Caloric requirements after operation

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It has become clear since the original studies of Cuthbertson (1930) that the change in metabolism after injury involves an alteration in the internal environment and in the composition of the body, which is reflected in fluid and electrolyte metabolism, energy and intermediary metabolism and endocrine function (Moore & Ball, 1952). This is appreciated by the patient as loss of weight, diminished muscular strength and activity, and a temporary but definite decrease in intellectual application. The nutritional implications of these metabolic changes have been a major source of inquiry and much of the work has been directed towards two objectives. The main part has aimed at achieving a clear understanding of the mechanisms involved and of the factors which may influence the metabolic response to trauma. The other aspect has been principally clinical, dealing with the effect of these metabolic changes on the response of the patient to his management with particular emphasis on nutrition. It is with these two objectives in mind that this paper deals with the caloric requirements after operation.

Mechanisms and modifications

The early observations of Cuthbertson (1932) included measurement of basal oxygen consumption in four patients who sustained fractures of the lower limbs. These patients were young men and would normally have had a resting oxygen consumption of 140–160 ml/m² per min (250 ml/min). In most of the measurements the resting oxygen consumption is in excess of 160 ml/m² per min during the first 4–10 d after trauma (Fig. 1). The peak of the increase was not immediate but was reached usually afer 3 d and normal levels were not regained until 14–21 d after the injury. A similar study was made of patients having moderate elective surgical

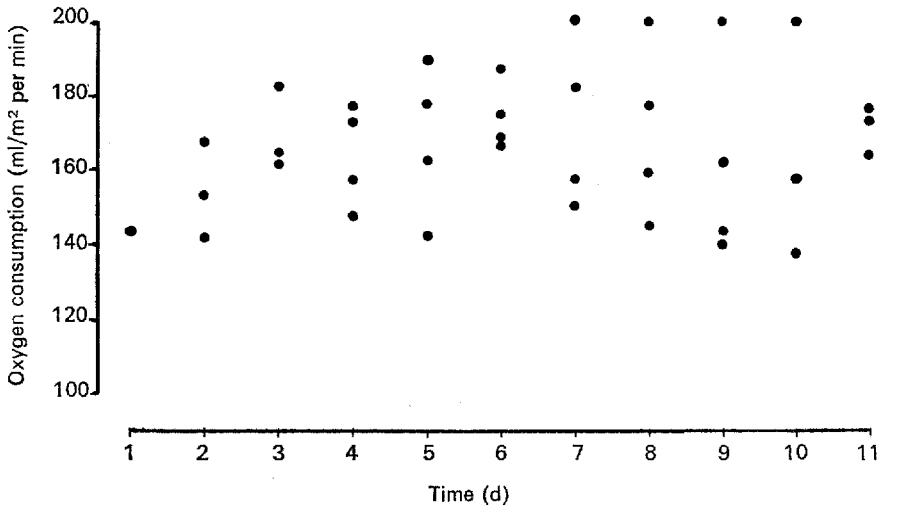

Fig. 1. Resting oxygen consumption of four men who sustained fractures of the lower limb. Values have been reduced to surface area (m²) for comparison (Cuthbertson, 1932).

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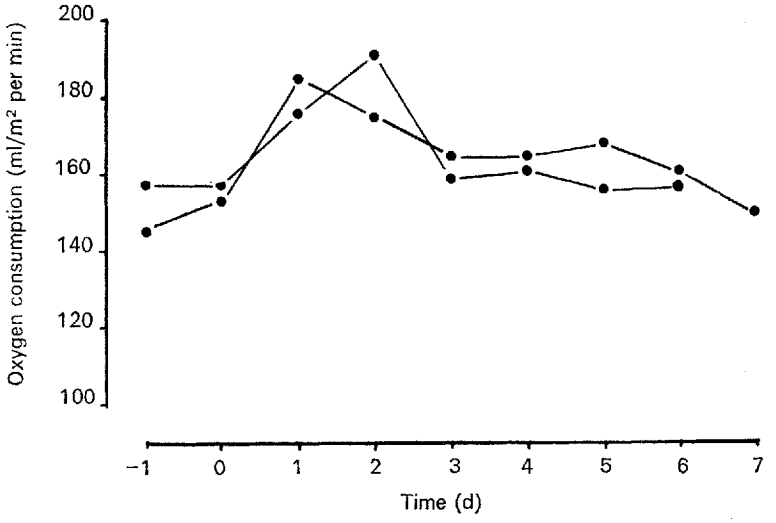

Fig. 2. Resting oxygen consumption of two men having moderate surgical trauma (Cuthbertson, 1932).

trauma, in this instance meniscectomy; this demonstrated that the resting oxygen consumption increased after operation to 110–120% of pre-operative values (Fig. 2), an increase of the same magnitude as the fracture cases showed, but elevation was sustained only until 3 or 4 d after operation. It was noted that there was a relation in time between the increase in resting oxygen consumption and the increase in urinary nitrogen excretion which followed the trauma, and it was concluded by the author that this was part of a 'response of general origin indicating a rapid catabolism of the body's substance in response to the exigencies of repair and maintenance'.

A different form of trauma was studied later by Cope, Nardi, Quijano, Rovit, Stanbury & Wight (1953), who measured resting oxygen consumption, expressed as 'metabolic rate', in patients with burns of differing severity. Burns of 20% body surface area were associated with an increase in 'metabolic rate' of 130–140%, whereas burns of greater severity, e.g. 65% surface area, were associated with a

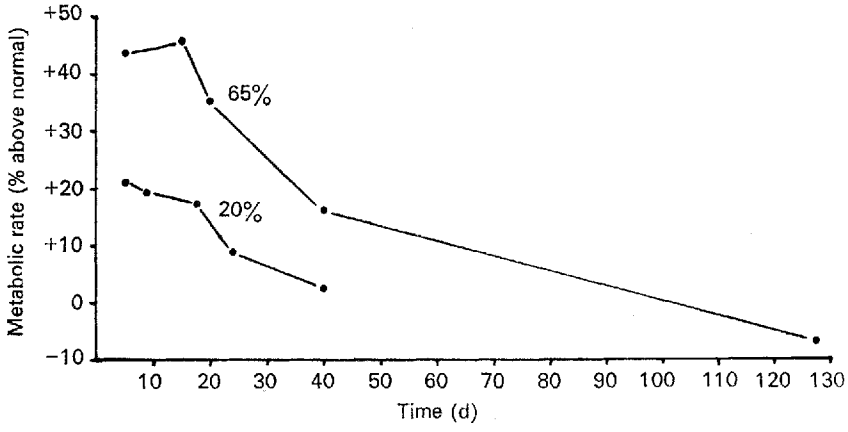

Fig. 3. Metabolic rate in patients with burns of 20% and 65% body surface area (Cope *et al.* 1953).

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'metabolic rate' up to 160% higher which was also of much longer duration (Fig. 3). Further studies by Cope *et al.* (1953) demonstrated that the magnitude of the increase in resting energy expenditure was related to the size of the traumatic stimulus.

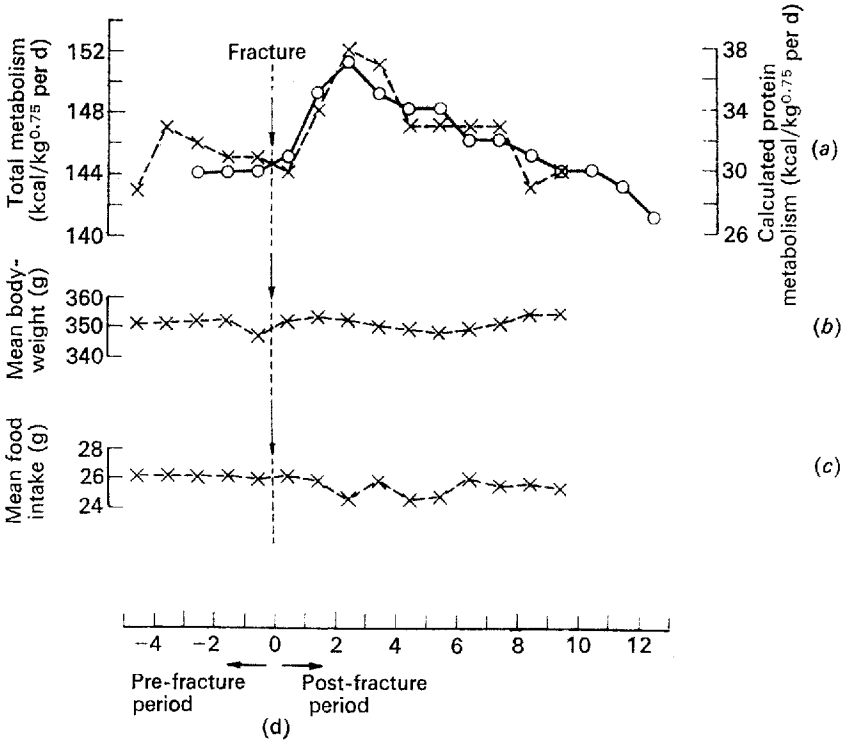

Fig. 4. *a*, Total metabolism (×— — —×), protein metabolism calculated from urinary nitrogen (○———○), *b*, mean body-weight and *c*, mean food consumption of nine rats on a high-protein diet (Cairnie *et al.* 1957). Reproduced by permission of the *British Journal of Experimental Pathology*.

Confirmation of the relationship between increased resting oxygen consumption and excess N loss was provided by studies on the effect of high and low protein intakes on total metabolism and urinary N excretion in rats subjected to hind limb fracture (Cairnie, Campbell, Pullar & Cuthbertson, 1957). Rats fed on high protein intakes responded to trauma with a significant elevation in both total metabolism and in urinary N (Fig. 4). The increase in total metabolism could be accounted for solely by the heat derived from catabolism and urinary N. Fig. 5 shows that in protein-deficient rats there is no obvious increase in total metabolism or urinary N. Thus, neither the increase in oxygen consumption nor in urinary N are obligatory parts of the metabolic response to trauma and are clearly dependent on an adequate nutritional status.

Further studies by Campbell & Cuthbertson (1967) showed that these two factors could also be modified by increasing the environmental temperature. The response

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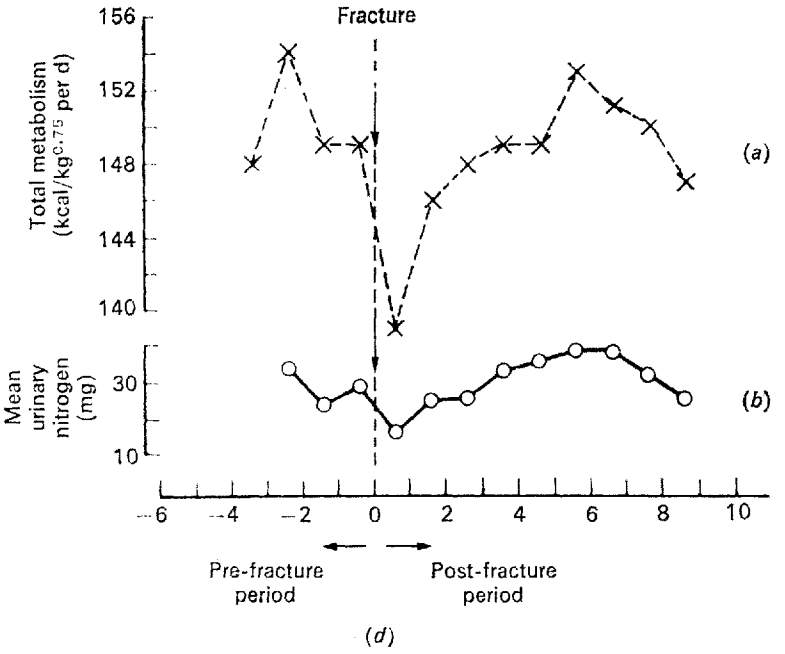

Fig. 5. Total metabolism (*a*) and urinary nitrogen (*b*) in protein-deficient rats subjected to fracture (Cairnie *et al.* 1957). Reproduced by permission of the *British Journal of Experimental Pathology*.

already described above in rats was observed at an environmental temperature of 20° but no significant increase in total metabolism or urinary N occurred after fracture when the animals were housed at 30°. The effect of the higher environmental temperature appears to be an abolition of the need to catabolize extra protein and similar findings have been reported in studies of the human (Barr, Birke, Liljedahl & Plantin, 1968; Tilstone & Cuthbertson, 1970).

In addition to the severity of the stress and the nutritional status of the patient, hormonal factors may also influence this aspect of the metabolic response to trauma. The most obvious relationship would be with alterations in thyroid activity, but early reports were unable to detect significant changes (Cope *et al.* 1953; Engstrom & Markardt, 1955). Recently, using modern techniques, Kirby & Johnston (1971) have demonstrated raised levels of free thyroxine in the plasma during the period when resting oxygen consumption and protein catabolism increase, and it has been shown by Miksche & Caldwell (1968) that thyroidectomy abolishes the energy response to fracture in rats. Hypophysectomy also prevents the rise in resting, oxygen consumption in humans (Clark, Rabelo & Kinney, 1961) (Fig. 6). In uncomplicated convalescence the basal metabolic expenditure decreases but is capable of responding to infection and secondary fracture. The hormonal mediators have not yet been precisely identified nor is the mode of action known. Whether or not there is a permissive action has not been established, nor is it clear whether the action is direct on energy expenditure or via the influence of protein metabolism.

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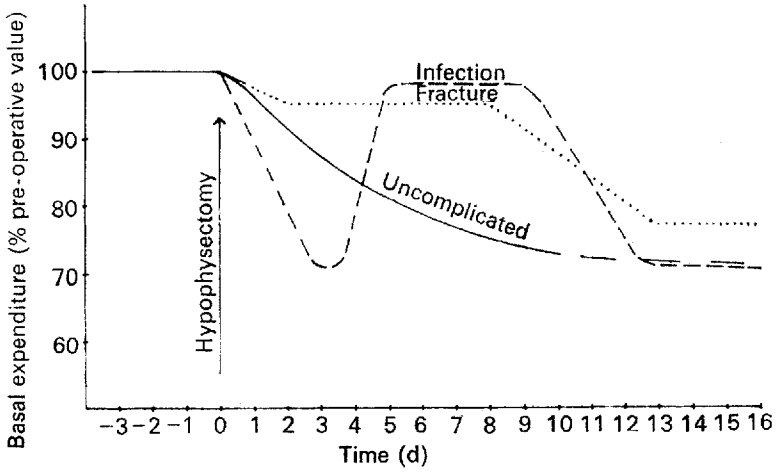

Fig. 6. Basal metabolic expenditure of patients after hypophysectomy (Clark *et al.* 1961).

Energy expenditure of the post-operative patient

Before 1960 no accurate information was available on the caloric expenditure of the patient after any form of injury. The belief current at that time was that the total energy needs of the injured were increased two–threefold. This was based on the known fact that resting metabolic expenditure was raised and also on the fact that high calorie and N intakes were unable to restore the patient to zero N balance.

Most of the information now available has come from the laboratories of Dr John M. Kinney. Using a method of indirect calorimetry (Kinney, 1960), he and his

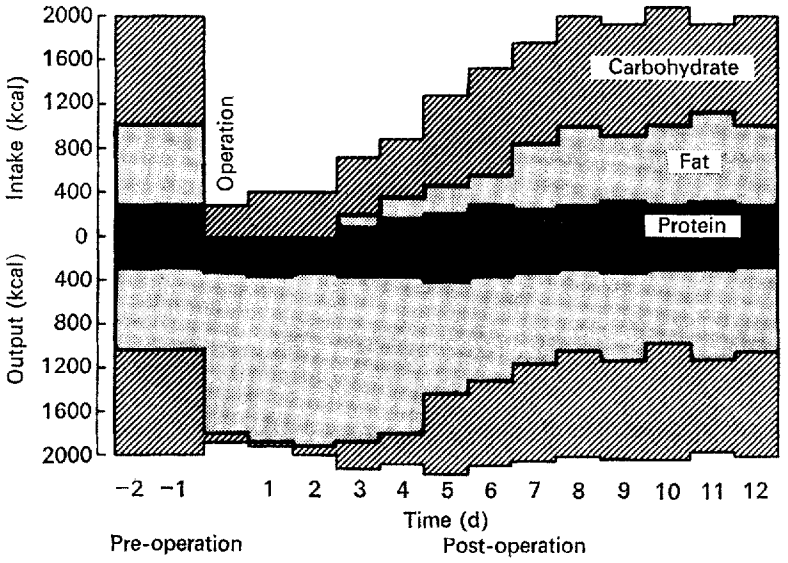

Fig. 7. Schema of energy exchange of patients after moderate operative trauma, showing utilization of fat, protein and carbohydrate (Kinney, 1960).

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co-workers have obtained a reliable estimate of daily caloric expenditure. Patients in hospital awaiting operation have a total caloric expenditure of 2000–2500 kcal/d, and in the first 2 or 3 d after operation there may be a slight decrease, but even in the days following this the output is rarely increased above 2500 kcal/d (Fig. 7). This finding did not conflict with the information already available on resting metabolic expenditure. Kinney also found increases in resting metabolic expenditure, but these were offset by the decrease in specific dynamic activity and muscular activity associated with the low caloric intake and the decreased mobility of the post-operative patient.

Injuries of greater severity such as multiple fractures were shown not to have significantly greater total caloric expenditure although the resting metabolic expenditure may be increased to 120–125% of normal. These findings relate to uncomplicated trauma. When sepsis supervenes, then considerable increases may occur in energy expenditure (Abbott & Albertsen, 1963; Roe, 1966).

These studies also reveal a change in the utilization of carbohydrate and fat in the immediate post-operative period. Immediately after operation the major source of calories appears to be fat and carbohydrate disappears almost completely from the expenditure (Fig. 8). During this time there is also a period of decreased glucose tolerance (Howard, 1955) and when the injury is more severe hyperglycaemia and glycosuria may be present. At the same time plasma insulin levels are also increased (Johnston, 1967), the usual duration of these changes after abdominal surgery being 3–4 d.

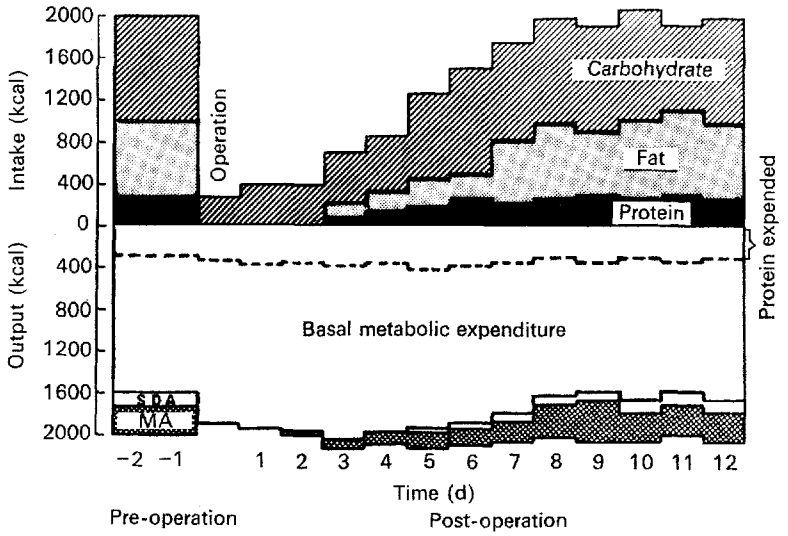

Fig. 8. Schema of energy exchange of patients after moderate operative trauma, showing increase in basal metabolic expenditure, decrease in specific dynamic activity (SDA) and muscular activity (MA) (Kinney, 1960).

However, this does not appear to interfere with the utilization of administered glucose. Measurement of glucose turnover and oxidation rates by the use of [^{14}C]-

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glucose in the post-operative period, however, showed no significant change (Long, 1966).

Nutrition of the post-operative patient

From the studies of Kinney (1960) and also from the work of Abbott & Albertsen (1963) and Dudley (1968) it became obvious that large caloric intakes are unnecessary in the post-operative state. Indeed, if replacement of expenditure were all that was required the caloric requirements would in many cases be less than those of a normal active individual. However, one of the differences between post-operative metabolism and starvation is that exact replacement of the expenditure in calories and N does not restore normal balance. It appears that to achieve a zero balance of N the caloric intake must be higher than the expenditure. Intraduodenal administration of a diet designed to compensate for the losses of patients having moderate operative trauma (30 kcal/kg per d) were unable to achieve zero N balance (J. T. Hindmarsh, personal communication). Calorie intakes of 45–55 kcal/kg per d, usually given by an intravenous route, are necessary to achieve this (Johnston, Marino & Stevens, 1966). This would suggest that the excess protein catabolism after trauma is not the response to an energy deficit and that the effect of high calorie intake is to induce an increase in protein retention without altering endogenous breakdown. It is also evident that the nutritional requirements of the patient after operation do not bear the same relationship to one another as before operation and further study is necessary to define this more clearly. Despite this gap in our knowledge common experience finds that the optimum levels of intake are 35–45 kcal/kg per d and 0·2 g N/kg per d.

The impression must not be gained that early feeding is advocated here in the post-operative patient. The majority of patients are endowed adequately with sufficient nutritional reserves to withstand the 3–5 d partial starvation which invariably follows operation. It is not therefore necessary to provide calories and N routinely either by intravenous or tube feeding. In most hospitals circumstances which necessitate these methods are found only in those patients requiring admission to intensive care facilities.

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The effect of environmental temperature on the metabolism and nutrition of burned patients

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The nutrition of patients with extensive burns usually requires daily intakes of fats, proteins and carbohydrates with a total calorific value in excess of 4000 kcal together with supranormal amounts of water and various ions, mainly sodium. These calorie requirements by a patient confined to bed are approximately those required by a normal adult male performing hard manual work. Recognition of the reasons for these large calorie requirements arose from the studies of Caldwell, Osterholm, Sower & Moyer (1959) with burned rats, who showed that these animals had a high rate of evaporation of water from the surface of the burned area. As the evaporation of water is an energy-consuming process, requiring 580 kcal/l, a burned patient losing 86 ml of water/h by evaporation from the burned surface requires 1200 kcal of energy/d for this process alone.

Precise measurements of the rate of evaporation of water from the burned surface have been made by Harrison, Moncrief, Duckett & Mason (1964), Roe & Kinney (1964), Barr, Birke, Liljedahl & Plantin (1968) and Zawacki, Spitzer, Mason & Johns (1970) using scales with a sensitivity of about 2 g. A change in body-weight over a period of 30 min indicated the total water loss from the body. In patients with burns of moderate severity between one-quarter and one-third of this water loss is from the lungs, the remainder from the burned area. In patients with very extensive burns the respiratory loss of water is only a small proportion of the total loss. Average values for the rate of water loss by evaporation from numerous burned patients with burns of different severity are shown in Table 1. The energy requirements for this evaporation of water are also shown and may be compared with the evaporative water loss in a normal person (mainly via the respiratory tract) and the energy production by a normal 70 kg male in the basal and moderately active states.

At the time of these increased requirements for calories for the evaporation of water, the patients showed substantially increased metabolic rates (see Davies & Liljedahl, 1971). In patients with burns of about 25% of the body surface the metabolic rate was about 50% above normal during the first 2 weeks after burning

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