computed tomography and magnetic resonance imaging of the brain showed no abnormality. A chest film showed bilateral hilar adenopathy. Transbronchial biopsy showed epithelioid noncaseating granulomas. The serum level of angiotensin-
converting enzyme (ACE) was 77 U/L. Pseudotumor cerebri can be caused by a wide variety of illnesses. The diagnosis of sarcoidosis in this case was made by characteristic chest roentgenographic findings, transbronchial biopsy, elevated ACE level, negative bacterial and fungal cultures of tissue biopsy and sputum specimens, and exclusion of other possible causes. The patient was treated with analgesics, diuretics, steroids, and immunosuppressive therapy and did well.

SARCOIDOSIS-ASSOCIATED HYPERCALCEMIA AFTER SUCCESSFUL TREATMENT OF CUSHING’S DISEASE. Capt Sean D. Fink, MD, and Maj James K. Rone, MD. Keesler Medical Center, Keesler AFB, Biloxi, Miss.

A 43-year-old woman with a history of Cushing’s disease was referred for evaluation of hypercalcemia. She came for treatment with pancreatitis, nephrolithiasis, and persistent hypercalcemia (>13.0 mg/dL) 10 months after curative pituitary surgery. Intact parathyroid hormone (PTH) levels were low (<10 pg/mL), and a workup for malignancy, including bone scan and PTH-RP, yielded negative results. Sarcoidosis was diagnosed after a biopsy of multiple subcutaneous nodules. Vitamin D studies revealed a low-normal 25-OHD level (9 ng/mL, normal 10 to 55), with a high-normal 1,25-OHD level (52 pg/mL, normal 18 to 62). The hypercalcemia was successfully treated with forced saline diuresis and a single dose of intravenous pamidronate. After 14 days without specific therapy for sarcoidosis, serum calcium levels remain normal.

Sarcoidosis associated with Cushing’s syndrome has been reported previously only once, and skin nodules were the sole manifestation in that case. Hypercortisolemia would suppress sarcoid activity; therefore, it is probably no coincidence that the onset of clinical sarcoidosis in our patient was delayed until after resolution of the cushingoid state.

Hypercalcemia occurs in up to 20% of patients with sarcoidosis. The mechanism appears to be granulomatous conversion of 25-OHD to 1,25-OHD. Sarcoidosis-associated hypercalcemia is usually treated with glucocorticoids, a treatment medically and psychologically undesirable in this patient, with her history of Cushing’s disease. Alternative therapies include chloroquine and ketoconazole. This is the first reported use of pamidronate in this setting. Our experience suggests that long-term control of sarcoid hypercalcemia may be achieved with a single dose of intravenous pamidronate.

EXPERIENCE WITH COMBINED TREATMENT OF HYPERTHYROIDISM OF GRAVES’ DISEASE WITH METHIMAZOLE AND THYROXINE. William W. Winternitz, MD. University of Alabama School of Medicine, Tuscaloosa Program, Tuscaloosa, Ala.

Management of Graves’ disease hyperthyroidism has been controversial over a long period of time. Thyroidologists around the world differ widely in their approach to this problem. Available treatments have been unsatisfactory because of undesired side effects, including frequent relapse, instability of thyroid status, and hypothyroidism. Hashimoto reported an unprecedented low relapse rate associated with a decline in thyroid-stimulating antibodies when patients were treated with thyrroxine and methimazole (T-M). We have had approximately 20 patients under treatment with T-M for up to 2.5 years. The relapse rate is low, and this may be an important benefit. The most significant benefit so far, however, is the stability of the condition while the patient is under treatment with T-M, with decreased fluctuation of thyroid activity, fewer office visits, and decreased frequency of thyroid testing. We believe that T-M has definite advantages, including patient and physician satisfaction and cost-effective control of the disorder, as well as the reported low rate of relapse.

HYPONATREMIA OF HYPOTHYROIDISM: NOT DUE TO SYNDROME OF INAPPROPRIATE ANTIDIURETIC HORMONE SECRETION. Hanna Khoury, MD, and Kaveh Ehsanipoor, MD. Memorial Medical Center, Department of Internal Medicine, Savannah, Ga.

Identification of the mechanism of hyponatremia of hypothyroidism is controversial. Inappropriate antidiuretic hormone (ADH) secretion, "reset osmostat," and reduced salt delivery to the loop of Henle (causing impaired free water clearance) have been implicated.

An 84-year-old woman came to our institution with weakness and slow mentation of 2 days’ duration. Medical history indicated depression, for which she was taking amitriptyline. On physical examination, she was oriented but slow in answering questions. Vital signs were normal. She was clinically euvoletic but had delayed relaxation phase of ankle jerks. Laboratory studies revealed the following values: sodium, 97 mmol/L; blood urea nitrogen, 11 mg/dL; T₄, 1.3 μg/dL; thyroid-stimulating hormone, 21.7 μU/mL; cortisol, 16.1 μg/dL; urinary concentration of sodium, 34 mmol/L; and urinary osmolality, 362 mOsm/kg.

Further studies revealed inappropriate elevation of ADH that resolved when amitriptyline was discontinued, despite the presence of hypothyroidism and hyposmolality. Treatment consisted of fluid restriction, levothyroxine, 25 μg/day, 2 doses of furosemide, and discontinuation of amitriptyline.
This was an extremely severe case of hyponatremia with relatively mild symptomatology, likely because of the gradual development of hyponatremia and probably associated hypothyroidism. It shows that hyponatremia of hypothyroidism is not due to syndrome of inappropriate antidiuretic hormone. In this case, amitriptyline worsened the degree of hyponatremia due to hypothyroidism.

MULTIPLE ENDOCRINE GLAND FAILURE: A RARE COMBINATION. Charles Harris, MD, and Kaveh Ehsanipoor, MD. Memorial Medical Center, Department of Internal Medicine, Savannah, Ga.

Eine biglandulare Erkrankung (a 2-gland illness) was first reported in 1926 by Schmidt. He described the clinical course and autopsy findings of two patients who had died of adrenal insufficiency and were found to have lymphocytic infiltration of the thyroid and adrenal cortex. This polyglandular autoimmune hypofunction was later expanded to include other endocrine glands. Pituitary involvement, shown in the following case, is rare.

A 43-year-old woman was admitted with acute psychosis. Past history included hypothyroidism of unknown cause. She had been treated with levothyroxine, thiothixene, and benztrapine mesylate; compliance was unknown. Pertinent physical findings included mild galactorrhea, absence of axillary hair and scant pubic hair, and delayed relaxation phase of ankle jerks. Laboratory data included the following values: thyroid-stimulating hormone, 12.6 μU/mL; cortisol, 0.9 μg/dL; corticotropin, < 3 pg/mL; prolactin, 173 ng/mL (normal < 33 ng/mL). Testing for antinuclear antibodies yielded positive results, and the cortisol response to corticotropin infusion was subnormal. Positron-emission tomography yielded negative results. After therapy with levothyroxine and hydrocortisone, the prolactin level normalized; the estradiol level was low, with elevated levels of luteinizing hormone and follicle-stimulating hormone. Her condition improved significantly, although her delusional state did not change.

This is an unusual case of Hashimoto's thyroiditis with primary gonadal failure and isolated corticotropin deficiency, most likely autoimmune in origin. Presence of an endocrine autoimmune disease should always alert the physician to the existence of other masked endocrinopathies.

UNUSUAL ORGAN INVOLVEMENT IN IDIOPATHIC HYPEREOSINOPHILIC SYNDROME. Hiem Thong, MD, and Robert J. DiBenedetto, MD. Memorial Medical Center, Department of Internal Medicine, Savannah, Ga.

Hyper eosinophilic syndrome (HES) is a rare disorder characterized by idiopathic proliferation of mature eosinophils; it was first recognized by Stillman in 1912. In 1975, Chusid established diagnostic criteria: eosinophilia greater than 1,500/mm³, lack of known causes, and signs and symptoms of parenchymal organ involve-

EFFECT OF TREATMENT OF HYPOTHYROIDISM ON LIPOPROTEIN(a). Dan Dan, MD, and Kaveh Ehsanipoor, MD. Memorial Medical Center, Department of Internal Medicine, Savannah, Ga.

Hypothyroidism is associated with coronary artery disease and is assumed to result from deteriorated metabolism of the atherogenic lipoproteins, particularly low-density lipoprotein cholesterol and apolipoprotein B-100. Lipoprotein(a) is considered a mediator of atherogenesis. This prospective study was designed to assess the influence of treatment of hypothyroidism on plasma lipoprotein(a).

Nineteen patients with documented primary hypothyroidism participated. Plasma levels of total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and lipoprotein(a) were measured before and after treatment with levothyroxine. No patient was treated with lipid-lowering agents. Euthyroidism was confirmed by detectable thyroid-stimulating hormone levels before the second lipid profile was drawn.

Total cholesterol level decreased significantly from a mean of 259.4 ± 60.6 mg/dL to 207.9 ± 38.7 mg/dL. Mean concentration of low-density lipoprotein cholesterol decreased significantly from 181.8 ± 63.3 mg/dL to 140.5 ± 43.2 mg/dL. Mean concentration of high-density lipoprotein cholesterol decreased significantly from 54.7 ± 11.7 mg/dL to 45.7 ± 9.5 mg/dL. Mean lipoprotein(a) concentration decreased significantly from 34.1 ± 43.0 mg/dL to 29.4 ± 38.4 mg/dL. Triglyceride levels did not change significantly.
The small but significant difference we found suggests that thyroid hormones play a role in the metabolism of lipoprotein(a).

EFFECT OF GLUCOSE CONTROL IN DIABETES MELLITUS ON LIPROPROTEIN(a) LEVELS. Marianne Fleming, MD, Lisa Adams-Mount, MD, and Kaveh Ehsanipoor, MD. Memorial Medical Center, Department of Internal Medicine, Savannah, Ga.

Lipoprotein(a) is a low-density lipoprotein (LDL) particle that has been recognized as a mediator of atherogenesis. Hyperlipidemia has been reported in patients with poorly controlled diabetes. This prospective study measures the effect of glucose control, reflected by hemoglobin A1c, on lipoprotein(a) in patients with type II diabetes who are receiving insulin or oral agent therapy.

Nineteen patients, aged 31 to 78, with type II diabetes and hemoglobin A1c > 8.0% at the baseline completed this study. Eight patients were male, and 11 were female; 10 were white, 8 were black, and 1 was Asian Indian. Fasting serum glucose level, lipid profile, and hemoglobin A1c and lipoprotein(a) levels were obtained at entry and 3 months later, with clinic visits at intervals for glucose monitoring and adjustment of therapy for glycemic control.

Glycemic control improved in all 19 patients. Mean hemoglobin A1c levels dropped from 10.3% at baseline to 6.4% at 3 months. Mean triglyceride levels dropped from 168.1 mg/dL to 122.4 mg/dL at 3 months. No significant change occurred in total cholesterol, LDL-C, HDL-C, triglyceride, or lipoprotein(a) levels.

This longitudinal study shows that excellent glycemic control did not improve lipoprotein(a) levels in these patients with type II diabetes.

FIBRINOGEN AS AN INDICATOR OF INFLAMMATORY DISEASE. Charles A. Baten, MD, and Thomas E. Herchline, MD. Department of Medicine, Keeler Medical Center, Biloxi, Miss.

Positive acute phase proteins are the result of humoral and cellular changes in response to inflammatory stimuli. Inflammatory stimuli are associated with acute infections and tissue insults. One widely available acute phase protein is fibrinogen. Modern coagulation evaluation instruments make quantitative fibrinogen levels rapidly available and inexpensive. Clinical usefulness of this assay is not well documented.

We retrospectively reviewed the admission fibrinogen level, white blood cell (WBC) count, gender, age, and discharge diagnosis of 32 consecutive patients admitted to the surgical service. Inflammatory conditions were defined as documented infections or acute inflammatory conditions.

Results showed that 9 of 32 patients were diagnosed with inflammatory conditions. The mean fibrinogen level for those patients was 503 mg/dL, compared to 319 for the remainder. The mean WBC count was 11,600/mm³ in the group with inflammatory conditions, compared to 8,200/mm³ for the remainder. The sensitivity of detecting an inflammatory condition based on an elevated fibrinogen level, elevated WBC count, or either condition was calculated. Elevated fibrinogen alone showed a sensitivity of 67%, specificity of 87%, positive predictive value of 67%, and negative predictive value of 87%.

Results for the leukocytosis or elevated fibrinogen groups were found to be 56% and 89% for sensitivity, 87% and 74% for specificity, 83% and 94% for negative predictive value, and 63% and 57% for positive predictive value, respectively. Each of these findings was statistically significant.

Further evaluation may prove that fibrinogen level is a valuable addition to established clinical and laboratory clues in the diagnosis of inflammatory disorders. Fibrinogen was a more accurate clinical predictor of inflammatory disease than the WBC count in this study.

CUTANEOUS TUBERCULOSIS CAUSED BY NEEDLE STICK IN A LABORATORY TECHNICIAN. Shariar Arasteh, DO, MS, and Ricardo S. Lemos, MD. Tulane University School of Medicine, Department of Medicine, Infectious Diseases, New Orleans, La.

A laboratory technician accidentally inoculated herself in the finger while transferring a sample of cerebrospinal fluid (CSF) culture fluid from a 13A Bactec bottle to a Nap bottle. The CFS sample had been obtained from a 31-year-old man with a past medical history of seropositivity for human immunodeficiency virus (HIV) who was brought to the emergency department by friends because of changes in mental status and combativeness for 2 weeks. On physical examination, the patient had a temperature of 103°F, no neck stiffness, and no photophobia. Computed tomography of the head revealed no abnormalities except generalized atrophy. CSF was obtained, revealing an opening pressure of 170 mm Hg, a glucose level of 9 mg/dL, a white blood cell count of 103/mm³, and a red blood cell count of 16/mm³; testing for acid-fast bacilli (AFB) and a VDRL test yielded negative results. Cryptococcal antigen testing also yielded negative results. Therapy with antituberculosis medications, as well as ceftriaxone and acyclovir, was begun. In spite of aggressive therapy, the patient died, and autopsy of the brain revealed many AFB. The laboratory technician had no significant past medical history. Within a month, a wartlike lesion developed in the finger; the lesion was removed surgically and tested positive for AFB. A culture from the finger specimen grew Mycobacterium tuberculosis that was sensitive to all antituberculosis drugs. Cultures of the CSF yielded M tuberculosis that was sensitive to all antituberculosis medications as well. She received therapy with antituberculosis medications for 9 months. The technician remained negative for HIV infection 6 months after the accident.
There have been reports of disseminated cutaneous infection due to *M. tuberculosis* in patients with acquired immunodeficiency syndrome. There have also been reports of cases of inoculation with cutaneous mycobacteriosis due to *M. tuberculosis* (e.g., prospector's wart), but the disease did not come from a patient with HIV infection and meningitis caused by *M. tuberculosis* infection.

This case underlines the need for awareness among laboratory technicians of the current epidemics of HIV and *M. tuberculosis* infection and the importance of taking adequate precautions when handling clinical specimens. Newer techniques (such as the use of gene probes) that minimize the risk of needle sticks may decrease the risk of accidents.

**SUBMANDIBULAR TUBERCULOUS LYMPHADENITIS (SCROFULA).** Orlando A. Cuadra, MD, and Kavch Ehsani-poor, MD. Memorial Medical Center, Department of Internal Medicine, Savannah, Ga.

Tuberculosis is on the rise in the United States. Diagnosis of extrapulmonary tuberculosis presents a formidable challenge when pulmonary symptoms are absent. Scrofula has been called "the dangerous masquerader" because it tends to mimic other diseases.

A 31-year-old man, a Korean immigrant, had a 1.5-month history of progressive enlargement of the left submandibular gland without pain or purulent drainage. His father had had cancer of the submandibular gland. Examination was unremarkable except for a 3-cm left submandibular mass and a positive PPD skin test. Results of a chest radiograph were normal.

The patient had exploratory surgery of the left submandibular gland, and the submandibular mass was removed. Frozen section showed granulomatous lymphadenitis with necrosis and abscess. Tissue biopsy culture for acid-fast bacilli isolated *Mycobacterium tuberculosis*. He was then referred to the health department for 6 months' therapy with pyrazinamide and rifampin/isoniazid (Rifamate). His weight increased after surgery. Mycobacteria susceptibility testing indicated no resistance to drugs.

Peripheral tuberculosis lymphadenopathy is the most common form of extrapulmonary tuberculosis; it usually affects the cervical lymph nodes. Tuberculosis outbreaks, particularly among the population infected with human immunodeficiency virus and among recent immigrants, should alert physicians to consider scrofula in the differential diagnosis of neck or submandibular mass. These nodes must be studied histologically and microbiologically.

**A CASE OF ERYTHEMA INDURATUM RESOLVED WITH ANTIMYCOBACTERIAL THERAPY.** R. Demaree Inglese, MD, Barbara T. Sitton, MD, and Robert Middleton, MD. Keesler Medical Center, Keesler AFB, Biloxi, Miss.

Erythema induratum (Bazin's disease) is a chronic benign vasculitis, caused by previous or active infection with *Mycobacterium tuberculosis*, that causes recurrent subcutaneous nodules. We report a case of a 73-year-old woman who came to us with a 10-year history of recurring, painful skin nodules that would occasionally suppurate. The lesions occurred mostly on the lower extremities but also developed on the buttocks, arms, and shoulders. A typical nodule would become indurated and painful over a period of 1 to 2 months and then would suppurate, leaving an atrophic, hyperpigmented scar.

This patient had received no definitive treatment for the 10-year duration of her illness. Multiple serologic testing was unrevealing. She did report exposure to active tuberculosis infection some 25 years previously. However, she gave no history of active pulmonary tuberculosis. Previous chest x-ray films and computed tomography revealed evidence of old granulomatosus disease, as well as transient, recurring, small pulmonary nodules of uncertain cause. PPD testing proved markedly positive, despite persistently negative smears and cultures for acid-fast bacilli. A skin biopsy revealed "necrotizing granulomatous dermatitis and panniculitis" consistent with erythema induratum.

A standard short course of antimycobacterial therapy was prescribed. Within 1 month of treatment, the frequency and duration of nodule occurrence had markedly diminished. By the third month of treatment, the patient had become lesion free for the first time in 10 years. Three months after completion of treatment, she remained free of recurrence. The patient's treatment was fraught with complications due to her other medical problems. A case discussion, including these treatment difficulties, will be presented, along with a review of the literature.

**EPSTEIN-BARR INFECTION MANIFESTING AS ACUTE AUTONOMIC NEUROPATHY.** T. Jackson, MD, and E. Holmboe, MD. Naval Medical Center, Portsmouth, Va.

Infection with Epstein-Barr virus can manifest with atypical features in young adults. We report the rare development of acute autonomic neuropathy in a previously healthy 24-year-old man as the result of Epstein-Barr infection.

The patient came to our hospital after 5 days of fever, nausea, vomiting, and syncpe. The initial diagnosis of gastroenteritis was questioned when aggressive intravenous hydration failed to resolve severe orthostatic hypotension. Despite hydration, a supine systolic pressure of 110 mm Hg fell to 50 mm Hg on standing without any change in heart rate. Near-syncpe developed repeatedly when the patient was upright. Although no objective decrease in strength was elicited, the patient complained of lower extremity weakness. He could not tolerate solid or liquid food, which caused intense nausea. An initial lumbar puncture revealed an elevated protein level without cells. A repeat lumbar puncture done 7 days later showed a tripling of the cerebrospinal fluid protein and the presence of lymphocytes. An upper gastrointestinal
series showed slow transit of the barium meal. Lower extremity nerve conduction studies showed normal conduction velocity with small reductions in amplitude. An Epstein-Barr serum titer was positive at only a 1:10 dilution.

Acute autonomic neuropathy was the presumptive diagnosis. Additional intensive investigation failed to reveal the cause for the neuropathy. He was discharged after 18 days of hospitalization. Persistent orthostatic hypotension and weight loss resulted in a second hospital admission 6 weeks later. A repeat Epstein-Barr titer was now elevated at 1:2500, indicating recent infection. This patient is only the second reported in the literature with acute autonomic neuropathy as the primary manifestation of Epstein-Barr infection.

PULMONARY-RENAL SYNDROME DUE TO MEMBRANOPROLIFERATIVE GLOMERULONEPHRITIS, TYPE I. Aubrey W. Ziegler, MD, Geoffrey H. Golembiewski, MD, and Joseph D. O'Gorman, DO. Keesler Medical Center, Department of Medicine, Keesler AFB, Biloxi, Miss.

We describe the case of a 29-year-old white man who was in excellent health until fever, dyspnea, and non-productive cough developed. Oral erythromycin failed to improve his symptoms, and subsequent lower extremity edema and scant hemoptysis prompted hospital admission. Initial physical examination revealed mild hypertension (150/90 mm Hg), absence of peribronchial exudate, and bibasilar crackles; there was no murmur or cardiac gallop. The extremities showed pitting edema bilaterally without rash. Significant laboratory data included the following values: blood urea nitrogen, 62 mg/dL; creatinine, 2.6 mg/dL; and hematocrit, 30.6% with a platelet count of 173,000/mm³. Titters for antistreptolysin-O and antinuclear antibodies/double-stranded DNA were negative. There was profound hypocomplementemia (C3/C4 = 37/<8). Additional negative/norma studies included blood and urine cultures, hepatitis panel, human immunodeficiency virus, cryoglobulins, antineutrophil cytoplasmic antibodies (ANCA), and anti-GBM Ab. Urinalysis revealed 4+ protein and blood, multiple red blood cell (RBC) casts, dysmorphic RBCs and 4.1 g protein/day. Admission chest film showed mild cardiomegaly and interstitial edema. While we were awaiting results of renal biopsy, substantial hemoptysis, hypoxia, and smoky urine developed. Ventilation/perfusion scan and duplex ultrasonography showed no pleural effusion or deep vein thrombosis. A renal biopsy specimen showed diffuse global hypercellularity without significant necrosis, diffuse global granular capillary wall and mesangial staining for multiple immunoreactants, and mesangial interposition with numerous mesangial and subendothelial electron-dense deposits consistent with membranoproliferative glomerulonephritis (MPGN), type I. While awaiting results of renal biopsy and anti-GBM Ab/ANCA serologic testing, empiric therapy with intravenous cyclophospha-

mide/pulse methylprednisolone and plasmapheresis was instituted. Continued hemoptysis prompted open lung biopsy, which showed extensive acute alveolar hemorrhage with negative immunostaining and negative bacterial/fungal/acid-fast bacilli cultures. With long-term prednisone therapy alone, the patient's glomerular filtration rate remains normal (creatinine level 1.2 mg/dL), although hypocomplementemia and microhematuria persist. A literature review showed only two (pre-ANCA) cases of pulmonary hemorrhage and MPGN, which should be included in the differential diagnosis of pulmonary-renal syndromes.

IDIOPATHIC RETROPERITONEAL FIBROSIS MANIFESTING AS RENAL FAILURE: A CASE PRESENTATION AND REVIEW OF THE LITERATURE. Marcus Cranston, MD, and John Eyre, MD. Keesler Medical Center, Keesler AFB, Biloxi, Miss.

Idiopathic retroperitoneal fibrosis is an uncommon disease that rarely manifests with renal failure. First described in 1948, its cause remains obscure.

This is a case of a 45-year-old black woman with a history of hypertension for 1 year and low back pain for 6 months who came to our emergency department with complaints of nausea and emesis for the previous 2 weeks. Her past history was unremarkable for any secondary causes of retroperitoneal fibrosis (ie, drug exposure, tumor, radiation, vascular disease, or trauma). On initial evaluation, she had a blood pressure of 206/118 mm Hg; results of the physical examination were unremarkable. Laboratory tests revealed a blood urea nitrogen level of 108 mg/dL and a creatinine level of 25.8 mg/dL. Renal ultrasonography showed bilateral hydronephrosis. Cystoscopy was done to place bilateral ureteral stents. After osmotic diuresis, the creatinine level dropped to 3.4 mg/dL, and abdominal computed tomography revealed a retroperitoneal mass, most consistent with fibrosis. Results of retroperitoneal biopsy were also consistent with this diagnosis.

This case is particularly interesting because it displays many of the classic historical findings associated with idiopathic retroperitoneal fibrosis. It also shows a rare cause of postobstructive renal failure. After presentation of the case report, we will discuss our review of the literature, including controversies in diagnostic imaging and treatment.

AUTOSOMAL DOMINANT POLYCYSTIC KIDNEY DISEASE COMPLICATED BY FOCAL SEGMENTAL GLOMERULOSCLEROSIS. Capt Samuel P. Gore, MD, Capt Jorge J. Scheirer, MD, and Maj Geoffrey Golembiewski, MD. 81st Medical Group, Keesler AFB, Biloxi, Miss.

A 54-year-old man with a history of autosomal dominant polycystic kidney disease (ADPKD) since 1978 was found to have 5,200 mg of protein in a 24-hour urine
collection and an increase in his serum creatinine level from 1.7 mg/dL to 2.4 mg/dL. Physical examination was remarkable for a blood pressure of 128/80 mm Hg, palpable left kidney, and bilateral lower extremity pitting edema. Antinuclear antibody testing yielded negative results, and serum protein electrophoresis complement were normal. Repeat 24-hour urine collection revealed 2,524 mg of protein and the serum creatinine level rose to 3.0 mg/dL. A specimen obtained by open renal biopsy revealed focal segmental glomerulosclerosis. Immunofluorescence stains were positive only for mesangial deposition of IgM and C3. Electron microscopy revealed foot process effacement and mild focal increase in mesangium. Prednisone, 60 mg every other day, was initially administered. Further renal decline necessitated the addition of cyclophosphamide (Cytoxan), 100 mg every day, resulting in a stabilization in the serum creatinine level and urinary protein excretion.

The cases of nine patients with ADPKD and nephrotic syndrome that were further evaluated by renal biopsy have been reported. All patients were found to have glomerulonephritis and usually had an accelerated decline in renal function that led to dialysis. We will discuss the salient features of the reported cases and the implications for management of nephrotic syndrome in patients with ADPKD.

COMBINED USE OF H-2 ANTAGONISTS AND SUCRALFATE: A RETROSPECTIVE STUDY. Capt Daryl M. McGlendon (ACP Associate), and Maj Michael H. Weiss (ACP Member), Department of Internal Medicine and Gastroenterology Service, 81st Medical Group, Keesler AFB, Biloxi, Miss.

H-2 antagonists are the single leading cost item of the pharmacy at Keesler Medical Center. The total expenditure for all H-2 antagonists and sucralfate for a 6-month period from January 1 through June 30 1993 was $569,704. Pharmacy records indicate that 213 patients concurrently filled prescriptions for both H-2 antagonists and sucralfate during the same period. One hundred twenty-three (58%) patient charts were available for review, and 92 (75%) of these charts had adequate information regarding indication for treatment, evaluation, duration, and outcome.

Historical review revealed that 84% of the patients were taking both sucralfate and ranitidine, and 16% were taking both sucralfate and cimetidine. The average age of these patients was 59 years; 32% were male and 68% were female. The most common clinical indication for treatment was gastroesophageal reflux (40%) followed by dyspepsia (37%). Other clinical indications included presumed peptic ulcer disease (PUD) (7), gastric ulcers (5), history of PUD (4), nonsteroidal anti-inflammatory drug prophylaxis (2), gastrointestinal bleeding (1), atypical chest pain (1), and irritable bowel syndrome (1). Sixty-two percent of the patients had no diagnostic evaluation during the course of their treatment, 12% had esophagogastroduodenoscopy (EGD), and 26% had upper gastrointestinal series (UGIS). The most common clinical diagnosis for patients who had an EGD was gastroesophageal reflux disease (GERD), and esophagitis was the most frequent finding in all patients who had this procedure. Of patients who had UGIS, the most common clinical diagnosis was GERD, but the most common finding was a normal study. Sixty percent of all patients whose cases were reviewed had subjective improvement in their condition with combined therapy; in 19%, the condition was unchanged, and 22% had no documented follow-up. Of the patients who were clinically diagnosed with GERD, 77% had subjective improvement, compared with 47% of those diagnosed with dyspepsia.

Long-term concurrent use of both H-2 antagonists and sucralfate is costly and is of questionable efficacy, especially for patients with dyspepsia. In patients with GERD, omeprazole may be a more efficacious and cost-effective alternative. Long-term studies are needed to evaluate duration of treatment and maintenance therapy.

ARE THERE MORE PSYCHOLOGIC SYMPTOMS AND CHRONIC ILLNESSES ASSOCIATED WITH ELDERLY PATIENTS SUFFERING FROM INSOMNIA THAN WITH THOSE WHO DO NOT? Francis Amede, MD, Jerry O. Ciocon, MD, Cleveland Clinic Florida, Ft. Lauderdale, Fl; and Daisy G. Ciocon, PhD, RN, GRECC, University of Miami, Miami, Fl.

Elderly patients usually have multiple chronic illnesses, and insomnia is a common complaint. The purpose of this study was to determine whether elderly people with insomnia have more psychologic symptoms and chronic illnesses than those who do not have insomnia. Twenty-five patients who were 65 years or older without end-stage dementia or terminal illness and suffering from insomnia were compared with 25 patients who did not have insomnia. Twenty-five patients who had insomnia defined as a sleep latency > 30 minutes, more than two awakenings, and staying awake for 30 minutes or more were interviewed. The charts were reviewed to determine demographics, diagnoses, and medications. All patients were seen at the Cleveland Clinic Florida. Twenty-five consecutive patients without insomnia were similarly interviewed, and their charts were reviewed. Thereafter, all patients were asked to complete questionnaires on Folstein mini-mental status (0 to 30), Yesavage geriatric depression scale (0 to 15), Sheehan patient-rated anxiety scale (0 to 140), symptom profile with SP-36 questionnaire (0 to 100), fatigue score (0 to 27), and quality of sleep using the Pittsburgh sleep quality index scale (0 to 21). The scores were recorded and compared using paired t test and a P < .05 considered statistically significant. Twenty-five patients (aged 76 ± 4 years; 10 men, 15 women) with insomnia and 25 patients (aged 77 ± 3 years; 13 men, 12 women) without insomnia were compared, and results were summarized. Anxiety and depression were the most common problems identified
in the elderly with insomnia. The number of chronic illnesses and the number of medications were the same in both groups. The Pittsburgh sleep quality index scale was normal in those without insomnia. However, the symptom profile was the same in both groups. Fatigue was a consequence of insomnia. Elderly patients with insomnia commonly are depressed and anxious and frequently complain of fatigue. Their chronic illness, medications, and generalized symptoms are no different from those of individuals without insomnia.

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NEW ONSET OF SEXUAL DYSFUNCTION IN HIV-SEROPOSITIVE WOMEN: RESULTS OF A PROSPECTIVE STUDY. George R. Brown, MD, Mountain Home VAMC, Johnson City, Tenn; and Rebecca Ledsky, MBA, Klemm Analysis Group, Washington, DC.

During a 5.5-year biopsychosocial study, we prospectively assessed the psychiatric status and sexual functioning of women during the natural history of human immunodeficiency virus (HIV) infection detected as part of an HIV screening program in the military. Participants were serving on active duty or were spouses of servicemen who have tested HIV-positive since 1986. Patients were psychiatrically evaluated every 6 to 18 months for four assessments (T1-T4). Initial evaluation (T1) was completed by 54 HIV-positive women without acquired immunodeficiency syndrome (AIDS) (average length of knowledge of seroconversion, 8.9 months). Thirty-eight had re-evaluation at T2 (average knowledge, 26.5 months), 28 at T3 (average knowledge, 52.3 months), and 18 at T4 (> 5 years’ knowledge). The most prevalent DSM-III-R psychiatric diagnosis at each evaluation, based on criteria published by Brown et al, was hypoaffective sexual desire disorder (HSDD) (Psychosomatic Medicine 1992; 54:588-601). At T1, 21% met threshold criteria for this diagnosis; an additional 25% had more than a 33% decrease in desire. At T2, 50% had HSDD (the disorder was of new onset in 71%). HSDD was persistently present in 50% at T3 and 50% at T4. A total of 64% (N = 18) of those seen at three visits were diagnosed with HSDD on at least one visit. Only two women recovered to baseline levels of desire during the time of the study. By the fourth evaluation, 25% of the women seen three times had been pregnant at least once. Twenty-nine percent reported that their male partners actively discouraged condom use.

We concluded that there is persistent, chronic impairment in sexual functioning in HIV-positive women, independent of HIV-related medical symptoms, as reflected in high rates of new onset of sexual desire phase disruptions. Potential causes are discussed, including potential biologic, interpersonal, and psychologic contributors.

PHARMACOLOGIC CHARACTERISTICS OF THE IDEAL ANTIDEPRESSANT. Elliott Richelson, MD. Department of Research, Mayo Clinic Jacksonville, Jacksonville, Fla.

The purpose of this study was to define many pharmacologic effects of antidepressants from their in vitro synaptic pharmacology and, from this information and from a review of the literature, to define the pharmacologic characteristics of the ideal antidepressant. The experimental methods used were blockade by antidepressants of [3H]norepinephrine, [3H]5-hydroxytryptamine (5-HT), and [3H]dopamine transport into rat brain synaptosomes prepared from hippocampus, frontal cortex, and striatum, respectively; and radioligand binding assays to determine equilibrium dissociation constants for antidepressants at human histamine H1, muscarinic, α1-adrenergic, and dopamine D2 receptors. The animal studies were approved by our IACUC, and normal human brain tissue was obtained at autopsy with our institutional review board’s approval. The results showed that newer second-generation antidepressants were more potent and selective for blocking uptake of serotonin than uptake of norepinephrine. Rank order of potency at blocking uptake of norepinephrine for the newer drugs was as follows: paroxetine > venlafaxine > sertraline > fluoxetine > bupropion. For these drugs, rank order of potency at blocking uptake of serotonin was as follows: paroxetine > sertraline > fluoxetine > venlafaxine > bupropion. Serotonin selectivity for the most potent 5-HT uptake inhibitors was as follows: sertraline > paroxetine > fluoxetine > venlafaxine. Venlafaxine was without activity at all four receptors. However, from a practical standpoint, bupropion, fluoxetine, and sertraline would not likely affect any of these receptors directly in vivo.

In general, the newer compounds were more selective for uptake blockade of serotonin and were weak antagonists at all four receptor sites. These synaptic effects of newer antidepressants predict a side effect profile for these compounds different from that of the older antidepressants.