# **CURRICULUM BASED CLINICAL REVIEWS**

# A practical approach to the management of high-output stoma

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Received 14 August 2013 Revised 4 October 2013 Accepted 5 October 2013 Published Online First 31 October 2013

### ABSTRACT

The development of a high-output stoma (HOS) is associated with water, electrolyte and nutritional complications. Prompt, careful assessment and management is required to avoid rapid clinical deterioration in this patient population. A multidisciplinary approach to management ensures the best possible outcome and quality of life for patients who experience HOS. This article outlines the important considerations in the identification and pathophysiology of HOS. A systematic approach to the management of the condition is outlined, considering fluid and electrolyte requirements, nutrient deficiencies and manipulation of gastrointestinal absorption, motility and secretions using medical and surgical therapies.

### INTRODUCTION

It is estimated there are 102 000 people with a stoma in the UK, and approximately 21 000 new stomas are formed every year.<sup>1</sup> Around 16% of patients with a stoma will suffer from early highoutput stoma (HOS) and 7% of these treatment.<sup>2</sup> will require ongoing Common misconceptions exist in the management of HOS and it is essential that healthcare professionals involved in the care of these patients are appropriately trained to assess and manage the problem. Core competency 2.d. of the 2010 Gastroenterology curriculum stipulates that UK trainees should be able to define the pathophysiology of HOS, describe the clinical consequences and manage the fluid, electrolyte and micronutrient disturbances associated with the condition. In addition to these core competencies, trainees seeking to achieve advanced nutrition competencies must demonstrate a more detailed understanding of the management, outcomes and longer-term problems of patients with HOS (box 1).

### **DEFINING A HOS**

A stoma is an opening, either naturally or surgically created, which connects a portion of the body cavity to the outside environment. Stomas formed from the bowel are named in relation to their location within the gastrointestinal tract. A stoma formed from proximal small bowel (less than 200 cm remaining of small bowel) is referred to as a jejunostomy; one formed from the distal small bowel, an ileostomy, and if formed from the colon, a colostomy.

There is no agreed definition of HOS. It has been variably defined as an effluent of over 1000-2000 mL/24 h. However, the effluent from a HOS is likely to be clinically significant when the output exceeds 2000 mL/24 h, causing water, sodium and magnesium depletion, with malnutrition occurring as a late complication.<sup>3</sup> In practice, a HOS is seen most commonly in jejunostomy patients, and is unlikely to occur in those with a colostomy with retained small bowel.<sup>2</sup> This is due to the large capacity for water resorption in the remaining colon of patients with a colostomy. However, in patients with jejunocolic anastamosis with downstream colostomy, HOS can occur.

### **CAUSES OF HOS**

Surgery, resulting in less than 200 cm of remaining proximal short bowel and the formation of jejunostomy is likely to result in a HOS. This can be referred to as anatomical short bowel syndrome (SBS). Common causes of SBS vary between paediatric and adult populations and are outlined in table 1.

However, other factors may cause a HOS in patients with a stoma, but without a short bowel (functional SBS). These include intra-abdominal sepsis,

2014;**5**:203–207. HOS (b

To cite: Mountford CG,

Manas DM, Thompson NP. Frontline Gastroenterology

## Box 1 Gastroenterology Curriculum 2010

Core competency 2.d. Malabsorption

- Defines the pathophysiology of fluid and nutrient malabsorption, including causes, for example, anatomical and functional short bowel syndrome and high-output stomas
- Describes the clinical consequences of malabsorption, including malnutrition, fluid and electrolyte disturbance and micronutrient deficiency
- Manages fluid, electrolyte and micronutrient disturbances associated with short bowel syndrome or high-output stomas

Advanced nutrition competency 3.c. Short bowel: jejunostomy/high-output stoma

- Understands the underlying diseases that result in a jejunostomy being fashioned
- Has a systematic approach to investigating the causes of high-output stoma
- Understands the principles of treatment including restricting oral hypotonic fluid, drinking a glucosesaline solution and the use of drugs
- Knows when parenteral support is needed
- Able to predict patient outcome in terms of fluid and nutritional needs from knowledge of how much functional bowel remains
- Knows the long-term problems of having a jejunostomy

enteritis (eg, clostridium or salmonella), intermittent obstruction due to strictures, bacterial overgrowth, recurrent disease in the remaining bowel (eg, Crohn's disease), sudden cessation of drugs (eg, steroids or opiates) and administration of prokinetics (eg, metoclopramide). These potential causes must be considered and excluded at the outset.

### PATHOPHYSIOLOGY OF HOS

Approximately 4 L of endogenous secretions pass the duodeno-jejunal junction daily (0.5 L saliva, 2 L gastric juice and 1.5 L pancreaticobiliary secretions).<sup>4</sup> Digestion in the upper jejunum (up to approximately 100 cm distal to duodeno-jejunal flexure) adds further secretions until the mid-jejunum, when absorption becomes the predominant action, resulting in approximately 1–2 L entering the colon. The clinical significance of this is that in patients with a stoma sited in

 Table 1
 Common causes of anatomical short bowel syndrome

| Adults               | Children                       |
|----------------------|--------------------------------|
| Crohn's disease      | Necrotising enterocolitis      |
| Mesenteric ischaemia | Mid-gut volvulus               |
| Neoplastic diseases  | Multilevel small bowel atresia |
| Radiation enteritis  |                                |

the upper 100 cm of jejunum, effluent volume is likely to be significantly greater than the volume taken by mouth.

The sodium content of a meal is approximately 10-40 mmol/L. Salivary, pancreatico-biliary and intestinal secretions are sodium rich and increase the concentration gradient between lumen and plasma in the jejunum to around 90 mmol/L.<sup>5</sup> Jejunal mucosa is highly permeable resulting in rapid water and sodium fluxes, so the jejunal contents become iso-osmolar. Sodium absorption can only occur against a small concentration gradient and is coupled to the absorption of glucose, as well as some amino acids, with the result that there is a net efflux of sodium from plasma to bowel lumen unless the concentration of solution within the jejunal lumen is greater than 90 mmol/L. This physiology is in contrast with the ileum, where sodium can be absorbed against a concentration gradient and is not coupled to glucose, hence, why fluid and electrolyte balance problems are less likely to occur with an ileostomy.

Intestinal adaptation is a physiological response to HOS caused by extensive small bowel resection. This process attempts to restore gut absorption of macronutrients, minerals and water to the 'pre-insult' state. This may be by increasing the absorptive area of remaining bowel (structural adaptation) and/or by slowing gastrointestinal transit (functional adaptation). In practice, functional, but not structural, jejunal adaptation occurs after an ileal resection that leaves the colon in situ. This is thought to be due, in part, to high circulating levels of peptide YY and glucagon-like peptide 2 released from the right colon, which reduce gastric emptying and small bowel transit.<sup>6</sup> <sup>7</sup> These patients may show a gradual reduction in nutritional requirements over time. However, there is no evidence for structural or functional adaptation in patients with a jejunostomy, and their fluid and nutritional needs are unlikely to alter with time.<sup>3</sup>

### CLINICAL ASSESSMENT

Assessments of water, sodium, magnesium and nutritional status are essential. A trial of nil by mouth may be informative to assess baseline stoma output (stoma output problems may be exacerbated in some patients who have been wrongly advised to increase their normal fluid intake or done so because they feel thirsty). Symptoms may include thirst, lethargy, feeling faint, muscle weakness and cramps. Examination should include weight (as well as assessment of weight change), examination of mucous membranes, postural blood pressure measurements, anthropometric measures (eg, triceps skinfold thickness, and mid-arm muscle circumference) as well as an assessment of fluid balance. Referral to a specialist dietician should be made for their assessment and to assist the development of a nutrition plan.

Laboratory measures, including serum urea and creatinine, sodium, potassium, magnesium and urinary sodium, help quantify the extent of water and electrolyte disturbances. The most helpful measure of sodium depletion is a random urinary sodium which, if less than 10 mmol/L suggests sodium depletion. It is helpful to measure these parameters every 1–2 days initially and ultimately every 2–3 months in those with long-term problems. Causes for HOS other than an anatomical SBS should be considered, including signs of intra-abdominal sepsis. Cross-sectional abdominal imaging may be appropriate to exclude this.

An assessment of residual small bowel length (either from operation notes or from radiological measures) can help predict outcome in those patients with a jejunostomy or jejunocolic anastamosis with HOS (table 2). Plasma citrulline can be measured as a marker of small bowel absorptive capacity.<sup>8</sup> However, difficulties extrapolating this information to calculate parenteral nutritional requirements in adults mean that its use in clinical practice is limited.

# MANAGEMENT OF SODIUM AND WATER DEPLETION

Jejunostomy patients have a large stomal output volume, which increases after eating and drinking. Each litre of jejunostomy fluid contains approximately 100 mml/L of sodium.<sup>9</sup> As referred to earlier, consumption of fluids with a sodium concentration of less than 90 mmol/L of sodium (hypotonic solutions) results in a net efflux of sodium from plasma to lumen until equilibrium is reached. Hypertonic fluids containing sorbitol or glucose can also cause stomal losses of water and sodium. Examples of these fluids are listed below (table 3).

Therefore, patients should be advised to restrict hypotonic/hypertonic fluids to less than 1000 mL daily. In practice, this step is often difficult for patients because they feel thirsty and their instinct is to drink more. The remainder of their fluid requirements

| Table 2      | A guide to r  | esidual smal  | l bowe   | el length | and  | long-term  |    |
|--------------|---------------|---------------|----------|-----------|------|------------|----|
| fluid/nutrit | ional support | : requirement | ts for p | oatients  | with | high-outpu | Jt |
| stoma        |               |               |          |           |      |            |    |

|   | Likely long-term fluid/nutritional requirem |  |  |  |  |
|---|---|--|--|--|--|
| Jejunal Jejunum-colon with<br>length (cm) colostomy |   | Jejunostomy  |  |  |  |
| <75   | Parenteral nutrition*                       | Parenteral nutrition<br>+parenteral fluids             |  |  |  |
| 75–100  | Oral (or enteral)<br>nutrition*             | Parenteral fluids±parenteral nutrition                 |  |  |  |
| 100–150   | None  | Oral or enteral nutrition+oral glucose/saline solution |  |  |  |
| 150–200   | None  | Oral glucose/saline                                    |  |  |  |

\*Requirements may reduce with time due to the effects of functional intestinal adaptation.

This table is adapted from reference 3.

 Table 3
 Examples of hypo/hypertonic fluids that should be restricted

| Hypotonic fluids |                       | Hypertonic fluids                             |  |  |
|------------------|-----------------------|---|--|--|
|                  | Water                 | <ul> <li>Coca cola</li> </ul>                 |  |  |
|                  | Теа                   | <ul> <li>Most commercial sip feeds</li> </ul> |  |  |
|                  | Coffee                |   |  |  |
|                  | Fruit juice           |   |  |  |
|                  | Alcohol               |   |  |  |
|                  | Dilute salt solutions |   |  |  |

should be met by consumption of a glucose-saline solution (1 L or more with a sodium concentration of at least 90 mmol/L) sipped in small quantities throughout the day (box 2). Dioralyte mixed to the manufacturer's recommended concentration (5 sachets with 1 L of water) achieves a sodium content of only 60 mmol/L. Mixing Dioralyte to 'double strength' achieves a sodium concentration above the level required. However, increasing concentrations do result in reduced palatability and in circumstances where this is a problem, mixing eight sachets of dioralyte with 1 L of water is an acceptable compromise. It is essential that healthcare professionals are educated in this area of management to avoid mixed messages on appropriate fluid intake.

In the initial phase of treatment, if there is marked dehydration, it is often necessary to rehydrate the patient first with intravenous normal saline, keeping the patient 'nil by mouth', subsequently withdrawing intravenous fluids as restricted oral fluids are reintroduced. Intravenous saline may also be required as a long-term therapy in those patients unable to maintain hydration with the above measures. A random urinary sodium of >20 mmol/L should be the target of treatment.

### **DRUG THERAPIES**

If fluid restriction and consumption of glucose-saline solution are not adequate to maintain fluid balance, drugs that reduce intestinal motility or secretions may

| Box 2 Examples of oral glucose-saline solutions  |
|--|
| Modified World Health Organization cholera solution<br>(also known as St Mark's solution)<br>Sodium chloride 60 mmol (3.5 g)<br>Sodium bicarbonate 30 mmol (2.5 g)<br>Glucose 110 mmol (20 g)<br>Water 1 L       |
| <ul> <li>Double strength' Dioralyte (10 sachets)</li> <li>Sodium 120 mmol</li> <li>Potassium 40 mmol</li> <li>Chloride 120 mmol</li> <li>Citrate 20 mmol</li> <li>Glucose 180 mmol</li> <li>Water 1 L</li> </ul> |

be needed (box 3). As intestinal output rises after meals, especially in net 'secretors' (those patients who lose more water and sodium from their stoma than they take by mouth) it is important to take the drugs before meals. Although octreotide has been shown to reduce large-volume jejunostomy output, injection may be painful and its long term-use has been associated with increased risk of gallstone problems.<sup>10</sup>

### MANAGEMENT OF NUTRIENT DEFICIENCIES

Magnesium deficiency is common in patients with HOS, due to a combination of reduced absorption (because of chelation with unabsorbed fatty acids) and increased renal excretion (due to secondary hyperaldosteronism).<sup>10</sup> Oral magnesium supplementation, given as magnesium oxide may be necessary. Alternative oral agents include magnesium aspartate and magnesium glycerophosphate, though both are unlicenced for this indication. Where oral replacement is insufficient to correct magnesium levels, intravenous magnesium sulfate, usually mixed in saline can be administered.

Patients with HOS require a large, total, oral, energy intake of a diet, in which osmolality is kept low using large molecules which are relatively high in fat/carbohydrate content.<sup>12</sup> Generally, patients should be advised to take a low fibre diet, avoiding nuts, wholemeal products and fruits and vegetables with skins in particular. Hyperosmolar elemental diets should also be avoided as they will also exacerbate the problem of HOS. If enteral feed is given, sodium chloride needs to be added to make the total sodium concentration of the feed to approximately 90 mmol/L while maintaining osmolality close to 300 mOsm/kg. Parenteral nutrition is required if a patient absorbs less than one-third of their oral energy intake, and is usually necessary when less than 75 cm of small bowel remains sited as a jejunostomy.<sup>13</sup>

Most patients with HOS will also require long-term vitamin B12 replacement. Other micronutrient deficiencies are common, in particular selenium; zinc and vitamins A, D, E and K deficiencies should be considered and replaced as necessary.

### SURGICAL CONSIDERATIONS

Patients with long-term HOS as a consequence of SBS, who have difficulty managing their hydration

### Box 3 Drug therapies for high-output stoma

Antimotility drugs

- Loperamide (dose 4–16 mg four times daily)
- Codeine phosphate (dose 30–60 mg four times daily)
   The effect may be greater if both are taken together<sup>11</sup>
   Antisecretory drugs
- Omeprazole (40 mg once or twice daily)
- Octreotide (50 mcg twice daily as subcutaneous injection)

and or nutritional status, may be suitable to be considered for surgical treatment options. The main aims of surgery are to correct mechanical obstruction in order to decrease bacterial overgrowth, and to maximise bowel length.

Surgery to restore intestinal continuity should be considered where there is viable ileum or colon distal to the stoma so that all potentially functional bowel is used. The colon has additional absorptive function as well as a 'braking' effect on intestinal motility.<sup>7</sup> Conversion of a jejunostomy to restore complete continuity, or even to a colostomy in such circumstances, can make a considerable difference in relation to hydration, sodium balance and, therefore, quality of life. There are, however, potential complications associated with restoration of continuity including diarrhoea, which may require dietary modification and the addition of bile-salt-binding agents.

Isolated dilated stagnant sections of bowel are a site for bacterial overgrowth. If symptoms of bacterial overgrowth are present, treatment of dilated segments with tapering should be considered, especially in the duodenum and jejunum. This procedure involves excision of the antimesenteric border of the dilated portion of bowel. This enables more effective peristalsis, thus reducing stasis and bacterial overgrowth.

Bowel lengthening procedures are possible, but rely on the presence of dilated bowel resulting from intestinal adaptation and should therefore be reserved until at least 6 months to 1 year following initial bowel resection. Procedures include the longitudinal intestinal lengthening and tailoring procedure described by Bianchi,<sup>14</sup> where the bowel is divided longitudinally between the mesenteric and antimesenteric borders along its dual blood supply, dividing the bowel into two limbs, each with a blood supply. These two limbs are then closed and anastomosed end to end, doubling that length of bowel. More recently, serial transverse enteroplasty<sup>15</sup> has been performed; a procedure that reduces bowel diameter, increases bowel length, and establishes isopropulsive bowel continuity without loss of mucosa increase functional small bowel length. to Complications of bowel-lengthening procedures are high, including anastomotic and staple line leaks, bowel obstruction from adhesions or ischaemic strictures, bleeding, abscess formation and death. These limitations of bowel-lengthening procedures have led some authors to advocate that they should be reserved for those patients who, after 6 months of bowel adaptation, are tolerating more than half of their feeds enterally, and would therefore have a greater chance of successfully becoming fully enterally fed following a lengthening procedure. In practice, until now, these procedures have been reserved for selected paediatric cases.

More recently, intestinal transplant has become a reality in selected centres worldwide. Frequent severe dehydration associated with HOS in SBS is one of the indications for referral for consideration of intestinal transplantation. However, given that 5 year survival for small intestinal transplantation in the UK is approximately 50%,<sup>16</sup> whereas long-term parenteral nutrition is approximately 73%,<sup>17</sup> transplantation is generally reserved for those with severe intractable problems associated with long-term parenteral nutrition given for SBS and intestinal failure, such as intestinal failure-associated liver disease and recurrent line sepsis or thromboses resulting in loss of central venous access.

### OTHER MANAGEMENT CONSIDERATIONS

Gallstones are common in patients with a jejunostomy or those with a jejunocolic anastamosis, with a prevalence of approximately 45%.<sup>18</sup> Those with a jejunocolic anastamosis are also at risk of developing renal stones as a result of increased absorption of dietary calcium oxalate.<sup>3</sup> They should be advised to follow a low-oxalate diet, which includes avoiding spinach, rhubarb, parsley, cocoa and tea.

Patients with HOS often experience social difficulties in relation to the large volume of effluent. Practical problems may be associated with emptying the bag and embarrassment may result from occasional leakage. Skin care around a stoma site can be problematic in cases of HOS. Stoma care nurse specialists provide a valuable source of support and advice in dealing with such problems.

Novel therapies, including intestinal growth factors may offer a useful therapeutic strategy in select patients. Teduglutide (a recombinant analogue of GLP-2) has been shown to increase small intestinal weight and promote villous hyperplasia in preclinical studies, and may reduce diarrhoea output, increase intestinal fluid absorption and reduce parenteral requirements.<sup>19</sup>

### SUMMARY

Poor knowledge among clinicians and healthcare professionals also often leads to inappropriate and conflicting advice to patients to increase their existing oral fluid intake in response to the problem of HOS. Careful assessment of fluid balance and nutritional status and requirements are necessary before instituting specific fluid and nutritional management plans in this population. A systematic and multidisciplinary approach is required to manage this problem effectively. Where the facilities for this do not exist locally, specialist regional nutrition support teams exist to support local physicians in their management.

**Contributors** CGM devised the idea and drafted the manuscript. DMM and NPT provided additional ideas and review. CGM, DMM and NPT all contributed to the subsequent revision to produce the final version.

### Competing interests None.

**Provenance and peer review** Not commissioned; externally peer reviewed.

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