Carbohydrate has been the predominant source of energy in the human diet since the beginning of history. The largest source of dietary carbohydrate has traditionally been the polysaccharide, starch, derived from cereals. Although cereals have been cultivated for over 10000 years, primitive man obtained glucose, fructose and sucrose from fruits, berries and honey. During the 19th and 20th centuries, large amounts of various sugars have been obtained from agricultural products, such as sucrose from sugar canes and sugar beets and glucose–fructose mixtures from cornstarch. These purified mono- and disaccharides contribute a significant amount to the total energy consumption of contemporary western man, particularly in the United States. The consumption of sucrose and glucose has risen as a percentage of total carbohydrate in the diet from around 23% in 1900 to over 50% by 1965. A parallel decrease has occurred in the amount of carbohydrate consumed as starch. The rising dietary intake of refined simple sugars has prompted criticism from those who feel that other nutrients which are present with starch in natural foods may be deficient in a modern diet.

The uncertainties which relate to the optimum content of carbohydrate in parenteral nutrition also involve questions concerning the proper chemical form of the carbohydrate and the total amount of carbohydrate that should be given. Some European investigators have recommended that parenteral solutions include fructose, sorbitol and xylitol and there are scattered references to maltose. Since each of these sources of carbohydrate have been shown to be either impractical, or to have undesirable side-effects, the use of glucose has been the common form of carbohydrate for the majority of parenteral nutrition in Europe and essentially all of parenteral nutrition in the United States. All body cells have the capacity to oxidize glucose, either aerobically via the Embden–Meierhoff pathway to pyruvate, or anaerobic-
ally to lactate. Pyruvate and lactate can then be further oxidized via the tricarboxylic acid cycle to yield energy. The metabolic utilization of glucose, including oxidation, is dependent upon the presence of insulin to facilitate its entrance into body cells. When insulin synthesis and output is inhibited during periods of increased catecholamine activity, the uptake of glucose into cells is reduced with an associated tendency toward hyperglycaemia.

GLUCOSE METABOLISM – THE DEPLETED PATIENT

The infusion of excessive quantities of glucose above that required to meet the resting energy expenditure (REE) results in lipogenesis. This conversion of glucose to fatty acids is associated with a rise in the non-protein respiratory quotient, which is largely the result of an increased CO₂ production. The magnitude of these changes is a function of the patient’s clinical state and the amount of the glucose load. In the depleted patient, lipogenesis occurs readily and the non-protein respiratory quotient commonly rises to 1.1 or 1.2, at a time when there is essentially no increase in the resting energy expenditure as represented by the O₂ consumption despite the large increase in CO₂ production³. In a study with graduated glucose intakes given to depleted patients, Elwyn et al.⁴ showed that there was no increase in resting energy expenditure with increasing energy intake below energy equilibrium. However, when glucose intake achieved a positive energy balance, the REE was found to increase by 1 kcal for each 5 kcal of intake. At zero energy balance, nitrogen balance in these depleted patients was only slightly positive at an intake of 173 mg N per kg. This is about twice the intake of nitrogen required to maintain zero nitrogen balance in normal adults. These authors concluded that improving the nitrogen balance by increasing glucose intake above energy expenditure restored mainly the portion of the lean body mass associated with fat deposition.

GLUCOSE METABOLISM – THE HYPERMETABOLIC PATIENT

The administration of large amounts of glucose in lipid-free total parenteral nutrition to the hypermetabolic patient, who is injured or septic, reveals a different metabolic response from the depleted patient. The hypermetabolic patient shows major increases in not only the resting CO₂ production, but also the resting O₂ consumption, so that the non-protein respiratory quotient remains below 1.0³ (Figure 23.1). This indicates that either lipogenesis has been inhibited or that some fat oxidation persists in the face of the large carbohydrate intake which would normally abolish fat oxidation and produce net lipogenesis. These acute patients have an elevated urinary excretion of norepinephrine, while receiving 5% dextrose and water, and the