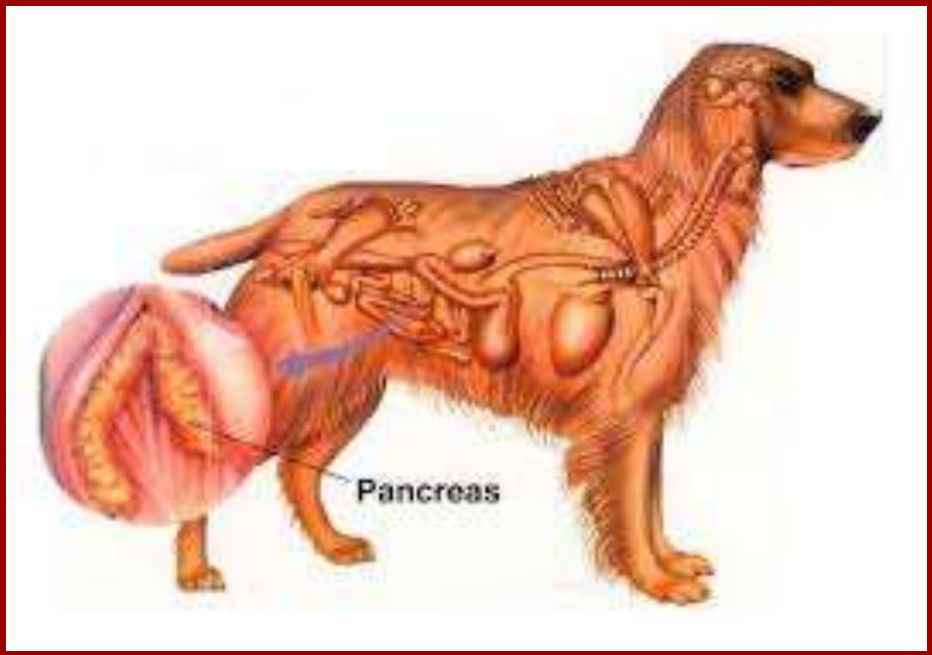

Acute pancreatitis in the dog

Jennifer Kyes, DVM DACVECC
Oct 30, 2014

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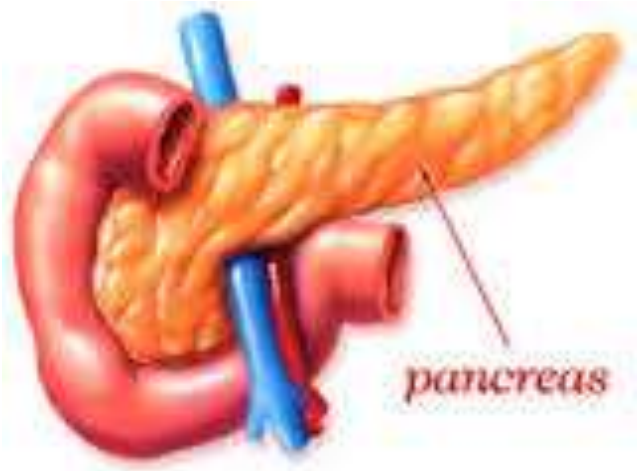
Acute pancreatitis in the dog

- Pancreatic physiology
- Pathophysiology of acute pancreatitis
- Clinical signs
- Diagnostics
- Treatment
  - Supportive care
  - Fasting
  - Re-feeding



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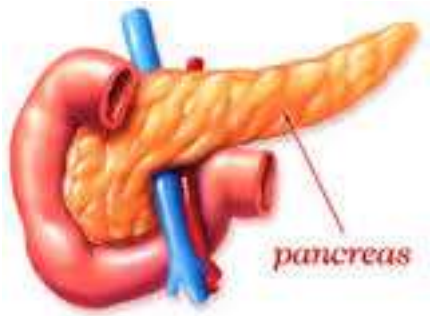
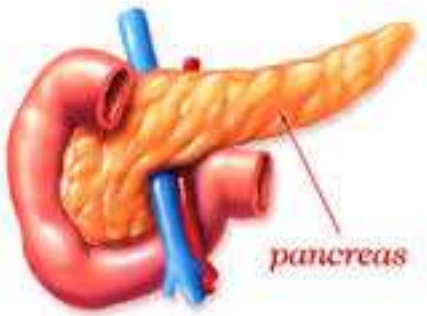
Physiology

- Complex release of secretory granules triggered by hormones and neuronal activity.
- Cholecystokinin (CCK) plays a central role in regulating pancreatic secretions.
- Most important stimulator of CCK is lipids
- Low levels when fasted. Increase 20-fold during digestion.
- 3 phases of pancreatic secretion
  - Cephalic
  - Gastric
  - Intestinal



Physiology

- The pancreas is able to protect itself from auto-digestion via:
- Enzymes stored as zymogens
- Zymogen granules remain separate from lysosomal granules enclosed in membrane bound organelles
- Activated AFTER secretion into duodenum
- *Pancreatitis-associated protein* increases 100-fold during injury and is bacteriostatic preventing infection during pancreatitis.

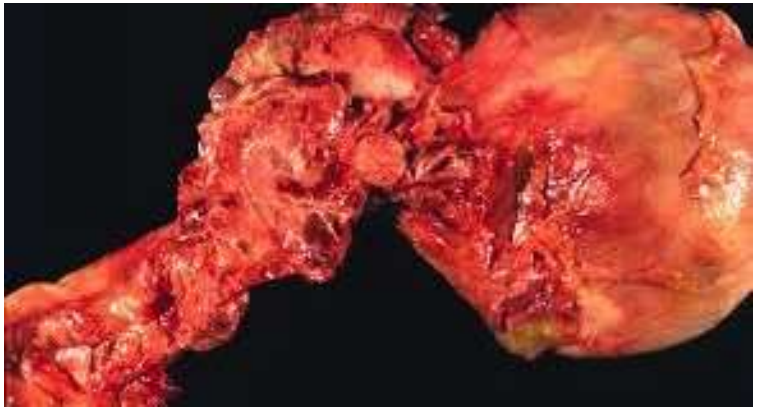


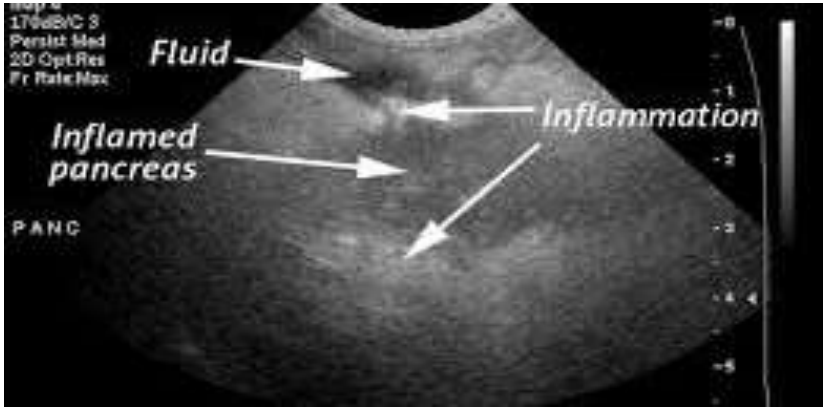
3 phases of pancreatic secretion

Phase	Stimulant	Regulatory pathway	% max enzyme secretion
Cephalic	Sight, smell, taste, mastication	Vagal	25%
Gastric	Distention, gastrin	Vagal-cholinergic	10-20%
Intestinal	Amino acids, fatty acids, H+	CCK, secretin	50-80%

Acute pancreatitis

- Acute pancreatitis is an inflammatory condition that may cause extensive local damage to the pancreas, as well as compromising the function of other organs.





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Pathophysiology of acute pancreatitis

Injury is linked to 3 events

1. Protective mechanisms preventing activation of zymogens are overwhelmed and enzymes are activated within the acinar cells of the pancreas
2. Inhibition of pancreatic secretion OUTSIDE of the pancreas. Enzymes are retained within the cell.
3. Secondary factors perpetuating injury; inflammation, vascular injury and decreased perfusion

Clinical signs

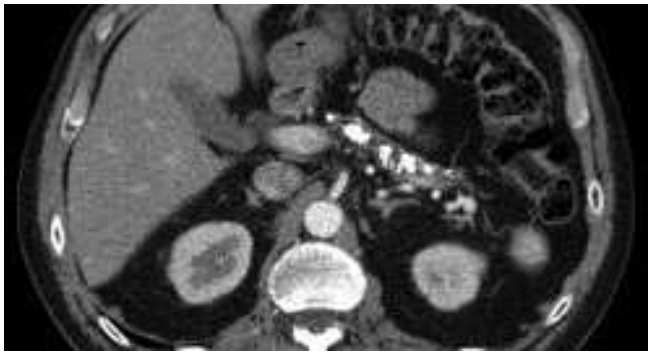
- Abdominal pain*
- Vomiting*
- Diarrhea*
- Inappetence*
- Hematochezia
- Melena
- Weight loss
- Hematemesis

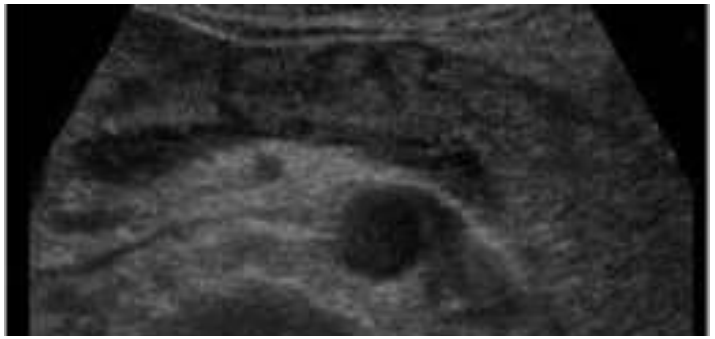
Risk factors

- Obesity
- High fat dietary exposure*
- Diabetes mellitus*
- Hyperadrenocorticism
- Hypothyroidism
- GI disease
- Hyperlipidemia

Diagnostics

- Amylase/Lipase
- Trypsin-like immunoreactivity
- Spec cPL / Snap cPL
- Radiographs
- Ultrasound
- CT





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Diagnostics

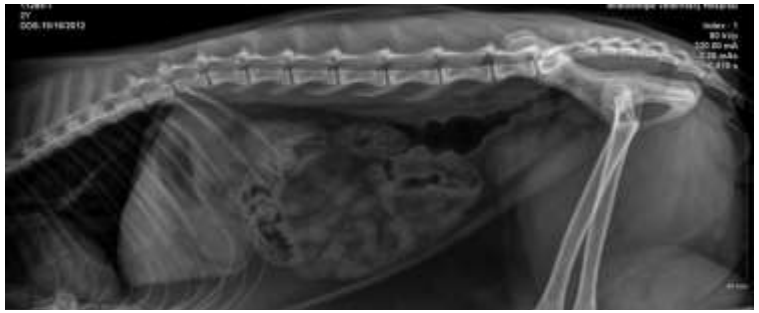
- **Amylase/Lipase**
  - Non specific, multiple sources outside pancreas
- **Canine Trypsin-Like Immunoreactivity (cTLI)**
  - Low sensitivity. Represents total amount of pancreatic tissue present and better for a diagnosis of EPI than pancreatitis.
- **Canine Pancreatic Lipase Immunoreactivity (cPLI)**
  - Best overall performance compared to other serum markers for diagnosis of pancreatitis
  - Spec – quantitative Snap- normal/abnormal

Diagnostics

Test	Outcome	Sensitivity (%)	Specificity (%)
Amylase (>1240 U/L)	Mild Moderate-severe	43 71	86 86
Lipase (>750 U/L)	Mild Moderate-severe	54 71	43 43
cTLI (>25 ug/L)	Mild Moderate-severe	30 29	100 100
Spec cPLI (>200ug/L)	Mild Moderate-severe	43 71	86 86

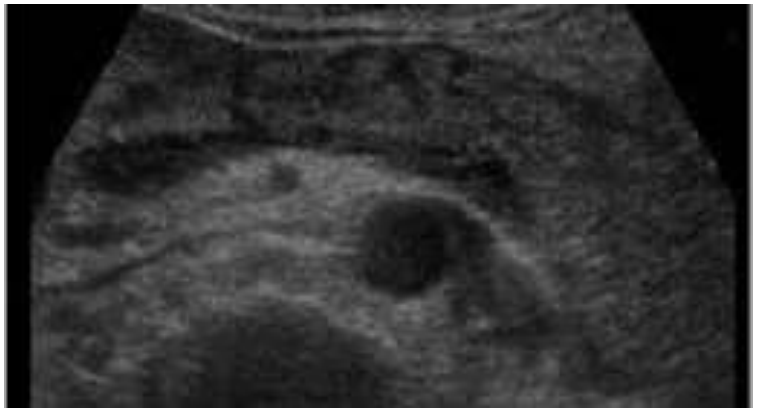
Diagnostics

- **Radiographs**
  - Non specific and supportive at best with lack of detail in peri-pancreatic area
  - Increased opacity in the right cranial abdomen
  - Displacement of descending duodenum or stomach
  - Gas-filled descending duodenum
  - Widening of gastric-duodenal angle creating a mass effect
  - Fluid accumulation leading to loss of detail



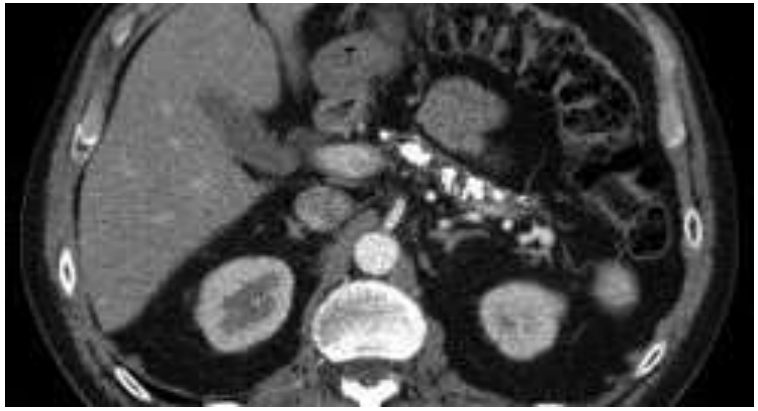
Diagnostics

- **Ultrasound**
  - Increasing popularity but heavily dependent on operator skill and experience.
  - U/S findings consistent 68% of the time with clinical diagnosis
  - Hypoechoic pancreatic parenchyma
  - Hyperechoic mesentery
  - Enlargement of the pancreas
  - Peritoneal effusion



Diagnostics

- **Computerized tomography (CT)**
  - Important in human medicine
  - Accurate in diagnosing extent of necrosis
  - Not commonly used in veterinary medicine



Pancreatitis can be:

- Mild – outpatient, supportive
- Moderate – inpatient, short duration, supportive.
- Severe – inpatient, longer duration, aggressive therapy, 1-2 CRI's, consider referral.
- Life-threatening – Referral case. Multiple CRIs, plasma, advanced supportive care and monitoring, multiple organ dysfunction.

Treatment options

- IV fluid therapy; balanced solution
- Pain medications; injections, CRIs, oral, transdermal
- GI support; famotidine, maropitant, ondansetron
- No antibiotics
- NPO vs feeding
- Plasma (severe)
- Multimodal analgesics (severe)

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What does NPO mean?

NO FOOD BY MOUTH

NO WATER BY MOUTH

NO MEDICATIONS BY MOUTH

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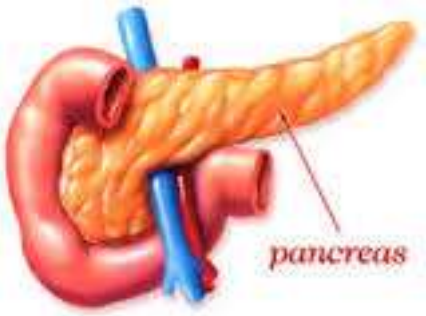
MY NPO RULE OF 3!

1. Patient is comfortable on abdominal palpation
2. CPLI<500.
3. Ultrasound is same or better than previously noted.



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#1 – The patient is comfortable on abdominal palpation

- Can comfortably palpate the dogs abdomen with minimal or no analgesics on board.
- Monitor behavior; moving, active, BAR?
- Trend pain score. Is it lower?
- If the patient palpates better, behaves better, and pains scores are better…… the pancreatitis is usually better! ☺

#2 - CPLI<500. Is trending valuable?

In the acute case….I think so!

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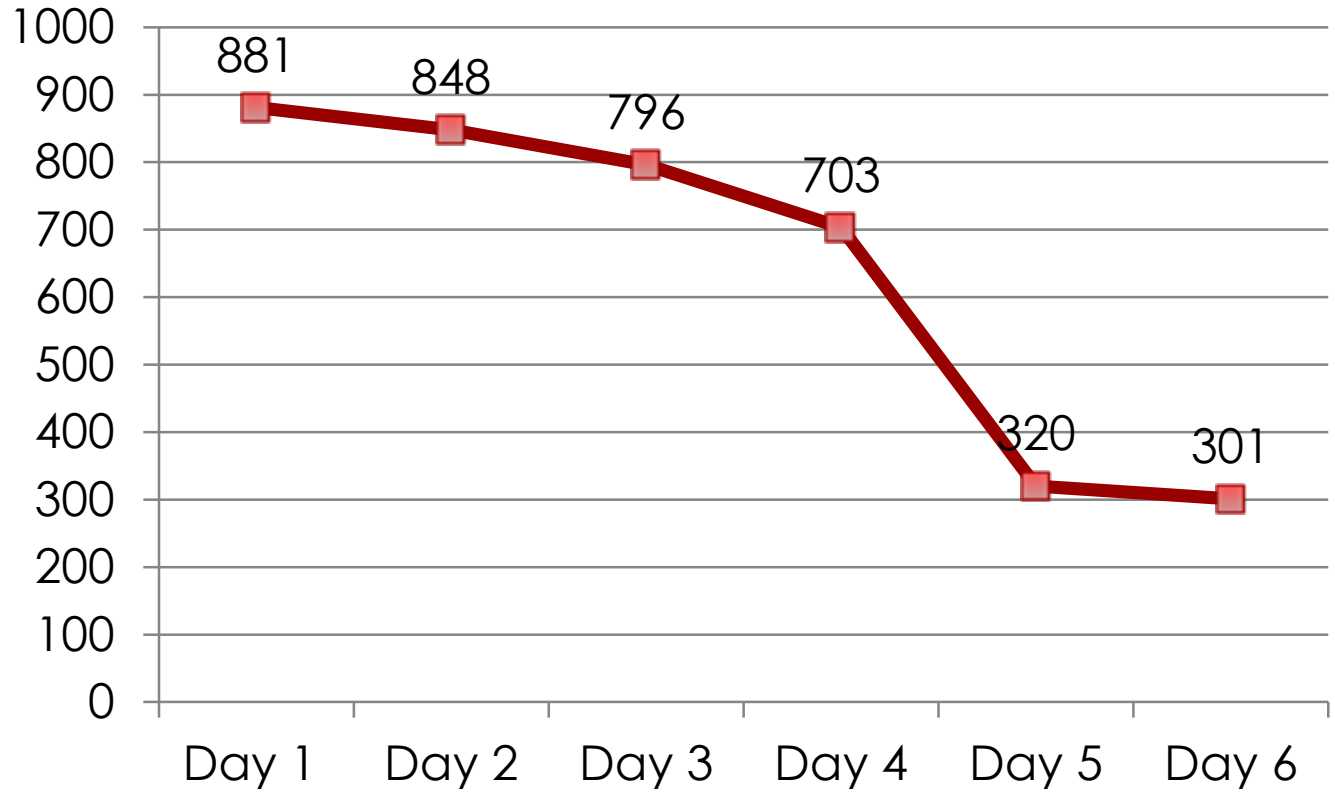
#2 - CPLI<500. Is trending valuable?



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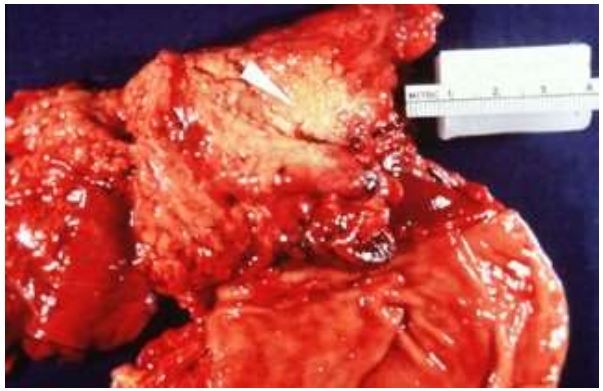
#3 - Ultrasound is same or better than previously noted.

- Trend the hypoechoic parenchyma
- Trend the hyperechoic mesentery
- Trend the peritoneal effusion
- Note the level of discomfort during the exam.
- Look for development of an abscess or walled-off sections



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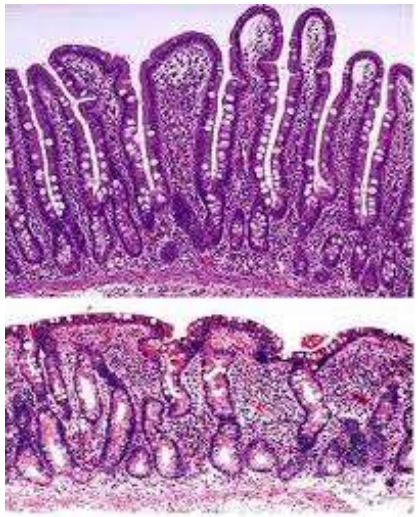
Why do I NPO??

- Primary signal for pancreatic secretions is food, if you stop stimulating in it…..you can stop the ongoing damage and allow it to heal
- Animal can survive without nutrition for extended periods
- Less RELAPSES
- Minimal adverse effects to weight and albumin
- Enteral and parenteral nutrition can be complicated

So what happens when you are fasted for <7 days

- Decreased rate of intestinal epithelial cell migration from crypts to villi
- Decreased intestinal cell apoptosis and shedding at villus tips (<4 days of fasting = villous atrophy)
- Energy sparing (use fat stores as energy source)
- Preservation of mature enterocytes
- Intestines prepare for restoration maximizing nutrient absorption for future.
- Decrease in BMR by 20%



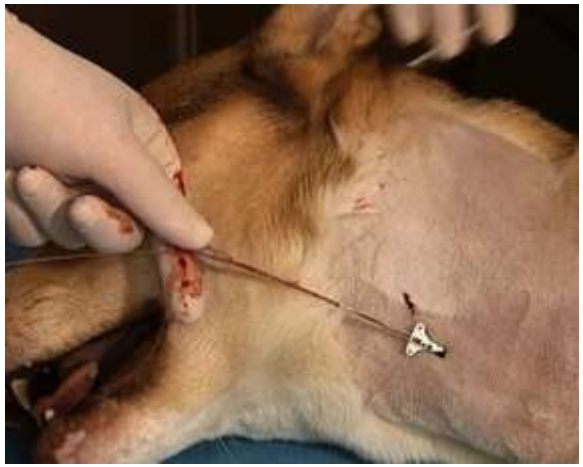
So why do I NPO?

- Because the GI tract prepares for starvation. Maximizes mature functioning enterocytes and reduces BMR.
- Because the side effects of NPO are minimal.
- Because the GI tract is completely restored after 3 days of refeeding
- Cellular activity is completely restored after 3 days of refeeding

Average weight loss was 0.6kg or 1.32 lbs over 6 days.

So why don't I use parenteral nutrition?

- Invasive
- Central line
- Product contamination
- Time consuming
- Unnecessary for most patients
- Additional $ to owners
- Break the *criticalist* stereotype



When do I feed my patients?

Refer back to my rule of 3!

1. When my patient is comfortable on abdominal palpation
2. When the CPLI<500.
3. When the ultrasound is same or better than previously noted.
4. When my patient looks and feels like it wants to eat!!!!

How to feed…….. my personal opinion

- What diet should you select? **LOW FAT!**
- How much should you feed? **¼ Normal amount**
- How often should you feed? **Twice a day**

ROYAL CANIN

DIETS

Diet	Dry (% fat)	Canned (% fat)
Low Fat ****	6.6	9.0
Weight control	8.2	10.0
Hypo	10.4	18.2
Calorie Control	10.9	n/a
Fibre	12.2	29.5
Preventative	16.5	20.1
S/O	17.0	n/a
Reduced protein	18.2	22.1

DIETS

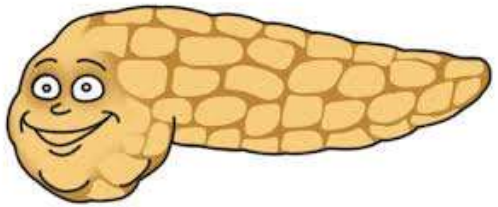
Diet	Dry (% fat)	Canned (% fat)
W/d	9.0	12.7
R/d ****	8.6	8.6
z/d	13.0	13.9
l/d	14.2	14.9
T/d	16.4	n/a
U/d	20.5	26.9
S/d	n/a	26.0

PURINA DIETS

Diet	Dry (% fat)	Canned (% fat)
OM****	7.24	11.21
HA Hypoallergenic	10.54	n/a
EN Gastroenteric	12.3	14.9
Dual Fiber Control	12.9	n/a

Summary

- Support, Support, Support
- Analgesic appropriate for severity of disease
- Trend CPLI q 48-72 hours
- Trend ultrasound appearance in severe cases
- Fast until CPL<500 and comfortable
- Re-feed low fat, low volume, low frequency



And you will have a happy pancreas

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Questions?