

BONE AND JOINT INFECTION

Dr. Jónás Zoltán

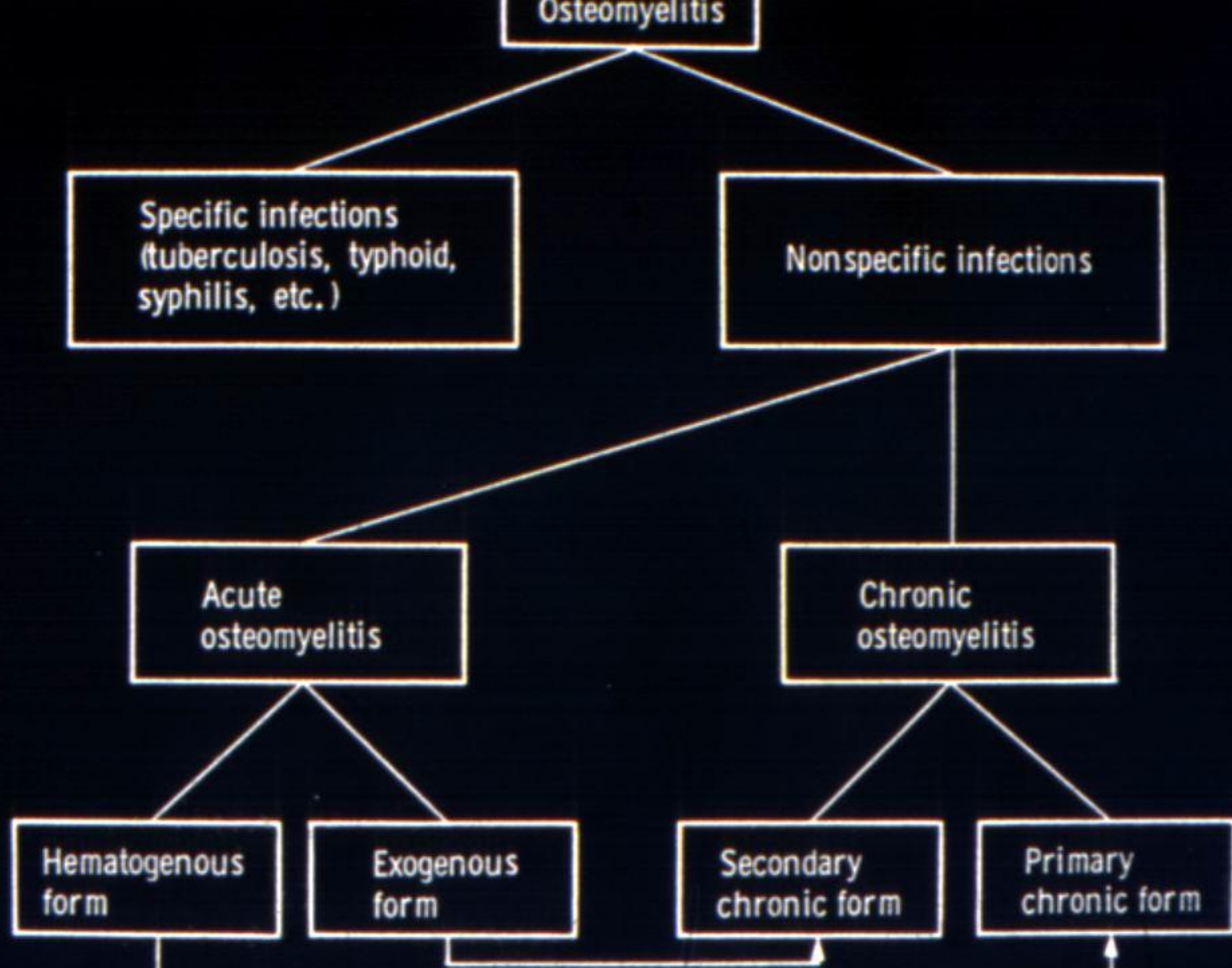
Dept. of Orthopaedics

www.ortopedia.dote.hu

- Order of verbal exams:
The students are able to register for the exam on the Neptun system. The students pick two titles, from the title list available at the beginning of the Semester. This list can be also found on the web site of the Orthopaedic Department.
Those students who attended at least 70 % of the lectures will pick two titles, but will only be examined on the one of their choice.
In case of a B or C exam the student is not entitled to the above advantage.

TERMINOLOGY

- INFECTION OF THE BONE: OSTEOMYELITIS
- INFECTION OF THE JOINT:
SEPTIC ARTHRITIS



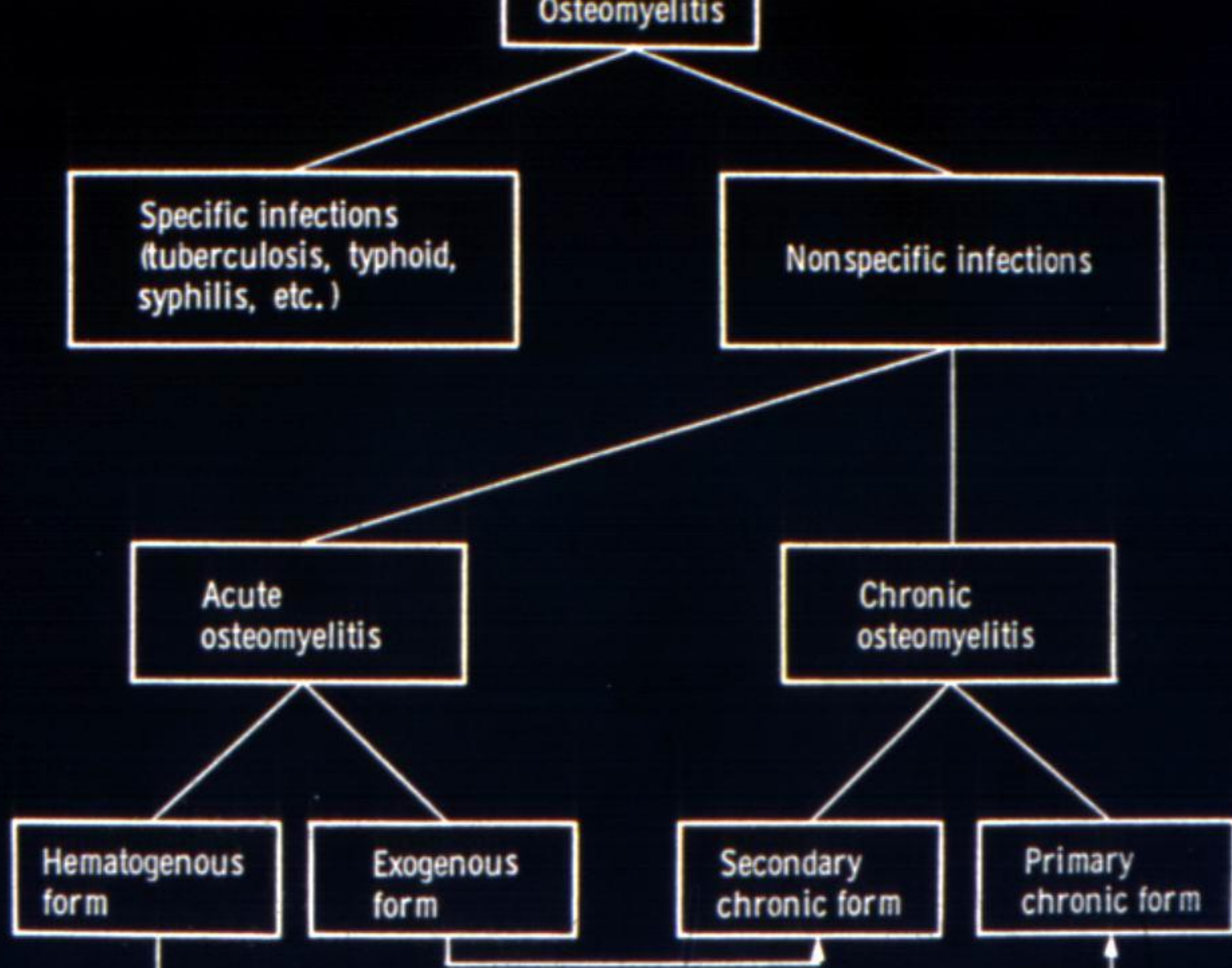
Outcome of bone infection

depends on two factors:

1. Virulence of bacteria
2. Resistance (defensive ability) of the host

If virulence is high or resistance is low-**ACUTE**.

If virulence is low or resistance is high-**CHRONIC**.



Osteomyelitis

Specific infections
(tuberculosis, typhoid,
syphilis, etc.)

Non-specific infections

Acute
osteomyelitis

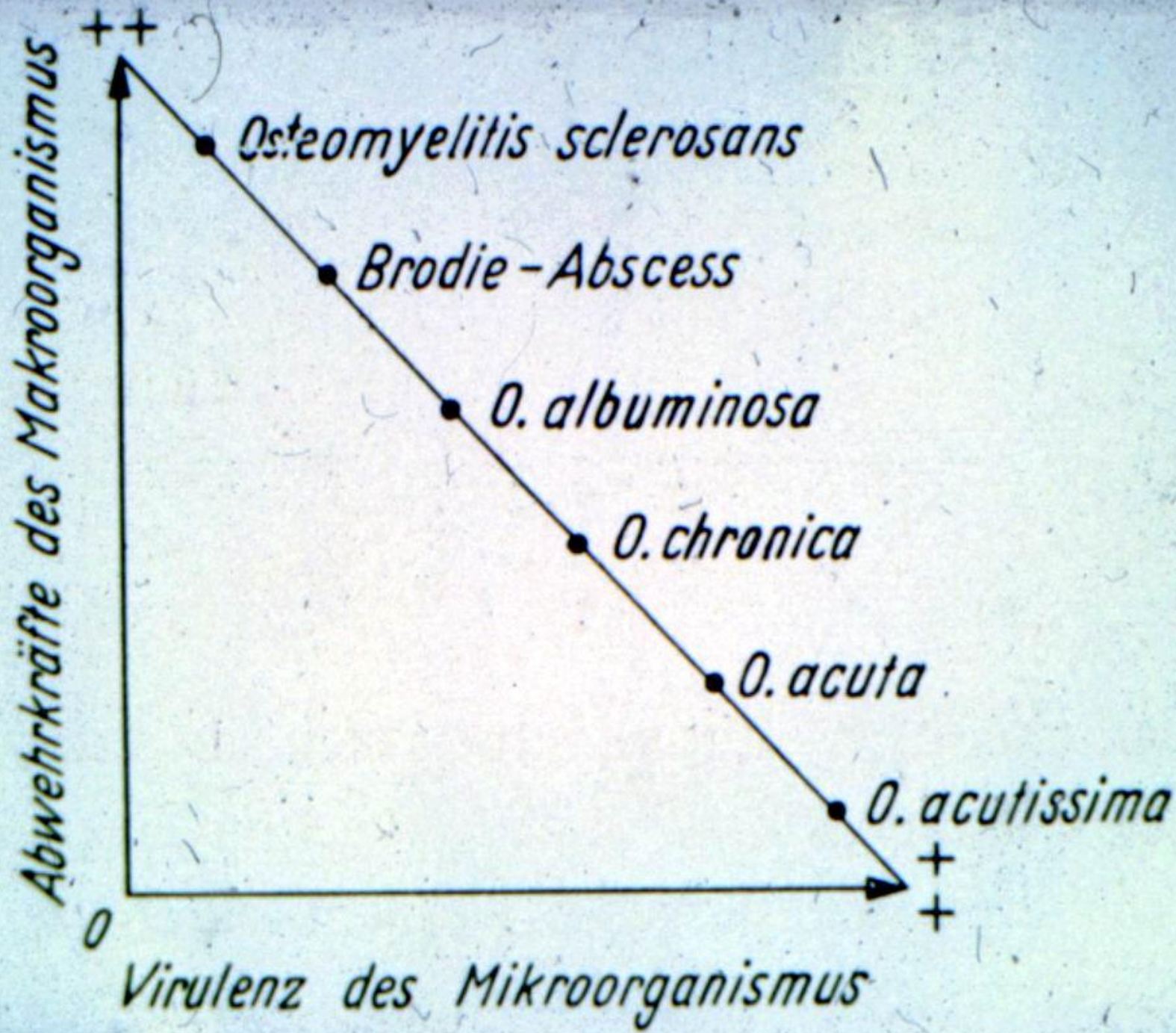
Chronic
osteomyelitis

Hematogenous
form

Exogenous
form

Secondary
chronic form

Primary
chronic form



ACUTE HAEMATOGENOUS OSTEOMYELITIS

- Most important form.(Model for other forms of bone infections).
- Bacteria reach the bones from a distant infected site of the body via the blood stream.
- Mainly children are affected.
- If adults - their resistance are lowered
(diabetes,immunosuppression,organ transplantation,systemic steroid medication,AIDS,etc.)

ACUTE HAEMATogenous OSTEOmyELITIS

ETIOLOGY

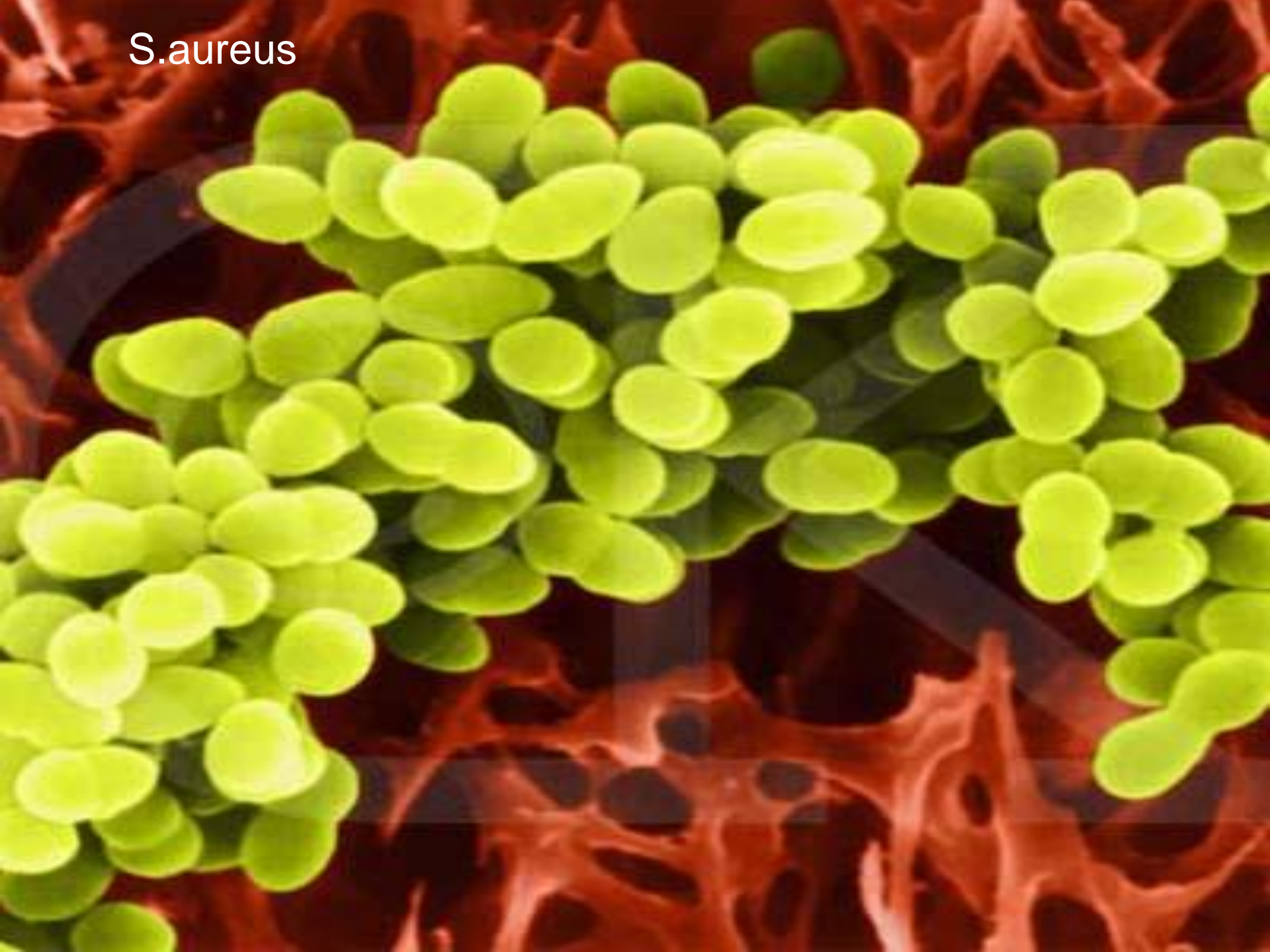
Most frequent causative organisms:

(staphylococci, (mainly *S.aureus*), streptococci (young children), pneumococci, *H.influenzae*.)

FOCI (distant infected site):

Otitis, infected umbilical cord, tonsillitis, urinary tract, pyoderma

S.aureus



S.aureus



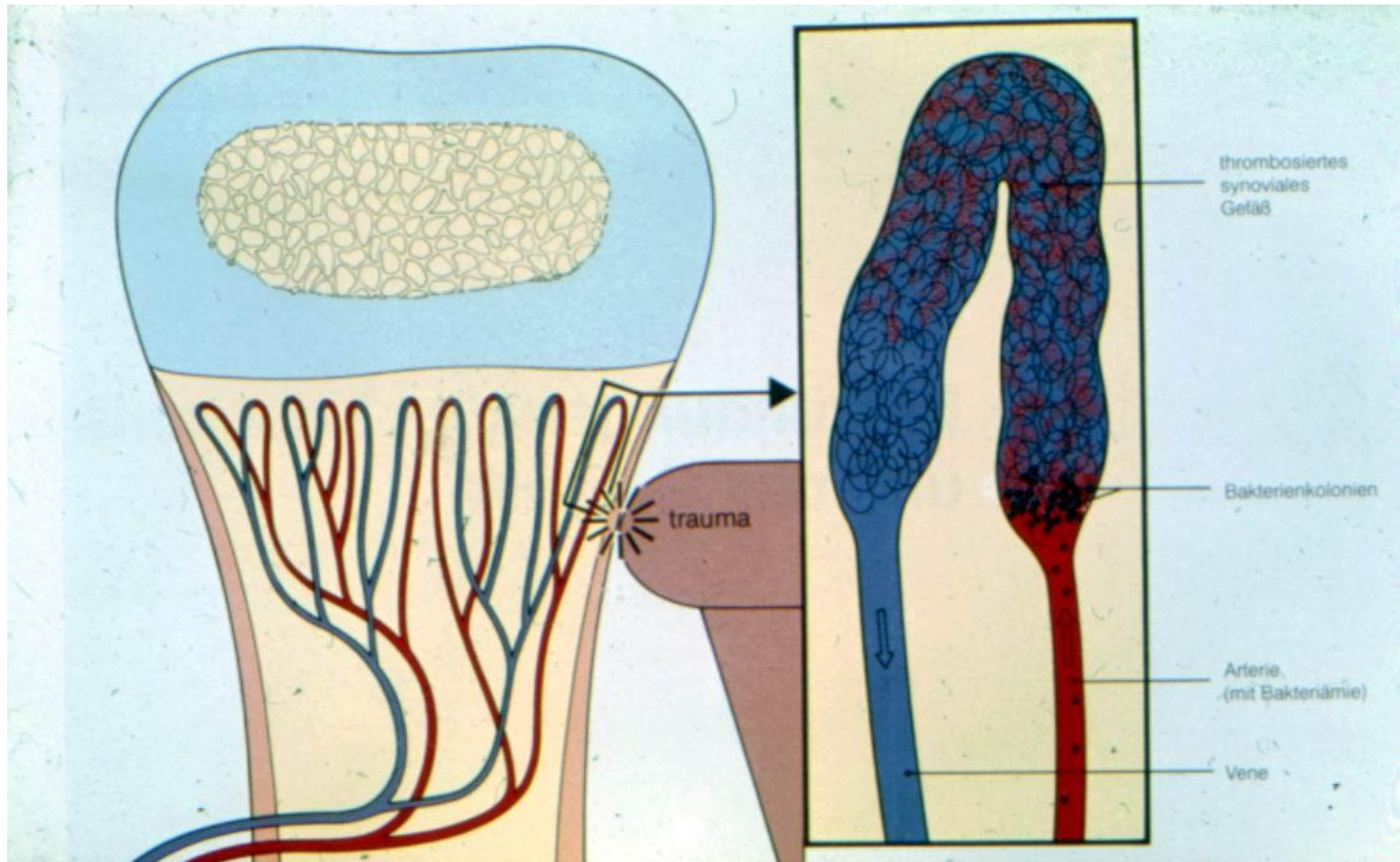
STEPS OF BONE INFECTION

- **I.** Blood stream infection and bacterial settlement in the bone
- **II.**
 - inflammation** (colonization) -local infective thrombosis, toxins, intraosseal pressure raises rapidly. /1-2 day/
 - suppuration** (pus formation) -subperiosteal abscess formation /3-4 day/
 - necrosis** (bone death)-sequester formation /by 1 week/
 - new bone formation** (involucrum) - periosteal stripping-cambium layer /by 2 week/
 - resolution** (remodelling).

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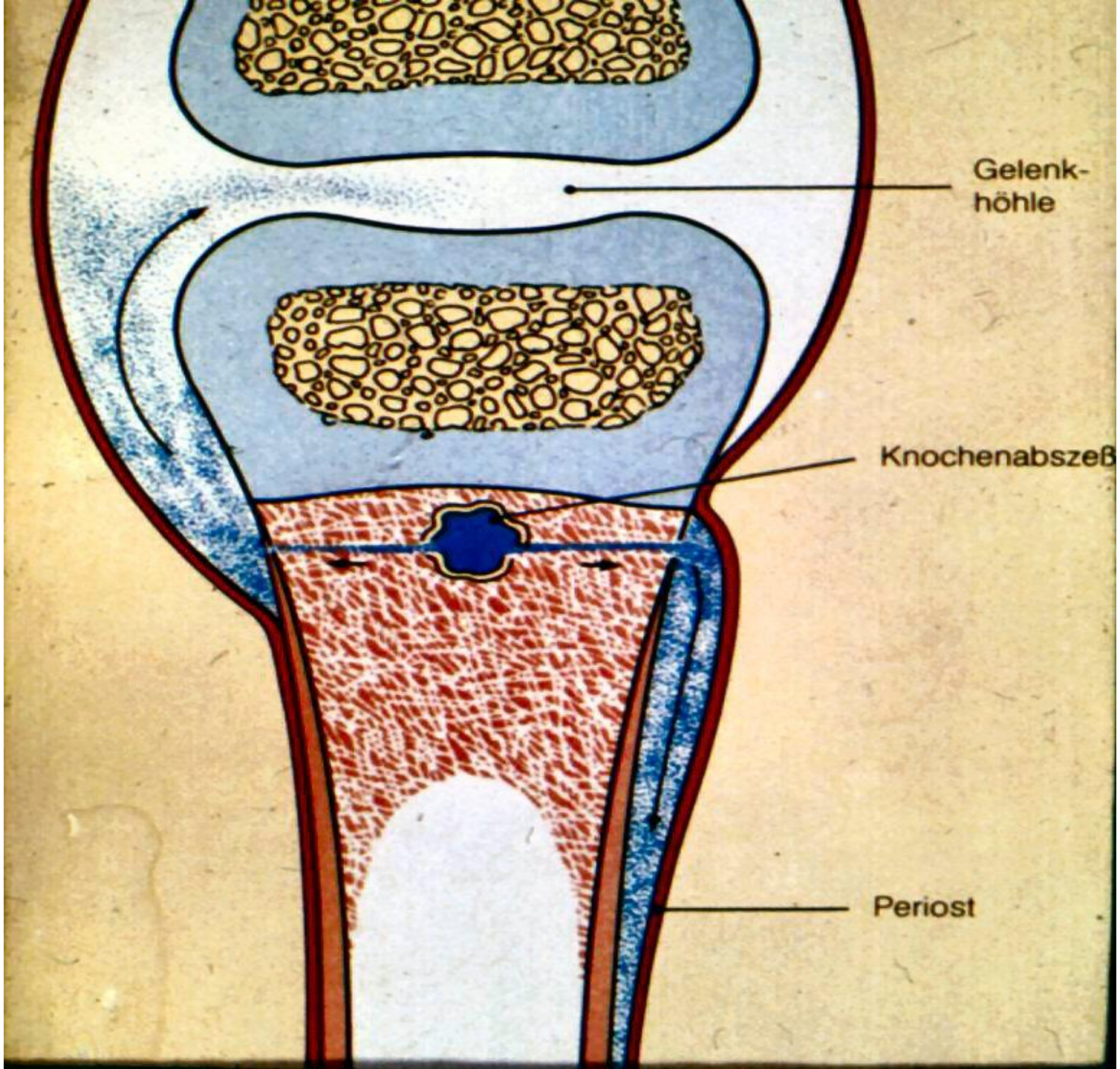
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inflammation(colonization)



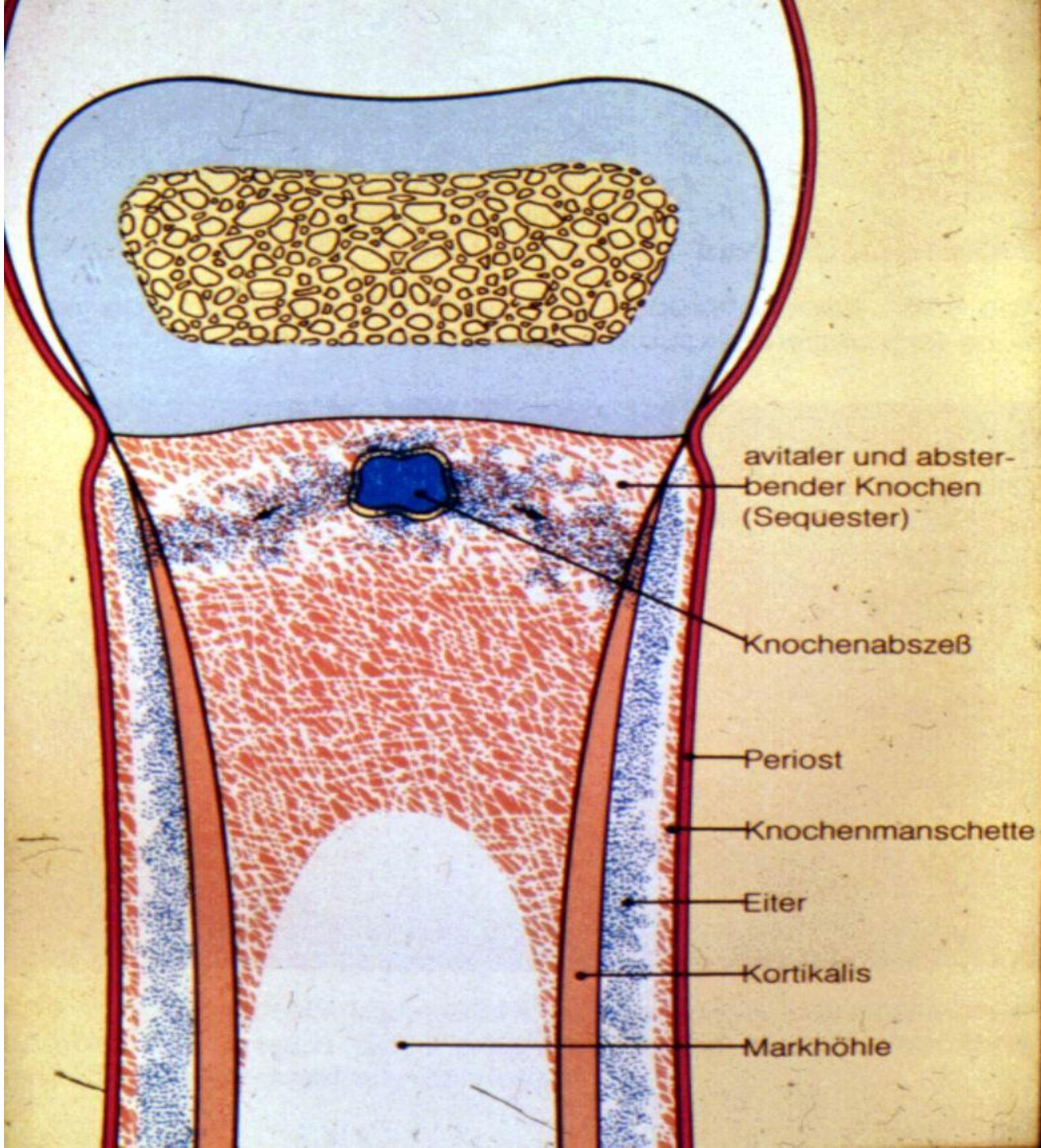
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Orthopaedic infections in children

ACUTE HAEMATOGENOUS OSTEOMYELITIS

Pathology

1. Inflammation (colonization)-bacterial embolization and infected thrombosis

Acute inflammatory reaction with vascular congestion

Rise in intra-osseous pressure causing intense pain

2. Suppuration

At 3-4 days pus forms within the bone and forces its way down the haversian canals to the surface where it forms a sub-periosteal abscess

The pus can spread from here back into the bone, into an adjacent joint or into the soft tissues (Where there is an **intra-articular metaphysis - prox.femur, prox.humerus,prox.radius- joint involvement!!!**)

Vertebral infection can spread through the end plate, disc and into the next vertebral body

3. Necrosis

At 7 days, rising pressure, vascular stasis, infective thrombosis and periosteal stripping and **toxins** compromise the blood supply to the bone resulting in bone death which results in a **sequestrum** .

4. New bone formation

At 10-14 days this forms from the deep surface of the stripped periosteum forming the **involucrum**

5. Resolution

With release of the pressure and appropriate antibiotics healing can occur

There may be permanent deformity

Unpublished work (quoted in Dee) shows that experimentally bacteria injected intravenously will settle in the metaphyses of bone preferentially

NB - in 10% of cases there is more than one site of infection.

Clinical Features

Children (invariably)

Pain, malaise, fever

Limp or not weight bearing

Infants

Failure to thrive, drowsiness, irritable

Adults

The commonest site is the *thoracolumbar spine* (Batson's venous complex from the pelvis)

Other bones involved especially in DM, IVDA, immunosuppressed

Examination

Local erythema, swelling and tenderness indicates that the pus has broken through the periosteum

ACUTE HAEMATOGENOUS OSTEOOMYELITIS- PATHOGENESIS

Localisation: metaphysis

Special vascular pattern (non-anastomosing terminal branches), slow blood flow, growing cells are vulnerable for infection

ACUTE HAEMATOGENOUS OSTEOMYELITIS- CLINICAL FEATURES

Rapid onset

Generally bad condition

Fever

GENERAL SIGNS OF INFECTION:

Pain (dolor)

Redness (rubor)

Swelling (tumor)

Warmth (calor)

Restricted movements (functio laesa)

BONE and JOINT INFECTIONS- laboratory investigations

- ESR- (100 mm/h)
- CRP- elevated
- Blood culture
- Aspiration (gram stain,culture, antibiogram)

ACUTE HAEMATOGENOUS OSTEOMYELITIS- IMAGING

PLAIN X-RAY

Periosteal new bone (10-14 days postinfection, too late)

RADIOISOTOPE UPTAKE

Very sensitive but not specific

MRI

ULTRASOUND

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sequester



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ULTRASOUND



STEPS OF BONE INFECTION- TREATMENT

- **I.** Blood stream infection (**bacteraemia**) and bacterial settlement in the bone
ANTIBIOTICS
- **II.**
inflammation (colonization) -local infective thrombosis,intraosseal pressure raises rapidly /1-2 day/
DRILLING
suppuration (pus formation) -subperiosteal abscess formation /3-4 day/
EVACUATION and DRILLING
necrosis (bone death) -sequester formation /by 1 week/
CURETTAGE and SEQUESTRECTOMY
new bone formation (involucrum)-periosteal stripping -cambium layer /by 2 week/
CURETTAGE and SEQUESTRECTOMY
resolution (remodelling)

ACUTE HAEMATOGENOUS OSTEOOMYELITIS- COMPLICATIONS

METASTATIC INFECTION

SUPPURATIVE ARTHRITIS

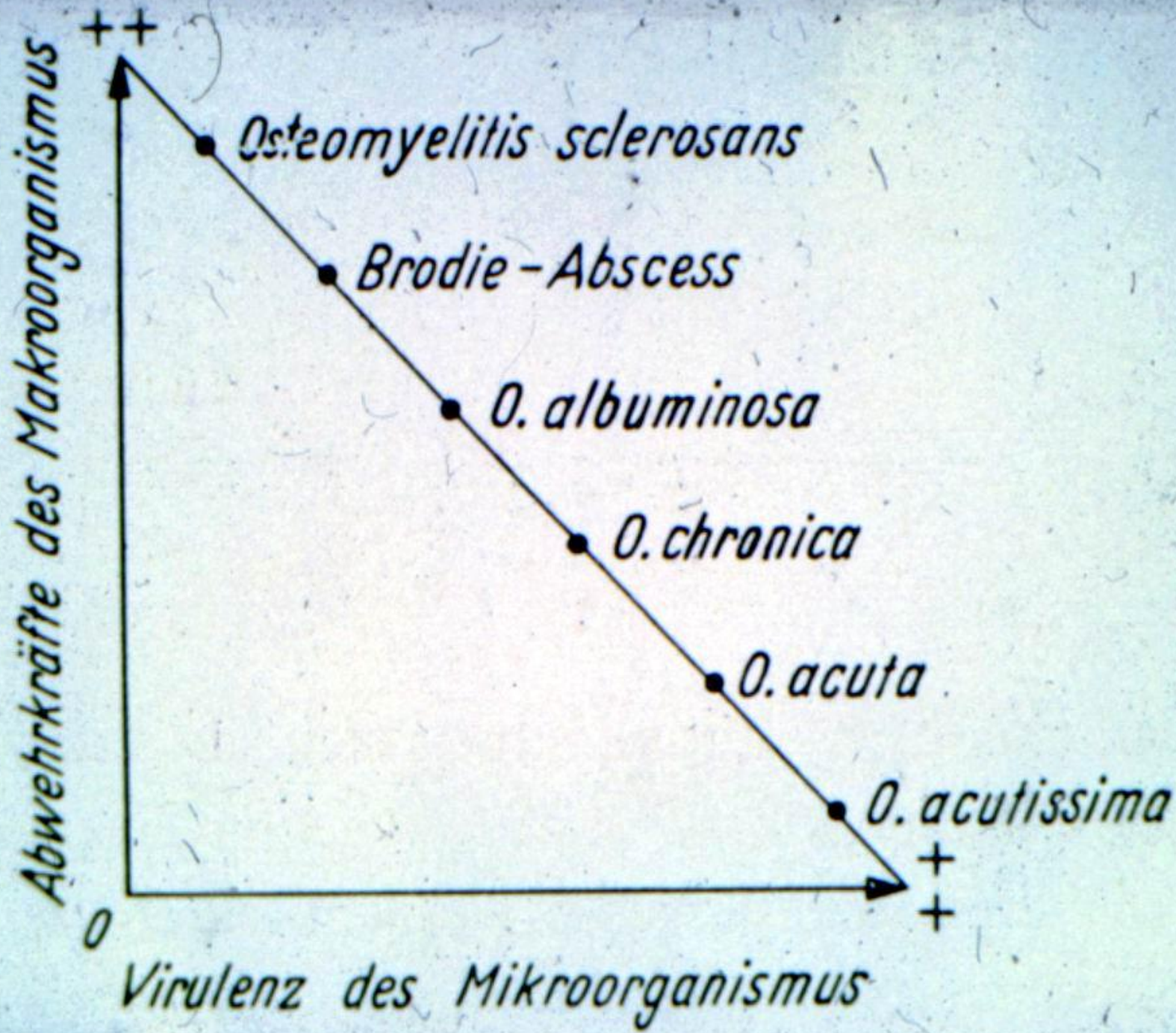
GROWTH DISTURBANCES (altered length, axial
deformities)

CHRONIC OSTEOOMYELITIS

LETHAL OUTCOME

SUBACUTE FORMS

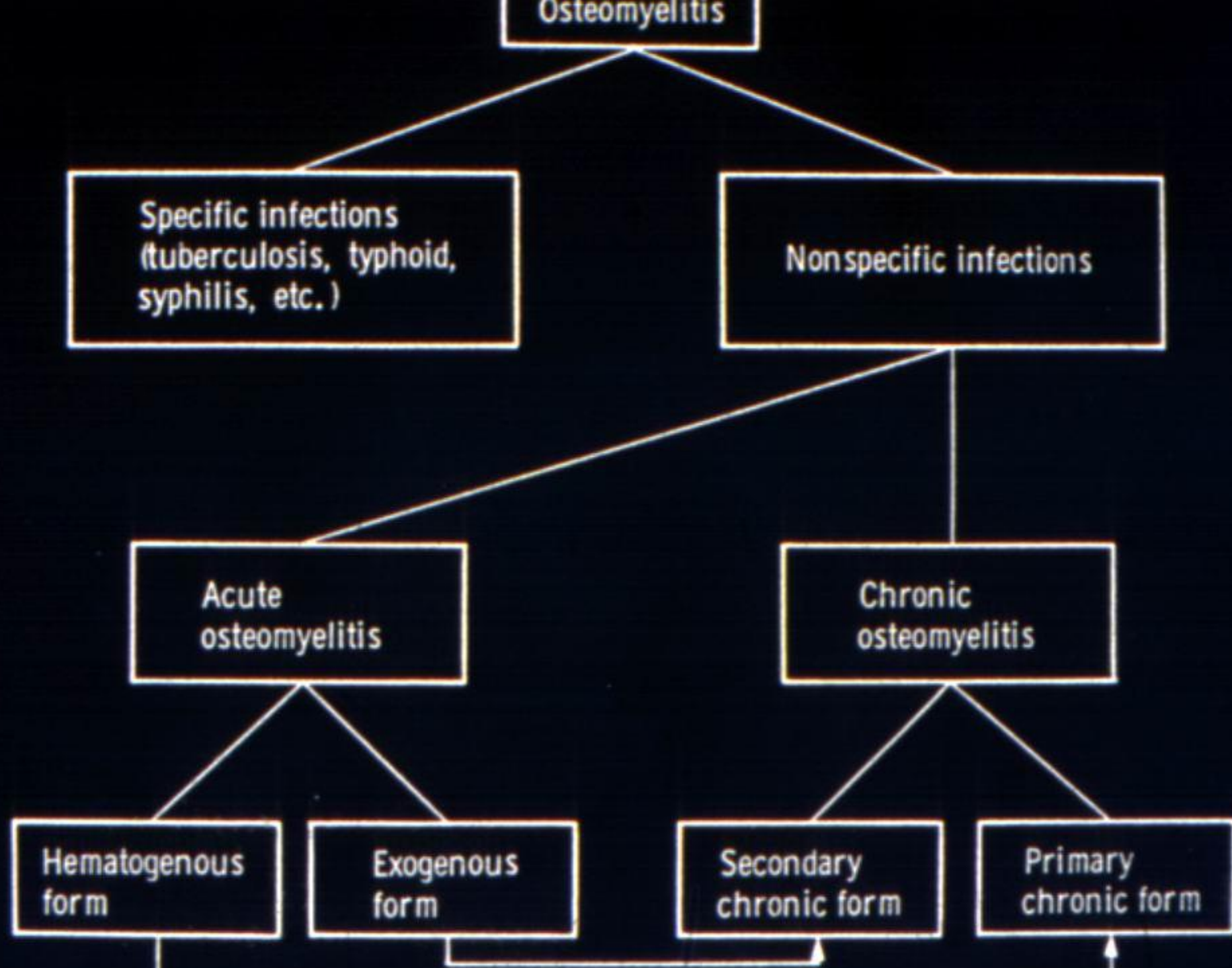
- Virulence of bacteria is not high.
- Defensive ability of host is good.
- X-ray: well-defined laesion surrounded by sclerotic rim.
- Means differential diagnostic problems.
- Needs no surgery (if dg.is sure), antibiotic treatment alone is sufficient.



BRODIE ABSCESS



CHRONIC FORMS



Osteomyelitis

Specific infections
(tuberculosis, typhoid,
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Non-specific infections

Acute
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Secondary
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Outcome of bone infection

depends on two factors:

1. Virulence of bacteria
2. Resistance (defensive ability) of the host

If virulence is high or resistance is low-**ACUTE**.

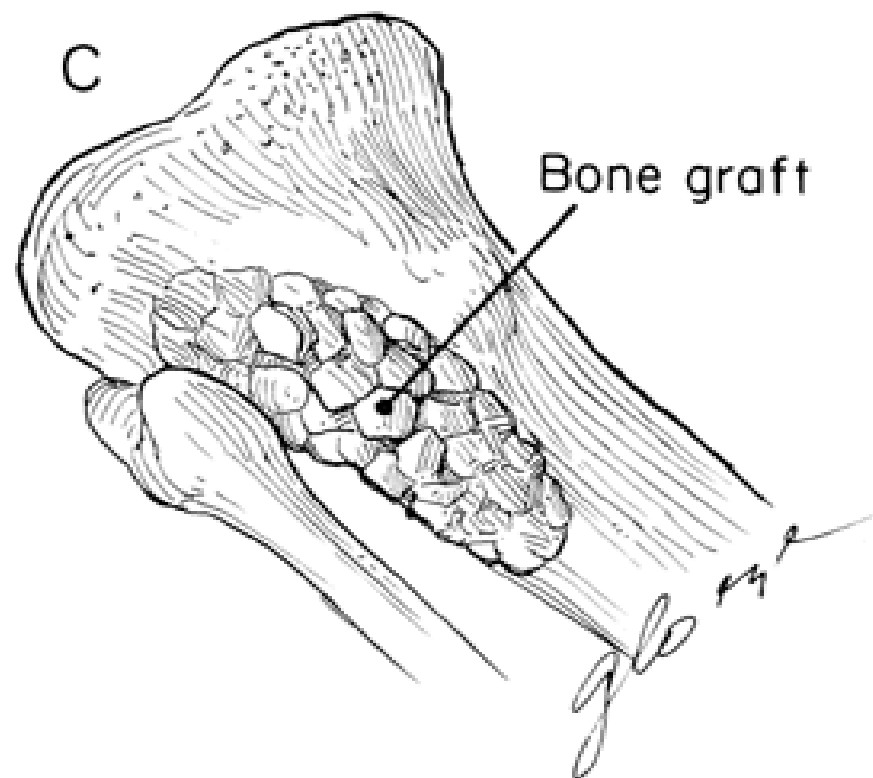
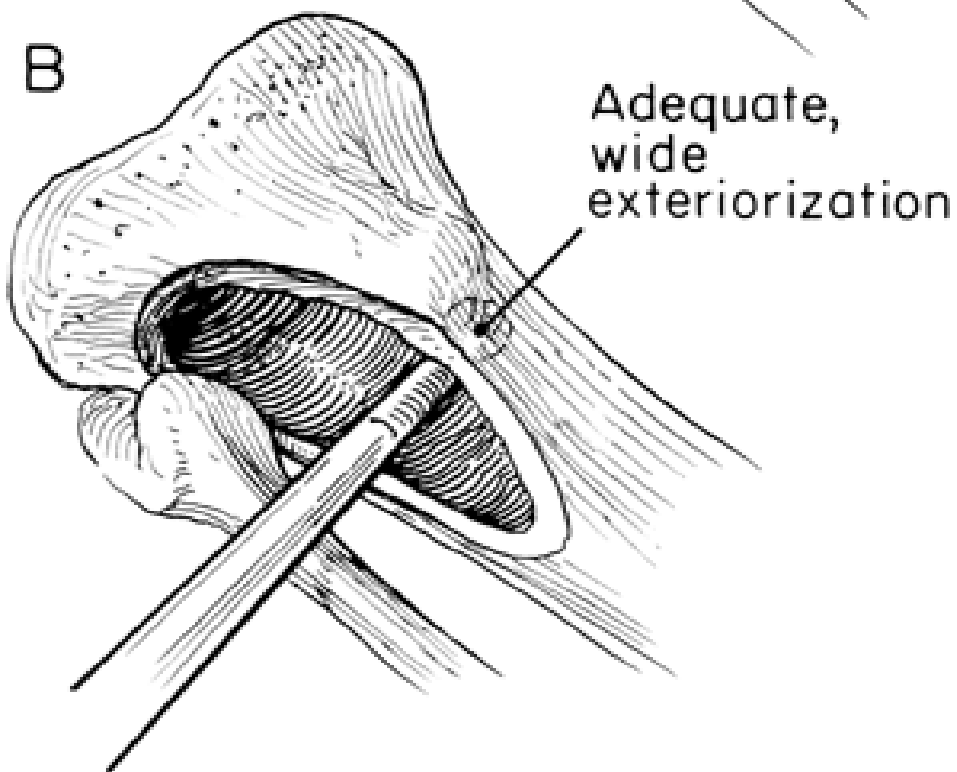
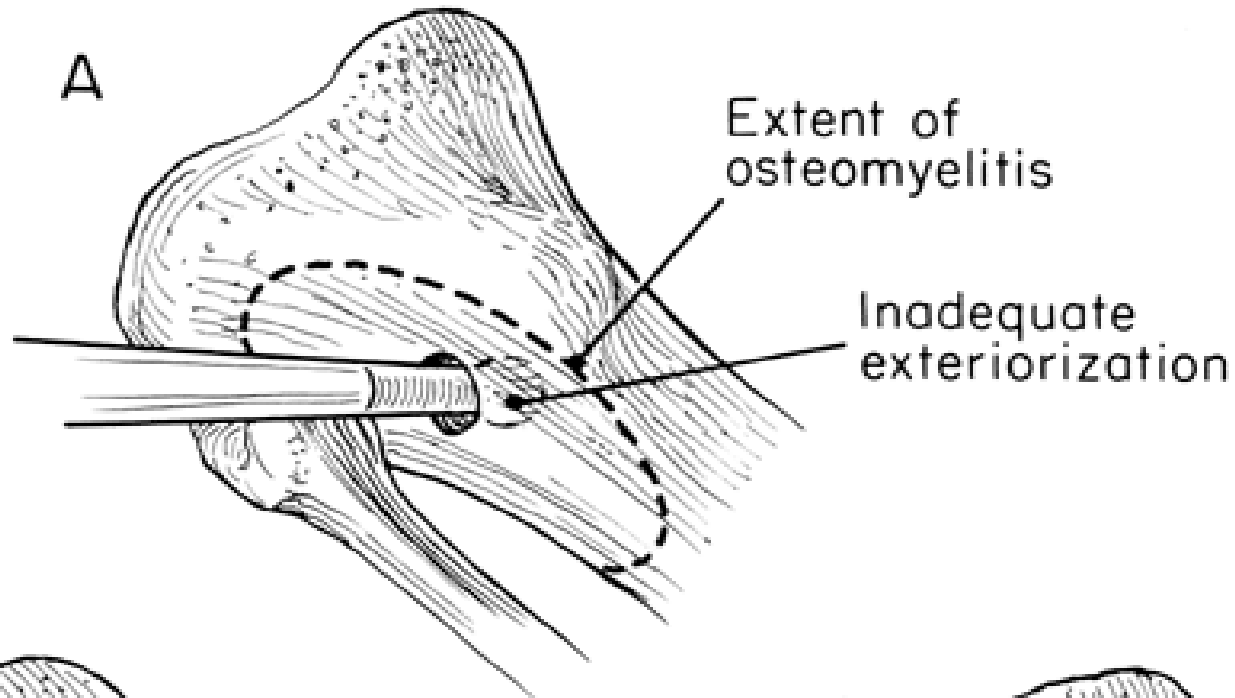
If virulence is low or resistance is high-**CHRONIC**.

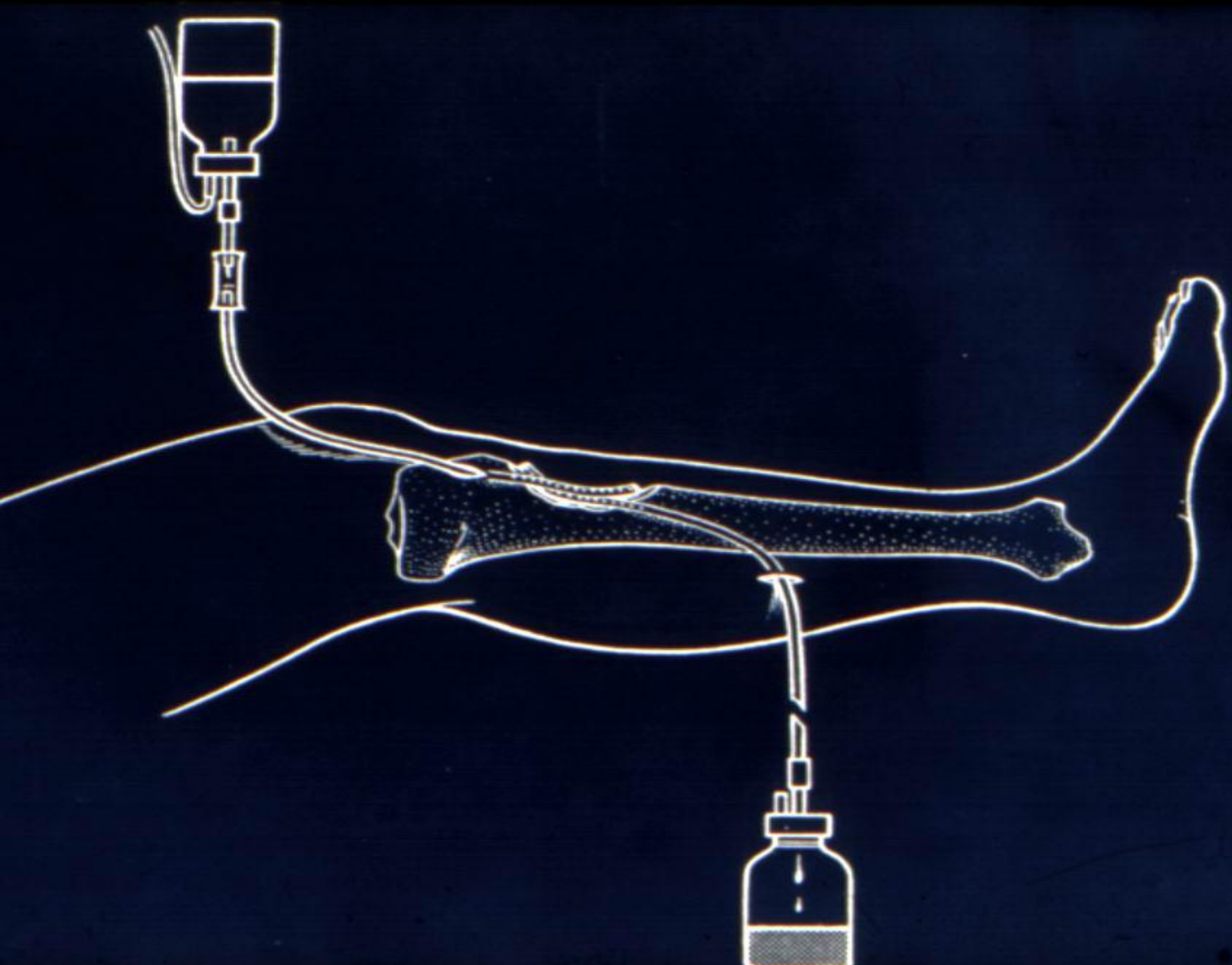




Treatment of chronic osteomyelitis

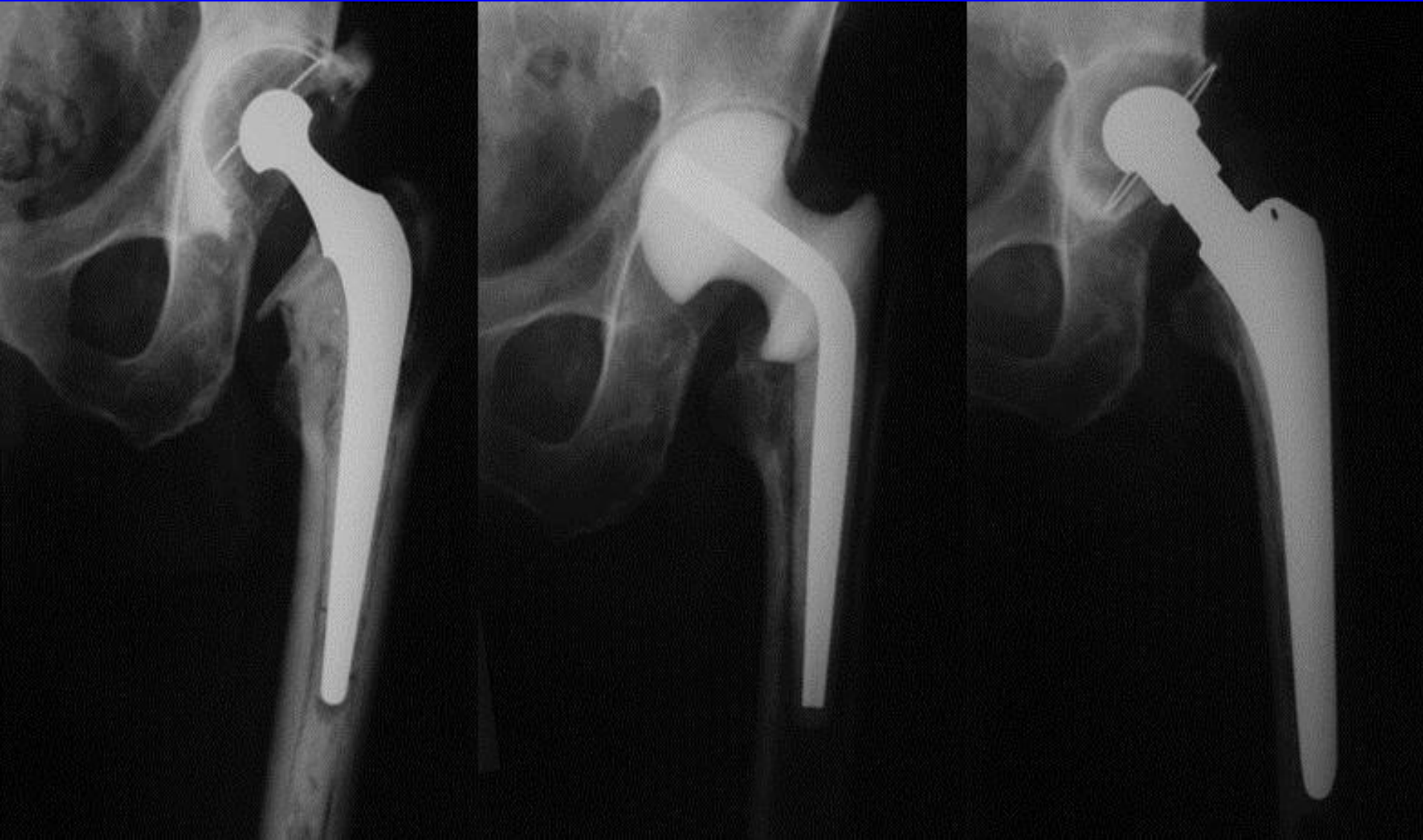
Almost exclusively surgical :
radical debridement
(mechanical removal of infected tissues)







Antibiotic-loaded spacer



Modell "szított gyári" spacer



SEPTIC ARTHRITIS

Route of spread

- Haematogenous (infected umbilical cord, otitis, tonsillitis, etc.).
- Spread from metaphyseal osteomyelitis where the metaphysis is intra-articular (prox. femur, prox. humerus, prox. radius).
- Spread from contiguous soft tissue infection .
- Direct inoculation .
- Spread through the growth plate (infants!)

SEPTIC ARTHRITIS

ETIOLOGY

Most frequent causative organisms:

(staphylococci, streptococci (young children), pneumococci,
E.coli

Bacteria settle down in the synovial membrane.

Cartilage and bone also can be affected.

SEPTIC ARTHRITIS- CLINICAL FEATURES

Rapid onset

Fever

GENERAL SIGNS OF INFECTION:

Pain (dolor)

Redness (rubor)

Swelling, fluctuation (tumor)

Warmth (calor)

Restricted movements (functio laesa)

SEPTIC ARTHRITIS- LABORATORY FEATURES

- ESR- (100 mm/h)
- CRP- elevated
- Blood culture
- Aspiration (gram stain,culture, antibiogram)

SEPTIC ARTHRITIS- TREATMENT

ANTIBIOTICS, ANALGESICS, SPLINTAGE

ASPIRATION (repeated)

DRAINAGE

CONTINUOUS SUCTION-IRRIGATION

SYNOVECTOMY

CORRECTIVE OSTEOTOMY

ARTHRODESIS