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gate. As I arrived, a technician was warming up the ultrasound machine just as Dr. Clyde and 3 assistants arrived by ambulance with Linda on a stretcher, anesthetized with ketamine. Bonobos are well known for their "human" characteristics, their communication skills and expressions of empathy, and their exuberant sexuality. They have been conditioned to voluntarily submit to some medical procedures. Although bonobos are phylogenetically Homo sapiens' closest relative, they are wild animals who will bite each other or their keepers. They must be placed under general anesthesia for extensive examinations or treatment. General anesthesia is risky and unpleasant for the bonobos and is only used when essential.

A quick ultrasound examination showed markedly reduced left ventricular function with anteroapical thinning and dyskinesis, ascites, and large bilateral pleural effusions. Blood pressure was 90/60 mm Hg. The electrocardiogram showed frequent premature ventricular complexes. Serum creatinine was 1.4 mg/dl. Serum potassium was slightly elevated. Enzymes were consistent with passive hepatic congestion.

Linda's overall condition seemed to improve slightly after thoracentesis of 250-ml straw-colored fluid, but her cardiovascular and respiratory function remained tenuous. Dr. Victoria Clyde, a zoo veterinarian who has worked with the bonobos for many years, convened a "family" conference with the head primate curator and several zookeepers. Many of the treatments available to humans, such as intensive nursing care, intravenous inotropic therapy or mechanical circulatory assist, myocardial revascularization, and heart transplantation are simply not feasible for bonobos. Issues of prognosis, quality of life, and the compassion of those caring for Linda Bonobo were similar.

As Linda was unable to return to living independently in the bonobo quarters, and there were no other reasonable options, she was euthanized. Post-mortem examination showed extensive coronary atherosclerosis and an extensive, old anteroapical myocardial infarction.

Linda Bonobo will be sorely missed by zoo staff and visitors, and especially by the 21 remaining members of the bonobo colony, where Linda served as a matriarch for 14 years.

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Perfusion Cardiovascular Magnetic Resonance in the Clinical Scenario of Patients With Coronary Artery Disease

In the recent paper by Patel et al. (1), the investigators stated that cardiovascular magnetic resonance (CMR) and coronary multislice computed tomography angiography "do not currently provide information on ischemic burden and are not assumed to be present in the clinical scenarios;" in other words, they are not suitable to assess appropriateness of indications for coronary revascularization.

An increasing amount of literature supports the clinical application of stress CMR (performed with adenosine/dipyridamole, or more frequently, dobutamine infusion) in patients with coronary artery disease (CAD) (2–4). Perfusion CMR has been validated against positron emission tomography and quantitative coronary angiography in patients with ischemic heart disease (5,6). Schwitter et al. (5) demonstrated a sensitivity and specificity of 91% and 94% versus positron emission tomography and of 87% and 85% versus quantitative coronary angiography. A myocardial perfusion reserve index derived by perfusion CMR images can distinguish between normal subjects and patients with CAD (7–10). Nandalur et al. (11) recently conducted a meta-analysis of 37 studies on the diagnostic efficacy of stress CMR in the detection of CAD and concluded that stress CMR, using either vasodilator or dobutamine, had high sensitivity and specificity for the diagnosis of CAD.

Possible advantages of perfusion CMR over myocardial perfusion scintigraphy include higher resolution imaging that discriminates between subendocardial and transmural perfusion defects, and no need for additional radiation burden in patients who are often in need of subsequent angioplasty (12). The MR-IMPACT (Magnetic Resonance Imaging for Myocardial Perfusion Assessment in Coronary Artery Disease Trial) multicenter study recently demonstrated that CMR perfusion has similar, if not superior, diagnostic performance to myocardial perfusion scintigraphy in the detection of CAD (13). Although myocardial perfusion scintigraphy is a robust technique with extensive prognostic data, there is now increasing evidence of the prognostic value of stress CMR in patients with known or suspected CAD (14–18).

Both the American College of Cardiology Foundation/American Heart Association's 2005 Clinical Competence Statement on Cardiac Imaging with Computed Tomography and CMR and the 2008 Training Statement on Multimodality Noninvasive Cardiovascular Imaging published in previous issues of the *Journal* refer to perfusion CMR as a valid clinical diagnostic tool for guidance of coronary revascularization therapy (19,20).

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Declining In-Hospital Mortality and Increasing Heart Failure Incidence in Elderly Patients With First Myocardial Infarction

Ezekowitz et al. (1) report a decreasing overall mortality rate but an increasing incidence of heart failure (HF), especially late-onset HF, in a large cohort of patients with a first myocardial infarction in Alberta between 1994 and 2000. They found that in most patients (76%) who had suffered a myocardial infarction, HF developed. We have published similar data from the United Kingdom in patients from a single, large hospital service who had a first or recurrent myocardial infarction in 1998 (2), and reported that in 63% HF developed over the subsequent 6 years. Interestingly, we reported that 84% of those who died during follow-up first developed HF. Recalculation of data provided by Ezekowitz et al. (1) yields a similar figure (92%) in a somewhat older population (Fig. 1). However, some disparity does exist. Whereas Ezekowitz et al. (1) suggest a relatively modest increase in 5-year mortality in those in whom HF developed, our data suggest a striking difference (Table 1). This most likely reflects differences in diagnostic criteria. We defined HF as intervention with diuretic agents for symptoms or signs of HF; Ezekowitz et al. (1) used hospital codes and billing information, and <25% of patients coded as HF received diuretic agents.

The high proportion of patients in whom HF develops after a myocardial infarction might seem surprising because ischemic heart disease is common and the prevalence of HF is only about 1% (3). This reflects the poor prognosis of HF and suggests that the burden of HF may be better described by its incidence rather than prevalence (4). HF is also often under-represented in health care statistics because events such as death or hospitalization are ascribed to the cause of HF rather than to its presence. Death is usually a complex process, and attributing death to only one reason often is inappropriate. For instance, a patient could die of a lethal arrhythmia in the setting of worsening HF induced by a recurrent ischemic event. This patient died as a consequence of a constellation of events. What is important is to identify which interventions might produce worthwhile benefits for patients.

Clearly, reducing the incidence of HF is an important goal of treating myocardial infarction because it may improve well-being as well as extend life. In most patients in whom HF develops after a myocardial infarction, it occurs shortly after an initial or recurrent coronary event, suggesting these as possible therapeutic targets. However, treatment with aspirin (5), statins (6), or revascularization (7) have so far all proved disappointing in randomized trials of patients with chronic HF, although these data should not be extrapolated to acute care of myocardial infarction. More attention needs to be paid to HF as an end point in trials of treatment for myocardial infarction. Because HF is often a difficult and subjective diagnosis, trials need to develop standard objective criteria, possibly including measurement of natriuretic peptides and the requirement for therapy with loop diuretic agents.