SHORT COMMUNICATION

Substrate oxidation shortly after accidental injury in man

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Summary

1. Oxidative metabolism has been studied by indirect calorimetry in 42 patients within the first 6 h after accidental injury.

2. The level of energy expenditure bore no relationship to the severity of the injury and varied more widely from patient to patient than in normal subjects.

3. After minor and moderate injuries the respiratory exchange ratio (mean 0.86) was not different from that in normal subjects on a mixed diet. After severe injuries the ratio (mean 0.78) was significantly lower \( (P < 0.005) \), indicating that the main substrate for oxidation in these patients was fat, despite their hyperglycaemia.

Key words: fat oxidation after trauma, indirect calorimetry, injury, metabolism, trauma.

Introduction

Cuthbertson [1] first described how the metabolic responses to a serious injury could be divided into an 'ebb' phase, lasting some hours, followed, in those who survived, by a 'flow' phase which could last for days. The 'ebb' phase is characterized by the mobilization of the stores of carbohydrate and fat in the body [2] but, while there is experimental evidence that carbohydrate oxidation is reduced at this time [3, 4], little is known about the preferred fuel for oxidation in recently injured man. We have now used indirect calorimetry to study substrate oxidation during the first 6 h after an injury.

Methods

Forty-two patients were studied shortly after admission to the Accident and Emergency Department and before definitive treatment of their injuries had begun. The patients were allocated an Injury Severity Score (ISS) according to the method of Baker et al. [5]. Measurements of gaseous exchange were made with the patient semi- or fully supine with a variable degree of exposure to the environment. Oxygen consumption, carbon dioxide production and the volume of the expired air were measured with a Metabolic Measurement Cart (Beckman R1IC, High Wycombe, Bucks.). The patients were connected to the apparatus with either a mouth-piece and valve (part no. 875830) with a nose-clip or a Royal Air Force face mask and helmet. Recordings were made for periods of 10–25 min. Equilibrium is reached after 4 min and the metabolic rate \( (\text{kcal h}^{-1} \text{m}^{-2}) \) was calculated from the data printed out at the end of the recording period, and from height and weight where these were known or could be measured. Core temperatures were measured with a zero-gradient aural thermometer (Addison Process Control Ltd, Beckenham, Kent). Environmental temperatures were measured with either a whirling hygrometer of a Light Minilab (Light Laboratories Ltd, Brighton, Sussex). The conditions were similar for all patients: median dry bulb 23.8°C (range 18.9–25.8°C), wet bulb 16.4°C (range 12.1–20.5°C). Glucose, non-esterified fatty acids and insulin in venous plasma were measured by methods described previously [2]. The results were analysed statistically by standard methods [6]. These studies were approved by the Local Ethical Committee.

Results

The patients have been divided into two groups according to the severity of the injury. Thirty patients (16 males) with ISS \( \leq 12 \) formed one
group of minor and moderate injuries and 12 patients (nine males) with ISS > 12 another of severe, usually multiple injuries. Their ages ranged from 19 to 90 years (median 50) in the first group and 15–76 years (median 35) in the second. Examples of injuries at the top of the first group would be a fracture of the tibial plateau or a compound fracture of the femur. There was one death in the series and this patient (female, age 76 years) died with multiple head injuries (ISS 25) 3 h after the accident. Two patients with moderately severe injuries (ISS 9 and 10) showed evidence of recent ingestion of ethanol. The measurements of metabolic rate and respiratory exchange ratio were made mostly 2–3 h after the accident. At that time only one patient (ISS 29) was hypotensive (blood-pressure 95/60 mmHg). Twenty-seven per cent of the patients had diastolic pressures >100 mmHg.

The results are shown in Table 1. The respiratory exchange ratio was significantly lower in the severely injured patients despite their hyperglycaemia. The median values for these data were very similar to the mean values. The difference in the respiratory exchange ratio was not related to the individual’s level of energy expenditure or to the interval from the last meal. In both groups of patients energy expenditure was very variable. This variability was significantly greater (F test: P < 0.05) than that in 21 healthy control subjects studied in the same environment with about 1 clo insulation (range 72–110% predicted [7]). These differences could not be related to the severity or type of injury, the patient’s age, fluid replacement, environmental temperature or the interval from the last meal.

Although there is a correlation between energy expenditure and pulse rate in normal subjects [8] this relationship was lost after injury. The responses did not appear to be influenced by analgesia (pethidine 50–150 mg intramuscularly, in eight patients; Omnopon 10 mg intravenously in one patient; O₂/N₂O (1:1) Entonox, in three patients).

**Discussion**

Our recently injured patients were not in a steady state, either physiologically or metabolically, so that the short collection periods used for the indirect calorimetry were appropriate. Although the level of energy expenditure shortly after injury was very variable it is clear that after serious injuries the respiratory exchange ratio was low. Indeed, as the respiratory rate in these patients was high (median, 23 min⁻¹) the ratio may have been overestimated. These ratios have not been corrected for protein oxidation since relevant urine samples cannot be obtained. If increased amounts of protein were being oxidized so early after injury the uncorrected ratios would over-estimate the rate of glucose oxidation. The low value of the ratio indicated that the main substrate being oxidized was fat and, thus, that glucose oxidation was reduced after severe injury. This was particularly remarkable in view of the hyperglycaemia found after severe injuries and could not be explained by a decrease in the mean plasma insulin concentration (Table 1). These changes are very similar to those recorded in animal experiments [4, 9]. It is of interest that Askanazi et al. [10] have recently shown that fat is also the principal substrate for oxidation in the flow phase after injury.

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References