Symposium 1

“Feeding in Pancreatitis”
BAPEN Annual Conference

Feeding in Acute Pancreatitis
Clinical presentation

A 45 year old man with a history of binge drinking of alcohol is admitted with acute upper abdominal pain. There are no signs of chronic hepatocellular disease but he has upper abdominal tenderness and has an elevated serum amylase concentration of 1250 (normal <100).

His Imrie score is 4 and a CT scan confirms pancreatic inflammation. There is no evidence of gall-stones on CT or on ultrasound scanning.
Clinical presentation

He is transferred to HDU for further care. By day 2, there has been some nausea and a little vomiting, for which metoclopramide is prescribed. Artificial nutritional support is considered.
Question 1

Would you provide artificial nutrition at this stage?

1) Yes  
2) No  

Red

Yellow
MUST score at this stage is 2 (BMI 26; no prior weight loss; acute illness and no expectation of oral intake for >5 days).
Question 2

How would you provide artificial nutrition?

1) Naso-gastric feeding Red
2) Naso-jejunal feeding Yellow
3) Intravenous feeding Green
Discussion

What is the best route for providing artificial nutrition in acute pancreatitis?

Mr Ross Carter
What is the best route for providing artificial nutrition in acute pancreatitis?

BAPEN Annual Conference
Harrogate
27th November, 2007
Open debate?

Session programme

What is the best route for providing artificial nutrition in acute pancreatitis?

What evidence guides the prescription of naso-jejunal feeds?

The effect of different routes of nutrient administration on human pancreatic exocrine function?
<table>
<thead>
<tr>
<th>Problem</th>
<th>Traditional</th>
<th>Current</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prediction of severity</td>
<td>Imrie/Ranson/CTSI</td>
<td>Organ support</td>
</tr>
<tr>
<td>Nutrition</td>
<td>TPN</td>
<td>?</td>
</tr>
<tr>
<td>ERCP</td>
<td>&lt;48hrs in severes</td>
<td>none (exc.cholangitis)</td>
</tr>
<tr>
<td>Drug modulation</td>
<td>Optimism</td>
<td>none</td>
</tr>
<tr>
<td>Early Tx for sterile necrosis</td>
<td>Open surgery</td>
<td>none</td>
</tr>
<tr>
<td>Prevention of infection</td>
<td>Prophylactic Ab’s</td>
<td>none</td>
</tr>
<tr>
<td>Surveillance for infection</td>
<td>CT guided FNA</td>
<td>none</td>
</tr>
<tr>
<td>1y surgery for sepsis</td>
<td>Open surgery</td>
<td>Perc necrosectomy</td>
</tr>
<tr>
<td>2y Tx for sepsis</td>
<td>More open surgery</td>
<td>Re-do necrosectomy</td>
</tr>
<tr>
<td>Treatment of OPN</td>
<td>Open cyst gastro</td>
<td>Lap cyst gastrostomy</td>
</tr>
<tr>
<td>Treatment of late pseudocyst</td>
<td>Open cyst gastro</td>
<td>EUS cyst gastrostomy</td>
</tr>
</tbody>
</table>
Nutritional issues in acute pancreatitis

Who needs fed?
.....only those that need it!

What morbidity is associated with each modality?

Can feeding / the feed composition affect the clinical course of pancreatitis?
Spectrum of disease in acute pancreatitis

Mild acute pancreatitis

- NO organ failure
- No necrosis
- Rapid resolution
- Secondary prevention

Severe acute pancreatitis

- MOF
- Extensive necrosis
- Prolonged catabolic illness
- Treatment of complications
Spectrum of disease in acute pancreatitis

Mild acute pancreatitis
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- Rapid resolution
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Severe acute pancreatitis
- MOF
- Extensive necrosis
- Prolonged catabolic illness
- Treatment of complications

80+%
“Rule of engagement”

*Do no further harm:*

- *through neglect*

- *through establishing the delivery system*

? *through exacerbation of the disease process*
What is standard management of acute pancreatitis?

“Aggressive conservative therapy”

“drip and suck(?)” – concept of gut rest
oxygen($P_{sO_2}$) and urinary catheter
CVP
Organ support
pethidine analgesia
Interval cholecystectomy
Nutritional support as required

-how did it become standard
Historical management of acute pancreatitis

1878  Friedrich first described the association of alcohol with abdominal pain, vomiting and pancreatic inflammation

1887  Socin was the first to describe an operation for acute pancreatitis

1894  Werner Korte advised a conservative approach limiting surgery for “suppuration in the lesser sac (Arch Klin Chir 1894;48,720)

1902  Kempe performed the first debridement

Diagnosis was generally at laparotomy / post mortem
Mortality following surgery was 40-60%
Nutrition by diet as tolerated
Historical management of acute pancreatitis

1921  Levin designed the single lumen NG tube for feeding  (*JAMA* 1921; 76(15), 1007)

1933  Wangensteen and Paine used the Levin tube for intestinal decompression (obstruction)  *JAMA* 1933;101(20), 1532-1539.

1938  development of rapid amylase estimation  (Somogyi M J Biol Chem 1938;125,399-414)


Nutrition by diet or NG
Historical management of acute pancreatitis

1960  John Howard – nurse through initial illness surgery for infectious complications
(JAMA. 1960 Nov 26;174:1687-9)

Overall mortality down to ~10%

1968  TPN a realistic option for nutritional support
(Dudrick SJ Surgery 1968 64(1) 134-142)

1970  Feeding by a combination of diet, enteral (NG) gastrostomy / jejunostomy of TPN as required

Nutrition by diet or NG or occ. TPN
Historical management of acute pancreatitis

1970’s concern enteral feeding may exacerbate AP
- Konturek SJ Am J Physiol 1972;222, 16-20
- Cassim MM Ann Surg 1974, 180, 228-231
- Vidon N Gut 1978;19, 194-8
- Mitchell CJ Scand J Gastro 1983;18, 5-8
- Evander A Digestion 1982;24, 159-167

1973 Earlier TPN feeding started the better

1977 Concern enteric feeding may increase septic complications
- Ranson J Surgery 1977;82(1), 99-106

Feeding by (early TPN)
Historical management of acute pancreatitis

1980’s Decade of Dogma

- Gastric decompression
- Gut rest
- Early TPN

Debridement for sepsis
- open laparostomy /packing

Bradley EL Ann Surg 1987;206(4),542-550

- closed lavage

Beger HG Br J Surg 1988;75(3),207-212
“striking back”

Nasogastric suction

Navarro S, Ros E, Aused R, et al
Comparison of fasting, nasogastric suction and cimetidine in the treatment of acute pancreatitis
Digestion 1984;30, 224-230

The use of nasogastric suction was associated with a delay the resumption of bowel activity, prolong the duration of pain, analgesic requirements and hospital stay.
“striking back”

Nasogastric suction
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The use of nasogastric suction was associated with a delay the resumption of bowel activity, prolong the duration of pain, analgesic requirements and hospital stay

NO role for the routine prolonged use of NG suction / decompression
Climate of change

Early parenteral support is beneficial
Sax HC, Warner BW, Talamini MA et al
Early total parenteral nutrition in acute pancreatitis: lack of beneficial effects

TPN associated with a significantly higher rate of catheter-related sepsis no difference in the number of days to oral intake, total hospital stay, or number of complications
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TPN associated with a significantly higher rate of catheter-related sepsis no difference in the number of days to oral intake, total hospital stay, or number of complications

Early (<7 days) TPN is worse than no feeding
Is gut rest essential?

McClave SA, et al
Comparison of the safety of early enteral vs parenteral nutrition in mild acute pancreatitis.

Nakad A, et al

Kalfarentzos F, et al
Enteral nutrition is superior to parenteral nutrition in severe acute pancreatitis: Results of a randomized prospective trial. Br J Surg 1997;84(12):1665-9.

Abou-Assi S, et al
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Comparison of the safety of early enteral vs parenteral nutrition in mild acute pancreatitis.

Nakad A, et al
Is early enteral nutrition in acute pancreatitis dangerous? About 20 patients fed by an endoscopically placed nasogastrojejunal tube.

Kalfarentzos F, et al
Enteral nutrition is superior to parenteral nutrition in severe acute pancreatitis: Results of a randomized prospective trial.

Abou-Assi S, et al
Hypocaloric jejunal feeding is better than total parenteral nutrition in acute pancreatitis: results of a randomized comparative study.

Early enteral feed is tolerated and avoids the TPN associated morbidity
Other benefits of enteral nutrition

Mucosal barrier defence mechanism
Ammori B et al J Gastrointest Surg 1999;3,252-262

Improvements in metabolic control
Petrov MS et al, Clin Nutr 2007;26,514-523

Immunomodulation
Gupta R et al Pancreatology 2003,3,406-413

Enhanced feeding
Bengmark S Curr opin Clin Nutr Metab Care 2005;8,557-561
Feeding must be distal to the Ligament of Treitz

Eatock FC et al.
Nasogastric feeding in severe acute pancreatitis may be practical and safe.

Eatock FC et al.
A randomized study of early nasogastric versus nasojejunal feeding in severe AP

Eckerwall GE et al.
Early nasogastric feeding in predicted severe AP - a randomised controlled study
*Ann Surg* 2006;244,959-967

Kumar A et al
Early enteral nutrition in severe acute pancreatitis – a prospective randomised Controlled trial comparing nasojejunal and nasogastric routes
*J Clin Gastroenterol* 2006;40,431-434
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Proximal feeding is possible without
apparent exacerbation of disease
Potential problems with enteral feeding

*Theoretical prolongation of disease*

*Tolerance (Peptide / fibre feeds / aspiration)*

*Risks of tube insertion (endoscopic / radiological)*

*Adequate absorption of delivered feed?*

*Pain on refeeding? (25%)*

*Petrov MS*

*Oral refeeding after onset of acute pancreatitis – a review of the literature (Levy, Pandey and Chebli studies)*

*Am J Gastroenterol 2007;102,2079-2084*
Theoretical advantages relating to feed delivery

Measurable clinically relevant morbidity of nutritional delivery
Theoretical advantages relating to feed delivery

Pragmatic approach to maintenance of nutritional integrity

Oral diet  →  NG  →  NJ  →  TPN

Measurable clinically relevant morbidity of delivery system
Clinical presentation

A dual-lumen naso-jejunal tube is passed to enable naso-gastric aspiration and naso-jejunal feeding.
Question 3

What type of feed would you use via a naso-jejunal tube?

1) Polymeric Red
2) Peptide-based Yellow
3) Elemental Green
What evidence guides the prescription of naso-jejunal feeds?

Miss Olivia Boyd
What evidence guides the prescription of naso-jejunal feeds?

Olivia Boyd
Manchester Royal Infirmary
What we will cover

- Background
- Literature search
- Results from the trials
- Nutritional assessment
- Current clinical practice
- Future developments
Background

• Conventional treatment - resting the pancreas
• Pancreatic rest enables pain relief
• Associated with increased catabolic state
• Leads to increased energy expenditure
• Decreased nutritional status
• Nutritional support prevents malnutrition
• **Debate:** when using the enteral route, what feed should we use?
Literature search

• Aim to look for RCT on enteral feeding in pancreatitis comparing feeds

• Databases searched included medline1950-onwards, EMBASE from 1974, CINAHL from 1982, Allied & Complementary medicine from 1985

• Pancreatitis, enteral nutrition, nutritional support, words truncated using MESH terms

• Limited to English & human studies

• 340 hits, 48 related, 3 met criteria
## The studies

<table>
<thead>
<tr>
<th></th>
<th>Author</th>
<th>Sample</th>
<th>Route</th>
<th>Feeds</th>
<th>Regimen</th>
<th>Outcome measures</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Lasztity et al, 2004</td>
<td>14 treatment (T) 14 control (C)</td>
<td>NJ</td>
<td>T = n-3 PUFA C = polymeric</td>
<td>Started with half strength feed, increased over 3 days</td>
<td>Time receiving NJ feed LOS Complications</td>
</tr>
<tr>
<td>2</td>
<td>Tiengou et al, 2006</td>
<td>15 treatment (T) 15 control (C)</td>
<td>NJ</td>
<td>T = semi elemental C = polymeric</td>
<td>35kcal/kg over 18hrs Increased by 500ml/d until goal achieved</td>
<td>Weight loss &amp; LOS</td>
</tr>
<tr>
<td>3</td>
<td>Pearce et al, 2006</td>
<td>15 treatment (T) 16 control (C)</td>
<td>NJ</td>
<td>T = immune enhancing C = polymeric</td>
<td>As per dietetic protocol Fed for a minimum of 3 days</td>
<td>↓ CRP by 40mg/L</td>
</tr>
</tbody>
</table>
Polymeric Vs. n-3 PUFA’s

Lasztity et al, 2004

- Moderate - severe pancreatitis
- Enteral nutrition - commenced within 24hrs
- Total of 3.3g n-3 PUFA plus additional Vit E given/day
- Feed tolerance was similar between the 2 groups
- LOS & duration of jejunal feed was reduced in the n-3 PUFA group (p<0.05)
- Tendency for fewer complications in the n-3 PUFA group
Polymeric Vs. Peptide

(Tiengou et al, 2006)

- Moderate - severe acute pancreatitis requiring NJ feeding
- Patients were NBM for 7-8 days prior to NJ feeding
- 50% of patients in each group received PN for approximately 8 days
- Tolerance of the feed was similar between both groups
- Semi-elemental feed resulted in less weight loss (p=0.01) and shorter length of stay (P=0.006)
  - ITT LOS: remained significant (p< 0.03)
Polymeric Vs. Immune

(Pearce et al, 2006)

- Patients predicted to develop severe acute pancreatitis

- Enteral nutrition commenced within 72hrs of onset of symptoms

- CRP reduced in 2/15 treatment vs. 6/16 control
  - By day 3 CRP significantly less in control vs. treatment (p=0.028)

- Tolerance of feeds was similar
  - Vomiting – 7 treatment / 1 control (p=0.029)
Nutritional assessment

- Initial screening MUST
  - Body mass index
  - Percentage weight loss in previous 3-6 months

- History of oral intake

- Weight history

- Subjective global assessment
  - Estimated dry weight
  - Physical assessment
Assessing requirements

• 30% will be malnourished presenting with acute pancreatitis \( (\text{Meler et al., 2002}) \)

• Basal metabolic rate increases due to:
  – Inflammatory stress
  – Pain
  – 80% of patients are catabolic \( (\text{Shaw et al., 1986}) \)
Calculating requirements

• Standard equation to calculate BMR e.g. Schofield

• Stress factors vary:
  – 3% chronic pancreatitis
  – 10% acute pancreatitis
  – 20% sepsis & abscess

(PENG Clinical Handbook, 2004)
Current clinical practice

• Most trials have used peptide-based feeds

• Demonstrated to be safe

• Pragmatic view due to lack of robust clinical trials

• It is common to start with a polymeric feed

• If this is not tolerated change to a peptide formula (ESPEN Guidelines 2006)
Future developments

• Role of probiotics

• Novel substrates use

• n-3 fatty acids

• Specific micronutrient supplementation
Clinical presentation

One of the factors in treating acute pancreatitis is the concept of resting the pancreas to reduce its exocrine secretion and limit autodigestion.
Question 4

Which of the following methods of feeding stimulate pancreatic exocrine secretion?

1) Naso-gastric  Red
2) Naso-jejunal  Yellow
3) Intravenous   Green
4) None of these Blue
Discussion

What is the evidence that pancreatic secretion is influenced by different types of artificial nutrition?

Is the concept of pancreatic rest important in treating acute pancreatitis?

Professor Peter Layer
The effect of different routes of nutrient administration on human pancreatic exocrine function

Peter Layer
Interdigestive And Postprandial Pancreatic Secretion*

*Coordinated with gastric and bile output, and GI motility
Oral Nutrients
Pancreatic Enzyme Response To A Mixed Meal In Humans

Layer et al, Gastroenterology 1997;112:1624-34

**Graph:**
- **Y-axis:** Lipase kU/min
- **X-axis:** Postprandial Hours
- **Legend:**
  - Cephalic Phase
  - Gastric Phase
  - Intestinal Phase

The graph shows the lipase activity over time, with peaks and troughs indicating different phases of digestion.
Pancreatic Enzyme Response To A Mixed Meal
In Humans

Layer et al, Gastroenterology 1997;112:1624-34
*Beglinger et al, J Clin Invest 1985;75:1471-76
Pancreatic Secretion In Response To A Carbohydrate Meal In Humans
Layer P et al, Gastroenterology 1986;91:41-48
Role of Duodenal Nutrients
Human Pancreatic Enzyme Output During Intraduodenal Essential Amino Acids

Go VLW et al, Gastroenterology 1970;68:321-328

Duodenal EAA Perfusion

Wash Out

Stimulated Secretion

kU/20min

0 1 2 3 4 5 6 10 min Periods

Lipase
Trypsin

0 50 100
Human Trypsin Response to Graded Duodenal Amino Acid Acid Perfusion


Human Subjects
n = 23
Mean ± SE
p = 0.003

Essential Amino Acids, µmol/min i.d.
Pancreatic Response to Oral vs Duodenal Nutrients


![Bar graph showing amylase output comparison between Placebo, Oral, Duodenal, and Duod Elemental groups.](image-url)
Duodenal Triglyceride Perfusion ± Lipase Inhibitor Orlistat (THL)


Lipase Activity

Free Fatty Acid Generation

CCK Release

Trypsin Response

NaCl  THL

*p<0.01
Layer et al, Gastroenterology 1997;112:1624-34

Digestion and Gastric Emptying of a Mixed Meal in Severe Pancreatic Insufficiency (PI)

- Intraluminal Nutrient Digestion
  - *p < 0.001

- CCK Release
  - *p < 0.01

- Gastric Emptying Time
  - *p < 0.01

Healthy vs. PI untreated controls for intraduodenal digestion, CCK release, and gastric emptying time.
Digestion and Gastric Emptying of a Mixed Meal in Severe Pancreatic Insufficiency (PI)  
Layer et al,  
Gastroenterology 1997;112:1624-34

**Intraduodenal Nutrient Digestion**

- Healthy Control vs. PI untreated: *p < 0.001
- Healthy Control vs. PI treated: *p < 0.01

**CCK Release**

- Healthy Control vs. PI untreated: *p < 0.01
- Healthy Control vs. PI treated: *p < 0.01

**Gastric Emptying Time**

- Healthy Control vs. PI untreated: *p < 0.01
- Healthy Control vs. PI treated: *

% kJ

- 80
- 60
- 40
- 20

CCK, Δpmol/l.180 min

- 500
- 400
- 300
- 200
- 100

90% Emptying

- 180
- 160
- 140
- 120
- 100
- 80
Regulation of Postprandial Pancreatic Secretion: Role of Duodenal Nutrients

- Duodenum: sufficient to elicit the full pancreatic stimulatory response to oral nutrient administration
  - cephalic, gastric contributions adjuvant
  - Marginal stimulatory contribution of jejunum
    Miller LJ et al, Dig Dis Sci 1979;24:150-54

- Stimulation stronger in response to complex vs elemental nutrients

- Stimulatory response to lipid requires predigestion (i.e., presence of FFA)
Regulation of Pancreatic Functional States: Induction of Fed Responses

- Cephalic
- Gastric
- Duodenal Mediation

*Vagal cholinergic
*Peptide hormones

Interdigestive State → Fed State
Stimulation of Pancreatic Enzyme Secretion
By Duodenal Nutrients

**Duodenal Lumen**

- **Nutrients**
  - Osmolarity
  - Distension
  - etc.

- ± **Releasing Peptides**?

**Pancreatic Acinus**

**CCK**

**CNS**

**Neural Reflexes**

**Afferent Neurons**

**Efferent Neurons**

**GI Regulatory Peptides:**
- Hormones
- Neuromodulators
CCK Links Postprandial Motor and Secretory Responses

- Bile Output increases Nutrient Digestion
- Nutrient Digestion increases CCK Release
- CCK Release stimulates Fed Motility Pattern
- Fed Motility Pattern stimulates Pancreatic Enzyme Output
- Pancreatic Enzyme Output increases Bile Output
- Bile Output inhibits Intraduodenal Nutrient Delivery
- Intraduodenal Nutrient Delivery determines Gastric Emptying

Parenteral Nutrients
Parenteral Nutrients Do Not Stimulate Exocrine Pancreatic Secretion

- **Animal experiments (dogs)**
  Traverso LW et al, 1981; Fried GM et al, 1982

- **Human studies**
  Stabile BE 1984; Niederau C 1984; Variyam EP 1985
Pancreatic secretion in response to duodenal vs IV feeding

Jejunal Nutrients
Effects of Jejunal Nutrients on Human Pancreatic Secretion Depend on Perfusion Site

Vu MK et al, Eur J Clin Invest 1999

% Maximum

60 cm

Basal Jejunal
Pancreatic enzyme response to jejunal vs duodenal feeding

Kaushik N et al, Pancreas 2005;31:353-59
Ileal Nutrients

Duodenal Amino Acids + Ileal Exposure

Amylase U/min

N = 9
p < 0.001

-15 0 15 30 45 60 75 90 105 120 min
Effect of Ileal Nutrients on Plasma Glucagon-Like Peptide-1


Duodenal Amino Acids

Ileal NaCl / CHO / Lipid

GLP-1 pmol/ml

N = 9
p<0.001

min
Lipase Output In Response To A Mixed Meal In Humans


"Switch-Off": Induced by ileal nutrients
Regulation of Gastrointestinal Fed and Fasting States: Contribution of Ileum

Cephalic Gastric

Duodenal Stimulation

Interdigestive State

Fed State

Ileal Inhibition ("Brake")
Summary
Regulation Of Human Pancreatic Secretion By Intestinal Nutrients


Oral/Gastric, Duodenal: (Sub-)maximal stimulation
Beglinger 1985
Layer 1997
O’Keefe 2003

Oral vs Intraduodenal: no difference
O’Keefe 2003

Jejunum: Moderate (proximal) or no (distal) stimulation
DiMagno 1973
Miller 1979
Vu 1999
Kaushik 2005

Ileum: Submaximal inhibition
Layer 1990
Keller 1997
Nutrient-Induced Integration of Postprandial Motor and Pancreatic Responses

Camilleri M, Gastroenterology 2006;131:640-58

Duodenal Nutrients:

**Stimulate**
- Pancreatic Secretion
- Intestinal Fed Motor Pattern

**Inhibit**
- Gastric Emptying
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Induce, Regulate & Integrate Postprandial Response
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Ileal Nutrients ("Brake"): **Inhibit** Fed Motility and Secretion
Nutrient-Induced Integration of Postprandial Motor and Pancreatic Responses

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Duodenal Nutrients: **Stimulate**
- Pancreatic Secretion
- Intestinal Fed Motor Pattern

**Inhibit**
- Gastric Emptying

Induce, Regulate & Integrate Postprandial Response

Ileal Nutrients (“Brake”): **Inhibit**
- Fed Motility and Secretion
- Switch Off Fed Response
- Induce Subsequent Interdigestive Pattern
Digestion and Gastric Emptying of a Mixed Meal in Pancreatic Insufficiency
Layer et al, Gastroenterology 1997;112:1624-34

Intraduodenal Nutrient Digestion

CCK Release

Gastric Emptying Time

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CCK, Δpmol/l.180 min

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- Healthy Control
- CP untreated
- CP treated

90% Emptying

- *p < 0.01
CCK Links Postprandial GI Motor and Secretory Responses

Intraduodenal Nutrient Delivery 

- Bile Output increases
- Lipid Digestion increases
- CCK Release stimulates

- Pancreatic Enzyme Output increases

Gastric Emptying inhibits

determines
Clinical presentation

The patient makes a gradual recovery and resumes ad libitum food intake but is advised to avoid alcohol.
Discussion