ent paper are essentially similar to those previously obtained from studies of the rat. In both tissues a halothane-induced negative inotropism is accompanied by an interference with glucose metabolism prior to completion of the initial steps of glycolysis. This interference may be the result of impairment of glucose uptake by the cell or inhibition of glucose breakdown within the cell at an early point in the glycolytic pathway (prior to the phosphofructokinase step). The similarity of results from rat and human tissues is suggestive evidence that the presence of pathologic changes in the hearts from which these preparations were excised (not detailed by the authors but assumed) did not alter the basic response. At first glance this would seem to counter the argument presented earlier in this article. However, from my experience with isolated human preparations, and from the published data of others, I would predict that had their attention been focused upon transmembrane electrical phenomena in these preparations, or had Ko and Paradise used atrial appendages obtained from the adult rather than the child (in a study of either electrical or mechanical phenomena), the fit between the data obtained from human preparations and the laboratory analog would have been less convincing. This is not meant to be a criticism of their work, and I do not question the relevance of their findings. As Ko and Paradise have stated, it is much more difficult to do quantitative experiments on human (rather than animal) tissues owing to the difficulty in obtaining samples and their heterogeneity.

A major point that I would emphasize, and I am certain that Ko and Paradise would agree, is that if physiologic and pharmacologic studies of human cardiac tissue of this type ultimately are to be useful to clinicians, we must not permit the experimental designs to be rigidly limited by the problem of tissue heterogeneity. Rather, we must eventually undertake the difficult task of broad-spectrum analyses of detailed drug responses in each of the various types of cardiac fibers (*i.e.*, the myocardium as well as specialized conducting tissues) under each of the various conditions presented clinically. It is probable that ultimately a large number of laboratory analogs will be employed to simulate human cardiac responses, for we can be almost certain, even at this preliminary point, that no single species can adequately serve as a model for man.

RICHARD A. JENSEN, PH.D.
Assistant Professor
Department of Pharmacology
University of California
San Francisco, California 94122

Anesthesia

HEAT BALANCE The use of air conditioning in the modern operating theater to provide comfortable conditions for the surgical team has led to declines in the body temperatures of patients. Heat can be lost by radiation to surroundings, by evaporation from the wound, lungs and skin, and by the use of cold intravenous fluids. During lobectomies and pneumonectomies in 15 patients, the deep body temperatures decreased by 1.15 degrees C over three and a half hours, and the average surface temperature decreased by 2.6 C, corresponding with a total heat deficit of 78 kcal, or 21 kcal per hour. This deficit must be repaid later. The comfort of the patient is also affected by operative heat deficit. In the presence of cold stimuli from the surface, the recovering surgical patient will feel cold and shiver, and this discomfort often produces restlessness, which requires repeated doses of narcotic sedation. One of the results of observing central and surface temperatures is to emphasize the necessity for measuring both in all operations where heat balance is likely to be affected. (*Vale, R. J., and Lunn, H. F.: Heat Balance in Anaesthetized Surgical Patients, Proc. Roy. Soc. Med. 62: 1017 (Oct.) 1969.*)