diac enzyme criteria occurred in patients who received thrombolytic therapy within 1 hour of symptom onset; that rate decreased with time, to 10% at 3 hours.

The trouble is, a mere 3% of ASSENT-3 participants were treated within 1 hour of MI symptom onset; 27% received thrombolytics within 2 hours. Getting more patients to come to the hospital or call an ambulance early after symptom onset has proved a daunting task. "So far every campaign to do that both here and abroad has failed," Dr. Gersh noted.

Transfer mania—the urge to transport everyone with an acute MI for primary PCI—is driven by half a dozen studies showing lower rates of death, stroke, and recurrent MI, he said. However, many of these trials were conducted in small European countries where transfer times are so short that the applicability of the results to U.S. patients becomes questionable.

This point was driven home by a recent report from the U.S. National Registry of Myocardial Infarction investigators (Circulation 2005;111:761-7). In analyzing nearly 4,300 MI patients transferred from one hospital to another for primary PCI during 1999-2002, they found only 4.2% had a less than 90-minute interval between time of arrival at the initial hospital to balloon inflation at the PCI center, as is recommended by current American College of Cardiology/American Heart Association guidelines for the use of primary PCI.

The Mayo Clinic has two helicopters and a fixed-wing airplane for transfer of MI patients from outlying hospitals. Here's what Mayo cardiologists recommend to physicians at community hospitals in their region without primary PCI capability: If a patient's duration of symptoms is less than 120 minutes, give full-dose thrombolytics and then transfer so the patient can undergo either routine elective angiography or, in the event of persistent ischemia, rescue PCI, Dr. Gersh said.

Beyond 2 hours, Dr. Gersh and his colleagues suggest direct transfer for primary PCI without preceding thrombolytics. This is a situation where facilitated PCI—that is, giving lytics and/or platelet glycoprotein IIb/IIIa inhibitors locally followed by transfer for PCI to maximize vessel opening—is very attractive. The results of ongoing trials of this approach are eagerly awaited, Dr. Gersh said.

If facilitated PCI proves effective, it will be particularly advantageous when transfer delays occur. For instance, last year the Mayo Clinic's air transport service was grounded by severe weather for some part of 58 days. "That's a fact of life in many parts of the United States," he noted. ■

L-Arginine After MI Linked to Higher Mortality

The addition of L-arginine to standard post-MI therapy does not decrease vascular stiffness or improve ejection fraction and may be related to increased postinfarction mortality, according to the results of the Vascular Interaction With Age in Myocardial Infarction trial.

Dr. Steven P. Schulman and colleagues randomized 153 patients following a first ST-segment elevation MI to receive L-arginine (with a goal dose of 3 g, three times daily) or placebo. Of the patients, 77 were aged 60 years or older. All the patients were followed up at 1, 3, and 6 months.

The amino acid L-arginine is a substrate for nitric oxide synthase. The results of previous studies suggest that it is associated with a reduction in vascular stiffness. As such, the investigators' objective was to establish whether the addition of the amino acid to standard treatment in post-MI patients, and especially older patients, would reduce vascular stiffness and improve left ventricular function (JAMA 2006;259:58-64).

In patients aged 60 years and older, ejection fraction and vascular stiffness did not change during the 6 months of follow-up in either group. However, six (9%) patients who had been randomized to L-arginine died, compared with none of those who received placebo. As a result, the data and safety monitoring board closed enrollment, the authors reported.

The participants had normal L-arginine levels at baseline, and Dr. Schulman, an associate professor of medicine and director of the Coronary Care Unit at Johns Hopkins University in Baltimore, and his associates speculated that the L-arginine level could explain the lack of efficacy. "The lack of any dose response in plasma L-arginine levels from 0 to 9 g suggests that higher doses of L-arginine would not have resulted in any biological effect in this population," the authors wrote, adding that many of the patients were already taking medications such as ACE inhibitors to improve vascular function.

The authors concluded that "L-arginine therapy should not be given to patients following a myocardial infarction. It neither alters noninvasive measures of vascular stiffness nor improves left ventricular function."

—Martha Kerr