

Severe Acute Malnutrition

Wasting, marasmus, kwashiorkor and organ pathology

An integrated clinicopathologic lecture

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Learning objectives

Define severe acute malnutrition using the criteria reported.

Distinguish somatic from visceral protein depletion.

Explain the marasmus and kwashiorkor phenotypes mechanistically.

Connect liver, intestinal and marrow lesions to clinical findings.

Severe wasting is a life-threatening disorder

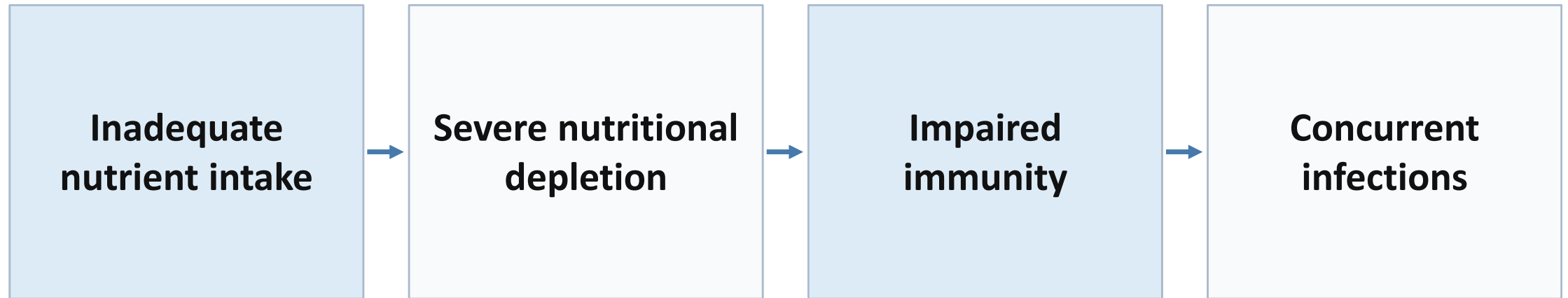
Severe acute malnutrition is a pediatric wasting syndrome associated with inadequate nutrient intake and/or recurrent illness.

Immune defenses are weakened.

Long-term developmental delays are possible.

Severe wasting increases the risk of death.

Inadequate intake and illness reinforce each other



Infections add inflammation and metabolic stress, worsening malnutrition.

Primary versus secondary malnutrition

Primary insufficiency

One or more essential dietary components are missing.

Food supply, poverty and inadequate dietary variety can contribute.

Secondary insufficiency

Intestinal absorption, utilization or storage may fail.

Excess loss or increased metabolic need can create deficits.

Wasting burden in 2022

45 million

Children under 5 affected by wasting

6.8%

13.6 million

Children under 5 affected by severe
wasting

2.1%

These are historical 2022 estimates reported, not updated 2026 surveillance.

Wasting and stunting are distinct phenomena

Wasting

An acute or recent deficit manifested by low weight relative to height.

Severe wasting is the focus of this lecture.

Stunting

A consequence of malnutrition beginning in utero or early childhood.

Approximately 148 million children under 5 were stunted in 2022 (textbook estimate).

Severe wasting is concentrated geographically

>75%

Of children with severe wasting live in
Asia

2022 estimate

22%

Live in Africa

2022 estimate

The geographic distribution of children with severe wasting.

Three diagnostic pathways identify severe wasting

MUAC

<115 mm

Weight-for-height

**More than 3 SD below
normal**

Bilateral pitting edema

Lower limbs

The source presents these as alternative criteria for severe acute malnutrition.

Mid-upper arm circumference: Severe threshold

Mid-upper arm circumference (MUAC) <115 mm

MUAC is a direct anthropometric measurement.

Reduced midarm circumference reflects loss of skeletal-muscle mass.

Interpret in the pediatric assessment context.

Weight-for-height: Marked deviation from reference



This as a severe anthropometric criterion; No unverified cutoffs are added.

Bilateral pitting edema is a diagnostic clue

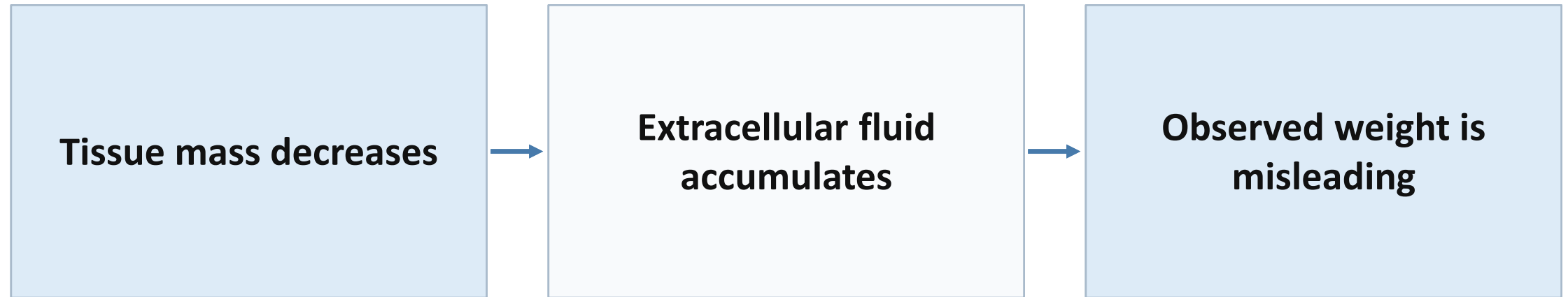
Bilateral pitting edema of the lower limbs is a diagnostic criterion.

Examine for fluid accumulation rather than judging by apparent body size.

Edema is especially characteristic of kwashiorkor.

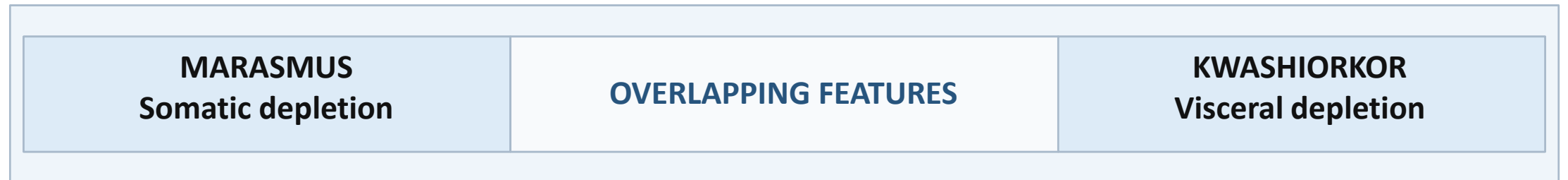
Excess fluid can conceal the underlying tissue loss.

Weight can conceal depletion when edema is present



Children with kwashiorkor may have substantially more tissue loss than the scale suggests.

Marasmus and kwashiorkor define a clinical spectrum



These endpoints overlap; Individual children may show features of both.

The two functional protein compartments

Somatic compartment

Proteins in skeletal muscle.

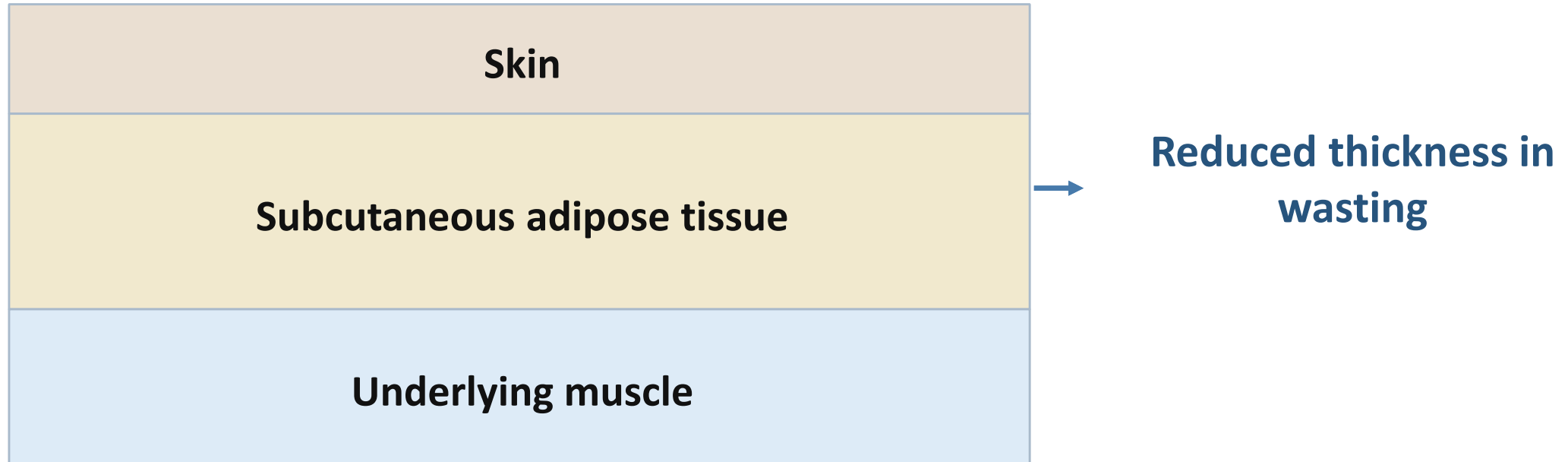
Prominent depletion produces visible muscle wasting.

Visceral compartment

Protein stores in visceral organs, particularly the liver.

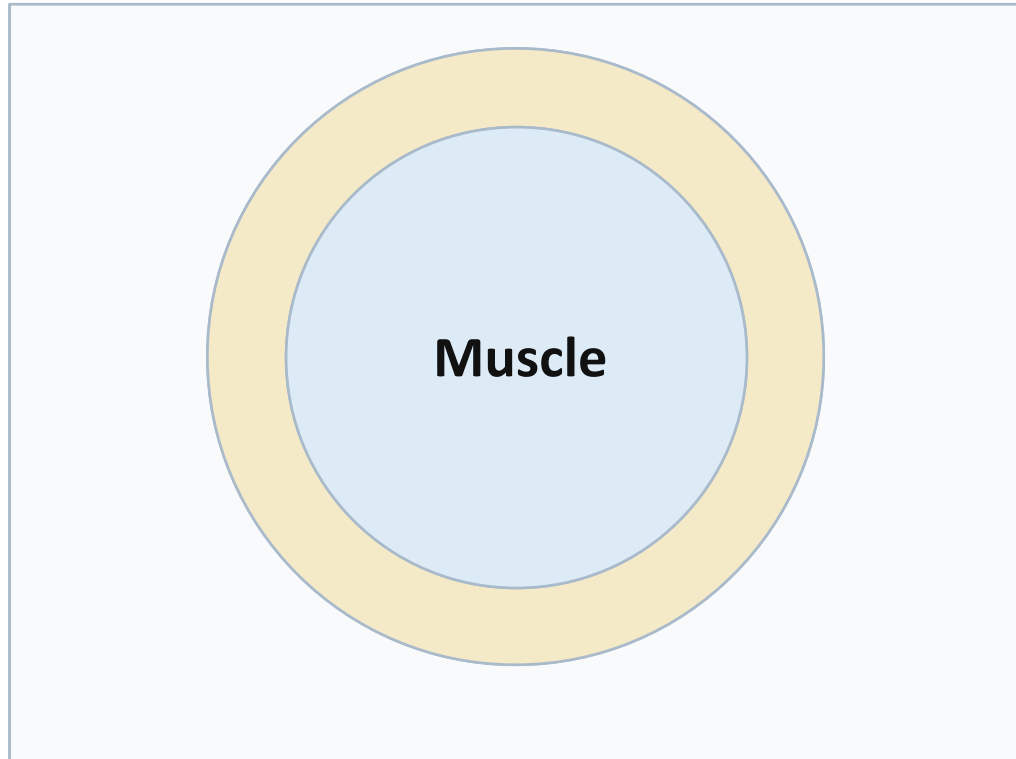
Marked depletion is associated with low serum proteins and edema.

Skinfold thickness reflects subcutaneous fat



Skinfold measurement includes skin and subcutaneous tissue; Fat loss reduces the measured thickness.

Midarm circumference reflects muscle depletion



Subcutaneous fat

Skeletal muscle

Circumference decreases when muscle is lost

A smaller midarm circumference can reflect loss of skeletal-muscle protein.

Serum proteins provide visceral compartment indices

Albumin and transferrin as serum markers.

Their concentrations can help estimate visceral protein status.

They do not replace examination of muscle, fat and edema.

Interpret the laboratory measurements within the overall clinical picture.

Assessment integrates complementary observations

Observation	What it informs
Weight relative to height	Anthropometric wasting
MUAC / midarm circumference	Somatic muscle depletion
Skinfold thickness	Subcutaneous fat loss
Bilateral pitting edema	Fluid-retaining phenotype
Albumin, transferrin	Visceral protein compartment

Gut microbiome patterns differ in affected children

Observed association

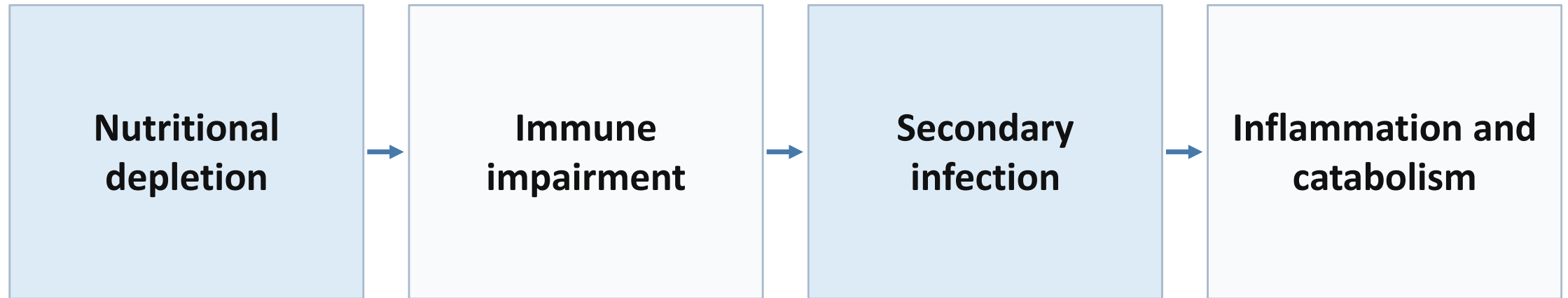
Children with severe acute malnutrition have microbial flora differing from those of well-nourished children.

Mechanistic uncertainty

A contributory role in pathogenesis is possible.

The source does not establish that a specific bacterial change causes SAM.

Infection increases the burden of malnutrition



The infection–malnutrition cycle worsens physiologic reserve.

Marasmus arises from severe caloric deprivation

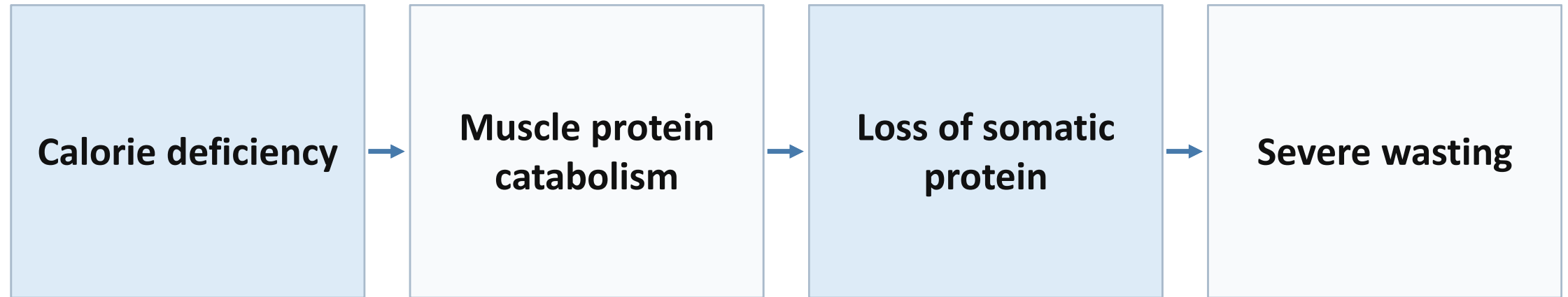
Marasmus is the energy-deficient end of the severe malnutrition spectrum.

Severe calorie insufficiency.

Marked loss of fat and muscle is characteristic.

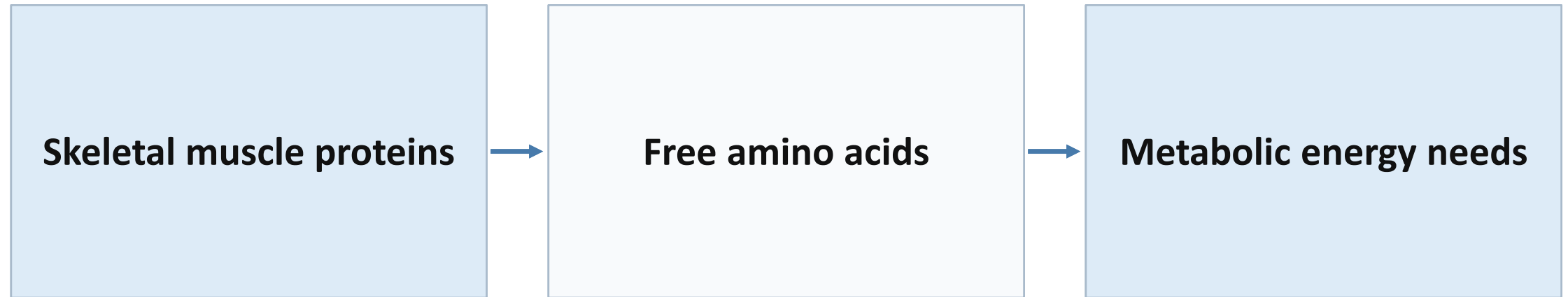
It reports a historical weight criterion of 60% of normal for sex, height and age.

Marasmus preferentially depletes somatic protein



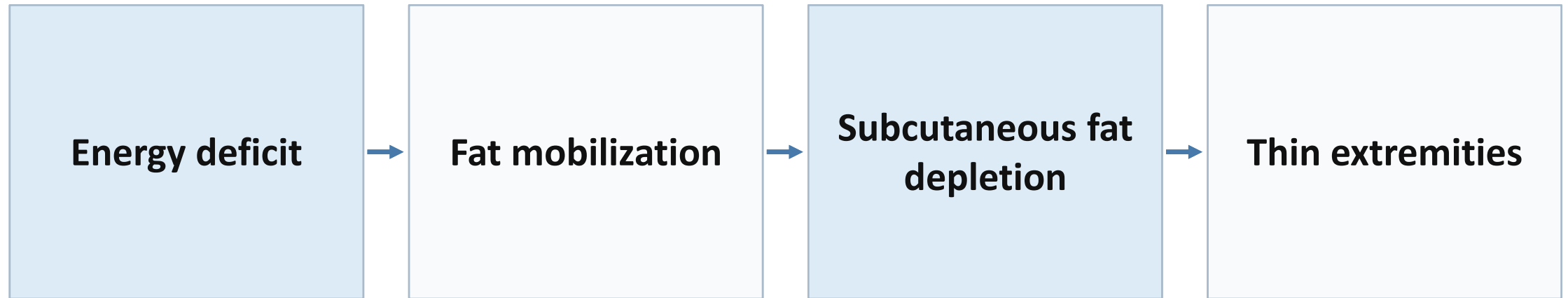
Muscle protein is mobilized as part of the adaptive response to energy deficiency.

Catabolized muscle supplies amino acids



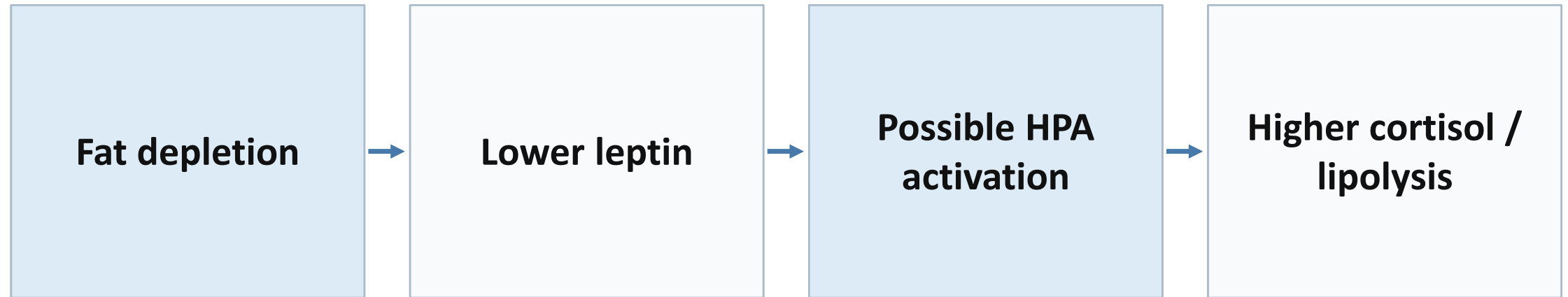
This energy-supporting adaptation occurs at the expense of muscle mass.

Subcutaneous fat becomes an energy source



Marasmus produces severe losses of both muscle and subcutaneous fat.

Leptin and cortisol: A proposed endocrine link



The leptin–HPA–cortisol sequence as a possible mechanism, not a proven universal pathway.

Albumin can be relatively preserved in marasmus

Marasmus

Visceral protein compartment is depleted only marginally.

Albumin is often normal or only slightly reduced.

Kwashiorkor

Visceral depletion is severe.

Hypoalbuminemia is associated with edema.

The marasmic body habitus reflects tissue loss

Extremities are markedly emaciated.

Skeletal-muscle loss is accompanied by subcutaneous fat depletion.

The head appears disproportionately large relative to the wasted body.

Growth retardation accompanies the chronic energy deficit.

T-cell immunity is impaired in marasmus



Intercurrent infection imposes additional stress on a physiologically weakened child.

Marasmus often has additional deficits

Anemia is a common associated manifestation.

Multiple vitamin deficiencies may coexist.

Cell-mediated immunodeficiency increases infectious complications.

A marasmic phenotype does not mean isolated calorie deficiency without other deficits.

Kwashiorkor reflects disproportionate protein lack

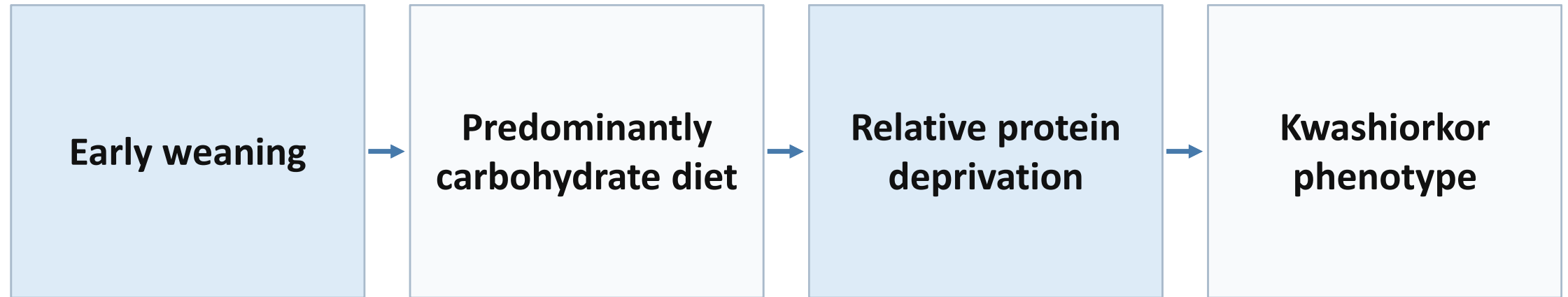
Protein deprivation is relatively greater than the fall in total calories.

Visceral protein is markedly depleted.

Fluid retention produces the characteristic edema.

The two classical phenotypes are endpoints, not separate sealed categories.

Dietary transitions can contribute to kwashiorkor



This feeding pattern is the textbook's classic setting; It is not the only etiologic pathway.

Protein loss and malabsorption create other settings

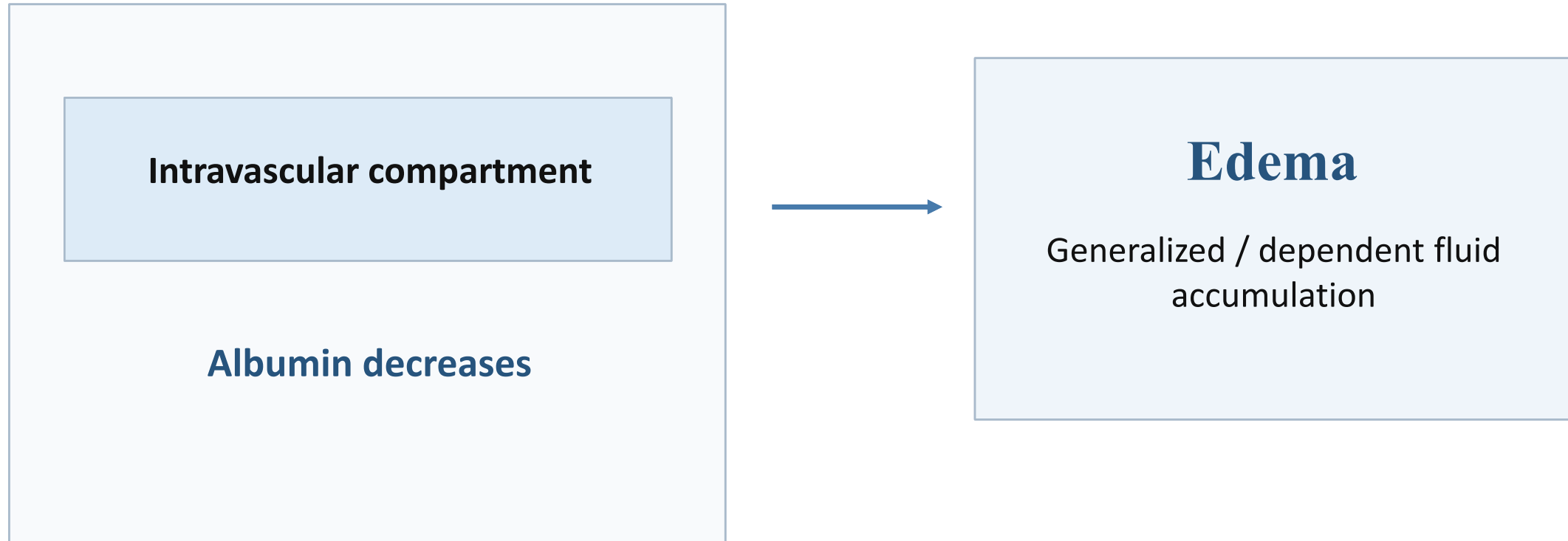
Clinical context	Mechanism identified in Robbins
Chronic diarrheal states	Impaired protein absorption
Protein-losing enteropathy	Gastrointestinal protein loss
Nephrotic syndrome	Renal protein loss
Extensive burns	Loss of body protein
Restrictive / unusual diets	Marked dietary protein inadequacy

Visceral protein depletion is central in kwashiorkor



The pathophysiology directly links a visceral-compartment deficit to the edematous phenotype.

Hypoalbuminemia accompanies fluid accumulation



Generalized or dependent edema can be prominent, including facial and limb puffiness and ascites.

Retained fluid masks the extent of tissue loss

A child with severe kwashiorkor may appear heavier because of edema.

Historical weights of 60%–80% of normal.

Observed weight can overestimate remaining tissue mass.

Examination must account for edema when interpreting nutritional status.

Muscle and fat may seem less reduced in kwashiorkor

Relative preservation

Compared with marasmus, muscle and subcutaneous fat are less depleted.

Important caveat

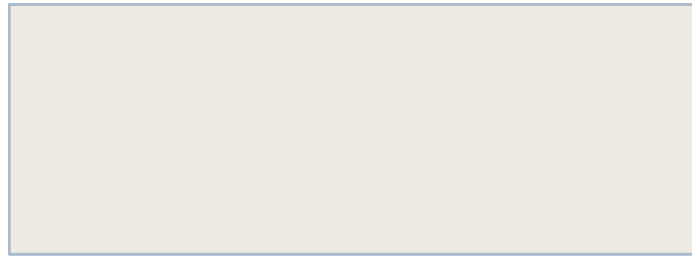
Edema can also mask losses in these compartments.

Relative sparing does not mean normal tissue reserves.

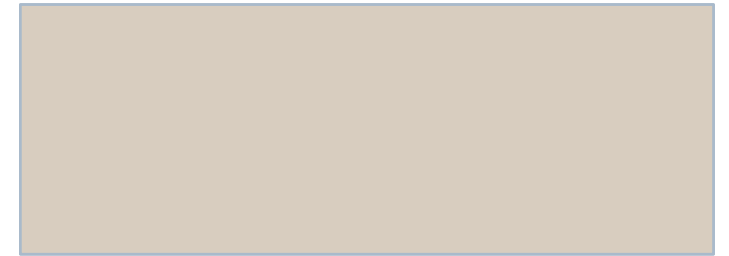
Kwashiorkor dermatosis has a “flaky paint” pattern



Hyperpigmentation



Desquamation



Hypopigmentation

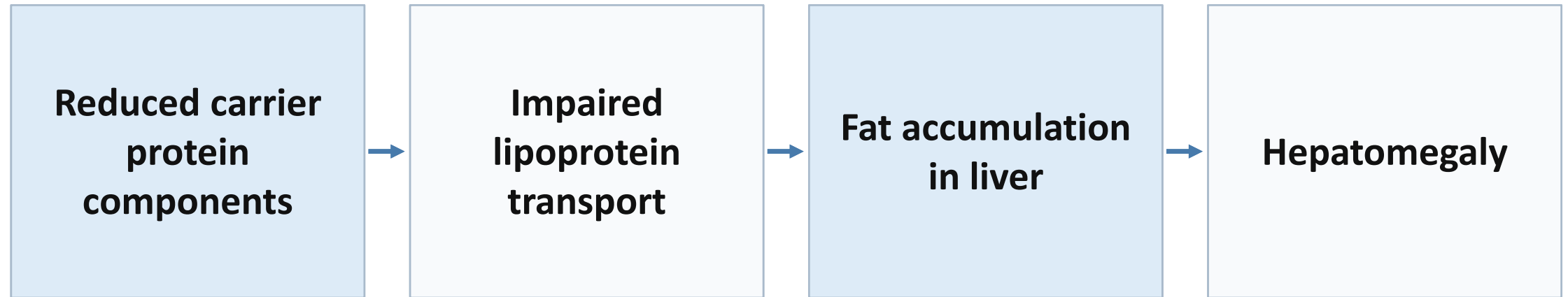
Schematic of descriptive features — not a clinical photograph

Alternating hyperpigmentation, desquamation and hypopigmentation form the characteristic described appearance.

Kwashiorkor changes hair texture and pigmentation

Feature	Textbook description
Pigmentation	Loss of color or alternating pale and dark bands
Architecture	Hair may straighten and become fine
Attachment	Hair is less firmly attached to the scalp

The enlarged fatty liver is diagnostically useful



Fatty hepatomegaly in kwashiorkor, in contrast to marasmus.

Apathy and poor appetite distinguish kwashiorkor

Apathy and listlessness are characteristic clinical changes.

Loss of appetite may accompany the visceral protein deficit.

Edema, dermatosis, hair changes and fatty liver strengthen the pattern.

Both phenotypes share nutritional and immune harm

Marasmus

Anemia and multiple vitamin deficiencies.

Defects in cell-mediated immunity.

Concurrent infection may be present.

Kwashiorkor

Associated vitamin deficiencies.

Immune defects and secondary infection.

Infection-driven catabolism worsens the state.

Marasmus and kwashiorkor compared

Feature	Marasmus	Kwashiorkor
Dominant deficit	Total calories	Protein disproportionate
Protein store	Somatic / muscle	Visceral / liver
Subcutaneous fat	Markedly lost	Relatively spared
Albumin	Normal / mildly low	Markedly decreased
Edema	Not a defining feature	Prominent
Liver	Usually not fatty	Fatty, often enlarged
Skin / hair	No classic pattern specified	Flaky paint; color / texture changes

Three principal anatomic findings in severe malnutrition

Growth

Growth failure

Fluid

**Peripheral edema:
Kwashiorkor**

Tissue

**Fat loss + muscle
atrophy**

The relative prominence of these findings varies by phenotype.

The liver is fatty in kwashiorkor, not marasmus

Kwashiorkor

Enlarged, fatty liver is characteristic.

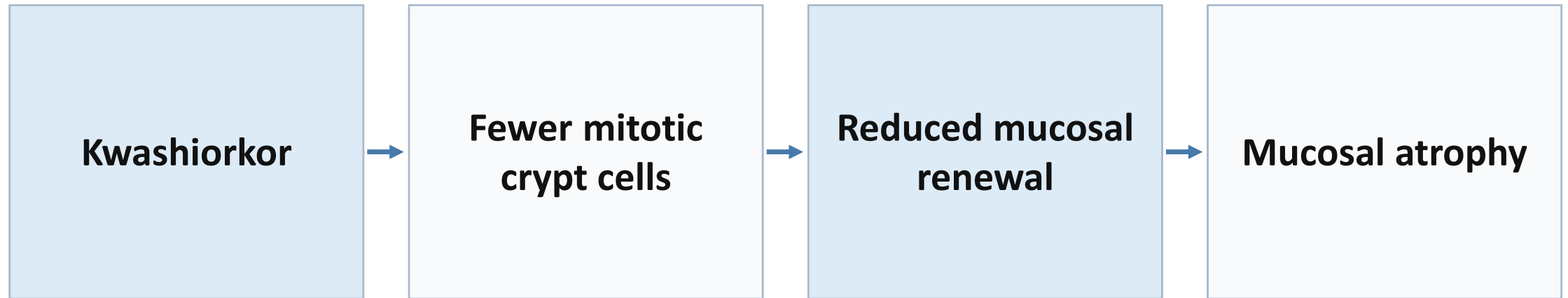
Superimposed cirrhosis is rare.

Marasmus

Fatty hepatomegaly is not characteristic in the cited morphologic description.

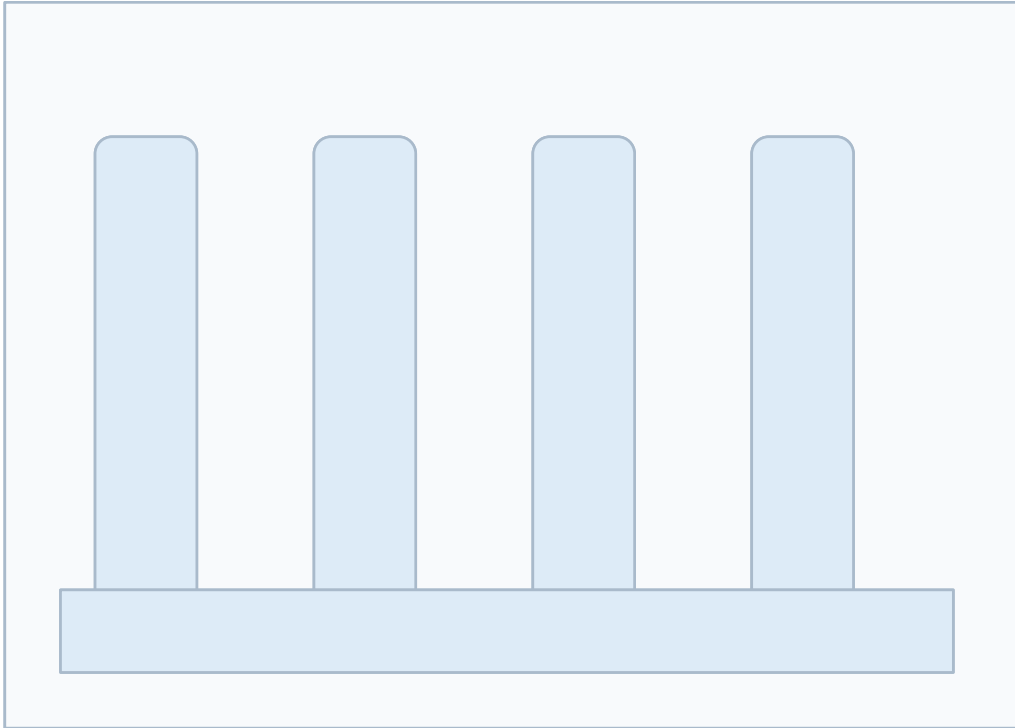
Marked peripheral fat and muscle loss predominate.

Intestinal crypt proliferation may fall

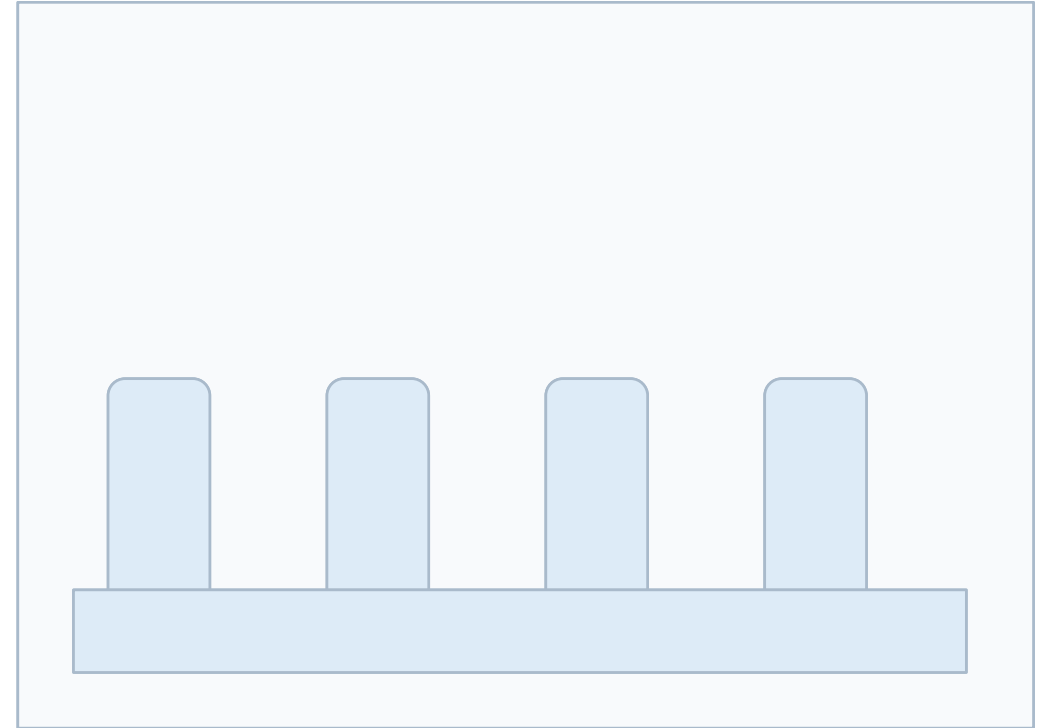


This crypt abnormality is described in kwashiorkor, rarely in marasmus.

Small bowel villi and microvilli may atrophy



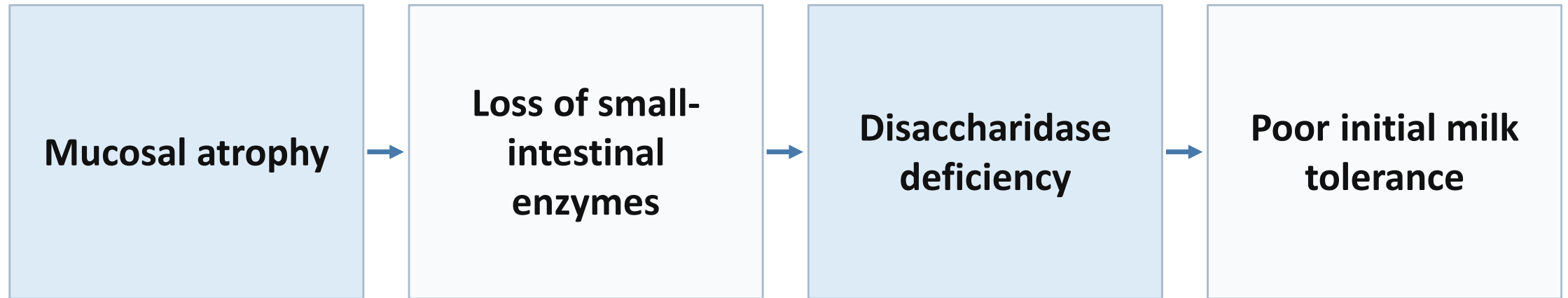
Reference mucosa



Kwashiorkor-associated atrophy

Schematic only: Relative villous height is illustrative; Microvilli are not drawn

Disaccharidase deficiency follows mucosal injury



Full-strength milk-based diets may initially be poorly tolerated.

Intestinal mucosal changes can reverse

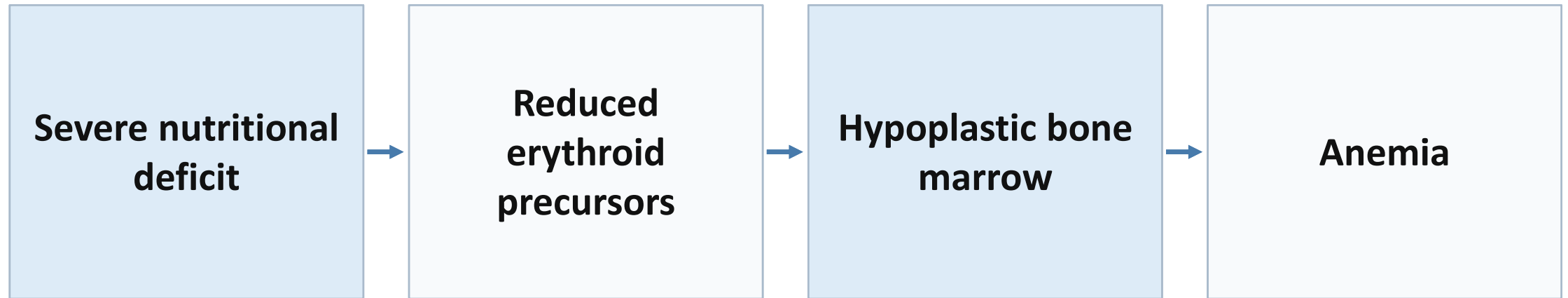
The intestinal mucosal changes described in kwashiorkor are reversible with treatment.

Crypt cell activity and mucosal structure can recover.

Enzyme depletion helps explain temporary feeding intolerance.

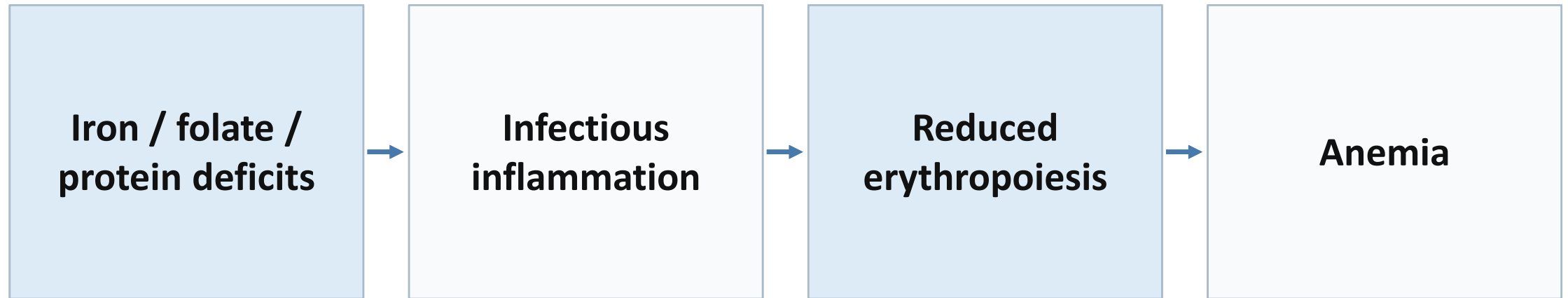
This slide reports pathology and does not prescribe a treatment protocol.

Bone marrow may become hypoplastic



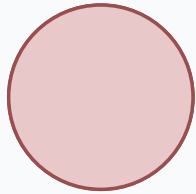
The marrow change is reported in both marasmus and kwashiorkor.

Anemia has overlapping etiologies

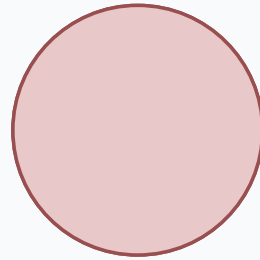


Robbins characterizes the anemia as usually mild to moderate and multifactorial.

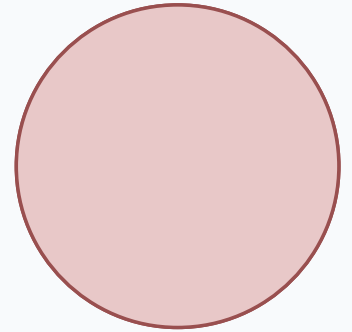
Red-cell size depends on the dominant factor



Microcytic



Normocytic



Macrocytic

Microcytic, normocytic or macrocytic anemia may occur; Size alone does not identify the nutritional syndrome.

Schematic: Not peripheral blood-smear imagery

Read organ morphology as a connected process

Site	Lesion	Clinicopathologic implication
Skeletal muscle	Atrophy / catabolism	Visible wasting
Liver	Steatosis in kwashiorkor	Enlargement
Small bowel	Villus/microvillus loss	Disaccharidase deficiency
Bone marrow	Reduced red-cell precursors	Anemia
Extracellular space	Edema in kwashiorkor	Apparent mass can be misleading

Case: Severe wasting without edema

A young child has strikingly thin limbs, very little subcutaneous fat, growth failure, and no prominent edema. Albumin is not markedly reduced.

Which protein compartment is most depleted, and which phenotype predominates?

Case interpretation: Marasmus-predominant

Somatic (skeletal-muscle) protein loss predominates.

Energy deficiency mobilizes muscle protein and adipose stores.

Visceral proteins may be relatively preserved.

The phenotype is marasmus-predominant.

Case: Edema, hair change and enlarged liver

A child has bilateral pitting edema, facial puffiness, sparse pale hair, skin desquamation and hepatomegaly despite a less strikingly low apparent weight.

What explains the edema, and which phenotype is most likely?

Case interpretation: Kwashiorkor-predominant

Visceral protein loss with hypoalbuminemia explains the edema.

Fluid retention masks actual tissue depletion.

Fatty liver, dermatosis and hair changes support kwashiorkor.

Clinical syndromes can still overlap.

Pathogenesis connects to the characteristic lesions

Mechanism	Principal lesion or sign
Somatic protein catabolism	Skeletal-muscle atrophy
Fat mobilization	Decreased subcutaneous fat
Visceral protein depletion	Low albumin, edema
Lipoprotein-component deficit	Hepatic steatosis
Reduced intestinal crypt renewal	Mucosal atrophy / enzyme loss
Reduced erythroid precursors	Anemia

Primary references and scope

Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th ed. Environmental and Nutritional Diseases (nutritional disorders section).

Chapter sections: Dietary Insufficiency; Severe Acute Malnutrition; Marasmus; Kwashiorkor; Morphology.

Burden estimates correspond to 2022 data quoted by the textbook; no external updates or treatment protocols have been inserted.