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Nutritional Support in the Management of Critically Ill Patients in Surgical Intensive Care

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Nutritional support is given to many patients in surgical intensive care after major trauma and serious sepsis but rarely after major elective surgery. We have quantified the changes in body composition that occur in these patients and have found that serious losses of body protein still occur after trauma and sepsis despite nutritional support. Correct nutritional management of critically ill patients in surgical intensive care depends on an understanding of the underlying physiology, drainage of sepsis, a high standard of general intensive care measures, and appreciation of sound principles of administration of intravenous nutrition.

Seriously ill surgical patients frequently require a period of intensive care after major elective surgery, severe trauma, or serious sepsis. During this time they often require respiratory and inotropic support and are at high risk of other organ failures. Under such circumstances, there is rapid loss of considerable amounts of body protein which may lead to impairment of wound healing, muscle function, and other organ function, and so lengthen the period of intensive care and of convalescence. Nutritional support, commonly as intravenous nutrition (IVN), is given to many such patients but the efficacy of this practice in reducing net protein loss or in improving clinical outcome remains uncertain.

Accepted clinical practice varies widely, with some clinicians using IVN liberally and others subscribing to the belief that such expensive therapy is ineffective and unwarranted. Considerable debate still remains as to the amount of nonprotein energy required, the place of glucose and fat, the amount of nitrogen required, the efficacy of branched chain amino acids, and even the practicalities of providing IVN to such patients. In this article, the physiology of the metabolic changes seen in response to critical surgical illness and intensive care treatments is examined together with the effects of current clinical practice and the principles and practical aspects of providing nutritional support. In view of the failure of current nutritional support to control the catabolic state, further clinical research is required on intermediary metabolism in critically ill patients in intensive care as well as the effectiveness of alternative nutritional support and of altering the hormonal milieu in preventing net protein loss.

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Metabolic Effects of Surgery, Trauma, Sepsis, and Intensive Care Treatments

Critical surgical illness results in a series of metabolic and body compositional changes. In response to operation, injury, or infection, there is increased production of many hormones including glucagon, insulin, cortisol, catecholamines, aldosterone, and vasopressin with resultant profound effects both on energy and protein metabolism and on the metabolism of salt and water [1].

Energy Metabolism

Resting energy expenditure (REE) rises by 5–50% after major trauma [2–4] and serious sepsis [5], and may remain elevated for a considerable period. In contrast, we have found that there is approximately an 8% fall in REE by 1 week after major uncomplicated elective surgery. In sepsis and after trauma, energy needs are met predominantly by the oxidation of fat [6, 7] and also from glucose derived by increased gluconeogenesis [8] from body protein. Total body glycogen stores are small (approximately 500 g) and are unlikely to provide a significant contribution to energy supply after the first day or two.

Protein Metabolism

Critically ill patients in surgical intensive care all have high urinary urea nitrogen loss—often 15–20 g of urea nitrogen per day being found in the absence of nutritional support [9]. Other sources of nitrogen such as creatinine and ammonia also contribute, such that urea accounts for about 75% of total urinary nitrogen. Unlike the situation in simple partial starvation (and perhaps in patients within a few days of major elective surgery), the starving patient with sepsis or after trauma does not have an adaptive reduction in urinary nitrogen loss and instead rapidly becomes severely depleted of body protein. In addition, the efficient, almost complete, suppression of gluconeogenesis from protein seen after glucose infusion in nonseptic

starvation is not seen in septic patients and larger amounts of glucose are required to suppress glucose production even partially [8]. The protein-sparing effect of fat infusion in sepsis is at present unknown. Despite considerable recent work in this area [10–14], the superiority of branched chain amino acid solutions over conventional balanced amino acid solutions in moderating net protein loss remains unproven. The applicability and effectiveness of other clinically feasible approaches to reducing energy expenditure and net protein loss in such patients including epidural anesthesia, high-dose narcotic infusion, and adrenergic or other hormonal blockade are presently uncertain although some very recent work from this department suggests that endogenous glucose production may be lessened by epidural anesthesia in surgical patients.

Salt and Water Metabolism

In addition to the changes in energy and protein metabolism, there are (in septic and injured patients) profound changes in the metabolism of salt and water and in the volume and composition of body fluid compartments. The presence of sepsis, profound shock, and tissue damage leads to impaired capillary permeability [15–17] with resultant obligatory expansion of the interstitial fluid space [18] during treatment of shock.

Clinically then, the surgical patient in intensive care has rapid wasting of muscle and, to a lesser extent, fat which is initially not seen because of an expanding layer of extracellular water.

Intensive Care Treatments

If the patient needs ventilatory support, he/she also necessarily gets bed rest, opiates, sedatives, and often muscle relaxants (curarization). The resultant immobility and perhaps even curarization (denervation) itself may further increase the loss of body protein. Other intensive care treatments such as inotropic agents, particularly epinephrine [19–21], hemodialysis [22], and steroids [23] may all also contribute to the catabolic process.

Functional Effects of Protein Catabolism

Recent work in this department [24] has shown that, in association with a loss of about 35% of body protein in surgical patients, there is impairment of skeletal muscle strength and endurance, reduced respiratory muscle strength, low levels of plasma proteins, and impaired wound healing. Clearly, these functional aspects are of crucial importance in the recovery from critical surgical illness.

Surgical Patients in Intensive Care and the Effects of Currently Accepted Clinical Practice on Body Composition

Surgical patients in intensive care fall into 3 main groups: postoperative major elective surgery, major trauma, and serious sepsis.

Major Elective Surgery

It is not our practice to admit patients to intensive care electively after clean major elective surgery (e.g., pancreaticoduodenectomy) and such patients will be discussed in this article only to highlight differences between them and the patient in intensive care after major trauma or with sepsis. Most patients who are admitted to intensive care after major elective surgery are not problematic from a nutritional point of view and receive nutritional support after transfer to a surgical ward according to standard accepted clinical guidelines [25]. Most such patients, in fact, do not receive postoperative IVN.

Table 1. Early body composition changes in patients after major elective surgery, severe blunt trauma, and serious sepsis

	Major surgery	Trauma	Sepsis
Number of patients	8	10	8
Protein index[a]	0.76 ± 0.05	0.92 ± 0.035	1.12 ± 0.03
Energy intake (Kcal)	–[b]	1,691 ± 141	2,750 ± 150
Protein intake (g)	–[b]	88 ± 13	127 ± 16
Study interval (days)	14	10	10
Changes in body composition			
Weight (kg)	−5.3 ± 0.9	−4.2 ± 1.2	−6.2 ± 2.9
Water (kg)	−3.8 ± 1.3	−3.3 ± 1.4	−6.8 ± 2.6
Protein (kg)	−0.6 ± 0.35	−1.1 ± 0.2	−1.5 ± 0.3
Fat (kg)	−0.7 ± 1.2	+0.2 ± 0.6	+2.2 ± 0.8

[a] All values as mean ± SEM.
[b] Elective surgery patients received no specific postoperative nutritional support but were allowed oral feeding as soon as clinically appropriate.

Recently, a longitudinal study of the changes in body composition that occur after major surgery has been conducted in this department. The results from 8 patients are presented in Table 1. Each patient gave informed consent for the study, which had the approval of the Auckland Hospital Ethicals Committee. The patients underwent measurement of total body water (TBW), protein (TBP) [26], and fat (TBF) [27] by tritiated water dilution and in vivo neutron activation analysis (IVNAA) [28] on the day prior to operation and again 14 days postoperatively. The accuracy and precision of these methods for the measurement of changes in these components of body composition has recently been established. [29]. For each patient, a protein index (PI), being an index of protein depletion [30], was calculated as the ratio of measured to predicted TBP and the mean preoperative value of PI for the group of 8 patients is shown in Table 1. Predicted TBP was obtained from published regression equations [31] from another center involving age, sex, and height (but not weight). Of the 8 operations, there were 3 total gastrectomies with intraabdominal esophagojejunal anastomosis, 2 pancreaticoduodenectomies, 2 mucosal proctectomies with ileo-anal pouch formation and ileostomy, and 1 panproctocolectomy. No patient had IVN preoperatively or during the 14-day postoperative period and none developed a serious complication.

As a group, these patients were depleted of approximately 24% of TBP immediately preoperatively (mean PI 0.76, 95% confidence limits 0.64–0.88). In the first 2 weeks postoperatively, there was a loss of TBP [mean loss 0.64 ± 0.35 (SE) kg, ns] or 5% of TBP, and at this time the patients were approximately 29% depleted of TBP (mean PI 0.71, 95% confidence limits 0.63–0.79). There was also a loss of fat (0.67 ± 1.23 kg, ns) in keeping with a postoperative caloric deficit met by the

oxidation of fat as well as protein. Weight losses were moderate (mean weight loss 5.3 ± 0.9 kg, $p = 0.001$) and were predominantly due to losses of body water (mean loss 3.8 ± 1.3 kg, $p = 0.02$).

Thus, our small study suggests that patients undergoing major elective surgery are frequently moderately depleted of body protein preoperatively (about 25%) but, even without nutritional support, lose only approximately another 5% of body protein in the first 2 weeks following surgery.

Major Trauma

Major trauma is the single most common reason for intensive care admission in our institution. We have reviewed the data on the 292 trauma patients (out of 962 total intensive care admissions) admitted to intensive care in 1984. In contrast to most North American experience, 90% of our patients are suffering from blunt trauma and only 10% have knife or gunshot wounds. These trauma patients are young (median age 19), seriously injured (187 of the 292 patients admitted had Injury Severity Scores (ISS) [32] of 25 or above and 46 had ISS of 41 or above) and were previously in excellent health. Fewer than 10% (25/292) in total and only 50% (8/15) of patients with severe abdominal injury [Abbreviated Injury Scale (AIS), 5] [33] required IVN. It is noteworthy that in our experience about 15% (12/77) of surviving patients with severe brain injury (AIS, 5) required IVN. Those patients who need IVN after trauma usually require prolonged intensive care, the median intensive care stay being 22 days for the 25 patients in 1984.

We have recently completed a study of the changes in body composition seen in these very severely injured patients [34] and the results are shown in Table 1. These 10 patients were chosen for study on the basis that they were likely to require intensive care for more than 10 days after reaching hemodynamic stability. They were young (median age 23) and all had severe blunt injury (ISS range 16–57, median 34). All were ventilator dependent throughout most of the 10-day study period and all survived to leave the hospital. After informed consent had been obtained from the patients' next-of-kin, measurements were made of TBP, TBW [35], and TBF. PI was also calculated for each patient. The first measurement was on post-injury day 4–12 (median 6), the second measurement 10 days later. IVNAA was performed in duplicate on each occasion to improve the precision of the protein and fat measurements [26]. IVN was given to 7 patients, 4 of whom received small amounts of additional enteral feeding via nasogastric tube. The 3 other patients had enteral feeding alone. Mean daily nutritional intakes for the patients as a group were 1,691 ± 141 nonprotein Kcal and 88 ± 13 g of protein. This prescription would have given [9] approximately 2,000 nonprotein Kcal and 100 g of protein to 70 kg Reference Man [36].

As can be seen from Table 1, the patients were probably slightly protein depleted at the time of the first body composition study (mean PI 0.92, 95% confidence limits 0.84 – 1.00) and lost a mean of 1.1 ± 0.2 kg ($p = 0.0004$) of protein or 11% of TBP to end up approximately 19% protein depleted by the time of the second assessment 10 days later (mean PI 0.81, 95% confidence limits 0.73 – 0.89). There was a small insignificant gain of fat for the group of patients (mean gain 0.2 ± 0.6 kg, ns). An estimate of the daily total energy expenditure (TEE) over the 10-day period was made for each patient from the nutritional intakes and the body composition data. In view of the inability of our body composition method [27] to distinguish changes in glycogen (with a caloric equivalent of 4 Kcal/g) from changes in fat (with a caloric equivalent of 9 Kcal/g), we have calculated 2 estimates of TEE for the group. They suggest that TEE lay between 2,286 ± 487 and 2,410 ± 255 Kcal or approximately 2,700–2,850 Kcal for 70 kg Reference Man [9, 36]. Most of the weight loss (mean 4.2 ± 1.2 kg, $p = 0.006$) was due to loss of water (mean 3.3 ± 1.4 kg, $p = 0.04$).

Thus, our study suggests that patients with major trauma in intensive care are not depleted initially but, despite moderate nutritional support, lose a large amount of body protein (about 1% of TBP per day) in the first 2 weeks after trauma. The possibility that higher nutritional intakes (sufficient to increase body fat) may reduce this loss requires investigation.

Serious Sepsis

The other large group of patients in whom nutritional problems are seen are postoperative and have sepsis as a major contributing factor to their requirement for intensive care. They are often old and some may have preexisting nutritional depletion as a result of cancer, recent major trauma, multiple operations, or semistarvation. They have serious acute diseases such as fecal peritonitis, infarcted small bowel, toxic megacolon, pancreatic abscess, and the like and they consume intensive care resources at a rate vastly disproportionate to their numbers.

In order to establish the changes in body composition found during currently accepted clinical practice, we measured TBW, TBP, and TBF in 8 postoperative patients in intensive care with serious sepsis before and after 10 days of IVN [9]. Mean daily nutritional intakes for the patients as a group were 2,750 ± 150 nonprotein Kcal and 147 ± 16 g of amino acid. This prescription would have given approximately 2,450 nonprotein Kcal and 128 g of protein to 70 kg Reference Man [36]. All patients had recovered from the septic shock syndrome but were still ventilator dependent and most were still receiving inotrope infusions at the start of IVN. Six patients survived and left the hospital. The results are shown in Table 1. These patients were, as a group, not depleted of TBP at the time of the first body composition assessment (mean PI 1.12 ± 0.03) but lost a mean of 1.5 ± 0.3 kg ($p = 0.001$) of protein or 12.5% of TBP over the 10-day period. There was a significant gain of fat for the group of patients (mean gain 2.2 ± 0.8 kg, $p = 0.026$). An estimate of the daily total energy expenditure (TEE) over the 10-day period was made for each patient from the nutritional intakes and the body composition data and the best estimate of TEE for the group was between 2,027 ± 468 and 3,265 ± 325 Kcal or approximately 1,800-2,900 Kcal for 70 kg Reference Man [36]. Again, the weight loss (mean 6.2 ± 2.9 kg, ns) was predominantly due to loss of water (mean 6.8 ± 2.6 kg, $p = 0.036$) with several patients losing more than 10 kg of water.

Clearly, large losses of TBP still occur in patients with sepsis in intensive care despite aggressive nutritional support sufficient to result in a gain in TBF. It is particularly in this group of patients that the effects of alternative nutritional substrates and hormonal manipulation must be assessed.

Table 2. Total body fat (TBF), measured by IVNAA and by skinfold anthropometry, together with estimates of total body water prior to illness $(TBW)_{pi}$ and measured TBW in intensive care patients.

Patient	Weight (kg)	TBF (IVNAA) (kg)	TBF (Anthropometry) (kg)	TBW_{pi} (kg)	TBW (kg)
R1	79.4	24.0	21.7	43.3	39.9
F1	81.5	14.0	23.4	39.9	52.0
F2	79.4	13.5	26.3	39.9	50.1
M1	83.5	15.6	26.7	41.6	50.8
B1	68.6	9.8	19.3	35.3	44.0
B2	65.0	12.3	16.4	35.3	39.2
H1	72.9	17.4	30.8	32.0	43.1
H2	59.7	20.7	22.5	32.0	29.1
N1	89.9	22.0	29.6	46.5	50.8
Mean		16.6	24.1	38.4	44.3
SE		1.6	1.6	1.7	2.5
Differences					
Mean			7.5		5.9
SE			1.8 (p = 0.003)		1.9 (p = 0.016)
Mean				1.7	
SE				0.6 (p = 0.026)	

Lessons Learned from Body Composition Research in Patients in Intensive Care

Weight Change is Nearly all Water Change

In critically ill surgical patients weight changes cannot be used as a form of nutritional assessment since they are predominantly due to large changes in TBW greatly in excess of changes in protein and fat and often in the opposite direction. In order to quantify the extent of the fluid excess for each of the 2 groups of patients (trauma and sepsis) in intensive care shown in Table 1, we have calculated an estimate of their TBW prior to illness (TBW_{pi}) [35] assuming that there had been no loss of TBP over the few days prior to the first measurement of TBP (according to the following regression equation [35] based on data obtained from 68 normals): $TBW_{pi} = 2.56 \times TBP + 8.95$ ($r = 0.925$, cv = 7.1%). As a group, the trauma patients had an estimated excess of 7.6 ± 1.8 liters of water at the time of the first body composition study (median 6 days after injury) and 4.4 ± 1.2 liters above their TBW_{pi} 10 days later. The septic patients had a mean excess of approximately 13.8 ± 2.0 liters of water at the beginning of IVN and 7.1 ± 3.7 liters 10 days later. Clearly, some patients lost very large volumes of water (10–15 liters) over this interval. Thus, for both groups of patients, the observed weight changes were predominantly due to changes in body water.

Skinfold Anthropometry Cannot Accurately Measure Fat

We have shown that skinfold anthropometry is an inaccurate method of assessment of TBF in patients presenting for nutritional support in surgical wards [27]. We found that skinfold anthropometry underestimated body fat by about 3 kg (or 19% of TBF) in those patients. Because of large changes in weight (water), the use of skinfold anthropometry is even more inappropriate in the patient in intensive care. We measured TBF by the IVNAA and tritium dilution method [26, 35] and derived TBF from skinfold anthropometry [37] in critically ill patients in intensive care. Nine studies were performed on 6 patients and the results are shown in Table 2. Shown also are estimates of the excess body water on each occasion by the method outlined above. The highly significant (p = 0.003) mean difference between TBF measured by the IVNAA technique (16.6 ± 1.6 kg) and that derived from skinfold anthropometry (24.1 ± 1.6 kg) was 7.5 ± 1.8 kg. Furthermore, this was significantly (p = 0.026) more than the mean excess TBW of 5.9 ± 1.9 kg for this group of patients implying that the discrepancy between the methods for the measurement of fat is due both to weight gain and to "wet" skinfolds.

Nitrogen Balance May Be Grossly Misleading

We have recently shown that nitrogen balance has serious deficiences and hidden imprecision [38]. We measured changes in total body nitrogen by IVNAA and by careful ward nitrogen balance over 44 two-week intervals in 40 postoperative patients in the surgical ward receiving IVN as their only nutritional intake. The nitrogen balance technique suggested that every patient was in positive nitrogen balance but underestimated true nitrogen losses by a mean of 77 ± 25 (SE) g over the 14-day period or about 5 g per day. Furthermore, the true precision of nitrogen balance is unknown (unlike IVNAA) since the nitrogen losses that account for the systematic inaccuracy seen in a group of patients are not themselves measured. In patients in intensive care, the errors of nitrogen balance (usually due to unmeasured or immeasurable losses of nitrogen-containing body fluids) are likely to be both more variable within patients and of greater magnitude for such patients as a group.

Clinical and Nutritional Management of Critically Ill Surgical Patients

Control of Sepsis

The single most useful "nutritional therapy" in patients in intensive care is the drainage, when present, of a septic focus. It is salutary to reflect that when sepsis develops late after

trauma with its catastrophic effect on metabolism [8], cardiopulmonary function, and mortality, it is often as a result of early surgical failure to provide appropriate or adequate debridement, drainage, or defunctioning. The majority of patients with abdominal sepsis do not develop abscesses if the initial surgery has been definitive. We have found, as have others [39, 40], that nosocomial pneumonia is a common cause of a persistent septic state in such patients [9] and that, in patients with persistent respiratory failure, cultures obtained by fiberoptic bronchoscopy will often identify a responsible gram-negative bacillus not found on conventional tracheal aspiration. Nevertheless, as such organ failure is known also to be a marker of a remote septic focus [40], an intraabdominal septic focus should always be suspected. We have found computed tomographic (CT) scanning to be of great clinical value in confirming the presence of an abscess which may be amenable to percutaneous drainage under CT or ultrasound control. Multiple or complex abscesses are better managed by direct surgical drainage [41]. Failure to demonstrate a drainable collection by CT scan is usually very reassuring but can occasionally be misleading [39] and repeat laparotomy may be required. The disturbingly high early septic mortality in complex gastrointestinal fistulas [41] and pancreatic abscesses may be reduced by the use of the "open abdomen" with daily laparotomy, often in the intensive care unit, until effective drainage has been established.

General Intensive Care Management

It is obviously important that the critically ill patient receive a high standard of intensive care because without this, the addition of nutritional support is likely to produce complications itself. In our own practice [9], all aspects of intensive care management including respiratory care, fluid and cardiovascular management, analgesia, nutritional support, microbiological surveillance, and instrumentation are the responsibility of full-time intensive care specialists. There are a number of aspects of management with potentially relevant metabolic consequences on which we place particular emphasis.

Respiratory Management. Because of the risk of barotrauma [42] and possible unfavorable effects on salt and water metabolism [43] and intracranial pressure (ICP) [44], we avoid high positive end expiratory pressure (PEEP) (> 12–15 cm H_2O) unless indicated for severe frothing pulmonary edema or for refractory hypoxemia despite $F_IO_2 > 0.8$.

Although the effects of curarization (denervation) itself on muscle loss are unknown, we believe that they are more likely to be harmful. Therefore, we do not routinely use curarization in the management of the ventilated patient except for patients with severe head injury in whom curarization itself can lower ICP [45] and ameliorate rises in ICP consequent on coughing during tracheal suctioning or against the ventilator. In all other patients, relaxants are given for facilitation of IPPV only if respiratory failure is severe (e.g., $F_IO_2 > 0.6$, PEEP > 10 cm H_2O) or to facilitate cooling in hyperpyrexia.

Because of possible favorable effects on salt and water metabolism and respiratory muscle function that may result from early use of intermittent mandatory ventilation (IMV) [46], we dominantly use this mode of ventilation, often in association with continuous positive airway pressure (CPAP) and thoracic epidural anesthesia.

Cardiovascular Management. We believe that the prevention of renal failure is of paramount importance in the survival of the critically ill patient in surgical intensive care and we are relatively less concerned by the appearance of peripheral edema, which can sometimes be massive [18], during resuscitation from shock when guided by appropriate hemodynamic monitoring. We frequently use dopamine in low doses (2–5 μg/kg per min) in such patients. When further inotropic support is required, we increase dopamine to 10 μg/kg per min before adding epinephrine. Because of the unfavorable metabolic effects of this drug, it is weaned off as soon as possible. Diuretics (furosemide) are used infrequently; the removal of excess salt and water after resuscitation is complete being accomplished by sodium restruction and judicious albumin infusion [9].

Prevention of Opportunistic Sepsis. Continued microbiological surveillance, especially of the respiratory tract, minimal use of invasive monitoring [47], and restrictions on the use of antibiotics to those for which there is a defined indication are all important in this regard.

Nutritional Support

The data in Table 1 suggest that the efficacy of nutritional support in preventing protein loss in critically ill patients with trauma and sepsis is poor; however, for ethical reasons, we have not had the opportunity to establish the effect of complete starvation in this context—indeed opportunities to demonstrate an effect of nutritional support by randomization of patients are few in the critically ill. We will review each of the following aspects of nutritional support—energy requirements, the nature of the nonprotein energy source, nitrogen requirements, the route of administration (enteral or parenteral), and the practical aspects of intravenous nutrition including vascular access and the prevention of catheter sepsis.

Energy Requirements. Energy requirements in critically ill ventilated patients are notoriously difficult to measure for technical reasons [5, 48, 49]. There may be wide variations in energy expenditure within an individual patient from day to day and at different times of the day [2], and the relationship between measured energy expenditure and that predicted on the basis of standard predictor equations is so variable [5, 50] as to make such equations almost useless as a guide to the energy requirements of the individual patient. Nevertheless, although resting energy expenditure is increased in critical illness, it is probably not as high as was previously thought and by virtue of the reduction in that component of total energy expenditure due to muscular work, total energy expenditure in critically ill patients is not greatly increased from normal—35 Kcal/kg of body weight per day is likely to be a somewhat generous allowance [2, 50]. Body weight is, of course, a rather crude index of metabolic body size, particularly in these patients who have large changes in body water as shown above and there will be few patients who will require more than about 2,500 nonprotein Kcal/day.

Table 3. Specific incompatibilities in IVN mixtures containing fat, glucose, and amino acids.

PVC bags cause leaching of plasticizer and cracking of fat emulsion.
Total divalent cation concentration > 4 mmol/l causes cracking of fat emulsion.
Total monovalent cation concentration > 150 mmol/l causes cracking of fat emulsion.
Low amino acid doses can cause cracking since cracking is pH dependent.
Bacterial filters cannot be used since fat droplets are larger than filter pore sizes.
Dopamine and albumin together cause a purple Biuret reaction.
Epinephrine is converted to pink, inactive, adrenochrome by trace elements and zinc.

Glucose, Fat, and the "One Bag" System. It is difficult to meet energy needs in these patients with glucose alone [6] without pharmacologic doses of insulin and close monitoring. Although insulin promotes clearance of glucose from plasma, it does not increase glucose oxidation [51]. Hyperglycemia, fatty liver, and increased heat production are likely to result. We have found, even in the most seriously ill patients, that equicaloric mixtures of glucose and fat can be given simply and safely and that lipemia and hyperglycemia do not occur [9]. Using this system, insulin is often not required and is not routinely given. When needed for hyperglycemia, moderate doses (40–60 units day) will suffice. Although there are some who suggest that lipid infusions can result in reticuloendothelial cell and hepatocyte lipid uptake [52] and prostaglandin-mediated hypoxemia [53], in clinical practice this must be very rare indeed.

We give 50% of the nonprotein energy as continuous fat infusion, mixed with glucose, amino acids, vitamins, trace elements, electrolytes, and certain drugs in large (up to 4.8 liters capacity) plastic (ethylene vinyl acetate, EVA) bags [54]. Although, as a matter of principle, we avoid adding drugs to the IVN solution when there is a reasonable alternative we have safely added dopamine, dobutamine, epinephrine, cimetidine, ranitidine, pirenzipine, insulin, hydrocortisone, and dexamethasone. A number of specific incompatibilities have been identified by our pharmacists as resulting either in "cracking" of the lipid emulsion or inactivation of drug additives and these are listed in Table 3. Many other potential or theoretical incompatibilities can be avoided by the correct sequence of dilution and addition of the components of the IVN bag. Amino acid solutions, together with some additives are added first, followed by dextrose solution, water, and other additives and finally fat emulsion. No additives are ever added directly to fat emulsions.

Nitrogen Requirements. In addition to the considerations discussed above pertaining to protein loss from catabolism and starvation, critically ill patients frequently lose protein from wounds, drains, and via the arterial lines. For these reasons, when giving IVN we use a high protein intake of 125–200 g/day or about 2–3 g of protein per kg of body weight. We have shown that critically ill ventilated patients have insensible water losses of about 1,200 ml/day [35] and relatively large volumes of free water (3–5 l/day) may be required to prevent azotemia during IVN when such high protein intakes are given. It may be difficult to provide these protein intakes if a high intake of free water is contraindicated by brain injury or renal impairment. Although careful fluid management has resulted in our ability to give these intakes to the great majority of patients, substantial protein loss still occurs [9].

Enteral Feeding. Because enteral feeding is much cheaper and simpler to administer in the critically ill patient than equivalent intravenous nutrition, it is always preferred if the gut is available for use. Enteral feeding is, however, not without the risk of pulmonary aspiration in critically ill patients unless the airway is protected by a cuffed endotracheal tube, and diarrhea may also limit its use. The additional obligatory sodium intake (50–100 mmol/day) in most commercially available preparations is not usually problematic.

Vascular Access for IVN. It is extremely difficult to care properly for a central venous line in the neck of a patient in intensive care and such sites are easily contaminated from the adjacent tracheostomy site, which is often a source of gram-negative bacteria. For this reason, an infraclavicular subclavian approach without tunnelling (in the vast majority of patients) or an internal jugular line which has been tunnelled subcutaneously to exit below the clavicle (in patients in whom the risk of pneumothorax is very high) are preferable. In ventilated patients, a temporary increase in inspired oxygen to 100% together with reduction in or removal of PEEP is strongly recommended to minimize the risk of pneumothorax during line insertion. This complication should have an incidence of less than 3% but can be fatal in a ventilated patient with poor lung function. A strict protocol prohibiting the use of the central line for anything but IVN together with a careful dressing technique which prevents contamination of either the skin entry site or the junction of the line to the giving set will keep catheter sepsis to a rarity. We do not use multiple lumen central venous catheters for IVN since their relative safety over single lumen catheters in this situation is not yet established. We have demonstrated that the use of such a protocol in patients in surgical wards is associated with an incidence of catheter sepsis of 2.3 episodes per 1,000 patient-days [55]. In 20 patients in intensive care with serious sepsis who had at least 10 days of IVN, there were no instances of catheter sepsis and no catheter required to be changed. We do not recommend changing these lines "as a routine."

What of the Future?

Our current research projects include a prospective, blind, randomized, isocaloric, isonitrogenous trial of branched chain, amino acid-enriched IVN versus standard IVN in critically ill patients in intensive care and the establishment of the extent to which loss of body protein can be minimized in trauma patients by nutritional manipulation.

Our body composition laboratory is being extended to include the measurement of total body potassium in a shadow shield counter which is designed to allow the measurement of critically ill patients in intensive care. In order to derive compositional entities such as total body cellular protein and to allow more accurate compartmentalization of body water [30], we are measuring tissue elemental composition in biopsy ma-

terial and muscle membrane potential difference in our sickest patients.

In association with measurements of body composition, we are carrying out various measures of clinically relevant organ function which will help to establish the functional consequences of the body composition changes. Furthermore, in this department, methods for studying intermediary metabolism [8] in critically ill patients are being used to establish guidelines that might be useful either for nutritional management or hormonal manipulation. It is here that we think that major advances are likely to occur in the near future.

Résumé

Dans les unités de soins intensifs de nombreux malades victimes d'un traumatisme grave ou en proie à une infection sévère bénéficient d'un apport nutritif important. Il n'en est pas toujours de même pour les opérés qui ont subi une intervention importante. Les auteurs ont mesuré les changements de la composition corporelle chez les patients traités dans l'unité de soins intensifs et ont ainsi constaté une perte protéique importante chez les blessés et les infectés malgré un apport nutritif important. Le traitement correct des malades dont l'état est critique et qui sont soignés dans les unités de soins intensifs repose sur l'appréciation exacte de l'état physiologique du malade, du drainage de l'infection, de la haute qualité des soins intensifs et d'une alimentation parentérale adéquate.

Resumen

El soporte nutricional es administrado a muchos pacientes en cuidado intensivo quirúrgico por trauma o sepsis grave, pero en muy raras ocasiones después de cirugía electiva mayor. Al cuantificar los cambios en la composición corporal que ocurren en pacientes en cuidado intensivo hemos hallado que serias pérdidas de proteína corporal continúan presentándose como consecuencia del trauma y la sepsis a pesar del soporte nutricional. El correcto manejo de pacientes en estado crítico en la unidad de cuidado intensivo depende de la comprensión de la fisiología involucrada, del drenaje adecuado de colecciones sépticas, de un alto estándar del cuidado intensivo, y de la observación de principios racionales para la administración de la nutrición intravenosa.

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