Control of Neglected Tropical Diseases

TO THE EDITOR: Hotez et al. (Sept. 6 issue) present an excellent review of current global approaches to neglected tropical diseases. However, vigilant ongoing (post-intervention) surveillance to ensure that these diseases do not rebound should not be ignored. The integration of disease-control programs using a “rapid-impact package of drugs” is a feasible and probably cost-effective way to improve the quality of life for billions of people. The authors advocate monitoring and evaluation to judge the success of these programs but do not emphasize ongoing surveillance. Although surveillance may present a resource challenge in many environments, it also poses a statistical challenge as these heterogeneously distributed parasitic diseases become less common. A comprehensive, integrated surveillance plan should be incorporated into the cost estimates for the control or elimination of neglected tropical diseases. New approaches for determining the burden of these diseases as they become less prevalent should include improved diagnostic tools and novel epidemiologic techniques.

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THE AUTHORS REPLY: Huppatz and Durrheim comment on the need for surveillance. For the neglected tropical diseases, control and surveillance strategies are determined by the epidemiology and by the precise objectives of the intervention. For example, human African trypanosomiasis was controlled yet resurged during the period from the 1960s to the 1990s in Angola, Democratic Republic of the Congo, and Sudan, because surveillance stopped, health systems collapsed, and the mobile-team approach was abandoned. Today, human African trypanosomiasis is again under control, and effective surveillance is crucial in order to avoid a new resurgence. In contrast, soil-transmitted helminthiasis and schistosomiasis require regular preventive mass chemotherapy to reduce severe morbidity, and subsequent monitoring of lot quality assurance sampling is enough. With lymphatic filariasis, onchocerciasis, and trachoma, surveillance after control must monitor reduction of transmission. The critical tools referred to by Huppatz and Durrheim are being developed or deployed. However, cost and expertise are constraints on routine use, because health systems are overburdened and human resources are scarce where neglected tropical diseases are prevalent. Sadly, introducing routine surveillance...
of neglected tropical diseases in health management information systems will be challenging, given the diversity of epidemiology and health systems.

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Myocardial Reperfusion Injury

TO THE EDITOR: In the article by Yellon and Hausenloy (Sept. 13 issue) on myocardial perfusion injury, I would like to challenge the statement about therapeutic hypothermia, since this method is emerging as a novel way to reduce final myocardial infarct size. Therapeutic cooling showed a significant ST-segment resolution in the group with anterior myocardial infarction in the Cooling as an Adjunctive Therapy to Percutaneous Intervention in Patients with Acute Myocardial Infarction (COOL-MI) trial.1 Patients with anterior-wall infarction and a core body temperature of 35°C or less before angioplasty had a reduction in the final infarct size, as compared with patients with a temperature of more than 35°C. Observations from the COOL-MI trial suggest that the heart needs to be cooled optimally before reperfusion in order to provide optimal myocyte and microvascular protection.

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TO THE EDITOR: Yellon and Hausenloy discuss mechanisms of myocyte death. Readers may underestimate the relationship between the “no reflow” phenomenon and myocardial reperfusion injury.1 Tissue perfusion is an independent predictor of death after reperfusion and is associated with infarct size, ventricular function, and the presence or absence of congestive heart failure.2 Therefore, the no-reflow phenomenon should be considered prominently in discussing mechanisms of myocardial reperfusion injury.

The authors refer to adenosine as an antiinflammatory agent; however, adenosine has other actions (antiplatelet, vasodilatory, angiogenic, vasculogenic, and antifibrotic).3 Preclinical studies have shown cardioprotective effects of adenosine. The Acute Myocardial Infarction Study of Adenosine (AMISTAD) I and II showed reductions in infarct size in anterior infarction (67% and 57%, respectively).1,3 AMISTAD II showed that adenosine given within 3 hours after the onset of symptoms decreased mortality at 6 months (7.3% in the adenosine group vs. 11.2% in the placebo group).4

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Drs. Forman and Jackson report being coinventors on a patent owned by Vanderbilt University. No other potential conflict of interest relevant to this letter was reported.