INTENSIVE CARE

Edited by
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Preface

A comprehensive text of intensive care would readily fill the equivalent of the *Shorter Oxford English Dictionary*. This is because the diseases treated are both numerous and varied; thus the patient can be medical, surgical, trauma or obstetric. It follows that the sum total of knowledge which needs to be available is truly encyclopaedic. This compact volume represents only a fragment of such information. The contributors were chosen because of their experience and because their methods were well-tried. The text therefore summarizes the best of current therapy and includes the controversial. The contributors come from four countries, adding an international flavour. One topic – The Recovery Room – outside the confines of intensive care has been included for two reasons. The recovery room is an important but neglected aspect of care, and it also seems important to define its relationships with the intensive care unit. It is hoped that the book will help the nurses and doctors involved in intensive care and, therefore, the patient.

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Contents

1
Intravenous feeding

HARRY LEE

The purpose of this chapter is to emphasize the practical aspects of intravenous feeding, rather than review the relevant pathophysiology. If such a therapy is to succeed, it must be capable of being practised both in specialized units and general wards which may be understaffed. In the last 20 years intravenous nutrition has advanced rapidly from being a somewhat erudite, complex form of treatment, to one that now should be considered commonplace, flexible and easy to manage. Indeed, unless it is accepted that any clinician can treat any patient on any ward at any time, the value of intravenous nutrition must be in doubt. The origins of intravenous feeding probably began following discussions and experiments by Sir Christopher Wren and Dr Robert Boyle. They experimented with ethanol and wine given intravenously to dogs, who actually survived! Fortunately, we have come a long way since then! Intravenous bolus feeding was probably first practised by Dr Latta in Scotland in the 1830s. Comprehensive parenteral feeding was first attempted by Frederich (1905) using a mixture of peptones, sugars, electrolytes and oils. Over the past century there has been a flurry of activity; first this dwelt upon the provision of electrolytes and water and then, at the turn of the century, the need to provide energy and nitrogen was recognized[1,2].

(I) AIMS AND OBJECTIVES

Intravenous nutrition must be seen as a therapy which provides complete nutritional support. It must be considered as the means of supporting nutrition for short or long periods when the gastrointestinal route cannot be used. As with any other form of therapy, an awareness of the specific need is essential and a team approach is vital if optimal patient care is to be achieved. A close analogy can be made with detection and treatment of infection. Here, the first line of observation and awareness often lies with the nurse, who notices the patient is unwell and records a raised temperature and tachycardia. The doctor is then informed, clinical examination and appropriate investigations are undertaken, and as a result antibiotics may be prescribed. Then a protocol is designed to assess the efficacy of the antibiotic. Why then should it be so different for the recognition of malnutrition? For it is true that in hospitals in 1981 'starvation in the midst of plenty' does occur and will continue to happen. This is because both medical and nursing staff fail to appreciate the importance of nutritional support for the critically ill and especially when there is partial or total gastrointestinal failure. One of the reasons for this lack of appreciation is the failure to adopt procedures by which malnutrition can be readily recognized. Furthermore, any treatment used to alleviate this malnutrition is not assessed by specific investigations and observations.

As a result of the metabolic response to trauma many critically ill patients will lose between 10 and 20 g of nitrogen per day. The greatest proportion of this nitrogen is lost in the urine as urea derived principally from the breakdown of muscle proteins. It is certain that any acutely ill patient who loses more than 30% of his initial body weight in an acute metabolic illness has only a small chance of survival[3]. Many of these patients will lose up to 20 or 30 g of nitrogen per day, which is equivalent to 0.6–0.9 kg of muscle. Is it surprising therefore that so many critically ill patients waste rapidly with resulting secondary complications? It is a shocking fact that, despite advances in medicine, improved surgical techniques and better anaesthesia, patients still die because of inadequate nutritional support.

Man, unlike the car engine, cannot switch off his metabolic processes because he is not provided with exogenous fuel. If fuel is not provided from without, then the body homeostatic mechanisms are so set that energy will be derived from the body tissues with harmful consequences[4,5]. That is why wasting is inevitable if substrates are not provided and why a negative nitrogen balance will occur. This latter is neither an ivory tower concept nor the repository of academic units. If only clinicians when doing ward rounds would recognize the condition of 'negative nitrogen balance', or perhaps, simply starvation, then appropriate treatment might follow.

What is the incidence of severe malnutrition requiring intravenous feeding? At this stage I must emphasize there is no competition between

Table 1.1 Tests for and evidence of malnutrition

Tests for malnutrition	*Equipment required*	*Result in malnutrition*
Body weight in kg and recent loss	various scales	$>$ 10%
Triceps skin fold thickness (TST) (fat energy reserves)	Holtain skin fold calipers	$<$ 10 mm in males $<$ 13 mm in females
Mid-arm circumference (MAMC) (muscle protein reserves) (MAMC = arm circumf. $-\pi$ (TST))	tape measure in cm	$<$ 23 cm in males $<$ 22 cm in females
Serum albumin (visceral protein)	routine lab. test	$<$ 35 g/l
Serum transferrin Complement C_3 } short half-life proteins	routine lab. test	$<$ 2 g/l
Retinol binding protein Thyroxine binding prealbumin	special tests	
Urinary hydroxyproline	special test	↑ collagen turnover
Lymphopenia	routine lab. test	$< 1.2 \times 10^9$/l
Plasma amino acid profile	specialized tests	changing valine/glycine ratio
Urine 3-methylhistidine	specialized tests	increased muscle breakdown
Hair root morphology	tweezers and microscope	more telogens and dysplasic hairs
Visual assessment of patient	eyes! and clinical acumen	

enteral and parenteral nutritional methods. For parenteral nutrition there is only one absolute indication, namely gastrointestinal failure, which is also the only contraindication to enteral nutrition[6]. Of course, there will be times when partial parenteral nutrition combined with enteral nutrition can be given. Probably not more than 15% of all acute hospital admissions require active nutritional support by one way or another, of which probably only one third require intravenous nutrition.

(II) RECOGNITION OF MALNUTRITION

It is most important that malnutrition can be diagnosed as such[7]. In Table 1.1 some of the measurements are shown, and I wish to emphasize that these should be used in the assessment of all inpatients. It is an unfortunate part of modern hospital practice that the amount that a patient eats, or is offered, is not under the control of medical and nursing staff, but in the hands of ward orderlies or maids. This practice is to be condemned because the patient suffers as a result. It is considered routine for nursing staff to document temperature, pulse rate, respiration and, hopefully, weight on all hospital admissions, so I would advocate that the 'vital sign' charts should include additional measurements. These can be made equally well by nursing or medical staff, but not by non-professional staff. Furthermore, it might be argued that if dietitians were to pay more attention to the problems of hospital malnutrition and its diagnosis, they might improve the shining hour of many of our critically ill patients.

A careful history indicates how much weight has been lost recently, and weighing is mandatory. Whilst one knows that there are changes between the body cell mass and the fluid compartments in the critically ill patient, nevertheless, weight is a useful guide. In addition, simple measurements of mid-arm circumference and skinfold thickness provide useful indicators of muscle protein and adipose tissue (fat energy) respectively[7–9]. These measurements are capable of reproducibility in the ward and should not be regarded as research innovations. Then there are simple biochemical measurements of serum albumin, serum transferrin and C3 complement. The additional measurement of retinol binding protein and thyroxine binding pre albumin are probably of value[10]. Albumin has a relatively long half-life and is a crude indicator of severe malnutrition. The serum transferrin may take a week or 10 days before a fall will point to underlying malnutrition. The other two proteins referred to may be better indicators of rapid onset malnutrition, and, since their measurement is not too difficult, they should become part of routine hospital laboratory practice.

It is a fact that clinicians constantly underestimate the total nutritional requirements of their patients. Whilst it may be of some use to have so-called 'guestimate tables' (Table 1.2) [11,12], nevertheless the range within each

Table 1.2 Catabolic rates and estimated requirements

	Protein (nitrogen) (g/day)	*Energy* (kcal/day)	*g N/kg body wt.*	*kcal/kg body wt.*	*kcal/g N*
Apyrexial medical patient	45–75 (7.2–12)	1500–2000	0.16–0.20	30–37	= 170
Post-operative (uncomplicated)	75–100 (12–16)	2000–3500	0.20–0.22	37–45	= 190
Hypercatabolic, e.g. burns	〉100 (〉 16)	〉 3500	0.22–0.30	46–52	= 210

1. Wide range of requirements for each group accentuates the risk of underestimating.
2. 1 g nitrogen ≡ 6.25 g protein ≈ 30 g muscle
3. Many patients breaking down 20 g nitrogen per day ≈ 0.6 kg muscle – hence potential for rapid wasting.

Table 1.3 Assessment of daily nitrogen losses and requirements

Urine urea nitrogen = 80% of total urine nitrogen across wide range
1 g (16.6 mmol) urea = 28/60 g nitrogen
(mol. wt. of urea = 60)
Total body water = 60% body wt. in kg
Urea equally distributed throughout body water
(i) 24 h urine urea in $g \times 28/60 \times 6/5^* = X \times 0.56 = (A)$g
(ii) Measure proteinuria, if any $= Y \times 4/25^{**}$
$= Y \times 0.16 = (B)$g
(iii) Correction for any rise of blood urea assuming no change in body weight in kg.
Rise in blood urea = Z g/l
$Zg \times 60\%$ body wt. $\times 0.28 = (C)$g
$(A) + (B) + (C)$ = nitrogen loss
= minimal nitrogen requirement

* This factor is correct for untreated patients and those receiving synthetic crystalline amino acid solutions. Use 4/3 for patients receiving protein hydrolysate solutions, as only 60% of total urine nitrogen is present as urea nitrogen, some is excreted as peptide nitrogen

** 1 g nitrogen = 6.25 g protein

N.B. This formula takes no account of any extra renal losses e.g. intestinal fistula. 1 litre of such fluid loss can represent an extra 2-4 g of nitrogen requirement/day

Table 1.4 Consequences of a negative nitrogen balance

Loss of body weight
Less cold tolerance
Impaired humoral and cellular immunocompetence
Increased susceptibility to infections
Increased incidence of wound dehiscence
Loss of muscle mass - poor mobility - compromised ventilatory performance
Hypoproteinaemic oedema
Apathy, depression
Increased mortality

group is so wide that gross underestimates are inevitable. Central to the construction of any parenteral nutrition regimen is the daily nitrogen requirements. It is fortunate that there is a linear relationship over a wide range between total urinary nitrogen and urinary urea[13]. This applies both to the untreated patient and to those receiving crystalline amino acid solutions. The formula for estimating daily nitrogen requirements is given in Table 1.3. Clearly, such a formula assumes normal renal function and makes no allowances for extra renal losses. Studies have shown that for each litre of fluid lost daily from an intestinal fistula (the principal source of extra renal

loss) an additional allowance of 3 g of nitrogen should be made[14].

A number of other tests have been examined, such as hair root morphology[15–17], skin testing with recall antigens, e.g. DNCB, candida[18,19], measurement of urinary 3-methyl histidine[20], and serum amino acid profiles. In the latter, the ratio of valine to glycine has been a useful indicator of impending malnutrition. Urinary hydroxyproline has also been used as an estimate of malnutrition[21] and, likewise, urinary zinc[22]. Whilst no one test is absolute in diagnosing malnutrition, those listed in Table 1.1, which are within the grasp of all clinicians, will suffice to measure the nutritional status and the response to intravenous nutrition.

It is important to emphasize again that intravenous nutrition is neither 'a drop of water with a dash of salt', nor 'nitrogen with a little sweetener'. It must be a comprehensive nutritional programme, which meets the requirements of water, electrolytes, nitrogen, energy, vitamins, essential fatty acids, essential biological elements and acid–base requirements. Let there be no misunderstanding; in the critically ill patient the first considerations are to maintain acid–base homeostasis and tissue perfusion. It is safe to let 48 hours pass before deciding on the need for intravenous nutrition. If, after 3 days of a critical illness, enteral feeding cannot be used, then failure to recognize the need for parenteral feeding and failure to start this therapy, are signs of clinical negligence. The metabolic response to trauma can be likened to the snowball going down the mountainside. If stopped early, little damage is done, but if it gathers momentum, not only may it be impossible to stop, but the damage becomes considerable. So too is the problem of an increasing negative nitrogen balance, as indicated in Table 1.4.

(III) DELIVERY SYSTEMS

Nowadays there should be little problem in gaining vascular access and in the last 5 years there has been a considerable improvement in the delivery systems. It is a matter of individual choice whether one uses a long peripheral catheter, e.g. Abbott's drum catheter[23] which ends centrally, or a central venous catheter[24,25]. My preference is to use the percutaneous infraclavicular subclavian vein catheter with the tip ending just above the right atrium. This method has the advantages of allowing the patient unrestricted limb movements and furthermore, there is no limitation on the osmolar loading. I personally feel that there is little need nowadays for the short peripheral line delivery system[26]. Indeed, that method imposes certain limitations which are not permissible. For these patients vascular access is just as much a lifeline as the artificial kidney is for the patients on regular haemodialysis. Skilled insertion using an aseptic technique and meticulous after care are vital in the care of these lines[27,28]. I feel it is important for the