

Osteochondroma

OSTEOCHONDROMA

Cartilage-capped surface lesion of bone

Morphology · Molecular pathogenesis · Clinical correlation

Dr Abu Suraih Sakhri

MBBS, MD (Pathology)

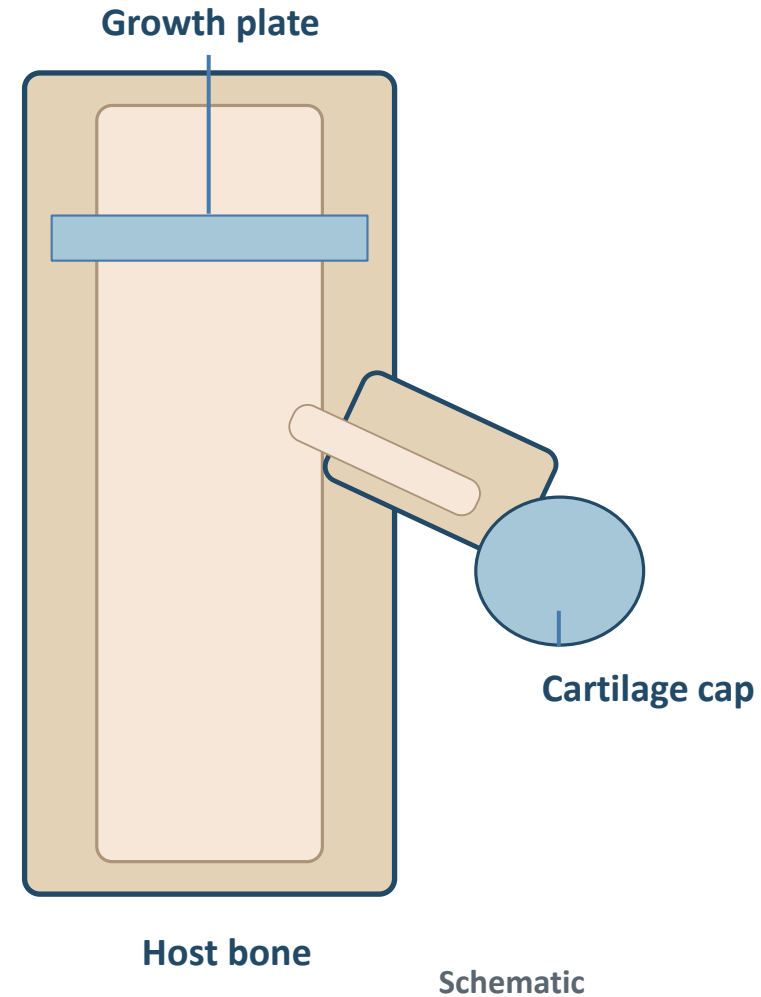
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Learning objectives

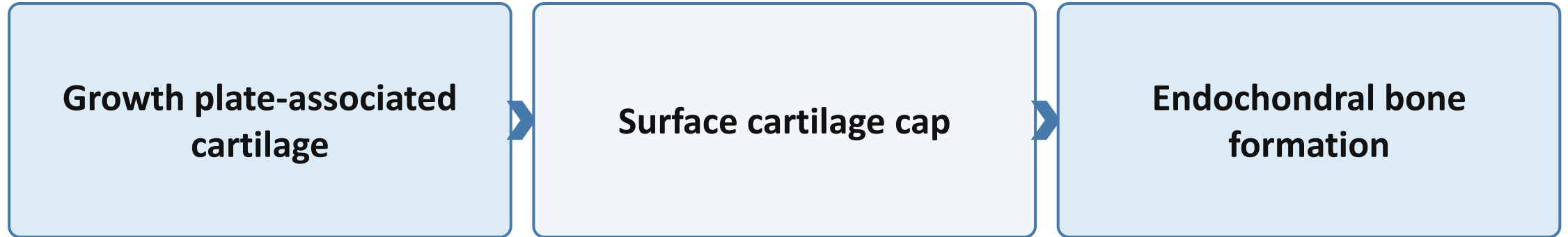
- Recognize the defining architecture of an osteochondroma.
- Explain the connection to the growth plate and EXT1/EXT2.
- Integrate gross, microscopic, and radiographic morphology.
- Distinguish solitary disease, hereditary disease, and malignant transformation.

Osteochondroma: Essential definition

- Benign cartilage-capped lesion arising from the bone surface.
- A bony projection carries the hyaline cartilage cap.
- Both sessile and pedunculated forms are recognized.



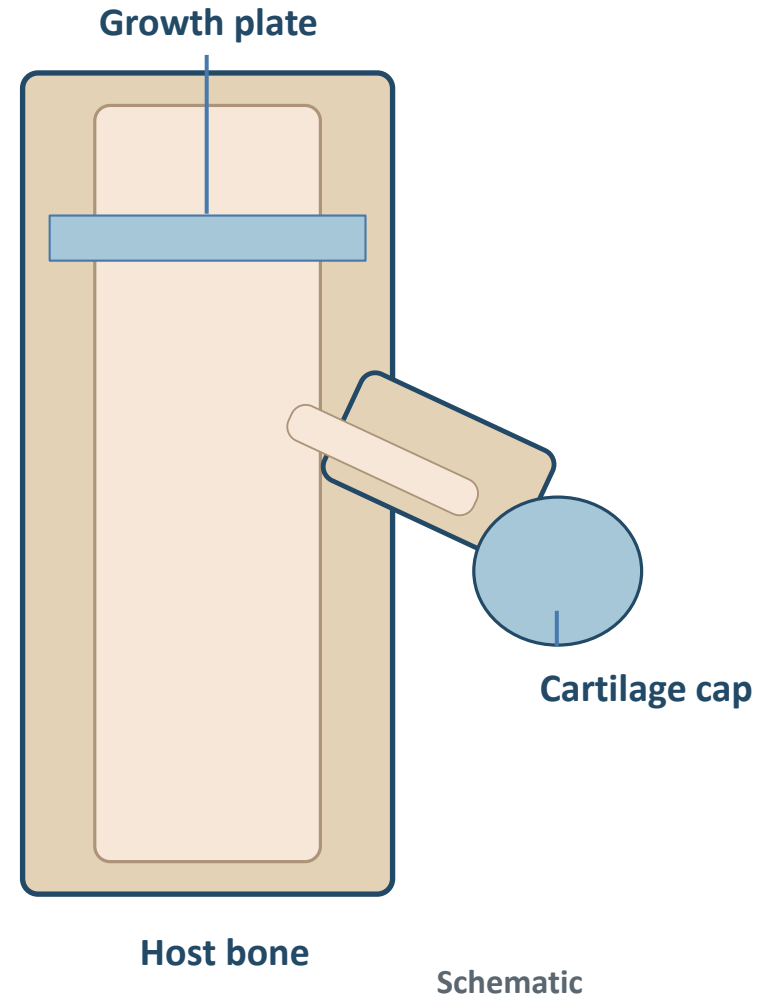
Why “cartilage-capped” matters



Schematic sequence

Anatomical origin: An endochondral bone

- Osteochondromas occur in bones formed by endochondral ossification.
- The metaphysis near a growth plate is the characteristic site.
- The lesion is topographically related to developing cartilage.

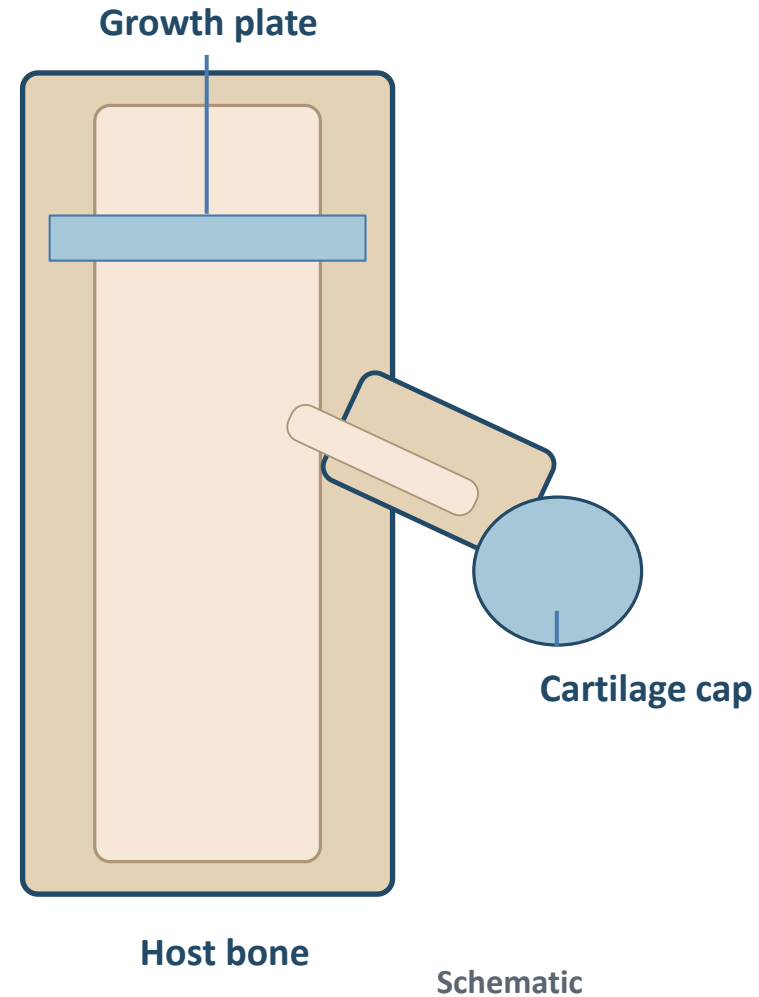


Two clinically important presentations

Solitary / sporadic	Hereditary multiple exostoses
Approximately 85% of cases	Remaining cases occur with syndrome
Usually recognized in late adolescence / early adulthood	Often apparent during childhood
Single osteochondroma	Multiple lesions may cause skeletal deformity

Distribution: Long tubular bones

- The metaphyses of long tubular bones are the commonest location.
- The region around the knee is particularly frequent.
- A surface projection may be readily visible on imaging.



Other locations: Pelvis, scapula, ribs

- After long bones, pelvic bones, scapula, and ribs are important sites.
- Osteochondromas at these sites often have relatively short stalks.
- A broad or short attachment can make the projection less obvious.

Age patterns reflect skeletal growth

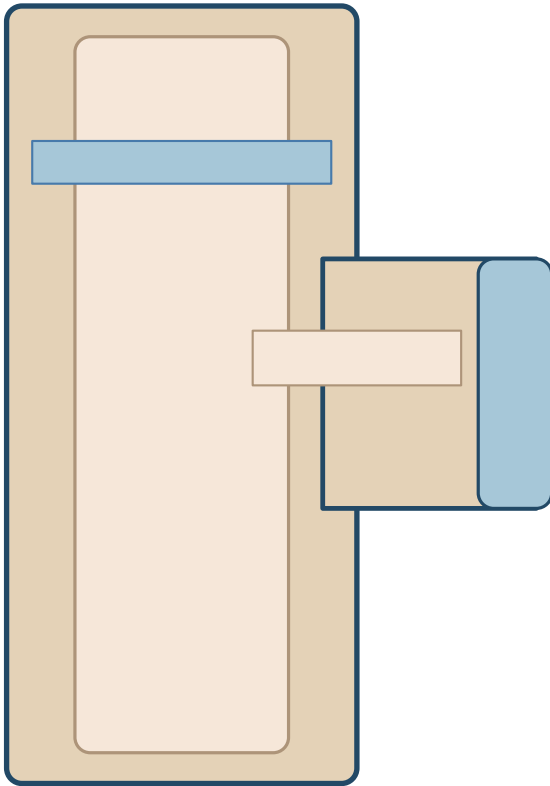
Sporadic osteochondroma	Multiple hereditary exostoses
Usually diagnosed in late adolescence or early adulthood	Typically recognized during childhood
Often one slow-growing lesion	Multiple lesions during skeletal development
Expansion usually ceases after growth-plate closure	Deformity reflects altered epiphyseal growth

Sex distribution in sporadic disease

Male predominance

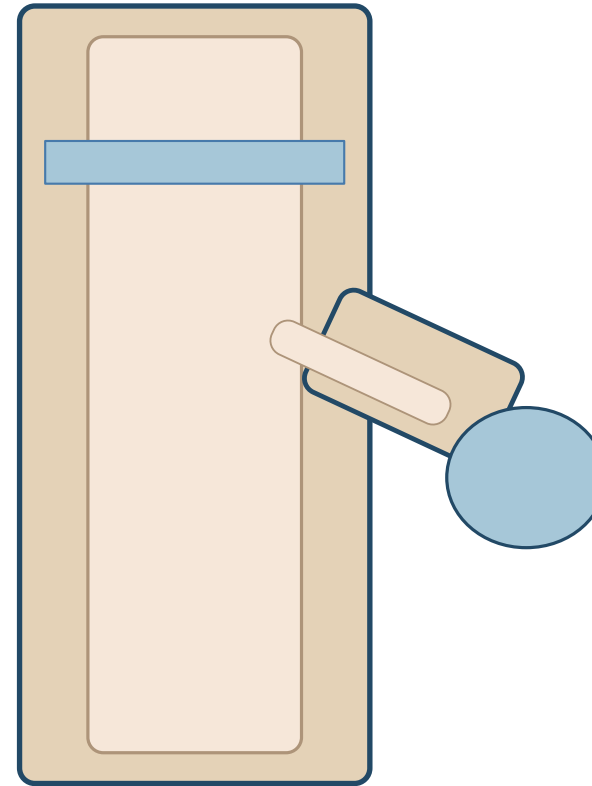
Males affected approximately three times more often than females in sporadic osteochondroma.

External configuration: Sessile or stalked



Schematic

Sessile = broad-based projection

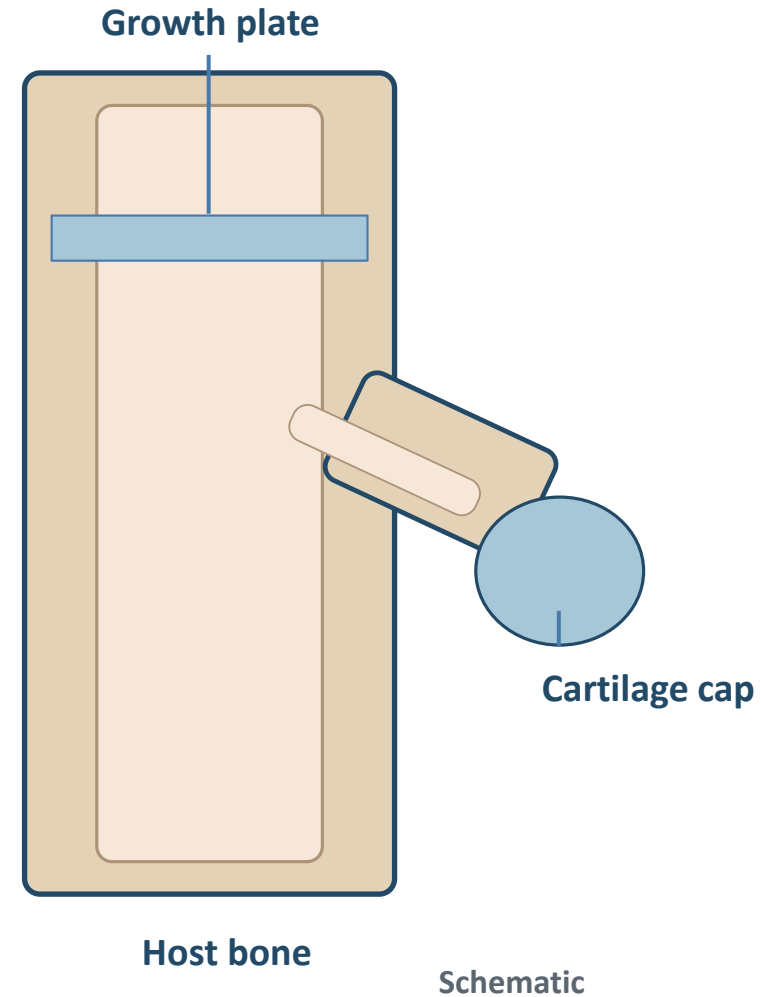


Schematic

Pedunculated = projection with bony stalk

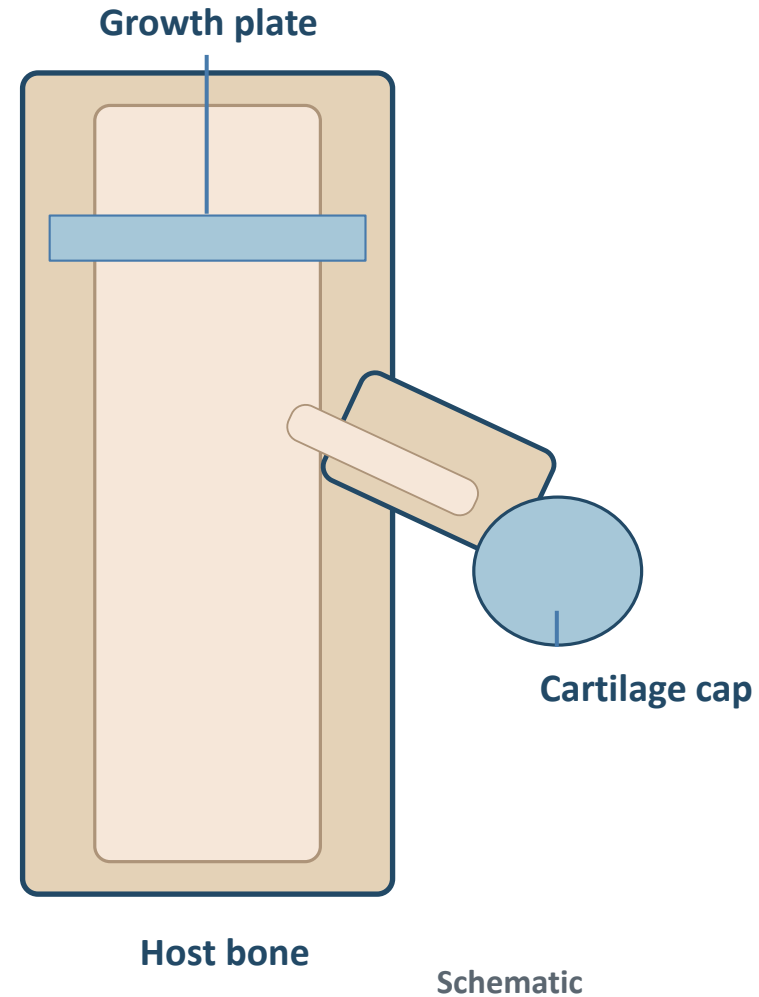
The decisive structural relationship

- Cortex of the lesion merges with host-bone cortex.
- Medullary cavity of the lesion continues with host marrow.
- This architecture is the key to recognizing osteochondroma.

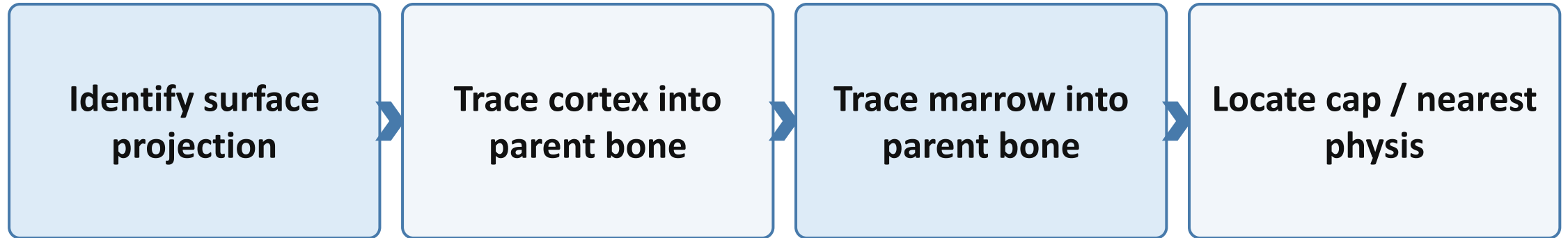


Growth plate and direction of the cap

- Lesions originate near the metaphyseal growth plate.
- On plain radiographs the cap points away from the nearest physis.
- Orientation is a clue; Continuity remains decisive.



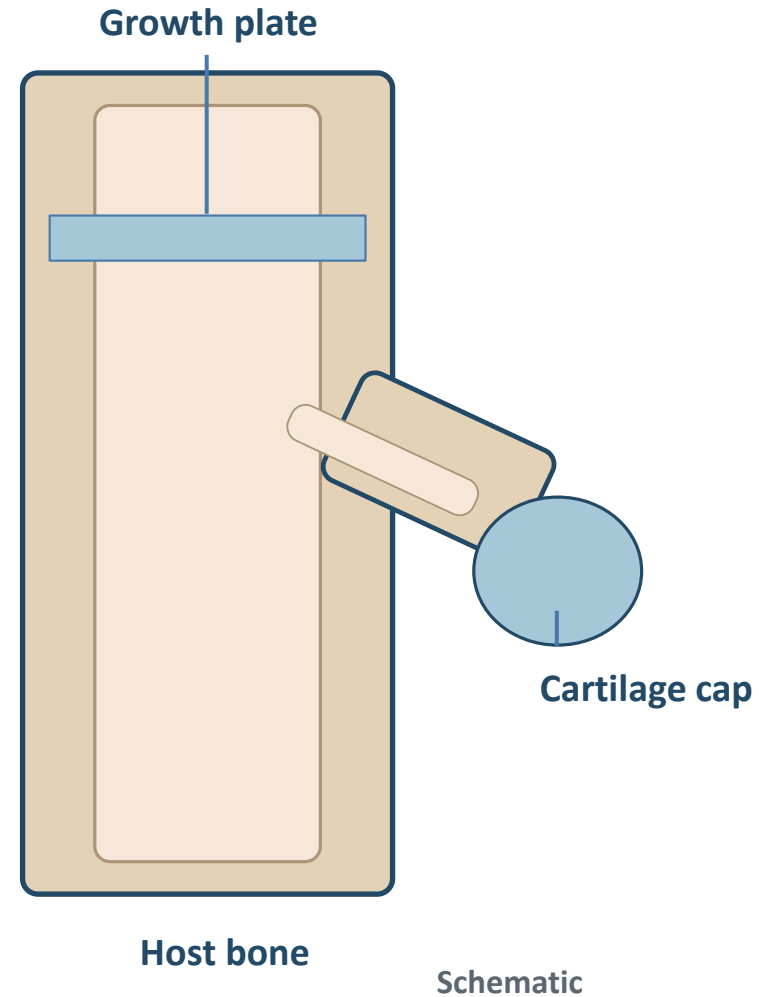
Reading an osteochondroma on radiographs



Schematic sequence

Metaphyseal anatomy: A location-based clue

- The metaphysis lies next to the epiphyseal growth plate.
- Osteochondroma morphology develops in relation to this zone.
- Location, outward growth, and continuity reinforce the diagnosis.



Solitary lesion versus syndromic multiplicity

One lesion

A solitary bone-surface mass is the usual presentation.

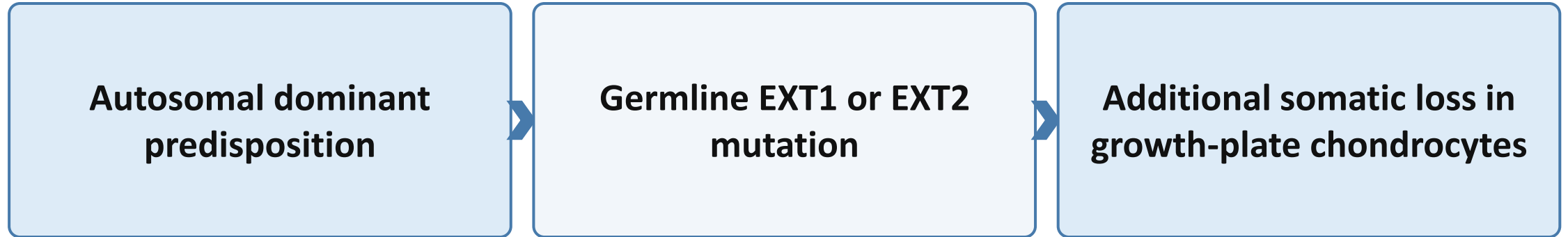
Multiple lesions

Multiple exostoses prompt consideration of the hereditary syndrome.

Skeletal effects

Multiple lesions may disturb growth and cause bowing or shortening.

Hereditary multiple exostoses: Inheritance



Schematic sequence

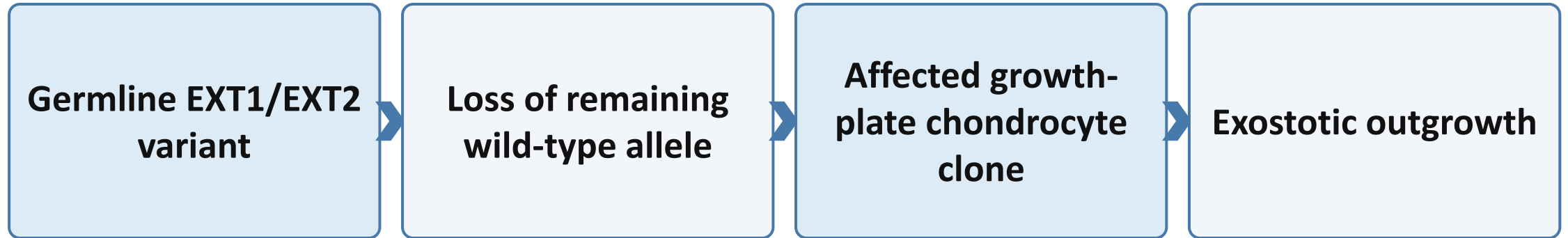
EXT1 and EXT2: Molecular function

- EXT1 and EXT2 encode glycosyltransferases.
- These proteins participate in heparan sulfate biosynthesis.
- Disruption affects growth-plate chondrocyte behavior.



Schematic molecular relationship

Hereditary disease: The second hit



Schematic sequence

Why a growth-plate progenitor matters

Cell of origin

The relevant somatic event occurs in a growth-plate chondrocyte.

Proliferation

The altered progenitor population may proliferate inappropriately.

Polarity

Loss of normal directional organization may favor outward growth.

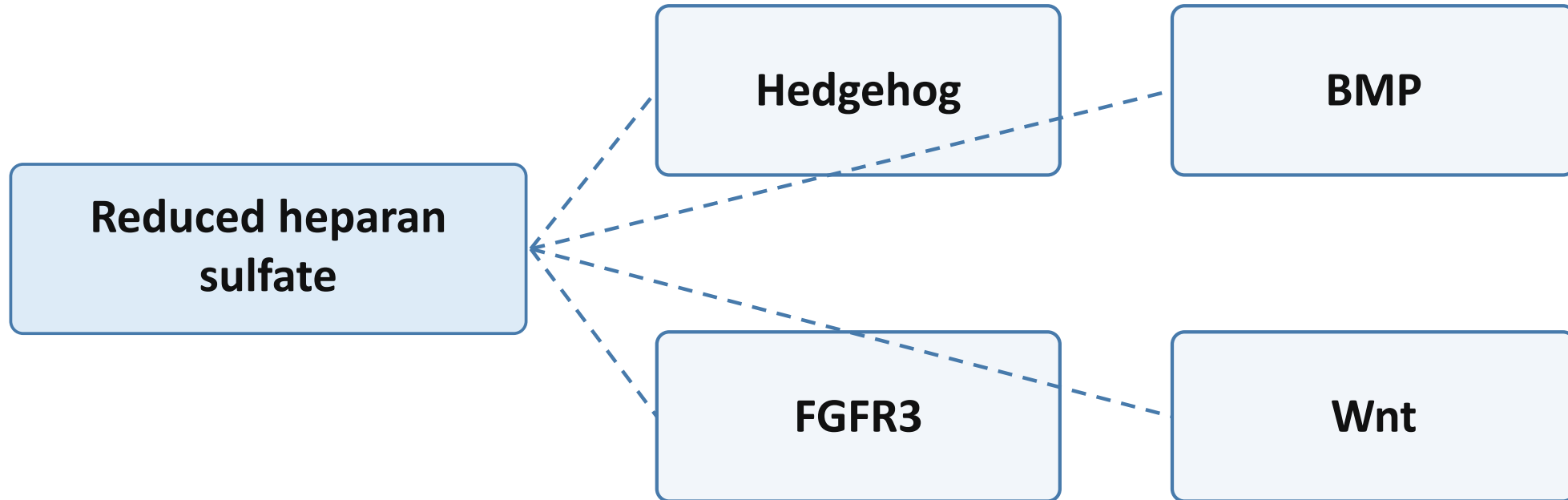
Loss of heparan sulfate: Upstream event

- EXT-related loss impairs heparan sulfate synthesis.
- Heparan sulfate normally contributes to local growth-factor signaling.
- Its reduction may disrupt growth-plate organization.



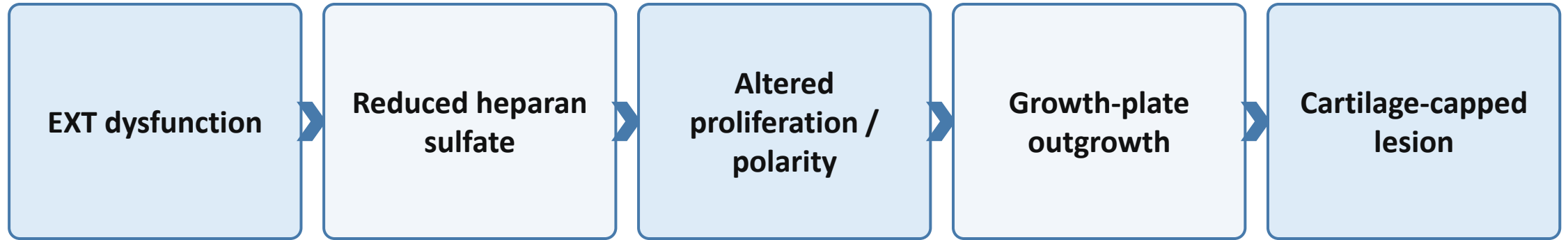
Schematic molecular relationship

Signaling pathways secondarily perturbed



Pathway perturbation (not a proven linear cascade)

Pathogenesis: Integrated proposed sequence



Schematic sequence

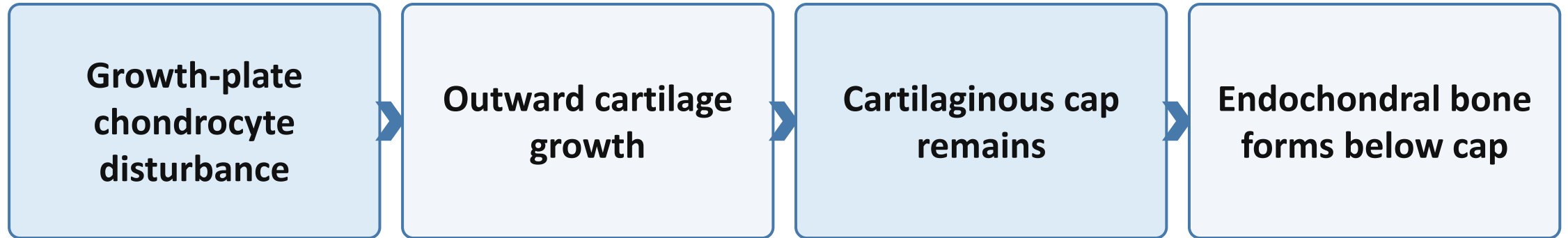
Sporadic osteochondroma: What is known

- Reduced expression of EXT1 or EXT2 has been observed in sporadic lesions.
- This is distinct from the established inherited two-hit model.
- Robbins does not assign one universal mutation pattern to all sporadic cases.

Molecular mechanism: Evidence versus inference

Established	Mechanistic interpretation
Hereditary lesions associate with EXT1 / EXT2 loss	Growth-plate signaling perturbation is downstream
EXT enzymes produce heparan sulfate	Progenitor proliferation and loss of polarity may result
Several pathways are implicated	Not a single direct linear signaling pathway

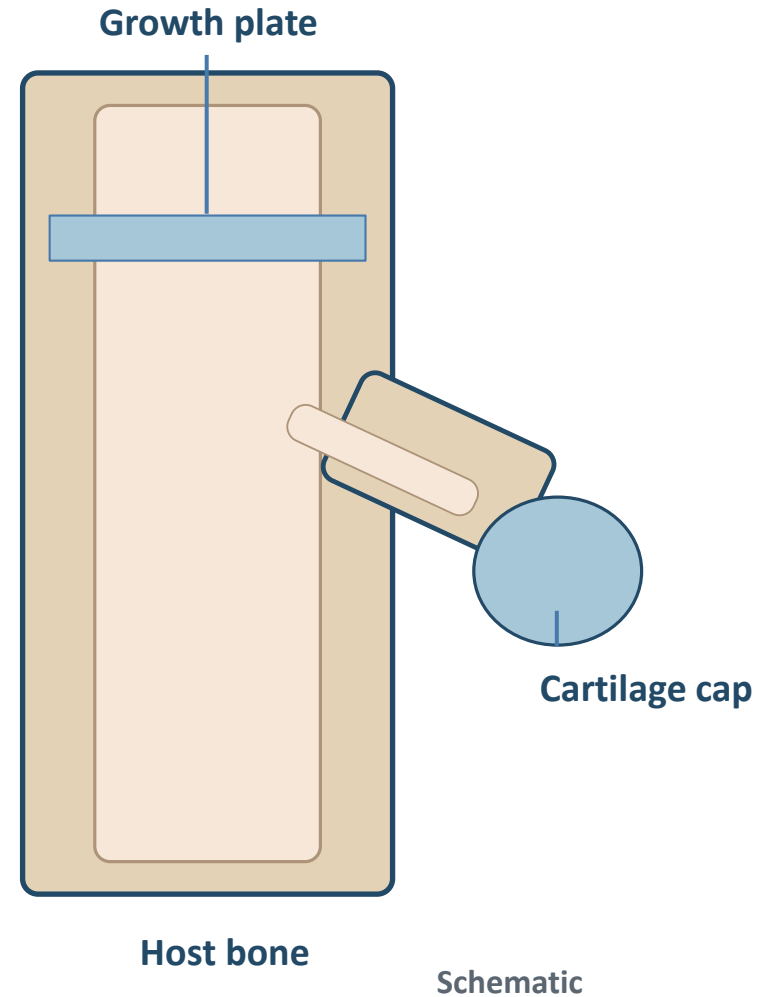
How a surface outgrowth develops



Schematic sequence

Osteochondroma and growth-plate closure

- Osteochondromas ordinarily stop expanding as the growth plate closes.
- Persistent enlargement after skeletal maturity deserves evaluation.
- Growth arrest supports dependence on developmental cartilage.



Gross size and shape

Size

Lesions ranging approximately 1–20 cm.

Attachment

Sessile forms have broad bases;
Pedunculated forms have stalks.

Composition

The projecting bone is covered by a cartilage cap.

Gross anatomy: Cartilage, bone, marrow

Perichondrium

Cartilage cap: Benign hyaline cartilage

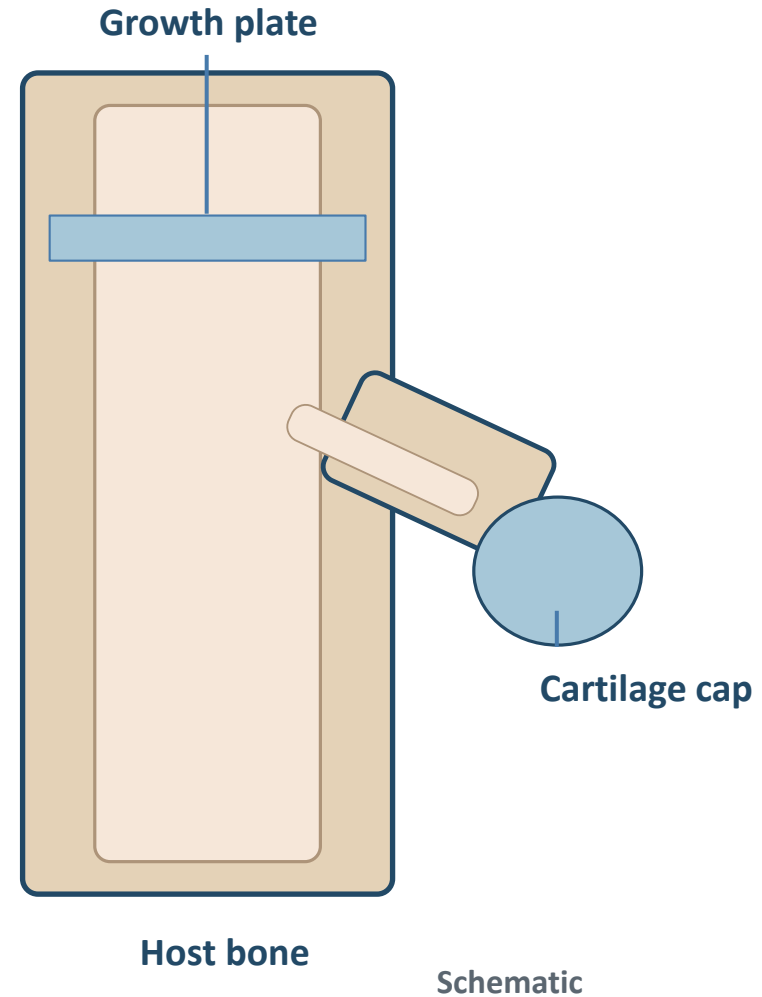
Endochondral ossification zone

Bone and continuous marrow

Schematic cross-section; Not a photomicrograph

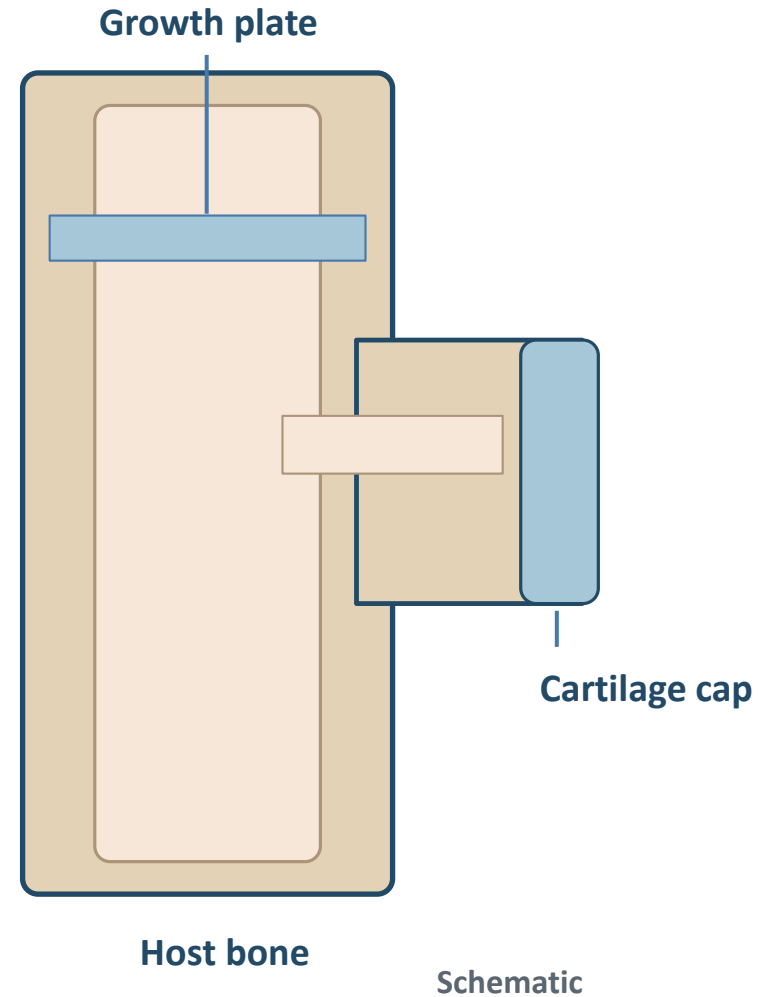
Pedunculated osteochondroma: Anatomy

- A projecting bony stalk arises from the host cortex.
- Marrow continues into the projection.
- The cartilage cap covers the head of the outgrowth.



Sessile osteochondroma: Anatomy

- The lesion has a broad attachment to the host bone.
- The continuity of cortical and medullary components still applies.
- The cartilage cap overlies the surface.



Cartilage cap: Tissue identity

- The cap is composed of benign hyaline cartilage.
- It is invested by perichondrium.
- It resembles an abnormally organized growth plate.

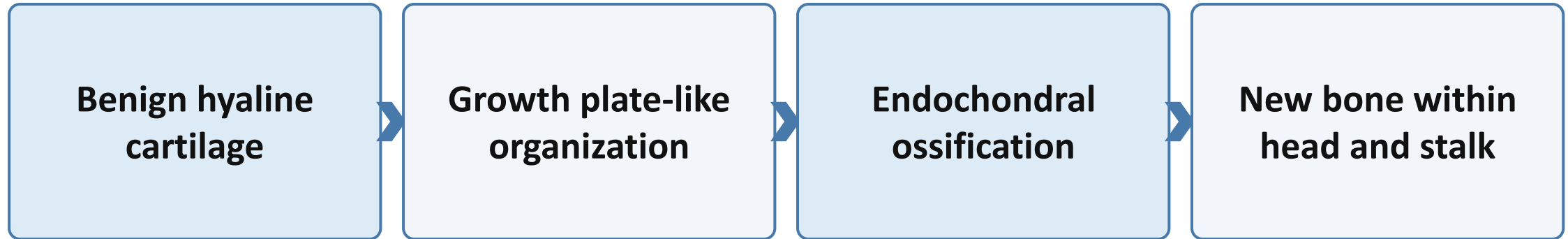
Perichondrium

Hyaline cartilage

Endochondral bone

Schematic

Endochondral ossification beneath the cap



Schematic sequence

Microscopic architecture: Interpret by zones

Perichondrium

Cartilage cap: Benign hyaline cartilage

Endochondral ossification zone

Bone and continuous marrow

Schematic cross-section; Not a photomicrograph

Why the cap looks like a growth plate

Normal growth-plate principle	Osteochondroma cap
Organized cartilage supporting longitudinal growth	Disorganized growth plate-like cartilage
Endochondral ossification at a defined interface	Cartilage undergoes endochondral ossification
Bone extends along skeletal axis	Bone forms within protruding head and stalk

Perichondrium is part of the cap

- The cartilage cap is covered by perichondrium.
- Recognition requires considering the surface covering and the underlying cartilage.
- It is a structural layer rather than proof of malignancy.

Perichondrium

Hyaline cartilage

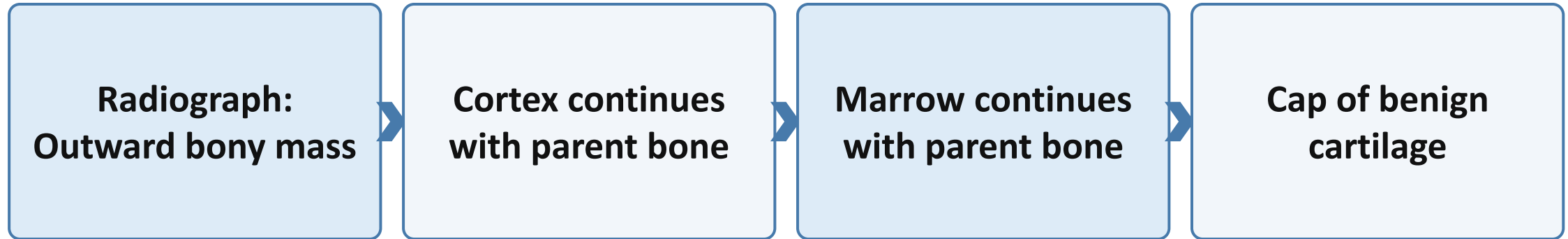
Endochondral bone

Schematic

Histologic cellularity: Interpret in context

- Hereditary multiple osteochondromas may be more cellular than sporadic lesions.
- Greater cytologic atypia can also occur in hereditary lesions.
- Neither finding alone establishes secondary chondrosarcoma.

Anatomic continuity links imaging and histology



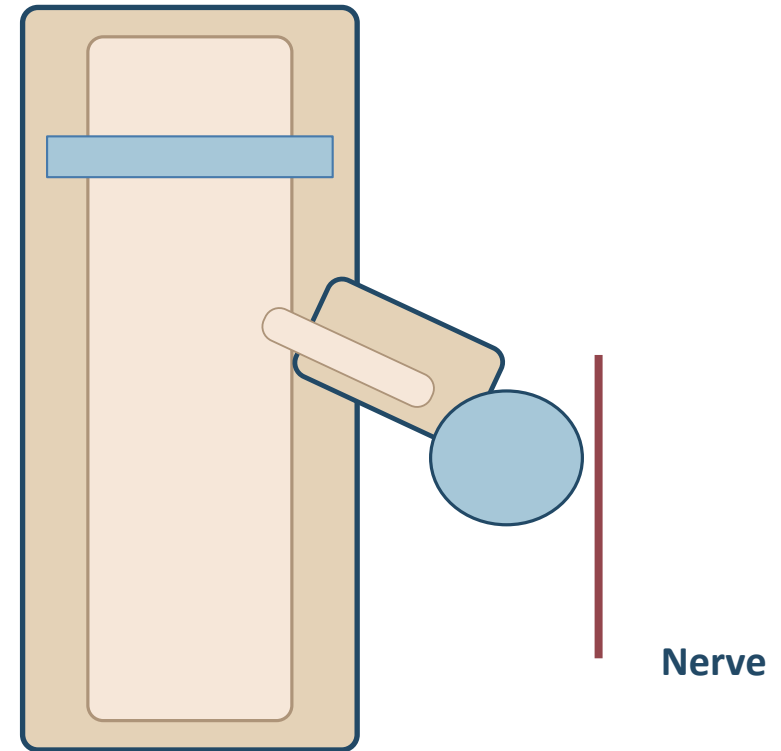
Schematic sequence

Clinical presentation: Painless growth

- The typical lesion is a slow-growing bony mass.
- Many lesions are discovered incidentally.
- A symptomatic lesion often produces local mechanical effects rather than systemic illness.

Mechanical pain: Compression of a nerve

- Pain may result when the protrusion impinges on a nerve.
- The mechanism is local anatomy, not necessarily aggressive tumor growth.
- Correlate lesion position with the symptomatic structure.

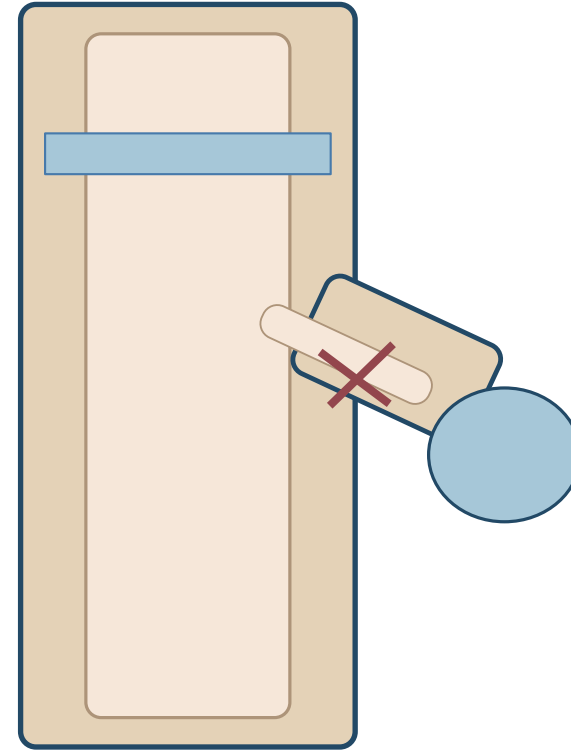


Schematic

Adjacent nerve impingement

Mechanical pain: Fracture of the stalk

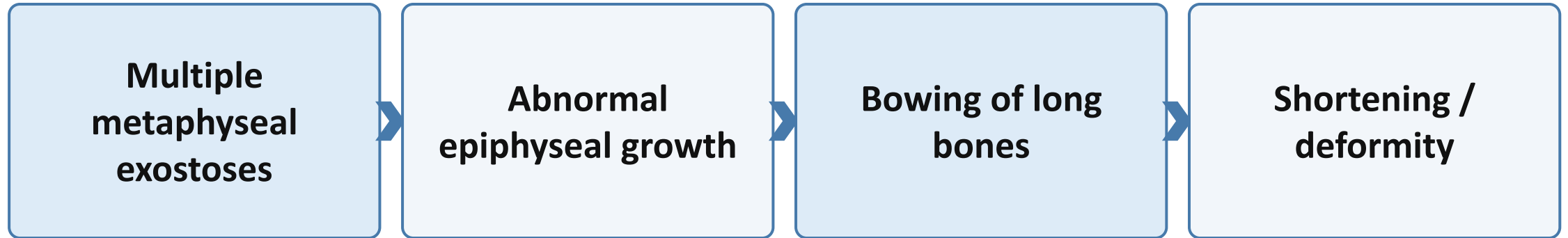
- A pedunculated lesion can sustain fracture of its bony stalk.
- A previously painless lesion may then become painful.
- A fractured stalk is distinct from malignant transformation.



Schematic

Possible stalk fracture

Growth disturbance in hereditary disease



Schematic sequence

Sporadic versus hereditary: Clinical phenotype

Solitary sporadic	Multiple hereditary
Single slow-growing surface lesion	Numerous lesions may appear in childhood
Bowing is not a defining feature	Bowing and shortening may occur
Malignant transformation is rare	Secondary malignancy more often arises in this setting

What happens at skeletal maturity?

Growth usually stops

Osteochondromas commonly cease enlargement with growth-plate closure. This temporal pattern helps interpret subsequent enlargement.

When is simple excision relevant?

- Symptomatic lesions are generally cured by simple excision.
- Clinical effects can include neural impingement or stalk fracture.
- Management follows symptoms and anatomy; The lecture does not prescribe a universal surgical threshold.

Secondary chondrosarcoma: Key risk context

- Secondary chondrosarcoma is a rare complication.
- It usually occurs in association with multiple hereditary exostoses.
- Do not treat all benign cellularity or atypia as malignant transformation.

Differential: Surface versus medullary chondroma

Osteochondroma	Enchondroma
Surface bone projection with cartilage cap	Cartilage nodule within medullary cavity
Cortical and marrow continuity into outgrowth	Circumscribed intramedullary lucent lesion
Usually metaphysis of long bone; Knee common	Often small tubular bones of hands / feet

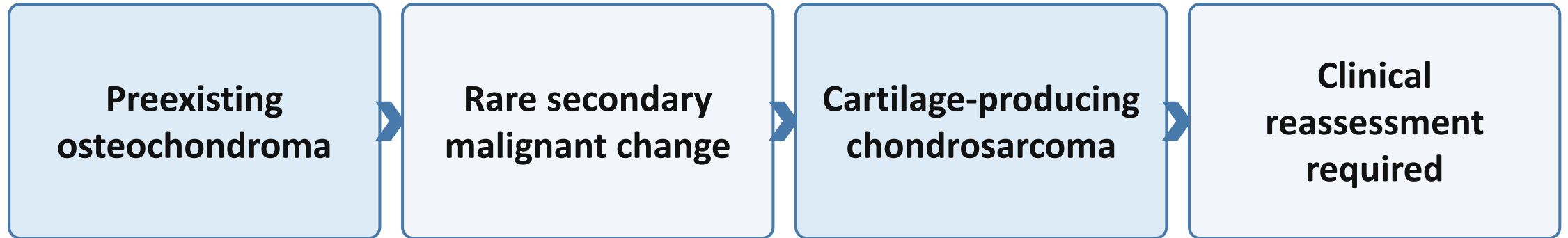
Differential: Juxtacortical chondroma

Osteochondroma	Juxtacortical chondroma
Surface growth has cortical–medullary continuity	Cartilage tumor located on bone surface
Contains bone and cartilage cap	Classified as a chondroma rather than a cartilage-capped exostosis
Growth-plate association typical	Not characterized by the osteochondroma continuity pattern

Chondrosarcoma: What is different?

Typical osteochondroma	Conventional chondrosarcoma
Benign cartilage cap on bone-surface projection	Malignant cartilage-producing tumor
Growth usually stops with skeletal maturity	May produce painful progressively enlarging mass
Cortex and medulla continuous with host bone	Aggressive tumors can destroy cortex and form soft-tissue mass

Chondrosarcoma arising in exostosis



Schematic sequence

EXT genes across benign and malignant lesions

Hereditary osteochondroma	Associated secondary chondrosarcoma
EXT1 / EXT2 disruption is fundamental to syndrome	Chondrosarcomas in this syndrome can harbor EXT1 / EXT2 variants
Growth-plate clonal outgrowth	Malignancy cannot be inferred from EXT status alone
Usually benign behavior	Requires morphology and clinical–radiologic correlation

Integrated diagnosis: Information to combine

Clinical

Patient age; Single versus multiple lesions; Symptoms; Growth course.

Radiologic

Surface location and cortical–medullary continuity.

Morphologic

Cartilage cap with perichondrium and underlying endochondral bone.

Diagnostic exercise: Simple stalked lesion

A late adolescent has a slowly enlarging metaphyseal projection near the knee. Imaging shows uninterrupted cortex and medulla extending into its stalk.

- What diagnosis is best supported?
- Which structural feature is decisive?

Case interpretation: Typical osteochondroma

- Most consistent diagnosis: Osteochondroma.
- The lesion is surface-based with cortex and marrow continuity.
- The location and stage of skeletal growth support the interpretation.

Diagnostic exercise: Multiple deforming lesions

A child has several exostoses of long bones with bowing and shortening. The lesions have cartilage caps and arise near metaphyses.

- Which syndrome is most likely?
- Which genes and mechanism are involved?

Case interpretation: Hereditary exostoses

- The pattern is compatible with hereditary multiple exostoses.
- The inherited predisposition involves EXT1 or EXT2.
- Subsequent loss of the wild-type allele in growth-plate chondrocytes is implicated.

Diagnostic exercise: Pain after a fall

A young person with a pedunculated osteochondroma develops focal pain following minor trauma. The lesion was previously asymptomatic.

- What mechanical complication should be considered?
- Does pain automatically imply malignant change?

Case interpretation: Fracture of the stalk

- A stalk fracture is a recognized cause of pain.
- Pain alone cannot establish malignant transformation.
- Clinical and radiologic correlation must distinguish local injury from a new growth pattern.

Diagnostic exercise: Adult enlargement

An adult with a longstanding hereditary exostosis reports that the mass is growing again after skeletal maturity.

- Why is this history important?
- Which complication deserves consideration?

Case interpretation: Re-evaluate growth

- Osteochondromas usually stop growing at skeletal maturity.
- New growth in an adult warrants reassessment.
- Rare secondary chondrosarcoma should be included in that evaluation, especially in hereditary disease.

Three discriminators that carry the diagnosis

Surface anatomy

Cartilage-capped outward projection.

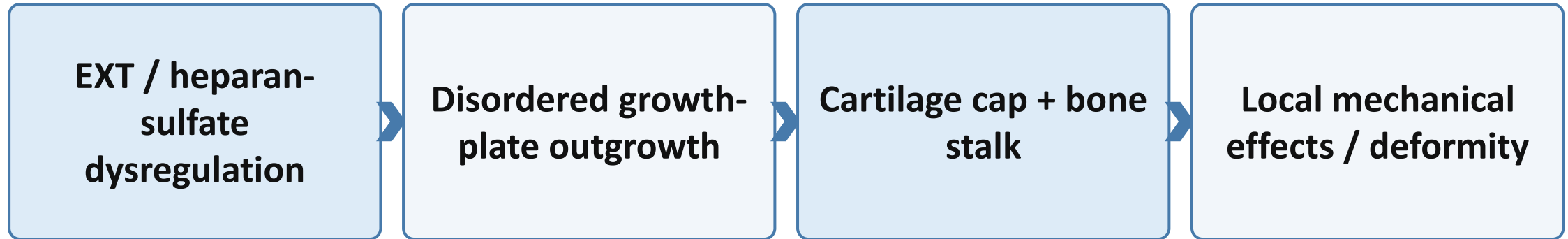
Internal continuity

Cortex and medulla continuous with parent bone.

Developmental behavior

Growth-plate association and usual cessation at maturity.

Final synthesis: Mechanism to consequence



Schematic sequence

High-yield review questions

- Which two anatomical continuities define the lesion?
- How do EXT1 / EXT2 alterations affect the growth plate?
- How does hereditary disease differ from sporadic presentation?
- What explains pain and which complication is rare but important?

References and source scope

Primary teaching source

- Robbins, Cotran & Kumar: Pathologic Basis of Disease, 11th ed. (2026).
- Chapter 26, “Bones, Joints, and Soft Tissue Tumors,” pp. 1080–1083.
- Osteochondroma section, including Fig. 26.21 and morphology; adjacent chondroma and chondrosarcoma for comparison.

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