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## NUTRITION AFTER INJURY

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### Chairman's opening remarks

By D. P. CUTHBERTSON, *Department of Pathological Biochemistry, University of Glasgow and Royal Infirmary, Glasgow*

Physical injury results in damage to and loss of body protein, both at the site of injury and, also, as part of a general catabolic response to injury which is related both to the nature and severity of the injury and to the level of dispensable protein reserve. In addition, through inaction, there results a loss of tissue protein – mainly muscle – through disuse atrophy. Apart from the shattering of bone in fractures, the major early and obvious disturbance is nevertheless in heat production and protein metabolism.

An early feature of the general response to severe injury is hyperglycaemia and glycosuria coupled with a short period of depressed heat production ('ebb' period) frequently with oliguria lasting 24 h or so, followed later by increasing outputs of nitrogen, sulphur, phosphorus, potassium, magnesium, zinc and creatine in the urine – the 'flow' period – reaching a peak within the first 10 d and constituting a marked loss of tissue substance. It is generally accompanied by a slight rise in body temperature. There is also increased heat production paralleling these changes in the adequately nourished and, in the first 10 d or so, this increment is apparently derived from increased oxidation of protein. This is all part of a generalized inflammatory response and involves both the injured area which is undergoing continuous change and the dispensable and labile reserves of the body generally.

Mechanical traumatic agents may also damage cell membranes and disturb spatial arrangements of enzymes and their substrates at the site of injury. In burns there can be a marked loss of protein in the exudate from the weeping surface. In burns also there frequently develops an anaemia of a rather intractable type. Blockage of blood supply to a protein of the tissues may cause an infarct in which cells are deprived of oxygen and the supply of other substances that are necessary for the balance of synthesis and breakdown. The changes in plasma proteins which occur at normal environmental temperature are reduced at 30–32°.

In otherwise reasonably well-nourished individuals there is probably no immediate need to worry about an initial low appetite following injury provided the inability to ingest, digest or absorb sufficient food is not likely to be longer than a few

days, otherwise recovery may be endangered. Those previously well nourished appear to have the most labile protein reserve to catabolize.

Following this 'flow' period of increased general catabolism with local anabolism in the healing area there is, in those who survive, a period of continuous or discontinuous anabolism depending on the course of the illness.

Recent observations in the USA and Sweden and by several of us in this country demonstrate that, at a higher environmental temperature (around 30–32°) and in relatively low humidity conditions, the charge on the organism to catabolize its reserves to supply energy for maintenance and healing is reduced, and intake is not appreciably diminished at the higher temperature (Caldwell, 1962, 1970; Barr, Birke, Liljedahl & Plantin, 1968; Cuthbertson, Smith & Tilstone, 1968). The changes in serum proteins are also reduced at this higher temperature (Cuthbertson, Tilstone & Green, 1969; Ballantyne, Cuthbertson, Fleck & Tilstone, 1969; Davies, 1970).

Nutrition begins with the adequate replacement of the body fluid loss, the precise nature of the replacement being dictated by the nature of the loss. Everything should then be done to encourage the patient to eat as much as he can of a good mixed diet of sufficient energy concentration and amount to avoid dietary protein or tissue protein being used for heat production. In the case of a fracture of the femur or both bones of the leg about 1 kg of protein may in 10 d be lost, in a muscle wound equivalent to four hands in volume (Medical Research Council, 1951) about 1.5 kg protein; in a 35% burn involving a considerable part of full thickness skin about 1.5 kg, and after a gastrectomy about 0.75 kg (Ministry of Health, 1964).

Because carbohydrate stores are the most readily available source of utilizable substrate from which energy can be derived, the stores in the liver of glycogen are rapidly utilized but these do not last more than a few hours unless the carbohydrate is replaced. The initial hyperglycaemia and glycosuria are due to increased sympathetic and adrenal medullary outflow: occurring later it may indicate a temporary resistance to insulin. You will hear from our speakers about the relative advantages of different energy sources including fat and of amino acid, mineral and vitamin supply.

Where there is difficulty in the ingestion of food but there is adequate digestion and absorption, I would recommend the introduction of homogenized whole diets by tube into the digestive tract, if necessary by the intraduodenal route. Where, however, the capacity to digest and or absorb or both is defective to the extent of imperilling speedy recuperation or life, then parenteral nutrition is indicated.

Supposing a net retention of protein be 20 g/d it will take 2–3 months to return to normal – where this is still possible – in the kind of cases of the injuries I have referred to earlier.

I would remind members that vitamin C is closely related to protein metabolism and that in the vicinity of wounds there is an accumulation of ascorbic acid. There is some evidence of an increased turnover of vitamin B<sub>12</sub> following injury. Finally, I would recommend that apart from head and chest injuries, and as soon as the shock period is over, severely injured patients should be put in a warm environment (28–32°) with relative humidity not exceeding 45% (Caldwell, 1962, 1970; Cuthbert-

son *et al.* 1968; Cuthbertson & Tilstone, 1968; Cuthbertson, 1970; Tilstone & Cuthbertson, 1970). Drier conditions (20% relative humidity) are indicated in burns (Barr *et al.* 1968).

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### Protein metabolism after injury

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Two authoritative reviews of various aspects of trauma have appeared recently (Porter & Knight, 1970; Sevitt & Stoner, 1970). For this reason it seemed apposite to consider mainly the nutritional aspects of the effects of injury on protein metabolism.

The first observations, which demonstrated altered metabolism after trauma, were made on humans (patients in Glasgow Royal Infirmary) by Sir David Cuthbertson (Cuthbertson, 1929). In a series of papers (see Cuthbertson, 1964), he demonstrated that the increased urinary excretion of nitrogen, sulphur and phosphorus was maximal between the 3rd and 8th days after injury and that there were parallel increases in body temperature, pulse rate and oxygen consumption. He also found that the daily loss of N could be up to 23 g after severe injury (Cuthbertson, 1930, 1932). It now seems to be generally accepted that the magnitude of the response is related direct to the degree of trauma, although environmental temperature (Caldwell, 1970; Tilstone & Cuthbertson, 1970; Davies, Liljedahl & Birke, 1969) and previous diet (Munro & Cuthbertson, 1943; Fleck & Munro, 1963) modify the response. Human males after a moderate injury such as closed fracture of the tibia and fibula have a net loss of about 5 g N/d for several days (Cuthbertson, unpublished observations). These patients before injury were generally normal fit men and were maintained in an environmental temperature of