Problem Summary:

infant not feeding well; failing to thrive.
Cries during day and wakes at night → FAILURE TO THRIVE

DEFN OF FAILURE TO THRIVE

~ SYMPTOM, NOT A DIAGNOSIS ~

CURRENT GROWTH

→ weight < 3° percentile (NCHS growth chart)
→ weight for height <5° percentile
→ weight ≤ 20% ideal weight for height

GROWTH OVER TIME

→ slowing of weight gain
  <20g/day 0-3m
  <15g/day 3-6m
→ fall-off from prev estab growth curve downward crossing ≥ 2 percentiles
→ documented weight loss

RELATIONSHIP OF GROWTH PARAMETERS TO EACH OTHER

↓ WEIGHT
  ↓ WT & HT
  ↓ WT, HT, head
  ↓ intake
  ↓ loss
  ↓ struct dystrophy endocrine const shortness

In utero insult
Genetic abN

HISTORY What is the cause??

CONTEXT

→ onset +/- Rx ***
→ overall health
→ pregnancy
→ birth (preterm, growth)
→ perinatal
→ current illness/meds/Sx
→ systems:
  • vomiting / appetite
  • stool patts
  • distress/tingr c feeds

PSYCHOSOCIAL

→ 1° environment
→ support systems
→ finances
→ domestic sitn
→ bonding
→ child’s routine

NUTRITIONAL

→ intake
  (schedule, amt, preparation)
→ breast-fed:
  • milk control
  • maternal diet, rest, meds
→ formula-fed:
  • type
  • mixed:
  • 3 day diet Hx
→ infant hunger/satiety cues

PHYS EXAM

(1) ORGANIC DISEASE or ∆
(2) SEVERITY OF MALNUTRITION
(3) ABUSE

COMPLETE PHYSICAL

ABDO: liver, spleen, distension, pain, guarding → peritonitis, IBD, GITI
CARDIO: murmurs → congenital defect, cyanotic heart disease, failure
RESP: → infxn, asthma, bronchiolitis

DEVELOPMENT:

→ milestones
→ behaviour

INVESTIGATIONS DETERMINED BY FINDINGS

→ FBC anaemia, infxn, kidyne
→ serum electrolytes
  & creatinine
→ LFT’s
→ urinalysis (± culture) kidney damage, UTI, starving
CATHETER FOR SAMPLE
→ UEC
  → urinalysis
  → stool analysis
  → hormone assays
  → bone age

↓ INTAKE

↓ appetite
  • chronic disease
  • psychosocial
  • ingestion probs
  • neuro disorder
  • craniofacial dfx
  • dysphoea
  • muscle weakness
  • food unavailable
  • poor feeding technique
  • ↓ volume
  • inapprop food for age
  • abuse

↓ ABSORPTION

Sml intestine
  • Coeliac Δ
  • food intolerance
  • inflamm bowel Δ
  • enzyme defics
Colon
  • Hirschsprung Δ
Pancreas
  • CF
Liver
  • chronic cholestatic Δ
Drugs
  • infections

EXCESS LOSS

Vomiting
  • CNS path (1ICP)
  • GI obstruction
  • metabolic probs
  • gastroesoph, reflux Δ
  • drugs/toxins
Diarrhoea
  • inflamm bowel Δ
  • infxn
  • immunodeficiency
  • Coeliacα / CF (fatty)
Renal loss
  • diabetes
  • tubular acidosis

ABNORMAL USE

Endocrine probs
  • diabetes
  • hypopituitarism
  • inborn errors of metab
  • mitochondrial Δs
Chromosomal Δ
Prenatal insult
  • foetal alcohol Δ

↑ NEED

Congenital heart
Δ

Chronic respiratory Δ
Neoplasms
Chronic/recurrent infxn

Chronic anaemia
Hyperthyroidism

FAMILY

• poverty
• lack of support
• education / experience
• priorities
• social isolation

SOCIETAL

• poverty / chomage
• poor health services
• discrimination

MATERNAL

• bonding because
  • irritability
  • poor

put together by Alex Yartsev: Sorry if i used your images or data and forgot to reference you. Tell me who you are.
aleks.igorevich@gmail.com
<table>
<thead>
<tr>
<th>GOAL</th>
<th>OPTIONS</th>
<th>comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>↑ WEIGHT &amp; GROWTH&lt;br&gt;- weight gain with ↑ intake confirms diagnosis, keep monitoring</td>
<td>(1) ↑ caloric intake (aprop diet for catchup)&lt;br&gt;(2) supplementation: ↑fats, polyjoule etc.&lt;br&gt;(3) hospitalization&lt;br&gt; - high risk abuse / severe neglect&lt;br&gt; - medically unstable (severe malnutrition)&lt;br&gt; - failed trialled outpatient mgt</td>
<td>Cost</td>
</tr>
<tr>
<td>TREAT UNDERLYING DISEASE</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PARENTAL SUPPORT</td>
<td>(1) Antenatal (preventive)&lt;br&gt;(2) Education programs (+/-residential)&lt;br&gt; → Tresilian / Karitane&lt;br&gt;(3) Early childhood support services&lt;br&gt;(4) Social network (mothers’ groups)&lt;br&gt;(5) Encourage paternal/family involvement</td>
<td></td>
</tr>
<tr>
<td>EVALUATE MATERNAL DEPRESSION</td>
<td>(1) Edinburgh Post-natal depression scale&lt;br&gt;(2) Anti-depressant meds / Counselling</td>
<td></td>
</tr>
<tr>
<td>MONITOR GROWTH</td>
<td>Weekly visits: GP &amp; Community Nurse&lt;br&gt; Multidisciplinary team - 1 ° caregiver&lt;br&gt; - nutritionist&lt;br&gt; - social worker&lt;br&gt; - child behaviour&lt;br&gt; specialist&lt;br&gt; services&lt;br&gt; Monitor ALL CHILDREN</td>
<td></td>
</tr>
<tr>
<td>PROTECT CHILD</td>
<td>DOCS</td>
<td></td>
</tr>
<tr>
<td>REFER TO PAEDIATRICIAN</td>
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</tbody>
</table>
The GIT develops as an outpouching of the yolk sack.

**WEEK 3:**
the yolk sack is fully formed.

**WEEK 3.5:**
The yolk sack protrudes through into the embryo and forms two pockets which are the very first bits of the foregut and hindgut. The Midgut is still part of the yolk stalk.

**WEEK 4:**
The embryo is now roughly a tube; the ventral cavity (coelom) contains the now-complete fore, mid and hind-guts. The yolk sack grows smaller.

**WEEK 5**
A loop of the midgut begins to protrude through into the umbilical stalk, while the rest distends and prepares to separate into the stomach, spleen, pancreas and liver. The yolk sac has become one with the midgut.

**WEEK 6**
The midgut loop elongates rapidly; its superior limb becomes the jejunum, and the inferior limb becomes the colon and appendix. The liver grows from the ventral mesentry; the spleen and pancreas grow from the dorsal mesentry.

Later in **WEEK 6:** section through midgut
The organs rotate 90 degrees to the right. This process begins in week 5.

**RELATIONSHIP OF ADULT TO EMBRYO:**
- **Foregut:** supplied by coeliac trunk
  - Oesophagus, stomach, proximal half of duodenum
- **Midgut:** supplied by superior mesenteric artery
  - Distal duodenum, jejunum, ilium, caecum + appendix
  - ascending colon, 2/3rds of transverse colon until the splenic flexure
- **Hindgut:** supplied by inferior mesenteric artery
  - Left colic flexure, descending + sigmoid colon, rectum and upper anal canal.

**Distribution of pain referral:**
Refers via sympathetic afferents
- foregut
- liver
- spleen
- appendix
- kidney + ureter
- hip
- midgut

**Rotation of the midgut loop**
Thus the colon assumes its ascending-transverse-descending position

**The Loop of Midgut**
Anticlockwise rotation of this loop around the axis of the Superior Mesenteric Artery will position the ascending, transverse and descending colon in its normal adult position.

**Later in WEEK 6:** section through midgut
The organs rotate 90 degrees to the right. This process begins in week 5.

**!! the ANUS and the MOUTH arise from different structures, are innervated separately from the gut, and receive a different blood supply.**
POVERTY AND CHILD HEALTH

DEFINITIONS:

**ABSOLUTE poverty**
- Income insufficient to maintain basic subsistence; **cannot afford food or shelter** eg. Dickens
- 1995: one QUARTER of the total world population fits into this category: **less than 1 dollar per day**
- this figure has **DOUBLED SINCE 1975**

**RELATIVE poverty**
- income insufficient to pay for basic social roles, participate in relationships etc.
- the exact figure is determined by the experts, eg. the “HENDERSON POVERTY LINE”
- henceforth “poverty” in this summary will allude to this estimate

How to determine who’s poor and who’s not:
- “expert” estimate of how much it costs to subsist in Australia, at a given time
- a conservative estimate: NOBODY WILL ARGUE that those below this line are “very poor”
- lecturer quoted about $290 as being the poverty line for a single white female living alone

**Distribution in Australia**
12% live in poverty: 1.7 million people
13% of children live in poverty: 440,000 kids

**Specific groups:**
- of single parents 29% are in poverty
- of couples with 1 child 5% are in poverty; ...of couples with 3 kids- 11%

**Why does this matter?**
- There exists a direct relationship between income and life expectancy
- Chronic illness and hospital admissions are more frequent in unemployed population
- Chronic illness is more common among children of the unemployed
- Children of the poor are less likely to be breastfed

**How has this changed?**
- Rates of breastfeeding, child mortality (including indigenous population) and birth weight have improved
- Not much else has

**Policies and Programs directed at child health: a timeline for a hypothetical infant**

<table>
<thead>
<tr>
<th>Conception</th>
<th>Birth</th>
<th>2 years</th>
<th>5 years</th>
<th>12 years</th>
<th>18 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Advocacy - Enhance social, political, economic and physical environment, legislation (eg. Gun control), structural changes (eg housing design)</td>
<td>Promote socio-economic equity</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>
**DIGESTION & ABSORPTION OF NUTRIENTS**

**WATER & SALT - ABSORPTION @VILLUS**
Water follows solutes: Na, Cl, HCO₃⁻. Solutes transported by ACTIVE TRANSPORT & LEAKY TIGHT JNCS
Main players: Na enters cell unwarranted & pumped out by SODIUM PUMP (Na⁺K⁺ ATPase) @ basolateral membrane
(1) Na cotransported with glucose/aa OR
(2) Na exchanged for H⁺ etc. via NHES
→CREATES OSMOTIC GRADIENT FOR WATER TO FLOW DOWN
(3) Aquaporins (no Na in), DRA mutin (no Cl in) → DIARRHOEA

**WATER & SALT - SECRETION @CRYPT**
(1) osmotic gradient established by fluid in lumen OR
(2) crypt secretes Na⁺Cl⁻, water follows
CFTR = CAMP dependent Cl⁻ channel
Main players:
SUPPLY = Na⁺K⁺2Cl⁻ cotransporter
ACTIVATOR = VIP
CHANNEL = CFTR
→CF=CFTR mutation malabsorption
→bacterial toxins (cholera)
(1) keeps cAMP high,
(2) blocks vili/crypt differentiation → MASSIVE DIARRHOEA
(3) stops at cell turnover (days)

**GLUCOSE**
POLYSACCHARIDES →salivary AMYLASE
MOUTH →pancreatic AMYLASE
SMI INTESTINE →OLIGOSACHS & DISACCHS
→brush border E's:
MALTASE, LACTASE, SUCRASE
SMI INTESTINE →MONO-SACCHS

**PEPTIDES**
PROTEIN →PEPSIN STOMACH (HCI)
SMI INTESTINE →LGEPOLYPEPS →pancreatic E's TRYPSIN
CHYMOTRYPSIN, CARBOXYPEPTIDASE
SMI INTESTINE →SMI POLYPEPS / PEPTIDES
→brush border E’s
SMI INTESTINE

**LIPIDS**
3-GLYS (FAT GLOBULES) →BILE SALTS emulsification
→pancreatic LIPASE
SMI INTESTINE →MONO-GLYS & FA’S (MICELLES)

**IONS**
IREG-1 = Fe regulated transporter
→low Fe = open, Fe enters blood
→high Fe, stored as Ferritin
HEPHAESTIN regulates IREG expression

**LARGE ORGANICS**
B12 + IF
Endocytosis
B12 + transcobalamin
Lysozome recycled

**TYPICAL ABSORPTION in sml intestine**
TIGHT JNCS at luminal surface means nutrients must go THROUGH epithelial cells, not BETWEEN

**VILLUS EPITHELIUM**

**To capillaries**

**Lumen SML INTESTINE**

**3-GLY resynth**
If >10 C chains in FA straight to blood

**FERRITIN**

Fe binds to Transferrin in Blood

**ACTIVE TRANS**

**ACTIVE TRANS**

**SIMPLE DIFFUSION**

**VESICLE TRANS**

**NA+**
**K+**

**Glucose mannose galactose**

**Fructose**

**Glucose mannose fructose**

**AA’s**

**3-GLY resynth**
If >10 C chains in FA straight to blood

**Peptides**

**H+**

**H+**

**Fe**

**B12 + transcobalamin**

**IF**

**Lysozome** recycled
MICROSCOPY OF THE GUT:

**OESOPHAGUS**
- 2x layers of muscularis externa: upper 1/3 = striated muscle
  - lower 1/3 = smooth muscle
  - middle 1/3 = mixed histology
- Foamy-looking glands in the submucosa, secreting mucous for lubrication
- Submucosa mainly made of connective tissue
- Papillae are present to anchor the stratified squamous epithelium
- PLUS there is a properly visible muscularis mucosae

**STOMACH**
- 3 layers of muscularis: oblique (inner), circular, longitudinal (outer)
- Muscularis also contains the glands at the bottom of the pits
- Glands are filled with parietal (acid) and chief cells (enzymes)
- Plus the neck has mucous cells and there are enteroendocrine cells throughout
- Mucosa is a simple columnar epithelium, “The Surface Mucous Cell” Cells migrate from the bottom of the pit (where the stem cells are) out to the top of the hill where they die and slough off; this is a common path for all the columnar cells in the GIT

**DUODENUM**
- Is dominated by villi and the crypts between them: V. absorb, C. secrete
- Villi are filled with loose connective tissue, strands of smooth muscle, lacteals and plasma cells
- 2 layers of muscularis, transverse and longitudinal (for peristalsis)
- **CHARACTERISTIC FEATURE!!** are the Brunner’s Glands, secrete mucus
- There is also a lot of goblet cells throughout the small intestine

**JEJUNUM**
- Is unremarkable by anything except its lack of features which distinguish the duodenum and the ilium;
- ...but this is where most of your absorption happens
- Villi are rather longer and more finger-like in the jejunum than in the duodenum

**ILIUM**
- Ayerbach’s plexus: between the layers of muscularis, there are ganglia of the enteric nervous system; neurons project to the circular muscle, to other myenteric ganglia, to submucosal ganglia, or directly to the epithelium, and play an important role in regulating and patterning gut motility.
- **CHARACTERISTIC FEATURE!!** Peyer’s patches: pieces of MALT, (Mucosa-Associated Lymphoid Tissue) nodular, contain antigen-presenting cells and lymphocytes.

**COLON**
- Distinguished from the above by presence of longitudinal tenia coli muscle,
- And sack-like projections called haustra (plus there are fatty epiploic appendages which hang off the colon like Christmas ornaments)
- PLUS there is a properly visible muscularis mucosae
- Straight tubular glands (crypts of Lieberkühn) extend through the full thickness of the mucosa
- The appendix is characterised by large numbers of lymph nodules. In many adults, the normal structure of the appendix is lost and replaced by fibrous scar tissue.

**ANO-RECTAL JUNCTION**
- Rapidly does the simple columnar epithelium shapeshift into stratified columnar,
- Then stratified cuboidal
- Then stratified squamous
ALIMENTARY CANAL

DIFFERENT TISSUE TYPES PREDOMINATE @ DIFFERENT AREAS → FUNCT DDS

MUCOSA (MUCOUS MEMBRANE) 3 SUBLAYERS

EPITHELIUM: SIMPLE COLUMNAR EPITHELIUM, ++ GOBLET CELLS
- mucus: protects against autodigestion, lubricates passage
(@ stomach, small intestine ENZYME/MUCOUS-SECRETING CELLS)
Lamina Propria: LOOSE AREOLAR CONNECTIVE TISS. CAPILLARIES
- nourish & absorb nutrients
LYMPH NODULES: protection against gut pathogens
MUSCULARIS MUCOSAE: SMOOTH MUSCLE (thin) - motility of mcosa

SUBMUCOSA: mod dense CONNECTIVE TISS.
1) BLOOD & LYMPH VESSELS
2) LYMPHOID FOLLICLES
3) NERVE FIBRES

- mobility of mcosa

MUSCULARIS (EXTERNA): SMOOTH MUSCLE 2 LAYERS
- peristalsis & segmentation
INNER CIRCULAR - thicken to form sphincters
OUTER LONGITUDINAL

SERRA = VISERAL PERITONEUM
AREOLAR CONNECTIVE TISS. covered with MESOTHELIUM (single layer of squamous epithelial cells) → protection
- oesoph. replaced by ADVENTITIA FIBROUS CT
- esoph. to surrounding strucuts
- retroperitoneal organs: both serra & adventitia

TONGUE
Ant 2/3 = Oral cavity, post 1/3 = oropharynx
- NO SUBMUCOSA
SKELETAL MUSCLE: runs in 3 perm planes
LINGUAL FRENULUM: severs to floor
STRAITFED SQUAMOUS EPITHELIUM
(keatitized)
PAPILLOA (ant 2/3)
- filiform: lots, keratin
- fungiform & circumvallate: Taste buds
SEROUS GLANDS: flush f & for taste buds

SALIVARY GLANDS
MAJOR: (OUTSIDE ORAL CAVITY)
PAROTID (serous), SUBMANDIBULAR (mixed), SUBLINGUAL (mucus mostly)
= COMPOUND TUBULOCALYCEAL GLANDS
serous acini + mucous tubules = ducts (continuous w/ oral epithelium distally)
MINOR: (ORAL CAVITY MUCOSA) continuous secretion for moisture
**Serous: watery, enzymes, ions, ~mucin
**Mucous: (mucin)

(ORO) PHARYNX
STRAITFED SQUAMOUS EPITHELIUM (no keratin)
MUCOSA: LP = DENSE FIBROELASTIC MUSCULARIS ext = SKELETAL (pharyngeal constrictors)
ADVENTITIA

OESOPHAGUS
*STRONG ELASTIC (conduction & protection)
* MUCOSA (tubulocarinal mucous glands)

STOMACH
CHEM DIGESTION (secretion) MECH (muscle churning)

SMALL INTESTINE
CHEM DIGESTION (secretion) ABSORPTION
3 ways to max surface area!
B. folds + plicae circulares
- mucus & submucosa
- villi + epithelium
C. Cell folds = microvilli
- brush border

COLON
FLUID ABSORPTION
MUCUS
MUSCULARIS SPHINCTERS

RECTUM

STRATIFIED SQUAMOUS EPITHELIUM
Non-keratized, THICK
APC’s (Langerhans)

LP: *oesoph cardiac glands
(ends)

ELASTIC network of LONGIT & CIRCULAR s.m.

COARSE COLLAGENOUS FIBRES, ELASTIC FIBRES
LONGIT FOLDS: allow expansion
oesoph: glands proper (sub)

+++ Locytes, epithel. mast cells

SIMPLE COLUMNAR
RUGAE (folds) → expand SECRETORY SHEET of columnar cells = neutral MUCIN
- protect from acid peptic
- lubrication
GASTRIC PITS tiny tubular infoldings of epithl - glands at bottom of pits
- cardia: MUCOUS
gastric: (+++ thru-out)
MUCOUS NECK CELLS - acid mucin
PARIEL CELLS = HCl, IF
- CHIEF CELLS = pepticogen
ENDOCRINE = G/H, Glucagon, pancreatic peptide
Panel: MUCOUS ENDODERM - gastrin
+++ Locytes, epithils, mast cells

SIMPLE COLUMNAR
VILLI, CYRTOPS, GLANDS ENTEROCYTES = main type of absorptive cell
- digestive: enzymes for disaccharides + polypeptides
- absorptive: protein, a/s, carbo’s (to capillaries in villi)
- lipids (to lymphatic channels)
- goblet cells = least
- mucus (lubricates, protects)
- lymphatic vessels
- navigate through villus

SIMPLE COLUMNAR
CRYPTS ~ NO VILLI ~ GLANDS bigger, closer
ENTEROCYTES
GOBLET CELLS
- no enzymes secreted
- digestion still occurs
- enzymes within

SIMPLE COLUMNAR
RECTAL COLUMNS: longi folds
ANAL VALVES distally: transverse folds
GLANDS
GOBLET CELLS
mucous mucosae
→ ALL END HERE!!

→ STRATIFIED SQUAM
EPITHELIUM (nokaterinized)
→ S4E (keratized)
@: External anal sphincter
!! SKIN HERE!!

CIRCUMANAL GLANDS
→ oily material

HAEMORRHoidal
PLEXUS ++veoids
(haemorrhoids-distension & protrusion into mcosa)

INTERNAL ANAL
SPHINCTER
inner CIRCULAR thickens
- outer folds away
EXTERNAL ANAL
SPHINCTER
skeletal m
surrounds dist anal canal

HAEMORRHoidal
PLEXUS ++veoids
(haemorrhoids-distension & protrusion into mcosa)

INTERNAL ANAL
SPHINCTER
inner CIRCULAR thickens
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skeletal m
surrounds dist anal canal
GLANDS

VILLI = PROJECTIONS INTO LUMEN covered with MATURE ABSORPTIVE ENTEROCYTES also GOBLET CELLS: short-lived, shed

CRYPTS = INVAGINATIONS OF EPITHELIUM AROUND VILLI lined with YOUNG SECRETORY EPITHELIAL CELLS

@ BASE = STEM CELLS constantly dividing to replace all epithelial cells on villi & crypts.

DYNAMICS

Stem cells commit to 1 of 4 lines: (1) ENDOCRINE (2) GOBLET (3) PANETH (4) ENTEROCYTE; these differentiate as they ascend SECRETORY → ABSORPTIVE ABSORPTION

→ capillary bed → systemic circulation

→ (fats) lymphatic vessel; CENTRAL LACTEAL → thoracic duct → circulation

STOMACH

GASTRIC PITS = tubular infoldings of epithelium, replaced every 4-5d by MIGRATING CELLS

THESE OVERLAY

GASTRIC GLANDS: the most abundant glands in stomach. More than one may open into each pit.

**Endocrine cells (apart from G-cells) Secrete → blood / intercell space (not lumen)

PLURIPOTENT STEM CELLS at the bottom of the pit

→ most migrate to lumen to become gastric lining epithelial cells (exfoliation)

→ some go downwards

-MUCOUS NECK differentiate into CHIEF CELLS
- ENDOCRINE
- PARIETAL go both ways

(if necrotic→ shed, apoptosis→ eaten)

OESOPHAGUS

SML INTESTINE

STOMACH

COLON
NO FORMULA WILL EVER APPROACH THE PERFECTION OF MOTHERS' MILK

Hard to copy: Human milk varies with the gestational and chronological age of the baby, the timing of sample collection both during the feed and during the day, with the diet, nutritional status, menstrual cycle and genetic make-up of the mother, and with the content and time elapsed from the mother's last meal.

bioactive components:
- live cells
- immunoglobulins related to infections the mother has been exposed to,
- large variety of oligosaccharides (about130)
- varying growth modulators which include hormones and enzymes (e.g. insulin, growth factors (IGF1), and lipases)
- PLUS long chain polyunsaturated fatty acids and proteins such as human lactoferrin

Compared with other mammalian milks, human milk is
- low in protein
- high in substances necessary for brain maturation, eg.
  - lactose
  - cholesterol
  - long chain polyunsaturated fatty acids
  - readily absorbed iron.

With the exception of infants with congenital lactase deficiency, an extremely rare disorder inherited as an autosomal recessive condition, virtually all infants are capable of digesting lactose in mammalian milks.

In most mammals, lactase activity declines after weaning; in contrast, in man lactase activity persists into adult life in approximately 30% of the world's population.

Human milk contains all the nutrients which are required for a healthy term baby of a well nourished mother for the first four to six months. No supplements are necessary other than vitamin K given at birth to prevent haemorrhagic disease of the newborn, a condition almost exclusively seen in breast-fed infants.

BF reduces the risk or severity of pyloric stenosis, respiratory illness, gastrointestinal tract disease, inflammatory bowel, some childhood cancers, otitis media, urinary tract infections, Sudden Infant Death Syndrome (SIDS) and juvenile onset diabetes mellitus. Breastfeeding helps THE MOTHER: by protecting against premenopausal breast and ovarian cancer and osteoporosis. It also helps the mother to regain her pre-pregnancy body weight and provides a contraceptive effect.

COMPOSITION OF BREASTMILK favours the colonisation of the infant colon with the non-pathogenic lactobacillus bifidus and discourages colonisation with pathogenic bacteria.

The major macronutrients in rank order are
- water
- lactose
- fats
- oligosaccharides.

Fat provides nearly 50% of the energy from milk.

DO NOT feed them whole cows milk!

HAVE TO USE INFANT FORMULA for the 1st year
…until they can drink from a cup
then you can start on full cream cows milk.
SUCKLING:
The baby places the whole areolar in the mouth with the nipple next to the back of the tongue. The baby's tongue presses against the roof of the mouth, and starting at the anterior (gum) margin rolls back against the palate. This action allows the stored milk to be squeezed into the mouth (oropharynx).

Suckling induces release of prolactin that induces milk secretion, and oxytocin causes the let-down response (causing the myoepithelium to contract and eject the milk). The baby swallows about 2 ml at a time.

!! BEWARE !! HIV and Hep C are transmissible through milk!!

LACTATION HAS A CONTRACEPTIVE EFFECT:
The high prolactin levels associated with suckling inhibit ovulation.

NORMAL NUTRITION IN THE FIRST 6 MONTHS:
SO, you’re an infant. Your gut and kidneys are immature. You cannot tolerate solids and high renal solute loads. THESE is the best thing ever.

Breastfeeding practices in Australia
Not every mother chooses to breastfeed. In
From the 1940s to 1960s, breastfeeding prevalence rates fell in many westernised countries as breastfeeding became less popular in line with the drive to modernity.
The 1970s saw a return in popularity of breastfeeding, at first among educated women. Breastfeeding initiation rates rose from then around 70% to now 90%, and rates at one year from less than 10% to now 20%.

The most common reason given for early cessation of breastfeeding is an "inadequate supply of breastmilk", probably reflecting a lack of support in the early stages.

Infant formulas
If breast milk is unavailable, a standard infant formula should be substituted.

Cow’s milk, sweetened condensed milk, or powdered cow’s milk, are inappropriate.

Amount of formula
A normal growth rate is the best sign of adequate nutritional intake.
A formula fed infant generally needs 100-120 kcal/kg/day,
..which is provided by 150-180 ml of formula/kg/day.
As babies grow older they feed less frequently but have larger volumes at each feed.
At the age of 6 months, an infant would usually be having 4-5 feeds per day of 180-240 ml per feed.

Water and other fluids:
If breastfed: NO NEED FOR FLUID SUPPLEMENT
If you water down breast milk, the infant will fail to thrive and go jaundiced

Introduction of solid foods @ 6 months
From 6-7 months through to 11-12 months = progress from sucking to chewing and biting.

MUST TRY EVERYTHING!! Learn different tastes and textures, and the mouth-feel of different foods.
If babies miss out on this experience, they tend to resist eating lumpy food, which may lead to significant feeding difficulties in the second year.

BUT: NOT TOO SALTY OR TOO SWEET! Don’t want your baby habituated to these flavours

Early introduction to solids = world of harm:
- undernutrition (decreased nutrient intake through immature gut)
- increased morbidity (diarrhoea, allergies)
- unnecessary stress on kidneys
- infant becomes used to high levels of sugar and salt

Late introduction to solids = not so good either
- growth-faltering
- difficulties in introducing new foods into the diet.

NORMAL BIRTH WEIGHT: 3.4kg
... should be
!! DOUBLED AT 5 months !!
...
!!! TRIPLED !!!
at 1st birthday
NUTRITION and DEVELOPING COGNITIVE FACULTIES

HUNGRY BABY = STUPID BABY
Preterm infants = most at risk

Mothers who elect to breast feed in developed countries are of a higher social and education status than those who do not.

But.. Differences observed between breast and bottle feeding may be due to the differences in mother-child contact and attachment (bonding) rather than human milk constituents.

BEST RESULTS: good nutrition AND mental stimulus
Although either one of these alone is still pretty good
BREAST MILK CONTAINS NERVE GROWTH FACTOR AND IGF-1, + iron, zinc etc

CONTROVERSIAL QUESTIONS: poly-unsaturated fatty acids. Apparently good for brain, but → conflicting results; SO nobody is allowed to add these PUFAs to their formula.

WORLD SCALE: IRON and IODINE = most scarce micro nutrients. IODINE ESPECIALLY IMPORTANT!
→ 20 million preventable cretins swarm the globe !! all for lack of Iodine
IRON DEFICIENCY = commonest deficiency worldwide, 40-45% of ALL children are anaemic (10% in developed countries! )
= poor attention span, lower intelligence scores and perception.

Poor performance by iron deficient adolescent girls in developed countries is improved with iron supplementation.

…But… Should making a child smarter be promoted as another reason for breast feeding?
ENERGY BALANCE
ENERGY IN = ENERGY OUT.

4 components to total energy expenditure over 24 hours:

<table>
<thead>
<tr>
<th>Energy cost of growth</th>
<th>Basal metabolic rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>... it costs energy to make breast milk, and its very expensive to grow flesh.</td>
<td>= largest single component influenced by:</td>
</tr>
<tr>
<td>Gain of 1 g lean body mass = = 5.4 - 7.1 kJ</td>
<td>body composition (lean tissue has a higher metabolic rate than adipose tissue)</td>
</tr>
<tr>
<td>Gain of 1 g fat mass = = 50 kJ</td>
<td>SEX (largely through differences in body composition, with males generally having more lean tissue for a given weight than females)</td>
</tr>
<tr>
<td>Overall, each gram of weight gain requires ~21 kJ of energy.</td>
<td>AGE (decreases with advancing age eg ~210-220 kJ/m²/h at age 5 years; 180-190 kJ/m²/h at age 15 years; 140-150 kJ/m²/h at age 80 years - note that BMR is here adjusted for surface area which is easier to measure than lean body mass)</td>
</tr>
<tr>
<td>Note: 1 metabolic equivalent (MET) is equivalent to BMR. Thus, an activity that is twice the energy cost of BMR is 2 METs.</td>
<td>PHYSIOLOGICAL STATE eg increased in pregnancy</td>
</tr>
<tr>
<td>The composition of weight gain is not the same at all times during growth. For example, during the first 4 months about 40-45% of weight gain is body fat, whereas by age 2 years it is much lower (less than 10%)</td>
<td>PSYCHOLOGICAL STATE eg increased with acute anxiety</td>
</tr>
<tr>
<td>Therefore, the energy cost of weight gain is much higher in early infancy than by age 2 years. This is a major reason why a decrease in energy intake in very young children can have a more significant impact on</td>
<td></td>
</tr>
</tbody>
</table>

Thermogenesis consists of cold thermogenesis (shivering and non-shivering) and post-prandial thermogenesis. One is to maintain the body temperature at the homeostatic set point; the other is a result of energy being sued to digest and absorb food.

Energy cost of activity
Physical activity accounts for 20-40% of total daily energy expenditure in most individuals. ➔ more for athletes; ➔ less for sloths

The amount of energy expended depends upon the intensity and duration of the different activities carried out in a day.

Physical activity incorporates both sports-like exercise as well as non-exercise activity thermogenesis (NEAT), which includes such things as fidgeting and incidental movement.

In adults, energy requirements can be estimated by
1) first calculating basal metabolic rate using published equations (eg Harris-Benedict equation) 2) and then multiplying by a factor to account for the level of physical activity eg 1.3 for sedentary activity, 1.5 for moderate physical activity and 2 for strenuous activity.

Below:
Estimates of energy costs of selected activities, expressed as a multiple of BMR, or metabolic equivalents [METs]
WHO recommended anthropometric indices:

- **weight-for-height**, Low weight-for-height, also known as wasting or thinness (<~3rd centile of weight for height), indicates in most cases a recent and severe process of weight loss, which may be associated with severe disease or acute starvation.

- **height-for-age**, Low height-for-age, or stunted growth (height <~3rd centile of height for age), reflects a process of failure to reach linear growth potential as a result of suboptimal health and/or nutritional conditions. For children in the age group below 2-3 years, low height-for-age probably reflects a continuing process of "failing to grow" or "stunting"; for older children, it reflects a state of "having failed to grow" or "being stunted".

- **weight-for-age**, Low weight-for-age, or underweight (weight <~3rd centile of weight for age), can reflect both a chronic process (ie long-term poor nutrition) as well as an acute process.

**FAILURE TO THRIVE**

= a description, not a diagnosis.
= describes a toddler or infant whose growth rate is abnormally low
= causes include all kinds of illness, malnutrition and lack of love (?)
= LOOK AT PERCENTILE CHART: but in first few months of life, the average weekly weight gain is better!! Nutritional influence on growth takes place on a background of the baby’s genetic potential for growth.

**SYMPTOMS AND SIGNS:**

- Mild:
  - child is irritable and unhappy,
  - hair is lack lustre, coarse or sparse,
  - the skin is dry.

- Moderate/severe:
  - wasting which is best seen by observing the child from side on,
  - reduction in muscle mass in the buttock
  - loose skin evident on the medial aspects of the thighs and buttock,
  - Generalised muscle wasting can be associated with oedema, reflecting hypoproteinaemia.

**Table 1 shows the minimum average acceptable weight gain (grams/week) between 6 and 12 weeks according to the weight percentile at 6 weeks.**

<table>
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<th>Wt percentile at 6 wks</th>
<th>Boys</th>
<th>Girls</th>
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<tbody>
<tr>
<td>p3</td>
<td>170</td>
<td>145</td>
</tr>
<tr>
<td>p10</td>
<td>160</td>
<td>137</td>
</tr>
<tr>
<td>p75</td>
<td>133</td>
<td>112</td>
</tr>
<tr>
<td>p97</td>
<td>130</td>
<td>103</td>
</tr>
</tbody>
</table>

The lower limits for weight gain for boys and girls together for small babies is about 150 g per week, and for bigger babies 120 g per week. Overall the lower limit for acceptable weight gain is greater for the smaller babies.

**WHY**

- A BABY SHOULD GAIN ABOUT 150g per week

**SYMPTOMS AND SIGNS:**

**Table 2 shows the minimum average acceptable weight gain (grams/week) between 6 and 12 weeks according to the weight percentile at 6 weeks.**

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chronic undernutrition in children may present as stunting only, history, physical examination and longitudinal growth data will show whether the child is thriving or not. COMPARE TO PARENTS: not abnormal for little tiny people to have little tiny children.

Pre-term children will appear short and wasted if plotted according to actual age; **THUS**: plot their graph according to EXPECTED age (i.e when they were due) rather than actual age

Correcting the age to the expected, rather than actual, birthdate should be made for weight until 24 months, and for height until 40 months.

**Table 1** shows the minimum average acceptable weight gain (grams/week) between 6 and 12 weeks according to the weight percentile at 6 weeks.

<table>
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<th>Age (mo)</th>
<th>Weight gain (g/day)</th>
<th>Length gain (mm/day)</th>
</tr>
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<tbody>
<tr>
<td>0-3</td>
<td>25-30</td>
<td>0.9</td>
</tr>
<tr>
<td>3-6</td>
<td>20</td>
<td>0.7</td>
</tr>
<tr>
<td>6-9</td>
<td>15</td>
<td>0.5</td>
</tr>
<tr>
<td>9-12</td>
<td>10</td>
<td>0.4</td>
</tr>
<tr>
<td>12-18</td>
<td>8</td>
<td>0.4</td>
</tr>
<tr>
<td>18-24</td>
<td>6</td>
<td>0.3</td>
</tr>
</tbody>
</table>
**Conceptual Overview of Gastrointestinal Anatomy**

**ARTERIAL SUPPLY**
- **T12**: Coeliac trunk
- **L1**: Sup. mesenteric
- **L3**: Inf. mesenteric

**VENOUS DRAINAGE**
- **FOREGUT** → Splenic vein
- **HINDGUT** → Inf. mesenteric
- **MIDGUT** → Sup. mesenteric

**LYMPHATIC DRAINAGE:**
- FOREGUT → coeliac nodes
- MIDGUT → sup. mesenteric nodes
- HINDGUT → inf. mesenteric nodes

**INNERSATION:**
- FOREGUT + MIDGUT → Vagus (parasympathetic), T5-T12 thoracic splanchnic (sympathetic)
- HINDGUT → S2-S4 pelvic splanchnic (parasympathetic); L1-L2 Lumbar Splanchnic (sympathetic)

**ANKASTOMOSIS between the portal and caval venous systems:**
- Site of rupture!! If portal pressure is too high!
  - Oesophageal wall: bleeding into stomach, haematemesis, black stool
  - Anal canal: hemorrhoids
  - Abdo wall around umbilicus: "Caput Medusae" radiating veins
  - Posterior Abdominal Wall

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**The CAVITIES**
- Epiploic foramen, connects lesser and greater sacs
- Lesser Sack (omental bursa)
- Transverse mesocolon
- Retrovesicular pouch: "behind the bladder"
  - In an upright position, when there is pus in the abdominal cavity, it will collect here and this is very convenient as it is right next to the rectum, thus one can palpate the pouch (pain and irritation are abundant when exudate collects there)

**Structures:**
- Falciform ligament
- Lesser omentum
- Supracolic space
- Greater omentum
- Sigmoid mesocolon
- Infracolic space
WHO OWNS THE BREAST? Breastfeeding CDT

breast feeding = the number one Dietary Guideline for Children in Australia.
= GOLD STANDARD; keep going as long as you can, at least 6 months

According to the World Health Organisation:
- Of 64% of the world's infants, 35% are breast fed for 4 months.
- only 32 per cent of Australian babies were being breastfed exclusively at six months. :( 
- Still not good enough; some mothers only breastfeed while in hospital
- The recommended duration is 6 months \( \Rightarrow \) THEN INTRODUCE SOLIDS, eg. CEREALS
  (By the age of six months, iron and zinc stores are falling in infants who have been exclusively milk-fed (either breast or formula).
- At this time also, the development of feeding behaviour has progressed from sucking to biting
  (and then to chewing by 7 - 9 months).
- Delayed introduction of solids may result in growth-faltering and difficulties in being able to introduce a variety of foods into the diet.

LIST OF FOODS in order of appropriateness:
- **Rice cereal** from 6 months; iron fortified (50 mg per 100g, but not heme form)
- **Mashed banana** from 6 months
- **Minced chicken** from 6-9 months
- **Finely chopped chicken** from 12 months, if already chewing
- **Cows milk** from 12months; approximately 4.0 g fat per 100 g

FACTOIDS:
- The energy needs per kg of body weight are greater in infancy than during adolescence.
- The greatest energy needs (adjusted for body weight) are during the first few months of life

Mature human milk has slightly more fat content than cow's milk: human milk 4.2 g/100g and cow's milk 3.9 g fat/100g.

Breast milk energy content ranges from 271 - 285 kJ per 100g. (same as formula)
ANTERIOR ABDOMINAL WALL

Below: viewed posteriorly (male)
Superficial Anatomy of the Abdomen: Localisation

Diagram showing the localization of various abdominal structures including the liver, stomach, and intestines.