phenobarbital-induced rat model. Immediate transsubstantiation of such data to humans, a dangerous gambit at best, would imply that certain individuals may be more susceptible genetically to this complication than others. Halothane hepatitis would thus be cast in the lot of a multifactorial event, involving variables such as splanchnic blood flow, hepatic oxygenation, qualitative and quantitative aspects of enzyme induction, but pivoting about a pharmacogenetic axis. This speculation is made more appealing because there is information that certain racial characteristics may produce a high rate of jaundice following halothane.*

In spite of advances in knowledge made by the Flinders group and others dedicated to clarifying this arcane issue, the allergy theory is alive and well. A recent publication notes evidence of hepatocyte reacting antibodies gathered from patients who suffered fulminant hepatic failure following halothane anesthesia.[5] The final mosaic produced by melding the biochemical activation hypothesis and the allergy theory, like Carroll's dark and obscure passageway, is certainly not effulgent at this time. It may be, as so often happens, that both these seemingly adversarial concepts are partially true, partially untrue. An initial biochemical event, *e.g.*, increased "reductive" biotransformation, environmental and/or genetic in origin, leads to early hepatocyte damage. This destruction may be aided and abetted by an immune response which intensifies and perpetuates the lesion. Such an hypothesis could not only unravel the Gordian Knot of challenge tests, second administrations, age and obese proclivities etc., but could explain the differences between mild and reversible cases of hepatic disturbance *vs.* the frequently lethal fulminant hepatic destruction observed. Certainly the plenary truth is still obscured, but work such as this of the group from Australia, however irrelevant it may seem to the uninitiated, is accretion of knowledge eventually contributing to patient safety, our ultimate goal.

BURNELL R. BROWN, JR., M.D., PH.D.
Professor and Head,
Department of Anesthesiology
University of Arizona College of Medicine
Tucson, Arizona 85724

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Blood Glucose Control during Surgery

THE MANAGEMENT OF DIABETES MELLITUS, including its acute complications of ketoacidosis and hyperosmolar coma, has undergone and is continuing to undergo revision. Rediscovery of old programs, return to rigid control of hyperglycemia, and attempts to achieve normalization of blood glucose have occupied the energies of clinicians and investigators over the past decade. Amidst this whirlwind of activity in diabetes management, it is not surprising that the sacrosanct arbitrary insulin regimens utilized for control of blood glucose during surgery should be scrutinized for their effectiveness and scientific merit. Insulin resistance occurs during surgery due to a combination of factors involving the secretion of hormones antagonistic to insulin action,[1] the administration of glucose solutions during surgery and in the postoperative period, and an apparent cellular hyporesponsiveness to endogenous insulin in nondiabetics[2,3] and to exogenous insulin in diabetic patients.[3] Logically, to meet this challenge of surgery, the insulin dosage to control hyperglycemia should be determined by the level of glucose during surgery as in the routine management of diabetic patients. For convenience, clinicians have accepted the alternative of arbitrary insulin regimens

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for glucose control for diabetic patients during surgery. However, in this issue of ANESTHESIOLOGY, Walts *et al.*[4] have nicely demonstrated that arbitrary insulin programs to control blood glucose during surgery fall short of their intended goals and should be abandoned. The authors conclude and recommend that control of blood glucose during surgery should be individualized and depend on the level of blood glucose determined at periodic intervals during the surgical procedure and in the immediate postoperative period. This conclusion and recommendation should come as no surprise as this approach has been the hallmark of clinical practice. The best approach in any treatment program is monitoring of the variable that is being corrected and adjustment of the level of medication on the basis of how effective the variable is controlled. Why convenience superseded logic in the management of blood glucose during surgery is not clear. The observations of Walts *et al.*,[4] however, have set the record straight. The use of controlled insulin-glucose infusions makes it possible for the anesthesiologist and the primary care physician or diabetologist to maintain the plasma glucose in a tight range between 80 to 120 mg/dl during surgery and in the postoperative period.

Perhaps the most exciting recommendation made by these investigators is the proposal for an endocrine intensive care unit for diabetic patients for preoperative and postoperative management until normal oral intake is resumed. Patients would be admitted to this unit before surgery to establish the monitoring of blood glucose and to initiate the insulin-glucose infusion.The patient would be returned to the unit after surgery for stabilization prior to returning to routine hospital care. The length of stay in the endocrine intensive care unit would be variable depending on clinical status and stabilization of blood glucose control. An endocrine intensive care unit has great merit not only for the surgical patient but for all diabetic patients, particularly insulin-taking diabetics, admitted to a hospital. Insulin-taking patients require a great deal of attention and monitoring. Fragmentation of such care and management throughout a variety of hospital units leads to an unevenness of the quality of diabetic care. Therefore, an endocrine intensive care unit should receive serious consideration by hospital administrators and physicians involved in the care of diabetic patients. Such a unit would promote optimum control of blood glucose in a variety of medical conditions through centralization of appropriate personnel for the management, instruction, and care of the diabetic patient. Complications of hyperglycemia such as infection, poor healing, and negative nitrogen balance would be minimized and optimum control of the hyperglycemia and diabetes mellitus promoted. Afterall, such an approach is what clinical practice is all about.

P. J. PALUMBO, M.D.
Professor of Medicine
Mayo Medical School
Consultant in Internal Medicine
and Endocrinology and Metabolism
Department of Internal Medicine
Mayo Clinic/Foundation
Rochester, Minnesota 55901

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