Communications continued from page 776

photon absorption densitometry) in affected diabetics has been shown to exist[2]; however, a number of investigators have found no role of osteopenia in diabetic fracture production.[2-4] Heath et al[2] compared with age matched controls the incidence of skeletal fractures in 1,000 diabetics. Although they did not identify those with peripheral neuropathy, their data did not support a role for osteopenia in skeletal fracture production. The only bone at increased risk for fracture in diabetics was found to be the medial malleolus.[2]

Sinha et al[3] studied 101 patients with diabetic neuropathic joints. The most common sites of involvement were the tarsal joints (47 percent), tarsal-metatarsal joints (34 percent), and ankle joints (11 percent).[3] Bilateral disease was present in 24 percent. The most frequent presenting complaints were bony deformity, ulceration, and soft tissue swelling. The most prominent signs of arthropathy were bony deformity, callus formation, ulceration, soft tissue swelling, and limp. All patients demonstrated anesthesia or hypoesthesia of the feet. The most common radiographic sign was disruption of articular surfaces as evidenced by irregular, narrowed, or obliterated joint spaces. Other radiographic signs included fragmentation, periosteal new bone formation, dislocation, vascular calcification, and bone resorption.[3]

Treatment of neuropathic joint disease includes immobilization and nonweight bearing of the extremity.[3,4,7] Continued trauma is a prerequisite for progression of joint destruction; thus, if repeated trauma is prevented, the joints will usually heal. Duration of protection of the joint must be based on clinical and radiological response. Pain is a poor guide of response, since these patients have diminished pain sensation. Premature resumption of activity may lead to further joint destruction.

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Pasteurella Multocida Meningitis in an Infant Following Occipital Dog Bite

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It has been estimated that approximately one million people are victims of dog bites each year in the United States, and children are bitten more frequently than adults.[1] Children under four years of age are at an increased risk of being bitten on the head, neck, or face.[2] This report describes the case of a ten-month-old boy who developed Pasteurella multocida meningitis 48 hours after being bitten in the occiput by the family dog.

Case Report

A ten-month-old white boy was admitted to the pediatric unit of Riverside Hospital, Toledo, Ohio, with the diagnosis of bacterial meningitis.

Forty-eight hours prior to admission the infant was attacked by the family's German shepherd dog and sustained three lacerations of the scalp: a 3-cm laceration over the left mastoid, a 3-cm laceration

Communications continued on page 780

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KLOTRIX®
(POTASSIUM CHLORIDE) SLOW-RELEASE TABLETS, 10 mEq

DESCRIPTION KLOTRIX is a film-coated (not enteric-coated) tablet containing 750 mg potassium chloride (equivalent to 10 mEq) in a wax matrix. This formulation is intended to provide a controlled release of potassium from the matrix to minimize the likelihood of producing high localized concentrations of potassium within the gastrointestinal tract.

INDICATIONS—BECAUSE OF REPORTS OF INTESTINAL AND GASTRIC ULCERATION AND BLEEDING WITH SLOW-RELEASE POTASSIUM CHLORIDE PREPARATIONS, THESE DRUGS SHOULD BE RESERVED FOR THOSE PATIENTS WHO CANNOT TOLERATE OR REFUSE TO TAKE LIQUID OR EFFERVESCENT POTASSIUM PREPARATIONS OR FOR PATIENTS IN WHOM THERE IS A PROBLEM OF COMPLIANCE WITH THESE PREPARATIONS.

1. For therapeutic use in patients with hypokalemia with or without metabolic alkalosis; in digitalis intoxication and in patients with hypokalemic familial periodic paralysis.
2. For prevention of potassium depletion when the dietary intake of potassium is inadequate in the following conditions: Patients receiving digitalis and diuretics for congestive heart failure; hepatic cirrhosis with ascites; states of aldosterone excess with normal renal function; potassium-losing nephropathy, and certain diarrheal states.
3. The use of potassium salts in patients receiving diuretics for uncomplicated essential hypertension is often unnecessary when such patients have a normal dietary pattern. Serum potassium should be checked periodically, however, and, if hypokalemia occurs, dietary supplementation with potassium-containing foods may be adequate to control milder cases. In more severe cases supplementation with potassium salts may be indicated.

CONTRAINDICATIONS In patients with hyperkalemia, since a further increase in serum potassium concentration in such patients can produce cardiac arrest. Hyperkalemia may complicate any of the following conditions: chronic renal failure, systemic acidosis such as diabetic acidosis, acute dehydration, extensive tissue breakdown as in severe burns, adrenal insufficiency, or the administration of a potassium-sparing diuretic (*eg*, spironolactone, triamterene).

Wax-matrix potassium chloride preparations have produced esophageal ulceration in certain cardiac patients with esophageal compression due to enlarged left atrium.

All solid dosage forms of potassium supplements are contraindicated in any patient in whom there is cause for arrest or delay in tablet passage through the G.I. tract. In these instances, potassium supplementation should be with a liquid preparation.

WARNINGS Hyperkalemia: In patients with impaired mechanisms for excreting potassium, administration of potassium salts can produce hyperkalemia and cardiac arrest. This occurs most commonly in patients given potassium intravenously but may also occur when given orally. Potentially fatal hyperkalemia can develop rapidly and be asymptomatic. Use of potassium salts in patients with chronic renal disease, or any other condition which impairs potassium excretion requires particularly careful monitoring of the serum potassium concentration and appropriate dosage adjustment.

Interaction with potassium-sparing diuretics: Hypokalemia should not be treated by the concomitant administration of potassium salts and a potassium-sparing diuretic (*eg*, spironolactone or triamterene), since the simultaneous administration of these agents can produce severe hyperkalemia.

Gastrointestinal lesions: Potassium chloride tablets have produced stenotic and/or ulcerative lesions of the small bowel and deaths. These lesions are caused by a high localized concentration of potassium ion in the region of a rapidly dissolving tablet, which injures the bowel wall and thereby produces obstruction, hemorrhage, or perforation. KLOTRIX is a wax-matrix tablet formulated to provide a controlled rate of release of potassium chloride and thus to minimize the possibility of a high local concentration of potassium ion near the bowel wall. While the reported frequency of small-bowel lesions is much less with wax-matrix tablets (less than one per 100,000 patient-years) than with enteric-coated potassium chloride tablets (40-50 per 100,000 patient-years) cases associated with wax-matrix tablets have been reported both in foreign countries and in the United States. In addition, perhaps because the wax-matrix preparations are not enteric-coated and release potassium in the stomach, there have been reports of upper gastrointestinal bleeding associated with these products. The total number of gastrointestinal lesions remains less than one per 100,000 patient-years. KLOTRIX should be discontinued immediately and the possibility of bowel obstruction or perforation considered if severe vomiting, abdominal pain, distention, or gastrointestinal bleeding occurs.

Metabolic acidosis: Hypokalemia in patients with metabolic *acidosis* should be treated with an alkalinizing potassium salt such as potassium bicarbonate, potassium citrate, or potassium acetate.

PRECAUTIONS Potassium depletion is ordinarily diagnosed by demonstrating hypokalemia in a patient with a clinical history suggesting some cause for potassium depletion. In interpreting the serum potassium level, the physician should bear in mind that acute alkalosis *per se* can produce hypokalemia in the absence of a deficit in total body potassium, while acute acidosis *per se* can increase the serum potassium concentration into the normal range even in the presence of a reduced total body potassium. Treatment of potassium depletion particularly in presence of cardiac disease, renal disease, or acidosis, requires careful attention to acid-base balance and appropriate monitoring of serum electrolytes, electrocardiogram and clinical status of patient.

ADVERSE REACTIONS Most common to oral potassium salts: nausea, vomiting, abdominal discomfort, and diarrhea. These symptoms are due to irritation of the gastrointestinal tract and are best managed by diluting the preparation further, taking the dose with meals, or reducing the dose. One of the most severe adverse effects is hyperkalemia (see Contraindications and Warnings). There also have been reports of upper and lower gastrointestinal conditions including obstruction, bleeding, ulceration and perforation (see Contraindications and Warnings); other factors known to be associated with such conditions were present in many of these patients. Skin rash has been reported rarely.

DOSAGE AND ADMINISTRATION The usual dietary intake of potassium by the average adult is 40 to 80 mEq per day. Potassium depletion sufficient to cause hypokalemia usually requires the loss of 200 or more mEq of potassium from the total body store. Dosage must be adjusted to the individual needs of each patient but is typically in the range of 20 mEq per day for the prevention of hypokalemia to 40-100 mEq per day or more for the treatment of potassium depletion.

Note: KLOTRIX® slow-release tablets must be swallowed whole and never crushed or chewed. Following release of the potassium chloride, the expended wax matrix, which is not absorbed, may be observed in the stool.

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Communications continued from page 778

over the right occiput, and a 1-cm laceration superior to the left mastoid wound. At that time the infant was taken to an emergency room, where the lacerations were cleansed with a povidone-iodine scrub (Betadine) and sutured with nylon. He was discharged on 125 mg of cephalexin (Keflex) orally every six hours. Skull x-ray films obtained at that time were negative for perforation or fracture.

Twenty-four hours later the infant developed a temperature of 39.4 °C and was reexamined in the same emergency room. Acetaminophen (Tylenol), 120 mg, was prescribed every four to six hours orally for fever, and the cephalexin was discontinued. The following day the infant was seen in the Riverside Family Practice Center and immediately admitted to Riverside Hospital.

On admission the child appeared lethargic and irritable. The rectal temperature was 37.6 °C. The pulse was 140 beats per minute, respirations were 56 per minute, and blood pressure was 108/80 mmHg in the right arm. Head circumference was 45.5 cm (25th percentile), height was 70 cm (10th percentile), and weight was 9.1 kg (25th percentile). The anterior fontanelle was tense. The sutured lacerations of the posterior scalp did not appear inflamed; however, a small hematoma was noted over the left occiput. The tympanic membranes were not inflamed, and the oropharynx was normal. The neck was supple, but Kernig's sign was present. The chest was clear and the cardiovascular examination was normal. Abdominal examination was unremarkable. The neurological examination revealed no focal neurological deficit.

A complete blood count revealed 21,400 white blood cells with 19 percent band forms, 64 percent segmented neutrophils, 13 percent lymphocytes, and 4 percent monocytes. Serum sodium was 136 mEq/L, serum potassium was 3.4 mEq/L, and chloride was 96 mEq/L. A lumbar puncture produced a markedly cloudy cerebrospinal fluid with 3,270/mm^3 white blood cells of which 95 percent were polymorphonuclear leukocytes and 4 percent were mononuclear leukocytes. Cerebrospinal fluid glucose was 20 mg/100 mL and the spinal fluid protein was 171 mg/100 mL. Gram stain of the cerebrospinal fluid revealed many polymorphonuclear leukocytes, but no organisms were seen. Counterimmunoelectrophoresis (CIE) was negative for group B streptococcus, Hemophilus influenzae type B, pneumococcus polyvalent, and meningococcus

polyvalent A-D. The culture of the cerebrospinal fluid grew Pasteurella multocida. Blood cultures were sterile.

Immediately after the lumbar puncture, antimicrobial therapy was begun with ampicillin, 400 mg/kg/day, and chloramphenicol, 100 mg/kg/day intravenously. Following 48 hours of combined therapy, the organism was reported to be very sensitive to ampicillin in vitro. Chloramphenicol was discontinued.

Repeat lumbar puncture 48 hours after initiation of therapy revealed a mildly cloudy cerebrospinal fluid with a white cell count of 2,750/mm^3 (85 percent polymorphonuclear leukocytes and 13 percent mononuclear leukocytes). Gram stain was negative and the cerbrospinal fluid cultures were sterile. Cerebrospinal fluid glucose was 45 mg/100 mL, and protein was 18 mg/100 mL. Simultaneous blood glucose was 78 mg/100 mL. Repeat skull x-ray examination, including tangential views of the occiput, were negative for fractures or depressions.

Clinically the child became less lethargic and less irritable during the first three days of hospitalization, although shortly after admission the temperature rose to 39.0 °C rectally. The child was afebrile 48 hours after admission. By the fifth hospital day the child was not irritable and had appropriate social behavior for ten months of age.

A lumbar puncture performed on the tenth day of hospitalization revealed clear spinal fluid with 13/mm^3 white blood cells. The cerebrospinal fluid glucose was 50 mg/100 mL with a simultaneous blood glucose of 80 mg/100 mL. Gram stain was again negative, and the cultures remained sterile. The child was discharged on the 11th hospital day.

Office follow-up at two weeks after discharge revealed no recurrence of symptoms.

Discussion

For over a century Pasteurella multocida has been recognized as a significant veterinary pathogen that is infrequently encountered in human infectious disease.[3] Identification of the bacterium is relatively uncomplicated. It is a nonmotile, gram-negative coccobacillus that does not grow on MacConkey's agar but grows well on nutrient agar under increased carbon dioxide tension. It exhibits a positive catalase test and is inhibited by penicillin in vitro.[3]

Since Pasteurella multocida is part of the normal respiratory flora of cats and dogs, most infections with this organism are a sequela of domestic animal bites.[4-6] A few human Pasteurella multocida infections have been reported with nonbite animal exposure or no known animal exposure.[7,8] The most frequent type of Pasteurella multocida infection is a cellulitis in the area of the animal bite.[4] Complications may occur from contiguous spread or bacteremia. Complications include peritonitis,[9] pneumonia,[10] empyema,[10] septic arthritis,[11] and osteomyelitis.[12]

As of 1980 only four cases of Pasteurella multocida meningitis had been reported from the United Kingdom.[13] In the United States 19 cases of Pasteurella multocida meningitis had been reported through 1980.[14-19]

The treatment of choice for infections due to Pasteurella multocida is penicillin G.[4] The organism is also very sensitive to other penicillin analogues, but it is usually not sensitive to erythromycin.[4] An appropriate treatment regimen for meningitis has not been established because of the small number of cases reported, but this experience suggests that 400 mg/kg solidus day of ampicillin, given intravenously for ten days, is efficacious.

Although Pasteurella multocida is an infrequently reported cause of human disease, injury from dog bites is commonly seen in the younger child. Physicians should provide anticipatory guidance to the family with a young child who may have a dog. Physicians should also consider Pasteurella multocida when treating a child who develops cellulitis, septicemia, or meningitis after a cat or dog bite.

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An Approach to Relearning the Pelvic Examination

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For 14 years the University of Tennessee College of Medicine has sponsored a week-long review course for the family physician. Originally, the course consisted of a series of didactic lectures given by University of Tennessee faculty members in a theatre-style auditorium. Since 1977 in response to participant requests, multiple small group sessions are offered in the afternoon segment of the program. The participants choose those groups most relevant to their own day-to-day office practices.

The Patient Instructor Program

As plans progressed for the 1981 course, word of a successful teaching concept used in the Department of Obstetrics and Gynecology reached the course director. This department teaches pelvic examination techniques to medical students with lay women as "patient instructors."[1,2]

The patient instructors are trained by faculty members of the Department of Obstetrics and Gynecology faculty members to teach students in both procedural and interpersonal aspects of the examination. Patient instructors work in teams of two with small groups of students.[3] One patient instructor demonstrates a pelvic examination on her partner; then, each student repeats the examination. The patient instructor in the patient role does most of the teaching for the bimanual component of the examination while her partner does most of the teaching for the part of the examination concerned with inspection of the genitalia and speculum insertion. Both women comment throughout on the student's interpersonal approach. The program is described in detail by Wheeler et al and Hale and Schiner.[4,5]

Student evaluation of the experience overwhelmingly indicated that the time spent with the patient instructors was valuable.[6-8] Consequently, the program was expanded to include obstetrics and gynecology residents. Most were pleased with

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