Welcome to the Spring Edition of *Best Practice*. We have listened to your feedback, and in this edition we include articles on some of the topics suggested by you in our latest readership survey: nutrition in pancreatic disease, protein requirements in critical illness, and an overview of tube feeding devices. We also include a section dedicated to metabolic disease, and a research corner to bring you a selection of the latest clinical papers and guidelines.

We hope you find this newsletter useful to your clinical practice, and that you enjoy looking at the enclosed photographs taken from a selection of the educational events run by Nutricia Medical in recent months.

As always we welcome your feedback and suggestions which can be sent to the Dietetic Resource Centre team at dietitians.ireland@nutricia.com

Sandra Wilkinson, Senior Dietitian

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**Pancreatic Disease**

**Sinead Duggan, Research Dietitian**

Sinead works in the Centre for Pancreatico-Biliary Disease, based in the Trinity Centre for Health Sciences, Adelaide & Meath Hospital incorporating the National Children’s Hospital (AMNCH). She is a Health Research Board (HRB) Fellow, being funded by a health professional’s research grant (2009). Prior to this she worked for 8 years as a dietitian in AMNCH, for 4 years in pancreatic disease. This is the first part of her article on pancreatic disease, the second part on acute pancreatitis will be published in the next edition of Best Practice.

**Part 1 – Chronic pancreatitis and malnutrition: an under-recognised problem?**

**Background**

The pancreas is an important organ in nutrition and metabolism, having both an exocrine and endocrine role. In response to various stimuli, the pancreas produces up to 2.5L of exocrine fluid per day. Chronic pancreatitis (CP) is defined as the continuing inflammatory disease of the pancreas, characterised by irreversible morphological changes, which typically cause abdominal pain, and / or permanent impairment of pancreatic function. Exocrine dysfunction, manifesting as diabetes, usually occurs late in the disease process.1 The incidence of CP is estimated at 3.5-10 per 100,000 population per year.2 It is more common in males, and usually begins in adulthood.3 The majority of cases in industrialised countries are attributed to alcohol excess. Repeat attacks of acute pancreatitis may result in CP. Other causes include pancreatic duct obstruction, cystic fibrosis, hypercalcaemia, hypertriglyceridaemia and gene mutations. A certain proportion are classed as idiopathic, with no apparent identifiable cause.4 A limited consensus exists on the best method for diagnosing, staging and classifying CP.5 It may be suspected following presentation with suggestive symptoms (typically weight loss, malnutrition, pain, fat malabsorption), but clinical presentation alone may be insufficient to reach a firm diagnosis.

**Malnutrition in chronic pancreatitis**

*Exocrine dysfunction*

Progressive loss of pancreatic cell function results in reduced production and secretion of pancreatic enzymes. This causes malabsorption of carbohydrate, protein and particularly fat. Excretion of more than 15g of fat per day is considered severe.6 The presence of visible oil in the stool is virtually pathognomonic of CP representing a loss of 30-40g per day of fat. Stool in CP tend to be bulky and formed as opposed to watery as in other malabsorptive conditions such as coeliac disease. Clinical tests to detect and measure the presence of faecal fat include the Van de Kamer test (gold standard) and the use of Sudan III fat stain. However, these tests are impractical for clinical use. The Faecal Elastase test is a simple, inexpensive, widely available and risk-free method of assessing exocrine function. Levels between 100 and 200 suggest moderate exocrine dysfunction, while levels of less than 100 suggest severe dysfunction.

Continued overleaf...
Endocrine dysfunction
Diabetes complicates CP in 30-50% of cases, due to a loss of islet cell function.5 Dietary management of CP-related diabetes may be complicated by malabsorption, alcohol abuse and erratic dietary intake.7 There is a higher than normal risk of hypoglycaemia due to the reasons above, which is exacerbated by destruction of glucagon-producing cells. Arguably, dietary restriction should be less stringent in this population than in ‘normal’ diabetes, due to the combined risks of malnutrition and hypoglycaemia.

Micronutrient deficiency
Micronutrient deficiency may be present in even the apparently well-nourished CP patient, driven by suboptimal dietary intake, increased losses, increased requirements, impaired binding of nutrients, anti-oxidant activity and malabsorption. Prevalence of vitamin E deficiency may be as high as 75%, and may be sub-clinical or overt (with neurological manifestations).8 Some studies report that vitamin E deficiency occurs more frequently than vitamin A or vitamin D deficiency.9 Vitamin B12 deficiency may occur due to inadequate protease secretion by the pancreas, which is required to release the vitamin for absorption.10 Zinc and copper deficiencies should also be considered.

Vitamin D and bone health
Osteopathy (osteoporosis, osteopenia and osteomalacia) may occur in 1 in 4 patients.11 This may be due to vitamin D deficiency; calcium malabsorption, poor diet or high smoking history. In CP, vitamin D deficiency tends to be correlated with the degree of exocrine insufficiency and therefore, to the severity of disease.12 Bone health guidelines exist for other gastro-intestinal conditions (coeliac disease13 and inflammatory bowel disease14) recommending routine bone mineral density assessment and supplementation. No such guidelines exist for patients with CP. However, extrapolating from the guidelines for similar malabsorptive conditions, perhaps calcium and vitamin D supplementation should be routine, and regular Dual X-ray Absorptiometry (DXA) may be warranted.

Nutritional requirements
A high energy intake of 35kCal/Kg/24hours is recommended for patients with CP.15 Resting energy expenditure may be higher than normal by 30-50%, especially in underweight or septic patients.16 CP patients will usually have a significantly lower body weight and body mass index than age-matched controls, despite possibly consuming more calories.17 CP patients may salvage energy by colonic bacterial metabolism.18 An extra 10% of energy may be salvaged by bacterial action on malabsorbed carbohydrate. Despite the tendency to over-restrict fat, 30% of calories may be given as fat, which may be well-tolerated, especially from vegetable sources. A severe restriction may exacerbate malnutrition and may not be warranted. Supplementation with medium chain triglyceride (MCT), which are absorbed even in the absence of pancreatic lipase, may aid weight gain.19 MCT fats should be introduced slowly, as they can cause cramps, nausea and diarrhoea. Commercial products containing MCT fats as oil emulsions or in enteral feeds may be useful. A diet rich in carbohydrate and protein is generally advised, although carbohydrate may need to be restricted in the case of concurrent diabetes.20 A protein intake of 1-1.5g/Kg is sufficient and well-tolerated. A low fibre diet is generally advised, as fibre may absorb enzymes, delaying nutrient absorption.21

Pancreatic enzymes
A reduction in steatorrhoea, along with ensuring an adequate energy intake, is the most important principle of nutrition therapy in patients with CP.22 Pancreatic enzymes are usually required as exocrine function deteriorates. The maximal post-prandial delivery of lipase into the duodenum in the normal state is 140,000 IU per hour for 4 hours, and maldigestion is thought to occur when less than 5-10% of enzyme output is delivered.23 Gastric acid denatures enzymes and therefore enteric coated preparations which will dissolve only at a higher pH are preferable. Acid suppression therapy may also be useful. To gauge the accuracy of treatment, a full assessment of symptoms and compliance should be undertaken.

Supplementation and nutrition support
In most cases, normal food will be sufficient to maintain nutritional status.24 Where intake is low, whole protein ONS may be provided; where tolerance is poor, peptide-based ONS could be trialled. A recent randomised controlled trial (RCT) compared the efficacy of an MCT-enriched supplement with dietary counselling by a dietitian on malnourished CP patients over 3 months.25 Both groups showed improvements in body mass index, muscle and fat stores, nutrient intake, and a decrease in fat excretion and pain. This demonstrated that dietary counselling and ONS may be equally effective in improving the nutritional status of CP patients. Enteral feeding may be required in 5% of CP patients, and where not possible, parenteral feeding will be required in <1% of cases.20 Table 1 summarises the indications for nutrition support in CP.

Antioxidants
Pain is a major issue for patients with acute pancreatitis, for which there is no effective medical treatment. Pain may be greatly debilitating, affecting quality of life. In recent years, attention has been given to the theory that taking an oral antioxidant preparation may alleviate free radical damage which is thought to cause intractable pancreatic pain. An RCT on 127 patients demonstrated a reduction in pain, a decrease in analgesia requirements, and a reduction in man-days employment lost in the antioxidant group compared to a placebo.26 The antioxidant preparations, which contain selenium, beta-carotene, L-methionine, vitamin C and vitamin E, are a promising therapy that may offer a simple treatment of pain and its issues.

Conclusion
Nutrition in CP has been described as a ‘problem area’. A review of the literature suggests that patients with CP are at risk of multiple deficiencies and nutritional problems.20 Bone health is likely to be a concern; however there are no guidelines on the assessment or monitoring of bone health in CP. To optimise the management of patients with this progressive disease, we suggest that regular nutritional assessment and follow-up is vital (figure 1).

Table 1: Guidelines on the use of nutrition support in patients with chronic pancreatitis (CP)

<table>
<thead>
<tr>
<th>Indications for enteral nutrition in CP</th>
<th>Indications for parenteral nutrition in CP</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Where patient cannot ingest sufficient calories (e.g. due to pain, obstruction)</td>
<td>• Where enteral nutrition is not possible e.g.</td>
</tr>
<tr>
<td>• Where weight loss continues despite apparently sufficient intake</td>
<td>• Gastric outlet obstruction</td>
</tr>
<tr>
<td>• In the presence of acute complications (e.g. acute pancreatitis)</td>
<td>• Complex pancreatic fistulae</td>
</tr>
<tr>
<td>• Prior to surgery</td>
<td>• Severe malnutrition pre-surgery (where no enteral access)</td>
</tr>
</tbody>
</table>

Taken from ESPEN guidelines on enteral nutrition: Pancreas (2006)20
Protein Catabolism and Requirements in Critical Illness

Valerie Patterson, Critical Care Dietitian, Antrim Area Hospital

Valerie has worked in Antrim Hospital since 1995 and as Specialist Dietitian in critical care since 2000.

Introduction

The critical illness response arises mainly from the systemic inflammatory response syndrome (SIRS) following trauma, infection, burns, pancreatitis and other causes. Leading from this is severe sepsis (acute organ dysfunction secondary to infection), or one step further is septic shock (severe sepsis plus hypotension not reversed with fluid resuscitation). The associated mortality rate is high with an incidence of one in four.1

Nutrition support has progressed from an adjunctive therapy to a proactive one. Much interest now focuses on the combination of specific nutrients and how these can potentially manipulate the inflammatory and immune responses.

Metabolic response

To effectively provide adequate nutrition support to critically ill patients it is important to understand how these responses differ from 'normal' metabolism in healthy individuals.

Critically ill patients are highly catabolic due to an excessive release of inflammatory mediators and a marked release of stress hormones. The result is a distinctly different metabolic and clinical response to carbohydrate, protein and fat administration. The features include hypermetabolism, increased resting energy expenditure and oxygen consumption, reduced ketogenesis and hypercatabolism which exceeds anabolism despite aggressive nutritional support.

If there is an inadequate supply of exogenous energy under 'normal' metabolic circumstances the process of ketogenesis is stimulated which elicits a sparing effect on endogenous protein. In critical illness this response is ineffective leading to increased endogenous glucose production to meet both the requirements of the brain, other glucose-utilising tissues and the extra requirement of glucose for glycolytic metabolism of inflammatory and wound tissues. Coupled with this is impaired suppression of glucose production due to insulin resistance caused by the stress mediators.

The primary casualty of this excessive glucose production is skeletal muscle protein. It is both the largest protein reservoir and the component most affected by protein malnutrition. The metabolic fate of dietary protein usually encompasses a balance of both protein turnover (synthesis and breakdown) and the oxidation of amino acids to produce CO$_2$, ammonia and urea and also synthesis of conditionally essential amino acids (e.g. alanine and glutamine). Since protein synthesis is inhibited there is persistent negative nitrogen balance and generalised catabolism with only the central nervous system being spared.

Overall energy expenditure increases proportionally to the severity of the initial injury and nitrogen balance cannot be achieved without achieving energy balance. This is a conundrum for calculating requirements because there is a trend emerging in the literature recommending hypocaloric feeding especially in obese patients. Overfeeding non-protein calories in the acute phase of critical illness is associated with poor outcomes.

Protein catabolism

The amino acids derived from skeletal muscle are either recycled via hepatic gluconeogenesis, or else used to spare or increase the size of the vital tissues like the liver. This increase in size occurs to facilitate the increased gluconeogenesis, increased reticuloendothelial activity and synthesis of acute phase proteins, leucocytes and proteins involved in healing wounds. Consequently, although detrimental to skeletal...
muscle function, its catabolism is an important substrate source post injury. This magnitude of depletion combined with immobility is in the region of 0.5-1.0kg per day and greatest when the injury is severe and persistent.

Elderly patients naturally have a different body composition due to the aging process whereby even though there appears to be the same ‘bulk’ of tissue the muscle portion has been gradually replaced with fat. This is described as sarcopenia (loss of flesh) but a better term would be rhadopenia (loss of muscle). Research carried out in healthy elderly patients has demonstrated that 10 days ‘bed rest’ leads to reduction in lean body mass (>3%), isokinetic muscle strength reduction (>15%) and muscle synthetic rate reduction (30%). Thus when assessing elderly patients this should be considered as when they become critically ill they have very little ‘reserve’ and altered ability to synthesise muscle.

### Timing of nutrition support

Regarding the timing of nutrition support there are no data showing improvement in relevant outcome parameters in a heterogeneous cohort of critically ill patients. However in adults following burns, surgery, trauma or head injury, early enteral feeding demonstrated significant reduction in infectious complications and length of stay.

Owing to this an expert committee has recommended that haemodynamically stable critically ill patients with a functioning gastrointestinal tract should be fed enterally with a whole protein feed within 24 hours of admission to the intensive care unit. The feed chosen should contain high protein concentrations contributing more than 20% of total energy as protein.

### Requirements

There is a wealth of formulae present in the literature for calculation of energy expenditure but for protein the recommendations are less controversial. It is generally accepted that the optimal intake is 1.2-1.5g/kg/day unless there is significant malnutrition (1.8g/kg/day) or in patients with severe losses, for example on CRRT (continuous renal replacement therapy) where there are extracorporeal losses of up to 10% (Table 1 + 2). Protein sparing was not improved with higher renal replacement therapy) where there are extracorporeal losses of or in patients with severe losses, for example on CRRT (continuous renal replacement therapy) where there are extracorporeal losses of.

### Table 1: Summary of recommendations for general requirements

<table>
<thead>
<tr>
<th>Nutritional status</th>
<th>Protein g/kg/day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-illness weight</td>
<td>1.2-1.5</td>
</tr>
<tr>
<td>Measured weight</td>
<td>1.0</td>
</tr>
<tr>
<td>BMI 30-40</td>
<td>&gt; 2.0</td>
</tr>
<tr>
<td>BMI &gt; 40</td>
<td>&gt; 2.5</td>
</tr>
<tr>
<td>Elderly patients</td>
<td>1.5-2.0</td>
</tr>
<tr>
<td>CRRT dependant patients</td>
<td>1.5-2.0</td>
</tr>
</tbody>
</table>

### Biochemical measure of protein catabolism

Visceral protein status is difficult to quantify because, as yet, in the presence of inflammation, there is a lack of valid laboratory indicators. The degree of catabolism coincides with the severity of illness. This can best be assessed by interpreting the biochemistry and clinical parameters (Table 3).

### Table 3: Some clinical indicators of degree of protein catabolism

<table>
<thead>
<tr>
<th>Increasing</th>
<th>Decreasing</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Urea + creatinine (+/- renal failure)</td>
<td>• Creatinine (Chronic critical illness)</td>
</tr>
<tr>
<td>• Temperature / pyrexia (Note: unreliable when on CRRT)</td>
<td>• Total protein</td>
</tr>
<tr>
<td>• WCC (&lt; 4 or &gt; 10)</td>
<td>• CRP (Except in decompensated liver disease)</td>
</tr>
<tr>
<td>• Inotrope + vasopressor dependence (e.g. dopamine, noradrenaline, vasopressin used for maintaining cardiac output and blood pressure)</td>
<td>• Albumin (Generally poor indicator of status but helps determine degree of infection, inflammation or injury)</td>
</tr>
<tr>
<td>• Oedema</td>
<td></td>
</tr>
</tbody>
</table>

The interpretation of biochemical parameters must be considered in the context of the degree of illness. Different parameters are useful in different conditions e.g. for decompensated liver disease in the absence of sepsis a coagulation screen may be the best indicator of liver synthetic ability because liver enzymes and acute phase proteins may not be elevated.

Overfeeding with protein can possibly be interpreted from uraemia, hypertonic dehydration and metabolic acidosis however each of these also have other causes. When tissue protein is catabolised, loss of nitrogen into the urine increases. Nitrogen balance can be calculated using measured urinary urea from 24 hour urine collections but this is often not practical (Table 4).

### Table 4: Calculations for determining nitrogen balance

\[
\text{Nitrogen Balance (g/day)} = \text{nitrogen intake} - \text{nitrogen output}
\]

\[
\text{Nitrogen output} = \text{urinary urea/mmol/24 hr} \times \text{0.33 + obligatory losses + any extra renal losses}
\]

(If laboratory measurement for urinary urea is in mg/dL then multiply this value by 2.808 and divide by 100 to convert to g/L)

As with delivery of all types of nutrition support in any patient once the calculations are made and the prescription is delivered, the most important element is the ability to effectively monitor the patient’s condition and determine when changes need to be made.

Reference list available on request from the Nutricia Dietetic Resource Centre
The clinical practice of tube feeding has developed significantly over the past number of decades. With this there has been an increase in the range of tube feeding devices designed to improve clinical outcome and patient quality of life. In this article I will take you through the different tubes, pumps and ancillary devices and discuss their indications, features and benefits. I will also provide some practical advice and guidance on the use of these devices in clinical practice.

Part 1 – “Feeding Tubes” is below, and Part 2 – Tube feeding accessories, medicine administration and tube blockage, will follow in the next edition.

**Part 1 – Feeding Tubes**

**Fine Bore Nasogastric Tube (NG)**
Designed for short-term enteral feeding.

**Insertion and placement**
Nasogastric tubes are made from polyurethane and most have a guide wire to facilitate insertion. The guide wire is removed once correct placement has been confirmed. The guide wire should NEVER be reinserted into the tube once it has been removed as it can cause perforation. Initial placement confirmation is usually by chest x-ray but may vary according to local policy.

**Daily care**
It is necessary to check correct tube position prior to each use. NPSA (National Patient Safety Agency) guidelines specify that feeding should only commence once tube aspirate has been confirmed at pH 5.5 or less. If there is no aspirate or if the aspirate is pH 6 or more it can help to lay the patient on their side where possible and retest. If this is unsuccessful it may be that the tube is sitting against the gastric mucosa. To dislodge it inject some air into the tube (10-20mls for an adult) and try to aspirate again. If unsuccessful leave the patient for 1 hour and try again. There are many factors which can affect the stomach pH, for example administration of proton pump inhibitors or recent feeding.

Many patients find the presence of an NG tube uncomfortable. Care of the skin around the nostril is very important. The skin should be clean and dry and securing tape changed regularly. If the nostril becomes ulcerated the tube may need to be changed to the other side.

**Percutaneous Endoscopic Gastrostomy (PEG)**
Description
A PEG tube is a plastic tube which connects the inside of the stomach to the outside to enable feeding to take place directly into the stomach. The tube is held in place by a retention bolster on the inside and a fixation device on the outside. There are a range of sizes: e.g. Corflo 12, 16, 20 FR. PEG Tubes can be polyurethane (PUR) or silicone. Most PEG tubes have more than one port at the end to facilitate the administration of feed and flushes or medication at the same time.

**Insertion and placement**
PEG tubes are usually placed in endoscopy. When the patient returns to the ward they will have a surgical dressing on the site. Once the site has healed there should be no further need for a surgical dressing.
**Daily care**

**Cleaning**

Once the PEG site has healed it is important to keep the skin clean and dry. Clean around the stoma site daily with mild soap and water starting at the PEG site and working outwards. The tube and external disc can be rotated to facilitate accessing all the area around the tube. It is important to dry the area thoroughly.

**Rotation**

In addition the tube and disc SHOULD be rotated 360° at least once a week. Please see tube manufacturer’s guidelines for specific details on when this should commence. This is to prevent a rare complication of long term PEG placement known as Buried Bumper syndrome. It will also prevent over granulation of the skin around the site.

**Clamp**

If there is a clamp on the PEG tube it is advisable to keep it closed when not feeding as this prevents stomach contents siphoning back into the tube and potentially causing a blockage. It is also important that the clamp position is changed regularly to prevent tube damage.

The PEG is designed to stay in place for ≥ a year and under normal circumstances will not come out. If, however it is pulled out inadvertently a dressing should be placed over the site and a replacement tube inserted as soon as possible by a suitably qualified person.

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**Gastrostomy Tube**

**Description**

The Gastrostomy Tube is a silicone tube that has a water filled balloon, which rests against the inside of the patient’s stomach wall, and a retention bolster that rests on the skin. The combination of these two features holds the tube in place. An extension set is normally available if a longer length is preferred for feeding. In general the life span of a replacement gastrostomy tube is 6 months and should be replaced by an appropriately trained person.

**Daily Care**

It is very important that manufacturer’s guidelines on care of the gastrostomy tube are followed. The water filled balloon should be checked regularly to ensure the balloon is intact and there is no risk of tube displacement. The volume of water in the balloon should be aspirated and discarded. The balloon should then be reinflated with sterile water. Local skin care around the site is the same as for a PEG.

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**Button PEG**

**Description**

A Button PEG is a low profile gastrostomy feeding tube. It is an alternative replacement device for patients who are looking for a more aesthetically pleasing device. It is also helpful for patients who have a history of pulling out their PEG tube. It is designed to be used in conjunction with an extension set for feeding. The button is held in place by a balloon of water which should be checked as per instructions for Gastrostomy.

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**Jejunal feeding tube**

For patients requiring feeding long-term where feeding into the stomach is not possible a Jejunal feeding tube is inserted. Skin care is the same as PEG management. As with NJ feeding, extra care should be taken when flushing/administering feed, to prevent contamination.

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**Part 2 – Tube feeding accessories, medicine administration and tube blockage, will follow in the next edition. If you have any questions, or would like any further information about feeding tubes, please contact us at dietitians.ireland@nutricia.com**
The SHS Metabolic Symposium was held for all metabolic healthcare professionals in the Republic of Ireland and Northern Ireland on Thursday the 7th of January in the Gresham Hotel in Dublin.

Dr Ardeshir Monavari, National Centre for Inherited Metabolic Disorders (NCIMD), Children’s University Hospital, Temple St, and Dr Joanne Hughes, from the Royal Belfast Hospital for Sick Children, started the day with an excellent overview on the current services they provide in their respective centres. This was followed by an extremely interesting update on the evaluation of the nurse/dietetic led PKU clinics currently ongoing in NCIMD, by Maria O’ Regan, Clinical Nurse Specialist. Maria gave feedback on how this new clinic model is working extremely well for the healthcare professionals but ultimately also for the patients who attend the clinic. Anne O’ Grady, Dietitian from SHS then gave an update on Anamix 6, a new compliance program that has been developed by SHS and is aimed at children aged one to ten years of age. This includes a number of fun activities and characters aimed at children which are linked to the nutritional benefits of their protein substitute.

Yvonne Rogers, Senior Psychologist, NCIMD, followed with an update on the transition between paediatric and adult health services. This was a thought provoking presentation on the difficulties that can be faced by teenagers as they transition between these services and highlighted how easily they can be lost to follow up, if this area isn’t carefully considered. Professor Eileen Treacy from NCIMD, then gave an extremely interesting presentation on Galactosaemia, which included the results from current studies ongoing at NCIMD and challenges the way we think about treating this condition.

After lunch, Caroline Fitzgerald from SHS gave some fascinating insights on market research that was carried out with metabolic patients in the area of compliance to their dietary treatment and their attitudes to home delivery of metabolic products.

We were joined by Professor Arnold Munnich from Hospital Necker Enfants Malades who gave an excellent presentation on mitochondrial diseases in childhood. He addressed the huge difficulties there are in the diagnosis of these conditions and gave an informative update on the approach he takes in his unit. A special thanks also goes to Anne Clark, Metabolic Dietitian Manager, NCIMD, who chaired the event.

This event could not have taken place without the tremendous assistance and guidance that we received from Dr Ardeshir Monavari and the staff in the metabolic unit in the Children’s University Hospital, Temple St. Their help on the day and in the run up to the event was invaluable.

We have had excellent feedback from everyone who attended the symposium and we look forward to being involved in the organisation of future metabolic educational events. If you would like further details on any of the presentations mentioned above, please contact Anne O’Grady at anne.ogrady@nutricia.com

Here is a selection of photographs from the 3rd Annual SHS Low Protein Living Weekend held in Trim, Co Meath from 26th-28th March. Many thanks to the staff from the Children’s University Hospital, Temple St., and the Royal Hospital for Sick Children in Belfast, for all their help with this event. A full report of the weekend will follow in the next edition of Best Practice.
Meeting report

Nutricia Educational Symposium for Dietitians, Belfast, March 2010

On the 11th March we held our 3rd Annual Educational Symposium for Dietitians in Northern Ireland. This year the symposium was held in the beautiful Ulster Hall in Belfast, followed by dinner in Coco’s Restaurant nearby.

The symposium was opened by Maurice Hickey, Managing Director of Nutricia Medical. He welcomed everyone to the venue, and introduced Karen Robinson, Specialist Dietitian in Nutrition Support in the Belfast City Hospital, who chaired the meeting.

The first presentation was given by Dr Pamela Mason, a pharmaceutical and nutrition writer and consultant, who spoke on the topic of Drug Nutrient Interactions. This was a very practical presentation which highlighted the many different ways that drugs can impact nutritional status, and also how certain food and nutrients can interact with medications and affect their action.

The second presentation came from Dr Sharon Madigan, community dietitian working in the COPD rehab team within North and West Belfast, who spoke on the role of nutrition in COPD. She gave an overview of the COPD condition, and described some emerging evidence for pharmacological interventions in patients at nutritional risk.

Brendan Foley, life coach and author, was the third speaker of the evening. He brought a different dimension to the symposium with his entertaining and thought provoking session entitled “Coping with the changing world of Dietetics”. Brendan identified practical ways in which attendees could adopt a more positive and optimistic approach to their working environment, and in so doing obtain greater job satisfaction. He also challenged everyone in the room to focus their energy on situations they have the ability to change, rather than on things they have no control over. Feedback to Brendan’s session was very positive, highlighting how important it is for dietitians to take a holistic approach to continuous professional development.

Noreen Roche, Head of Nutrition in Nutricia Medical, closed the meeting by giving an overview of the many different services and resources available to dietitians, including the new Nutricia website, www.nutricia.ie and the range of practice support tools provided by the Nutricia Dietetic Resource Centre.

For more information on the symposium, or to receive copies of the presentation handouts, please contact the Dietetic Resource Centre (see front page for contact details).
Clinical Research Corner

Nutrition support in Paediatrics


Background & aims
Children admitted to the hospital are at risk of developing malnutrition. The aim of the present study was to investigate the feasibility and value of a new nutritional risk screening tool, called STRONGkids, in a nationwide study.

Methods
A prospective observational multi-centre study was performed in 44 Dutch hospitals (7 academic and 37 general), over three consecutive days during the month of November 2007. The STRONGkids screening tool consisted of 4 items: (1) subjective clinical assessment, (2) high risk disease, (3) nutritional intake, (4) weight loss. Measurements of weight and length were performed. SD-scores < -2 for weight-for-height and height-for-age were considered to indicate acute and chronic malnutrition respectively.

Results
A total of 424 children were included. Median age was 3.5 years and median hospital stay was 2 days. Sixty-two percent of the children were classified “at risk” of developing malnutrition by the STRONGkids tool. Children at risk had significantly lower SD-scores for weight-for-height, a higher prevalence of acute malnutrition and a longer hospital stay compared to children with no nutritional risk.

Conclusions
The nutritional risk screening tool STRONGkids was successfully applied to 98% of the children. Using this tool, a significant relationship was found between having a “high risk” score, a negative SD-score in weight-for-height and a prolonged hospital stay.


The optimal enteral feeding regimen in children with short-bowel syndrome (SBS) is debated by clinicians. The purpose of this article is to present an overview of published data on feeding strategies in children with SBS. A structured literature search (years 1966 through 2007) was done to identify human studies in children directly addressing nutrition (or specified nutrients) in relation to SBS. Eight relevant studies retrieved were graded by seven experts according to the Scottish Intercollegiate Guidelines Network criteria. This grading system is based on the study design and methodological quality of individual studies. Recommendations were made based on the outcome according to the Scottish Intercollegiate Guidelines Network if appropriate and on expert opinion otherwise.

The most important recommendations are:
- Enteral nutrition should be initiated as soon as possible after bowel resection to promote intestinal adaptation.
- Enteral nutrition should be administered in a continuous fashion.
- Breast milk or standard polymeric formula (depending on the child’s age) is recommended as preferred type of nutrition.
- Bottle-feeding (small volumes) should be started as soon as possible in neonates to stimulate the suck and swallow reflexes. Solid food can be introduced at the age of 4 to 6 months (corrected for gestational age if necessary) to stimulate oral motor activity and to avoid feeding aversion behaviour.

The team of experts concluded that high-quality research on the preferred types of enteral and oral nutrition in children with SBS is scarce. Multi-centre prospective studies on the effects of feeding strategies on bowel adaptation, fecal production, linear growth, and clinical outcome are required to find the optimal feeding regimen in children with SBS.

Nutrition Support in Adults


Background & aims
To evaluate an intervention for improving the delivery of early enteral nutrition (EN) in patients receiving mechanical ventilation with prone positioning (PP).

Methods
Eligible patients receiving EN and mechanical ventilation in PP were included within 48 h after intubation in a before-after study. Patients were semi-recumbent when supine. Intolerance to EN was defined as residual gastric volume greater than 250ml/6 h or vomiting. In the before group (n = 34), the EN rate was increased by 500ml every 24 h up to 2000ml/24 h; patients were flat when prone and received erythromycin (250 mg IV/6 h) to treat intolerance. In the intervention group (n = 38), the EN rate was increased by 25ml/h every 6 h to 85ml/h, 25° head elevation was used in PP, and prophylactic erythromycin was started at the first turn.

Continued overleaf...
Best Practice

Frequently Asked Questions

We have decided to include an FAQ section in which we will comprehensively answer one or more of the common questions we receive through our Dietetic Resource Centre helpline. If you have a question you would like to see answered please email it to us at dietitians.ireland@nutricia.com quoting “Best Practice FAQ” in the subject line.

FAQ No 1. Should patients on Warfarin not take oral nutritional supplements because of the vitamin K content?

Acute and significant changes in Vitamin K intake in the diet can potentially affect warfarin action and INR (international normalised ratio). If the INR is too low, the warfarin will not be effective. If the INR is too high, bleeding times will be longer. Therefore anybody beginning a course of warfarin is advised to keep the vitamin K content of their diet constant.

All oral nutritional supplements (ONS) contain vitamin K, generally at a level of 3.5-20μg/100kcal. It is a common misconception that people on warfarin should avoid ONS because of their vitamin K content. In fact, it does not matter if a patient has a high or low intake of vitamin K once intake remains constant week to week, and the warfarin dose has been adjusted to match that intake. For this reason, two simple steps can help your patients to safely improve their nutritional status with ONS, without affecting their INR:

1. Inform the patients’ GP / warfarin clinic aware if they are commencing ONS
2. Make the patient aware of the importance of taking their prescribed ONS every day to maintain a constant vitamin K intake.

You may be surprised, but ONS may actually help to minimise the risk associated with fluctuations in vitamin K intakes. This is because patients who have a low intake of vitamin K have been found to have more fluctuation in their INR when they consume foods with a high vitamin K content. To explain: If your patient is consuming a low vitamin K diet, and they inadvertently consume a high vitamin K food, they may increase their vitamin K intake by over 50% which represents a huge change in vitamin K intake for the week. The result of this significant increase in vitamin K will be a significant drop in INR. However, if they were consuming an ONS providing 40μg/day vitamin K in addition to their normal diet, consumption of a high vitamin K food may only increase their vitamin K intake by 10%.

So the answer to the question is No – patients on warfarin can safely take ONS once their warfarin dose is adjusted accordingly, and their vitamin K intake remains constant.

Results

Compared to the before group, larger feeding volumes were delivered in the intervention group (median volume per day with PP, 774ml [IQR 513–925] vs. 1170ml [IQR 736–1417]; P < 0.001) without increases in residual gastric volume, vomiting, or ventilator-associated pneumonia.

Conclusions

An intervention including PP with 25° elevation, an increased acceleration to target rate of EN, and erythromycin improved EN delivery.


Background & aims

Many older adults and patients do not achieve sufficient nutritional intake to support their minimal needs and are at risk of, or are suffering from, (protein-energy) malnutrition. Better understanding of current treatment options and factors determining nutritional intake, may help design new strategies to solve this multifactorial problem.

Methods

Medline, Science Citation Index, ScienceDirect and Google databases (until December 2008) were searched with the keywords malnutrition, elderly, older adults, food intake, energy density, variety, taste, satiety, and appetite.
Changes to European food labelling regulations to affect clinical nutrition products

New legislation from the EU, published in November 2008, reports that fibre must be declared on food labelling as a contribution to total energy content. The Directive, 2008/100/EC, defines what is meant by fibre and assigns an energy value as below:

- Fibre means carbohydrate polymers with three or more monomeric units, which are neither digested nor absorbed in the human small intestine.
- The average energy value for fibre should be declared as 2kcal/g.
- All foods marketed in EU have to be labelled in accordance with these new requirements by November 2012.

Nutricia Medical will have to comply with the new regulations and declare the fibre calories on all our medical nutrition products. Although new labels can be phased in until 2012, any new products from here on will follow these guidelines.

Currently this can be seen in two of our newer products:

1. **Protison** contains 1.5g fibre per 100ml, which equates to 3 fibre kcals per 100ml. Protison is therefore labelled as having 128kcals per 100mls (125kcals per 100mls from protein, carbohydrate and fat, and 3kcals per 100ml from fibre).

2. **Fortini Multi Fibre Unflavoured** contains 1.5g fibre per 100ml, which equates to 3 fibre kcals per 100ml. Therefore the energy content listed on the label is 153kcals per 100ml, instead of the 150kcals listed on the other Multi Fibre flavours which have not been updated yet.

It’s important to highlight that the physiological effect of the fibre containing products in terms of energy provision will not change, it is merely the energy value of the product as listed on the label which will be increasing slightly. While this does make the practice of calculating enteral feeding regimens more difficult, we are recommending that you follow the labelled energy values when making your calculations rather than remembering old “non-fibre calorie” values. This is because all new labels and compendiums will quote the new values.
Programme

9.30            Registration

10.00 - 10.05  Welcome Address

10.05 - 10.10  Opening
Chairperson: Roberta McCarthy, Clinical Specialist Dietitian (Neonatology), National Maternity Hospital, Holles Street, Dublin

10.10 - 10.40  ‘Sip Back and Relax – Understanding Issues with Compliance in Children’
Chris Smith, Senior Paediatric Dietitian, Royal Alexandra Children’s Hospital, Brighton, UK

10.40 - 11.10  ‘Nutrition in Paediatric Cardiology’
Carina Kelleher, Senior Paediatric Dietitian, Our Lady’s Children’s Hospital, Crumlin, Dublin

11.10 - 12.00  ‘Enteral Feeding in Paediatric Patients – Clinical Practice Guidelines’
Dr Mark Dalzell, Senior Consultant Paediatric Gastroenterologist, Alder Hey Hospital, Liverpool, UK

12.00 - 12.30  Coffee Break

12.30 - 12.50  ‘Vitamin D in Pregnancy, Infancy & Beyond – How Much is Enough?’
Ana O’Reilly-Marshall, Senior Neonatal / Paediatric Dietitian, University College Hospital, Galway

12.50 - 1.10   ‘Safety in Enteral Feeding – Hang Times’
Deborah Griffin, Senior Paediatric & Neonatal Dietitian, Waterford Regional Hospital, Waterford

1.10 - 2.15    Lunch

2.15 - 2.20    Opening
Chairperson: Dr Trevor Brown, Consultant Paediatrician, The Ulster Hospital, Dundonald, Belfast

2.20 - 3.00    ‘Early Diagnosis & Rapid Treatment of CMPA: Why It Matters to Families’
Dr Rupert Smith, Senior Consultant Paediatrician, Rochdale Infirmary, Rochdale, UK

3.00 - 3.30    ‘Atopic Eczema & Food Allergy’
Dr Helen Cox, Consultant Paediatric Allergist & Immunologist, St Marys Hospital, London, UK

3.30 - 4.00    ‘Food Allergy in Infants – What Formulae To Use, What Formulae Not To Use & Why’
Carina Venter, Senior Dietitian, The David Hide Asthma and Allergy Research Centre, Isle of Wight

4.00 - 4.30    ‘Putting the Theory into Practice’
Sr Karen Orr, Allergy Nurse Specialist, The Ulster Hospital, Dundonald, Belfast

Places are limited so register today!
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Email: events.ireland@nutricia.com
or register online by clicking on the symposium advert on www.nutricia.ie