NUTRITION IN CRITICAL CARE

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In the beginning physicians and surgeons didn't feed patients. It became obvious that malnourished patients didn't survive surgery as well as nourished patients⁽¹⁾. So we started feeding patients enterally and parenterally and sometimes they got better. Now we tailor the protein, fat and energy requirements to try and fit patients' metabolic demands. Potentially in the future we will be altering the patients' immune response, biochemical pathways and disease process by what we feed them and more importantly by the way we feed them.

PHYSIOLOGICAL DEMANDS

Patients in the critical care environment have diverse underlying pathologies and coexisting illnesses. It is difficult to formulate a single nutrition policy to cover all eventualities. But it is possible to find common threads in patient management strategies. It is important to realise that these patients are not only starving but have a hypercatabolic process. This process involves the extensive breakdown of protein and mobilisation of amino acids; it is not a good adaptation within the intensive care setting. In starvation the body rapidly converts its metabolic processes to utilise fat and thereby maintains protein stores. If attempts are not made to reduce the protein breakdown during catabolism, respiratory and cardiac muscle will eventually be lost, significantly compromising recovery and wound healing, ultimately leading to death. I intend to review some of the problems faced with the nutritional management of patients in the critical care environment, outline our current practice and finally look towards future developments.

ASSESSMENT

Patients are often admitted to critical care environments in a nutritionally poor or malnourished state. Objective assessment of nutritional state is difficult; by the time resuscitation has been completed, many patients have received large volumes of fluid causing peripheral oedema and dilutional effects compounded by capillary leak and third space losses. Other objective tests of nutritional state require awake cooperative patients, which is unusual in the ICU. The best idea of the patient's premorbid condition can be obtained from the nursing evaluation on their initial hospital admission or from relatives. Simple clinical observation provides a good basis together with the history of their presenting illness and clinical course⁽²⁾.

TOTAL PARENTERAL NUTRITION

Modern concepts of nutrition in the critical care environment came about with developments in longterm central venous access. It became possible to sustain adequate nutritional intake via the parenteral route, which soon showed that changing the basic formulation of Total Parenteral Nutrition (TPN) could alter the way the body utilised the amino acids, fat and carbohydrate provided; it was not simply a matter of

providing energy. Initial feeding regimens based on large carbohydrate loads changed to include fat emulsions to decrease venous complications. Attempts to reverse the protein loss due to the hypercatabolic state by increasing the amino acid supply did not achieve the desired effect. It has become accepted that the aim of amino acid administration should be a decrease in the rate of protein breakdown, if possible. A positive nitrogen balance, which indicates a build up of muscle, will not be achieved until the catabolic process has been stopped and anabolism started. Initial attempts to correct malnutrition before surgery decreased morbidity, but continuing TPN into the postoperative period increased morbidity – predominantly due to septic complications⁽³⁾. A reduction in septic events can be achieved by the use of enteral instead of TPN nutrition in the early post-operative period⁽⁴⁾.

USING THE GUT

Enteral nutrition, or the use of the gut to provide a route of nutrition, has been in use for a long time. The advantages of a good chicken broth and other delicacies from grandma's kitchen have passed into folklore. Many dietary supplements and other basic foods are available to help with illness recovery. Within the critical care environment multiple feed types are available, simple at first in their composition and now increasingly sophisticated. Basic feed types can be tailored to various diseases. For example, in respiratory disease increased fat content reduces CO₂ production. In renal failure, decreasing the potassium and protein content reduces solute load; and increasing energy concentration reduces the water load. A variety of pre-digested feeds or elemental feeds are available to cater for those individuals with decreased digestive enzymes or short guts with reduced absorptive capacity.

The main limitation to the use of the enteral route for feeding is that the gut is required to function. Increasingly, it is recognised that the gut is one of the first organs to suffer decreased perfusion as a consequence of sepsis or cardiovascular compromise. Gut failure can be a primary or secondary event. It becomes a source of bacteria and endotoxins, which can translocate from the gut lumen and cause sepsis or the systemic inflammatory response (SIRS). Therefore, at the very time gut integrity is required, it is failing. Attempts are made to improve gut blood flow by rapid resuscitation and the use of Dopexamine, which increases splanchnic blood flow. Other policies such as maintaining gastric pH at normal levels and the use of selective gut decontamination have been advocated to reduce bacterial overgrowth.

The time-honoured way of administering enteral nutrition has been via a nasogastric tube. This is easy to insert with few side effects but with gastroparesis (a failure of gastric peristalsis common in sedated and ventilated patients) it is difficult to instigate feeding even with pharmacological support and standardised protocols. Some centres have

undertaken trials with excellent results from specific feeding regimens but are reluctant to share their methods. Nasojejunal tubes can be used but these require either radiological or endoscopic placement in the absence of gastric motility. Surgically-placed percutaneous gastrostomy or jejunostomy tubes have been advocated but in many cases intra-abdominal sepsis is the reason for the laparotomy and insertion is contraindicated. They may have a role in elective surgery, for example following oesophagogastrectomy.

FURTHER DEVELOPMENTS

Following the development of feeding strategies it became apparent that the provision of protein, whilst adequate for our healthy state, was inadequate for critically ill patients. The total provision of amino acids and essential amino acids was adequate, but evidence has become available that during sepsis intracellular glutamine is reduced. This has consequences in the utilisation of amino acids and reduced gut resistance to bacterial translocation. Leucocytes also rely upon glutamine as one of their main energy sources. Although not an essential amino acid, it is present in reduced quantities in enteral feeds and is absent from parenteral nutrition due to its pharmacological instability. Commercial enteral feeds are now available with added glutamine; clinical studies show a reduction in length of hospital stay but no improvement in mortality.

The word 'immunonutrition' has been coined to convey the concept that by altering the components of the feed and the route by which it is given we can produce changes in immune function and thereby the illnesses. The systemic inflammatory response is the fundamental process underlying many of the biochemical and physiological abnormalities within the acutely ill patient. It has multiple mediators including cytokines. As indicated previously the process is wasteful of amino acids and other substrates. The aim of immunonutrition is to modulate these effects by adding or increasing nutrients, possibly via a pharmacological effect. Groups of compounds thought to have this potential include amino acids (arginine and glutamine), nucleotides, anti oxidants and fatty acids (omega3). Clinical studies have indicated that there appears to be a reduction in mortality and septic episodes in those patients fed with immune-enhancing feed^(5,6) and though a recent meta-analysis supports the reduction in septic episodes there was no support for a reduction in mortality⁽⁷⁾. Provision of antioxidants has shown an improvement in *in vitro* assessments of oxidative stress⁽⁸⁾. There is obviously more research needed to define the effects of nutritional drugs.

WHAT WILL WE BE DOING IN 10 YEARS TIME?⁽⁹⁾

Parenteral nutrition has the advantages that its effects are seen rapidly and it doesn't require a functioning gut to work but there is an increase in septic episodes. Enteral nutrition has fewer septic episodes and is cheaper but may take many days to achieve adequate levels of nutrition. We will,

therefore, see combined strategies involving early, commencement of enteral nutrition covered by parenteral nutrition until the former is established. At the RLI we need to improve our ability to deliver nutrients beyond the pylorus using either endoscopic or manually-placed nasojejunal tubes thereby decreasing the delay in establishing enteral feeding. Improved protocols and better prokinetic agents (of which erythromycin is already showing promise⁽¹⁰⁾) will increase the effectiveness of enteral nutrition. Increased understanding of the interactions of chemical messengers will allow immunonutrition to target key metabolic pathways (to reduce the catabolic process) and antioxidants (to reduce cell damage). Nutrition will then be providing a therapy, not merely an energy and substrate source. Nutrition may become less of a supportive item and more of a core treatment, even a drug.

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