collateral vessels. Pathologic changes in the bowel that are associated with mesenteric venous thrombosis — especially bowel dilatation, bowel-wall thickening, reduced motility, and intestinal pneumatosis — can be assessed as readily by ultrasonography as by CT.

After the diagnosis of mesenteric venous thrombosis, frequent monitoring should be conducted by ultrasonography in order to avoid radiation exposure or contrast-induced damage.[1]

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To the Editor: In their review of mesenteric venous thrombosis, Kumar et al. could have added that the results of a D-dimer assay can help establish the diagnosis.

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The authors reply:

To the Editor: Thrombolytic therapy may be beneficial in patients with localized thrombus, especially if there is worsening bowel ischemia without obvious necrosis. Strategies include the systemic infusion of thrombolytic agents,[1] percutaneous transhepatic or transjugular infusion of thrombolytic agents directly into the thrombus,[2] the infusion of such agents into the superior mesenteric artery,[3] and mechanical thrombectomy.[4] However, reports of success should be interpreted with caution. Many of the reports have involved patients with thrombosis in the portal vein or in the superior mesenteric vein at its junction with the portal vein. In our experience, these patients are not likely to require surgical intervention, and most of them can be treated with anticoagulation and supportive measures alone. Many of the reports cited by the correspondents involve patients who did not require surgery and who may have done well with anticoagulation alone. Although a transjugular intrahepatic shunt has the potential advantage of allowing easy access to the mesenteric venous circulation, a high rate of stenosis and the permanence of the procedure argue against its use. Current consensus recommendations for the use of transjugular intrahepatic shunts do not include mesenteric venous thrombosis as an indication.[5]

We agree with Stadler et al. that transabdominal color Doppler ultrasonography may allow diagnosis of mesenteric venous thrombosis. However, ultrasonography is very much an operator-dependent technique, and the results are less reproducible than those of CT. Patients with acute mesenteric venous thrombosis often have ileus, gaseous distention, and abdominal tenderness, all of which preclude an adequate examination. CT should be the diagnostic test of choice for several reasons. In addition to allowing diagnosis of mesenteric venous thrombosis and visualization of the thrombus, CT permits complete examination of the structure of the bowel, the detection of potential complications such as bowel ischemia and perforation, and the identification of potential precipitating causes of the thrombosis.

In response to Dr. Cohen: an increase in the D-dimer level is not specific enough to diagnose mesenteric venous thrombosis.

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Severe Hyperosmolar Metabolic Acidosis Due to a Large Dose of Intravenous Lorazepam

To the Editor: In a 34-year-old woman with a history of alcohol abuse, cardiomyopathy, and AIDS, acute respiratory failure developed as a result of *Escherichia coli* sepsis and pneumonia. Initial laboratory studies revealed a pH of 7.2, a bicarbonate level of 8.8 mmol per liter, a partial pressure of carbon dioxide of 26 mm Hg, an anion gap of 15.4 mmol per liter, a lactate level of 6.9 mmol per liter, and a creatinine level of 1.8 mg per deciliter, with a normal blood sugar level and no ketones.

After four days of treatment, the patient's acidosis was worse (pH, 6.9; bicarbonate level, 6.2 mmol per liter; anion gap, 17.8 mmol per liter), despite a slow improvement in the lactate level (to 4.5 mmol per liter). The serum osmolality was 462 mOsm per liter with an osmolal gap of 165 mOsm per liter. Standard blood tests for methanol, ethylene glycol, and isopropyl alcohol were negative. It was noted that the woman was being sedated with increasing doses of intravenous lorazepam (up to 30 mg per hour), which contained 0.18 ml of polyethylene glycol–400 (PEG-400) in propylene glycol with 2 percent benzyl alcohol per 2-mg vial. She had received 1696 mg of lorazepam in 78 hours, for a cumulative PEG-400 dose of 153 ml. Despite treatment with fomepizole, a polyethylene glycol an-

tagonist that is administered intravenously, she required hemodialysis. After three hemodialysis sessions, the patient's pH and bicarbonate level had improved (7.37 and 26 mmol per liter, respectively), and her osmolal gap had decreased to 40 mOsm per liter. She was discharged after 26 days in the hospital. Her renal function was stable; the creatinine level was 1.7 mg per deciliter. It should be noted that our laboratory was unable to quantify the levels of PEG-400, its metabolites, or benzyl alcohol.

Polyethylene glycol is oxidized by alcohol dehydrogenase to hydroxy acid and diacid metabolites, possibly contributing to the development of metabolic acidosis.[1] In previous reports polyethylene glycol has been implicated in hyperosmolar states,[2,3] acute renal failure,[3-5] and mild metabolic acidosis[3,4] due to the topical or intravenous use of various drugs, including lorazepam.[5] Although our patient had preexisting metabolic anion-gap acidosis, we believe that the large dose of intravenous lorazepam that she received contributed to the development of severe, hyperosmolar metabolic acidosis.

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The Popliteal-Artery Entrapment Syndrome in a Patient Using Anabolic Steroids

To the Editor: The popliteal-artery entrapment syndrome is a potentially serious but rare cause of ischemia of the legs.[1] It occurs predominantly in young persons and is due to an abnormal anatomical relation between the popliteal artery and the tendinous insertion of the gastrocnemius muscle. Usually, symptoms arise when there is occlusion of the functional artery during contraction of the calf muscle; arterial thrombosis is a rare cause.[2] Abuse of anabolic steroids has increased in frequency during the past decade and is associated with a documented risk of acute coronary-artery and peripheral-artery thrombosis.[3-5] We describe the occurrence of thrombotic occlusion of the popliteal artery in an athlete with the popliteal-artery entrapment syndrome who abused anabolic steroids.

A 31-year-old male bodybuilder was referred to our emergency department with a three-day history of claudication and paresthesias of the left foot. Clinical examination revealed symmetric muscular hypertrophy of the legs, with discoloration of the left foot and decreased skin temperature below the left knee. The left popliteal, tibialis, and dorsalis

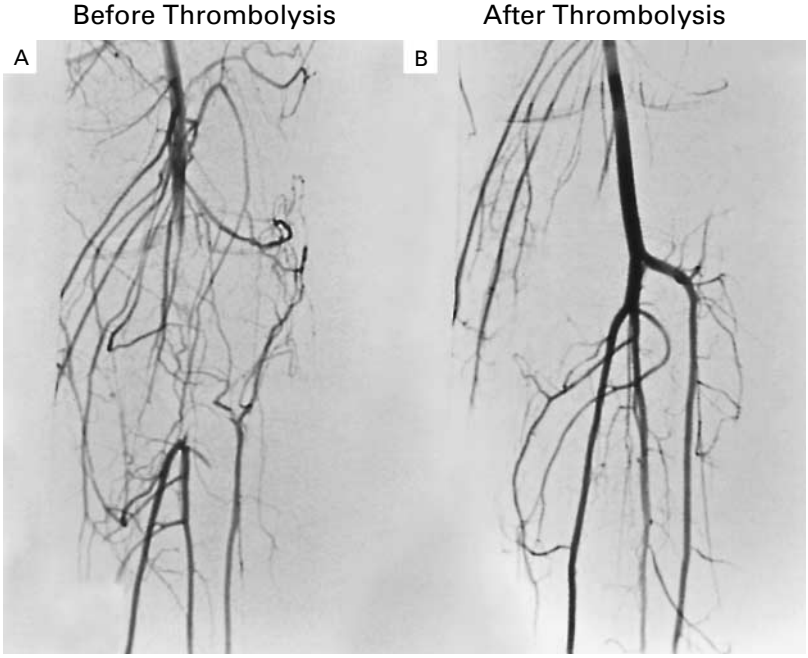

Figure 1. Angiograms of the Left Leg before Thrombolysis, Showing Complete Occlusion of the Popliteal Artery (Panel A), and after Thrombolysis, Showing Restored Patency (Panel B).