Electric Hazards in Hospitals

Of what value is the report of a symposium published two years after the event? Substantial—since the report concerns electricity, which has just been discovered to be a threat to hospitalized patients—or so it would seem from the recent barrage of articles, symposia, and pronouncements on the topic. Brilliantly edited by Carl Walter, "Electric Hazards in Hospitals" is the proceedings of a workshop sponsored by the Committee on Anesthesia and the Committee on Shock of the National Research Council, in April of 1968. A summary of the meeting appeared in Anesthesiology in 1968. Now the official and complete record is available. Handsomely-mounted, the 277-page volume merits interest and concern.

The Proceedings are a compendium of knowledge and opinion up to 1968. Concern is appropriate because of vigorous efforts now underway to "do something" about a problem that remains undefined with respect to numbers and importance. That there is a problem cannot be denied, but the fact is that nobody really knows how many hospitalized patients are injured by electricity each year. More important, nothing is known of the near misses and how they occur. Participants in the 1968 symposium were aware of this, and stressed the need for sound epidemiologic data as a base for intelligent application of funds and efforts toward the safe use of electrical apparatus. There were no such data in 1968, owing to absence of effective case-finding and reporting mechanisms coupled with well-founded fear of litigation. Accident reports were meagre in number and anecdotal in character. This situation has not changed materially in the past two years, yet the search for solutions has forged ahead. Some proposals are already acquiring "muscle" through incorporation into upcoming revisions of NFPA Bulletin 56 and the Standards for Hospital Accreditation.

It is indisputable that some things ought to be done. Device testing, improved maintenance, and user education are long overdue. The wisdom and practicality of other proposals are open to debate. Is there an established need for ungrounded power systems in patient care areas; for elaboration of equipment grounding; or for lock-in power plugs for equipment? Is one case of induced ventricular fibrillation per 1,000 beds per year too many if halving the number calls for diversion of half-a-billion health care dollars from other more critical areas?

As electrical safety becomes a hot commodity, not all the portents are reassuring. Answers are being supplied before the questions are defined. Relatively simple issues are being confused by "what if" hand-wringing over second- and third-order possibilities. The huckster is moving in, with meaningless verbiage such as "approved" and "safe," which appeared in a recent hospital journal advertisement for electrical isolation equipment. For the imaginative attorney, broad new horizons of claims-litigation are unfolding. Whatever happens, the cost will be substantial and, as always, ultimately will be borne by the patient and the taxpayer.

Involvement in this issue will be thrust upon the anesthesiologist, interested or not. It behooves us to become informed. The Na-
In Vitro Studies of Human Cardiac Tissue

AN IMPORTANT QUESTION that frequently confronts anesthesiologists concerns the effects of general anesthetics on the heart. For years, the safety of anesthetics has been dictated by the magnitude and nature of their effects on cardiac function. It is now clear that the response of the heart is the result of a complex interaction of factors, including reflex changes in activity of the autonomic nervous system, alterations in transmembrane electrical potentials, changes in myocardial contractility, and altered metabolic phenomena in cardiac fibers. In addition, there is little doubt that the response of the heart to anesthetics is determined to a substantial degree by the age of the patient, the presence or absence of pathologic changes, and drug interaction at receptor sites.

Most of our current knowledge of the principles involved in these phenomena has been derived from laboratory experiments (either whole animals or tissue preparations) and from data compiled from empirical clinical observations. In the practical application of anesthetic procedures and analysis of clinical data, the anesthesiologist certainly must question the extent to which the results of laboratory experiments are applicable to the patient. The problems of translating physiologic and pharmacologic responses from one species to another have been discussed many times. A related, but rather infrequently considered, problem highly relevant to anesthesia concerns the extent to which it is possible to extrapolate data from “normal” cats, dogs, and rats to human patients who are “abnormal” as a result of either premedication (e.g., with vagolytics, digitalis, sedative, or narcotics) or pathologic changes which modify, and perhaps seriously compromise, cardiac function, or both.

In most attempts to identify the mechanisms of the electrical and mechanical actions of drugs on the heart, it has been assumed that the same rules and limits apply to normal and to abnormal cardiac fibers, and that studies of normal fibers might be used to explain either abnormal activity or the mode of action of drugs. (The latter is particularly evident in studies of the mechanisms of cardiac arrhythmias and the responses to antiarrhythmic drugs.) With open-heart surgery and the consequent availability of samples of human cardiac tissue, it is now possible to test these assumptions and analyze cardiac drug response directly in the absence of the indirect factors inherent in the intact human heart.

In this issue of the Journal, Ko and Paradise describe the effects of halothane on myocardial contractile force and glucose metabolism in human atrial appendages isolated from 7-10-year-old patients undergoing cardiac surgery. This work is an extension of previous studies of a similar nature using isolated rat atria by Paradise and his co-workers, many of which have been reported in Anesthesiology. The data for human atria provided in the pres-