Refeeding Syndrome

The harm comes from the feeding, not the starving · Identify the risk before the first calorie goes in

PHOSPHATE
the sentinel electrolyte

WHY FEEDING IS THE DANGEROUS PART *the physiology explains every step of the protocol*

WHAT STARVATION DID

Prolonged starvation shifts metabolism to fat and protein and **depletes intracellular phosphate, potassium, magnesium and thiamine**. **The serum levels can look entirely normal** throughout, because the deficit is intracellular — which is precisely why a reassuring baseline panel gives false comfort.

WHAT FEEDING DOES

Carbohydrate triggers a surge of insulin, which drives **glucose, phosphate, potassium and magnesium rapidly into cells**. Serum levels then **collapse within 24 to 72 hours**. Insulin also causes **sodium and water retention**, precipitating edema and cardiac failure in an already atrophied heart.

WHY PHOSPHATE MATTERS MOST

Phosphate is required to make ATP. Severe hypophosphatemia therefore causes **respiratory muscle failure, cardiac failure and arrhythmia, rhabdomyolysis, hemolysis, seizures and delirium**. **Thiamine is consumed as a cofactor of carbohydrate metabolism**, so refeeding can precipitate **Wernicke's encephalopathy**.

IDENTIFY WHO IS AT RISK *score this before writing the feeding order, not afterwards*

Category	Criteria
High risk — ANY one of	**BMI below 16 · unintentional weight loss over 15% in the past 3 to 6 months · little or no nutritional intake for more than 10 days · low potassium, phosphate or magnesium before feeding starts.**
High risk — TWO or more of	**BMI below 18.5 · unintentional weight loss over 10% in 3 to 6 months · little or no intake for more than 5 days · a history of alcohol misuse, or of insulin, chemotherapy, antacids or diuretics.**
Extremely high risk	**BMI below 14, or negligible intake for more than 15 days.** These patients need **cardiac monitoring** and the slowest possible start, with specialist nutrition input from the outset and a clearly documented daily review of the electrolytes and the feed rate.
Populations to think of	**Anorexia nervosa, alcohol use disorder, chronic alcohol-related liver disease, advanced malignancy, post-bariatric surgery, prolonged nil-by-mouth or ICU stay, the frail elderly, homelessness, and post-operative patients whose feeding was repeatedly deferred.**
It is not just tube feeding	**Oral, enteral and parenteral nutrition can all cause it — and so can intravenous dextrose alone.** A starving patient started on maintenance dextrose is being refed, and that is a routinely missed route to the syndrome. **The risk is also carried into and out of hospital**: flag it clearly at handover and on discharge, since the vulnerable window extends across the first week of feeding wherever the patient happens to be.

BEFORE AND DURING FEEDING *correct alongside, do not delay nutrition to correct first*

1. **Check phosphate, potassium, magnesium, calcium, sodium, glucose and renal function**, and get a baseline weight and ECG in the higher-risk groups.
2. **Give thiamine before or with the first feed**, with a vitamin B complex and a multivitamin. **Dose and duration are below.**
3. **Start low: about 10 kcal/kg/day**, and **as little as 5 kcal/kg/day in the extremely high-risk patient**. **Increase slowly to full requirements over 4 to 7 days**, guided by the electrolytes rather than by the calorie target.
4. **Do not withhold feeding until the electrolytes are normal. Correct them in parallel with feeding** — starving the patient further to fix a phosphate is a common and harmful misreading of the protocol. Replace phosphate, potassium and magnesium aggressively and repeatedly.
5. **Monitor electrolytes at least daily for the first 3 days and through the first week**, with **fluid balance, daily weight and cardiac monitoring in the highest-risk patients**. **Restrict sodium and be cautious with fluid**: the atrophied heart tolerates volume poorly, and edema plus a rising weight in the first days is fluid, not nutrition.
6. **Address why they were not eating.** Refeeding is a symptom of something else — an eating disorder, alcohol dependence, dysphagia, depression, malignancy, or simply a hospital process that left someone NPO for days. **In anorexia nervosa involve the eating disorder service early** and consider **capacity and the relevant legal framework**, because under-feeding out of caution is itself a recognized cause of death in that group.

THE NUMBERS *what to give, and how slowly to feed*

THIAMINE FIRST

200-300 mg daily, IV or oral, starting before any carbohydrate and continuing at least 10 days. Glucose consumes thiamine as a cofactor, so **feeding a depleted patient can precipitate Wernicke encephalopathy**. It is cheap and harmless: give it rather than debating it.

START LOW, GO SLOW

10 kcal/kg/day, or **5 kcal/kg/day in extreme risk** (BMI under 14, or negligible intake for more than 15 days). **Increase slowly over 4 to 7 days** to full requirement. **The feed is the exposure**, so the rate of increase matters more than the target.

CORRECT AS YOU FEED

Do not delay feeding to normalize electrolytes; correct alongside. Check **phosphate, potassium and magnesium at baseline and daily for the first 3 days**, then as needed. **A falling phosphate after feeding starts is the diagnosis**, and it can fall precipitously in the first 72 h.

RECOGNIZING IT WHEN IT HAPPENS *the presentation is non-specific and easily misattributed*

WHAT YOU WILL SEE

Within 24 to 72 hours of starting feeding: falling phosphate, potassium and magnesium; peripheral edema and a rising weight; breathlessness and respiratory muscle weakness; tachycardia, arrhythmia and heart failure; confusion, weakness, seizures; and **rhabdomyolysis or hemolysis** in severe cases. **These are routinely blamed on the underlying illness**, which is why the risk assessment before feeding matters more than pattern recognition afterwards.

WHAT TO DO

Reduce or briefly pause the feed rate rather than stopping nutrition altogether, and **replace electrolytes intravenously and repeatedly** — single doses are rarely enough because the intracellular deficit is large. **Give thiamine if it has not already been given.** Treat fluid overload, monitor the rhythm, and rebuild the feed rate more slowly once stable. **Involve the dietitian and nutrition team**, who should ideally have been involved before the first feed.

PEARLS & PITFALLS

- **Normal baseline electrolytes do not mean low risk.** The deficit is intracellular and only appears once insulin drives the shift.
- **Thiamine goes in before the carbohydrate**, not after. Feeding first can precipitate Wernicke's encephalopathy.
- **Intravenous dextrose counts as refeeding**, and so does a high-carbohydrate oral supplement. A starving patient started on maintenance fluid is already being refed, and is already at risk of the syndrome.
- **Do not starve the patient to fix the phosphate.** Replace and feed at the same time, slowly. Withholding nutrition is itself a cause of harm and death.
- **Weight gain in the first few days is fluid.** Restrict sodium and watch for heart failure rather than celebrating the number.
- **Score the risk before you write the feed.** This syndrome is almost entirely preventable and almost never diagnosed early.

References: NICE clinical guideline on nutrition support for adults (risk criteria and refeeding protocol) · ASPEN consensus recommendations for refeeding syndrome · ESPEN guidance on clinical nutrition in the hospitalized patient.

Educational aid; verify against local nutrition and dietetic protocol, renal function and patient factors.