

Nosocomial scatology in the intensive care unit

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Although Western societies generate an extraordinary amount of humour from scatological matters, serious discussion of issues related to faeces (σκατᾶ= faeces in ancient Greek) is seen as bizarre, if not perverse. This is true not only in daily conversation, but also in hospitals and even intensive care units, where doctors and nurses typically take pride in their ability to deal with all sorts of unpleasant and unusual physical changes. This behaviour is understandable from a human point of view, as faecal incontinence can be embarrassing to patients and the nurses who have to clean them, diarrhoea may be a source of great discomfort to patients (and the associated smells to staff), and constipation can lead to abdominal distension, pain and, occasionally, more serious clinical problems, such as pseudo-obstruction or impaction.¹

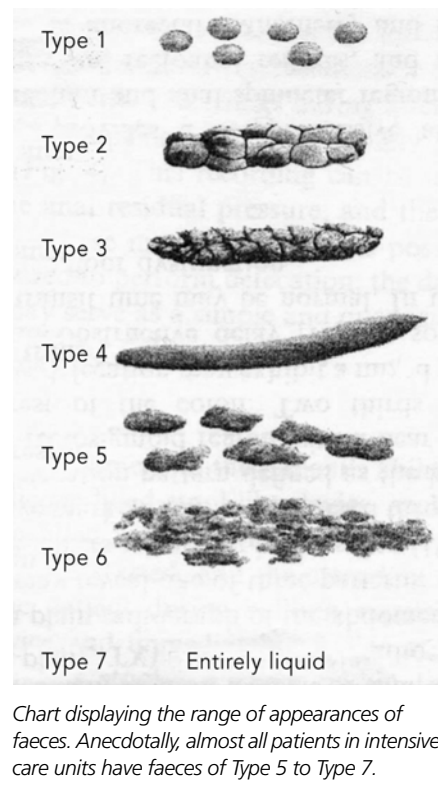
This prurience in the ICU and elsewhere in hospitals means little systematic, rational thought is applied to the scientific assessment of the epidemiology, pathophysiology, diagnosis, treatment and prognosis of these conditions or, of course, to the conduct of well designed, suitably powered, randomised controlled trials.

This is unfortunate for patients, because they may miss out on better management, and for nurses and doctors, because they are constantly exposed to the consequences of the lack of evidence-based management. Here, I will review some aspects of nosocomial scatology in ICU patients that I believe require reflection and investigation.

Is it constipation or non-defecation?

A common and disturbing phenomenon I have seen over the past 10 years has been the management of non-defecation as though it were the same as constipation. Typically, a patient is admitted to hospital with pneumonia, having had little oral intake for 24–48 hours because of the developing critical illness. The patient then undergoes endotracheal intubation and mechanical ventilation, and within 12–24 hours is started on enteral feeding with a

Figure 1. Bristol stool chart



standard formula. The illness continues, and the patient's condition is reasonably stable to improving over 3–4 days when, during the morning round, the bedside nurse reports that there is a problem that needs attention — constipation. The patient has not had a bowel motion for 3 days. Something must be done.

The patient is lightly sedated with propofol. On questioning, he or she denies abdominal pain. Physical examination reveals a lax non-distended abdomen. Rectal examination reveals an empty rectum. The doctor then, despite *no objective evidence of true constipation* (full rectum or colon with need to evacuate but failure to achieve evacuation), prescribes a laxative. Twenty-four hours later, the patient is reviewed in the morning round, and the nurse reports another problem — diarrhoea.

This new problem is trickier than one might expect, and several things may randomly happen depending on the medical and nursing staff

involved. First, the knowledge of recent laxative therapy may be seen as insufficient to exclude infectious diarrhoea, and the faeces may be sent for culture and *Clostridium difficile* toxin measurement. The patient may still have a fever and raised white cell count, which may have oscillated between 11 and $13 \times 10^9/L$. Concern may lead to initiation of nasogastric metronidazole. The nurse may even feel unhappy about frequently cleaning the bed and the patient, and reach for a \$200 rectal tube.

This somewhat chaotic activity is in part a response to the inability to differentiate non-defecation from constipation. Non-defecation seems to me a very reasonable physiological consequence of having had a period of no oral intake, followed by a fluid diet that contains no fibre and is mostly absorbed in the gut, and is therefore unlikely to generate the bulk necessary to form normal faeces and the desire to evacuate. The patient cannot defecate because there is little or nothing in the colon that needs to be evacuated. However, because we do not perform scatological research, we do not know how to reliably

differentiate true constipation from non-defecation, nor the natural history and typical appearance of bowel actions in ICU patients (see Figure 1), how often non-defecation leads to constipation or pseudo-obstruction, and how best to treat these conditions — both non-defecation and constipation.

This might be dismissed as trivial if evidence did not indicate that “constipation” and diarrhoea are so common in the ICU,^{2,8} and treated with a range of apparently random interventions based on incorrect diagnoses and perceptions, leading to patient discomfort and even morbidity, at a high cost. For example, if constipation is wrongly diagnosed in a patient with non-defecation, it may be treated with lactulose as a laxative. This may lead to several episodes of watery stools, in turn leading to perianal skin breakdown or the greater risk of groin-line contamination. This may lead to a pressure sore or line infection, and so on. Unsurprisingly, in all of this chaos, definitions of constipation vary from “failure of the bowel to open for 3 days”,⁴ to “no stools on the third day of ICU treatment”,² to “need for treatment with laxatives or enemas according to the treating physician’s criteria”. As might be expected, no data are available on epidemiology and current practice in the management of these problems in Australia and New Zealand.

Is it diarrhoea or normal bowel actions?

The diagnostic problems surrounding non-defecation/constipation are mirrored by the problems surrounding the differentiation of diarrhoea from normal bowel actions in ICU patients. In a healthy person eating a solid food diet with fibre and roughage, the lack of formed stools, the presence of watery stool, and frequent evacuation are obvious and reliable markers of diarrhoea. However, are these reliable and useful markers of “diarrhoea” in ICU patients receiving a fluid diet? It may well be (and anecdotal clinical observation strongly supports this) that formed stools simply do not occur in ICU patients. Their pursuit is doomed to failure (or iatrogenic diarrhoea). It may well be that the passing of two or three liquid or only partially formed stools is normal in a critically ill patient receiving any of the commonly used enteric feeding preparations. If this is the case, then the term diarrhoea is a misnomer, which often carries interventions that may not be helpful, and may even alter the microbiological flora of the ICU in an undesirable way. The definition of diarrhoea is, of course, arbitrary and varies from five or more liquid stools in a 24-hour period or an estimated volume of 2000 mL or more per day,¹ to “clinical opinion”,² to “ ≥ 3 loose liquid stools/day with a total volume > 250 mL/day”.⁶

Do we know anything about nosocomial scatology in the ICU?

A search of PubMed in July 2009 for randomised controlled clinical trials, using the terms “constipation” and “critical care”, yielded only a recent double-blind randomised controlled trial in two ICUs in the Netherlands.² In this study, 308 patients who had not defecated by Day 3 after admission were randomly allocated to receive placebo (103 patients), lactulose (110) or polyethylene glycol (95). This study provides much useful information. First, defecation occurred earlier with laxatives, at about twice the rate observed in the placebo group in the first 7 days. Second, laxatives “worked” in only 70%–75% of cases. Third, by Day 7 only 30% of the placebo group had defecated. This might seem a catastrophe, exposing patients to the risk of obstruction, pseudo-obstruction, perforation and other nefarious complications. However, the incidence of pseudo-obstruction was 4.1% with placebo, 5.5% with lactulose, and 1% with polyethylene glycol, with no difference between the groups in mortality, at 17%–18%. Interestingly, much is made in this report of a difference in duration of ICU stay between the lactulose group (156 hours) and the placebo group (196 hours). However, it seems unlikely that this difference was due to the beneficial effects of iatrogenically stimulated defecation, given that patients randomised to polyethylene glycol achieved early defecation but had a length of stay of 190 hours.

A skeptical view of this study might be that less non-defecation was exchanged for more diarrhoea by means of laxatives. However, incredibly for a study of laxatives, no information was provided on the incidence of diarrhoea. Yet, an important observation was that, if a patient has not defecated by ICU Day 3, he or she has a 30% chance of doing so by Day 7, and that waiting 4 days appeared to carry no additional clinical risk compared with immediate administration of a laxative. Predictably, early defecation was associated with shorter length of stay, a link most likely secondary to the fact that once people get better, morphine is stopped, the body recovers, things return to normal (gut included), and patients leave the ICU.

Prevention and treatment of non-infectious diarrhoea have been better studied, with negative studies of ispaghula husk,⁹ psyllium hydrophilic mucilloid¹⁰ and fibre-containing feed.¹¹ The best-conducted study was a double-blind, randomised controlled trial from France, involving 11 ICUs and 128 patients, which tested the effect of *Saccharomyces boulardii* and found a reduction in days of diarrhoea from 18.9% to 14.2% ($P=0.007$).¹² Two smaller studies found a limited benefit from the use of fibre,^{13,14} contradicting the results of larger studies.^{9–11} However, all positive studies showed a relatively minor impact.

