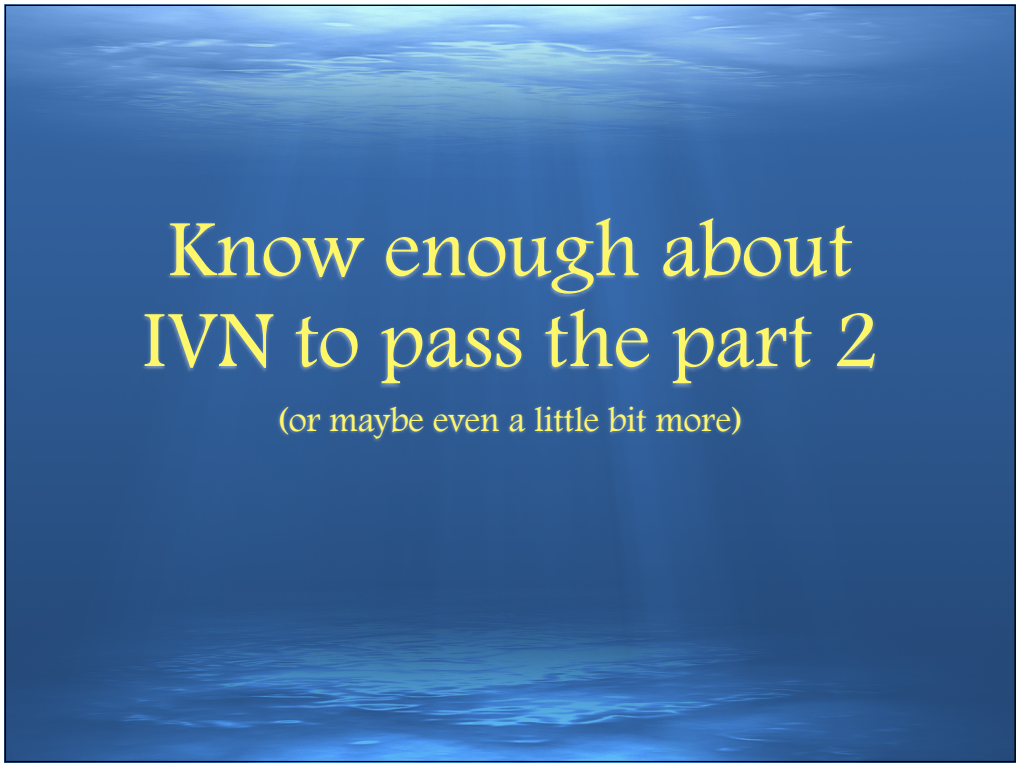


- We're going to talk a bit about surgical nutrition
- Complex topic with lots of side branches, and you just need to have a working knowledge to take to the exam and beyond
- I'm going to present some basic facts and then we'll do a few problem cases and see what comes about
- With part 2 there's no syllabus, so studying is like laying one layer on top of another
- As you get closer you have to narrow down and summarize what you know- a topic like TPN is summarized to an A4 page or maybe a page and a half
- So you end up with a set of cues that can help you remember more details in the heat of battle

This Sprinkler Guy TotallyLooksLike.com This Happy Elephant from the Disneyland Jungle Cruise

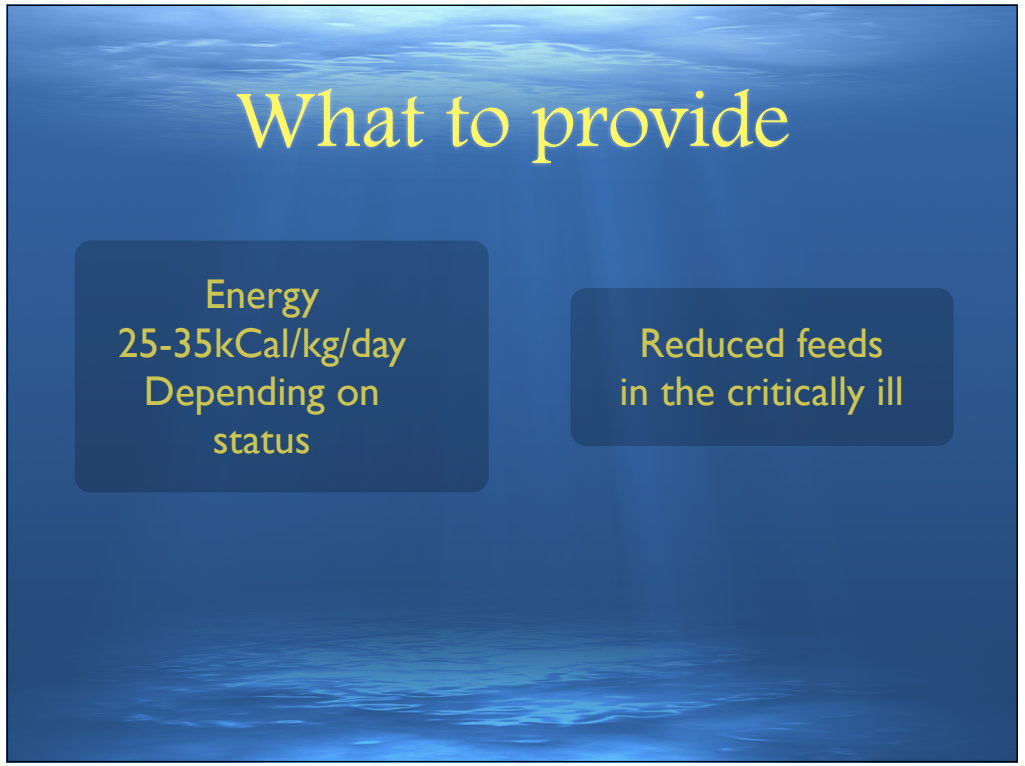
- firstly you've got to know why you're bothering to nourish your patient
-after all we know patients can starve for long periods, people go on hunger strikes and survive etc
- Wound healing- external wounds, anastomoses etc
- Resistance to infection
- The ability to mobilize which will reduce ileus, improve lungs, reduce DVTs
- Respiratory muscle function
- And just a sense of wellbeing

Some of the other effects of malnutrition may be• *a* *Decreased cardiac output*

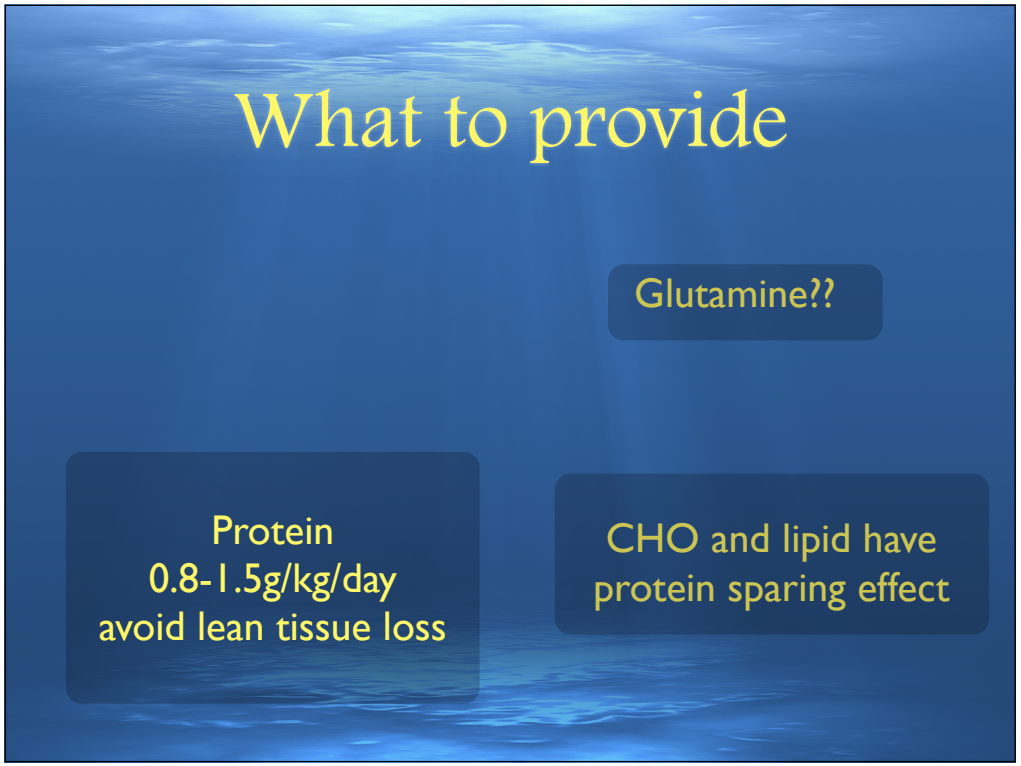
- *b Secondary anorexia*
- *c hypothermia*
- *d as well as the other effects above*

- The observation of ielus settling soon after IVN started

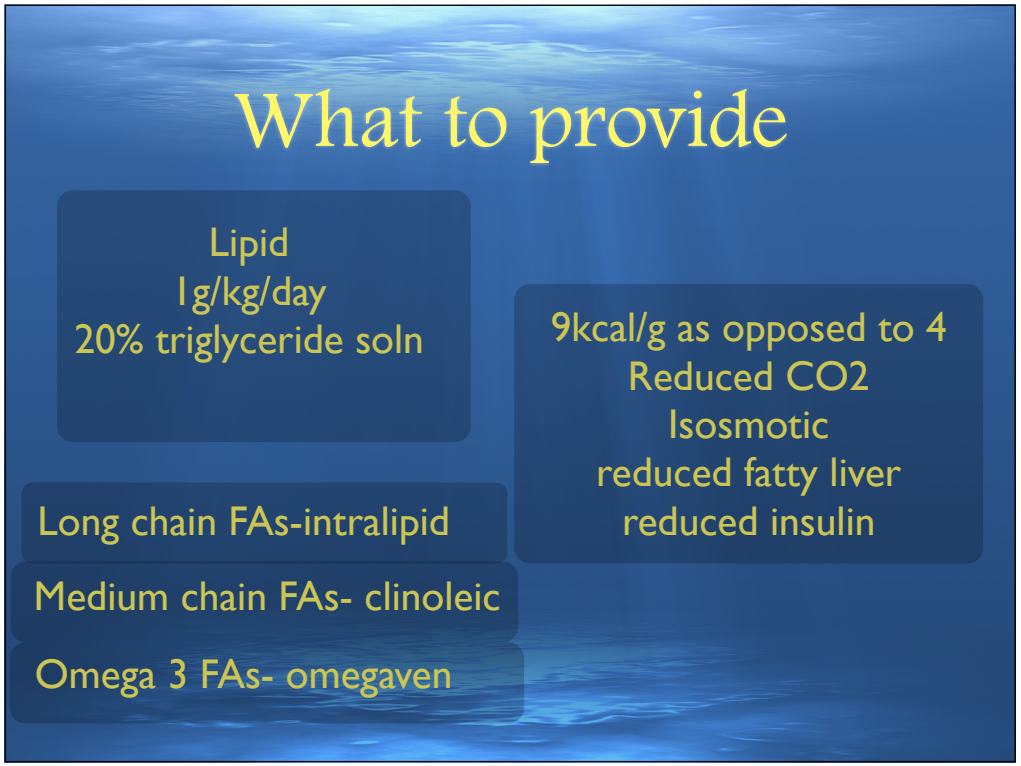
- so we need to be aware of what our goals are- maintenance of lean body mass rather than replacement, energy provision and nutrients for healing.



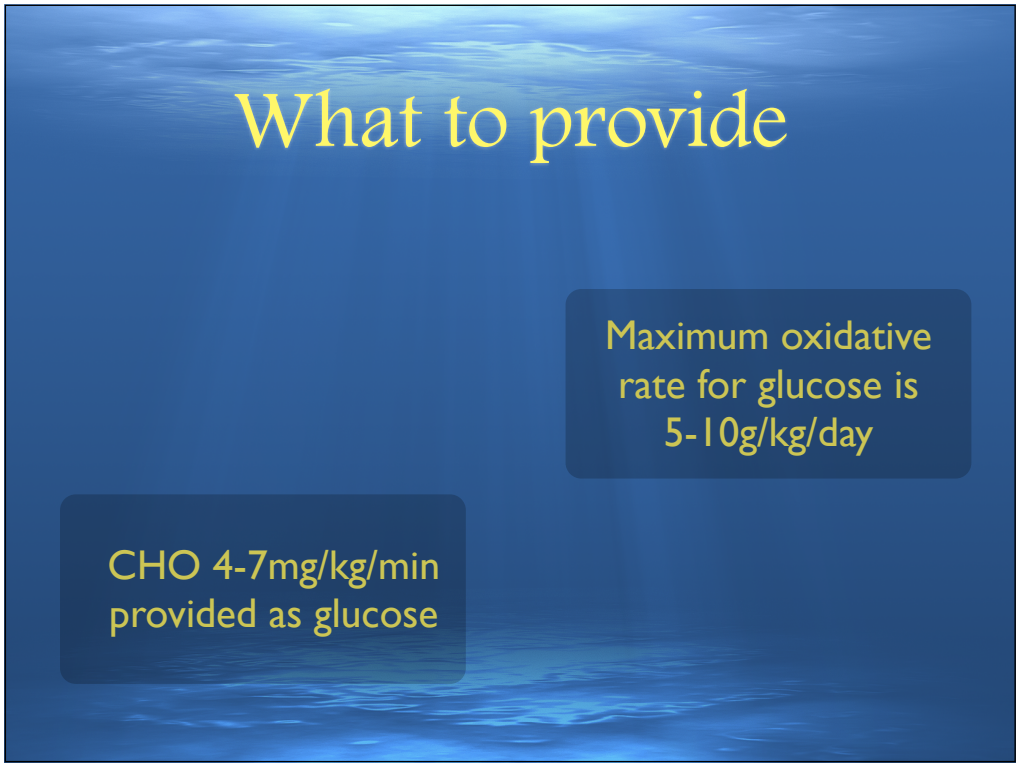
In terms of energy you need to be careful, overfeeding in the critical illness phase is dangerous- teleology favours rest and anorexia during illness- there is good reason for this, our metabolism is in acute phase and not ready to process nutrients- far better to underfeed macronutrients and energy (not micronutrients). Dangers of hypoglycaemia, toxicity of free amino acids, lipid peroxidation etc. tend lower in the acute phase other risk of refeeding may be *Increased CO2 production, hyperglycaemia, hepatic steatosis, increased renal solute load, refeeding syndromes.Using the Harris Benedict equation can lead to overfeeding in up to 30%*



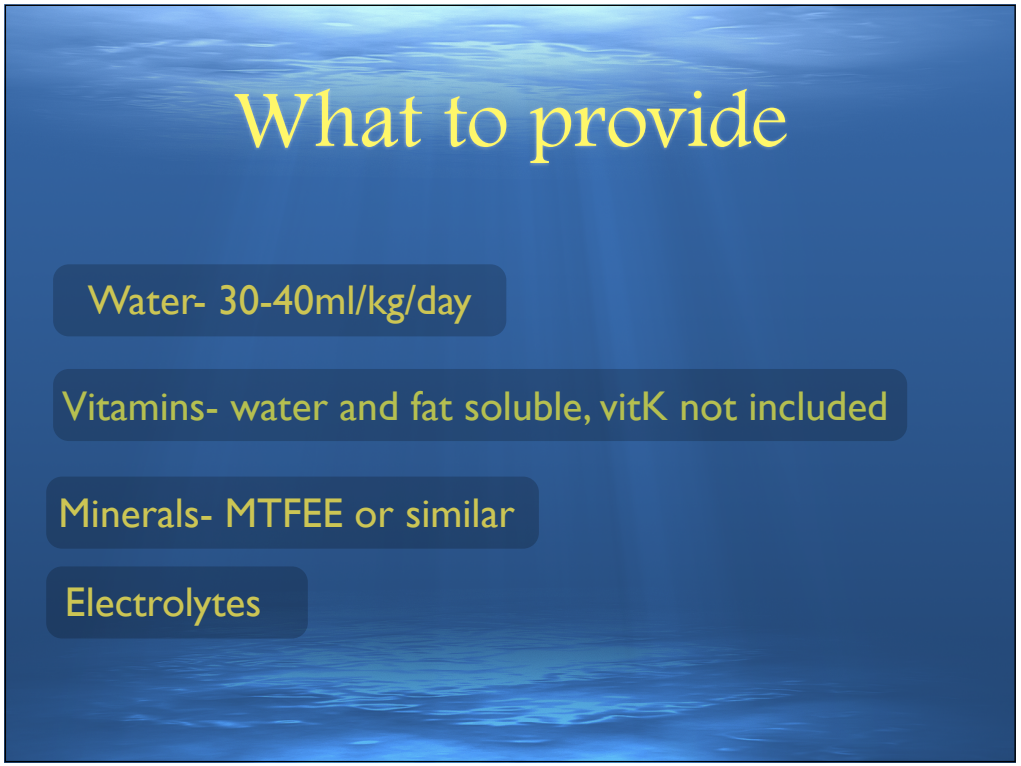
Protein- prefer higher protein and lower energy in acute phase. Remember that protein can be consumed as energy- cho and lipid will spare protein Glutamine is a whole issue in itself, its a conditionally essential amino acid, used as a nitrogen shuttle and an enterocyte food, synthesis of nucleic acids, becomes depleted in acute trauma and sepsis. Variable data on outcomes with its use in the acute setting, doesn't hurt probably.



lipid is usually given as a 20% triglyceride solution, there are a variety available. The basics are the long chain FA eg intralipid based on soybean and safflower oils, more recently MCTs based around olive oil and omega 3 FAs based around Fish oils. A range of purported benefits, none proven beyond reasonable doubt. There are however a range of other benefits to including lipid in your formula, no matter which one you decide on.

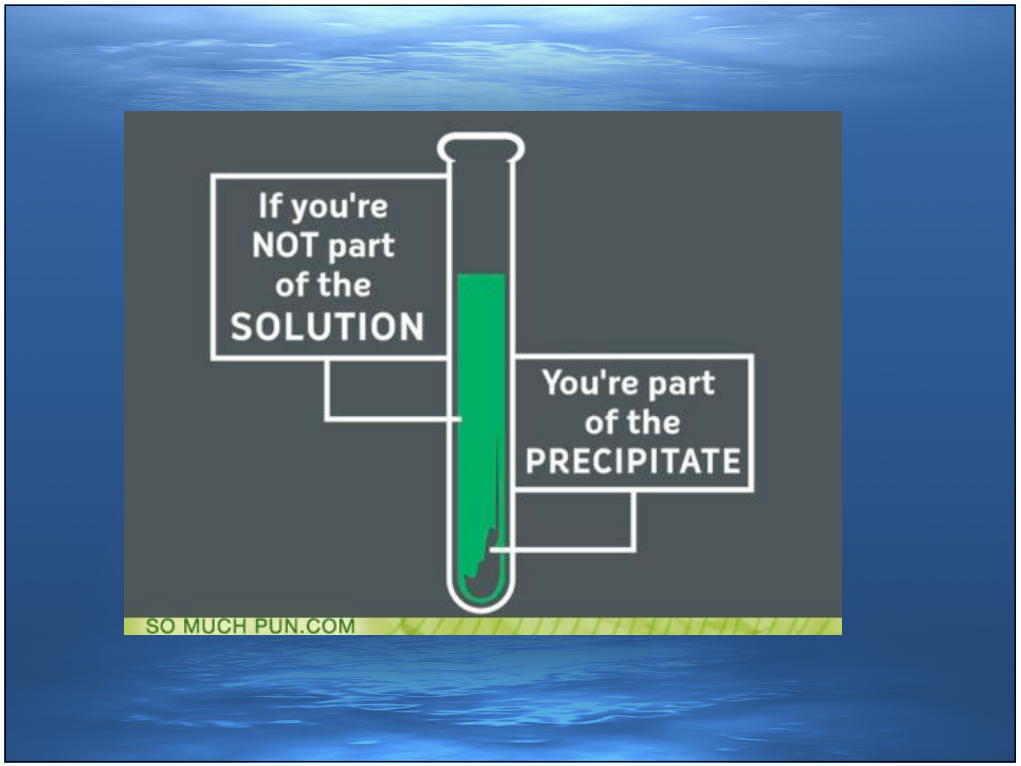


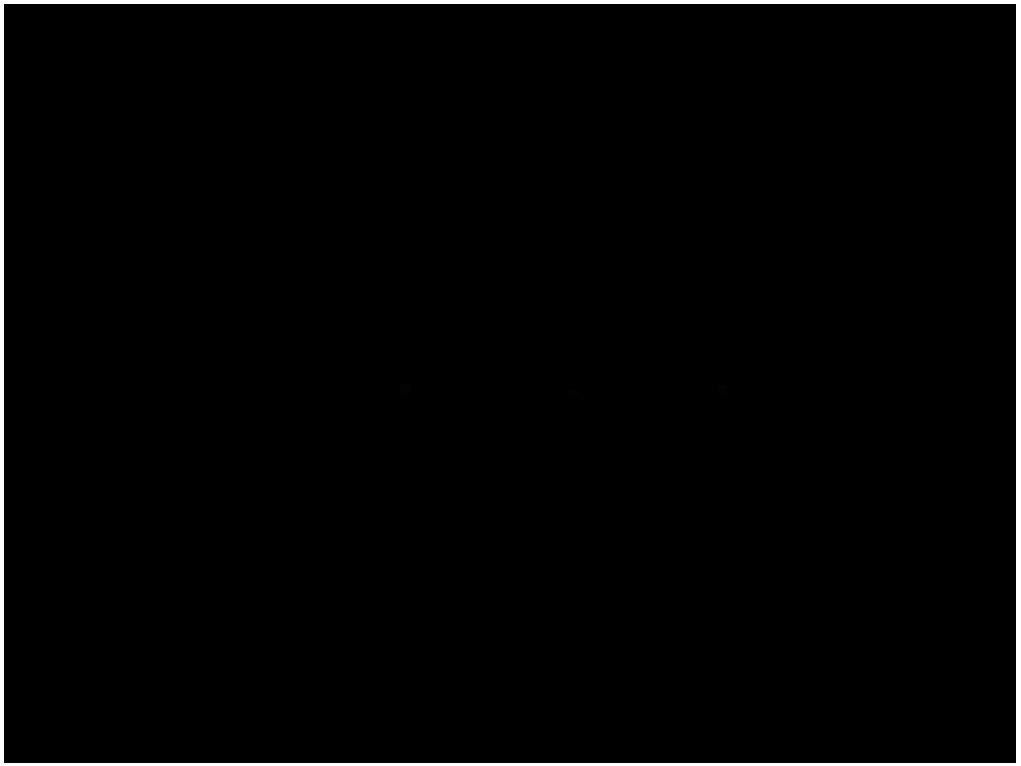
Its very important you don't exceed the maximum oxidative rate for glucose , this is set based on your body weight. Again you'll want to use fewer calories in your sickest patients.



Water provision will of course vary according to losses.
With micronutrients its really important to give the full provision right from the start, you should't start feeding without providing these, the more depleted the patient the more you should give. Vit K isn't included but remember that 60% of vit K is made in the colon anyway, so you just keep an eye on INR. Minerals- some preps don't have Fe so you need to check this. Some preps don't have much zn- often only 3mg/bag, you can lose 20mg/day with a fistula so watch this.

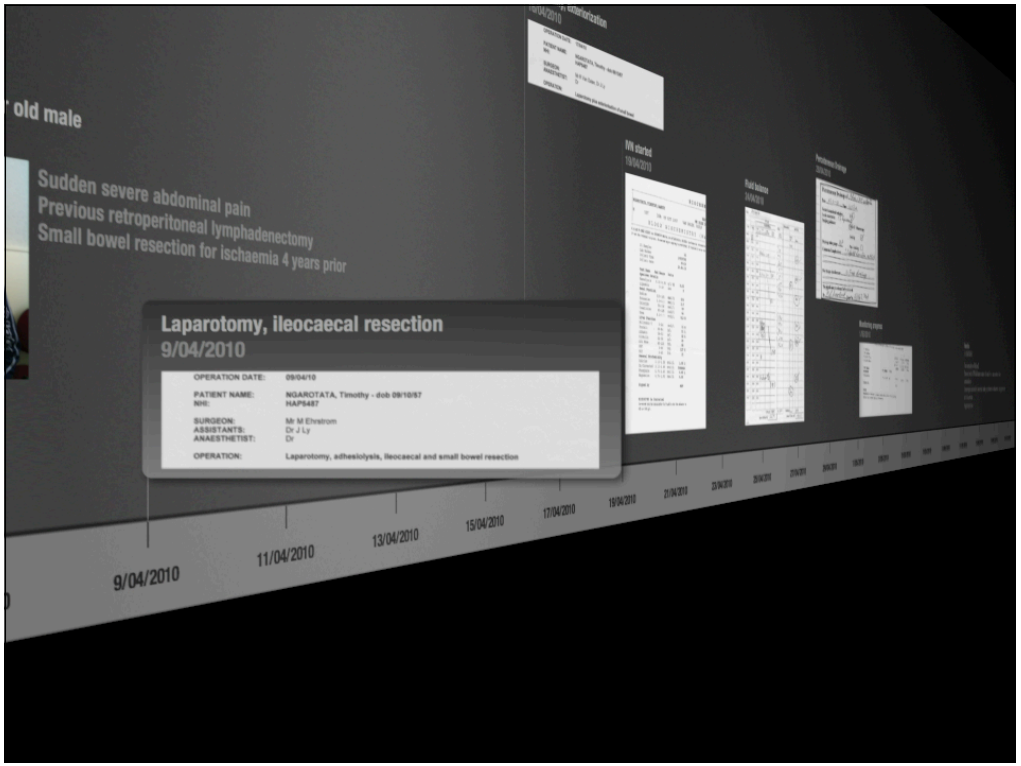
Electrolytes and sodium are a whole other talk so we won't go into that today.


If you're NOT part of the SOLUTION
You're part of the PRECIPITATE
SO MUCH PUN.COM

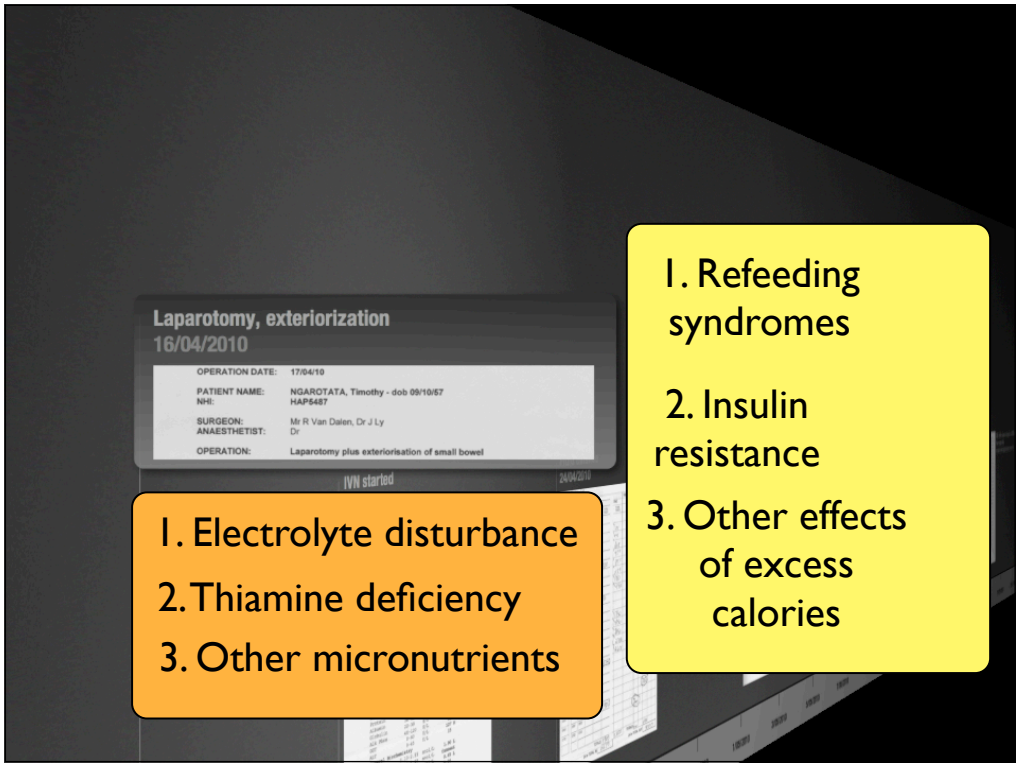


54yr old man, sudden pain, prev. retroperit lymphadenectomy for testicular cancer, 2006 lap and small bowel resection for adhesions, smoker, CT showed thickened bowel

3 days after admission he went to theatre and had some distal small bowel resected with some caecum, very dense adhesions, no mention made of length- good recovery, discharged 5 days later.



2 days later he was readmitteed with more pain, WCC 25 and he went back to theatre- anastomotic leak, bowel exteriorized as a double barrell, noted at that stage 75cm of small bowel remaining, exteriorized with the transverse colon on the right side.



So the next day intravenous nutrition is started, in anticipation of delayed gut function and short bowel syndrome.

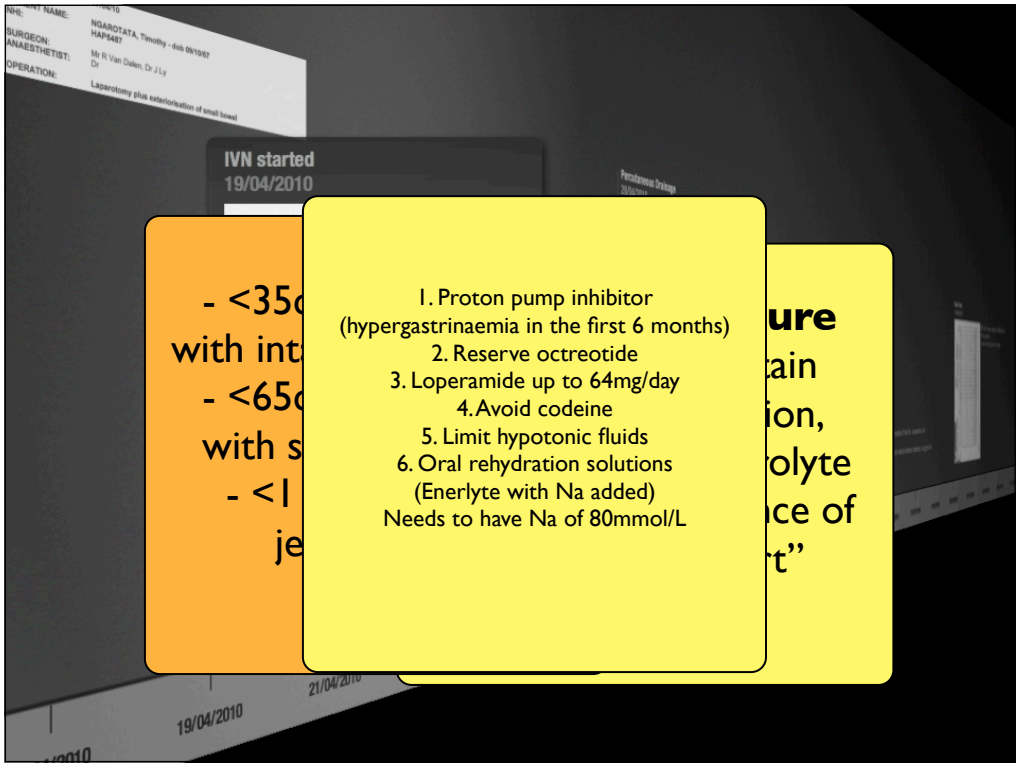
1. What are the concerns at this stage about starting intravenous nutrition.

Refeeding is the first thing that comes to mind

Insulin resistance of critical illness-

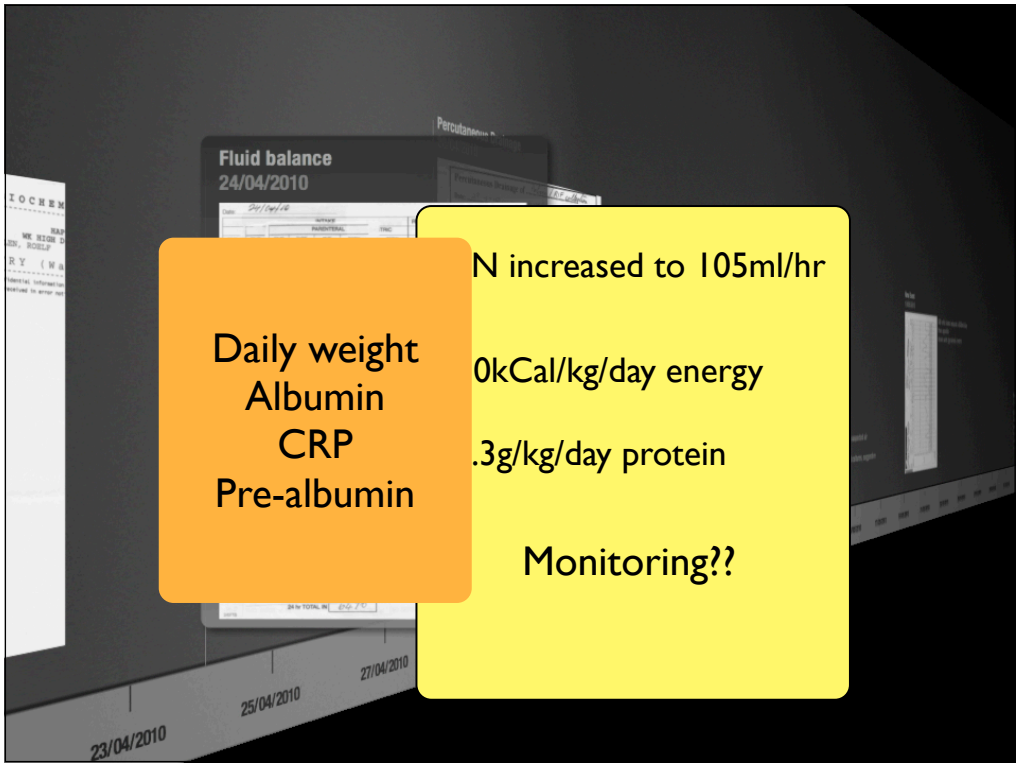
Teleology favours rest after trauma or illness, usually associated with anorexia, the body is in starved state metabolism, the liver is in proinflmmatory mode. Pooring in calories just doesn't make sense at this stage- hyperglycaemia, free aas may be toxic, lipid peroxidation, increased renal solute load from protein, increased CO2 production, heptatic steatosis

Dietician notes suggested 25% of his normal intake leading up to first admission, so a high risk of refeeding sydromes occuring. Also with terminal ileum resection we're thinking about B12 and bile salts- the fat sol. vitamins may take a hit here.



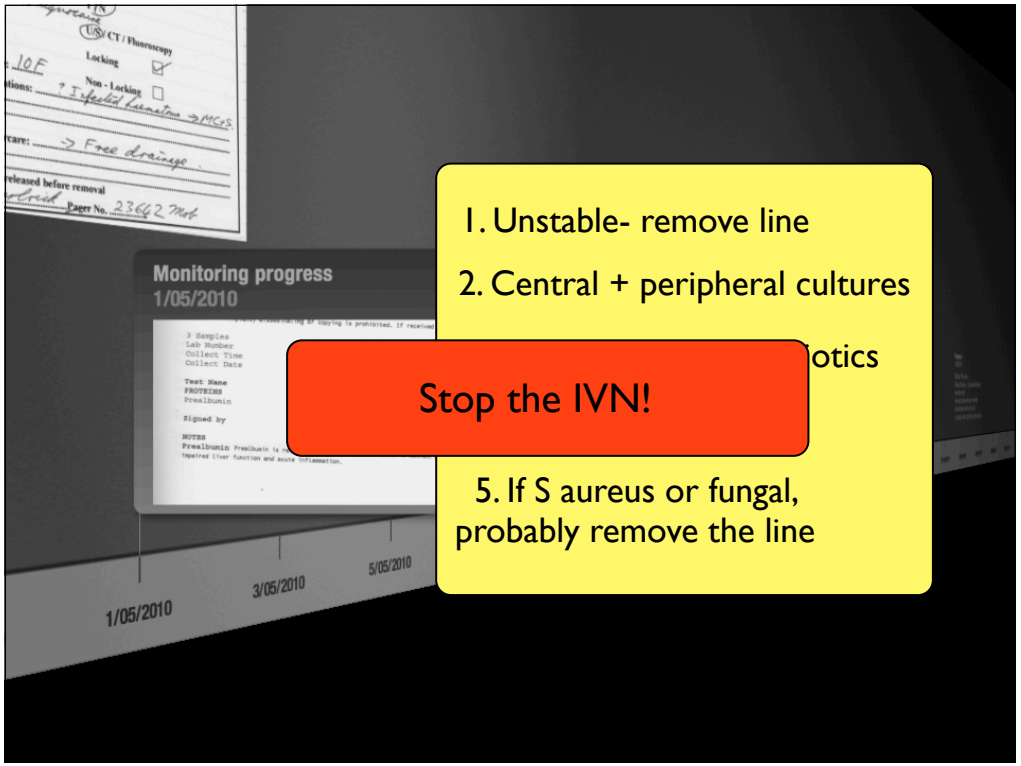
Ok so here we are a couple of days later and we've got issues with high output ileostomy Tim's got >4000ml/day. Before we talk about managing this, the first issues is this- who's at risk for permanent trouble from short bowel/intestinal failure. I would look at the lengths in terms of these features-
You kown in general you should be ok with >100cm small bowel, or >60cm of small bowel with intact colon. In SBS the colon can absorb water, sodium, some AAs and energy fromshort chain fatty acids. Significant variability in jejunal absorbtion can be found between individuals, in general the ileum can take over jeunal function but not the other way around

You get a hypergastrinaemia after massive enterectomy for 6 months- add PPI or H2 blocker. Reserve octreotide, may slow adaption and cause gallstones, loperamide- normally works through enterohepatic circulation so the higher doses are indicated, esp. if TI resected. I limit hypotoinic fluids to 500ml/day. Add in oral rehydration to leverage sodium/gluc cotransport mechanisms.
Remember someone in this situation is going to be a net sodium secretor,at least until they adapt.



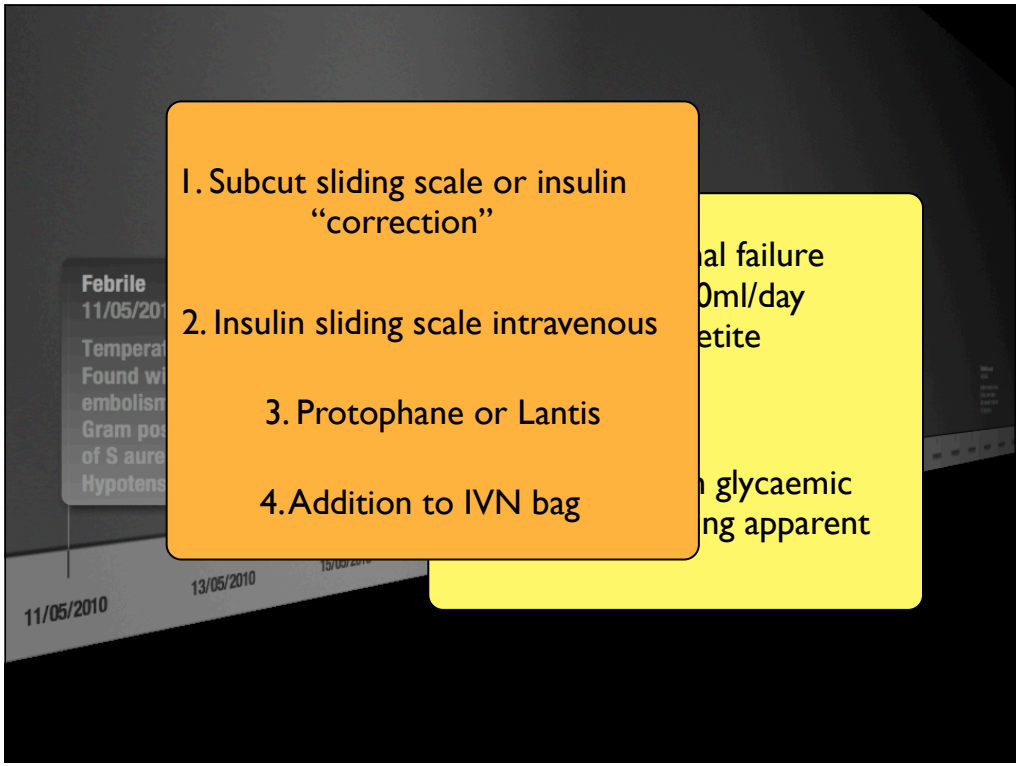
An intra-abdominal collection was picked up at one stage, of course its important to look for these with high output cases, as well as partial obstruction (which you often can't do anything about).

Things had stabilized a little, weight was down 2kg so TPN was increased to 105ml/hr over 24hr giving a total of 30kCal/kg/day and 1.3g protein/kg/day, this reversed the weight loss.
Daily weight important, especially once fluid problems over, albumin and CRP are markers of sepsis and catabolism rather than nutrition.



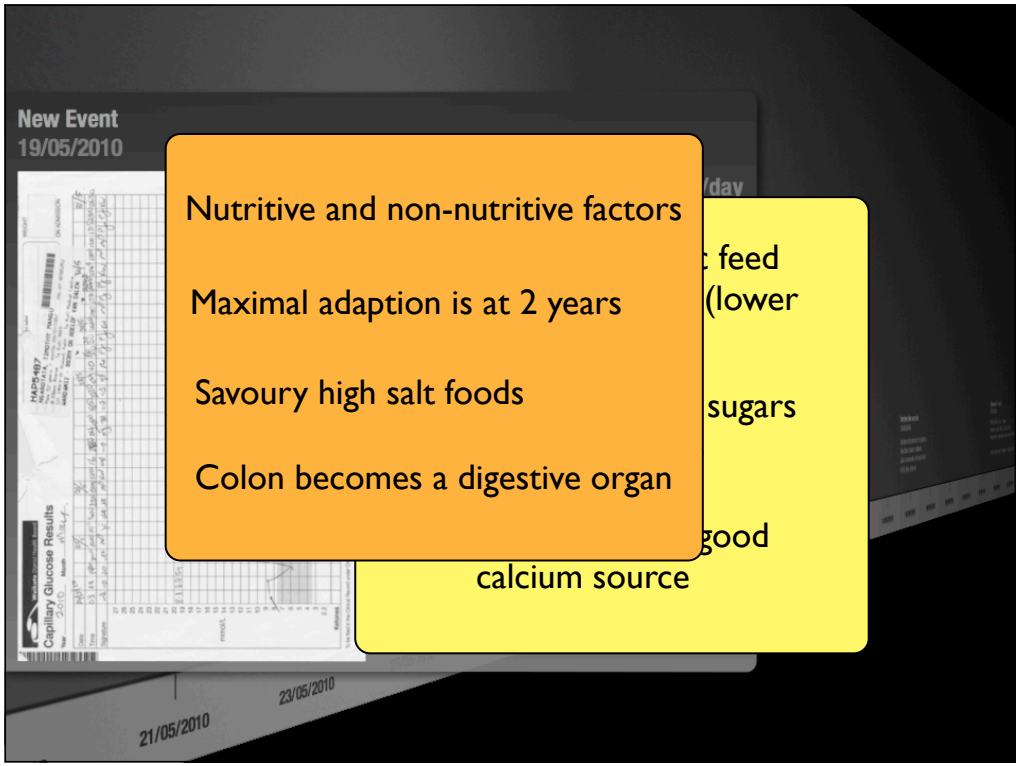
So we're all steaming along OK then on 11 May- Temp 40deg, line disconnection with suspected air embolism, chest pain ?MI, gram pos cocci ?s aureus in all cultures- central line removed. Another one placed 3 days later.

Line infections are a big ticket item. How to manage- they cause a lot of confusion so I'm going to simplify it a lot
Unstable and high index- high temps etc- remove the line
Not unstable- central and peripheral cultures, institute antibiotics and ethanol locks. Stop the IVN- its a perfect culture medium.
If its staph aureus or fungal they're hard to get rid of and you'll probably have to lose the line, other infections you may be able to eradicate, depending on how precious the line is of course.
When replacing the line i'd give thought to an antibiotic coated line,and definitlely institute 70% ethanol locks twice weekly.



So here we are a few days later, still in intestinal failure, poor appetite, still IVN dependant. Now for some reason we see glycaemic issues starting to occur, marked issues in fact. What sort of things are we thinking of in terms of cause and in terms of treatment of this??

Firstly sepsis of course- latent hyperglycaemia, always think of subclinical sepsis, then the gluc/lipid ratio- can you work with this on individualized bags. Preexisting diabetes and steroids can of course confound. There are basically four interventions- from minimally invasive and minimally effective subcut through to addition. Think about absorbtion of subcut, how oedematous the patient is, if brittle iv is best. Adding long acting is more for down the track on overnight cycles. With addition to bag, you'll need to remember a 25% adsorbtion to the bag and it will also mean individual bags.



Now we had a plan for sugar control,we started bringing the cycle down, first step to go to 20 hours- always 4 hour blocks. Feeding quite heavily now to maintain weight- 30 energy and 1.4 protein. We're using full dose loperamide at 64/day with octreotide, oral high salt fluids, iv insulin with some nighttime protophane.

So what about oral feeding, what role does it play in this relatively early phase of short bowel syndrome. There's often a lot of confusion around this, a tendancy to withold food because of the increased stoma outputs that you see.

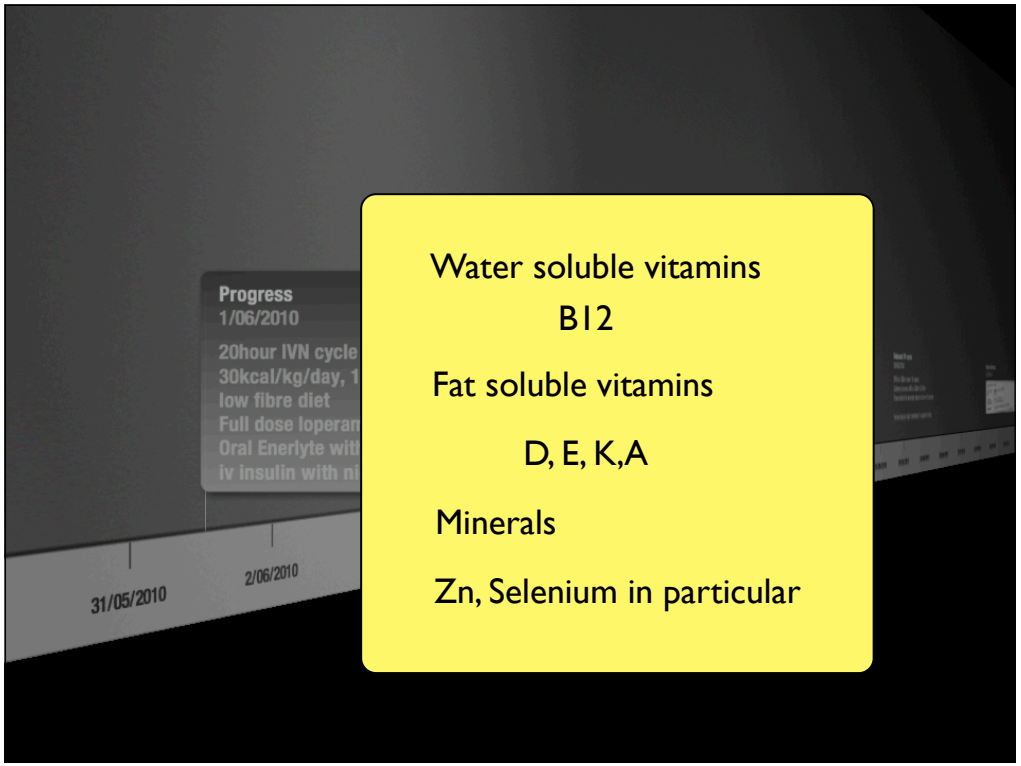
Firstly you need to keep the osmotic load low- complex polymeric feed rather than elemental or semielemental. Avoid simple sugars. Diarly is OK, most dissacharidases are in the proximal jejunum so you don't need lactose free. Mg deficiency when it occurs suppresses PTH, so its particularly important you have a good calcium source to limit metabolic bone disease.

Gut adaption maximises at about 2 years post resection,and it driven by what are called nutritive and non-nutritive factors. Non-nutritive factors would include such things as GH, enteroglucagon, IGF1 etc. There's no doubt that food in the gut is vital for adaption, so as soon as fluid balance is under reasonable control you should start feeding.

Is there anything that can help adaption? Glutamine and GH have been trialled but nothing definitive has come out so nothing is in routine use.

Also important to remember that the colon does become a digestive organ in short bowel syndrome. It produces about 60% of your vitamin K, unabsorbed CHO getting through to the colon can be converted to short chain fatty acids which provide extra energy. Oxalate is one unwanted absorbtion that you get however.

Citrulline <20umol/L suggestive of permanent intestinal failure- citrulline is a byproduce of glutamine metabolism by enerocytes, is used by the kidneys to make arginine.



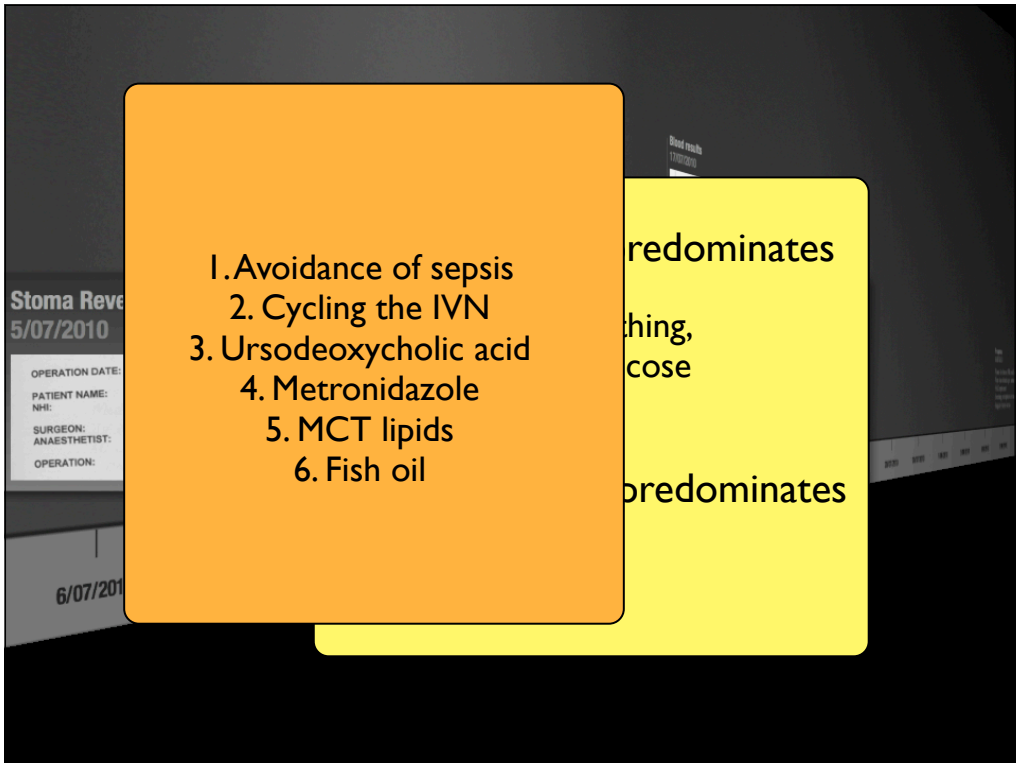
So further along the way he had another line sepsis episode and again the line was replaced, on the 25th of june we dropped to a 16 hour cycle, meaning 130ml/hr for that 16 hours. Plans began to reverse the double barrell stoma to utilize the colon functions that we've already alluded to.

So what about our micronutrients here- we're obviously giving standard multivitamins in our bag but what do we have to keep and eye on.

Water sol- B12 will need to be watched.Folate and B1 should be OK. Fat sol vitamins you will need to keep an eye on because of bile salt loss. VIt K isn't included so do your INR weekly, especially if you haven't got a colon in continuity.

What about minerals- Zn and selenium are the most at risk with high output, there is some in the standard MTFEE preps but not much- say 3mg zinc when you can lose up to 20mg so we check this monthly in high output patients like Tim. The others I'll only do 3 monthly- copper, urine chromium and manganese, vit D and E. Manganese is a funny one- its really a contaminant and accumulates in these patients, high levels can cause neurological problems that are supposedly irrersible.


Reduced IVN cycle
25/06/2010
IVN to 130ml over 16 hours
Ostomy losses still >3000ml/24hrs
Plans afoot to reverse double barrell stoma
?what about micronutrient requirements
Stoma Reversal
2010
24/06/2010
26/06/2010
28/06/2010
30/06/2010
2/07/2010
4/07/2010
6/07/2010
8/07/2010

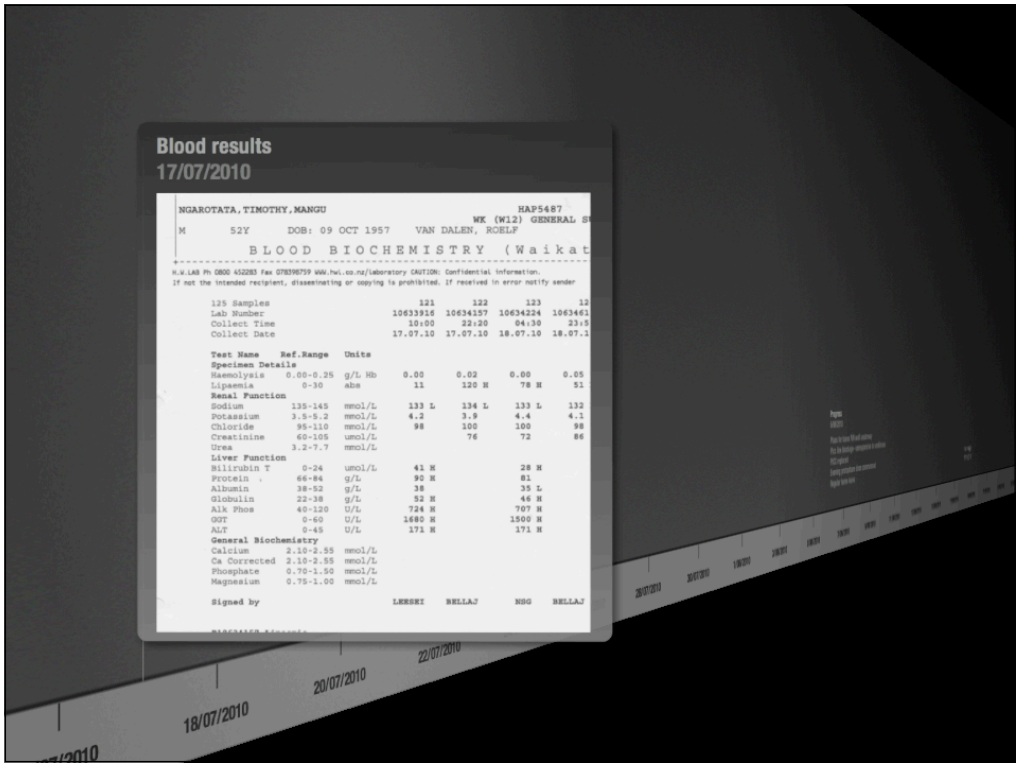


So on the 5th of the July about 3 months had gone by so we did a minilap and reversed the double barrell with an ileocolic anastomosis. At that point we also changed him over to individualized IVN with the sole aim of adding insulin into the bag. He made a reasonable recovery from this, then we started getting problems with the liver functions- here are some typical results from the time- you can see know a cholestatic picture developing.

Abnormal LFTs are part in parcel of TPN of course- and I have to stress the difference between early and late changes. Early changes usually occur very early and are ubiquitous- almost always reversible steatosis. Most of the time it can be ignored. If you're worried review your glucose infusion- if necessary increase the lipid and reduce glucose, maybe reduce total caloric load if you think you can.

Late changes are cholestatic thats what we're dealing with here, the problems are usually more severe and potentially dangerous.

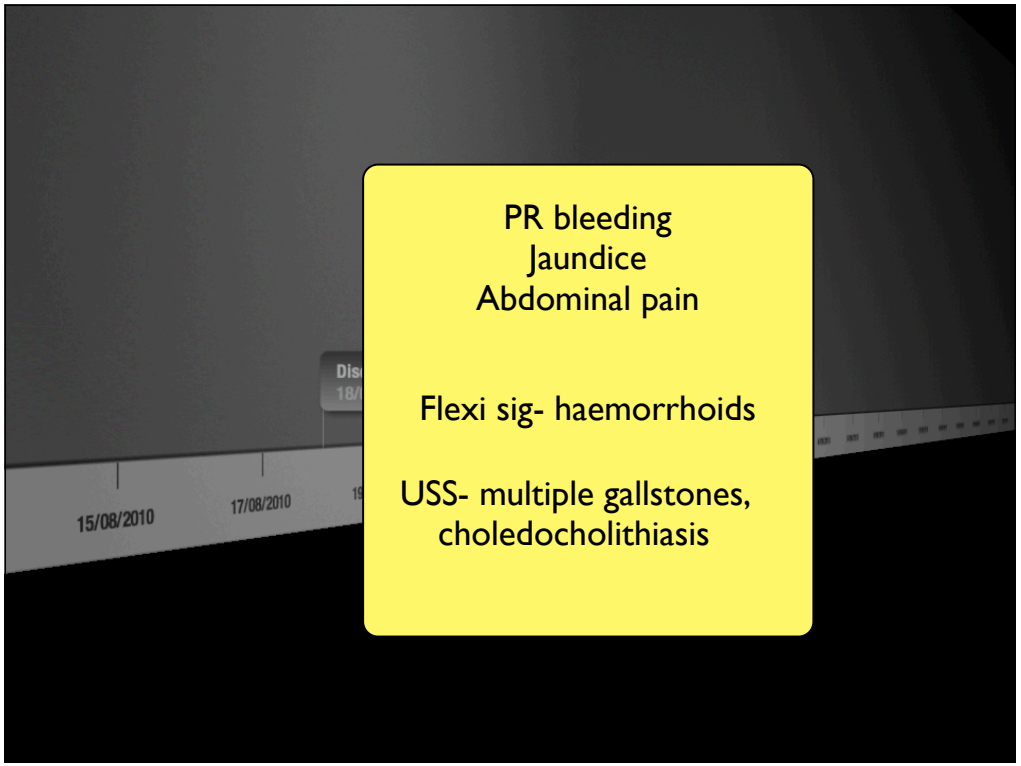
What are some of the interventions we can do for cholestasis, what are some of the strategies?
firstly - avoidance of sepsis, recurrent sepsis is the one thing guarenteed to make cholestasis worse.
Cycling the IVN becomes important- giving the body an IVN free period in the 24hrs for the liver to recover.
Urso- 6-15mg/kg/day- hard to find good evidence but as witnessed later in this patient it definitely has a role to play
Metronidazole- depends if you believe SIBO has a role to play
Different lipids- this is controversial, the drug companies want you to believe, some evidence for MCT based oils such as olive oil and fish oils with omega 3.
Short gut itself is definitely a risk factor for hepatic impairment.



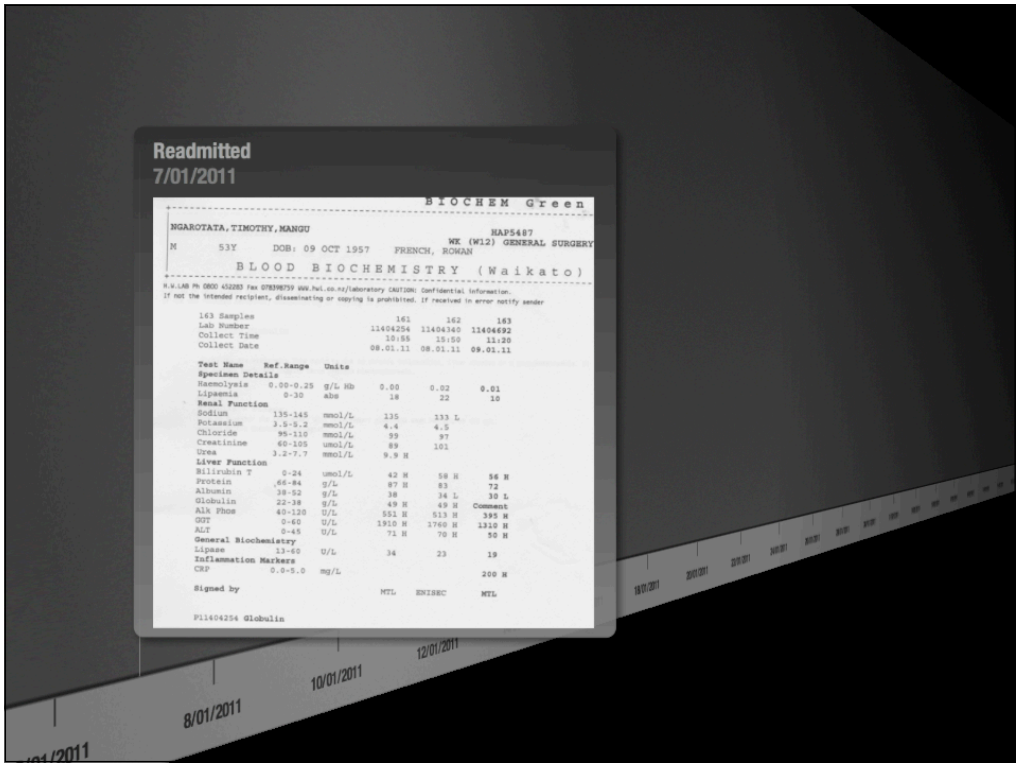
54yr old man, sudden pain, prev. retroperit lymphadenectomy for testicular cancer, 2006 lap and small bowel resection for adhesions, smoker, CT showed thickened bowel

So about 4 and a half months after the whole thing started we were able to discharge Tim home to Te Kuiti, and that should have been the end of the story, but like any good story of course there's a sequel.

At this stage his bowels were working 8-9 times per day, all his micronutrients were OK, h had a total of 55u actrapid in his bag and was self checking BSLs ok.



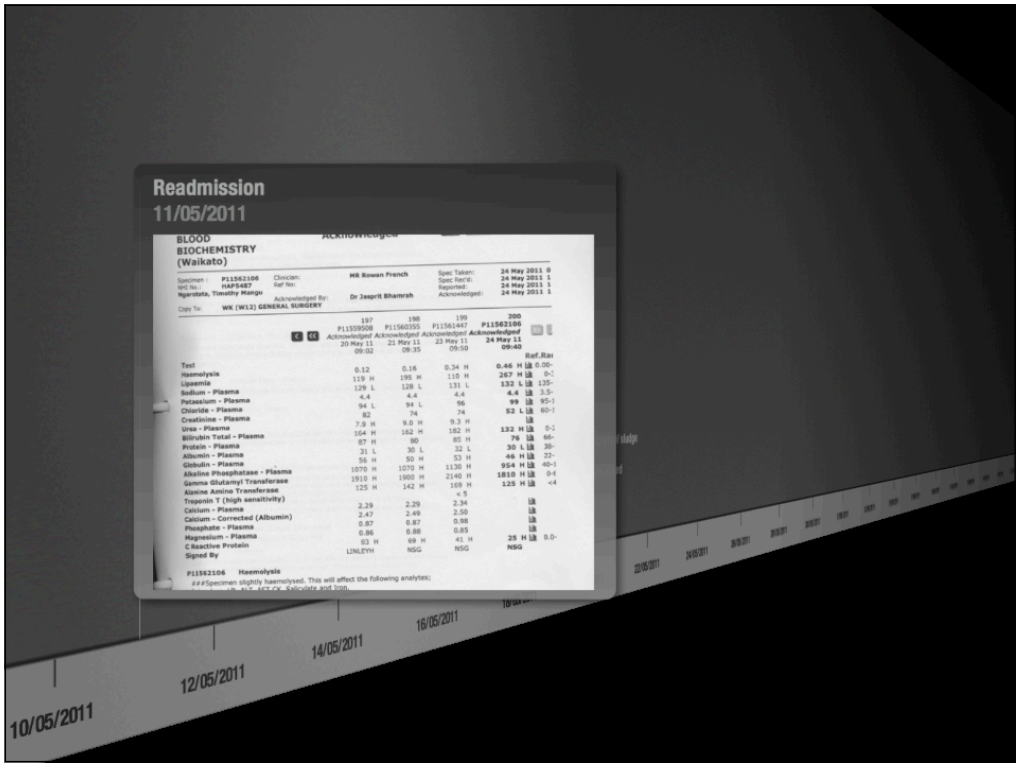
So about 5 months later on 6 Jan he's admitted with some pr bleeding jaundice and abdominal pain. The following blood pictures is shown. You can see there's a very significant bilirubin amongst other things. What are you going to do now- flexi sig then USS
Two ERCPs- multiple pigment stones, not all able to be removed, stent placed. Third ERCP unsuccessful- for surgery



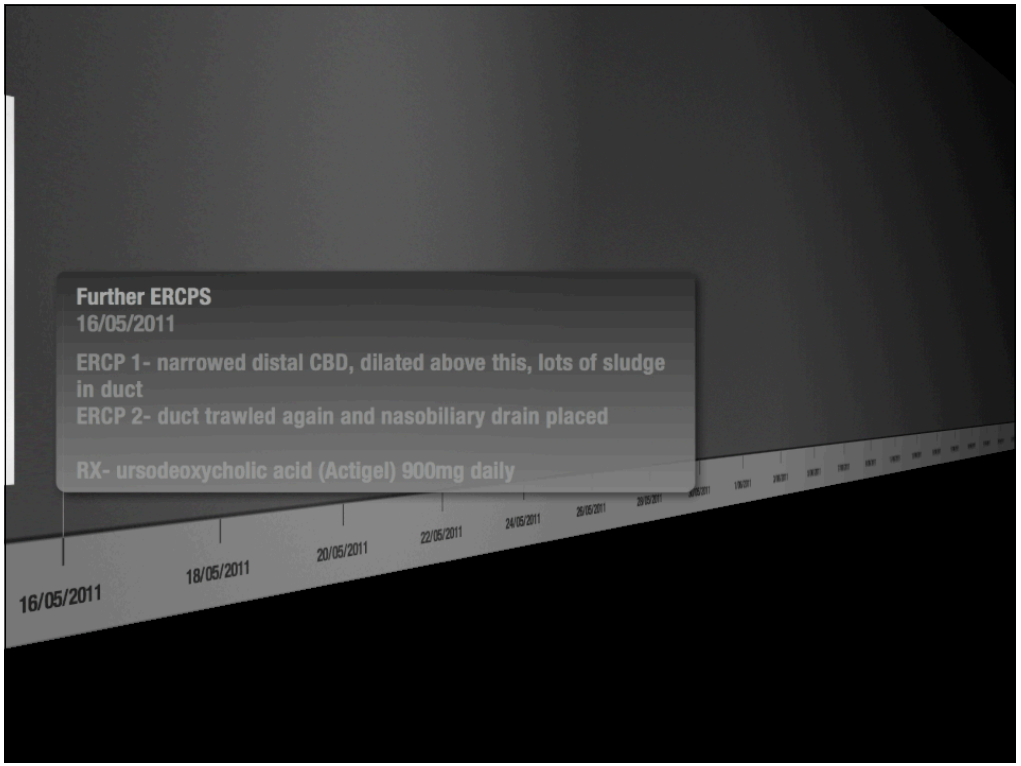
So he was discharged with his stent in situ, and came back on the 16th of march for open cholecystectomy, CBD exploration. What we found was a dilated upper duct, and quite a strictured lower duct around the stent with multiple stones. We talked about options- choledochoduodenostomy, couldn't do choledochojej (SBS). Decided not to leave T tube because of stent, but this probably would have been a good option.

Recovery was good, bili came down somewhat but then on the 11th he was readmitted with the following picture.

yes he was readmitted with even worse jaundice than before, again a cholestatic picture. We got MRCP and again we got this sort of picture, strictured lower duct with sludge blocking the top end. Two difficult ERCPs later and a nasobiliary tube, before we decided to start him on ursodexocholic acid 900mg daily



And slowly but surely the jaundice began to receed. We had planned to change to a metal stent if the actigel didn't work but so far that wasn't necessary. The other question is how long to keep things going. My thoughts were when enteral feeding predominated over IVN would be the right time.



And at last review 2 weeks ago Tim was down to 3 bags per week, stopped urso, good oral intake. The aim is to get him off IVN by 2 years, so far we're on target to dothis.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