An Integrated Analysis of Glucose, Fat, and Protein Metabolism in Severely Traumatized Patients

Studies in the Basal State and the Response to Total Parenteral Nutrition

JAMES H.F. SHAW, M.D., F.R.A.C.S.,* and ROBERT R. WOLFE Ph.D†

University Department of Surgery, Auckland Hospital, Auckland, New Zealand,* University of Texas Medical Branch at Galveston, and Shriners Hospital, Galveston, Texas†

A series of isotopic infusions were performed in 43 severely ill patients suffering from blunt trauma (mean injury severity score of 31). The patient data have been compared with data obtained from 32 normal volunteers, and in addition the metabolic response of the trauma patient to total nutritional support (TPN) has been assessed. The rate of VO₂ was elevated in the trauma patients compared with that of the volunteers (160 μmol/kg/minute vs. 103 μmol/kg/minute). Glucose production was significantly increased in the patients compared with the volunteers (21 ± 2 μmol/kg/minute vs. 14 ± 1 μmol/kg/minute), but the trauma patients had an impaired capacity to directly oxidize plasma glucose. The percentage of glucose uptake oxidized in the volunteers was 36 ± 2%, and the percentage of glucose uptake recycled was 10 ± 1%. By contrast, in the trauma patients, 23 ± 4% of the glucose uptake was directly oxidized, and 29 ± 11% was recycled. The rate of glycerol turnover in the trauma patients (5.3 ± 0.3 μmol/kg/minute) was significantly elevated compared with the volunteer value (2.2 ± 0.1 μmol/kg/minute), and the basal rate of fat oxidation was twice as high in the patients as in the volunteers (2 mg/kg/minute vs. 1 mg/kg/minute). The rate of whole body protein catabolism was significantly higher in the patients (5.8 ± 0.7 g/kg/day vs. 4.3 ± 0.3 g/kg/day), and as a result, the rate of net protein catabolism was significantly elevated in the patients. The response to TPN (amino acids and a 50:50 mixture of glucose and fat) included an increase in the percentage of glucose uptake oxidized (up to 45 ± 12%), a decrease in the oxidation of fat (up to 0.8 mg/kg/minute), and a significant increase in whole body protein synthesis (up to 6.1 ± 1.1 g/kg/day) so that the rate of net protein loss was minimized but not prevented. The rate of net protein catabolism during TPN was 1.3 ± 0.5 g/kg/day.) There was no correlation between the injury severity score (ISS) and the degree of metabolic abnormality. The rate of NPC in the patients with ISS < 20 was higher than in the volunteers (ISS = 0), but the values for NPC in patients with ISS 21–40, and ISS > 40 were virtually identical to the corresponding values in patients with ISS < 20. It is concluded from these studies that: 1) Trauma patients have a high rate of VO₂. There is an increase in glucose turnover and lipolysis in the basal state, but reliance on plasma glucose as an energy substrate is reduced. 2) Trauma patients are catabolic due to a significantly elevated rate of whole body protein catabolism that is in part compensated for by a minor increase in protein synthesis. 3) The use of TPN in trauma patients results in an increase in plasma glucose oxidation, a decrease in fat oxidation, and as a consequence of the significant increase in protein synthesis, the rate of protein loss is minimized but not eliminated. 4) The metabolic response to blunt trauma in clinical terms is an "all or none" response: the patient with an ISS of 15 is metabolically similar to the patient with ISS = 50.

T HE METABOLIC RESPONSE to trauma has been widely studied since the pioneering work performed by Cuthbertson and others, but despite this, a number of aspects remain unresolved. In particular, the capacity of the trauma patient to oxidize glucose and/or fat is unclear, and the kinetic basis for the increased protein loss seen in these patients has not been resolved. In addition, the efficacy of total parenteral nutrition (TPN) as a means of repleting the catabolic trauma patient is debated. In a general sense, it is clear that most types of trauma result in an increase in glucose turnover. Wolfe et al. have shown that burn patients have elevated rates of glucose production, and others have come to the same conclusion using animal models. In this report, we debate the capacity of trauma patients to oxidize glucose. Some investigators have reported that glucose oxidation is increased in the trauma patient, others conclude that trauma patients oxidize glucose to an extent similar to...