Although Western societies generate an extraordinary amount of humour from scatological matters, serious discussion of issues related to faeces (σκατα = 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 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 x 10⁹/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

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**Figure 1. Bristol stool chart**

Chart displaying the range of appearances of faeces. Anecdotally, almost all patients in intensive care units have faeces of Type 5 to Type 7.
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 mor-
bidity, 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 constipa-
tion 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 treat-
ing 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/con-
stipation 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 con-
trolled 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%. Interest-
ingly, 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 defeca-
tion 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 ispa-
ghula 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
myces 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 contradict-
ing the results of larger studies.9,11 However, all positive
studies showed a relatively minor impact.
Conclusions

As can be seen, in 2009, we have little knowledge about what to do for patients who do not defecate by ICU Day 3, or for patients with non-infectious diarrhoea. We have no knowledge of the epidemiology of non-defecation, true constipation (inability to evacuate despite a full bowel — however operationally defined) and diarrhoea (however operationally defined) in Australian and New Zealand ICUs, and we do not know what clinicians currently do about such conditions, let alone what they should do.

Several Australian and New Zealand ICUs have a fine record of publications and world-class research in the physiology and pathophysiology of the upper gastrointestinal tract. Perhaps it is time they moved their focus from the stomach and duodenum to the colon and rectum. If they did, I am sure they would help many patients and, once again, put Australian and New Zealand critical care medicine “ahead of the bunch”. Let’s hope this happens soon.

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References