

COMMENTARY

# Moving beyond the 'pancreatic rest' in severe and critical acute pancreatitis

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See related research by Chang *et al.*, <http://ccforum.com/content/17/3/R118>

## Abstract

Nasojejunal tube feeding is considered the current standard of care in patients with severe and critical acute pancreatitis. However, it is not known whether enteral nutrition is best delivered into the jejunum. This Commentary discusses recent clinical studies that have shown that tube feeding into the stomach is safe and well tolerated in the vast majority of patients with acute pancreatitis, thus overthrowing the notion of putting the pancreas at rest. Development of a new conceptual framework is warranted to further advance nutritional management of patients with acute pancreatitis.

Enteral nutrition is a rapidly evolving frontier in the management of acute pancreatitis (AP). In the previous issue of *Critical Care*, Chang and colleagues investigate whether nasojejunal tube feeding confers any tangible benefit compared with nasogastric tube feeding in patients with AP [1]. It has been 5 years since publication of the previous systematic review on the topic [2] and it is timely to review the progress. Further, the recent international multidisciplinary classification of AP has redefined the 'severe' category of severity and introduced the new 'critical' category of severity (Table 1), thus putting a high emphasis on the need to optimise management of these most challenging patients [3-6].

The study by Chang and colleagues [1] adds an important perspective to the discussion regarding the 'pancreatic rest' concept, which is perhaps the oldest dogma in the management of AP. The central tenet of this concept is that enteral nutrition delivered into any part of the upper gastrointestinal tract other than the jejunum stimulates pancreatic secretion and, consequently, exacerbates the severity of AP. The corollary is that

'non-stimulatory' nutrition had been widely advocated, being total parenteral nutrition two to three decades ago and nasojejunal tube feeding in the past decade. That is why the majority of randomised controlled trials in the past studied 'non-stimulatory' regimens as both intervention and comparator, that is, either parenteral nutrition versus nil per os, or parenteral nutrition versus jejunal tube feeding, or jejunal tube feeding versus nil per os [7,8]. It is argued that this has retarded progress in the field.

The systematic literature review by Chang and colleagues [1] has appraised the current best evidence regarding the use of nasogastric tube feeding (presumed to be 'stimulatory') in patients with AP. It demonstrates that the evidence base is (still) relatively small but does show that enteral nutrition given via the nasogastric route is well tolerated in more than 90% of patients with AP [9-11]. In line with the previous systematic review [2], it shows no statistically significant difference between 'non-stimulatory' and 'stimulatory' regimens in terms of morbidity and mortality. The new, and somewhat surprising, finding here is that both routes of enteral feeding appear to be equivalent in terms of delivery of target calories.

There are two possible explanations for the observed results. First, tube feeding into the stomach might have been 'non-stimulatory' in patients with AP. Unfortunately, little is known about the secretory response of the pancreas during the acute phase of clinical AP, let alone the effect of feeding on it [12]. But a study in healthy volunteers demonstrated that both oral and duodenal tube feeding stimulate pancreatic enzyme secretion in comparison with placebo [13]. Moreover, the degree of pancreatic stimulation is very similar between oral and duodenal tube feeding. Second, tube feeding into the stomach might have stimulated the pancreas in patients with AP but it has no clinical ramifications, essentially meaning that the concept of 'pancreatic rest' might have been fallacious. Although it has become deeply entrenched in the management of AP, it is worth noting that the 'pancreatic rest' concept was never proven in randomised controlled trials. Moreover, the recent MIMOSA (Mild to MOderate acute pancreatitis: early

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**Table 1. Definitions of the four severity categories according to the 2012 international multidisciplinary classification of acute pancreatitis [4]**

	Mild	Moderate	Severe	Critical
(Peri)pancreatic necrosis	No AND	Sterile AND/OR	Infected OR	Infected AND
Organ failure	No	Transient	Persistent	Persistent

(Peri)pancreatic necrosis is nonviable tissue located in the pancreas alone, or in the pancreas and peripancreatic tissues, or in peripancreatic tissues alone. It can be solid or semisolid (partially liquefied) and is without a radiologically defined wall. Sterile (peri)pancreatic necrosis is the absence of proven infection in necrosis. Infected (peri)pancreatic necrosis is defined when at least one of the following is present: gas bubbles within (peri)pancreatic necrosis on computed tomography; a positive culture of (peri)pancreatic necrosis obtained by image guided fine-needle aspiration; a positive culture of (peri)pancreatic necrosis obtained during the first drainage and/or necrosectomy. Organ failure is defined for three organ systems (cardiovascular, renal, and respiratory) on the basis of the worst measurement over a 24-hour period. In patients without pre-existing organ dysfunction, organ failure is defined as either a score of 2 or more in the assessed organ system using the SOFA (Sepsis-related Organ Failure Assessment) score or when the relevant threshold is breached, as shown: Cardiovascular, need for inotropic agent; Renal, creatinine  $\geq 171$   $\mu\text{mol/L}$  ( $\geq 2.0$  mg/dl); Respiratory,  $\text{PaO}_2/\text{FiO}_2$  (partial pressure of oxygen/fractional inspired oxygen concentration)  $\leq 300$  mmHg ( $\leq 40$  kPa). Persistent organ failure is the evidence of organ failure in the same organ system for 48 hours or more. Transient organ failure is the evidence of organ failure in the same organ system for less than 48 hours.

nasogastric tube feeding compared with pancreatic rest) trial compared in a randomized fashion early nasogastric tube feeding (commenced within 24 hours after hospital admission) with nil per os and found that nasogastric feeding does not exacerbate the course of AP and even reduces the risk of oral food intolerance [14]. Similarly, an earlier randomised controlled trial compared early nasogastric tube feeding (commenced within 24 hours after hospital admission) with parenteral nutrition and found no difference between 'non-stimulatory' and 'stimulatory' regimens [15].

In conclusion, accumulating evidence indicates that the site of enteral tube feeding does not affect major clinical outcomes in patients with AP. Given that tube feeding into the stomach is more practical than into the jejunum in the majority of clinical settings, it should be considered as the first-line approach for patients with severe and critical AP. The 'pancreatic rest' concept can now be put to rest. There is a need and justification for developing a contemporary conceptual framework concerning nutritional management of AP.

#### Abbreviations

AP, acute pancreatitis.

#### Competing interests

The author declares that he has no competing interests.

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