USMLE Nutrition Notes

Integrated review for Step 1, Step 2 CK, and Step 3

Official update: starting in June 2026, USMLE is enhancing nutrition content across all three Step exams and adding nutrition performance feedback to score reports. Nutrition will still appear inside integrated system-based questions rather than as a separate scored block.

Best way to use this note

- Start with the diagram pages. They give you the framework.
- Then go through the comparison tables and deficiency patterns.
- Finish with the memory cases, vignettes, and rapid review pages.

What is covered

- Macronutrients, protein-energy malnutrition, and severe acute malnutrition
- Fat-soluble, water-soluble vitamins, and trace mineral patterns
- Iron, calcium, vitamin K, anemia, and copper/zinc comparison patterns
- Pregnancy, postmenopausal bone health, geriatrics, and metabolic syndrome counseling
- Malabsorption, bariatric surgery, CKD, enteral vs parenteral nutrition, TPN, and refeeding syndrome

1. What the exam is really testing

Nutrition on the USMLE is usually not a standalone fact-recall block. It is commonly tested as physiology plus a clinical setting.

- Deficiency patterns in a patient stem: night blindness, macrocytosis, bleeding, neuropathy, bone pain, or edema.
- Malabsorption after pancreatic disease, cholestasis, gastrectomy, bariatric surgery, or terminal ileal disease.
- Hospital nutrition problems: refeeding syndrome, TPN complications, and when to prefer enteral feeding over parenteral feeding.
- Disease-state physiology: CKD and vitamin D activation, pregnancy and folate, vegan diet and B12, alcohol use disorder and thiamine.
- Treatment-order questions: what to give first, what to measure, and what complication to prevent.

Stem workflow

1. Identify the setting: newborn, pregnant patient, alcoholic, vegan, malnourished patient, post-bariatric patient, CKD, cholestasis, cystic fibrosis, or refugee camp.
2. Link the setting to the nutrient or physiology being stressed.
3. Predict the lab pattern before reading the answer choices.
4. Pick the next step that fixes the dangerous problem first.

2. Macronutrients and protein-energy malnutrition

Item	Key fact	Board clue
Carbohydrate	4 kcal/g. Main quick fuel. Adult AMDR 45%-65% of calories.	Carbohydrate reintroduction drives the insulin surge in refeeding syndrome.
Protein	4 kcal/g. Needed for enzymes, transport proteins, muscle, and oncotic pressure. Adult AMDR 10%-35% of calories.	Severe deficiency can cause wasting; edema suggests advanced protein-energy malnutrition.
Fat	9 kcal/g. Dense energy source. Needed for cell membranes, eicosanoid precursors, and absorption of A, D, E, and K. Adult AMDR 20%-35% of calories.	If bile or pancreatic enzymes are missing, think fat malabsorption and ADEK deficiency.
Alcohol	7 kcal/g but not an essential nutrient.	Alcohol use disorder is a strong clue for thiamine deficiency and poor overall nutritional status.

Fed vs fasting: the part that keeps showing up in stems

- Fed state -> insulin is high -> glycogen synthesis, lipogenesis, and protein synthesis.
- Early fasting -> glucagon is high -> hepatic glycogenolysis becomes important.
- Prolonged fasting -> gluconeogenesis and lipolysis increase; ketone production rises.
- Refeeding -> carbohydrate returns -> insulin rises -> phosphate, potassium, and magnesium shift into cells.

Feature	Marasmus	Kwashiorkor
Main deficit	Total calorie deficiency	Severe protein deficiency with relative energy preservation
Appearance	Marked loss of muscle and fat; "skin and bones"	Edema can hide weight loss

Edema	Absent	Present
Liver	No classic fatty liver pattern	Fatty liver is common
Skin / hair	Dry, thin tissue from wasting	Dermatosis and hair changes are classic clues
Exam tone	Severe wasting	Edematous malnutrition

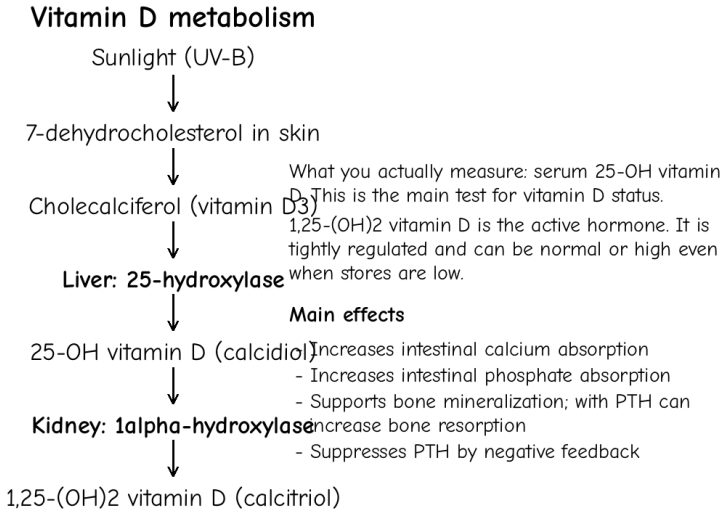
Extra clue

- Essential fatty acid deficiency is most likely in prolonged fat-free TPN or severe fat malabsorption.
- Look for scaly dermatitis, alopecia, poor wound healing, and growth failure in the right clinical setting.

3. Visual framework

These are the pages to memorize first. They are meant to be redrawn from memory.

3.1 Vitamin D metabolism



Exam clues

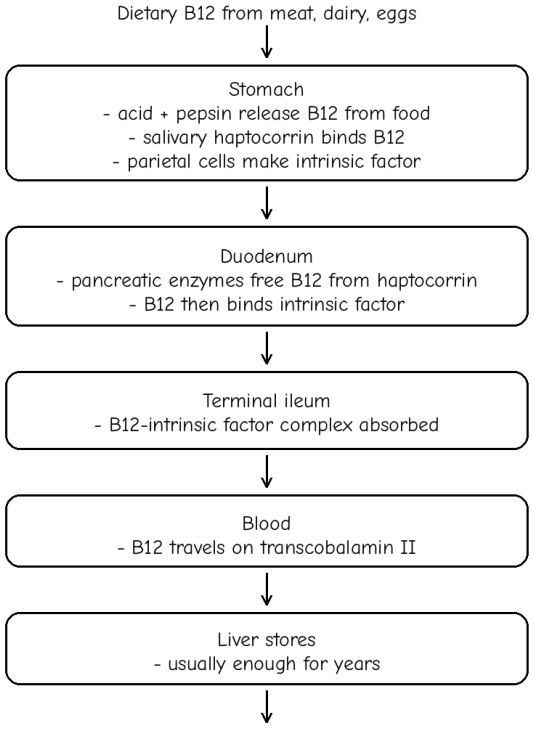
- CKD -> reduced renal activation -> hypocalcemia + secondary hyperparathyroidism + renal bone disease.
- Granulomatous disease (for example sarcoidosis or TB) -> ectopic 1alpha-hydroxylase -> increased calcitriol -> hypercalcemia.
- If the question asks which vitamin D test to order, choose 25-OH vitamin D.

Exam notes

- Order 25-OH vitamin D when the question asks about vitamin D status.
- CKD impairs activation in the kidney. Granulomatous disease can increase ectopic activation.

3.2 Vitamin B12 absorption and deficiency

Vitamin B12 absorption and where it fails



Failure points

Low stomach acid / no intrinsic factor

- Pernicious anemia
- Gastrectomy or gastric bypass
- Long-term acid suppression can reduce release from food

Pancreatic problem

- Pancreatic insufficiency: B12 may stay bound to haptocorrin

Terminal ileum problem

- Crohn disease
- Ileal resection
- Bacterial overgrowth
- Diphyllobothrium latum

Diet / drugs

- Strict vegan diet
- Metformin can reduce absorption
- Nitrous oxide functionally inactivates B12

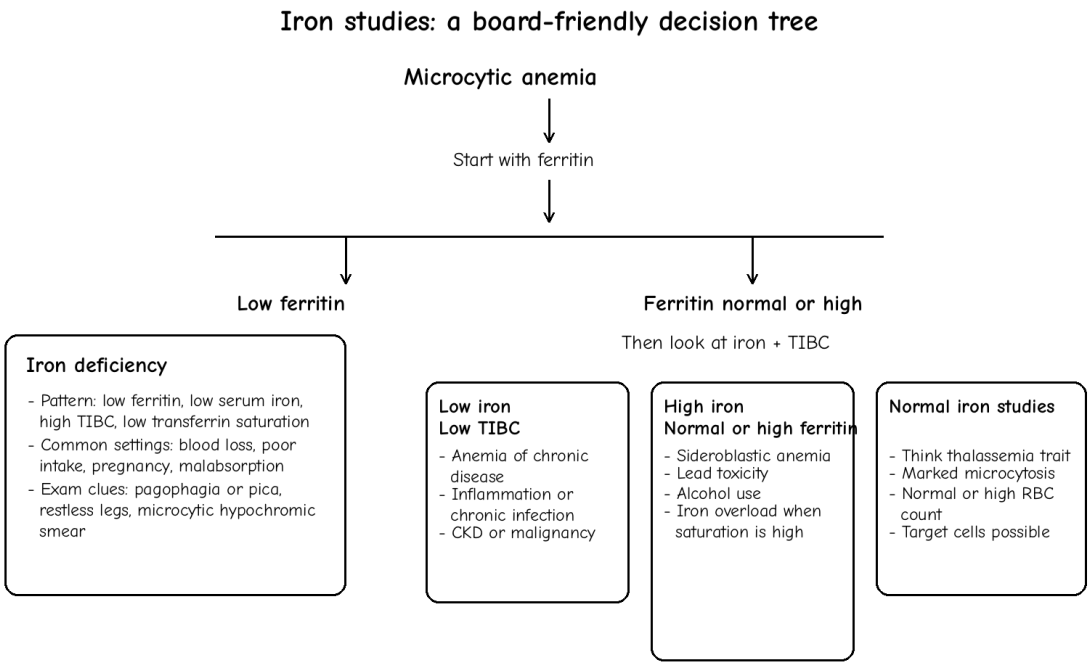
Lab pattern

- Low serum B12 -> elevated methylmalonic acid and elevated homocysteine.
- Macro-ovalocytes + hypersegmented neutrophils = megaloblastic anemia.
- Neurologic findings can include loss of vibration or proprioception, sensory ataxia, and corticospinal tract signs.

Exam notes

- Remember the duodenum step: pancreatic enzymes free B12 from haptocorrin before B12 binds intrinsic factor.
- Terminal ileum disease or resection is one of the classic board clues.

3.3 Iron studies decision tree



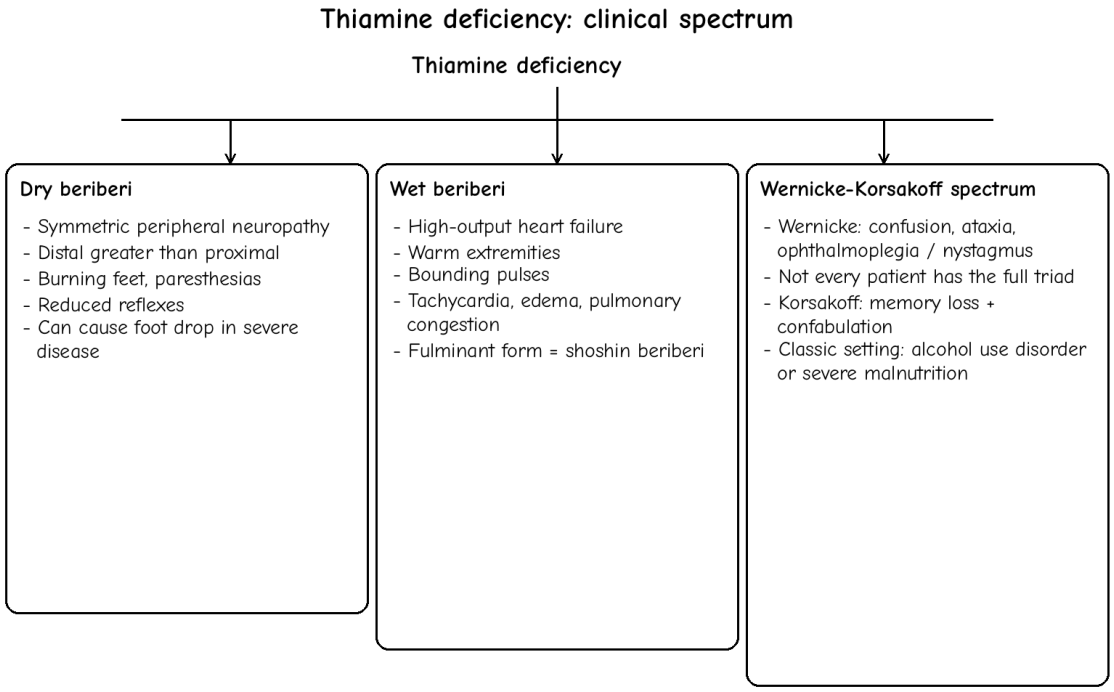
Remember this

- Low ferritin strongly supports iron deficiency. Ferritin can look falsely normal or high during inflammation, so read it in context.
- If transferrin saturation is clearly high, think iron overload or sideroblastic processes rather than simple iron deficiency.

Exam notes

- Ferritin low = iron deficiency until proven otherwise.
- Normal or high ferritin does not fully exclude iron deficiency if inflammation is present.

3.4 Thiamine deficiency spectrum



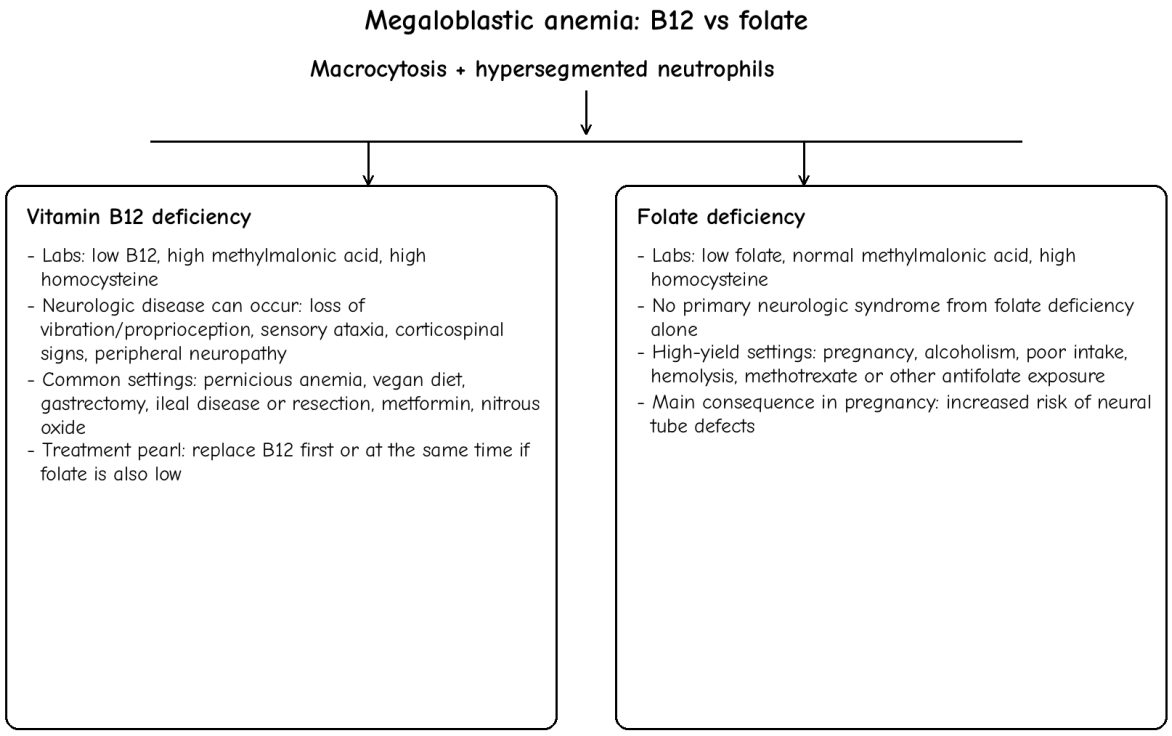
Biochemistry that actually matters

- TPP is a cofactor for pyruvate dehydrogenase, alpha-ketoglutarate dehydrogenase, branched-chain alpha-ketoacid dehydrogenase, and transketolase.
- Low thiamine -> impaired aerobic glucose metabolism -> lactate can rise.
- Take-home: give thiamine before carbohydrate or dextrose in a malnourished or alcohol-dependent patient.

Exam notes

- Wet beriberi = high-output heart failure. Dry beriberi = peripheral neuropathy.
- Wernicke should be treated immediately; do not wait for the full triad.

3.5 B12 vs folate



Practical rule

- Homocysteine rises in both. Methylmalonic acid points toward B12, but remember that renal failure can also raise MMA.
- Folate can improve the anemia while neurologic injury from unrecognized B12 deficiency continues.

Exam notes

- Both raise homocysteine. MMA is the cleaner differentiator.
- Macrocytosis plus neurologic findings = B12 until proven otherwise.

3.6 Refeeding syndrome

Refeeding syndrome: what happens and how to prevent it

Before feeding

- Insulin is low
- Catabolism: fat and protein are being used for fuel
- Total body phosphate, potassium, magnesium, and thiamine become depleted
- Serum levels may still look near-normal at baseline

After calories are restarted

- Carbohydrate intake increases insulin release
- Glucose, phosphate, potassium, and magnesium shift into cells
- Thiamine demand rises as carbohydrate metabolism restarts
- Serum electrolytes can fall quickly over the first few days

What you see clinically

- Hypophosphatemia = hallmark finding
- Weakness, rhabdomyolysis, hemolysis, respiratory failure
- Hypokalemia / hypomagnesemia -> arrhythmias, prolonged QT, ileus
- Fluid overload and heart failure can occur
- Wernicke encephalopathy can appear if thiamine is not replaced

Prevention

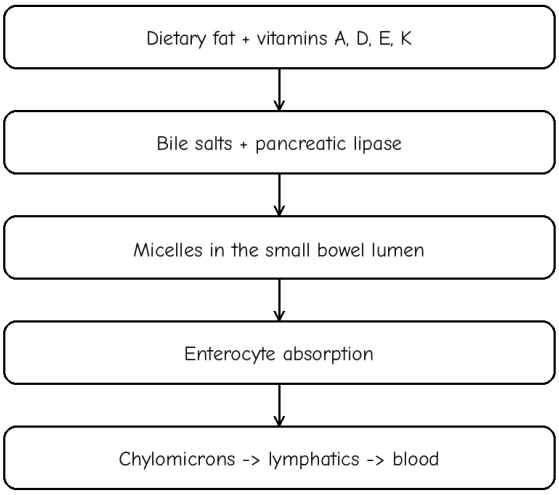
- Identify risk first: BMI less than 16, major weight loss, little or no intake for more than 10 days, or low phosphate/potassium/magnesium. Multiple moderate-risk features also count.
- Give thiamine before and during early feeding.
- In high-risk adults, start at no more than 10 kcal/kg/day and advance slowly; use 5 kcal/kg/day in extreme risk.
- Monitor phosphate, potassium, magnesium, glucose, and fluid balance closely during the first week.

Exam notes

- Hypophosphatemia is the hallmark.
- Start low, go slow, and replete electrolytes while monitoring closely.

3.7 Fat-soluble vitamin absorption

Fat-soluble vitamins: absorption and malabsorption



When absorption breaks

Bile problem
- Cholestasis
- Biliary obstruction
- Severe cholestatic liver disease

Pancreatic problem
- Cystic fibrosis
- Chronic pancreatitis
- Pancreatic insufficiency

Intestinal problem
- Celiac disease
- Crohn disease
- Short bowel syndrome

Drugs
- Orlistat
- Cholestyramine
- Mineral oil

Classic deficiency clusters
- A -> night blindness, xerosis, Bitot spots, keratomalacia if severe
- D -> osteomalacia / rickets, hypocalcemia, elevated PTH
- E -> hemolysis and neurologic dysfunction in severe deficiency
- K -> bleeding and prolonged PT first
- If the stem says cystic fibrosis or cholestatic disease with multiple deficiencies, think A, D, E, and K together.

Exam notes

- A, D, E, and K travel with fat, so bile and pancreatic problems can affect all four together.
- Cystic fibrosis and cholestatic disease are classic cluster stems.

4. Deficiency patterns you should know cold

Vitamin	Main role	Classic deficiency pattern	High-yield note
A	Vision and epithelial differentiation	Night blindness -> conjunctival xerosis -> Bitot spots -> keratomalacia if severe	Preformed vitamin A excess is teratogenic.
D	Calcium and phosphate homeostasis	Rickets in children; osteomalacia in adults; hypocalcemia can prolong QT	Order 25-OH vitamin D, not 1,25-(OH)2 vitamin D, for status.
E	Antioxidant in cell membranes	Hemolysis; severe deficiency can cause neurologic dysfunction with loss of vibration / ataxia	Think abetalipoproteinemia or severe fat malabsorption.
K	Gamma-carboxylation of factors II, VII, IX, X and proteins C/S	Bleeding; PT rises first	Newborns need prophylaxis at birth.

Nutrient	Classic deficiency clue	Useful association
B1 (thiamine)	Dry/wet beriberi; Wernicke-Korsakoff	Alcohol use disorder, hyperemesis, malnutrition, refeeding
B3 (niacin)	Pellagra: dermatitis, diarrhea, neurocognitive change	Think alcoholism, Hartnup disease, or carcinoid syndrome
B6 (pyridoxine)	Sideroblastic anemia, neuropathy, seizures	Isoniazid is the classic trigger
B9 (folate)	Megaloblastic anemia without the B12 neurologic pattern	Pregnancy raises importance because of neural tube defects
B12 (cobalamin)	Macrocytosis plus neurologic disease	Vegan diet, pernicious anemia, gastrectomy, ileal disease, metformin
C (ascorbic acid)	Scurvy: gum bleeding, perifollicular hemorrhages, corkscrew hairs, poor wound healing	Also improves nonheme iron absorption
Iron	Fatigue, microcytosis, low ferritin, pagophagia	Blood loss is the first thing to exclude
Calcium	Low calcium -> tetany, long QT; high calcium -> stones, constipation, short QT	Vitamin D and PTH questions often sit behind calcium stems

Micro-points that save questions

- B12 deficiency raises methylmalonic acid and homocysteine; folate deficiency raises homocysteine but not MMA.
- MMA can also rise in renal insufficiency, so interpret it in context.
- Vitamin K deficiency classically prolongs PT first because factor VII has the shortest half-life among the vitamin K-dependent clotting factors.
- Breast milk is low in vitamin K; newborns also start with low stores and limited placental transfer.

5. Comparison tables

Feature	Iron deficiency	Megaloblastic anemia
MCV	Low	High
Smear	Microcytic, hypochromic cells	Macro-ovalocytes + hypersegmented neutrophils
Best lab clue	Low ferritin	Check B12, folate, and MMA
Reticulocytes	Usually low unless acute blood loss is ongoing	Low because erythropoiesis is ineffective
Classic stem	Menstruation, GI blood loss, pagophagia	Vegan diet, pregnancy, alcohol use, gastrectomy, ileal disease
Complication to remember	Restless legs or pica	Neurologic injury if the cause is B12

Feature	B12 deficiency	Folate deficiency
MMA	High	Normal
Homocysteine	High	High
Neurologic findings	Yes	No primary B12-type neurologic syndrome
Classic patient	Vegan, pernicious anemia, gastrectomy, ileal disease	Pregnancy, poor intake, alcoholism, antifolate drug
Treatment pitfall	Do not miss it before giving folate	Treat, but make sure B12 deficiency is not being masked

Finding	Hypocalcemia	Hypercalcemia
Neuromuscular tone	Tetany, hyperexcitability	Weakness, reduced excitability
Reflexes	Hyperreflexia	Hyporeflexia
Exam signs	Chvostek and Trousseau positive	These signs are not expected
CNS	Paresthesias, seizures	Confusion, lethargy
ECG	Prolonged QT	Shortened QT
Common clue	Vitamin D deficiency	Hyperparathyroidism or malignancy

System	Complication	Mechanism / clue	How to prevent or think about it
Infectious	Catheter bloodstream infection	Central line colonization	Sterile technique and line care matter
Metabolic	Hyperglycemia	High dextrose load / insulin resistance	Monitor glucose and adjust insulin if needed
Metabolic	Hypoglycemia after abrupt stop	Exogenous glucose delivery stopped suddenly	Do not stop TPN abruptly; taper when appropriate
Metabolic	Refeeding syndrome	Insulin drives phosphate / K / Mg into cells	Identify risk before feeding and advance slowly
Metabolic	Essential fatty acid deficiency	Lipid-free or severely restricted fat delivery	Include lipid when clinically appropriate
Hepatobiliary	Steatosis, cholestasis,	Too much carbohydrate + no	If the gut works, use enteral

	gallstones	enteral stimulation	feeding whenever possible
Bone	Metabolic bone disease	Long-term PN	Use the gut when possible and monitor over time
Vascular	Thrombosis	Central venous catheter complication	Watch the line

Short rule to memorize: if the gut works, feed the gut.

6. Mnemonics and pattern hooks

Hook	Meaning
A D E K	The fat-soluble vitamins; think bile, pancreas, cystic fibrosis, cholestasis, and steatorrhea.
C A O	Confusion, ataxia, ophthalmoplegia: the classic Wernicke triad, even though many patients do not have all 3 at once.
4 Ds	Dermatitis, diarrhea, dementia or delirium, and death for pellagra.
MMA makes me think B12	Methylmalonic acid points toward B12 deficiency rather than folate deficiency.
TIBC = empty tables	In iron deficiency the body makes more binding capacity because there is not enough iron to occupy it.
Start low, go slow	The prevention line for refeeding syndrome.

Patterns worth memorizing

- Pagophagia plus microcytosis points hard toward iron deficiency.
- Macrocytosis plus neurologic findings points hard toward B12 deficiency.
- Night blindness plus conjunctival xerosis or Bitot spots points to vitamin A deficiency.
- Newborn bleeding in the first week points to vitamin K deficiency bleeding if prophylaxis was missed.
- Day 2 to 5 of refeeding plus weakness, confusion, or arrhythmia should make you check phosphate first.

7. Memory cases

Case 1. Ice craving and microcytosis

A patient says she chews ice constantly. CBC shows microcytic anemia and ferritin is low.

This is a classic pagophagia stem, and it should immediately push iron deficiency to the top of the list.

Take-home

- Pagophagia is one of the most useful symptom clues in iron deficiency stems.
- Low ferritin plus high TIBC fits iron deficiency unless another process is clearly confusing the picture.

Exam clue: Ice craving + microcytosis = iron deficiency until proven otherwise.

Case 2. Macrocytosis plus difficulty walking

A young strict vegan develops numb feet, poor balance in the dark, and macrocytic anemia.

The combination of posterior column symptoms and macrocytosis is the key pattern.

Take-home

- Loss of vibration and proprioception points to posterior column disease.
- Add hyperreflexia or Babinski and you are seeing subacute combined degeneration from B12 deficiency.
- B12 stores last years, so symptoms can take time to appear.

Exam clue: Macrocytic anemia + neurologic findings is B12 until proven otherwise.

Case 3. Alcohol use disorder and hypoglycemia

A malnourished patient with alcohol use disorder is hypoglycemic and needs dextrose.

The dangerous mistake is giving carbohydrate first and forgetting thiamine.

Take-home

- Low thiamine impairs carbohydrate metabolism and can precipitate Wernicke encephalopathy when glucose is given first.
- In a time-pressured question, the main answer is simple: thiamine before glucose.

Exam clue: Do not wait for the full Wernicke triad.

Case 4. Bleeding newborn on day 5

A newborn who did not receive prophylactic vitamin K returns with bleeding in the first week of life.

This is the classic board setup for vitamin K deficiency bleeding.

Take-home

- Newborns begin with low stores, limited placental transfer, low vitamin K content in breast milk, and minimal early bacterial production.
- Vitamin K deficiency raises PT first because factor VII falls fastest.

Exam clue: The board point is prevention: intramuscular vitamin K at birth.

Case 5. Day 3 of refeeding

A severely malnourished patient starts calories and then becomes weak, confused, or arrhythmic on day 2 or 3.

The lab you should want immediately is phosphate, followed by potassium and magnesium.

Take-home

- Hypophosphatemia is the hallmark lab abnormality.
- The right prevention move is to identify risk, give thiamine, replete electrolytes, and start calories slowly.

Exam clue: Start low. Go slow.

8. USMLE-style vignettes

Vignette 1. What has to come before dextrose?

Question: A patient with alcohol use disorder is found with glucose 40 mg/dL. Which intervention should come before IV dextrose?

Best answer: Thiamine

Why

- Chronic alcohol use and malnutrition deplete thiamine stores.
- Giving carbohydrate first can precipitate or worsen Wernicke encephalopathy.
- The exam point is treatment order, not a long differential.

Watch for

- Do not overthink this one. It is one of the highest-yield nutrition questions.

Vignette 2. Which lab best separates B12 from folate deficiency?

Question: A patient has macrocytosis and hypersegmented neutrophils. Which lab most cleanly separates vitamin B12 deficiency from folate deficiency?

Best answer: Methylmalonic acid

Why

- Both deficiencies raise homocysteine.
- Only B12 deficiency classically raises methylmalonic acid.
- MMA is not perfect in renal failure, but it is still the classic differentiator in board questions.

Vignette 3. Day 3 weakness during nutritional rehabilitation

Question: A severely malnourished patient develops weakness and confusion on hospital day 3 after calories are restarted. Which lab abnormality is the hallmark?

Best answer: Hypophosphatemia

Why

- Refeeding increases insulin, which pushes phosphate into cells.
- Low phosphate undermines ATP-dependent tissues and explains weakness, rhabdomyolysis, respiratory failure, and arrhythmias.
- Potassium and magnesium also matter, but phosphate is the key hallmark.

Vignette 4. Macrocytosis plus posterior column signs

Question: A patient has macrocytic anemia, loss of vibration sense, sensory ataxia, and mixed upper-motor-neuron plus peripheral-nerve findings. Which deficiency fits best?

Best answer: Vitamin B12

Why

- Posterior column plus corticospinal tract disease is the localization of subacute combined degeneration.
- Folate deficiency can mimic the anemia but not the classic cord findings.
- The next thought after choosing B12 is usually to check MMA and begin replacement.

Vignette 5. Night blindness, xerosis, and Bitot spots

Question: A child with night blindness, conjunctival dryness, and Bitot spots needs treatment for xerophthalmia. What is the key immediate therapy?

Best answer: High-dose vitamin A using the WHO age-based treatment schedule

Why

- Night blindness is an early ocular clue of vitamin A deficiency.
- Bitot spots are a classic exam finding in xerophthalmia.
- For children older than 12 months, the WHO treatment schedule uses 200,000 IU on day 1, day 2, and again at least 2 weeks later.

9. Common pitfalls

Pitfall 1. Seeing macrocytosis and stopping at "B12 or folate"

Common wrong move: Ignoring the peripheral smear and forgetting the non-megaloblastic causes.

What to do instead

- Hypersegmented neutrophils support megaloblastic anemia.
- If the smear does not fit, widen the differential: alcohol, liver disease, hypothyroidism, reticulocytosis, medications, or myelodysplasia.

Pitfall 2. Giving folate before you think about B12

Common wrong move: Treating the anemia first and missing the neurologic disease.

What to do instead

- Folate can improve the CBC while neurologic injury from B12 deficiency keeps progressing.
- If B12 is low or uncertain, replace B12 first or at the same time.

Pitfall 3. Ordering the wrong vitamin D test

Common wrong move: Checking 1,25-(OH)2 vitamin D when you really want vitamin D stores.

What to do instead

- For vitamin D status, order serum 25-OH vitamin D.
- Reserve 1,25-(OH)2 vitamin D for specific physiologic questions, such as altered activation or ectopic production.

Pitfall 4. Missing refeeding syndrome because the first labs looked okay

Common wrong move: Trusting a baseline normal phosphate, potassium, or magnesium in a severely malnourished patient.

What to do instead

- The body can be profoundly depleted even when the first serum values look close to normal.
- Risk assessment comes before feeding. Monitor daily once calories restart.

Pitfall 5. Thinking vitamin K reverses warfarin immediately

Common wrong move: Assuming vitamin K alone fixes urgent bleeding in minutes.

What to do instead

- Vitamin K restores synthesis of clotting factors, so it is not the fastest rescue maneuver.
- If bleeding is major or surgery is urgent, think immediate factor replacement such as PCC; plasma is an alternative when PCC is not available.
- Board version: vitamin K gives durable correction, but you may need clotting factors now.

10. Rapid review

Ten things you should not miss

1. Thiamine before glucose in the malnourished or alcohol-dependent patient.
2. 25-OH vitamin D is the main vitamin D status test.
3. Methylmalonic acid points toward B12, not folate.
4. Macrocytosis plus neurologic disease = B12 until proven otherwise.
5. Hypophosphatemia is the hallmark of refeeding syndrome.
6. All newborns should receive intramuscular vitamin K prophylaxis at birth.
7. Fat malabsorption puts A, D, E, and K at risk together.
8. Night blindness and Bitot spots point to vitamin A deficiency.
9. Low ferritin strongly supports iron deficiency.
10. If the gut works, use enteral feeding rather than parenteral feeding whenever possible.

Recognition patterns

- Ice craving -> iron deficiency
- Corkscrew hairs and gum bleeding -> vitamin C deficiency
- Macrocytosis + hypersegmented neutrophils -> megaloblastic anemia
- Macrocytosis + difficulty walking -> B12 deficiency
- Photosensitive dermatitis + diarrhea + neurocognitive change -> niacin deficiency
- Alcohol use disorder + confusion / ataxia / eye findings -> thiamine deficiency
- Newborn bleeding in the first week -> vitamin K deficiency bleeding
- Day 2 to 5 of refeeding + weakness or arrhythmia -> phosphate first
- Cystic fibrosis + easy bruising + bone pain + night blindness -> think A, D, E, K together
- CKD + hypocalcemia + bone disease -> impaired vitamin D activation

Condition	Best quick clue	One-liner
Iron deficiency	Low ferritin, high TIBC	Microcytosis, blood loss, pagophagia
B12 deficiency	High MMA	Macrocytosis plus neurologic findings
Folate deficiency	Normal MMA	Macrocytosis without the classic B12 neurologic pattern

Vitamin K deficiency	PT rises first	Bleeding, newborn or cholestatic clue
Vitamin D deficiency	Low 25-OH vitamin D	Rickets / osteomalacia, hypocalcemia
Refeeding syndrome	Low phosphate after calories restart	Start low, go slow

11. Exam approach

Four ways nutrition questions usually become easier

1. Use the epidemiology clue: newborn, pregnant patient, vegan patient, alcoholic patient, post-bariatric patient, cystic fibrosis, CKD, cholestasis, refugee camp, or anorexia nervosa.

2. Use the timeline clue: first week of life, day 3 of refeeding, months of macrocytic symptoms, years of B12 depletion, or slow vitamin A deficiency progression.

3. Use the cluster clue: multiple deficiencies in the same stem usually point to a shared absorption problem.

4. Use the treatment-order clue: sometimes the entire question is asking what comes first, not what the final diagnosis is.

High-yield treatment anchors

Scenario	Treatment anchor
Suspected Wernicke or at-risk hypoglycemia	Give thiamine before dextrose; suspected Wernicke requires high-dose IV thiamine by local protocol.
Xerophthalmia in child older than 12 months	Vitamin A 200,000 IU orally on day 1, day 2, and again at least 2 weeks later.
Newborn prophylaxis	Intramuscular vitamin K at birth; CDC cites AAP guidance to give it within 6 hours.
High-risk refeeding	Thiamine plus slow calorie advancement and close electrolyte monitoring.
Pernicious anemia or severe B12 malabsorption	Parenteral B12 replacement is the board-level move.
Iron deficiency	Each 325 mg ferrous sulfate tablet provides about 65 mg elemental iron; exact schedules vary clinically.

Short study plan

- If you have 4 weeks: week 1 diagrams + ADEK/B vitamins, week 2 anemias and calcium, week 3 refeeding/TPN/macronutrients, week 4 cases and vignettes.
- If you have 1 week: diagrams first, then comparison tables, then rapid review and vignettes.
- If you have 1 day: diagrams in the morning, anemia / vitamin K / calcium tables in the afternoon, rapid review at night.

12. Trace minerals and add-on deficiency details

This section folds in the trace-mineral and classic explanation details that are commonly buried inside long question-bank explanations.

Mineral	Common risk setting	Deficiency pattern	High-yield clue
Chromium	Long-term parenteral nutrition without adequate trace element replacement	Impaired glucose tolerance or harder-to-control glucose levels	Rare but board-recognizable
Copper	Bariatric / gastric surgery, celiac disease, inflammatory bowel disease, excess zinc	Anemia, leukopenia, sensory ataxia, peripheral neuropathy, brittle hair, skin depigmentation, osteoporosis	Can mimic B12 deficiency
Iron	Blood loss, poor intake, pregnancy, chronic bleeding	Microcytic anemia	Low ferritin is the strongest simple clue
Selenium	Deficient intake, malabsorption, long-term PN	Cardiomyopathy, thyroid dysfunction, myopathy, immune dysfunction	Think heart + thyroid
Zinc	Malabsorption, chronic diarrhea, bowel resection, gastric bypass, long-term PN	Alopecia, periorificial or acral dermatitis, dysgeusia, poor wound healing, hypogonadism, immune dysfunction	Rash + hair loss + taste loss

Copper deficiency

Think of copper deficiency in a post-bariatric or malabsorptive patient who looks neurologically like B12 deficiency but also has hematologic or hair / skin changes.

- Common settings: prior bariatric or other gastric surgery, chronic malabsorption such as celiac disease or inflammatory bowel disease, and excess zinc exposure.
- Neurologic findings can include distal paresthesias, loss of vibration sense, loss of proprioception, sensory ataxia, and peripheral neuropathy; advanced disease can add myelopathic or upper motor neuron findings.
- Hematologic abnormalities include anemia and leukopenia. The anemia is often microcytic, but normocytic or macrocytic patterns can also occur.
- Associated clues include brittle hair, skin depigmentation, edema, hepatosplenomegaly, and osteoporosis.
- Diagnosis is supported by low serum copper and low ceruloplasmin; treatment is copper replacement and removal of excess zinc exposure.

Clinical pearl: posterior-column symptoms plus anemia after bariatric surgery should make you think about copper deficiency, not only B12 deficiency.

Zinc deficiency

Zinc deficiency is one of the most testable trace-mineral patterns because the stem often gives you rash, alopecia, poor healing, or loss of taste all at once.

- Risk settings: malabsorption, bowel resection, gastric bypass, poor oral intake, chronic diarrhea, and prolonged parenteral nutrition without adequate trace element replacement.
- Typical findings are alopecia, periorificial or acral dermatitis, hypogonadism, impaired wound healing, dysgeusia, and immune dysfunction.
- Dietary sources include meat, nuts, and fortified cereals; absorption occurs mainly in the duodenum and jejunum, so proximal small-bowel disease increases risk.

Clinical pearl: a patient on long-term parenteral nutrition with rash, alopecia, and poor wound healing should make you think of zinc deficiency first.

Fat-soluble vitamin details that are easy to miss

The basic A, D, E, and K table is already covered earlier in the note. The points below are the extra details that often show up in long-form explanations.

Vitamin A deficiency

- Major causes are poor intake and fat malabsorption, especially pancreatic insufficiency, cholestatic disease, and post-bariatric surgery states.
- Ocular progression: night blindness -> conjunctival xerosis -> Bitot spots -> corneal xerosis or ulceration in severe xerophthalmia.
- Nonocular clues include follicular hyperkeratosis / phrynoderma, impaired immune function, and poor growth in children.
- Low serum retinol supports the diagnosis. In a post-Roux-en-Y patient with dry eyes, night blindness, or Bitot spots, urgent replacement matters because delayed treatment can cost vision.

Vitamin E deficiency

- Classically causes a posterior-column / spinocerebellar pattern: ataxia, hyporeflexia, loss of vibration sense, loss of proprioception, and peripheral neuropathy.
- Can also cause skeletal myopathy, hemolysis, and sometimes pigmentary retinopathy.
- A rare long-standing association is brown bowel syndrome due to lipofuscin deposition in intestinal smooth muscle.

Vitamin K deficiency in newborns

- Newborns start with low stores because placental transfer is limited, early intestinal vitamin K production is minimal, and breast milk contains relatively little vitamin K.
- Missed prophylaxis can lead to vitamin K-deficient bleeding, especially in the first week of life and especially in exclusively breastfed infants.
- PT prolongs first because factor VII falls fastest; severe deficiency can prolong PTT as well.
- The board move is straightforward: intramuscular vitamin K prophylaxis at birth prevents this syndrome; suspected deficiency improves after vitamin K replacement.

13. Life-stage nutrition and counseling topics

Postmenopausal bone health

Item	Board-level target / reminder	Sources and cautions
Elemental calcium	Women older than 50 usually need about 1,200 mg/day total intake.	Food first when possible: milk, yogurt, fortified foods, dark green vegetables. In older adults, avoid chronic excess; high intake can contribute to hypercalcemia, hypercalciuria, and nephrolithiasis.
Vitamin D	Age 51-70: 600 IU/day. Older than 70: 800 IU/day. Many review resources summarize this as 600-800 IU/day in women over 50.	Sources include fortified dairy, fatty fish, and sunlight exposure; adult upper limit is 4,000 IU/day.

- Bone loss accelerates after menopause because estrogen falls; counseling should emphasize weight-bearing or resistance exercise, smoking cessation, and limiting excess alcohol.
- For boards, the key message is prevention of osteoporosis through diet, activity, and fall-risk awareness rather than indiscriminate megadose supplementation.

Nutrition in pregnancy

Prepregnancy BMI	Recommended total weight gain
<18.5 kg/m^2	12.5-18 kg (28-40 lb)
18.5-24.9 kg/m^2	11.5-16 kg (25-35 lb)
25-29.9 kg/m^2	7-11.5 kg (15-25 lb)
>=30 kg/m^2	5-9 kg (11-20 lb)

- Give or continue a daily prenatal vitamin. Folic acid matters before conception and in early pregnancy; iron or other supplements are added as indicated clinically.
- Avoid high-mercury fish, unpasteurized dairy, and raw or undercooked meat, fish, or eggs; wash produce well.
- Counsel about substance use, keep caffeine moderate, and use local obstetric guidance when counseling on fish intake and supplements.

Metabolic syndrome: what nutrition counseling questions are really testing

- Weight loss is the central therapeutic goal because it improves insulin sensitivity, blood pressure, triglycerides, and HDL patterns.

- First-line treatment is lifestyle change: balanced calorie-appropriate diet, physical activity, more fruits and vegetables, and less sugar-sweetened beverages and saturated fat.
- In question stems, nutrition counseling is usually the best initial answer before medications when the patient is stable and the problem is newly recognized metabolic syndrome.

Older adults with unintentional weight loss

- Start with serial weights, dietary and appetite history, medication review, physical examination, and simple screening labs such as CBC, chemistry profile, and thyroid studies.
- Escalate the workup according to the story: early satiety may justify upper endoscopy; ongoing unexplained loss may justify imaging or broader malignancy evaluation.
- Treatment focuses on the cause plus practical diet changes: remove unnecessary restrictions, encourage calorie-dense and protein-dense intake, and use oral nutrition supplements if diet changes alone are not enough.
- Routine appetite stimulants are not favored for most older adults because benefit is limited and adverse effects can outweigh the gain.

14. Severe acute malnutrition and nutrition support

Hypometabolic vs hypermetabolic states

Feature	Hypometabolic / starvation state	Hypermetabolic stress state
Typical setting	Chronic undernutrition without major inflammatory stress; risk pattern is marasmus	Injury, infection, burns, sepsis, major inflammation, or critical illness; risk pattern is acute malnutrition or kwashiorkor if needs are not met
Inflammatory and counter-regulatory signals	Lower overall cytokine and stress-hormone drive	Higher cytokines, catecholamines, cortisol, glucagon, and insulin resistance
Metabolic rate and oxygen use	Lower	Higher
Proteolysis and gluconeogenesis	Reduced compared with the stressed state	Increased
Fat use	Fat catabolism rises as an adaptation to starvation	Absolute fat and protein catabolism both rise
Adaptation to starvation	Relatively preserved	Abnormal or overwhelmed

High-yield idea: the starved but nonstressed patient adapts, whereas the critically ill or inflamed patient is catabolic and loses lean mass rapidly.

Severe malnutrition: bedside pattern and first moves

Topic	Key point
Types	Marasmus (wasting), kwashiorkor (edematous malnutrition), or a mixed picture.
Appearance	Marasmus causes severe loss of muscle and fat. Kwashiorkor adds edema that can hide weight loss and may come with fatty liver, hair change, or dermatosis.
First priorities	Rewarm if hypothermic, treat presumed infection, rehydrate carefully, correct electrolytes, and refeed cautiously.
Hydration pearl	In a stable chronically malnourished child, oral rehydration is preferred. IV fluids are reserved for shock and should be given more cautiously than a standard pediatric bolus because fluid overload and heart failure are real risks.
Major complications of management	Heart failure / fluid overload and refeeding syndrome.

- Do not jump straight to aggressive high-calorie feeding or total parenteral nutrition in a chronically malnourished patient. Slow refeeding and early phosphate, potassium, and magnesium attention are safer.

Infant younger than 6 months: simple SAM pathway

Step	How to think about it
1. Community screen	Poor weight gain or bilateral edema in an infant younger than 6 months should trigger referral for formal assessment.
2. Confirm severe acute malnutrition	Use weight-for-length below -3 Z-scores or bilateral pitting edema as the core diagnostic criteria.
3. Decide on inpatient vs outpatient care	Admit if there are medical complications, inability to drink or breastfeed effectively, vomiting everything, lethargy / unconsciousness, convulsions, recent weight loss or failure to gain, pitting edema, ineffective feeding, or important medical / social concerns.
4. Uncomplicated SAM	If those danger features are absent, outpatient management with close follow-up is acceptable.
5. Complicated SAM	If danger features are present, manage as inpatient severe acute malnutrition with rehydration, antibiotics when indicated, careful feeding support, and close reassessment.
6. Discharge target	Feeding is effective, weight gain is adequate, and weight-for-length has recovered to at least -2 Z-scores; then continue community monitoring to prevent relapse.

Critically ill patient: enteral versus parenteral nutrition

Topic	Enteral nutrition (EN)	Parenteral nutrition (PN / TPN)
When to prefer it	Preferred when the gut works and the patient can be fed through the GI tract, often within the first 24-48 hours of critical illness once reasonably stable.	Reserved for patients who cannot use the GI tract safely or adequately, or when enteral nutrition is contraindicated or insufficient.
Why it matters	Helps maintain gut integrity and is associated with fewer infectious complications than early parenteral feeding in many ICU settings.	Bypasses the gut and can provide complete nutrition, but it usually requires central venous access and carries more line-related and metabolic complications.
Practical route	Nasogastric or orogastric tube; advance postpyloric if aspiration risk is high.	Usually central venous catheter because the osmotic load is too high for routine peripheral use.
Board pearls	Start low and advance as tolerated.	Watch for catheter bloodstream

	Normal bowel sounds or recent stool are not required before beginning EN. D50 or D5-containing maintenance fluids are not nutrition support.	infection, hyperglycemia, liver dysfunction, essential fatty acid deficiency if lipids are omitted, and refeeding syndrome in malnourished patients.

Bottom line: if the gut works, use it. Parenteral nutrition is valuable when needed, but it is not the first move in a patient whose intestine is usable.

15. Sources:

- USMLE: official June 2026 nutrition-content enhancement announcement and FAQ.
- NIH Office of Dietary Supplements fact sheets: vitamins A, D, E, K, copper, chromium, selenium, zinc, iron, and calcium.
- CDC material on vitamin K prophylaxis and vitamin K deficiency bleeding in newborns.
- WHO materials on xerophthalmia / vitamin A deficiency, severe acute malnutrition, and infants younger than 6 months with SAM.
- ACOG and National Academies pregnancy guidance for weight gain and counseling topics.
- FDA / EPA and CDC patient guidance for fish safety and food safety in pregnancy.
- NICE, ASPEN, and ESPEN guidance for refeeding risk, nutrition support, critical care feeding, and nutrition in older adults.
- AHA / NHLBI lifestyle guidance for metabolic syndrome and cardiometabolic risk reduction.