



Diets with modifications

Diets with mineral modification

Diets with vitamin modification

Diets with water modification

low sodium diet

hypertension: >160/95
normotension: <140/90
borderline hypertension: in between the above 2
added-risk hypertension: >130/85

it affects the heart, the brain and the kidneys

90-95% idiopathic. Correlated with sodium

EFSA → limit of 2,4 g of Na⁺/day ≈ 5g of salt

Swap Salt for spices and condiments. Eat K⁺ rich foods

Lower weight, exercise, limit alcohol and lower coffee & tea consumption

Calcium intake modification

most abundant mineral in the human body

renal lithiasis

4% of population. males over 30 y/o

- calcium oxalate 70% + calcium phosphate 6%.
- uric acid 7%.
- struvite 5%.
- cystine 2%.

Crystallisation processes:
1. **solute** saturation of the urine (hypercalciuria)
2. ↓ crystallisation **inhibitors**
3. changes in **pH** (acidity → corrosion of crystals)

↑oxalates ↑proteins ↑vit.C ↑Ca
↓citrates ↓Mg → kidney stones

Solution: ↑water ↓weight
~proteins ↓NaCl ↑citrate ↑Mg
↓stress ↓oxalates <100mg/day.
↓Ca only in **phosphocalcic** alone
↓Purines in **uric acid** lithiasis.
↓P & ↓Mg in **struvite** lithiasis.

osteoporosis

↓mineralisation
↓bone volume

types:
- primary type 1 → low estrogen
- primary type 2 → age
- secondary → disease or drug treatments
- idiopathic → unknown origin

acute pain, hip fractures and bone deformities

body weight and composition, Ca/P ratio 0.5, vitamin D3, vitamin K (mineralisation), high protein diet and Na (Ca lost in urine) and fluoride (fluorohydroxyapatite)

Adequate calcium intake throughout life → trying to compensate in old age is not helpful

Increase Mg intake and do physical exercise (cardio + strength + mobility movements)

rickets and osteomalacia

↓bone mineralisation

rickets in children
osteomalacia in adults

slowed new matrix formation

primary causes: ↓ vitamin D intake or decreased skin synthesis

secondary causes: problems of absorption or metabolism of vitamin D

Iron deficiency anaemia

decrease in haemoglobin quantity

4 successive stages:
1. ↓Fe
2. ↓Fe, ↓ferritin reserves
3. ↓Red blood cell production
4. ↓haemoglobin = ANEMIA

Causes:
- you don't **eat** enough iron (vegetarian, vegan, no red meat).
- you **need more** (at certain stages of life)
- you **lose** too much **blood** (menstruation, haemorrhage, infections)

from the 12mg ingested, only 1mg is actually absorbed

change the **diet:** liver, legumes + vitamin C, avoid anti-nutrients that reduce absorption and be careful with antacids and antibiotics. Iron **supplements** and **fortification** of foods with iron (flours, salts, cereals).

copper deficiency

enzyme intermediate

problems: deficit or excess (Wilson)

fructose and Zn lower Cu absorption → real problem: low intakes

NO copper → no Fe transitions (no hemoglobin, no ferritin, no erythrocytes)
Copper is the guy that changes the electrons when needed

EFSA → 1.3mg/day♀ and 1.6mg/day♂. Oysters and molluscs, nuts, cereals...

Megaloblastic anaemia

decrease in DNA synthesis

Cobalamin deficiency

blood cell formation and folate metabolism

Causes:
- insufficient intake (vegans)
- gastric disturbances (non IF)
- intestinal disturbances (non-separation of proteins)
- interactions with drugs and alcohol

stage 1: reserves exist, serum depletion
stage 2: tissue depletion, metabolic pathways not altered
stage 3: insufficient tissue deposition → reduced DNA synthesis and erythrocyte ↓folate
stage 4: metabolic disturbances → clinical damage. corrected with 5µg

bacterial synthesis, only in animal sources: viscera, egg yolk, salmon, meat, dairy products...

Folate deficiency

structure of pteroylglutamic acid (folic acid)

Causes:
- insufficient intake: age
- high requirements: pregnancy
- decreased intestinal absorption
- metabolic disturbances: alcohol and vitamin B12 deficiency

- stage 1: no cellular depletion but serum depletion, no metabolic alterations
- stage 2: erythrocyte folate decrease
- stage 3: impaired erythropoiesis, not enough DNA
- stage 4: megaloblasts macrovalocytes and reduced haemoglobin

monoglutamates 90% → direct absorption
polyglutamates 50% → to be hydrolysed

almost everywhere, mainly in veggies and legumes

Vitamin B6 deficiency

Vitamin B6 (pyridoxal phosphate) → amino acid metabolism

Rare in modern countries, Causes:
cereal-based food → glucoside (non-absorbable)
chronic **alcoholism** → ↓phosphorylation
↑dephosphorylation
treatment with various **drugs**
chronic **haemodialysis**

1.3-1.7mg/day♂ 1.3-1.5mg/day♀
→ liver, legumes, nuts and banana

thermal process losses up to 50%.

without water there is no life, it is involved in:

- digestion, absorption and excretion
- transport and elimination
- body temperature regulation
- osmotic balance and pH
- cell shape and lubricating fluids.

Water balance

total water intake → from food and beverages
available water = intake + macronutrient oxidation

drinking more than the maximum renal excretion (0.7-1.1 L/hour)

causes hyponatremia and possible cerebral edema

Water intoxication

adequate water intake: ~1ml/kcal/day

adjust more water in children, athletes (1L per lost kg), pregnant and breastfeeding women and elderly

Water losses

renal losses: ↑protein and mineral intake + diabetes / diuretics → more urine

cutaneous and pulmonary losses: body temperature and environment

gastrointestinal losses: diarrhoea and vomiting → altered thirst sensation (thirst = small dehydration)

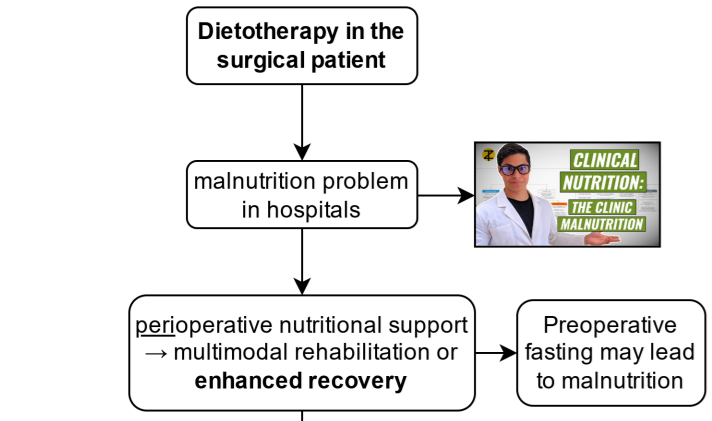
Dehydration

Types

by amount: mild 1-2%bw, moderate 3-5%, high 6-8%, severe 9-11%. over 11% → incompatible with life

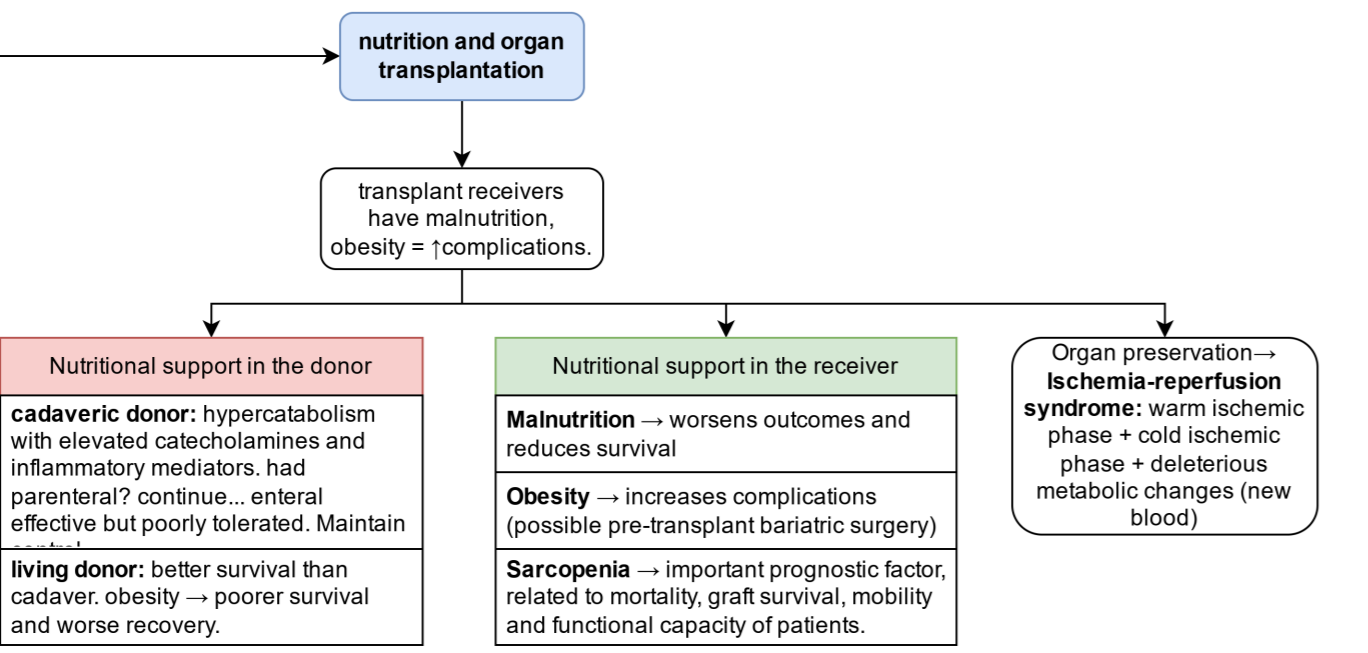
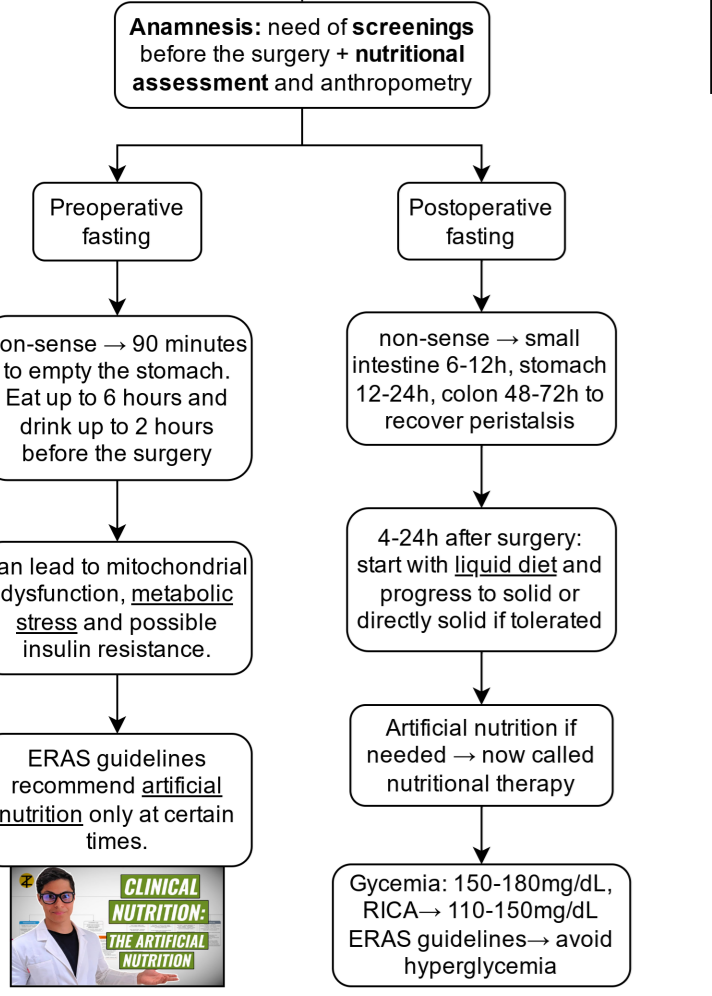
by speed of loss: if it is sudden it has more severe symptoms

by lost solute/water ratio: isotonic, hypertonic (↑water lost) or hypotonic (↑minerals lost)



Changes during surgery (aggression)

- Increased **energy** expenditure
- Protein** hypercatabolism with increased urinary excretion of nitrogen.
- Hyperglycaemia** with hyperinsulinaemia
- Mobilisation of **fatty acids** and triacylglycerols
- Water** and **sodium** retention
- Mineral** urinary losses of zinc, magnesium, phosphorus, and potassium



Transplant type	Liver	Kidney	Pancreas	Lung	Cardiac	Intestinal	haematopoietic precursor cells
pre-transplant	protein 1.2-1.5 g/kg/day. Carbs 50-60% and fats 30-35%. avoid prolonged fasting periods.	Obesity is not an absolute contraindication (increases complications), possible bariatric surgery prior to transplantation.	previous kidney transplantation, ↑protein-calorie intake and ↓weight. obesity worsens graft and individual survival.	Emphysema → malnutrition. Cystic fibrosis ↑kcal burned → ↑respiration. 30-50% malnutrition → oral or enteral nutrition to ↑muscle mass.	90-110% of ideal weight, ↑Na ⁺ to 2g, ↑protein (1.5-2g/kg/day). possible oral nutritional supplements	Short bowel syndrome due to mesenteric vasculopathies, trauma and Crohn's disease. infectious complications are the main cause of morbidity and mortality.	chemotherapy → individual assessment → screening → dietary advice → dietary advice or oral nutritional supplements
transplant	early enteral nutrition although complementary parenteral nutrition may be necessary.	do not require specific nutritional support.	immediate functioning. no insulin treatment required. restart oral intake in 2-3 days.	oral feeding usually begins after 1-2 days. Pancreatic enzyme administration (cystic fibrosis)	basal energy expenditure x 1.3-1.5 and protein 1.2-2 kg/day. oral nutritional supplements may be necessary.	parenteral or enteral tube feeding.	no hard, acidic or extreme temperature foods. hygiene and "very clean food". Enteral > parenteral nutrition (risk of infections).
post-transplant	↑Kcal & ↑Protein, moderate in fat, 1,500 mg of calcium and multivitamins until nutritional recovery.	frequent weight gain (possible diabetes) ↑ fat ↓ muscle ↓ bone. Undesirable gain not in everyone (avoid malnutrition).	recovers the functionality of both organs, general population recommendations. obesity leads to diabetes recurrence	Severe cases: enteral nutrition via gastrostomy or gastro-jejunostomy. In cystic fibrosis use of oral nutritional supplements.	calorie adjustment of diet and gradual exercise, keeping weight within recommended limits	progressive bowel rehabilitation. oligomeric or polymeric enteral diet, lipids are introduced from the fourth week onwards.	Acute → diet to control symptoms-chronic → treat diarrhoea, ↑fat ↓residues, oral supplements. Severe? artificial nutrition

