In this issue of the journal, the article by Narita et al. highlights the fact that leptospirosis can be acquired by anyone exposed to infected water. The water can be surface waters, floods, or rivers. Any activity that brings humans into contact will cause infection. Over time, there has been a proliferation of reports that are basically variations of the same theme: large outbreaks with unusual presentations. Zaki and Shieh reported pulmonary hemorrhage associated with leptospirosis, and this has expanded our knowledge and sensitized us to clinical variations of this disease. This report discusses the epidemiology of leptospirosis with special emphasis on recreational water-borne acquisition of the disease. Previous exposure appears to offer some protection to local individuals working on a river. This is illustrated by the majority of hospitalized cases occurring in tourists who were never exposed, while local individuals with previous exposure and thus immunity were less affected. The Jarisch-Herxheimer reaction occurred in 43% of the patients in the study after treatment with ampicillin.

The epidemiology of leptospirosis has been and continues to be well researched. Knowledge gained in this endeavor has allowed protective measures to be put in place to reduce the exposure to the disease. I would like to see more basic science research on leptospirosis. We need to understand more about the organism and how it produces disease. This knowledge, hopefully, would then translate into targeted and better treatment. In the clinical field, we have not made much progress. We still are willing to debate if antibiotics are useful in these patients. However, there have been few advances in the treatment of severe cases in the last 20 years. We know that intensive care with excellent support will reduce mortality. However, we have no specific treatments for cardiac arrhythmias or pulmonary hemorrhages. I would like to challenge our colleagues who see large numbers of leptospirosis patients in the hospital setting to be the catalysts in well-designed treatment trials that will help us evolve treatment of this disease. If atrial fibrillation is prevented or abolished by medication, does it prevent more severe arrhythmias and cardiac death? Is desmopressin (DDAVP) or nitrous oxide useful for treatment of pulmonary hemorrhage? Will platelet transfusions or early administration of DDAVP keep platelet counts elevated and prevent pulmonary hemorrhage? Which has the better outcome in renal failure, peritoneal dialysis, hemodialysis, or continuous veno-venous hemofiltration? These are but a few of the studies that would help us along the road to better treatment (and reduced mortality) of the severe form of this disease.

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