centration of anesthetic, including the rate at which the preparation is driven, the temperature, the initial tension applied to the muscle, the adequacy of oxygenation, and the calcium ion concentration in the bath. One cannot interpret an isolated data point without information about these matters and the degrees of depression found at other concentratons of anesthetics.

In summary, we are interested to learn that the concentrations of certain anesthetics can be different on the two sides of a sintered glass disc, and we look forward to full documentation of the claim, and particularly its quantitative importance. We think it unlikely that this problem influenced results with cyclopropane. Our data on diethyl ether suggest to us that little quantitative error occurred in overestimation of potency ratios with the volatile anesthetics, but the possibility of error in these ratios must be acknowledged. There is no reason to believe that even if a concentration gradient existed in our system it would alter the slopes of the dose–response curves we published or the configuration of the individual isometric twitches. Therefore, we do not view the finding of Vongvises *et al.* as a serious challenge to the major conclusion of the paper: that the inhalation anesthetics studied all depress contractility via a common mechanism.

BURNELL R. BROWN, JR., M.D., PH.D.
J. RICHARD CROUT, M.D.
Department of Anaesthesia
Harvard Medical School
Boston, Massachusetts 02115

Hyperthermia during a Second Anesthesia

To the Editor:—Increasingly numerous reports describe the development of malignant hyperthermia in patients who had undergone one or more uneventful anesthesias previously.[1-3] An additional case has been observed by us.

A healthy 30-year-old woman underwent a gynecologic operation under uneventful thiopental–nitrous oxide–oxygen–succinylcholine anesthesia lasting 75 minutes. Nine weeks later, while drunk, she lacerated one wrist severely. After three days of intensive antibiotic therapy (nafcillin, ampicillin, dicloxacillin), she was anesthetized for tendon repair. Anesthesia was induced with thiopental, followed by 50 mg succinylcholine and intubation of the trachea. Anesthesia was maintained with nitrous oxide–oxygen–halothane. A tourniquet was then applied to the arm. Thirty minutes later, blood pressure and heart rate started to increase; halothane was discontinued, but blood pressure rose to 200/90 torr and heart rate to 140 beats/min. The patient felt hot and dry, and the rectal temperature was found to be 105 F.

The operation was abandoned. External cooling brought the temperature to 102 F over a 30-minute period and to 99 F during the next hour. Hyperventilation with oxygen and intravenous fluids were employed as well. The temperature remained below 100 F, and a week later the operation was completed under brachial plexus block.

The question arises as to whether hyperthermia may have been triggered by halothane, excessive alcohol, or one of the antibiotics. Ethanol is capable of enzyme induction,[4] while antibiotics enhance the uncoupling effect of barbiturates on oxidative phosphorylation in liver and brain mitochondria.[5] Elliott[6] stated that anesthesia "must be integrated into the patient's total therapeutic regimen." I may be that a detailed retrospective history of drug and food intake as well as of exposure to pollutants in patients who have developed hyperthermia during anesthesia will provide information to elucidate the triggering factor or factors.

SIMON HALEVY, M.D.
GERTIE F. MARX, M.D.
Department of Anesthesiology
Albert Einstein College of Medicine
Bronx, New York 10461

REFERENCES

1. Capizzi LS, Phillips OC, Harris LC Jr: Malignant hyperthermia during anesthesia. ANESTHESIOLOGY 31:97–99, 1969

2. Daniels JC, Polayes IM, Villar R, *et al.*: Malignant hyperthermia with disseminated intravascular coagulation during general anesthesia. Anesth Analg 48:877–883, 1969
3. Thomford NR, Hamelberg WE, Wiederholt WC: Sudden hyperthermia during general anesthesia. Surgery 66:850–855, 1969
4. Conney AH: Pharmacological implications of microsomal enzyme induction. Pharmacol Rev 19:317–366, 1967
5. Killam KF, Brody TB, Bain JA: Potentiation of barbiturate hypnosis by certain uncoupling agents. Proc Soc Exp Biol Med 97:744–748, 1958
6. Elliot HW: Influence of previous therapy on anesthesia. Clin Pharmacol Ther 3:41–58, 1962

Burn Therapy

BURN THERAPY IN CHILDREN Children represent a large proportion of the 2,000,000 people hospitalized each year for burns. The first-aid treatment consists of reassurance and wrapping the involved area in clean cotton or linen until the extent and severity of the burn can be evaluated. No child should be transported until respiratory difficulties are relieved and control of the airway is obtained. Upon arrival at the hospital, the order of procedure is as follows. 1) Adequacy of the airway is checked, and oxygen, tracheal intubation, tracheostomy and ventilatory assistance are provided as indicated. 2) Sedation is given only if necessary, using the intravenous route. 3) All clothing is removed and the patient weighed. 4) An intravenous catheter that is adequate to deliver fluids at a high rate of flow is inserted. A central venous pressure catheter is desirable in patients with extensive burns. 5) The extent and depth of the burn are evaluated. 6) The burned area is covered with gauze dressings wet with 5 per cent silver nitrate, using sulfamylon for facial burns, and 7) areas of potential contractures are splinted. 8) Blood samples for baseline laboratory studies are obtained; 9) fluid requirements and fluid routines are established. 10) A Foley catheter is inserted and urine specimens are obtained for analysis. 11) A tabular chart of intake and output, vital signs and chemical values of blood and urine is prepared. 12) Prophylactic antibiotic therapy with penicillin is started, and 13) appropriate protection against tetanus is given. 14) A detailed history, including circumstances and time of injury, is obtained. 15) Consideration should then be given to means of achieving temporary and permanent skin cover and 16) nutritional support, treatment of anemia and hypoproteinemia, and rehabilitation, including physiotherapy and emotional support. A balanced salt solution rather than plasma is now used in the early treatment of burns. Two ml/per cent burn/kg plus 1,500 ml/m^2 of Ringer's lactate solution are administered every 24 hours. An increase in the rate of infusion is necessary if the urinary output decreases to below 30 ml/m^2/hour or if the hematocrit rises above 50 per cent. Serum albumin or plasma is not administered unless blood pressure, central venous pressure and urinary output cannot be maintained by an infusion of Ringer's lactate solution of 300 ml/m^2/hour. No more than the volume required for a 50 per cent burn should be infused even if the burn covers more than 50 per cent of the body surface. Half of the estimated first day's fluid requirement should be given in the initial eight hours after the burn. The rate of fluid administration must be decreased if anesthesia or surgery is necessary because these procedures commonly lead to antidiuresis. The aim of fluid therapy is to keep the urine flow at 40 ml/m^2/hour, urinary sodium at 20 to 80 mEq/l, serum sodium between 130 and 140 mEq/l, and serum potassium between 3.5 and 5.0 mEq/l. The requirements for sodium chloride are very much increased by the use of silver nitrate wet dressings. Chloride is lost at a rate of 3.5 mm/100 cm^2 of deep second- or third-degree burn/day. Alkalinization is an advantage if hemoglobinuria is present. (*Herrins, J. T., and Crawford, J. D.: Care of the Critically Ill Child—Major Burns, Pediatrics 45: 449 (March) 1970.*)