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CLINICAL PRACTICE GUIDELINES

ផ្នែក

FOR

សល្យសាស្ត្រ

SURGERY

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នាយកដ្ឋានសេវាសុខភាព

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Kingdom of Cambodia
Nation Religion King

CLINICAL PRACTICE GUIDELINES

FOR

SURGERY

Part *2*

Department of Health Services

December 2025

Ministry of Health

CLINICAL PRACTICEGUIDELINES FOR

SURGERY

Part

2

December 2025

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CLEFT LIP AND PALATE

Kao Chinphallin, Mam Sambo Vithya, Long Vanna, OU Cheng Ngiep

I. DEFINITION

Cleft lip and cleft palate are openings or splits in the upper lip, the roof of the mouth (palate) or both. Cleft lip and cleft palate result when facial structures that are developing in an embryon don't close completely.

The malformation can simply be divided into cleft lip (CL), cleft palate (CP), and cleft lip and palate (CLP); however, the surgical treatment plan requires a more complex classification scheme. The cleft lip deformity is also divided into unilateral or bilateral, and then subdivided into complete, incomplete, or microform.

Unilateral cleft lip

Unilateral cleft lip and palate *and* bilateral cleft lip and palate

The incidence of cleft lip and/or palate is approximately 1 in 700 live births. This incidence varies widely depending on geographic origin, racial and ethnic group, environmental exposures, and socioeconomic status.

In the Caucasian population, cleft lip with or without cleft palate occurs in approximately 1 in 1,000 live births. These entities are twice as common in the Asian population (about 2/1000 : Japanese 2.1/1000, Chinese 1,7/1000), and approximately half as common in African Americans (0.3- 0.4/1000).

II. ETIOLOGY

Both environmental teratogens and genetic factors are implicated in the genesis of cleft lip and palate; representing an interaction between genetics and environment during a critical stage of development.

The teratogens, such as alcohol, smoking, anticonvulsants (phenytoin and valproic acid), corticosteroids, and retinoic acid, as well as folic acid deficiency during the periconceptional period increase the incidence of cleft lip.

Recently several genes causing CL and palate have been discovered. The nature and function of these genes vary widely, illustrating high complexity within the craniofacial developmental pathways.

If the family has one affected child or parent with CLP, the risk of the child of the next pregnancy having CLP is 4%. If two previous children have CLP, the risk increases to 9%, and if one parent and one child were previously affected, the risk to children of subsequent pregnancies is 17%.

III. DIAGNOSTIC PROCEDURE

A cleft lip or cleft palate is easy to diagnose.

Currently an ultrasound examination during the 20 weeks of pregnancy can be performed to exclude an oral cleft of the face. Evaluation of the upper lip for possible CL/P is an optional element and has a sensitivity of 88% for detecting CL/P. However, the sensitivity for the prenatal detection rate of CP is only 0%-1.4%.

If the cleft has not been detected in an ultrasound prior to the baby's birth, a physical exam of the mouth, nose, and palate confirms the presence of cleft lip or cleft palate after a child's birth. Usually, a cleft in the lip or palate is immediately identifiable at birth. Cleft lip and cleft palate may appear as:

- A split in the lip and roof of the mouth (palate) that can affect one or both sides of the face.
- A split in the lip that can appear as only a small notch in the lip or can extend from the lip through the upper gum and palate into the bottom of the nose.
- A split in the roof of the mouth that doesn't affect the appearance of the face.

Less commonly, a cleft occurs only in the muscles of the soft palate (submucosal cleft palate), which are at the back of the mouth and covered by the mouth's lining. This type of cleft often goes un noticed at birth and may not be diagnosed until later when signs develop. Signs and symptoms of submucosal cleft palate may include:

- Difficulty with feedings, swallowing, with potential for liquids or foods to
- come out the nose.
- Nasal speaking voice.
- Chronic ear infections.

IV. COMPLICATIONS

They usually include not only cosmetic and dental abnormalities, but also speech, hearing and facial growth difficulties. The emotional, psychological impact and impact on quality of life for the child and the family can be severe and must be recognized.

- Difficulty feeding: One of the most immediate concerns after birth is feeding. While most babies with cleft lip can breast-feed, a cleft palate may make sucking difficult. Nasal reflux is irritating to the nasal mucosa and can predispose to sinusitis and ulceration. Weight gain and skeletal growth confirm success of the feeding regimen.
- Ear infections and hearing loss: Babies with cleft palate are especially at risk of developing middle ear fluid and hearing loss. Estimates are 20–30% incidence of pure tone hearing loss in cleft patients by audiography. Untreated children with clefts and severe effusions may have total deafness.
- Dental problems: If the cleft extends through the upper gum, tooth development may be affected.
- Speech difficulties: Because the palate is used in forming sounds, the development of normal speech can be affected by a cleft palate. Speech may sound too nasal.
- Growth disturbances: cleft infants have been shown to exhibit poor weight gain in early infancy. In later childhood, average weight and height of children with cleft appear to diminish compared with those of control subjects.
- Challenges of coping with a medical condition: Children with clefts may face social, emotional and behavioural problems due to differences in appearance and the stress of intensive medical care.

V. TREATMENT

Children born with cleft lip and or palate require coordinated care from multiple specialties to optimize treatment outcome. The ideal is in a centre with a multidisciplinary cleft team, dedicated to treating cleft-related issues from birth to adulthood. Typical members of a cleft team include an audiologist, dentist, geneticist, nurse, nutritionist/dietitian, oral surgeon, orthodontist, otolaryngologist, paediatrician, plastic surgeon, psychologist, social worker, and speech pathologist.

The idea of interdisciplinary care is to coordinate treatment by multiple specialists in a timely fashion with an aim of achieving normality in all aspects, including feeding, breathing, speech, hearing, alignment of teeth, appearance, and overall psychological and physical development. The timing of surgical and non-surgical cleft care is as follow:

Prenatal

Diagnosis and parental counselling.

0–6 months

General assessment for associated anomalies.

ENT evaluation – breathing, feeding, swallowing, and hearing. Presurgical orthopaedics (0–3 months).

Primary lip repair (3–4 months).

6 months – 2 years

Speech and oral sensory motor assessment.

Grommets/ear tubes (as needed).

Primary palate repair (9–12 months).

Preschool: 3–5 years

Dental care.

Speech assessment and therapy (continue as needed). Assess need for lip revision.

Childhood: 6–12 years

Correction of velo-pharyngeal dysfunction (as needed).
Orthodontic treatment – phase I.

Alveolar cleft repair (8–11 years).

Adolescence: 13–18 years

Orthodontic treatment – phase II.

Orthognathic surgery (if needed) – 14–16 years (female), 16–18 years (male).
Revision cheilo-rhinoplasty.

Replacement of missing teeth (as needed).

VI. ALGORITHM

Age	Treatment Cleft	Cleft team members
Prenatal	Prenatal imaging, diagnosis, and counseling	Multidisciplinary
Newborn	Feeding assessment, medical assessment, genetic counseling, treatment information	Multidisciplinary
0–3 months	Presurgical orthopedics	Orthodontist, plastic surgeon
3 months (or after presurgical orthopedics) [a]	Primary cleft lip repair and tip rhinoplasty ± gingivoperiosteoplasty	Plastic surgeon
12 months (delayed if airway or medical concerns) [a]	Primary cleft palate repair with intravelar veloplasty ± bilateral myringotomy and tubes	Plastic surgeon, otolaryngologist
Diagnosis of velopharyngeal insufficiency (3–4 years)	Secondary palate lengthening or pharyngoplasty, speech obturator	Speech pathologist, plastic surgeon, otolaryngologist, orthodontist
School-age years	Treatment of secondary lip and nasal deformities	Plastic surgeon
7–9 years (mixed dentition) [b]	Secondary alveolar bone graft	Orthodontist, plastic surgeon, oral surgeon
Postalveolar graft [a]	Presurgical orthodontics	Orthodontist
Puberty	Definitive open rhinoplasty	Plastic surgeon
Skeletal maturity	LeFort I ± mandible orthognathic surgery	Plastic surgeon, oral surgeon

a: Essential treatments of cleft lip and palate deformity.

b: Required if gingivoperiosteoplasty is not done or is unsuccessful.

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CLUBFOOT

Pium Pisey, Sok Youlay, Heng Sophea, Ou Cheng Ngiep

I. DEFINITION

Clubfoot, congenital talipes equinovarus, is one of the most common congenital anomalies. Babies are born with an inward-facing foot, fixed varus and equinus of the hind foot, high medial longitudinal arch (cavus) and abduction of the forefoot (metatarsus abductus) (Figure 1).

Without treatment, clubfoot will become permanent stiff deformity, with pain and walking difficulty, and physical & social consideration like school, marriage, job etc.

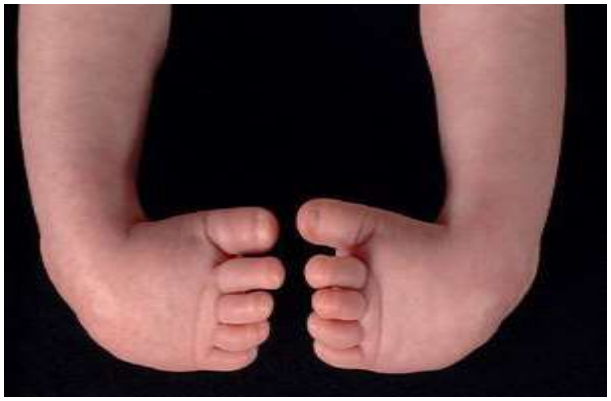


Figure 1. Typical posture for congenital talipes equinovarus.

Incidence of clubfoot

- The global pooled prevalence of clubfoot was 1.18 per 1000 births
- Cambodia 2021: prevalence: 1-1.21/1000 live birth
- About 50 percent affected both feet
- Sex ratio M:F= 2:1

II. ETIOLOGY

2.1 Pathophysiology

- muscle contractures contribute to the characteristic deformity that includes (CAVE)
  - Cavus (tight intrinsics, FHL, FDL)
  - Adductus of forefoot (tight tibialis posterior)
  - Varus (tight tendo-Achilles, tibialis posterior, tibialis anterior)
  - Equinus (tight tendo-Achilles)

- bony deformity consists of medial spin of the midfoot and forefoot relative to the hindfoot

2.2 Genetic

- genetic component is strongly suggested
- familial occurrence in 25%

2.3 Associated condition

- arthrogryposis
- diastrophic dysplasia
- myelodysplasia etc.

III. DIAGNOSIS OF CLUBFOOT

3.1 Physical examination

- characteristic deformity:
  - hindfoot in equinus and varus
  - midfoot in cavus
  - forefoot in adduction
- small foot and calf
- shortened tibia
- medial and posterior foot skin creases

3.2 Imaging

- ➢ Radiographs
  - often not taken
  - if taken, recommended views: dorsiflexion lateral and AP views.
- ➢ Ultrasound: clubfoot can be diagnosed during a pregnancy at 13 to 24 weeks.

3.3 Classification

- Positional Clubfoot: flexible foot held over time in an abnormal position in utero.
- Idiopathic Clubfoot: unknown cause and not related to any other medical problem.
- Secondary Clubfoot: usually Neurological such as Spina Bifida or Syndromic Disorders such as Arthrogryposis.

Table 1. The Pirani scoring system: used to evaluate the severity of deformity and progression		
Midfoot (midfoot contracture score)	Medial crease	Score (0, 0.5, 1)
	Curved lateral border	Score (0, 0.5, 1)
	Lateral head of talus	Score (0, 0.5, 1)
Hindfoot (hindfoot contraction score)	Posterior crease	Score (0, 0.5, 1)
	Empty heel	Score (0, 0.5, 1)
	Rigid equinus	Score (0, 0.5, 1)

IV. DIFFERENTIAL DIAGNOSIS

- Tibial agenesis
- Charcot-Marie disease
- Metatarsus adductus
- Congenital vertical talus etc...

V. TREATMENT

5.1 **Nonoperative:**

Consists of 2 phases: corrective phase (serial casting and tenotomy) and maintenance phase (foot abduction bracing).

5.1.1 Ponseti method of serial manipulation and casting

Techniques

- Ponseti method is the gold standard in most part of the world
- The goal is to rotate foot laterally around a fixed talus
- Order of correction (CAVE)
  1. **C**avus
  2. **Adductus**
  3. **Varus**
  4. **Equinus**

The casting is performed using a single layer of padding under quick-setting plaster of Paris. Casts run from above the knee to the exposed toes and are changed every 7 days. Each cast gradually corrects the foot deformity. The first cast lifts the first metatarsal and corrects the cavus deformity. The subsequent casts simultaneously correct the hindfoot varus and metatarsus adductus. This is achieved by abducting the forefoot while maintaining pressure on the lateral side of the talar head. The hindfoot itself is never manipulated but corrects because of the coupling of the movements of the tarsal bones. Once the hindfoot varus and metatarsus are corrected, there is usually residual hindfoot equinus, which can only be adequately treated with an Achilles tenotomy. Cast complications are rare, but include pressure ulcers and cast slips. Parents are taught how to identify a slip and how to remove the cast if this occurs. Onward referral or help should be sought if the child develops repeated pressure ulcers or slips, requires more than six or seven casts or their Pirani score is stalling (table 1).

5.1.2 5.1.2 Achilles tenotomy

Up to 95% of clubfoot cases require an Achilles tenotomy. This is a standard feature of the Ponseti method. The tenotomy is performed once the foot is ready (table2), or once the midfoot contracture score is 0.5 or less. This is usually achieved after three to six casts. The procedure can be performed percutaneously under local anesthetic for most babies under 6 months of age. Using an aseptic technique, the Achilles tendon is divided using a small blade. The procedure takes a few seconds to perform, and the leg is cast for 3 weeks after surgery.

Table 2. Features that suggest clubfoot is ready for tenotomy
Talar head is covered
Heel in neutral or valgus
Anterior process of os calcis has come out from under talus or foot abduction >50%

5.1.3 Foot abduction bracing

Following foot correction by the Ponseti method, the foot is held in an abducted and dorsiflexed position in boots that are connected by a bar. This is known as a foot abduction brace, or ‘boots and bar’ (Figure 2).

- For the first 3 months, it is recommended that the foot abduction brace is worn for 23 hours a day.
- The child will wear the foot abduction brace at night and during naptime, up to age of 5 years.
- The foot abduction brace is fitted and regularly checked in clinic:

- During the first 3-4 months after getting bracing shoe, follow up are usually every month.
- Up to the age of 2 years, checkups are usually performed every 3 months.
- up to the age of 5 years, this is performed every 6 months.
- After the age of 5 years, children are usually followed up annually in a dedicated pediatric foot clinic, until skeletal maturity. Function is assessed, alongside monitoring for any signs of relapse.

Figure 2. Foot abduction bracing

5.2 **2 Operative indications**

5.2.1 Posteromedial soft tissue release and tendon lengthening

- resistant and/or recurrent feet in young children which have failed Ponseti casting and bracing
- "Rocker bottom" feet that develop following serial casting which failed non-surgical intervention
- syndrome-associated clubfoot if casting fails

5.2.2 Medial column lengthening or lateral column-shortening osteotomy, or cuboid decancellation

- often combined with initial surgical clubfoot release in children more than 2-3 years old
- may be performed in 3-10 years old children with recurrent deformity and "bean-shaped" foot

5.2.3 Talectomy

- in severe, rigid recurrent clubfoot in children with arthrogryposis
- age typically 6-10 years

5.2.4 Multiplanar supramalleolar osteotomy

- rarely necessary
- salvage procedure in older children with complex, rigid, multiplanar clubfoot deformities that have failed conventional operative management
- salvage procedure in older children (8-10 yrs) with an insensate foot

5.2.5 Ring fixator (Taylor Spatial Frame) application and gradual correction

- complex deformity resistant to standard methods of treatment
- recurrence of deformity is very high after frame removal

5.2.6 Triple arthrodesis

- almost never indicated
- contraindicated in insensate feet due to rigidity and resultant ulceration

VI. COMPLICATIONS

6.1 Complications with nonoperative treatment

- Deformity relapse

- relapse in child < 2 years
  - early relapse usually the result of noncompliance with bracing
  - associated with lower parental level of education
  - treatment with repeat manipulation and casting

- relapse in child > 2 years
  - treat initially with casting
  - consider tibialis anterior tendon transfer (split or whole tendon transfer)
  - consider repeat Achilles tendon lengthening or gastrocnemius recession for recurrent equinus

- Dynamic supination
  - may occur in approximately one third of patients
  - begins between 3 and 5 years of age
  - occurs during swing phase of gait with subsequent weight bearing on lateral border of foot
  - treated with anterior tibial tendon transfer to lateral cuneiform

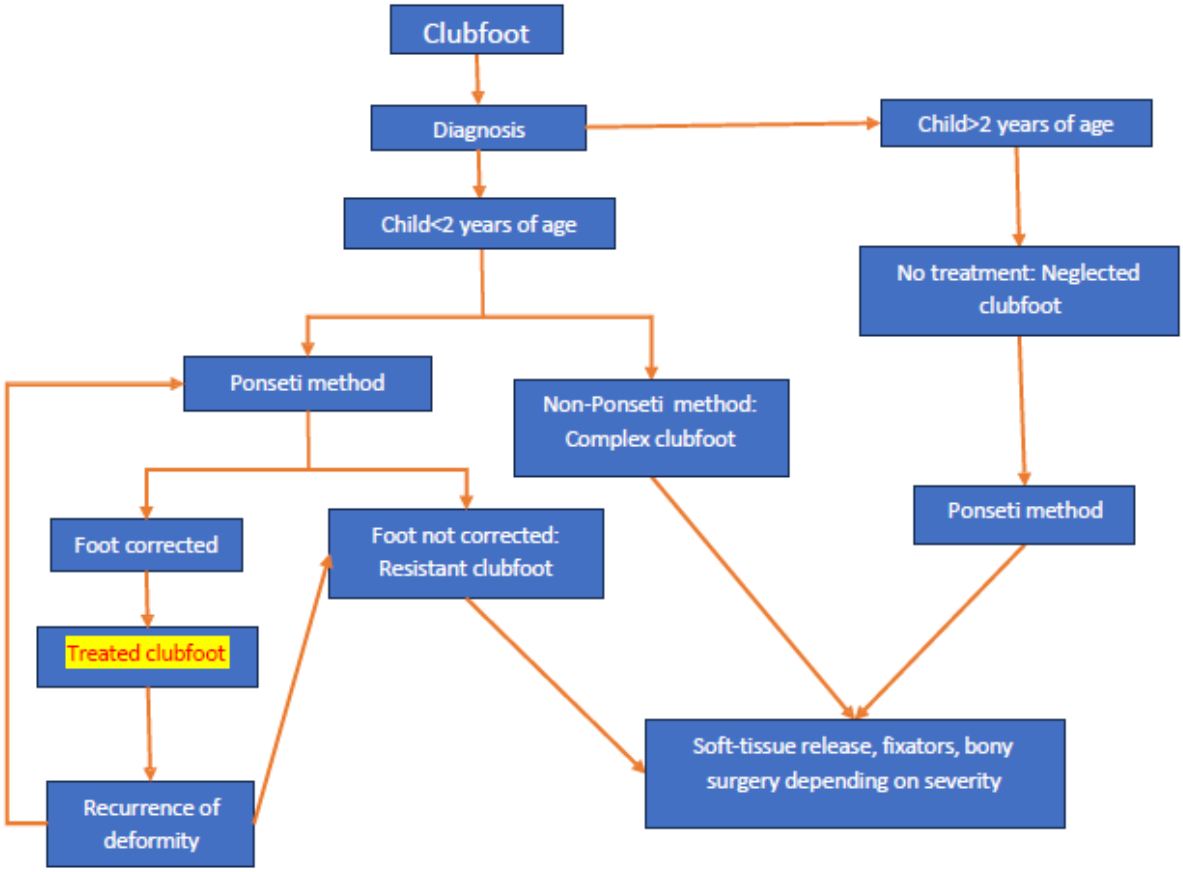
- Rocker bottom deformity
  - occurs when attempted correction of equinus contracture occurs before fully corrected hindfoot varus deformity

- dorsiflexion occurs through midfoot instead of through hindfoot.

6.2 Complications with surgical treatment

- residual cavus
  - result of insufficient plantar release and/or placement of navicular in dorsally subluxed position
- pes planus
  - results from overcorrection, often from extensive subtalar capsular release
- under correction
- in toeing gait
  - commonly due to internal tibial torsion and/or internal rotation of the talus within the ankle mortise
- osteonecrosis of talus
  - vascular insult to talus resulting in osteonecrosis and collapse dorsal bunion
  - caused by dorsiflexed first metatarsal (flexor hallucis brevis and abductor hallucis overpull secondary to weak peroneus longus) and overactivity of anterior tibialis
  - may be associated with inadvertent peroneus longus lengthening at the index procedure
  - treat with tibialis anterior lengthening or transfer, flexor hallucis longus transfer to the plantar aspect of the first metatarsal head, and possible plantarflexion osteotomy of the first ray.

VII. ALGORITHM



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PEDIATRIC SEPTIC ARTHRITIS

Chan Reaksmey, Cheng Samrech, Ros Rithyvorn, Ou Cheng Ngiep

I. DEFINITION

Septic arthritis is the intra-articular infection of a synovial joint. Bacterial arthritis usually occurs in a single joint, most commonly of the lower extremity. Polyarticular infections are more common in neonates and with certain pathogens (Neisseria meningitidis, Neisseria gonorrhoeae, and occasionally Staphylococcus aureus).

The incidence in developed countries is 4–5 cases per 100 000 children per year. It is more common in boys than girls with a ratio of 2:1.

II. ETIOLOGY

Entry of bacteria into the joint:

- In most cases, hematogenous spread.
- Infection may be introduced by direct inoculation via penetrating injury, or extension from bone infection (osteomyelitis).

Staphylococcus aureus is the commonest cause.

Other organisms should be considered based on:

- age (*Group B streptococci* in neonates),
- immunisation status (*Haemophilus influenzae)*
- underlying illness (*Salmonella* in children with sickle-cell disease).

Most common pathogens by age

Age	*Most common organism*
□3 months	Staphylococcus aureus Escherichia coli and other Gram-negative bacteria Group B Streptococcus Candida albicans Neisseria gonorrhoeae (newborns)
3 months to 5 years	Staphylococcus aureus Kingella kingae Group A Streptococcus Streptococcus pneumoniae (unimmunized children)
□5years	Staphylococcus aureus Group A Streptococcus

III. DIAGNOSTIC

III.1- Clinical examination Classic presentation:

acute onset (two to five days) of fever and joint pain, swelling and limited range of motion. However, the presentation varies depending upon the age of the child, the site of infection, and the causative organism.

In neonates and young infants, the typical presentation is that of septicemia, cellulitis, or fever without a focus of infection. Clues to joint involvement include lack of use of the involved extremity, aversion to or discomfort on being handled, postural changes, and unilateral swelling of the extremity, buttocks, or genitalia.

Older children generally have fever and constitutional symptoms in addition to swelling, tenderness, and limited mobility of the affected joint, but the joint findings may be subtle.

Physical examination:

Physical *examination* may confirm the presence and location of acute arthritis and may identify a source of bacteremia or entry point for direct inoculation. Examination also may provide support for conditions in the differential diagnosis.

- Observation of the child for restricted extremity use, a preferential position (antalgic attitude), and/or joint swelling and erythema.
- Palpation of ll bones and joints for local inflammatory signs.
- Assessment of range of motion of all joints.
- Examination of the skin and eyes.

III.2- Investigations

III.2.1- Laboratory test

- Complete blood count with differential (CBC), C-reactive protein (CRP), Erythrocyte sedimentation Rate (ESR): The peripheral white blood cell (WBC) count and CRP are usually elevated in patients with bacterial arthritis. Normalization of the WBC count and CRP is expected to occur with clinical improvement.

- Blood cultures: Blood cultures may provide the only confirmation of a pathogen and/or the only opportunity to obtain antibiotic susceptibility testing to guide therapeutic decisions.

- Synovial fluid WBC count, Gram stain, culture, and susceptibility testing: Synovial fluid WBC count of >50,000 cells/microL with >90 percent neutrophils are suggestive of bacterial arthritis, but the identification of organisms in the joint fluid is the primary criterion for diagnosis.
- Additional studies for select patients**:** Additional laboratory studies may be necessary in patients in whom particular organisms are suspected.

III.2.2- Imaging study

The radiography is rarely helpful in diagnosis. The widening of joint space and/or displacement of the normal fat pads may be the suggestive findings.

Ultrasonography is helpful in detecting joint fluid, but the presence of fluid is not specific for joint infection.

Magnetic resonance imaging is the most sensitive for the early detection of joint fluid and concomitant osteomyelitis but is not as readily available as ultrasonography and may require sedation for younger children.

IV. DIFFERENTIAL DIAGNOSIS

The differential diagnosis of bacterial arthritis includes other types of infectious and noninfectious arthritis, other infections (musculoskeletal and systemic), and other causes of joint pain.

- Juvenile Rheumatoid Arthritis,
- Kawasaki Disease
- Lyme Disease
- Rheumatic Fever
- Serum Sickness
- Transient Synovitis
- Trauma, including non-accidental injury
- Malignancy, including leukemia
- Other infection occurring near a joint, such as osteomyelitis, pyomyositis, septic bursitis, cellulitis, and abscess.

V. TREATMENT

Septic arthritis requires prompt recognition and management. Delays in treatment are associated with long-term damage to bones and joints.

Drainage of joint fluid and antimicrobial therapy are the cornerstones of therapy.

V.1- Drainage procedures:

Drainage and lavage are necessary to decompress the joint space and to remove inflammatory debris to preserve synovium and collagen matrix.Drainage can be accomplished through arthrotomy, arthroscopy, or needle aspiration (single or multiple).

Decisions regarding the optimal drainage procedure should be individualized according to the site and extent of involvement, duration of symptoms, the suspected organism, and other clinical features. It is preferred to do arthrotomy for children with bacterial arthritis of the hip or shoulder, penetrating trauma, concomitant osteomyelitis, and/or large amount of debris or loculations. For other joints, arthroscopy and needle aspiration are alternatives to arthrotomy.

Gonococcal arthritis seldom involves hip, or shoulder joints and usually resolves without surgical drainage.

V.2- Antibiotic Therapy

Antibiotic therapy is necessary to sterilize the joint fluid. Antibiotics should be administered as soon as possible after blood and synovial fluid cultures have been obtained.

Initial antimicrobial therapy is usually administered parenterally.

V.2.1- Empiric parenteral therapy:

Coverage for *S. aureus* should be included in the empiric regimen for children of all ages. Coverage for additional pathogens may be necessary based upon the child's age, particular clinical circumstances, and Gram stain. Empiric therapy can be altered when the susceptibility of the causative bacterium is known.

- *Children younger than three months:* Empiric therapy for bacterial arthritis in infants <3 months of age should be directed against *Staphylococcus*, group B *Streptococcus*, and gram-negative bacilli.
- *Children three months and older:* Empiric therapy for bacterial arthritis in children ≥3 months should be directed toward *S. aureus* and other gram-positive organisms (group A streptococci, *Streptococcus pneumoniae*). Additional coverage for other pathogens (*Kingella kingae*, *Haemophilus influenzae*, *N. gonorrhoeae*, *Salmonella spp*) may be necessary in select populations.

V.2.2- Pathogen-directed therapy:

The antimicrobial regimen can be tailored to a specific pathogen when culture and susceptibility results are available. Children whose cultures remain negative and improve with empiric therapy usually are continued on the empiric parenteral regimen.

V.2.3- Route and total duration of therapy:

In infants <1 month of age, the entire course of antimicrobial therapy is provided parenterally. Patients older than one month may be switched to oral therapy

if they have demonstrated clinical and laboratory improvement and meet prerequisites for oral therapy.

S. aureus arthritis is usually treated for at least three weeks; other causes of bacterial arthritis and culture negative arthritis are usually treated for two to three weeks. Antimicrobial therapy may be discontinued if the ESR and/or CRP have returned to normal by these time points and there is no radiographic evidence of unsuspected osteomyelitis.

V.2.4- Response to therapy:

Children receiving appropriate antimicrobial therapy generally demonstrate clinical improvement within three to five days. Clinical improvement is demonstrated by decreased fever; improved joint pain, swelling, erythema, and range of motion; and decreased peripheral WBC count, ESR and/or CRP, and synovial fluid WBC count and culture (if obtained).

Patients who do not respond to treatment as expected should be reevaluated. They may require arthrotomy and/or adjustment of antimicrobial therapy.

V.3- Adjunctive Therapies V.3.1- Analgesia:

Pain management is an important aspect of therapy for bacterial arthritis. Opioid therapy may be necessary during initial hospitalization. After discharge, acetaminophen or ibuprofen may be used for pain control.

V.3.2- Physical therapy:

Attention must be paid to joint position and rapid mobilization to prevent contractures and promote optimal nutrition to the articular cartilage. Once discharged home, the child initially may require a wheelchair or walker and continued physical therapy.

V.4- Patient Follow-Up

Patients should be seen approximately one week after discharge from the hospital and at 1-2 weeks interval thereafter. Clinical improvement and complications of high-dose antibiotic therapy should be monitored. CBC, ESR, and CRP should be also checked at each visit.

VI. COMPLICATIONS

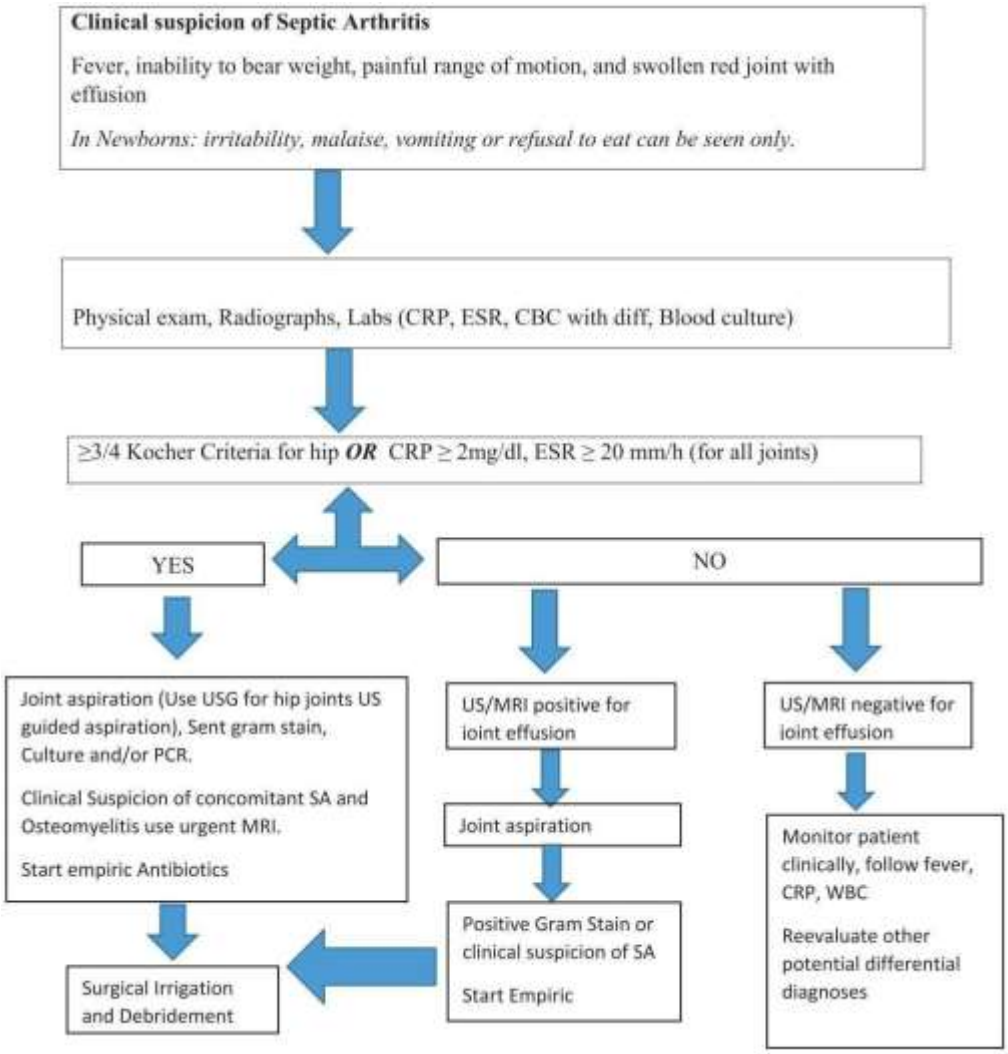
- Immediate complications: sepsis, septic shock, deep vein thrombosis, and septic pulmonary emboli.
- Long-term complication:

- Avascular necrosis
- Joint laxity, subluxation, or dislocation
- Limited range of motion of the joint
- Limb-length discrepancy or angular deformities
- coxa magna in bacterial arthritis of the hip
- Pathologic fractures
- Premature osteoarthritis

The following factors were associated with long-term complications:

- Duration of symptoms before treatment, particularly if >4 to 7 days.
- Involvement of the hip.
- Involvement of the hip or shoulder with concomitant osteomyelitis
- Age less than one year, particularly less than one month
- Isolation of S. aureus or Enterobacteriaceae compared with other pathogens

VII. ALGORITHM



Treatment approach decision-making algorithm for pediatric septic arthritis management (5)

CBC = complete blood count, CRP = C-reactive protein, ESR = erythrocyte sedimentation rate, SA = septic arthritis, PCR = polymerase chain reaction, US = ultrasound, USG = ultrasonography, WBC = white blood cell.

The 4 Kocher criteria: fever > 38.5°C, refusal to bear weight, leukocyte count >12,000/mm^3,ESR > 40mm/h.

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CHAPTERVI

PLASTICSURGERY

1. Bed sore
2. Burn
3. Skin tumor
4. Soft tissues infection

BEDSORE/ PRESSURE ULCERS

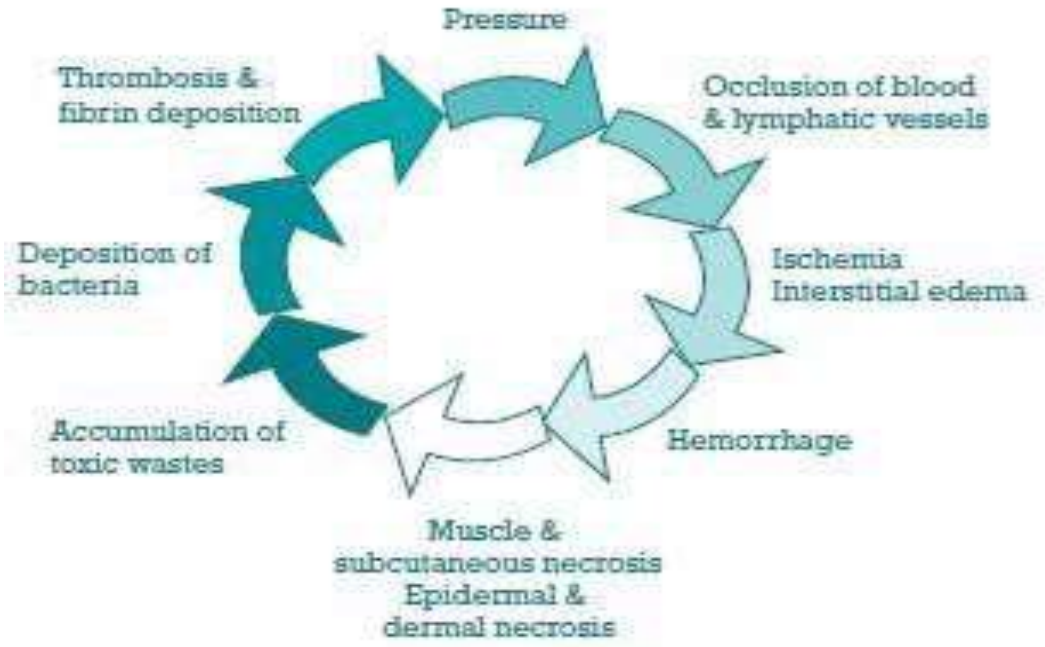
KY ChanmonyRaksmey, Tep Borin

I. DEFINITION

- Any lesion caused by unrelieved pressure that results in damage to underlying tissue
- Usually occurs over a bony prominence
- Staged to classify the degree of tissue damage observed (National Pressure Ulcer Advisory Panel, 1989)

II. CAUSES

- Area of greatest damage near bony prominences



(Goode & Allman, 1989)

- By the time inflammation becomes visible, necrosis of muscle, fat and subcutaneous tissue may have occurred
- Worsen between 1-2 days
- Underlying damage has begun as tissue destruction has been set in motion (Patterson & Bennett, 1995)

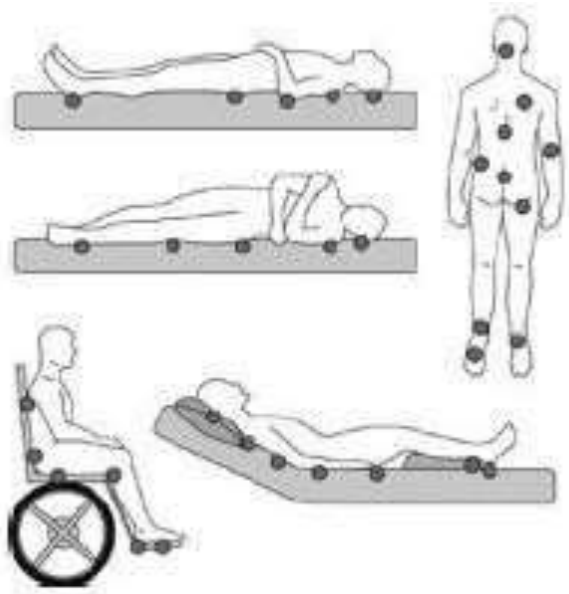
a) Risk Factors for Developing Pressure Ulcers

1. Mobility
2. Activity
3. Sensory Perception
4. Nutrition
5. Arteriolar Pressure
6. Pressure
7. Moisture
8. Friction
9. Shear
10. Age

(Braden, 1987)

b) Pressure Ulcer Sites

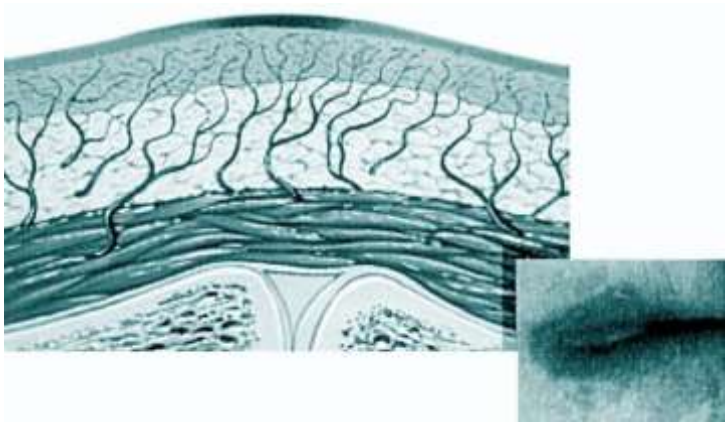
1. Supine Position: heels, sacrum, elbows, scapulae, back of head
2. Lateral Position: malleous, medial and lateral condyles, greater trochanter, ribs, acromion process, ear
3. Prone Position: toes, knees, genitalia (men), breasts (women), acromion process, cheek and ear
4. Sitting Position: elbow, sacrum, ischium



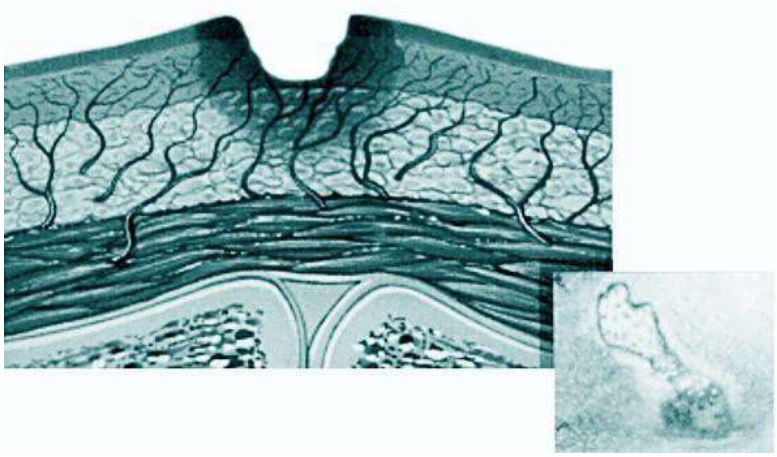
c) Pressure Ulcer Staging System

The HSE Wound Management Guideline (2018) 4 describes the following Pressure Ulcer Staging System (see Appendix 1): 4 HSE

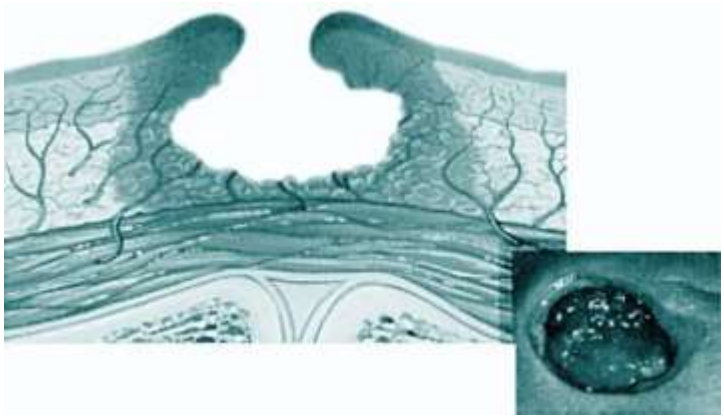
- Stage I: Intact skin with non – blanchable redness of a localised area usually over a bony prominence. Discolouration of the skin, warmth, oedema, hardness or pain may also be present. Darkly pigmented skin may not have visible blanching. The area may be painful, firm, soft, warmer or cooler as compared to adjacent skin.



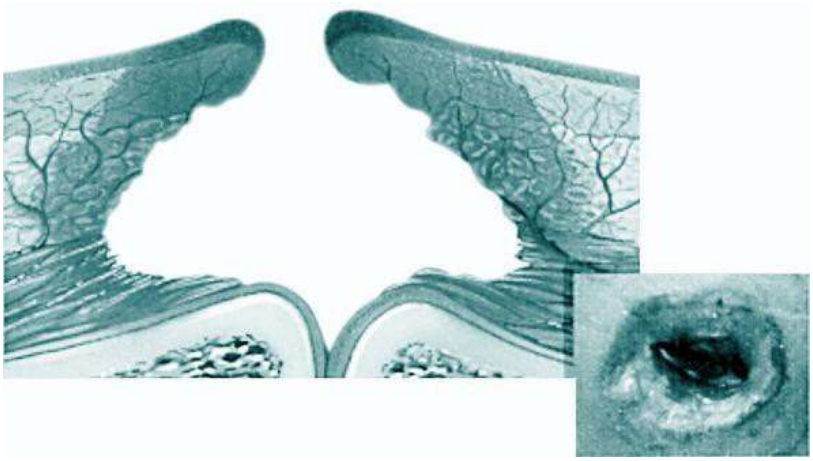
- Stage II: Partial thickness skin loss of dermis presenting as a shallow ulcer with a red pink wound bed, without slough. May present as an intact or open/ ruptured serum filled blister filled with serous or sero-sanguineous fluid. Presents as a shiny or dry shallow ulcer without slough or bruising.



- Stage III: Full thickness skin loss. Subcutaneous fat may be visible but bone, tendon or muscles are not exposed. Slough may be present but does not obscure the depth of tissue loss. The stage may include undermining or tunnelling.



- Stage IV: Full thickness tissue loss with exposed bone, tendon or muscle. Slough or eschar may be present. This stage often includes undermining and tunnelling. Exposed bone / muscle is visible or directly palpable.



Suspected deep pressure and shear induced tissue damage, depth unknown. For Service Users with non-blanchable redness and purple/maroon discoloration of intact skin combined with a history of prolonged, unrelieved pressure/shear, this skin change may be an indication of emerging, more severe pressure ulceration i.e. an emerging Stage III or IV Pressure Ulcer. Clear recording of the exact nature of the visible skin changes, including recording of the risk that these changes may be an indication of emerging more severe pressure ulceration, should be documented in the Service User's health record. These observations should be recorded in tandem with information pertaining to the Service User's history of prolonged, unrelieved pressure/shear.

It is estimated that it could take 3-10 days from the initial insult causing the damage, to become a Stage III or IV Pressure Ulcer. Stable eschar (dry adherent, intact without erythema or fluctuance) on the heel serves as the body's biological cover and should not be removed. It should be documented as at least Category / Stage III until proven otherwise.

See Appendix 1 for illustration of the HSE Pressure Ulcer Category/Staging System Recommendations.

The Braden Scale for Predicting Pressure Sore Risk

Educational Workshop for RNs and RPNs: Assessment and Management of Pressure Ulcers

Assessing Risk Factors for Developing Pressure Ulcers

The Braden Scale for Predicting Pressure Sore Risk

Sensory Perception Ability to respond meaningfully to pressure-related discomfort.	1. Completely limited Unresponsive (does not moan, flinch or grasp) to painful stimuli due to diminished level of consciousness or sedation, or limited ability to feel pain over most of body surface.	2. Very limited Responds only to painful stimuli. Cannot communicate discomfort except by moaning or restlessness, or has a sensory impairment that limits the ability to feel pain or discomfort over half of body.	3. Slightly limited Responds to verbal commands but cannot always communicate discomfort or need to be turned, or has some sensory impairment that limits ability to feel pain or discomfort in 1 or 2 extremities.	4. No impairment Responds to verbal commands. Has no sensory deficit that would limit ability to feel or voice pain or discomfort.	
Moisture Degree to which skin is exposed to moisture.	1. Constantly moist Skin is kept moist almost constantly by perspiration, urine etc. Dampness is detected every time patient is moved or turned.	2. Very moist Skin is often, but not always, moist. Linen must be changed at least once a shift.	3. Occasionally moist Skin is occasionally moist, requiring an extra linen change approximately once a day.	4. Rarely moist Skin is usually dry, linen only requires changing at routine intervals.	
Activity Degree of physical activity.	1. Bedfast Confined to bed.	2. Chairfast Ability to walk severely limited or non-existent. Cannot bear own weight and/or must be assisted into chair or wheelchair.	3. Walks occasionally Walks occasionally during day, but for very short distances with or without assistance. Spends majority of each shift in bed or chair.	4. Walks frequently Walks outside the room at least twice a day and inside room at least every 2 hours during waking hours.	
Mobility Ability to change and control body position.	1. Completely immobile Does not make even slight changes in body or extremity position without assistance.	2. Very limited Makes occasional, slight changes in body or extremity position but unable to make frequent or significant changes independently.	3. Slightly limited Makes frequent though slight changes in body or extremity position independently.	4. Walks frequently Makes major and frequent changes in position without assistance.	
Nutrition Usual food intake pattern.	1. Very poor Never eats a complete meal. Rarely eats more than 1/3 of any food offered. Eats 2 servings or less of protein (meat or dairy products) per day. Takes fluids poorly. Does not take a liquid dietary supplement, or is NPO and/or maintained on clear liquids or IVs for more than 5 days.	2. Probably inadequate Rarely eats a complete meal and generally eats only about half of any food offered. Protein intake includes only 3 servings of meat or dairy products per day. Occasionally will take a dietary supplement, or receives less than optimum amount of liquid diet or tube feeding.	3. Adequate Eats over half of most meals. Eats a total of 4 servings of protein (meat, dairy products) each day. Occasionally will refuse a meal, but will usually take a supplement if offered, or is on a tube feeding or TPN regimen, which meets most of nutritional needs.	4. Excellent Eats most of every meal. Never refuses a meal. Usually eats a total of 4 or more servings of meat and dairy products. Occasionally eats between meals. Does not require supplementation.	
Friction and Shear	1. Problem Requires moderate to maximum assistance in moving. Complete lifting without sliding against sheets is impossible. Frequently slides down in bed or chair, requiring frequent repositioning with maximum assistance. Spasticity, contractures or agitation lead to almost constant friction.	2. Potential Problem Moves feebly or requires minimum assistance. During a move skin probably slides to some extent against sheet, chair, restraints, or other devices. Maintains relatively good position in chair or bed most of the time but occasionally slides down.	3. No apparent problem Moves in bed and in chair independently and has sufficient muscle strength to lift up completely during move. Maintains good position in bed or chair at all times.	NOTE: Patients with a total score of 16 or less are considered to be at risk of developing pressure ulcers. (15 or 16=low risk; 13 or 14=moderate risk; 12 or less=high risk)	

Nursing Best Practice Guidelines Program

Registered Nurses' Association of Ontario

III. DIAGNOSTIC PROCEDURE

History and Physical Examinations:

Pressure ulcers should be assessed in the context of the patient/resident's overall physical and psychological health.

i. Physical health:

Intrinsic Factors – Relate to the aspects of the client's physical, medical or psychosocial condition.

- Physical
- Nutritional status
- Reduced mobility/immobility
- Posture/contractures
- Repetitive stress syndrome
- Neurological/sensory impairment
- Incontinence
- Age
- Level of consciousness
- Medical
- Acute illness
- History of previous pressure damage
- Vascular disease
- Chronic/terminal illness
- Psychosocial
- Stress and anxiety
- Sleep disturbances

Extrinsic Factors – Are derived from the environment

- Friction
- Pressure
- Shearing
- Hygiene
- Living Conditions
- Medication
- Garments
- Transfer slings
- Restraint use

- Support systems for pressure relief

ii. Psychological health:

- Mental Status
- Learning Ability
- Depression
- Social Support
- Polypharmacy/Overmedication
- Alcohol/Drug Use
- Goals/Values and Lifestyle
- Sexuality
- Culture and Ethnicity
- Pain as a symptom
- Stressors
- Resources (availability and skill of caregivers, finances, equipment) of individuals being treated for pressure ulcers in the home

iii. Quality of Life

- How do pressure ulcers affect the patient's quality of life?
- Ask patient to describe his/her current health status
- Ask patient how the pressure ulcer impacts on his/her day to day living

iv. Pressure Ulcer Assessment

- Location
- Depth/Stage
- Size (cm)
- Odour
- Sinus Tracts
- Undermining
- Tunnelling
- Exudate
- Wound Bed
- Appearance/Condition of Surrounding Skin (Periwound) and Wound Edges
-

v. Location (Barton & Parslow, 1996)

Most common sites: Sacrum (60% of all sores). Ischial Tuberosities in the sitting patient, greater trochanter, heel (15%)

Uncommon Sites: Elbow. Knee. Ankle. Occiput

vi. Measurement

- Size (how to measure)
- Use a ruler, transparency tracings or photography
- Measure the width/depth/breadth
- Weekly measurement are usually suffi cient
- Depth
- Sinus Tracts
- Undermining (clock measurement)
- Tunnelling

vii. Exudate

- Type
  - *Serous:* Clear fl uid with visual absence of blood, pus or other debris
  - *Sanguinous: Bloody, appearing to be composed entirely of blood*
  - *Serosanguinous: Blood mixed with obvious quantities of clear fluid*
  - *Purulent: Pus* like in appearance, cloudy and viscous
- Amount
  - *Dry: Wound does not produce exudates*
  - *Low: Wound is moist*
  - *Moderate: Surrounding skin is wet*
  - *High:*
    - Surrounding skin is saturated (sometimes macerated)
    - Wound is bathed in fluid

viii. Wound Base

black eschar, necrotic cleanse/debride yellow fibrin or slough cleanse/debride pink/red granulation protect

IV. THERAPEUTIC APPROACH

v. Stabilize patient

Prevent pressure and trauma in order to maintain skin integrity

- **Do's**

  1. Prevent local areas of pressure

- **Don'ts**

  1. Prevent local areas of pressure
  2. Provide pressure reduction via ues of mattress overlays, cushions, or foams

- Prevent maceration imitation and bacterial growth

  1. Keep skin hydrated and supple
  2. Keep skin clean and dry
  3. Wash skin gently with water; pH balanced soaps or skin cleansers
  4. Investigate and manage incontinence

- Prevent moisture retention and excessive warmth
  1. Avoid use of plastics (under pads and diapers) choose liner or fabric instead
  2. Increase vigilance when patient is diaphoretic
  3. Protect skin by applying barrier creams, gels or pastes
  4. Avoid applying lotion between toes

- Involvement of Physicians and Surgeons
  1. General Practitioners
  2. Geriatricians
  3. Dermatologists
  4. Plastic Surgeons
  5. Vascular Surgeons

vi. Empirical treatment

Wound Healing: A cascade of events of the biologic and immunologic system

(CREST, 1998). The recognized end point in healing is total wound closure (Robson et al., 1998).

A. Mechanical Impediment:

- Dead tissue in a wound slows wound healing by impeding the migration of epithelial cells from wound edges
- Eschar prevents the wound edges from drawing together and is a breeding ground for bacteria
- Foreign material (lint and pieces of dressing) can impede epithelial migration and increase likelihood of infection

- A dry wound site can impede the viability of the cells and tissues involved in wound healing When applied to healthy tissue, some antimicrobial preparations and cleansing agents may delay wound healing and may even be toxic to viable tissue

Example: Full-strength hydrogen peroxide can damage newly forming cells that remain in the wound. To reduce surface bacteria and tissue trauma, irrigate the wound gently with 100 to 150 ml of normal saline.

Use enough irrigation pressure to enhance wound cleansing without causing trauma to the wound bed. 4-15 psi is the safe and effective irrigation pressure range. To achieve 4-15 psi, use a 20-35 ml syringe with a 19 gauge angiocath.

B. Reminder:

Do not use skin cleansers or antiseptics to clean the wound. For example:

1. Povidine Iodine (Betadine)
2. Iodophor
3. Sodium Hypochlorite Solution
4. Hydrogen Peroxide
5. Acetic Acid

C. Debridement:

Debridement is often necessary to remove devitalized tissue and exudates, reduce the risk of infection, prepare the wound bed and promote healing. Debridement can be:

- Autolytic
- Mechanical
- Sharp
- Enzymatic

D. Assessment and Management of Infected Wounds:

a) Infection is diagnosed when >105 bacterial/gram tissue is present

b) Clinical Signs of Infection

- Delayed healing/dehiscence
- Increased wound pain
- Malodour
- Abscess/sinus formation
- Localized swelling/redness/heat
- Increased level of exudates/purulent discharge

- Pyrexia, rigours
- Tachycardia

E. Antibiotics:

- Systemic antibiotics are not required for pressure ulcers with only clinical signs of local infection
- Exceptions with locally infected wounds requiring systemic antibiotics
- Systemic antibiotics are used when the virulence of the organism is high and the host's defenses are compromised

F. Antiseptics:

- Use of cytotoxic antiseptics to reduce bacteria in wound tissue is not recommended
- Typical management of infected wounds includes the use of topical antimicrobials rather than antibiotics or antiseptics

G. Dressings

- Sterile dressings should be used in all care settings
- Avoid all occlusive dressings if anaerobic infection is suspected or cultured
- Protect non-infected ulcers with occlusive dressings

vii. Directed treatment

Stage I **Goals:** To reduce further skin breakdown and prevent skin loss. Protect against moisture and friction

- **Interventions:** Protect area from friction, shear, and maceration using a transparent film dressing or
- thin hydrocolloids. Provide pressure relieving devices to reduce friction and shearing forces
- **Goals:** To reduce further skin breakdown and prevent skin loss. To protect the surrounding skin from moisture by managing exudates and providing a moist wound environment to promote healing.
- **Interventions:** Clean the ulcer with normal saline. Protect the wound by covering it with a transparent dressing or hydrocolloid. For moderate amount of exudates, use an absorbent foam dressing. Use liquid or solid barriers to protect periwound skin from maceration damage.

Stage II

Stage III & IV

- **Goals:** To remove cell debris and promote autolysis. To provide clean, moist environment for the healing process to begin. To absorb exudates. To protect from contamination and trauma. To decrease dressing changes. To protect surrounding skin
- **Interventions:**
- **Dry Cavity:** Irrigate with normal saline using a 20-35 ml syringe and 19-gauge needle or angiocath. Protect periwound skin with a protective barrier.

  Fill dead space with appropriate filler (including sinus tracts). Line cavity with gel and place 4 x 4 gauze packed loosely. Protect from contamination by use of an absorbent outer semi-occlusive dressing
- **Interventions: Exudating Cavity:** Irrigate with normal saline using a 20- 35 ml syringe and 19-gauge needle or angiocath. Protect periwound skin using a protective barrier. Fill dead space with appropriate filler (including sinus tracts). Use absorbent foam dressings. Protect from contamination by use of an outer semi-occlusive dressing.

Surgery:

- **Goals:** To debride and remove dead tissue. To rehydrate the eschar by providing a clean, moist
- environment for the healing process to begin. To promote closure/healing
- **Interventions:** Clean with normal saline. Surgical debridement by MD or trained person. Autolytic debridement using gels. Protect

  periwound skin using a protective barrier. Cover with transparent dressing.
- **Antiseptics:** cytotoxic antiseptics is not recommended. Typical management: topical antimicrobials rather than antibiotics or antiseptics
- **Dressings:** Sterile dressings should be used in all care settings. Avoid all occlusive dressings if anaerobic infection is suspected or cultured. Protect non-infected ulcers with occlusive dressings.
- **Pain Assessment and Management Operative Repair of Pressure Ulcers:** Wounds can be closed by direct closure, skin grafting, skin flaps, musculocutaneous flaps and free flaps. Procedure is performed by MD and plastic surgeon. Candidates for operative repair are medically stable, adequately nourished, and can tolerate operative blood loss and postoperative immobility. Other considerations are quality of life, patient

preferences, treatment goals, risk of recurrence, and expected rehabilitative outcome

viii. Monitoring

- **PostoperativeCare: Havepatient slowly increaseperiods oftime sitting or lyingon theflap to increaseits toleranceto pressure. Monitoring the flap for pallor,redness,orboththatdonotresolvein10minutesor pressure relief.**
- **Discharge/Transfer of Care Arrangements: Patients/residents moving between care setting should** have the
  - following information provided:
  - Risk factors identified
  - Details of pressure points and skin condition prior to transfer
  - Type of bed/mattress required
  - Details of healed ulcers
  - Stage, site and size of existing ulcers
  - History of ulcers, previous treatments and dressings used
  - Type of dressing currently used and frequency of change
  - Allergies to dressing products
  - Need for on-going nutritional support

BURN

KY ChanmonyRaksmey, Tep Borin

I. DEFINITION

Burn is a tissue injury that results from thermal application (hot/cold) and from application of physical or chemical energy.

II. CAUSES

Burns are caused by dry heat, moist heat, cold injury, chemical burn, electrical burns, ionizing radiation and friction.

III. DIAGNOSTIC PROCEDURE

III.1. Clinical argument

III.1.A Histories

- Time of burn
- Histories medico-surgeries
- Last meal
- Events and environment of burn injury

III.2. III.1.B Degree of burn

1st degree: minor epithelial damage, present as redness, clinically insignificant 2nd degree: partial thickness burns and scalds. These burns are painful.

3rd degree: full thickness burn, usually painless.

4th degree: As the third but extends into fascia and/or muscle

Fig (1)

Depth of Burn	Appearance	Sensation
First degree	Red Blanches with pressure Dry **No** blisters	Painful
Second degree *Partial thickness* - **superficial**	Red Blanches with pressure Moist, weeping Blisters	Painful to temperature and air
Second degree *Partial thickness* - **deep**	Variable colour **No** blanching with pressure Wet, waxy or dry Blisters (easily unroofed)	Perceptive of pressure only
Third degree *Full thickness*	Waxy white, leathery gray, charred or black **No** blanching with pressure Dry, inelastic	Deep pressure only
Fourth degree	As with Third degree, but extends into fascia and/or muscle	Deep pressure

Fig 2

Depth of Burn	Appearance	Sensation
First degree	Red Blanches with pressure Dry **No** blisters	Painful
Second degree *Partial thickness* - **superficial**	Red Blanches with pressure Moist, weeping Blisters	Painful to temperature and air
Second degree *Partial thickness* - **deep**	Variable colour **No** blanching with pressure Wet, waxy or dry Blisters (easily unroofed)	Perceptive of pressure only
Third degree *Full thickness*	Waxy white, leathery gray, charred or black **No** blanching with pressure Dry, inelastic	Deep pressure only
Fourth degree	As with Third degree, but extends into fascia and/or muscle	Deep pressure

It is common to find all types of burns within the same wound and the depth may change with time, especially if infection occurs.

I.1.A. **Calculation of surface area** of burn Adults :

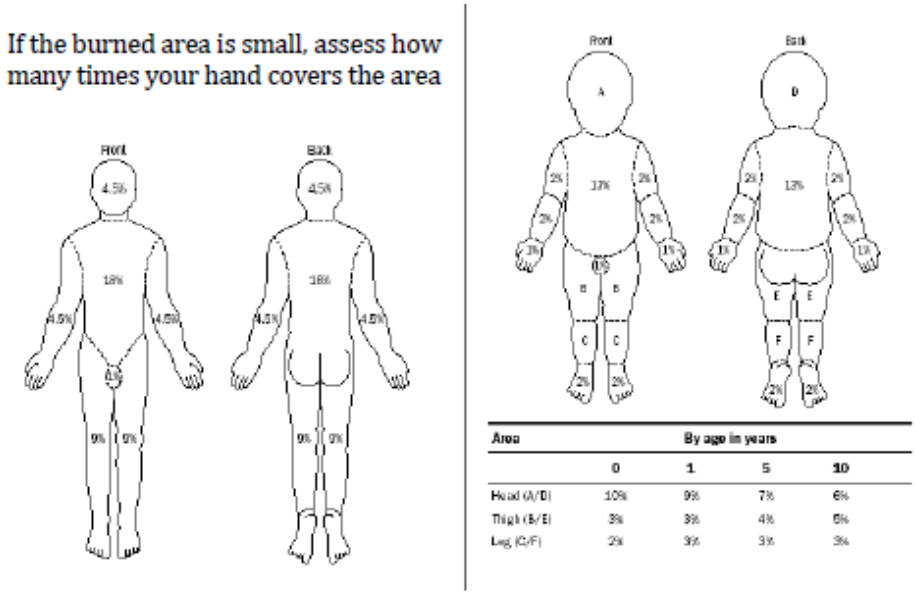
+ the Rull of 9 is commonly used to estimate the burned surface area in adults.
+ the body is divided into anatomical regions that represent 9% (or multiples of 9%) of the total body surface area.
+ the outstrectched palm and fingers approximate to 1%

Children: the rule of 9s is modified for infants and children since their heads and lower extremities represent different proportions of body surface area.

Technical procedure

Laboratory test: blood test Chest X-ray if necessary

Fig 3



III.2.Admission criteria

- Partial thickness burn of >25% of TBSA adults
- >20% burns in children below 10 years old and adults > 50 years old
- Full thickness burn > 10% TBSA
- Burn of face, eyes, ears, and perineum
- Chemical burns, electrical burns, inhalation injury, and underlying debilitating illness
- Moderate burns > 15% TBSA (TBSA=total body surface area)

IV. THERAPEUTIC APPROACH

- In all cases, administer tetanus (inj. TT 0.5cc IM) prophylaxis
- Remove with debride devitalized and loose tissue
- Pain management
- Airway: airway burns, smoke inhalation syndrome, severe burns of face and neck, laryngeal edema may need inhalation or tracheostomy
- O2 inhalation: started at 8-12 lit/min in patients having dyspnea, major burns, airway burns, and smoke inhalation syndrome
- Fluid resuscitation:
    - Insertion of large bore cannulae
    - oParkland formula is to be used for calculation or fluid requirement in first 24 hrsi.e 4ml/kg/% of burn.
    - The fluid of choice is Ringer lactate
    - 50% of the fluid is to be transfused in first 8hrs from the time of burns and the remaining 50% in next 16hrs
    - Adequate resuscitation is monitored by vital parameters and urine output of 0.5- 1ml/kg/hr.
    - oIf the urine output is in excess of 2ml/kg.hr the rate of infusion should be reduced
    - Next 24hrs:
        - Total volume = ½ of first day
        - Colloids (0.5ml/kg/%) and 5% glucose or isotonic glucose saline to make up the rest
- Nasogastric tube insertion if necessary
- Antibiotics:
    - Used of broad-spectrum antibiotics lead to colonization of wound by resistant organisms
    - However, Ceftazidime + Amikacin + Metronidazole canbe used after 48hrs pending sensitivity of organism isolated from the wound
- H2-receptor antagonists:
    - Acute upper GI erosions and ulcers (curling ulcer) may occur in patients with severe burn injuries. The common clinical finding in such patients is painless GI hemorrhage. Traitment of acute stress ulceration is principally preventive

- H2-receptor antagonists inhibit gastric acid secretion, this prophylaxis against acute stress ulceration is initiated immediately after admission. Commonly used drug is inj. Ranitidine 50mg x 8th hourly

- Nutrition (third day onwards)

oStart with oral fluids and Fortified milk:

- Calculation calories; Adults 25kcal x Kg + 40 x %burns; Child 60kcal x kg + 35 x %burns
- Protein; Adult 1gm/kg +3gm/%burns; Child 3gm/kg + 1gm/%burns
- Vitamin supplements

- Care of burn wounds:
  - Clean wound with dilute normal saline or ringer's lactate
  - Debride blisters and nonviable tissues
  - Use Silver sulphadiazine or Soframycine ointment to cover the wounds
  - Wounds can be managed by exposure method or by closed method by covering wounds with Vaseline gauze and bandages

- Others supportive therapy:
  - Physiotherapy for joints
  - Compression dressings to prevent hypertrophic scars
  - Psychotherapy
  - Rehabilitation therapy

V. COMPLICATIONS:

V.1. Early complications

- burn shock
- renal failure
- smoke inhalation syndrome
- septicemia

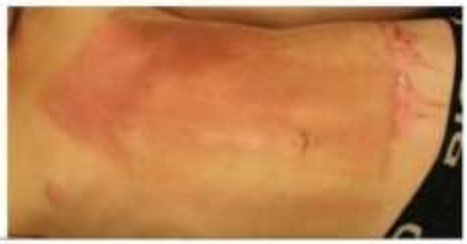
V.2. Late complications

- hypertrophic scars
- keloids
- contractures
- protein losing enteropathy

Appendix 2: recognising burn depths chart

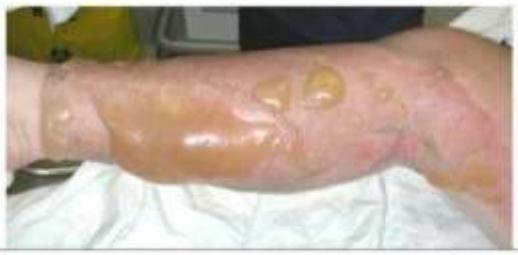
Epidermal burn (erythema)

- Damage to epidermis only. Skin intact, no blisters present
- Erythema. Red
- Brisk capillary refill
- Heal spontaneously within 3–7 days with moisturiser or protective dressing



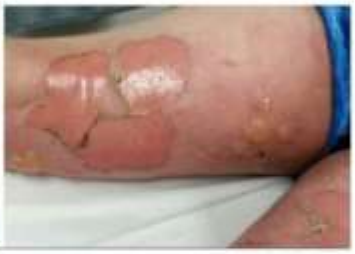
Superficial dermal burn

- Damage to upper layer of dermis
- Pink. Blisters present or absent
- Brisk capillary refill (under blister)
- Should heal within 7–10 days with minimal dressing requirements



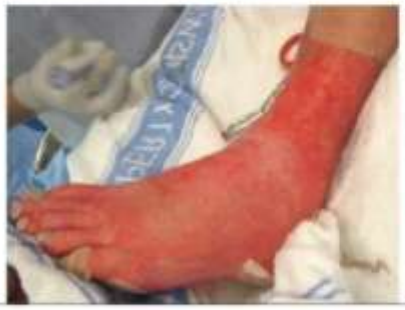
Mid dermal burn

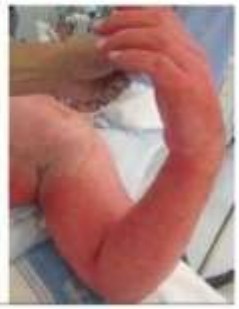
- Damage into mid dermis
- Dark pink
- Sluggish capillary refill
- Should heal within 14 days.
- Deeper areas may need surgical intervention and referral



Deep dermal burn

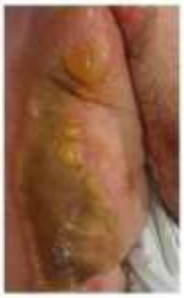
- Burn extends into the deeper layers of the dermis, but not through the entire dermis
- Blotchy red/white
- Sluggish to absent capillary refill
- Generally need surgical intervention.
- Refer to specialist unit



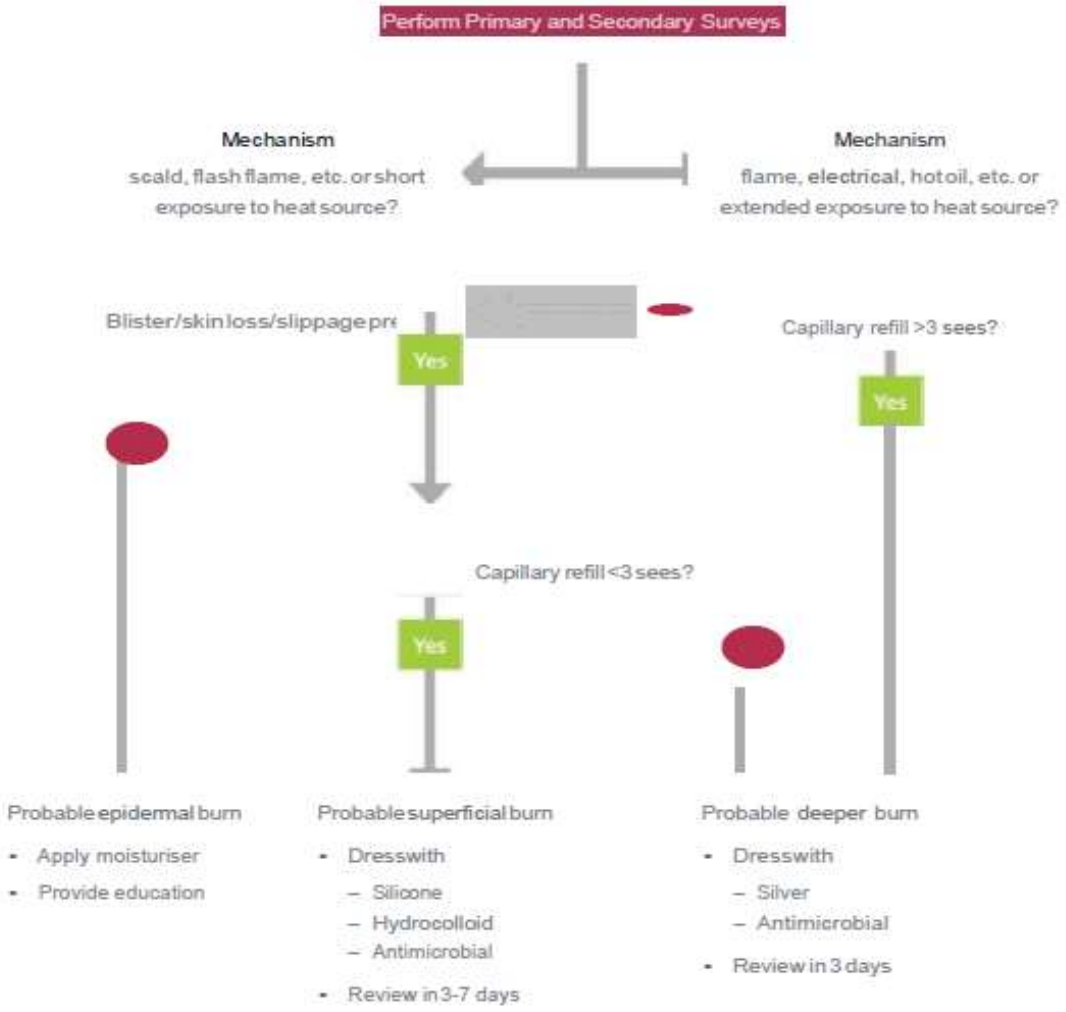


Full thickness burn

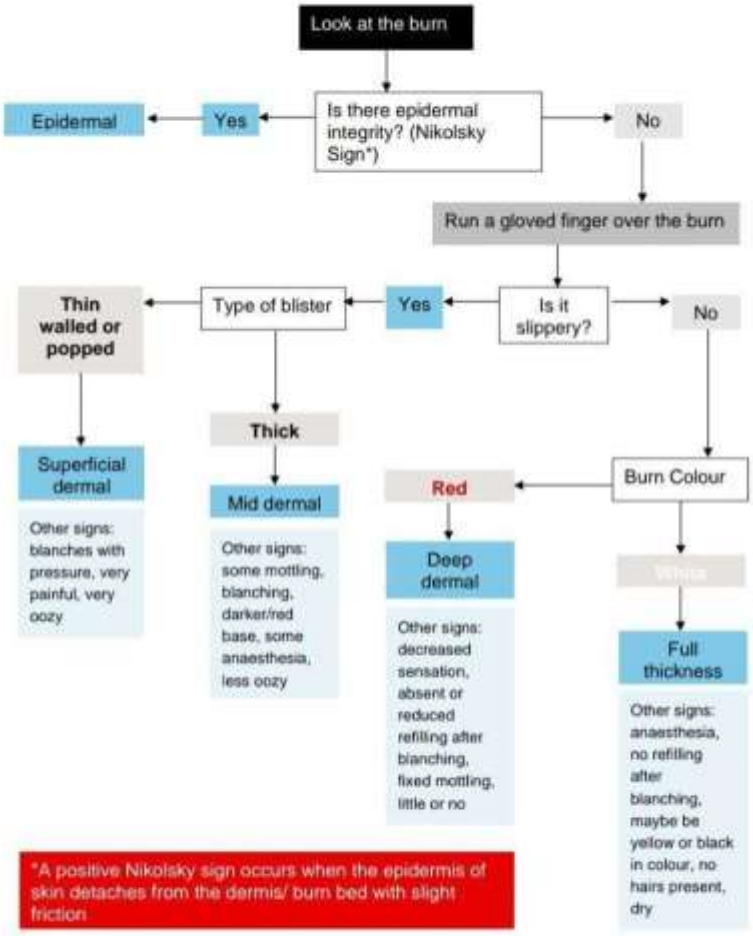
- Entire destruction of dermis, sometimes underlying tissue involved
- White, waxy, brown, black.
- No capillary refill
- Surgical intervention and long-term scar management required
- Refer to specialist unit



Burn patient dressing decision-making tree



Appendix I – Protocol for burn depth assessment



Appendix N – Management of facial burns

Initial care (may be done in theatre/technical suite)

- All non-viable tissue should be gently removed by picking and washing with gauze. Beard, moustache and sideburn hair will have been shaved completely and scalp hair similarly shaved away from the burn edge. Soft paraffin is applied. **Do not apply Flamazine- risk of corneal ulceration.**

Ongoing care (on ward, aseptic technique)

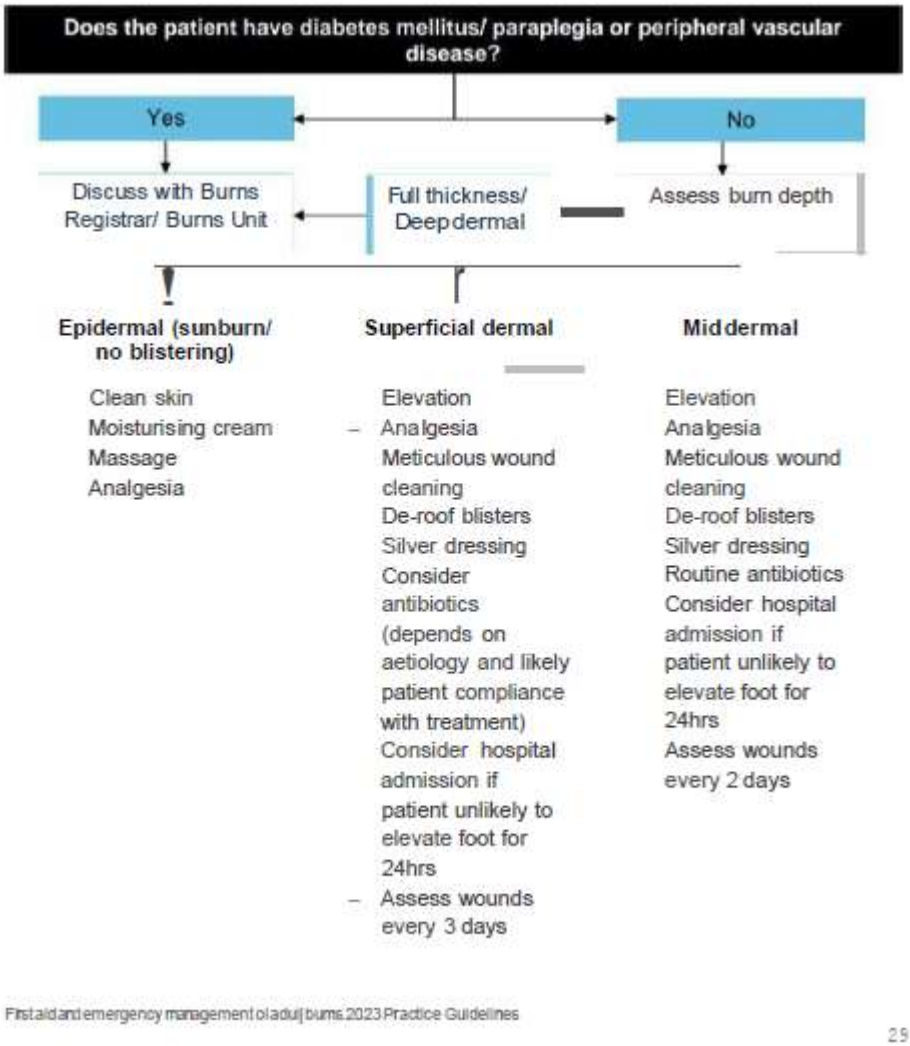
- Performed **6-hourly** with eye care (increase frequency if dry). Gentle cleaning and removal of existing paraffin and any newly declaring non-viable tissue. Forceps are to be avoided on the face.
  Application of a **thin** layer of white soft paraffin.
- **Performed 12-hourly** (with mouth and ear care) – male patients will undergo shaving of facial hair.
- **Performed daily** – hair washing.
- **Viral Swabbing** – Patients with facial burns (particularly those with a history of cold sores), virology swabs must be performed for Herpes Simplex Virus on days two and five **post-burn**. A positive result requires Famciclovir administration (herpes face burn infection is very painful, delays healing and leads to poorer healing and scarring).
- Patients with a strong history of HSV-1 and a facial burn should be considered for antiviral prophylaxis.

Appendix 0 Management of burns to the foot

Each foot is colonised by 1,000,000,000,000 bacteria. Inadequate management of foot burnsfrequently results inserious infection.This can lead to a need for skin grafting (where spontaneous healing was expected) and even digital/other amputation.

Avoid any constrictive/abrasive footwear - loose footwear should be worn. Initial elevationfor at least 24 hours is of utmost importance in preventing burn depth progression.

Time off work should be considered especially for those whose jobs entail standing or a hot,dusty dirty environment. As burns to the feet meet ANZBA referral criteria, consider contacting the Burns Unit/Burns Registrar for advice and how to best manage foot wounds .



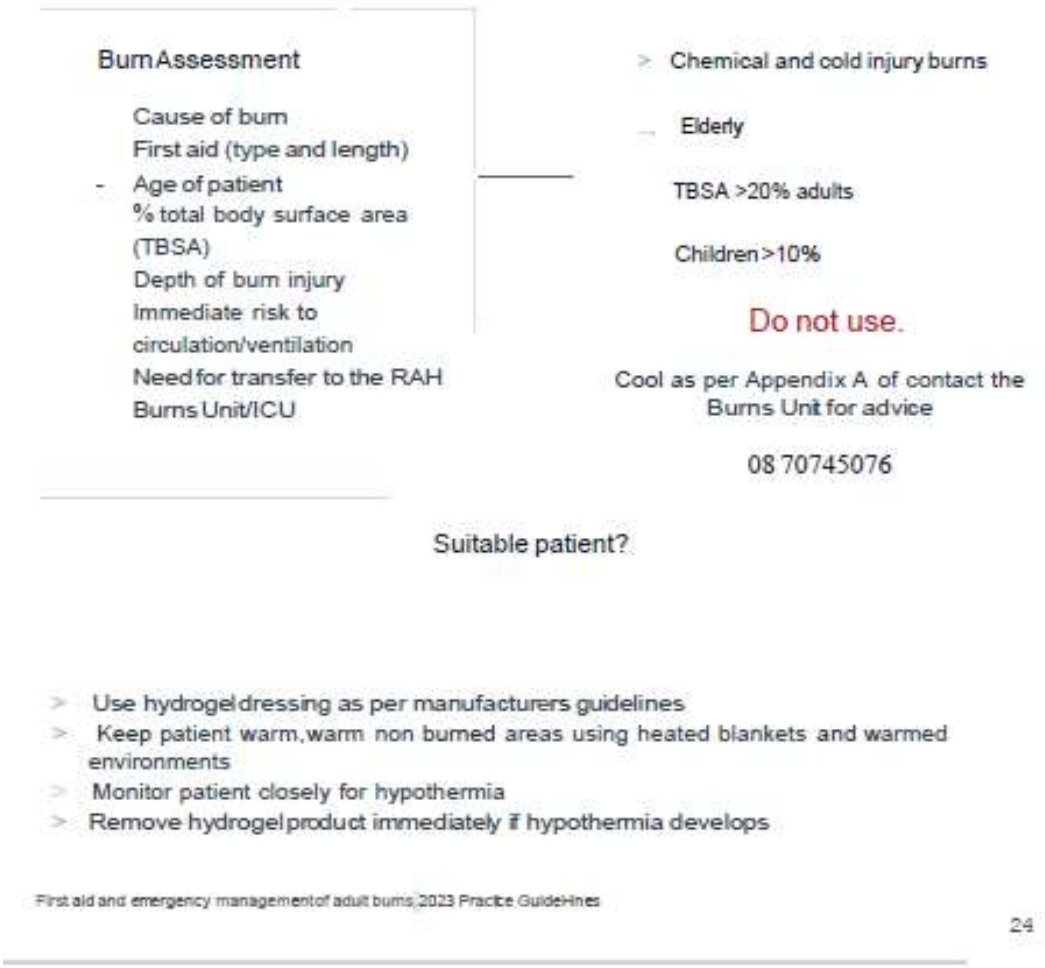
Appendix J - Protocol for the use of Hydrogel cooling products in burn injury first aid

Evidence indicates that the cooling function of Hydrogel dressing products is not as effective as cool running water.

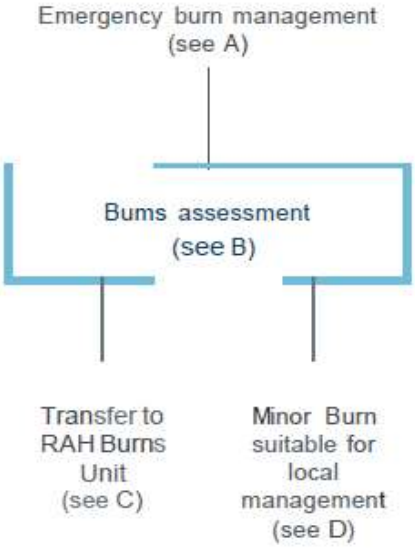
If no clean water available,application of Hydrogel may be useful as an analgesic but should be replaced by water as soon as available if within 3 hours of injury.

Patients with extensive burn wounds (>20% TBSA in adults or >10% TBSA in children) are at increased risk of hypothermia

Hydrogels,or any wet dressing, can also be associated with the development of hypothermia if exposed to the air and left in place for prolonged periods, particularly in the elderly or children with larger burns and SHOULD BE AVOIDED ANZBA Consensus Statement· Firat aid and the use of Hydrogel&, Aug 2021



Appendix K - Primary burn wound care guidelines - adults



A. Emergency Management
Refer RAH first aid and emergency management guidelines

B. Bum AaMument
- cause of bum
- First aid (type & length)
- Depth
- %totalbody surface area (TBSA)
- Site of bum
- ventilation
- Need for transfer/ consultation* to RAH Burns Unit

C.Transfer to RAH Burns Unit
Anticipated time to arrivalat RAH
<1hour
- Face- wet soaks
- Other burn areas- cling film

1 4 hours
- Face- wet soaks
- Other burn areas- cling film

4-24 hours
- Face- soft paraffin
- Other burn areas- Jelonet ™, Bactegras ™or !nadine ™

>24 hours
- Face- soft paraffin
- Other burn areas- Jelonet™, Bactegras ™or Inadine ™
- Actlcoat™ following discussion w h receiving clinician (temporary skin staining arising from Act1coat ™ applicalion may make luture burn assessmenl / Biobrane application diff icult).

For any chemical injuries please contact the Burns Registrar through RAH switchboard (08) 70740000 for advice

D.Minor Burn for local management
Follow RAH dressing guidelines for minor burn management – Appendix F

Appendix P- Hydrofluoric acid burn treatment protocol (burns <2°/o TBSA and HF concentration <10%)

Copious irrigation for 30-120 mins to remove H'ion effect (burn).
Ends with patient's subjective cessation of 'burning' sensation.
If Diphoterine/Hexafluorine available,follow local guidelines

+

Apply calcium gluconate 2.5% gel to skin of entire burn area. Wash and reapply every 30 mins until resolution of pain, then every 4 hours
Clinician to wear double gloves

If primary survey passed,transport to RAH, if not consult nearest Trauma Centre

No deep tissue discomfort — Burns Unit Consultant and toxicobgy consult — Deep tissue discomfort (aching/ pain subcutaneously)

Wash and reapply gel every 30mins until cessation of pain,then every 4 hours consider removal of nails and application of gel to nail bed if affected.

Contact Burns consultant regarding further treatment
(08) 7074 5076

No deep tissue discomfort develops

Burns Unit admission overnight for observation

Appendix Q - Hydrofluoric acid burn treatment protocol (burns >2% TBSA and HF concentration >10%)

Patient at risk of systemic fluoride poisoning

Immediate Burns and Toxicology consultation

!

Local burn management as per protocol for <2% TBSA flow chart

!

VBG or ABG (check Ca^{2+}, K^+) MBA 20 and Mg - ECG

Patient stable and investigations normal

1

Six hourly ECG and venous gas
Twice daily MBA 20

Patient unstable or investigations abnormal

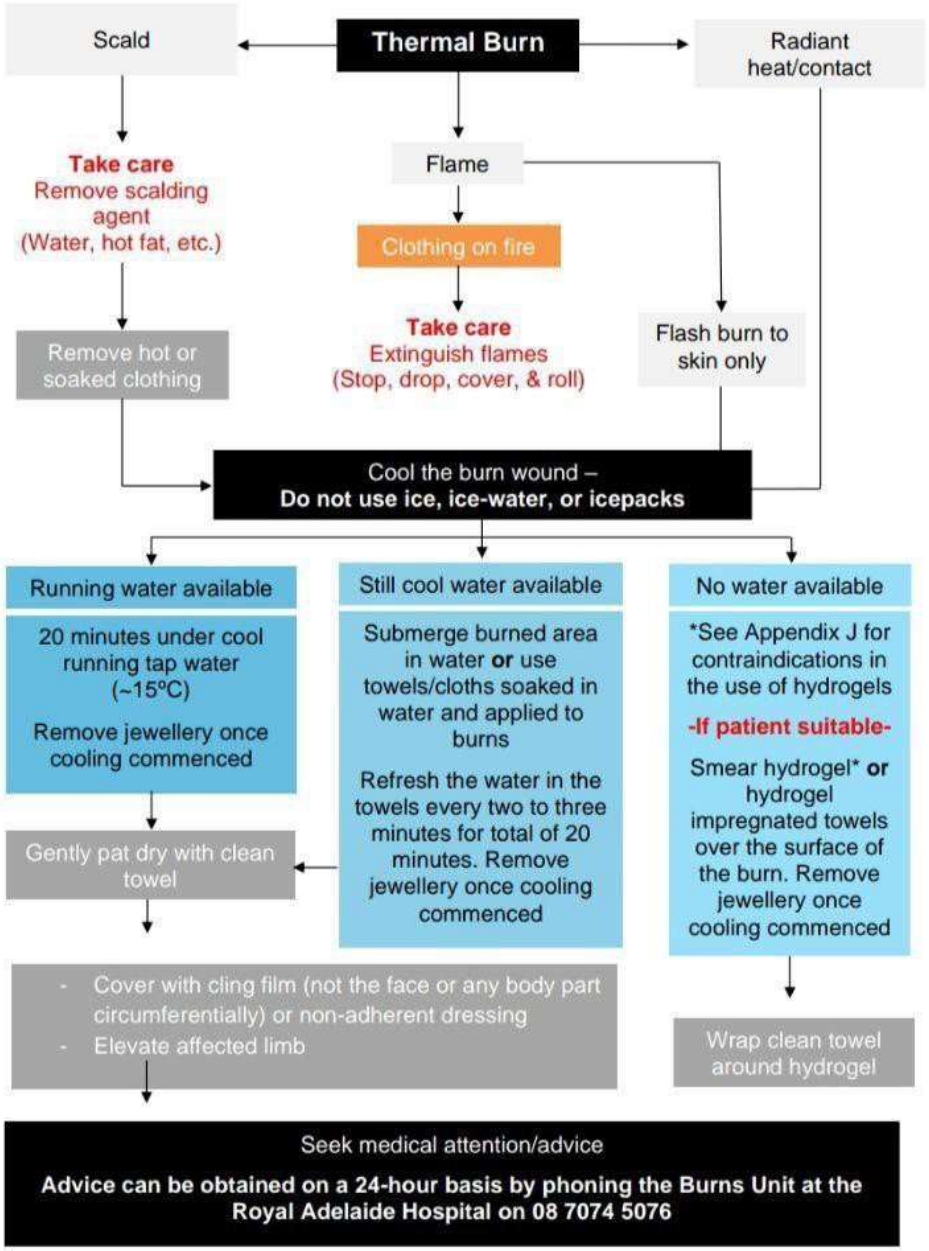
1

HDU/ICU

Aggressive replacement of Ca^{2+} and Mg^{2+}

Hourly VGB/ABG
Six hourly ECG, MBA 20 and Mg^{2+}

Appendix A – Community first aid protocol for thermal injury



SKIN TUMORS

Heng Monirath, Leng Sovannara

I. DEFINITION

Skin tumor are divided to 2 types:

- non-cancerous (benign) tumor of the skin is a growth or abnormal area on the skin that does not spread (metastasize) to other parts of the body.
- Skin cancer is the out-of-control growth of abnormal cells in the epidermis, the outermost skin layer, caused by unrepaired DNA damage that triggers mutations. These mutations lead the skin cells to multiply rapidly and form malignant tumors. The main types of skin cancer are Melanoma, Non - melanoma (Basal Cell Carcinoma (BCC), Squamous Cell Carcinoma (SCC)).

II. ETIOLOGY

- Benign skin tumor: Many benign skin growths are caused by years of exposure to the sun. Others run in families or appear to respond to hormonal changes. Pyogenic granulomas appear after damage to the skin from an injury.
- Skin cancer: The most commence are caused by the ultraviolet and radiation .

  The incidence of both non-melanoma and melanoma skin cancers has been increasing over the past decades. Currently, between 2 and 3 million non-melanoma skin cancers and 132,000 melanoma skin cancers occur globally each year. One in every three cancers diagnosed is a skin cancer and, according to Skin Cancer Foundation Statistics, one in every five Americans will develop skin cancer in their lifetime.

- Some individual risk factors for skin cancer
  - Fair skin
  - Blue, green or hazel eyes
  - Light-colored hair
  - Tendency to burn rather than suntan
  - History of severe sunburns
  - Many moles
  - Freckles
  - A family history of skin cancer

1. Bening skin tumor

Acrochordons

Acrochordons (skin tags) are extremely common, small, and typically

pedunculated benign neoplasms. They consist of hyperplastic soft dermis and epidermis, and are usually skin colored or brownish. They are generally 2 to 5 mm in size, although they may become larger. The most common locations are in skin folds (e.g., neck, axillae, groin), where skin irritation can be a causative factor. They occur in 25% to 46% of adults and increase with age and during pregnancy.[5]Studies have found that acrochordons are associated with the metabolic syndrome (obesity, dyslipidemia, hypertension, insulin resistance, and elevated C-reactive protein levels). This suggests they may be viewed as cutaneous clues for cardiovascular disease.

Acrochordons (skin tags) in a patient with metabolic syndrome.

Diagnosis is based on the appearance and location of lesions. They must be differentiated from neurofibromas, seborrheic keratoses, and pedunculated nevi. There have been rare case reports of skin tags that were found to be basal or squamous cell carcinomas. Treatment consists of cryosurgery, electrodesiccation, or simple scissor or shave excision. Electrodesiccation causes less hypopigmentation than cryotherapy and is the preferred treatment in nonwhite patients. An ear speculum placed over a small lesion may be helpful in directing the freeze pattern during cryosurgery.

Sebaceous Hyperplasia

Sebaceous hyperplasia is a benign disorder of the sebaceous glands that is common in middle-aged or older adults. Lesions present as asymptomatic, discrete, soft, pale yellow, shiny bumps on the forehead or cheeks, or near hair follicles. They typically appear as an umbilicated dome with multiple lobules resembling a cauliflower. There may be single or multiple lesions, ranging from 1 to 4 mm in diameter. They have no clinical significance except for cosmesis. Histologically, lesions consist of enlarged mature lobules of sebocytes around a central duct. It is important to rule out basal cell carcinoma, which is generally red or pink and increasing in size. Inspection of any surface vessels will show a haphazard arrangement in basal cell carcinoma, whereas the vessels in sebaceous hyperplasia occur only between lobules.

Sebaceous hyperplasia on the forehead with the typical umbilicated, lobulated appearance without haphazard blood vessels. No treatment is required for sebaceous hyperplasia, although patients may request removal of lesions for cosmetic reasons or because of concerns about malignancy. Therapeutic options include cryosurgery, phototherapy, shave excision, laser ablation, electrodesiccation with curettage, chemical cautery, or oral isotretinoin for widespread lesions.

Lipomas

Lipomas are slow-growing, benign mesenchymal tumors enclosed by a thin fibrous capsule. They closely resemble normal fat and are the most common

type of soft tissue tumor. They are usually subcutaneous but may occur in any organ because they are mesenchymal. They are generally asymptomatic but may become irritated with trauma or produce local obstructive symptoms in the airway or gastrointestinal tract. Their prevalence is 1%.[9]

Lipomas must be clinically differentiated from other tumors. The primary differential diagnosis in a subcutaneous mass is a sebaceous cyst or abscess. Lipomas are soft, flesh-colored nodules that are easily moveable under the overlying skin. Sebaceous cysts are generally identifiable by a central punctum, and abscesses can be identified by the presence of warmth, redness, and pain. Ultrasonography is increasingly used to aid in the diagnosis of lipomas. High-frequency ultrasonography (greater than 20 MHz) can provide high-resolution images of subcutaneous tumors and surrounding structures.[10] The differential diagnosis of lipomas also includes liposarcomas; risk factors for malignancy are size greater than 10 cm, older age, rapid lesion growth, location on the thigh, and invasion into deeper tissue, such as nerve or bone, leading to a firm or fixed feeling on examination. Lesions concerning for malignancy should be imaged with computed tomography or contrast magnetic resonance imaging.[11]

Patients commonly present with cosmetic concerns or symptoms related to compression of surrounding tissue. A single incision or punch excision (for smaller lesions) will generally allow manual expression of the lipoma without difficulty when standard excision is not required.[12]

Keratoacanthomas

Keratoacanthomas are rapidly growing, squamo proliferative benign tumors that resemble squamous cell carcinoma. They begin as round, firm reddish or skin-colored papules that develop into dome-shaped nodules with a keratin-filled crater. They may grow to 1 to 2 cm over weeks or months. There is a slower involution phase over several months, leaving a scar if not excised early in its course. Keratoacanthomas generally occur later in life on sun-exposed areas, primarily the face, arms, and legs. They are attributed to sun exposure, cigarette smoking, human papillomavirus infection, genetic factors, trauma, and chemical carcinogens.

Typical dome-shaped, keratin-filled plug of a keratoacanthoma on the dorsal arm.There is long-standing controversy over whether keratoacanthomas are benign, spontaneously self-limited tumors or a variant of cutaneous squamous cell carcinoma that have the potential for metastasis.[13] Keratoacanthomas share histopathologic characteristics that make them difficult to distinguish from squamous cell carcinoma. Shave biopsy may be inadequate to distinguish the conditions, whereas punch biopsy may be adequate because it obtains deeper tissue. Because no clinical or pathologic features can reliably differentiate keratoacanthoma from squamous cell carcinoma, early simple

excision of lesions is recommended, with margins of 3 to 5 mm. Mohs micrographic surgery may be considered if tissue sparing is desired.[14]

Medical treatment (systemic retinoids or intralesional injections of methotrexate, fluorouracil, or bleomycin) is reserved for nonsurgical candidates, patients with multiple lesions, and those with lesions on inoperable sites.

Pyogenic Granulomas

Pyogenic granulomas are rapidly growing nodules that bleed easily. Their name is a misnomer, however, as these lesions are neither pyogenic nor granulomas. They are an acquired benign tumor often found on mucous membranes. They tend to occur on the head or neck, or at sites of previous penetrating trauma. They are common in infancy and childhood, and approximately 2% of women develop a mucosal lesion in the late first to second trimester of pregnancy.

Pyogenic granulomas are yellow to purplish, pulpy vascular lesions often surrounded by a scaly collarette. They are usually removed because of their rapid growth and tendency to bleed. The differential diagnosis includes Spitz nevi, amelanotic melanoma, and squamous or basal cell carcinoma. Treatment options include shave excision with electrodesiccation of the base, and laser ablation.

Dermatofibromas

Dermatofibromas result from idiopathic benign proliferation of fibroblasts. Generally located on the lower extremities, they may develop at any cutaneous site and range in size from 3 to 10 mm. They are four times more common in women, and most develop between 20 and 50 years of age. They are usually asymptomatic, although pruritus and tenderness can be present. Dermatofibromas appear gradually over months and may persist for years. Although multiple dermatofibromas may be present, large numbers (15 or more) are rare. A multiple eruptive variant occurs in only 0.3% of patients, many of whom are immunocompromised (classically, those with human immunodeficiency virus infection or systemic lupus erythematosus).

Diagnosis is based on the appearance of firm, raised, papules or nodules, ranging from tan to reddish brown. They tend to be darker at the center and fade to normal skin color at the margin. Dermatofibromas exhibit dimpling or retraction of the lesion beneath the skin with lateral compression.

A typical dermatofibroma located on a lower extremity (A). The same dermatofibroma exhibits the dimple sign (B), a depression of the entire lesion (secondary to the dermal scarring seen as the major pathologic change) on lateral compression of the lesion with the thumb and index finger.

No treatment is required unless there is a change in size or color, or bleeding or irritation from trauma. A 2012 study found that 73% of patients who underwent laser ablation reported satisfaction with the results.

Epidermal Inclusion Cysts

Epidermal inclusion cysts are the most common type of cutaneous cyst. They are discrete nodules resulting from the implantation and proliferation of epidermal elements within the dermis. However, they display no sebaceous component and are not truly sebaceous cysts. They typically present on the head, neck, or trunk, and may remain stable or enlarge over time. Spontaneous inflammation and rupture can occur, with significant involvement of surrounding tissue. There is no way to predict which lesions will remain quiescent or become larger or inflamed. Infected cysts tend to be larger, more erythematous, and more painful than sterile inflamed cysts.

Multiple epidermal inclusion cysts are associated with Gardner syndrome, an autosomal dominant condition associated with colon cancer. Cysts that are unusual in number or location (e.g., fingers, toes) warrant screening for colon cancer. Malignancies (e.g., basal cell carcinoma, Bowen disease, squamous cell carcinoma, mycosis fungoides, melanoma in situ) can develop in cysts, but this is rare.

Diagnosis of epidermal inclusion cysts is based on appearance and palpation of a discrete, freely movable cyst or nodule. Careful inspection often reveals a central punctum. Treatment is unnecessary unless desired by the patient, and can be accomplished via simple excision with removal of the cyst and cyst wall. Inflamed or ruptured cysts often resolve spontaneously without therapy, although they tend to recur. Intralesional steroid injections can hasten resolution of inflamed cysts and should be followed by interval excision.23

Seborrheic Keratoses

Seborrheic keratoses are the most common benign epithelial tumor. They tend to be hereditary and occur after 30 years of age. They present as multiple, well-circumscribed, yellow to brown, raised lesions that feel slightly greasy, velvety, or warty and are described as having a “stuck-on” appearance

A well-circumscribed, yellow to brown, warty “stuck-on” seborrheic keratosis. The Leser-Trélat sign is the abrupt eruption of multiple seborrheic keratosis lesions in a patient with an underlying malignancy, usually an adenocarcinoma of the stomach. This is a rare sign supported mainly by case reports, but should prompt consideration of a paraneoplastic disorder. Seborrheic keratoses may resolve with treatment of the malignancy, then reappear with its recurrence. Seborrheic keratoses generally do not require treatment unless they become irritated or the patient has cosmetic concerns. They may be treated with

electrodesiccation, laser ablation, curettage, cryosurgery, or shave excision if biopsy is required.

Cherry Angiomas

Cherry angiomas are extremely common lesions that tend to appear with increasing age. They usually occur as multiple asymptomatic lesions, most commonly on the trunk and arms. They are dome-shaped, small (0.1 to 0.5 cm in diameter), bright red to violaceous, soft, compressible papules with smooth surfaces that blanch with pressure and bleed profusely with traumatic rupture. They can be treated effectively with electrodesiccation or laser ablation.

Table 1. Comparison of Common Benign Skin Tumors

Condition	Characteristics	Differential diagnosis	Treatment	Comments	Precautions and referral criteria
Acrochordon	Skin-colored to brown papules on narrow stalk	Senescent intradermal nevus	Cryosurgery, electrodesiccation, scissor or shave excision	Do not send multiple specimens in same jar	Cryosurgery should be performed with caution in persons with darker skin; refer patients with eyelid involvement
Cherry angioma	Dome-shaped, small, bright red to violaceous, soft, compressible papules	Pyogenic granuloma	Electrodesiccation, laser ablation	Numerous lesions (hundreds) and early onset can occur in Fabry disease	Genetic evaluation for Fabry disease in patients with multiple lesions
Dermatofibroma	Firm, raised, tan to reddish-brown papules or nodules; dimpling with lateral compression	Cellular dermatofibroma, dermatofibrosarcoma protuberans	Cryosurgery, intralesional steroid injection, laser ablation, punch excision	Abrupt appearance of multiple lesions may occur in persons with human immunodeficiency virus infection or systemic lupus erythematosus	Refer patients with cellular variant and dermatofibrosarcoma protuberans (deep invasion and metastases)
Epidermal inclusion cyst	Firm, mobile, subcutaneous nodule with central punctum; painless (unless inflamed)	Lipoma, abscess (vs. inflamed cyst)	Excision, intralesional steroid injection with interval excision for inflamed cysts	Presence of punctum helps differentiate cysts from lipomas; history helps differentiate between inflamed cyst and abscess (acute)	Inflamed cysts and those that have undergone previous incision and drainage can be more difficult to excise; refer patients with facial cysts
Keratoacanthoma	Rapidly growing, dome-shaped hyperkeratotic papule on sun-damaged skin	Squamous cell carcinoma, verruca, hypertrophic actinic keratosis	Excision, intralesional injection (methotrexate, fluorouracil, bleomycin), Mohs micrographic surgery	Cannot be histologically differentiated from squamous cell carcinoma	Refer patients with recurrence after complete excision
Lipoma	Soft, mobile subcutaneous nodules	Epidermal inclusion cyst, liposarcoma, deep hemangioma	Incision or punch excision and manual expression	Ultrasonography can help differentiate lipomas from other deep neoplasms	Use caution with facial lipomas and recurrent lesions after excision
Pyogenic granuloma	Rapidly growing, yellow to violaceous, friable nodule, often surrounded by scaly collarette	Amelanotic melanoma, Spitz nevus, basal cell carcinoma, squamous cell carcinoma	Laser ablation; shave excision with electrodesiccation of base	Send for histologic evaluation to rule out melanoma	Refer patients with recurrent lesions or facial lesions
Sebaceous hyperplasia	Dome-shaped papule with central umbilication and uniform yellow lobules on magnification	Basal cell carcinoma	Chemical cautery, cryosurgery, electrodesiccation, laser ablation, oral isotretinoin, photodynamic therapy, shave excision	Thin shave biopsy can rule out basal cell carcinoma	Basal cell carcinoma is generally red or pink and increases in size
Seborrheic keratosis	Well-circumscribed, yellow to brown, "stuck-on" papules and plaques	Atypical nevus, melanoma	Cryosurgery, curettage, electrodesiccation, laser ablation, shave excision	Consider malignancy workup for abrupt appearance of multiple lesions	Cryosurgery should be performed with caution in persons with darker skin

w/r/t: Removed or excised lesions should be sent for histologic evaluation, especially pyogenic granulomas, lesions with atypical presentations, and lesions that recur after reasonable treatment (e.g., cryosurgery).

Table summary of benign skin tumor

Clinical subtypes of basal cell carcinoma

Subtypes of BCC	Clinical characteristics
Nodular	Glossy pearly papules or nodules with smooth surface and curled edges, dendritic capillaries, occurring on the head and neck (46-48)
Superficial	Well-defined and erythematous thin patches or plaques with scales, clear in the center and thinning at the edges. Common in the trunk area (48)
Micronodular	Erythema or thin papules/plaques
Infiltrative	Poorly defined, sclerotic, flat or depressed plaques that are white, yellow or pale pink and may be covered with crusts, erosions, ulcers or papules
Morpheaform	Infiltrative plaques with faint borders and shiny surfaces, commonly on the head and neck
Infundibulocystic	Well-defined pearly papules on the head and neck are common in the elderly
Fibroepithelial	Sessile patches of skin color or erythema or pedunculated papules with a predilection for the trunk (49)
Basosquamous	Most found on the head and neck (50)

BCC, basal cell carcinoma.

2. Malignancy of skin tumor

The diagnosis of the malignancy of skin tumor is result of histology.

a. Basal Cell Carcinoma

Low- and high-risk features of basal cell carcinomas

Grading criteria	Features[1]	Low risk BCC	High risk BCC
Clinical	Forms	Primary	Recurrent, metastatic
	Immune status	Immunocompetent	Immunosuppressed
	Anatomic location	Area L and M	Area H
	Radiotherapy	No	Yes
	Tumor boundaries	Well-defined	Poorly defined
	Tumor dimensions	Surface area[2]: area L, <20 mm; area M, <10 mm	Surface area[2]: area L, >20 mm; area M, >10 mm
		Size/diameter: <5 cm	Size/diameter: >5 cm
	Involvement of specified nerves	Absent	Present
Pathologic	Histologic type/growth pattern	Superficial, nodular, keratotic infundibulocystic, fibroepithelioma of Pinkus	Micronodular, infiltrative, sclerosing morpheaform, basosquamous, metatypical/sarcomatoid
	Perineural invasion	Absent	Present, diameter of involved nerve ≥0.1 mm, multifocality, involvement of named nerves

[1], features as defined by the National Comprehensive Cancer Network; [2], human skin is divided into three zones according to the risk of invasive keratinocyte carcinoma: area H is a high-risk zone (frontal hairline, central face, nose, eyelids, chin, ears, genitalia, hands, feet, and bald scalp); area M is a medium-risk zone (cheeks, forehead, scalp, neck, and jawline); and area L is a low-risk zone (trunk and extremities, excluding areas H and M). BCC, basal cell carcinoma.

Treatment options for basal cell carcinoma

Treatment	Indications	Advantages	Disadvantages	Histological assessment of excised tissue/margins
Surgical excision	Primary treatment option for small, well-defined tumors or large, low-risk tumors in low-risk areas	High cure rates	Potential for recurrence due to incomplete excision; contraindicated or impractical for some patients	Postoperative analysis
Mohs surgery	Treatment of choice for large, high-risk, recurrent, and facial tumors	Very high cure rate; preserves healthy tissue and minimizes scarring	Time-consuming procedure requiring specialized training	Intraoperative complete en face margin evaluation
Curettage and electrodessication	Small, low-risk, primary tumors	Inexpensive and fast procedure	Wounds heal slowly and cause scarring; high rate of recurrence with high-risk and recurrent tumors	Not assessed after treatment
Radiotherapy	Patients in whom surgery is contraindicated or impractical or based on patient considerations	Noninvasive, painless	Low cure rates vs (Mohs) surgery; generally reserved for older patients due to potential toxicities	Not applicable
Field therapies (topical 5-fluorouracil, imiquimod, photodynamic therapy)	Patients with low-risk, shallow, or superficial tumors contraindicated for surgery and radiotherapy	Typically, good cosmetic outcomes; inexpensive	Lower cure rates vs surgery and radiotherapy	Not applicable
HPI therapy	Adults with laBCC that is recurrent or who are not candidates for surgery and radiation or with mBCC	Efficacious in patients with advanced BCC	Not well tolerated in some patients due to low-grade AEs	Not applicable

Abbreviations: AE = adverse event, BCC = basal cell carcinoma, HPI = hedgehog pathway inhibitor, laBCC = locally advanced BCC, mBCC = metastatic BCC.

b. Squamous Cell Carcinoma

Cutaneous squamous-cell carcinoma can develop on any surface of the skin. It is more common in men than in women (3:1 ratio), and the risk increases dramatically with age. Specifically, the incidence among persons older than 75 years of age is 5 to 10 times that among those younger than 55 years of age. Patients typically present with scaly, erythematous, or bleeding

lesions, most often on sun- exposed areas, and the appearance of these lesions differs according to histologic subtype.

Table 1. Tumor Staging and Risk Factors for Cutaneous Squamous-Cell Carcinoma.

Tumor Stage	Staging System*				
	AJCC	BWH	Salamanca Refinement	Tübingen	NCCN
Low risk					
T1	Tumor diameter <2 cm	0 risk factors	Tumor diameter <2 cm	Tumor diameter ≤2 cm, tumor thickness ≤6 mm	Tumor diameter ≤2 cm on trunk and arms and legs; well-defined; primary tumor, well or moderately differentiated, depth ≤6 mm
T2	Tumor diameter ≥2 cm and <4 cm		Tumor diameter ≥2 cm and <4 cm		
T2a		1 risk factor			
High risk					
T2b		2 or 3 risk factors			**High risk**: tumor diameter >2 cm and ≤4 cm on trunk and arms and legs; location on head, neck, hands, or feet, pretibial area, or anogenital areas, regardless of diameter; poorly differentiated, recurrent, immunosuppression, site of prior radiation therapy or chronic inflammatory process, rapid growth, neurologic symptoms, perineural involvement. **Very high risk**: tumor diameter >4 cm at any location, poor differentiation, desmoplastic squamous-cell carcinoma, depth >6 mm or invasion below subcutaneous fat, perineural invasion of a nerve lying below dermis or measuring ≥0.1 mm, lymphatic or vascular invasion
T3	Tumor diameter ≥4 cm or minor bone erosion, perineural invasion, or deep invasion	≥4 high-risk factors or bone invasion			
T3a			Tumor thickness >6 mm (with no invasion beyond subcutaneous fat), with or without tumor diameter ≥4 cm		
T3b			Invasion beyond subcutaneous fat or perineural invasion		
T3c			Combination of both T3b risk factors or AJCC T3 definition with ≥3 risk factors		
T4a	Tumor with gross cortical bone or marrow invasion				
T4b	Tumor invading skull bone or involving skull base foramen				

* The American Joint Committee on Cancer (AJCC) *Cancer Staging Manual*, 8th edition, defines deep invasion as invasion beyond subcutaneous fat or at a depth of more than 6 mm. Perineural invasion is defined as invasion in nerves that are 0.1 mm or more in diameter, invasion that is deeper than the dermis, or clinical and radiologic involvement of affected nerves, without involvement or invasion of the base of the cranium. The Brigham and Women's Hospital (BWH) staging system defines high-risk tumors as having a diameter of 2 cm or greater, poorly differentiated histologic features, perineural invasion of 0.1 mm or more, or tumor invasion beyond subcutaneous fat (excluding bone invasion, which automatically upgrades the tumor to stage T3). NCCN denotes National Comprehensive Cancer Network.

Tumor Staging and Risk Factors for Cutaneous Squamous-Cell Carcinoma

Table 2. Guideline-Based Management and Risk-Adjusted Follow-up Recommendations for Primary Cutaneous Squamous-Cell Carcinoma.*

Risk Level	Surgery	Workup	Radiation Therapy	Systemic Therapy	Follow-Up
Low	Curettage and electrodesiccation (excluding terminal hair-bearing areas), wide local excision (margins, 4–6 mm), Mohs micrographic surgery	Clinical lymph-node evaluation	For patients who are not surgical candidates or who have positive margins	For patients who are not surgical candidates	Skin self-examination monthly; full-body skin examination every 3–12 mo for 2 yr, then every 6–12 mo for 3 yr, then annually
High	Mohs micrographic surgery or PDEMA preferred, wide local excision (margins, 5–10 mm)	Consideration of nodal staging (imaging) for BWH T2b or T3 tumors or AJCC T3 or higher-grade tumors	For patients who are not surgical candidates or who have positive margins, recurrent disease, or extensive or named-nerve perineural invasion	Multidisciplinary consideration of systemic therapy if there is residual disease after resection or if surgery is not feasible	Skin self-examination monthly; full-body skin examination plus clinical nodal examination every 3–6 mo for 2 yr, then every 6–12 mo for 3 yr, then annually
Very high	Mohs micrographic surgery or PDEMA preferred, wide local excision (margins, 5–10 mm)	Consideration of nodal staging (imaging or sentinel lymph-node biopsy)	For patients who are not surgical candidates or who have positive margins	Multidisciplinary consideration of systemic therapy alone in complicated cases of locally advanced squamous-cell carcinoma in which curative surgery or radiation therapy is not feasible	Skin self-examination monthly; full-body skin examination plus clinical nodal examination every 3–6 mo for 2 yr, then every 6 mo for 3 yr, then every 6–12 months

* PDEMA denotes peripheral and deep exhaustive margin assessment.

Guideline-Based Management and Risk-Adjusted Follow-up Recommendations for Primary Cutaneous Squamous-Cell Carcinoma.

c. Melanoma

Staging a melanoma provides a description of its size and whether it has spread to other parts of the body. The stage of your melanoma helps to guide your treatment. There are three ways to stage a melanoma, and all patients will undergo at least two of these:

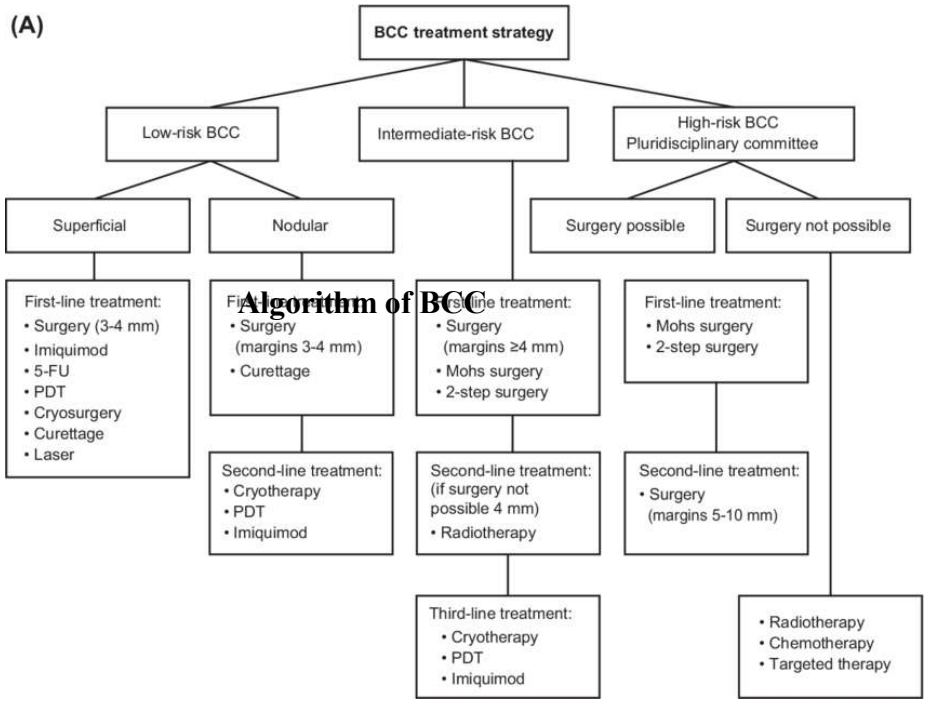
- **Microstaging** – A histopathologist looks closely at tissue under a microscope using special dyes and techniques to look for specific features that will help inform how far the disease has spread.
- **Clinical staging** – **The** lymph node groups that relate to the location of the melanoma are carefully examined to for any evidence of spread, usually seen by enlarged lymph nodes.
- **Staging after investigation** – This involves the use of imaging scans to see inside the body, including CT, MRI, and PET scans.

Stage	Definition	Main treatment options
In situ **Stage 0**	At this stage the tumour is confined to the cells in the top layer (epidermis) of the skin. The melanoma has not invaded deeper layers (dermis).	Surgical removal (wide local excision) is the main treatment.
Stage I	A Stage I melanoma can be up to 2 mm in thickness without ulceration; or up to 1 mm in thickness with ulceration.	Surgical removal is the main treatment. Sentinel lymph node biopsy (removal of nearby lymph nodes) may be considered to look for spread of melanoma to lymph nodes. It may be recommended if your melanoma is less than 1 mm in Breslow thickness or in some patients whose melanoma shows other adverse prognostic factors.
Stage II	Stage II melanoma is defined by thickness and ulceration. Tumours thicker than 2 mm with or without ulceration, and tumours between 1–2 mm with ulceration.	Surgical removal is the main treatment and sentinel lymph node biopsy may be considered to look for spread of melanoma to lymph nodes.
Stage III	A Stage III melanoma can be any thickness and has spread to nearby lymph nodes or tissues.	Surgical removal is the main treatment. Lymph node dissection (removal of all lymph nodes within the region concerned), drug and radiation therapies may be considered.
Stage IV	Stage IV melanoma can be any thickness, with spread (metastases) to distant lymph nodes or to distant sites (e.g. lung, liver, brain, bone).	Surgery or systemic (drug) therapies including immunotherapy and targeted therapy may be recommended. Radiation therapy may also be used.

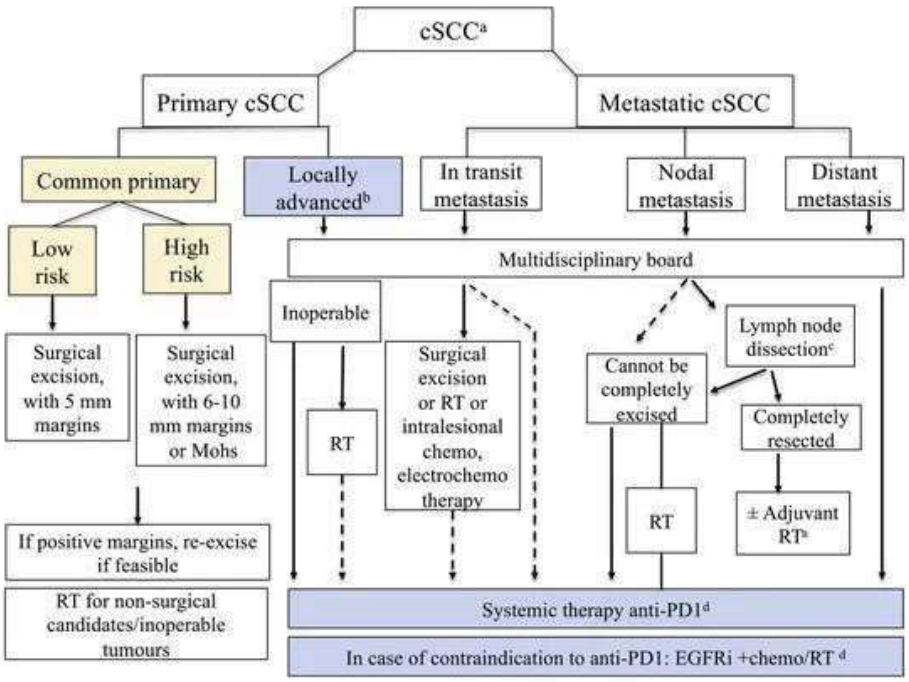
Management of melanoma

Stages	Definition	Main Treatment Options
In situ Stage 0	At this stage the tumour is confined to the cells in the top layer (epidermis) of the skin. The melanoma has not invaded deeper layers (dermis).	Surgical removal (wide local excision) is the main treatment.
Stage I	A Stage I melanoma can be up to 2 mm in thickness without ulceration; or up to 1 mm in thickness with ulceration.	Surgical removal is the main treatment. Sentinel lymph node biopsy (removal of nearby lymph nodes) may be considered to look for spread of melanoma to lymph nodes. It may be recommended if your melanoma is less than 1 mm in Breslow thickness or in some patients whose melanoma shows other adverse prognostic factors.
Stage II	Stage II melanoma is defined by thickness and ulceration. Tumours thicker than 2 mm with or without ulceration, and tumours between 1–2 mm with ulceration.	Surgical removal is the main treatment and sentinel lymph node biopsy may be considered to look for spread of melanoma to lymph nodes.
Stage III	A Stage III melanoma can be any thickness and has spread to nearby lymph nodes or tissues.	Surgical removal is the main treatment. Lymph node dissection (removal of all lymph nodes within the region concerned), drug and radiation therapies may be considered.
Stage IV	Stage IV melanoma can be any thickness, with spread (metastases) to distant lymph nodes or to distant sites (e.g. lung, liver, brain, bone).	Surgery or systemic (drug) therapies including immunotherapy and targeted therapy may be recommended. Radiation therapy may also be used.

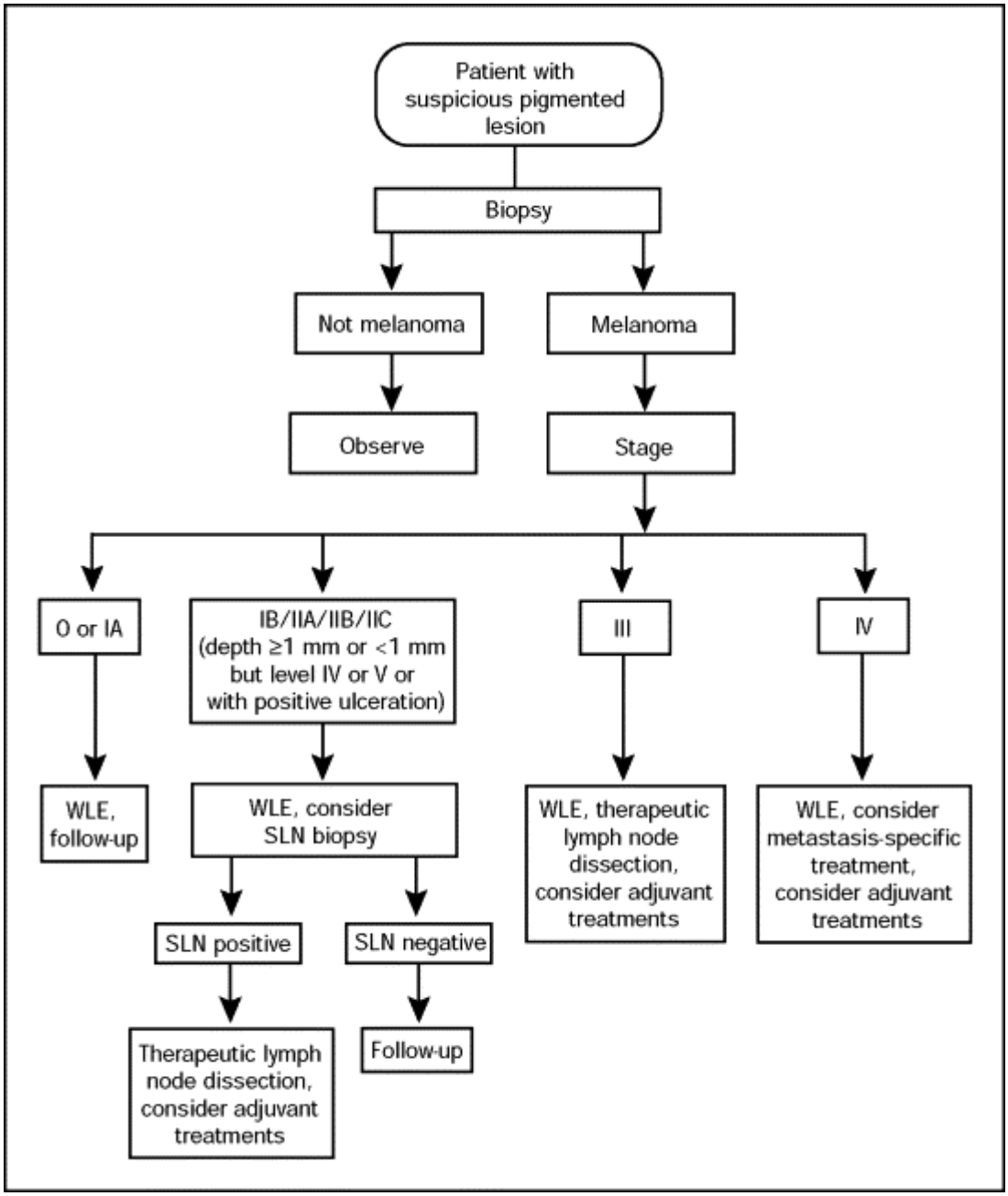
III. ALGORITHM



Algorithm of BCC



Algorithm of SCC treatment



Algorithm of melanoma treatment

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8. Dr. Wysong can be contacted at ashley.wysong@unmc.edu or at the Department of Dermatology, 985645 Nebraska Medical Center, Omaha, NE 68198.

SOFT TISSUE INFECTION

KOEUT Kundara, MOK Theavy, Ky Chanmonyraksmey, Tep Borin, RY Sina, HENG Monyroth

I. DEFINITION

Soft Tissue Infections (STIs) are variety of pathological that involve underlying subcutaneous tissue, fascia, or muscle, ranging from simple superficial infections to severe necrotizing infections. STIs may affect any part of the body and are a frequent clinical problem in surgical departments.

Soft Tissue Infections (STIs) are classified necrotizing or non-necrotizing character of the infection, the anatomical extension, anatomic location, causative pathogen(s), rate of progression, depth of infection, and severity of clinical presentation. the characteristics of the infection (purulent or not purulent).

Simple abscess

2.1 Cutaneous abscesses are collections of pus within the dermis and deeper tissues. (induration and erythema only to a defined area of the abscess and should not have extension into deeper tissues or multiloculated extension).

2.2 Epidermoid cysts, often named "sebaceous cysts," are due to infection of pilosebaceous gland and are present anywhere in the body lined by squamous epithelium.

2.3 Furuncles are superficial infections with suppuration of the hair follicle, usually caused by *S. aureus*. They extend through the dermis into the subcutaneous tissue, where form a small abscess. Furuncles can occur anywhere on hairy skin. A group of infected hair follicles with pus is named Carbuncle.

2.4 Carbuncles are larger and deeper than furuncles. Furuncles often rupture and drain spontaneously or following treatment with moist heat.
A recurrent abscess at a site of previous infection may be caused by local causes such as a pilonidal cyst, hidradenitis suppurativa, or foreign material. Therefore, it always requires research of a local cause.

2.5 Treatment
Incision and drainage

Antibiotic therapy only in selected patients for 5 days. You may extend therapy up to 7–10 days if lack of symptom resolution at 5 days.
Incision, evacuation of pus and debris, and probing of the cavity to break up loculations provide effective treatment of cutaneous abscesses. **The pus and debris must be sent to lab for culture and antibiogram**. The resultant wound should be left open and lightly packed with roll gage soaked with antiseptic solution.
Antibiotic therapy should be prescribed for abscesses greater than 5 cm, in an area difficult to drain (e.g., face, hand, and genitalia), if there is lack of response to incision and drainage alone, if there are multiple localizations and in patients with immunosuppression.
Empiric antibiotic regimens. Normal renal function

Target Pathogens: *S.aureus* and streptococci.

- One of the following oral antibiotics
  - Chloramphenicol: 12.5 mg per/kg every six hours.
  - Trimethoprim and Sulfamethoxazole 160/800 mg every 12 h
  - Minocycline 100 mg every 12 h
  - Doxycycline 100 mg every 12 h
  - Amoxicillin-clavulanate 1 g every 8 h
  - Cephalexin 500 mg every 6 h
- In patients at risk for CA-MRSA including immunocompromised status, personal or household contact with MRSA infection or colonization in the past 12 months, with prior antibiotic use for 5 days during the last 90 days or who do not respond to first-line therapy add one of the following oral antibiotics

  or
  - In patients with beta-lactam allergy
  - Clindamycin 300 mg every 8 h

In cases of recurrent skin abscess, it is necessary to look for the presence of foreign materials and identify and correct local factors that may cause recurring infection. For recurrent skin abscess bacterial cultureand antibiograme testing must be performed to verify the causative bacteria and antibiotics susceptibility to define a targeted therapy.

If an abscess is treated with prolonged antibiotics without drainage, it can lead to formation of sterile pus surrounded by thick fibrous tissue. It makes a hard lump that sometimes mimics malignancy. The treatment is surgical drainage with excision of fibrous wall.

1. Erysipelas

Erysipelas is a skin infection involving the dermis layer of the skin, but it may also extend to the superficial cutaneous lymphatics, a fiery red, tender, painful plaque with well-demarcated edges and is commonly caused by *Streptococcus spp.*, usually *S. pyogenes*. *S. aureus* rarely causes erysipelas.

Erysipelas is distinguished clinically from cellulitis by the following two features [1]:

- In erysipelas, the lesions are raised above the level of the surrounding skin, and
- Erysipelas is characterized by a clear line of demarcation between involved and uninvolved tissue.

Streptococci are the primary cause. The role of *S. aureus*, and specifically MRSA, remains controversial.

- Target Pathogens: *S. aureus* and streptococci, CA-MRSA is unusual.

Treatment

- Antibiotic therapy for 5 days. You may extend therapy up to 10 days if lack of symptom resolution at 5 days
- Use intravenous antibiotics if signs of systemic inflammation
- One of the following oral antibiotics:
  - Chloramphenicol: 12.5 mg per/kg every six hours.
  - Trimethoprim and Sulfamethoxazole 160/800 mg every 12 h
  - Minocycline 100 mg every 12 h
  - Doxycycline 100 mg every 12 h
  - Amoxicillin-clavulanate 1 g every 8 h
  - Cephalexin 500 mg every 6 h
  - In patients at risk for CA-MRSA including immunocompromised status, personal or household contact with MRSA infection or colonization in the past 12 months, with prior antibiotic use for 5 days during the last 90 days or who do not respond to first-line therapy add one of the following oral antibiotics

    or
  - In patients with beta-lactam allergy
  - Clindamycin 300 mg every 8 h

or

Inpatient therapy

One of following intravenous antibiotics:

- Chloramphenicol: 12.5 mg per/kg every six hours.
- Vancomycin 25–30 mg/kg loading dose then 15–20 mg/kg/dose every 12 h
- Linezolid 600 mg every 12 h
- Cefazolin 2 g every 8 h
- Amoxicillin-clavulanate 1.2/2.2 gr every 8 h

or

- In patients at risk for CA-MRSA including critically ill and immunocompromised status, personal or household contact with MRSA infection or colonization in the past 12 months, with prior antibiotic use for 5 days during the last 90 days or who do not respond to first- line therapy add one of following intravenous antibiotics

Because of their very low yield, blood cultures are not helpful in managing erysipelas, unless it is particularly severe. Culture by aspiration or punch biopsy is not recommended as principle for identifying the causative bacteria in typical erysipelas patients. However, in several cases including immunosuppressed patients or those with

neutropenia, streptococci or *S. aureus* are not the causative bacteria. For these cases, lesion aspiration, or punch biopsy may be helpful to identify the causative bacteria and define a targeted therapy.

2. Cellulitis

Cellulitis is an acute bacterial infection primarily of the dermal lymphatics and the subcutaneous tissue that most commonly affects the lower extremities, although it can affect other areas. It causes local signs of inflammation, such as warmth, erythema, pain, lymphangitis, and frequently systemic upset impact with fever and raised white blood cell count. Outpatient therapy should be recommended for patients with adherence to therapy, who do not have general signs of inflammation or hemodynamic instability.

Patients with a previous attack of cellulitis, especially involving the legs, can present recurrences. The infection usually occurs in the same area as the previous episode. oedema, especially lymphedema, venous insufficiency, prior trauma (including surgery) to the area, and tinea pedis can increase the frequency of recurrences. Addressing these factors may decrease the frequency of recurrences.

The pathogens involved are streptococci and *S. aureus*. Cellulitis associated with abscesses is usually caused by *S. aureus*. In contrast, typical (non-purulent) cellulitis is most commonly caused by both streptococcal species and *S. aureus*. MRSA is an unusual cause of typical cellulitis.

In neutropenic and immunocompromised patients, Gram-negative bacteria should be considered.

Treatment

- Antibiotic therapy for 5 days or extend therapy up to 7-10 days if lack of symptom resolution at 5 days.
- Incision and drainage in purulent cellulitis and send pus to lab for culture and anti-biograme

2.1 Typical (non-purulent) cellulitis

Empiric antibiotic regimens. Normal renal function

- Target Pathogens: *S. aureus* and streptococci, CA-MRSA is unusual.

Outpatient therapy or step-down

- One of the following oral antibiotics
  - Chloramphenicol: 12.5 mg per/kg every six hours.
  - Trimethoprim and Sulfamethoxazole 160/800 mg every 12 h
  - Minocycline 100 mg every 12 h
  - Doxycycline 100 mg every 12 h
  - Amoxicillin-clavulanate 1 g every 8 h
  - Cephalexin 500 mg every 6 h
  - In patients at risk for CA-MRSA including immunocompromised status, personal or household contact with MRSA infection or colonization in the past 12 months, with prior antibiotic use for 5 days during the last 90 days

or who do not respond to first-line therapy add one of the following oral antibiotics

or

- In patients with beta-lactam allergy
- Clindamycin 300 mg every 8 h

or

Inpatient therapy

- One of following intravenous antibiotics
  - Chloramphenicol: 12.5 mg per/kg every six hours.
  - Vancomycin 25–30 mg/kg loading dose then 15–20 mg/kg/dose every 12 h
  - Linezolid 600 mg every 12 h
  - Cefazolin 2 g every 8 h
  - Amoxicillin-clavulanate 1.2/2.2 gr every 8 h

or

- In patients at risk for CA-MRSA including critically ill and immunocompromised status, personal or household contact with MRSA infection or colonization in the past 12 months, with prior antibiotic use for 5 days during the last 90 days, with cellulitis associated with penetrating trauma especially from illicit drug use or who do not respond to first-line therapy one of the following intravenous antibiotics

- In patients at risk for Gram-negative infections or severe forms who do not respond to first-line therapy consider.

Piperacillin/tazobactam 4,5 g every 6 h.

2.2 Purulent cellulitis

Incision and drainage are recommended as primary management for abscesses with associated cellulitis. In these cases, antibiotics is generally suggested.

Empiric antibiotic regimens. Normal renal function

- Target Pathogen: *S. aureus* including CA-MRSA.

Outpatient therapy or step-down

- One of the following oral antibiotics

  - Chloramphenicol: 12.5 mg per/kg every six hours.
  - Trimethoprim and Sulfamethoxazole 160/800 mg every 12 h
  - Minocycline 100 mg every 12 h
  - Doxycycline 100 mg every 12 h
  - Amoxicillin-clavulanate 1 g every 8 h
  - Cephalexin 500 mg every 6 h
  - In patients at risk for CA-MRSA including immunocompromised status, personal or household contact with MRSA infection or colonization in the past 12 months, with prior antibiotic use for 5 days during the last 90 days or who do not respond to first-line therapy add one of the following oral antibiotics
    - or
  - In patients with beta-lactam allergy
  - Clindamycin 300 mg every 8 h

or

Inpatient therapy

- One of following intravenous antibiotics
  - Chloramphenicol: 12.5 mg per/kg every six hours.
  - Vancomycin 25–30 mg/kg loading dose then 15–20 mg/kg/dose every 12 h
  - Linezolid 600 mg every 12 h
  - Cefazolin 2 g every 8 h
  - Amoxicillin-clavulanate 1.2/2.2 gr every 8 h

or

- In patients at risk for Gram-negative infections or severe forms who do not respond to first-line therapy consider
- Piperacillin/tazobactam 4,5 g every 6 h.

Culture is recommended as principle for identifying the causative bacteria in cellulitis patients. However, in immunosuppressed patients or those with neutropenia or in severe forms associated with systemic signs of inflammation or that do not respond to first-line therapy, culture may be helpful to identify the causative bacteria and define a targeted therapy.

Cellulitis in following situations can be life-threatening thus requiring early diagnosis and prompt intervention:

Orbital cellulitis: The infection usually spreads from paranasal sinuses and the patient presents with proptosis, chemosis, ophthalmoplegia and diminished vision due to pressure on optic nerve. Uncontrolled infection may have intra-cranial extension leading to cavernous sinus thrombosis and meningitis. Early intravenous empirical antibiotic therapy is required. Surgical drainage is required in progressive disease to prevent loss of vision.

Ludwigs angina: It is cellulitis of submandibular region occurring deep to deep cervical fascia leading to brawny induration in this region with edema of floor of the mouth. Untreated cases may have laryngeal edema and stridor. In case of no response to antibiotics, liberal fasciotomy of deep cervical fascia in the submandibular region is required.

3. Perianal and perirectal abscesses

Perianal and perirectal abscesses are typically well-circumscribed and respond to incision and drainage.Common sites of origin of complex abscesses may be perineal or perianal, perirectal. Antibiotic therapy should be used if systemic signs of infection are present, in immunocompromised patients, if source control is incomplete, or in cases of great abscess or with significant cellulitis. Empiric broad-spectrum antibiotic therapy with coverage of Gram-positive, Gram-negative, Klepsiella pheumoniae and anaerobic bacteria should be considered.

Diagnosis

In patients with perianal and perirectal abscesses, the diagnosis is often based on

clinical assessment and examination under anaesthesia. However, in some patients several imaging techniques may be useful.

1. EUS (anal endosonography)
2. CT

They should be evaluated according to the specific clinical scenario and the available technology and resources.

- Radiological studies are not usually needed to diagnose a complex abscess but can be useful in some special situations. The use of imaging techniques in perianal and perirectal abscesses could be helpful in all those cases with an atypical presentation (e.g., lower back pain, severe anal pain in the absence of a fissure, urinary retention), when the physical examination suggests a supra-elevator or inter-sphincteric abscess or when there is suspicion of perianal Crohn's disease or colonic source.

- MRI has high detection rates for anorectal abscesses [2], while there is debate around the sensitivity and specificity of (EUS). Some studies suggest that EUS is more accurate than MRI in detecting abscesses and evaluating complex fistulas, especially when suspected of arising from underlying Crohn's disease [3].

Treatment

- Incision and drainage + antibiotic therapy for 5 days in selected patients. You may extend therapy up to 7–10 days if lack of symptom resolution at 5 days.
- In perianal and perirectal abscesses identification of eventual fistula tract, and either proceed with primary fistulotomy to prevent recurrence (only in cases of low fistula not involving the sphincter muscle) or place a draining seton for future consideration. Fistulotomy can risk continence if too extensive and placement of seton should only be performed if the tract and openings are very clear, as there is risk of creating a false internal orifice and complicating the condition.

Empiric antibiotic regimens. Normal renal function

- Target Pathogen: Gram-positive, Gram-negative and Klepsiella pheumoniae

Outpatient therapy or step-down

- One of the following antibiotics
- Cefepime1 g every 8 h or
  - In patients with beta-lactam allergy
  - Ciprofloxacin 500 mg every 8 h + Metronidazole 500 mg every 8 h
  - In patients at risk for CA-MRSA or who do not respond to first-line therapy add one of following oral antibiotics
  - Minocycline 100 mg every 12 h
  - Trimethoprim and sulfamethoxazole 160/800–320/1600 mg every 12 h
  - Doxycycline 100 mg every 12 h or

Inpatient therapy

  - One of following intravenous antibiotics
  - Ceftriaxone 2 g every 24 h + Metronidazole 500 mg every 8 h
  - Cefotaxime 2 g every 8 h + Metronidazole 500 mg every 8 h
  - Piperacillin/tazobactam 4,5 g every 6 h

or

- In patients with beta-lactam allergy
- Ciprofloxacin 400 mg every 8 h + Metronidazole 500 mg every 8 h
- In patients at risk for CA-MRSA or who do not respond to first-line therapy add one of following intravenous antibiotics
- Vancomycin 25–30 mg/kg loading dose then 15–20 mg/kg/dose every 12 h
- Linezolid 600 mg every 12 h

Culture is recommended as principle for identifying the causative bacteria in perianal or perirectal abscess. However, in immunosuppressed patients or those with neutropenia or in severe forms associated with systemic signs of inflammation or that do not respond to first-line therapy, it may be helpful to identify the causative bacteria and define a targeted therapy.

4. Necrotizing infections

NSTIs are life-threatening, invasive, soft-tissue infections with a necrotizing component involving any or all layers of the soft-tissue compartment, from the superficial dermis and subcutaneous tissue to the deeper fascia and muscle.

The vicious cycle of fulminant infection, toxin production, cytokine activation, micro thrombosis and ischemia, tissue dysfunction and death, and in turn, greater dissemination of infection is central to the rapidly progressive necrosis seen in NSTIs and differentiates it from that of the other SSTIs.

NSTIs have been described according to their anatomical locations (i.e., Fournier's gangrene) and the depth of infections: dermal and subcutaneous components (necrotizing cellulitis), fascial component (necrotizing fasciitis), and muscular components (necrotizing myositis).

Diagnosis

Conditions associated with NSTIs include diabetes mellitus, renal insufficiency, arterial occlusive disease, intravenous drug abuse, body mass index (BMI) > 30 kg/m^2, age < 65 years, liver disease, immunosuppression also in patients having tuberculosis and viral infections, recent surgery and traumatic wounds or incision of the skin, including minor lesions like insect bites and injections sites.

Diabetic patients exhibit impaired wound healing and increased susceptibility to infection, which may affect the course of SSTIs. It is thus reasonable to speculate that this chronic, debilitating disease contributes to a more serious nature of NSTIs. Diabetes mellitus is the most common co-morbidity associated with NSTIs. Up to 44.5% of patients with this condition are diabetic. Patients with diabetes generally present with polymicrobial and have poorer outcomes. Delay in diagnosis and delay in treatment of these infections increase the risk of mortality.

The initial differential diagnosis between cellulitis and NSTI that requires prompt operative intervention may be difficult. Most cases of NSTI are initially diagnosed as cellulitis. However, timely diagnosis is critical since time to operative debridement is an important determinant of outcome in NSTIs.

Patients with NSTI usually present with severe pain, which is out of proportion to the physical findings:

Local signs

- Edema
- Erythema
- Severe and crescendo pain out of proportion
- Skin bullae or necrosis (at a later stage)
- Swelling or tenderness
- Crepitus

The triad of swelling, erythema, and disproportionately severe pain should raise the suspicion of NSTI.

Systemic signs

- Fever
- Tachycardia
- Hypotension
- Shock

Laboratory tests are not highly sensitive or specific for NSTIs. A rapidly progressive soft-tissue infection should be treated as a necrotizing infection from the beginning. The clinical picture may worsen very quickly, sometimes during a few hours. But debridement necrotizing infection tissue culture or blood culture and antibiograme must be don early as possible.

To predict the presence of NSTI, the Laboratory Risk Indicator for Necrotizing infection (LRINEC) score was proposed (Table 1). LRINEC score assigns points for abnormalities in six independent variables: serum C-reactive protein level (> 150 mg/L), white blood cell (WBC) count (> 15,000/μL), hemoglobin level (< 13.5 g/dL), serum sodium level (< 135 mmol/L), serum creatinine level (> 1.6 mg/dL [142 mmol/l]), and serum glucose level (> 180 mg/dL [10 mmol/l]). With a score of 8 or higher, there is a 75% risk of an NSTI.

Table 1 Lrinec score *From: WSES/GAIS/WSIS/SIS-E/AAST global clinical pathways for patients with skin and soft tissue infections*

Variable (units)	Score points
C-reactive protein (CRP) (mg/L)	
< 150	0
≥ 150	4
White blood cell count (per mm^3)	

Variable (units)	Score points
< 15	0
15–25	1
> 25	2
Hemoglobin (g/dl)	
> 13.6	0
11–13.5	1
< 10.9	2
Serum sodium (mmol/L)	
≥ 135	0
< 135	2
Serum creatinine (mg/dl)	0
≤ 1.6	0
> 1.6	2
Serum glucose (mg/dl)	
≤ 180	0
> 180	1

Subsequent evaluation of the LRINEC score has demonstrated conflicting results. Several studies have assessed the utility of LRINEC for the early diagnosis of necrotizing infections.

Imaging

The diagnosis of NSTIs is primarily clinical. However, radiologic imaging may be able to provide useful information when the diagnosis is uncertain. A plain X-ray should not be used to rule out NSTI. However, it is important that if clinical suspicion of NSTI is high, radiologic imaging must neither delay nor deter surgery, because in this setting an early surgical debridement is essential to decrease mortality.

- Ultrasound
- CT

- MRI

CT has a higher sensitivity than plain radiography in identifying early NSTIs. Findings consistent with necrotizing infections are fat stranding, fluid and gas collections that dissect along fascial planes, and gas in the involved soft tissues. Additionally, fascial thickening and non-enhancing fascia on contrast CT suggests fascial necrosis.

MRI has been considered the imaging modality of choice for necrotizing fasciitis. However, MRI may be difficult to perform under emergency conditions and is not recommended as the first-choice imaging technique.

Treatment

- Surgical source control as soon as possible within 6 h after admission. Delay in early surgical increases mortality.
- Appropriate and effective debridement techniques. Skin-sparing debridement techniques focusing on tissue *directly involved in necrosis and send to laboratory culture and antibiograme.*
- Re-explorations should be repeated until the time when very little or no debridement is required.
- Empiric antibiotic therapy optimizing Pharmacokinetics (PK) and Pharmacodynamics (PD) targets.
- Deep samples collected at the interface between healthy and necrotized tissues during initial debridement and blood cultures allow the identification of causative pathogens in most cases.
- De-escalation of antibiotic therapy be based on clinical improvement, cultured pathogens, and results of rapid diagnostic tests where available.
- Hyperbaric oxygen therapy where it is available
- Intravenous immunoglobulin (IVIG) in patients with streptococcal NSTIs

Early source control, antibiotic therapy, and (organ) supportive measures are the cornerstone of treatment in patients with sepsis or septic shock caused by NSTIs.

Early surgical debridement with complete removal of necrotic tissue, including potential major amputation is essential to decrease mortality and other complications in patients with NSTIs.

It is the most important determinant of outcome in patients with NSTIs and should be performed as soon as possible, but at least within the first 6 h after admission.

Scheduled re-explorations should be done at least every 12–24 h after the initial operation or sooner if clinical local or systemic signs of worsening infection become evident, as well as with worsening laboratory parameters (WBC count, C Reactive Protein and Procalcitonin). Re-explorations should be repeated until the time when very little or no debridement is required.

After debridement and once the wound is stable, the subsequent use of negative pressure therapy allows reduction of the wound surface, extraction of wound exudate and cell residues, as well as induction of granulation.

Gas gangrene (clostridial myonecrosis), or type III NSTI, is an acute infection by clostridium or bacillus of healthy living tissue that occurs spontaneously or as a result of traumatic injury.

Occasionally in immunocompromised patients, NSTIs may also be caused by mycotic species. Empiric coverage against fungi should be started in high-risk patients. Since it is impossible to exclude with certainty a polymicrobial NSTI, an aggressive broad-spectrum empiric antimicrobial therapy should initially be selected to cover Gram-positive, Gram-negative, and anaerobic organisms until culture-specific results and sensitivities are available. An acceptable empiric antibiotic regimen should always include antibiotics, which cover MRSA with the additional benefit of inhibiting invasive hamolytic streptococci virulence proteins.

Selection of antibiotics that inhibit toxin production may be helpful, particularly in those patients who have evidence of toxic shock syndrome (TSS), potentially present in patients who have streptococcal and staphylococcal infections. Protein cytotoxins, such as superantigens, play an important role in the pathogenesis of various staphylococcal and streptococcal infections, and toxin production should be considered when selecting an antimicrobial agent for Gram-positive pathogens. Linezolid and clindamycin play an important role because they may significantly inhibit exotoxin production from Gram-positive pathogens [4]. Culture-specific results and sensitivities can direct both broadening of antibiotic regimen if it is too narrow and a de- escalation if it is too broad particularly in critically ill patients where de-escalation strategy is one of the cornerstones of antimicrobial stewardship programs [4].

In the absence of definitive clinical trials, antibiotic therapy should be administered until further debridement is no longer necessary. Patients with NSTIs are very complex and may lose fluids, proteins, and electrolytes through a large surgical wound. In addition, hypotension is caused by vasodilation induced by the systemic inflammatory response syndrome to infection and cytotoxins [5]. Fluid resuscitation and analgesia are the mainstays of support for patients with advanced sepsis, usually combined with vasoactive amines associated with mechanical ventilation and other organ function support, if needed. No ideal fluid has been proven: however, resuscitation therapy must be prompt and immediate as in any type of shock.

- As soon as possible after diagnosing sepsis (organ dysfunctions) associated with a NSTI, administer a 1-L bolus of a balanced crystalloid solution over 30 min. In hypotensive patients or those with an elevated serum lactate level additional fluid should be administered to achieve 30 ml/kg of initial volume resuscitation. This should be administered within 3 h.
- In patients who do not achieve a MAP $\geq$ 65 mmHg with initial volume resuscitation within one hour, start a norepinephrine infusion and titrate as needed. This can initially be administered through a peripheral IV while central venous access is being obtained.
- Simultaneously, administer within 1-h broad spectrum antimicrobial agent(s) to cover potential pathogens.
- If the norepinephrine infusion increases to $\geq$ 15 mg/min, add low dose vasopressin at infusion rate of 0.03 U/min. Do not increase this dose of vasopressin.
- Start low dose steroids (hydrocortisone 50 mg q 6 h) in patients requiring ongoing high doses of norepinephrine and vasopressin to achieve MAP $\geq$ 65 mmHg.
- Additional fluid resuscitation (beyond the initial 30 ml/kg) will likely be needed but should be based on the assessment that the patient will be fluid responsive.

- Patients with impaired cardiac function should inotropic agent started. Dobutamine is the preferred agent but will cause hypotension in hypovolemic patients.

New agents to treat NSTIs

Reltecimod (previously known as AB103 or p2TA), a peptide derived from the T-cell receptor CD28, modulates the host immune response by targeting the co-stimulatory pathway, which is essential for the induction of multiple pro-inflammatory cytokines. Consequently, reltecimod has demonstrated beneficial effects against different bacterial infections such as NSTIs.

A randomized, double-blind, placebo-controlled trial of single dose reltecimod (0.5 mg/kg) administered within 6 h of NSTI diagnosis was recently published [6]. Reltecimod was associated with improved resolution of organ dysfunction and hospital discharge status. Further studies are warranted to establish the real efficacy in clinical practice.

Wound management after source control

The rapidly spreading infection followed by aggressive surgical intervention and repeated debridement creates challenges for wound management.

Negative pressure wound therapy (NPWT) refers to wound dressing systems that continuously or intermittently apply sub-atmospheric pressure to the surface of a wound. NPWT has become a popular treatment modality for the management of many acute and chronic wounds [3]. In the setting of necrotizing infections once the necrosis is removed, NPWT can help wound healing physiologically. The negative pressure leads to an increased blood supply, increasing tissue perfusion, reducing edema, absorbing fluids and exudates, inhibiting infection, and finally drying the wound and thus the migration of inflammatory cells into the wound. Additionally, it promotes and accelerates the formation of granulation tissue by the removal of bacterial contamination and exudates. A modification of the original system added intermittent automated instillation of topical wound irrigation solutions to traditional NPWT.

Although skin grafting may fulfill this role, techniques higher on the reconstructive ladder, including local, regional and free flaps, are sometimes undertaken [7].

Empiric antibiotic regimens. Normal renal function

The initial empirical antibiotic regimen should comprise broad-spectrum drugs, including anti-MRSA and anti-Gram-negative coverage. Antitoxin active antibiotics such as clindamycin or linezolid should be included in the empirical antibiotic regimen to treat NSTIs.

In stable patients

- One of the following antibiotics
- Amoxicillin/clavulanate 1.2/2.2 g every 8 h
- Ceftriaxone 2 g every 24 h + Metronidazole 500 mg every 8 h
- Cefotaxime 2 g every 8 h + Metronidazole 500 mg every 8 h
- +

- Clindamycin 600–900 mg every 8 h

In unstable patients

- One of the following antibiotics
- Piperacillin/tazobactam 4.5 g every 6 h
- Meropenem 1 g every 8 h
- Imipenem/Cilastatin 500 mg every 6 h
- +
- One of the following antibiotics
- Linezolid 600 mg every 12 h
- Tedizolid 200 mg every 24 h or
- Another anti-MRSA-antibiotic as
- Vancomycin 25–30 mg/kg loading dose then 15–20 mg/kg/dose every 8 h
- Daptomycin 6–8 mg/kg every 24 h *
- Telavancin 10 mg/kg every 24 h
- +
- Clindamycin 600–900 mg every 8 h

*Approved at the dosage of 4–6 mg/kg/24 h, it is currently used at higher dosages.

5. Fournier's gangrene

Fournier's gangrene (FG) is a severe type of NSTI involving the genital area and or perineum. The origin of the infection is identifiable in the majority of cases and is predominantly from anorectal, genito-urinary or local cutaneous sources. The aggressive nature of the infection requires early recognition and immediate surgical intervention.

Patients with FG usually present with severe pain, which is out of proportion to the physical findings

Diagnosis Local signs:

- edema.
- Erythema.
- Severe and crescendo pain out of proportion.
- Skin bullae or necrosis (at a later stage).
- Swelling or tenderness.
- Crepitus.

Systemic signs:

- Fever.
- Tachycardia.
- Hypotension.
- Shock.

Fournier's Gangrene severity index (FGSI) is a standard score for predicting outcome in patients with FG and is obtained from a combination of physiological parameters at admission, including temperature, heart rate, respiration rate, sodium, potassium, creatinine, leukocytes, haematocrit, and bicarbonate. An FGSI score above 9 has been demonstrated to be sensitive and specific as a mortality predictor in patients with Fournier's gangrene [8, 9, 10, 11] (Table 2).

Table 2 Fournier's Gangrene severity index

Physiological variables	+4	+3	+2	+1	0	+1	+2	+3	+4
Temperature (C)	> 41	39–40	–	38–39	36–38.4	34–35.9	32–33.9	30–31.9	< 29.9
Heart rate (bpm)	> 180	140–179	110–139	–	70–109	–	55–69	40–54	< 39
Respiratory rate	> 50	35–49	–	25–34	12–24	10–11	6–9	–	< 5
Serum K + (mmol/L)	> 7	6–6.9	–	5.5–5.9	3.5–5.4	3–3.4	2.5–2.9	–	< 2.5
Serum Na^+ (mmol/L)	> 180	160–179	155–159	150–154	130–149	–	120–129	110–119	< 110
Serum creatinine (mg/1000 ml) (× 2 for acute renal failure)	> 3.5	2–3.4	1.5–1.9	–	0.6–1.4	–	< 0.6	–	–
Hematocrit (%)	> 60	–	50–59	46–49	30–35	–	20–29	− < 20	
WBC (mm^3)	> 40	–	20–39.9	15–19	3–14.9	–	1.2.9	–	< 1
Serum bicarbonate venous (mmol/L)	> 52	41–51	–	32–40	22–31	–	18–21	15–17	< 15

Treatment

- Surgical source control as soon as possible. Re-explorations should be repeated until the time when very little or no debridement is required and send to lab for culture and antibiograms.
- Diverting colostomy or rectal diversion devices
- Antibiotic therapy
- (Organ) supportive measures

Surgical debridement must be early and aggressive to halt the progression of infection. Cultures of infected fluid and tissues should be obtained during the initial surgical debridement, and the results used to tailor specific antibiotic management. Radical surgical debridement of the entire affected area should be performed, continuing the debridement into the healthy-looking tissue [12, 13].

In the setting of FG, diverting colostomy has been demonstrated to improve outcomes but are poorly controlled studies that do not consider the morbidity and impact of the colostomy and its potential reversal.

A transverse loop colostomy is preferred because it yields solid and formed stools with little contamination of the surrounding skin. The abdomen above the umbilicus is ideal because FG often extends into the lower abdominal wall [14].

It may help in minimizing bacterial load in the perineal wound, thus controlling infection [15]. Diverting colostomy does not eliminate the necessity of multiple debridements nor reduces the number of these procedures [15]. Diverting colostomy should be avoided as much as possible, mainly when there are other methods to avoid wound contamination. Recently, rectal diversion devices have been marketed. They are silicone tubes designed to divert fecal matter in patients with diarrhoea, local burns, or skin ulcers. The devices protect the wounds from fecal contamination and reduce, in the same way a colostomy does, the risk of skin breakdown and repeated inoculation with colonic microbial flora. fecal diversion tubes can be used in combination with negative pressure wound therapy to effectively isolate the wound from fecal contamination [16]. Most FG infections are polymicrobial, and since it is impossible to exclude with certainty a polymicrobial necrotizing infection, an aggressive broad-spectrum empiric antimicrobial therapy should initially be selected to cover Gram-positive, Gram-negative, and anaerobic organisms until culture-specific results and sensitivities are available. An acceptable empiric antibiotic regimen should always include antibiotics, which cover MRSA with the additional benefit of inhibiting invasive GAS virulence proteins. For the treatment of MRSA, we refer to the previous paragraphs.

To treat Gram-negative bacteria, the use of piperacillin-tazobactam in the setting without the high local prevalence of ESBL-producing Enterobacteriaceae optimizing pharmacokinetic / pharmacodynamic parameters is appropriate. Carbapenems, administered in adequate dosage, including meropenem, imipenem-cilastatin, or doripenem, may be used in the settings with a high local prevalence of ESBL-producing Enterobacteriaceae. Culture-specific results and sensitivities can direct both broadenings of antibiotic regimen if it is too narrow and the de- escalation if it is too broad, particularly in critically ill patients where de-escalation strategy is one of the cornerstones of antimicrobial stewardship programs.

Empiric antibiotic regimens. Normal renal function

The initial empirical antibiotic regimen should comprise broad-spectrum drugs, including anti-MRSA and anti-Gram-negative coverage. Antitoxin active antibiotics such as clindamycin or linezolid should be included in the empirical antibiotic regimen to treat NSTIs.

In stable patients

- One of the following antibiotics
- Amoxicillin/clavulanate 1.2/2.2 g every 8 h
- Ceftriaxone 2 g every 24 h + Metronidazole 500 mg every 8 h
- Cefotaxime 2 g every 8 h + Metronidazole 500 mg every 8 h
- +
- Clindamycin 600–900 mg every 8 h

In unstable patients

- One of the following antibiotics
- Piperacillin/tazobactam 4.5 g every 6 h
- Meropenem 1 g every 8 h
- Imipenem/Cilastatin 500 mg every 6 h
- +
- One of the following antibiotics
- Linezolid 600 mg every 12 h
- Tedizolid 200 mg every 24 h

or

- Another anti-MRSA-antibiotic as
- Vancomycin 25–30 mg/kg loading dose then 15–20 mg/kg/dose every 8 h
- Teicoplanin LD 12 mg/kg 12-hourly for 3 doses, then 6 mg/kg every 12 h
- Daptomycin 6–8 mg/kg every 24 h *
- Telavancin 10 mg/kg every 24 h
- +
- Clindamycin 600–900 mg every 8 h

*Approved at the dosage of 4 mg/kg/24 h, it is currently used at higher dosages.

6. Gas gangrene

Gas gangrene also named clostridial myonecrosis and is another highly lethal NSTIs, caused by *Clostridium species*, with *Clostridium perfringens* being the most common [17].

Clostridial infections usually arise in traumatized tissues. However, it can also arise spontaneously. The infection involves deeper tissue such as a muscle which can lead to a rapidly spreading infection along tissue planes, and patients often present with sepsis.

C. perfringens causes 80–90%, of gas gangrene cases, but other species can cause infection, including *C. novyi, C. septicum, C. histolyticum, C. bifermentans, C. fallax,* and *C. sordellii* [17].

The fulminant clinical and histological features of an infection with clostridia are mediated by potent bacterial exotoxins, making clostridial myonecrosis the most rapidly spreading and lethal infection in humans.

Clostridium spp. can produce alpha and theta toxins that cause extensive tissue damage.

The primary toxin to mediate the effect of *C. perfringens* is alpha-toxin, a zinc metallophospholipase with phospholipase C and sphingomyelinase activity. Alpha-toxin is thought to be the major factor for tissue pathology leading to muscle necrosis and hemolysis [18]. The second major toxin is theta-toxin, a pore-forming toxin [19].

The infection can spread quickly, and within a matter of several hours, the patient may develop overwhelming shock, sepsis, and death. The infection can develop slowly over weeks or rapidly over hours depending on the oxygen tension of the tissue and the amount of organism inoculated.

Increasingly severe pain beginning within 24 h at the injury site is the first reliable clinical symptom. The skin may initially appear pale, but quickly changes to bronze, then purplish-red. The infected region becomes tense and tender, and bullae filled with reddish-blue fluid appear. Gas in the tissue, detected as crepitus or by imaging, is usually present by this late stage. Signs of systemic toxicity, including tachycardia, fever, and diaphoresis, develop rapidly, followed by shock and multiple organ failure.

Because the infection is rapidly progressive, it is important to treat patients aggressively, by early surgical debridement, antibiotics and intravenous fluid resuscitation.

Treatment / Management

Because the infection is rapidly progressive, it is important to treat patients aggressively with antibiotics, early surgical consultation with debridement, intravenous fluid resuscitation, ICU monitoring, and adjuvant hyperbaric oxygen therapy.

It is important to get early surgical consultation without delay as this is a true surgical emergency. Providers should not delay antibiotics to get cultures but should begin empiric treatment with antibiotics. Reasonable broad-spectrum coverage includes vancomycin and tazobactam or a carbapenem or ceftriaxone with metronidazole. If the provider suspects gas gangrene or a necrotizing soft tissue infection, then penicillin plus clindamycin should be added which will also treat group A streptococcal necrotizing fasciitis. Clindamycin should be strongly considered because it inhibits the synthesis of clostridial exotoxins and will lessen the systemic effects of these toxins. Because clindamycin is bacteriostatic and not bactericidal, it should be used in conjunction with a second anti-microbial such as penicillin.[20][21][22][23][24]

Fasciotomy may be necessary to relieve compartment pressures. As the infection progresses into deep tissue along and under the fascia tissue compartment pressures increase, which perpetuates further tissue ischemia and necrosis. Surgical debridement should focus on removing all the necrotic tissue, and foreign bodies such as soil, debris, and shrapnel. It is also important to irrigate the wounds with copious amounts of sterile normal saline.

Hyperbaric oxygen therapy should be added to standard therapy of antibiotics and surgical debridement to help improve survival.[24][25] It is important to have coordinated care of these critically ill patients with an intensivist, general surgeon, orthopedic surgeon, urologist (in the setting of Fournier's gangrene of the testicles and perineal structures), gynecologist (in the setting of uterine gas gangrene),
infectious disease specialist, hematologist/oncologist, gastroenterologist (in the setting of spontaneous gas gangrene), and hyperbaric oxygen therapy specialist. The flow of consultation starts with usually an emergency department provider and early recognition of the disease.[26][27][26]

Early IV antibiotics with early surgical debridement followed by hyperbaric oxygen therapy can salvage patients with an otherwise nearly always fatal disease. Intravenous antibiotics and early surgical debridement of the necrotic tissue reduce the fatality rate to about 30%. With the addition of hyperbaric oxygen therapy, this can
be reduced down to 5 to 10%. Hyperbaric oxygen therapy helps by halting exotoxin production by the bacteria, helps to improve the bactericidal effect of the antibiotic, treats the tissue ischemia, improves reperfusion injury of the tissue, and promotes the activation and migration of stem cells and polymorphonuclear cells. Additionally, hyperbaric

oxygen induces vasoconstriction reducing tissue edema, while augmenting oxygenation. The oxygen tension of the tissue increases by a factor of 1000 and this increased oxygen in the tissue helps to resolve hypoxia, improve cellular activity, inhibit bacterial growth, and affect cytokinesis that increases migration of neutrophils to the injured tissue. Hyperbaric oxygen also increases the production of growth factors such as vascular epidermal growth factor (VEGF) which induces neovascularization and tissue repair with capillary budding. This is recognized clinically as increased granulation tissue formation and is usually seen after several hyperbaric oxygen treatments.[26][29][30][29]

Hyperbaric oxygen therapy involves placing the patient in a pressurized chamber which can be mono-place (single patient) or multi-place (multiple patients treated at the same time). The mono-place chamber can only treat one patient at a time, and the attendant is outside of the chamber with specialized equipment and pumps to run IVs and even mechanical ventilation equipment through ports in the chamber door or wall. The disadvantage of this setup is that it limits the therapies available in the chamber and if the patient requires direct contact with the attendant, the chamber has to be depressurized, and the patient is taken out of the chamber. The multi-place chamber has the added benefit of being able to treat multiple patients at the same time, and the attendant is in the chamber with the patients allowing easier access to the patient for ventilator support, IV therapy, placement of a chest tube, or needle decompression of a pneumothorax. The treatment pressure for gas gangrene is 3 atmospheres absolute (ATA). The patient will have air brakes about every half hour to help reduce the risk of oxygen toxicity. These air brakes are usually 5 to 10 minutes in duration. The total duration of the treatment at pressure is usually about 90 minutes with 10 minutes for descent and 10 minutes for the ascent.[22][31][32][22]

When treating gas gangrene, the treatments start twice a day for the first 5 to 10 treatments, reducing to once-daily treatments when stabilized. Continuing hyperbaric oxygen therapy beyond the initial stabilization can speed healing of tissue and preparation for eventual tissue grafting that is often necessary to close the large defects left after surgical debridement of dead tissue. The risk of hyperbaric oxygen therapy includes oxygen toxicity which can cause seizures, hypoglycaemia especially in insulin- dependent diabetics, and barotrauma which can affect the ears, lungs, or any gas-filled structures, such as the stomach, and gas embolism. These complications are rare, except for ear barotrauma which occurs approximately 43% of the time (84% of these are minor injections of the tympanic membrane).

It is crucial to get early surgical consultation without delay in the case of gas gangrene, as this is an immediate emergency. Broad-spectrum antibiotics should be initiated without any delay to get cultures. Reasonable coverage should include vancomycin, tazobactam or a carbapenem, or a third-generation cephalosporin (ceftriaxone) with metronidazole. Moreover, in case of any suspicion for gas gangrene or necrotizing fascitis, penicillin plus clindamycin should be added to cover group A

streptococcal necrotizing fasciitis. Clindamycin is strongly recommended. Adjunctive measures in the treatment of gas gangrene include hyperbaric oxygen (HBO) therapy. The function of the existing toxin is not affected by hyperbaric oxygen therapy; thus, debridement is of paramount importance. Hemodynamically unstable patients may not be candidates for HBO therapy. Moreover, animal experimental studies in animals failed to document the therapeutic efficacy of HBO.[32][20]

Providers should consider the use of negative pressure wound dressing therapy once adequate surgical debridement has resolved ongoing tissue necrosis.

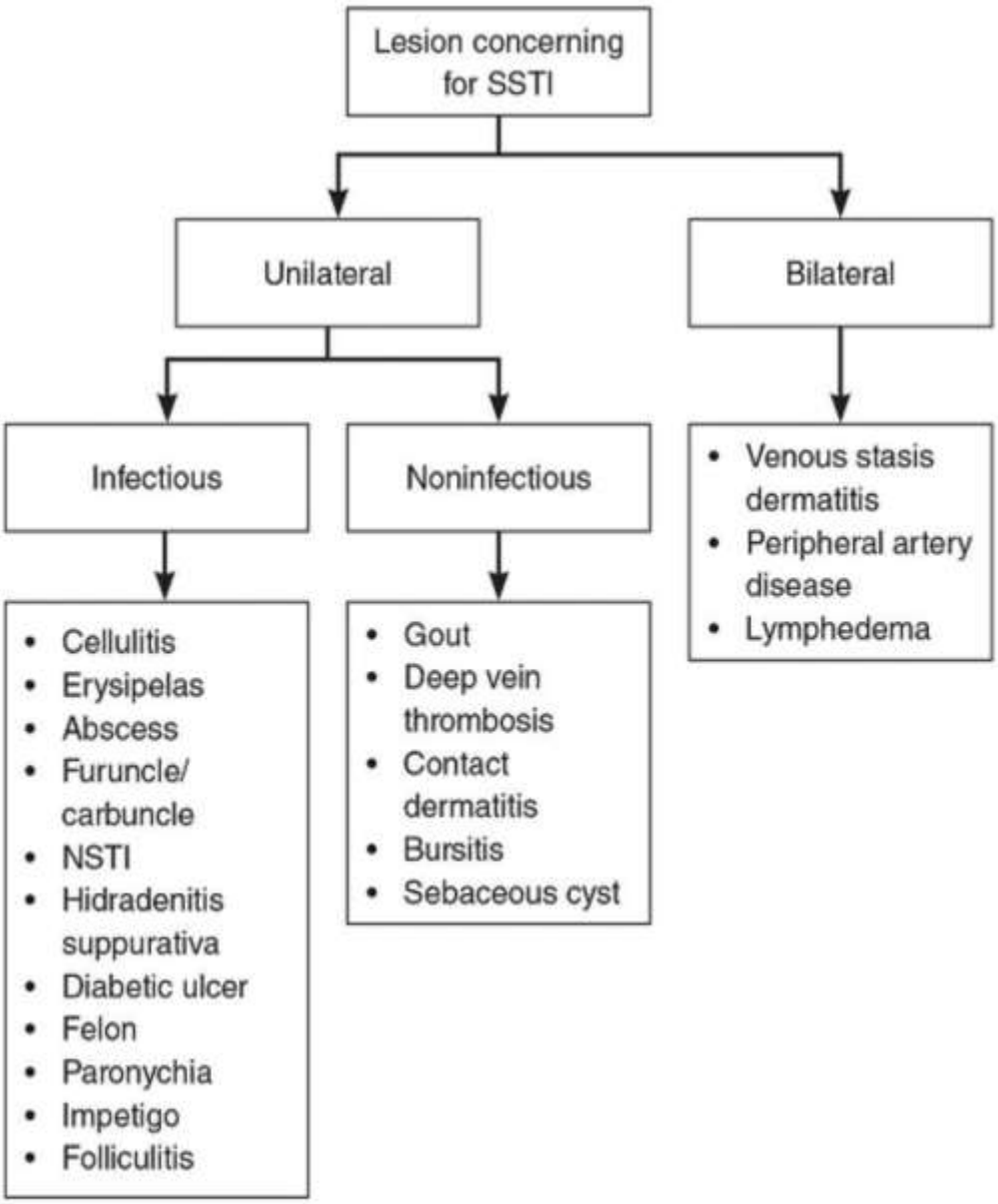
Table: clinical practice and CPA

Pathology	**Clinical signs**		**Bacteria**	**Indication**	**Anest**	**CPA**
Simple abscess	Skin abscess	Collection pus dermis and deep tissues, induration, Erythema	Sta. aureus, E.colie, Stre.pyogenes	Inci. drainage + open wound	LRA GA	CPA 2,3
	Epidermoid cysts infected	Or Sebaceous cysts Infection pilo-sebaceous gland: Lumps or bumps warmth tenderness and redness	Coci grame +, Stap, Anaerobie Fungus	Removal including capsule + open	LRA	CPA 2,3
	Furuncles	Superficial Infec. with suppuration of hair follicle: redish tender bumps + pus	Staphylococcus aureus	Inci drainage + open	LA	CPA 2,3
	Carbuncles	larger and deeper than furuncles or Clusters of Furuncles and fever	Staphylococcus aureus	Inci drainage + open	LA/GA LRA	CPA 2,3
Erysipelas	skin infection involving the dermis layer, but it may also extend to the superficial cutaneous lymphatics: fiery red, tender, painful plaque with well-demarcat- ed edges. distinguished clinically from cellulitis by the following two features : • the lesions are raised above the level of the surrounding skin, • Erysipelas is characterized by a clear line of demarcation between involved and uninvolved tissue.		Streptococcus spp., usually Staphylococcus pyogenes. Staphylococcus aureus	Medical treament		CPA 1,2,3
Cellulitis	infection primarily of the dermal lymphatics and the subcutaneous tissue. warmth, erythema, pain, lymphangitis, and frequently systemic upset impact with fever		Streptococcal species and *S. aureus*. Gram -	Incision drainag / Faciotomy + open wound	LRA GA	CPA 2,3

Perianal and perirectal abscesses	Anal pain, Fever, Chills, Constipation, or diarrhea Purulent discharge, Erythema and iduration in the skin around the perianal.	Gram-positive, Gram-negative, and anaerobic bacteria	Incision drainag + open wound	LRA GA	CPA 2,3
Necrotizing infection	Aggressive invasive, soft-tissue infections with a necrotizing component involving any or all layers of the soft-tissue compartment, from the superficial dermis and subcutaneous tissue to the deeper fascia and muscle. Local signs: - Edema, Erythema - Sever pains - Skin bullare or necrosis Swelling or tenderness - Crepitus Systemic signs: - Fever - Tachycardia - Hypotension - Shock	Staphylococcu Streptococcus A Polymicrobial - Gram (- and +), and anaerobic - Clostridium myonecrosis - Bacterioides coliforms, prot eus, klebsiella, peptostreptoc oc cus and pseudomona s	- Debridement surgical as soon as possible within 6 h after admission. Re-explorations should be done at least every 12–24 h after ini tial operation or sooner if clinical local/systemi c signs worsening	LRA GA	CPA 3
Fournier's gangrene	is a severe type of NSTI involving the genital area and or perineum. Local signs: - Edema, Erythema - Sever and crescendo pains - Skin bullare or necrosis - Swelling or tenderness - Crepitus Systemic signs: - Fever - Tachycardia - Hypotension - Shock	Staphylococcu Streptococcus A Polymicrobial - Gram (- and +), and anaerobic - Clostridium myonecrosis - E-coli and pseudomona s	- Debridement surgical as soon as possible within 6 h after admission. Re-explorations should be done at least every 12–24 h after ini tial operation or sooner if clinical local/systemi c signs worsening	LRA, SP/RA GA	CPA 2,3

			- Colostomy or rectal diversion devices		
Gas gangrene	- named clostridial myonecrosis and Clostridial infections usually arise in traumatized tissues. - spread quickly of several hours. - patient may develop overwhelming shock, sepsis, and death. - severe pain beginning within 24 h at the injury site. - skin may initially appear pale, but quickly changes to bronze, then purplish-red. - infected region becomes tense and tender, and bullae filled with reddish-blue fluid appear. - Signs of systemic toxicity, including tachycardia, fever, and diaphoresis, develop rapidly, followed by shock and multiple organ failure.	*Clostridium species* *Clostridium perfringens* causes 80–90%, but other species can cause infection, including *C. novyi, C. septicum, C. histolyticum, C. bifermentans, C. fallax,* and *C. sordellii*	- Debridement / Fasciotomy surgical as soon as possible. - Hyperbaric oxygen therapy	LRA SA/RA GA	CPA 3

DIFFERENTIAL DIAGNOSES OF SOFT-TISSUE INFECTION



Abbreviations: NSTI, necrotizing soft-tissue infection; SSTI, skin and soft-tissue infection.

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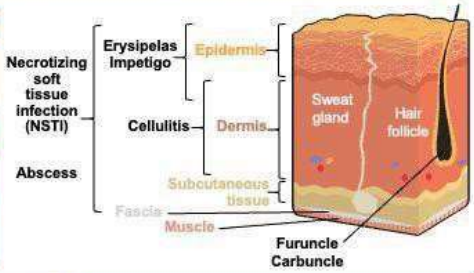
SKIN AND SOFT TISSUE INFECTIONS

SIGNS & SYMPTOMS

- Erythema, warmth, tenderness, pain, fever, purulence
- **Systemic signs:** Temp. >38°C, HR >90, RR >24 or WBC <12,000 or <300 cells/uL

HISTORY

- Onset of signs/symptoms? Progression?
- Association with trauma?
- Burn(s), frostbite, pressure ulcer, post-surgical?
- Environmental risks? Vaccination history?
- Severity of pain? Radiation?
- Loss of function? Joint involvement?



Non-Purulent SSTI			
Erysipelas	Associated with fever, well demarcated erythema	*Group A Streptococcus* (GAS)	
Impetigo	**Non-bullous:** painless, erythematous base w/ honey-crusted exudate on face/limbs	*S. aureus*, GAS	
	Bullous: clusters of bullae/solidary lesions of exudate ± desquamation	Toxin producing *S. aureus*	
Cellulitis	Edema, pain, poorly demarcated erythema. Orbital cellulitis is a medical emergency!	GAS, *S. aureus*	
Necrotizing Soft Tissue Infection (NSTI)	Generally, rapidly evolving, pain out of proportion, erythematous rash w/ fever, toxic appearance, & thrombocytopenia. Hemodynamically unstable.	**Monomicrobial** *S. aureus* most common	**Polymicrobial** GAS, MRSA, VRE, *Clostridium*
Purulent SSTI (Drainable Collection)			
Cutaneous / Deep Soft Tissue Abscess	Collection of pus	*S. aureus* (community-acquired MRSA), GAS	
Furuncles, Carbuncles ± Cellulitis	**Furuncles (boils)** are skin abscesses that involve a hair follicle. **Carbuncles** are clusters of furuncles.	*S. aureus* (including MRSA), GAS	

Cellulitis, Erysipelas, Necrotizing infection

Mild: cellulitis, erysipelas, ± purulence
Moderate: above w/ systemic signs
Severe: failed oral antibiotics, systemic symptoms, immunocompromised, signs of deeper infection (bullae, skin sloughing, hypotension, organ dysfunction)

Furuncle, Carbuncle, Abscess

MILD	MODERATE	SEVERE
Oral **Cephalexin** **Cloxacillin**	**Intravenous** **Cefazolin** *OR* **Cloxacillin** *OR* **Ceftriaxone** *OR*	**Emergent surgical inspection, debridement** *R/o necrotizing process* **Empiric Rx:** 1) Vancomycin 2) ***PLUS*** Piperacillin-Tazobactam 3) ***PLUS*** Clindamycin*

C&S for definitive management

MILD	MODERATE	SEVERE
Incision and Debridement (I&D)	**I&D** *PLUS* **Cephalexin PO** *OR* **Cefazolin IV**	**I&D** *PLUS* **Vancomycin IV**

C&S of purulent fluid for definitive management

NSTIs IN PEDIATRICS

1) No skin manifestations in up to 50% of cases.
2) A prolonged prodrome or perceived slow onset should NOT rule out an NSTI.
3) May present with multifocal sites. Always check for additional sites during the physical exam.

EAGLE EFFECT

The **Eagle Effect** is the paradoxical effect of reduced penicillin efficacy at higher antibiotic doses. **Clindamycin** also inhibits bacterial toxin production.

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Alexander Perry (Medical Student 2021, University of Alberta), Courtney Wilkes (Plastics PGY-5, University of Alberta) & Dr. Jaret Olson (Pediatric Plastic Surgeon, University of Alberta) for www.pedscases.com

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CHAPTER VII

TRAUMATO-ORTHOPEDIC SURGERY

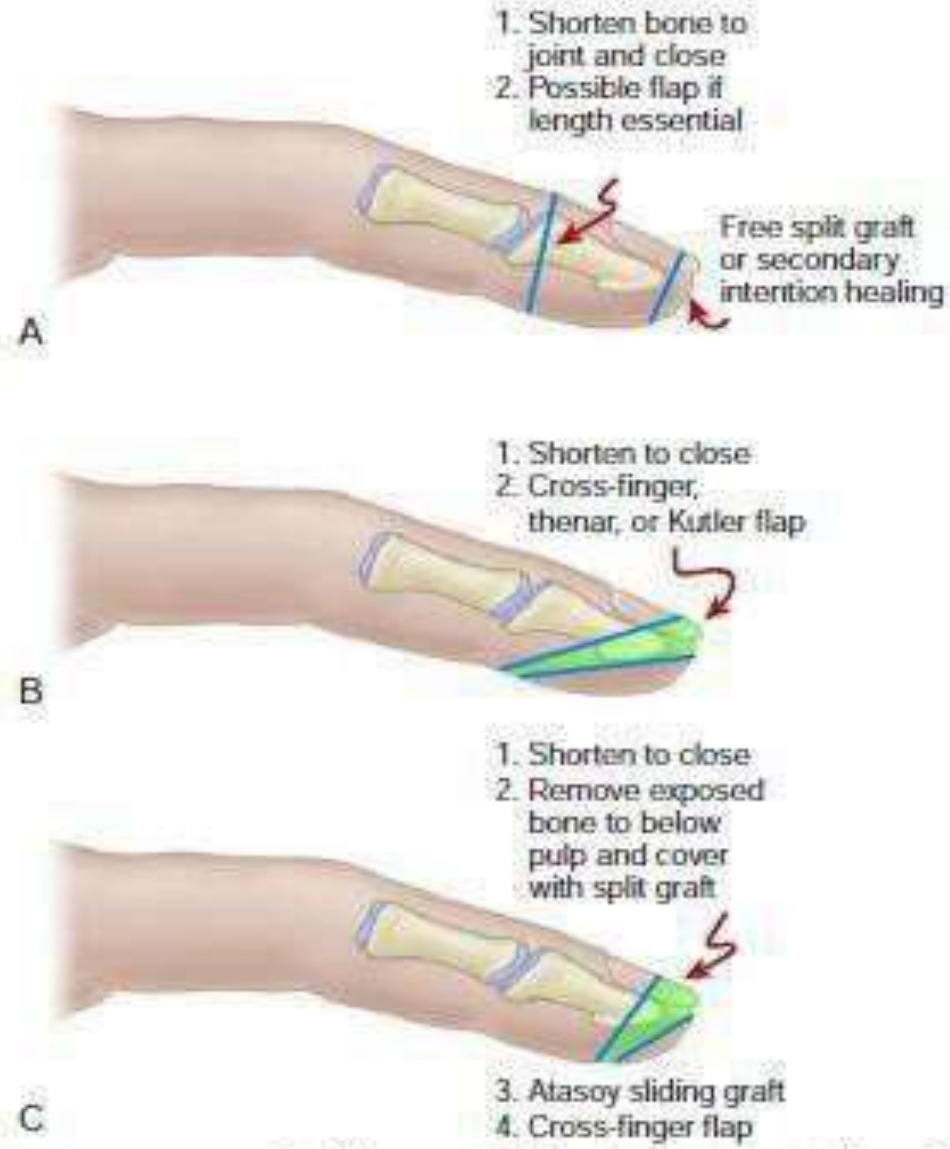
1. Amputation of hand, foot and ankle
2. Amputation of upper and lower limb
3. Clavicle fracture
4. Elbow dislocation
5. Femoral shaft fracture
6. Forearm fracture
7. Hip dislocation
8. Humerus shaft fracture
9. Lower limb cast
10. Open bone fracture
11. Open fracture management
12. Proximal tibia pin traction
13. Shoulder joint dislocation
14. Tibia shaft fracture
15. Trans calcaneus pin traction
16. Upper extremity splints and casts
17. Wrist fracture

AMPUTATION OF HAND AND FOOT AND ANKLE

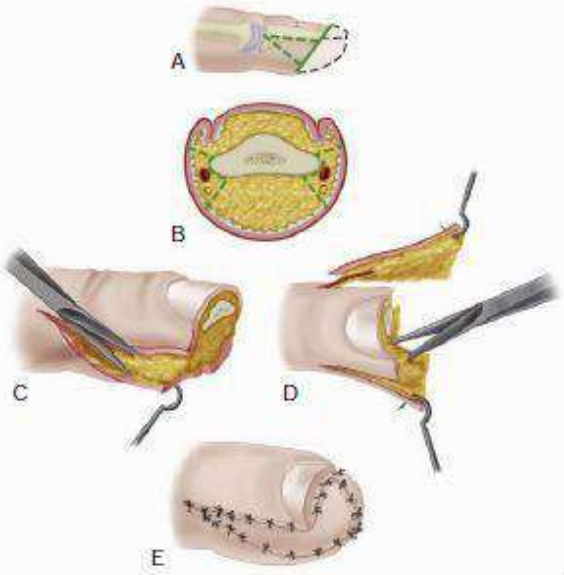
CHEA Huy, HENG Veasna, SONG Kimhai

I. AMPUTATION OF HAND

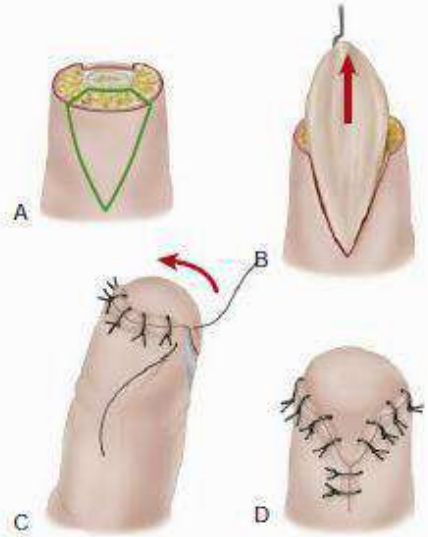
- Fingertip amputation: a split thickness graft is sufficient if the bone is only slightly exposed/ not exposed. Flaps/ full thickness grafts are desirable for better sensation and durability. Flaps available for fingertip cover include:
  - V-Y advancement flaps (Kutler/Atasoy)
  - Cross finger flap
  - Thenar flap
  - Island pedicle flap
  - Ulnar hypothenar flap
- Index/2nd ray amputation ideal level is through second metacarpal if amputation is anticipated proximal to PIP.
- Thumb amputation reconstruction of thumb can be done by pollicization of 2nd digit (Buck-Gramcko). 2nd toe may be used with micro-vascular technique to replace for thumb.



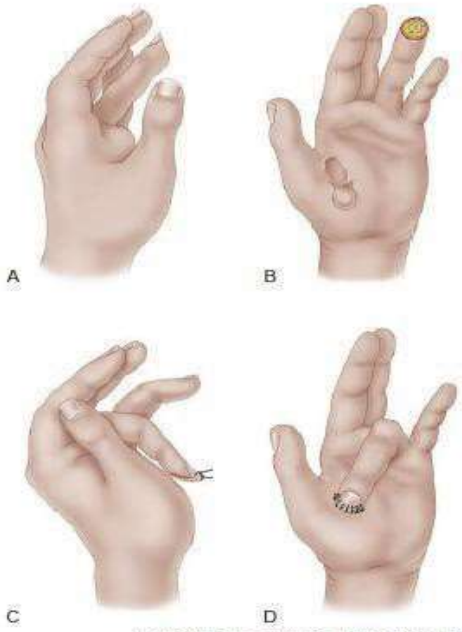
Techniques useful in closing amputations of fingertip. **A,** For amputations at more distal levels, a free split graft is applied; at more proximal levels, bone is shortened to permit closure, or if length is essential, dorsal flaps can be used. **B,** For amputations through green area, bone can be shortened to permit closure or cross-finger or thenar flap can be used. **C,** For amputations through green area, bone can be shortened to permit closure, exposed bone can be resected, and a split-thickness graft can be applied; Kutler advancement flaps can be used, or a cross-finger flap can be applied. In small children, fingertips commonly heal without grafts.



Kutler V-Y advancement flaps. **A,** Advancement flaps over neurovascular pedicles carried down to bone. **B–D,** Fibrous septa are defined **(B)** and divided **(C),** permitting free mobilization on neurovascular pedicles alone **(D). E,** Flaps advance readily to midline.

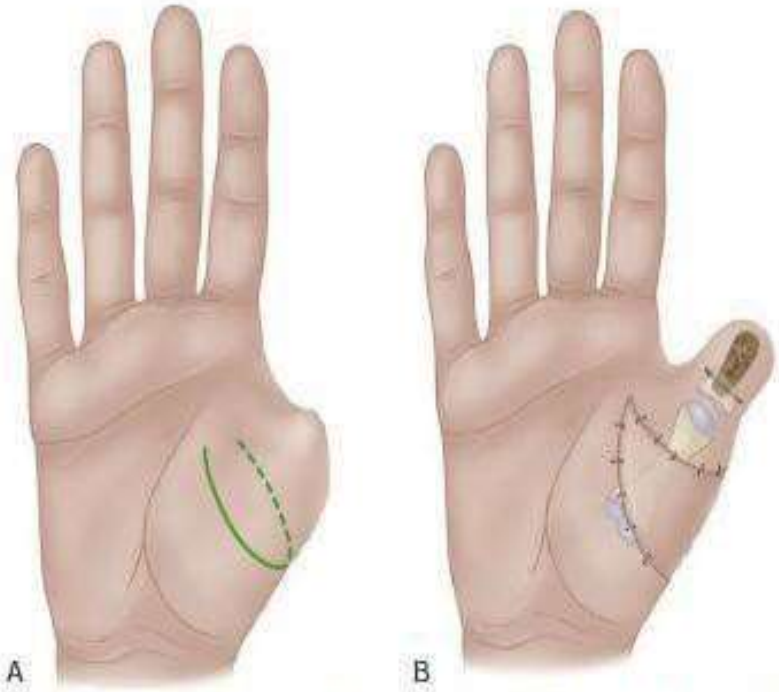


Atasoy V-Y technique. **A,** Skin incision and mobilization of triangular flap. **B,** Advancement of triangular flap. **C,** Suturing of base of triangular flap to nail bed. **D,** Closure of defect, V-Y technique.



Thenar flap for amputation of fingertip. **A,** Tip of ring finger has been amputated. **B,** Finger has been flexed so that its tip touches middle of thenar eminence, and thenar flap has been outlined. **C,** Split-thickness graft is to be sutured to donor area before flap is attached to finger. **D,** End of flap has been attached to finger by sutures passed through nail and through tissue on each side of it

Thumb tip amputation levels. Acceptable procedures by level are *1*, split-thickness graft; *2*, cross-finger flap or advancement flap; *3*, advancement flap, cross-finger flap, or shorten thumb and close; *4*, split-thickness skin graft; *5*, shorten bone and split-thickness skin graft, advancement flap, or cross-finger flap; *6*, advancement flap or cross-finger flap; and *7*, advancement flap and removal of nail bed remnant.



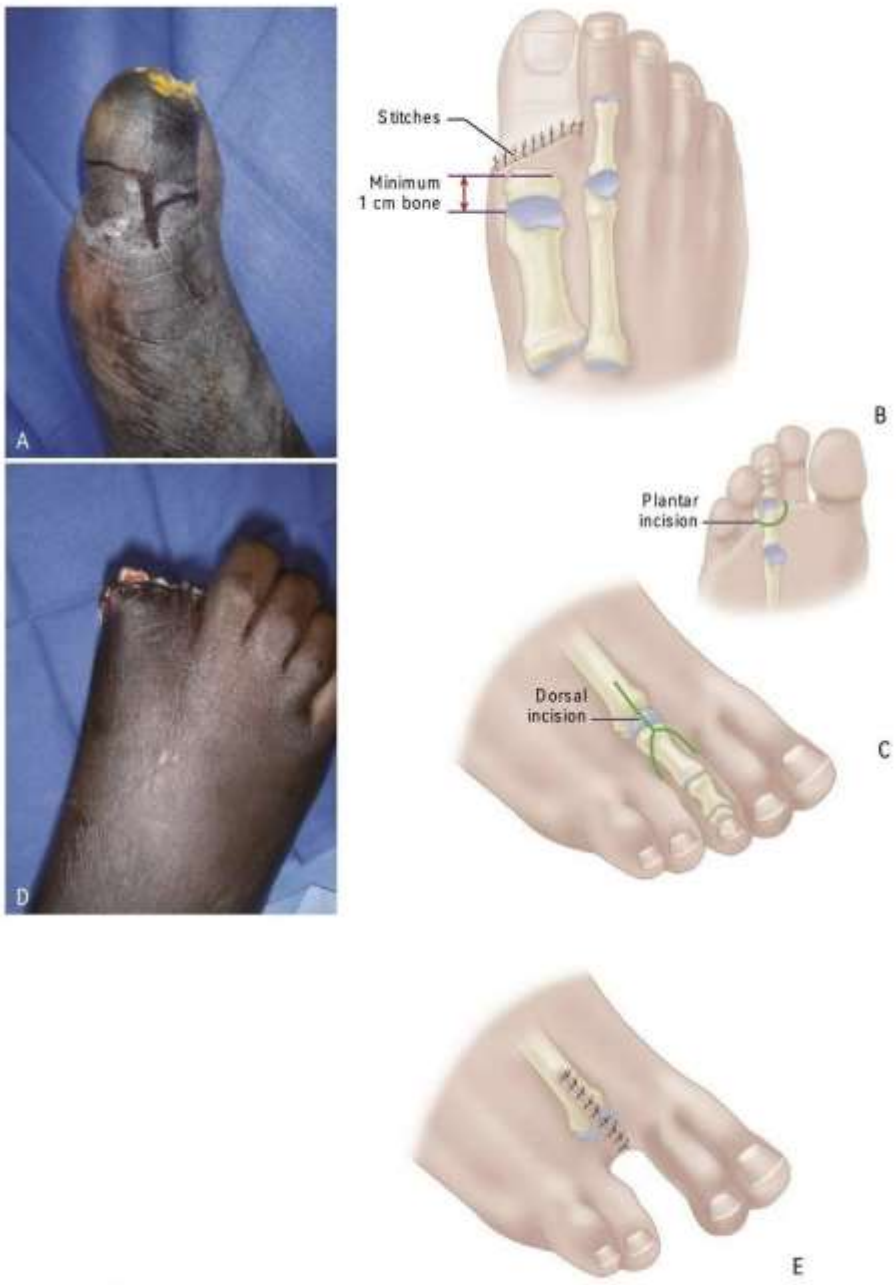
Reconstruction of thumb by technique of Gillies and Millard, modified. **A**, Outline of curved incision around dorsal, radial, and volar aspects of base of thumb. **B**, Hollow flap has been undermined and elevated, iliac bone graft has been fixed (this time to base of proximal phalanx), and raw area at base of thumb has been covered by split-thickness skin graft.

II. AMPUTATION OF FOOT AND ANKLE

- Toe amputation: disarticulation of toes from Interphalangeal joint (Single or multiple toes)
  - preserve 1cm at base of proximal phalanx
    - ✓ preserves insertion of plantar fascia, sesamoids, and flexor hallucis brevis
    - ✓ reduces amount of weight transfer to remaining toes
    - ✓ lessens risk of ulceration
- Metatarsophalangeal disarticulation
- Trans metatarsal amputation
  - more appealing to patients who refuse transtibial amputations
  - almost all require Achilles lengthening to prevent equinus
- Lisfranc amputation (midfoot amputation): at level of tarso-metatarsal joint
  - Equinovarus deformity is common
    - ✓ caused by unopposed pull of tibialis posterior and gastroc/soleus
    - ✓ prevent by maintaining insertion of peroneus brevis and performing achilles lengthening
    - ✓ a walking cast is generally used for 4 weeks to prevent late equinus contracture

- Energy cost of walking similar to that of BKA

- Chopart or Boyd amputation (hindfoot amputation): at level of midtarsal joint
  - a partial foot amputation through the talonavicular and calcaneocuboid joints
  - primary complication is equinus deformity
    - ✓ avoid by lengthening of the Achilles tendon and transfer of the tibialis anterior to the talar neck
    - ✓ leads to a propulsive gait pattern because the amputation is unable to support modern dynamic elastic response prosthetic feet
- Syme amputation (ankle disarticulation): through ankle at the level just proximal to malleoli.
  - patent tibialis posterior artery is required
  - more energy efficient than midfoot even though it is more proximal
  - stable heel pad is most important factor
  - used successfully to treat forefoot gangrene in diabetics
  - disadvantages include: poor cosmesis (bulbous stump) and migration of heel pad posteriorly).
- Pirogoff amputation (hindfoot amputation: involves sectioning of calcaneus vertically. The remaining posterior part of calcaneus is rotated to produce tibia calcaneal arthrodesis.
  - removal of the forefoot and talus followed by calcaneotibial arthrodesis
  - calcaneus is osteotomized and rotated 50-90 degrees to keep posterior aspect of calcaneus distal
  - allows patient to mobilize independently without use of prosthetic
- Ray amputation (lateral ray, middle rays and medial ray).



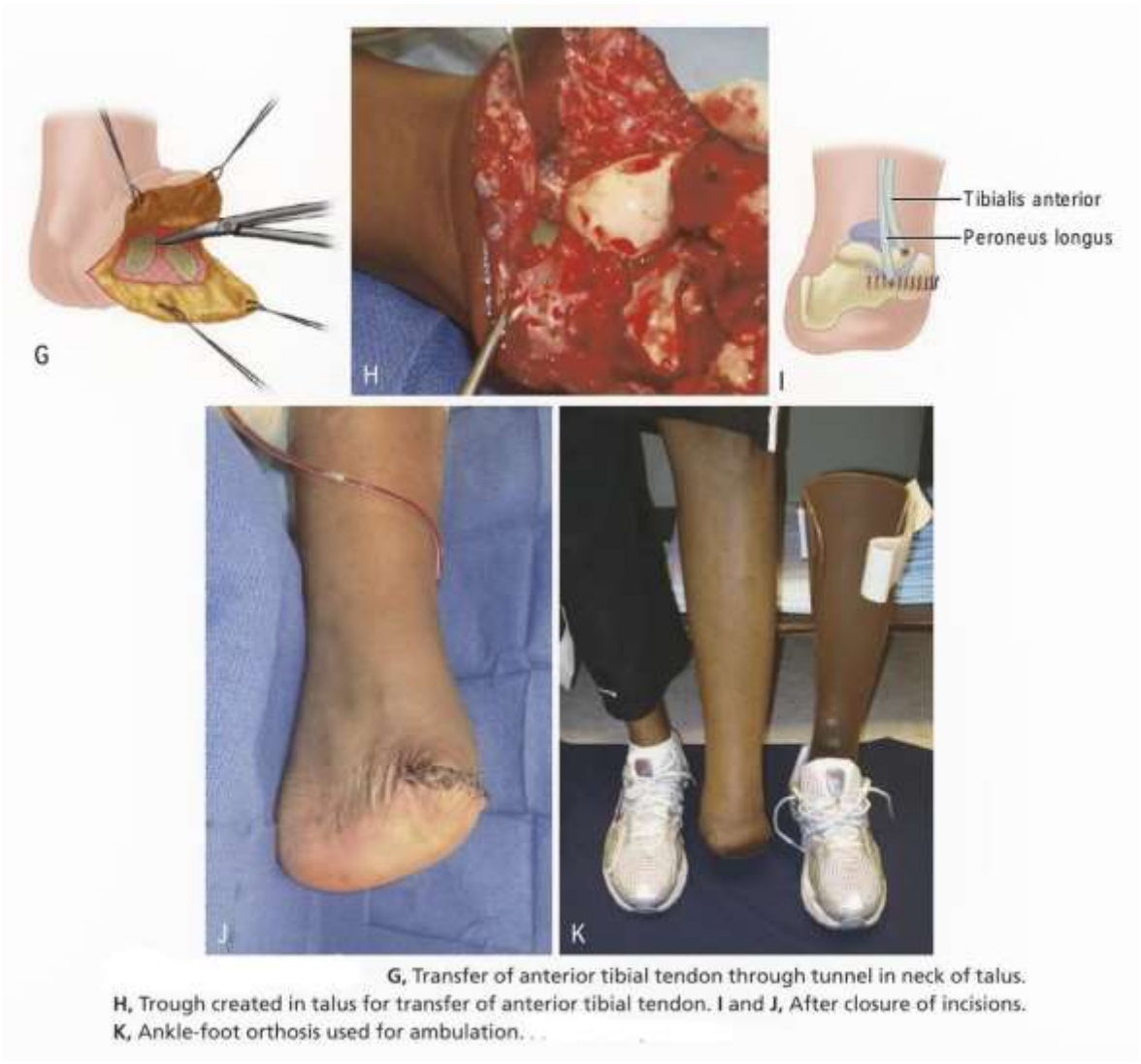
Amputation at base of phalanx. **A,** Incision. **B,** Osteotomy of first proximal phalanx 1 cm from base. **C,** Racquet-shaped incision. Osteotomy of lesser toes also is made 1 cm from the base of the proximal phalanx. **D** and **E,** Wound closure.

A

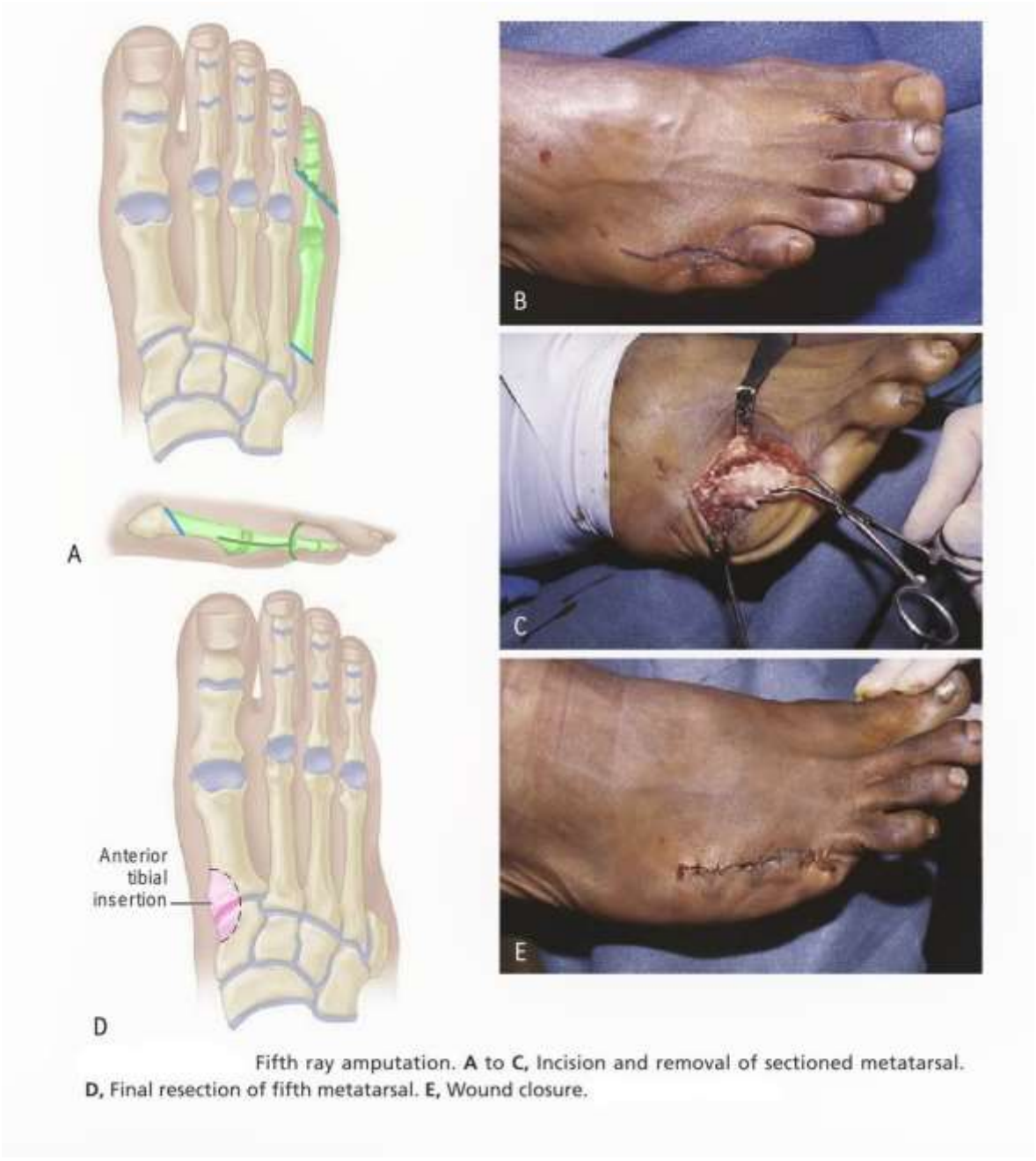
B

C

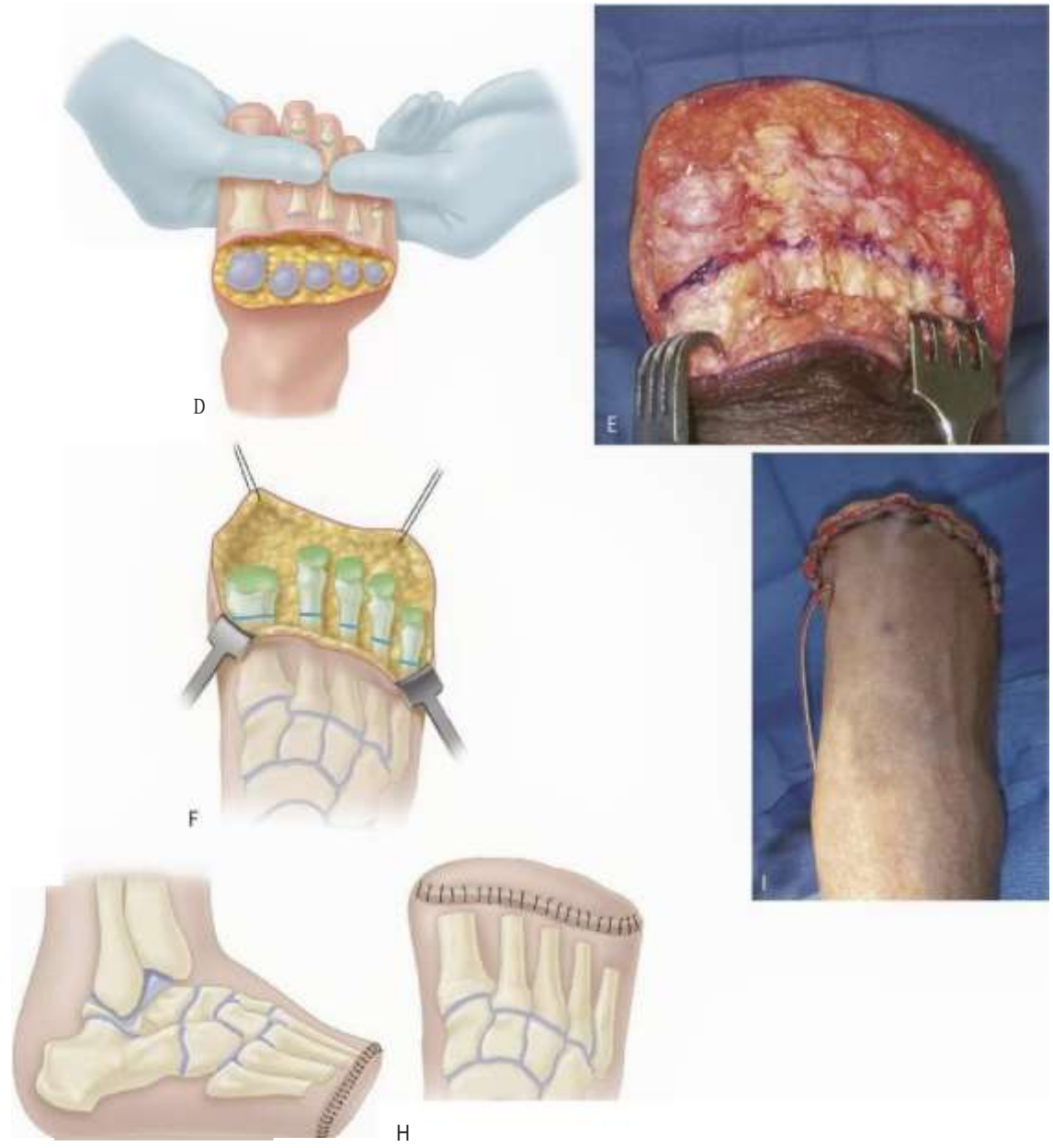
A, Disarticulation at metatarsophalangeal joint of great toe. **B** and **C,** Severe ischemia of hallux to level of metatarsophalangeal joint.



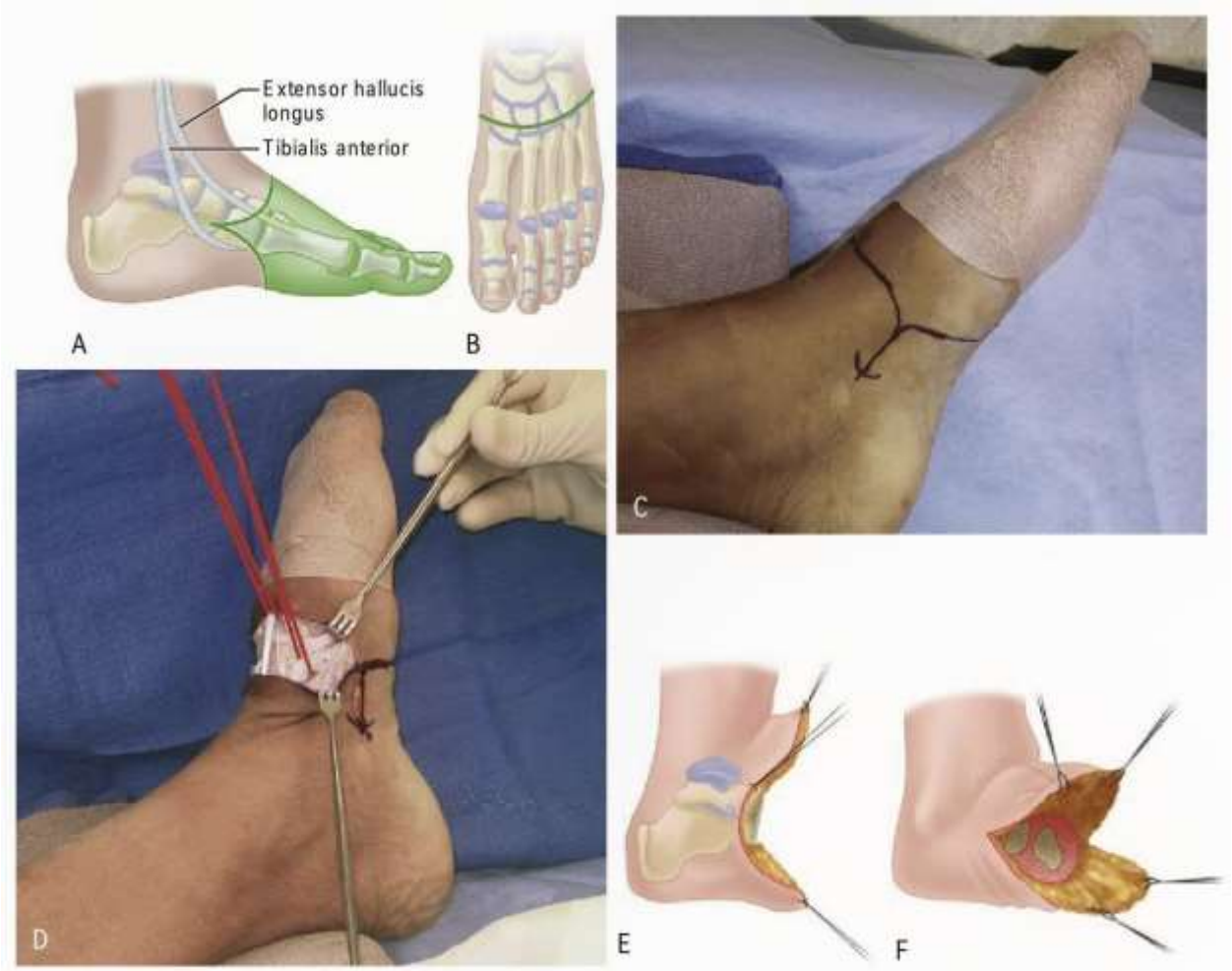
G, Transfer of anterior tibial tendon through tunnel in neck of talus. **H,** Trough created in talus for transfer of anterior tibial tendon. **I** and **J,** After closure of incisions. **K,** Ankle-foot orthosis used for ambulation.



Fifth ray amputation. **A** to **C,** Incision and removal of sectioned metatarsal. **D,** Final resection of fifth metatarsal. **E,** Wound closure.



DtoF. Levelofbonetransectionintransmetatarsal amputation. Gand H, Osteotomy locations are gently curved with flap brought over the ends of bones. I, One-layer closureusingmonofilamentnonabsorbablesuture.



Chopart amputation. **A,** Incisions: lateral view of dorsal and plantar flaps. **B,** Dorsal view of incision. **C,** Dorsal flaps outlined. **D,** Anterior tibial and extensor hallucis longus tendons (marked by vessel loops) will be transferred to the neck of talus to help counter equinus deformity. **E** and **F,** Flaps retracted after resection of distal foot.

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AMPUTATION OF UPPER AND LOWER LIMB

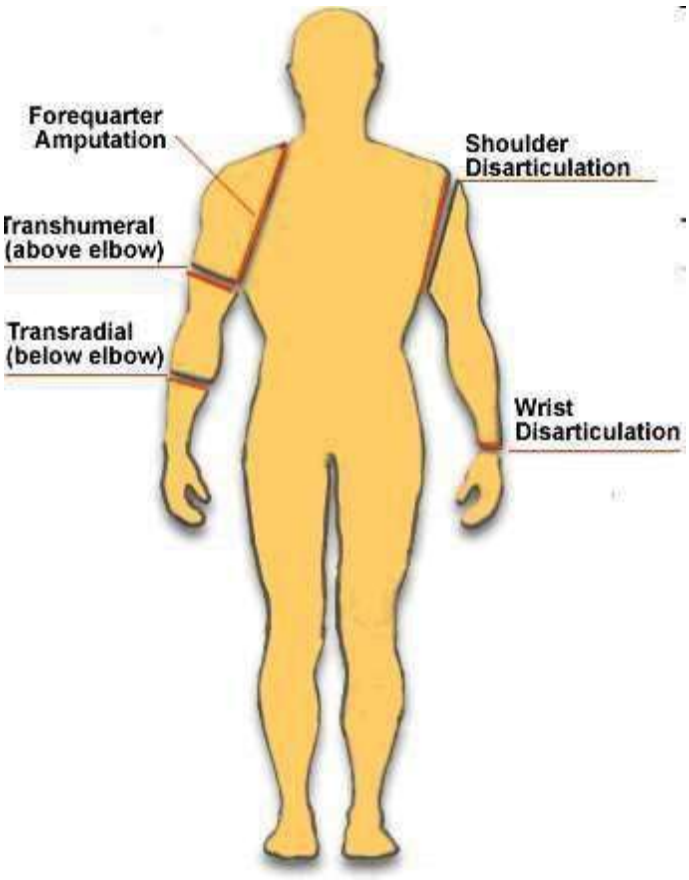
CHEA Huy, HENG Veasna, SONG Kimhai

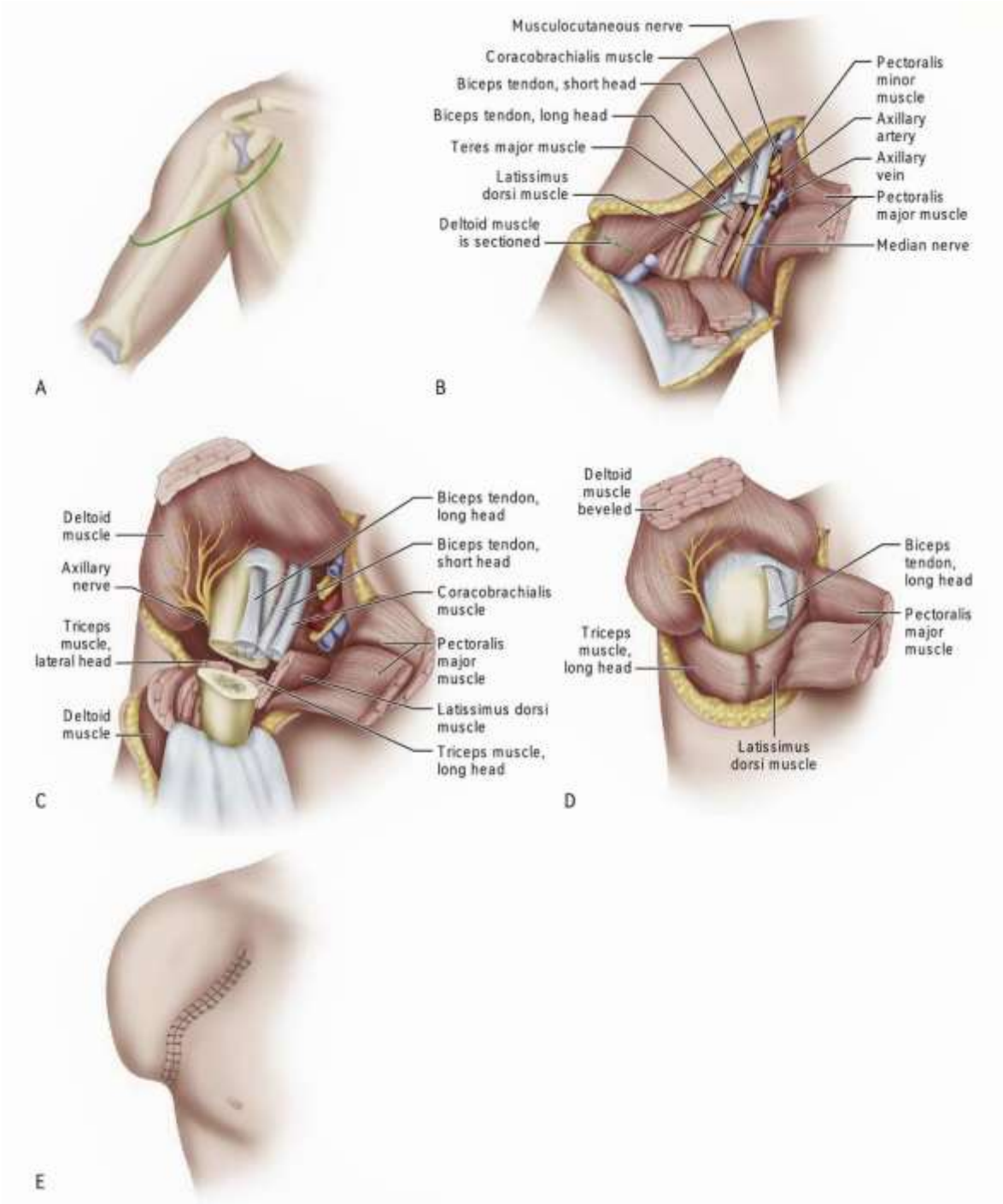
I. DEFINITION

Amputation is a removal of a part of limb completely or partially proximal to the lesion.

II. UPPER LIMBS AMPUTATION

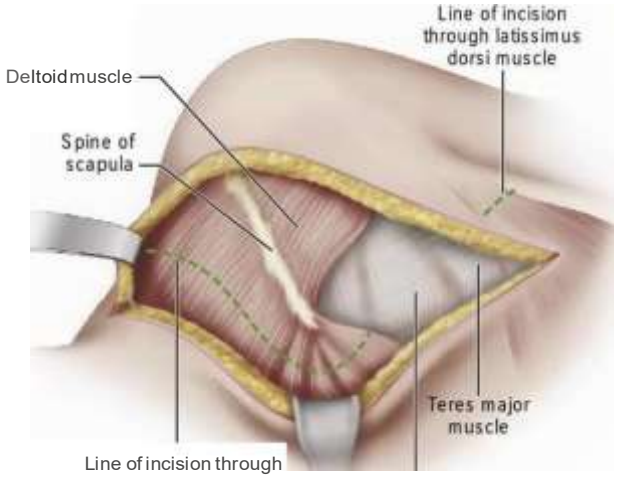
- Wrist Disarticulation: Limb is amputated at the level of the wrist
- Trans radial (below elbow amputations) Amputation occurring in the forearm, from the elbow to the wrist
- Trans humeral (above elbow amputations): Amputation occurring in the upper arm from the elbow to the shoulder
- Shoulder Disarticulation: Amputation at the level of the shoulder, with the shoulder blade remaining.
- Forequarter Amputation: Amputation at the level of the shoulder in which both the shoulder blade and collar bone are removed.





Amputation through surgical neck of humerus. A, Skin incision. **B,** Section of anterior muscles. **C,** Bone level and completed muscle section. **D,** Closure of muscle flap. **E,** Completed amputation.

A



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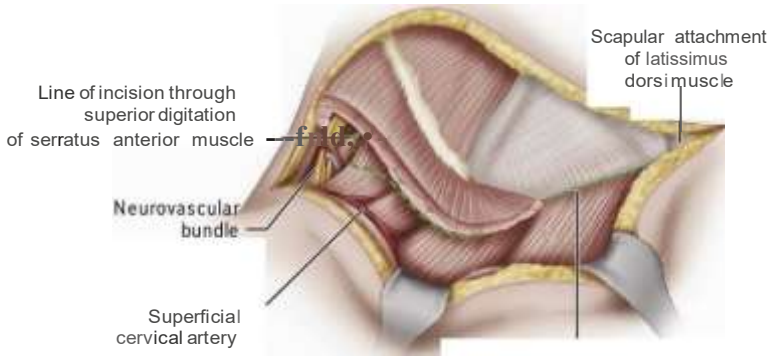

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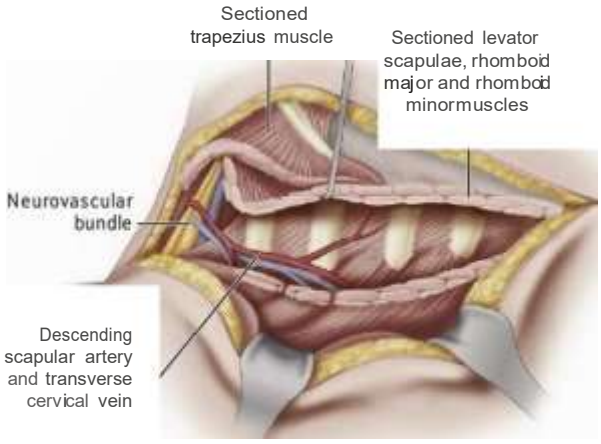
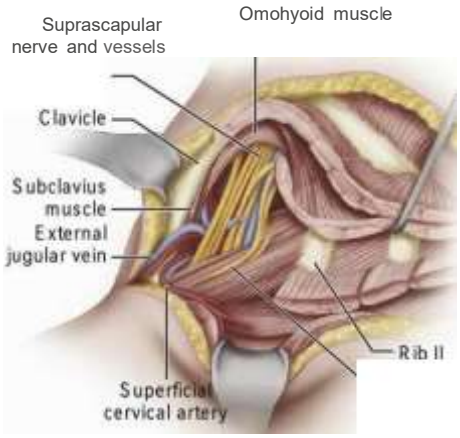


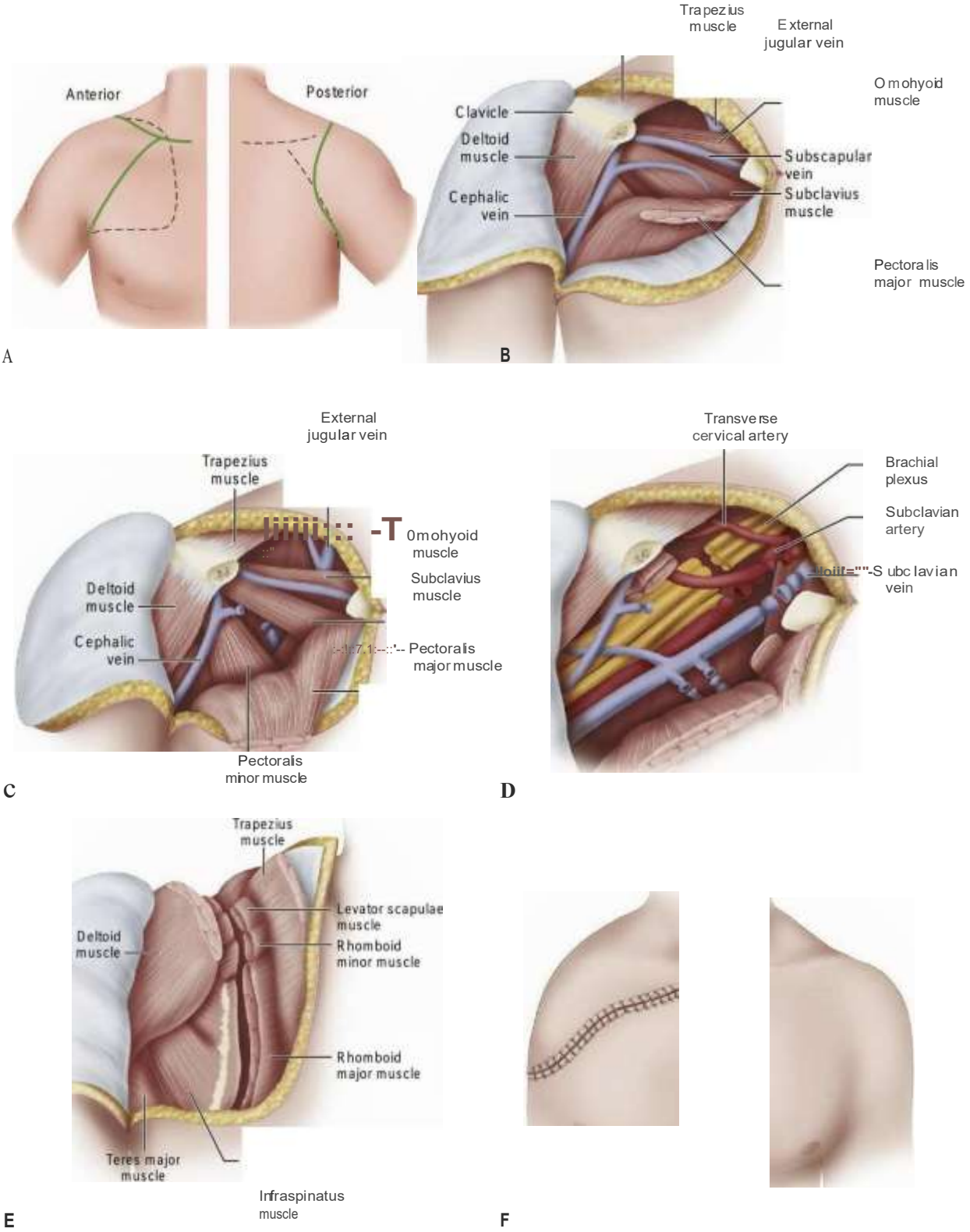
D



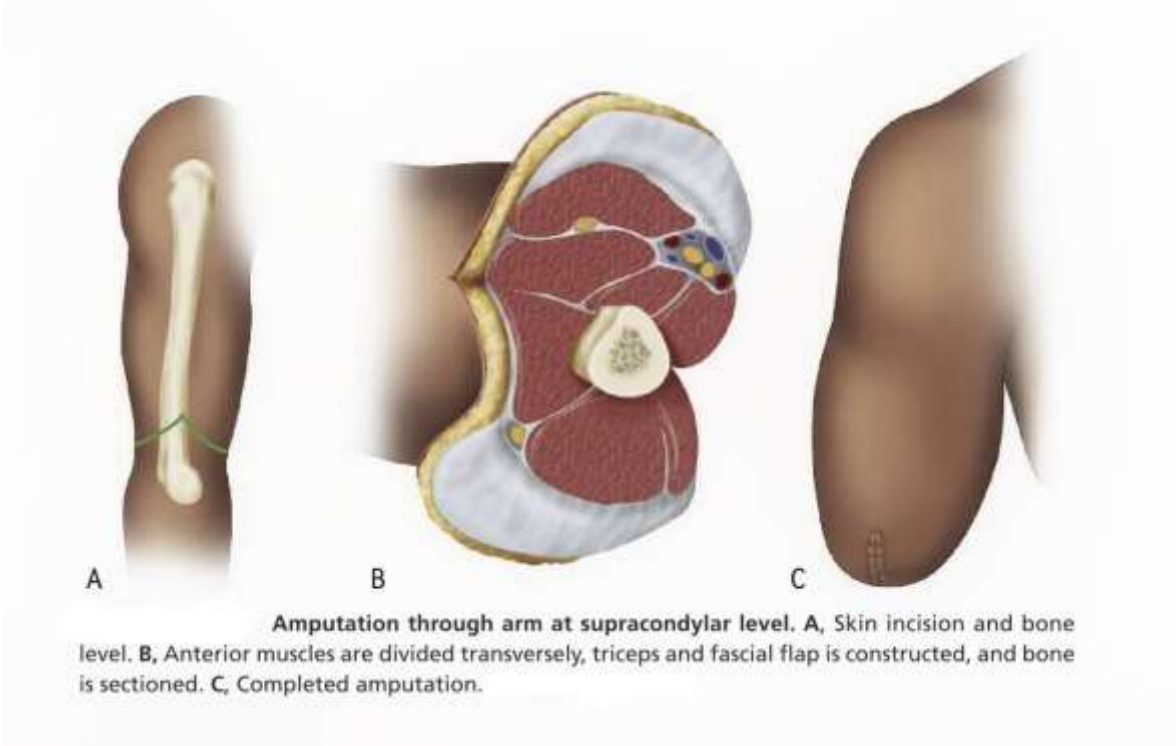
E

C5, 6 origin of long thoracic nerve piercing scalenus medius

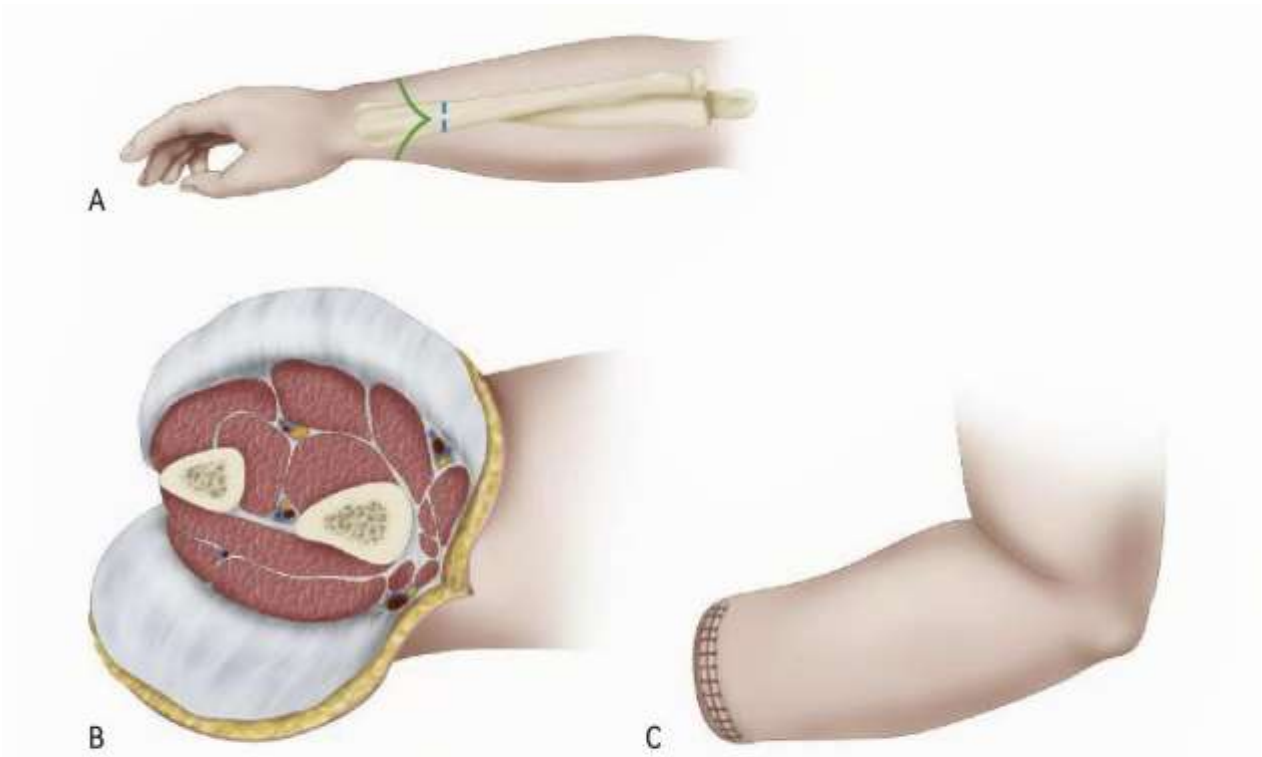
Littlewood technique for interscapulothoracic (forequarter) amputation. A, Incision. **8,** Skin flaps undermined from clavicle. **C,** Scapula drawn away from chest wall with hook or retractor; levator scapulae and rhomboids minor and major divided. **0,** Exposure of neurovascular structures. **E,** More detailed view of neurovascular structures.



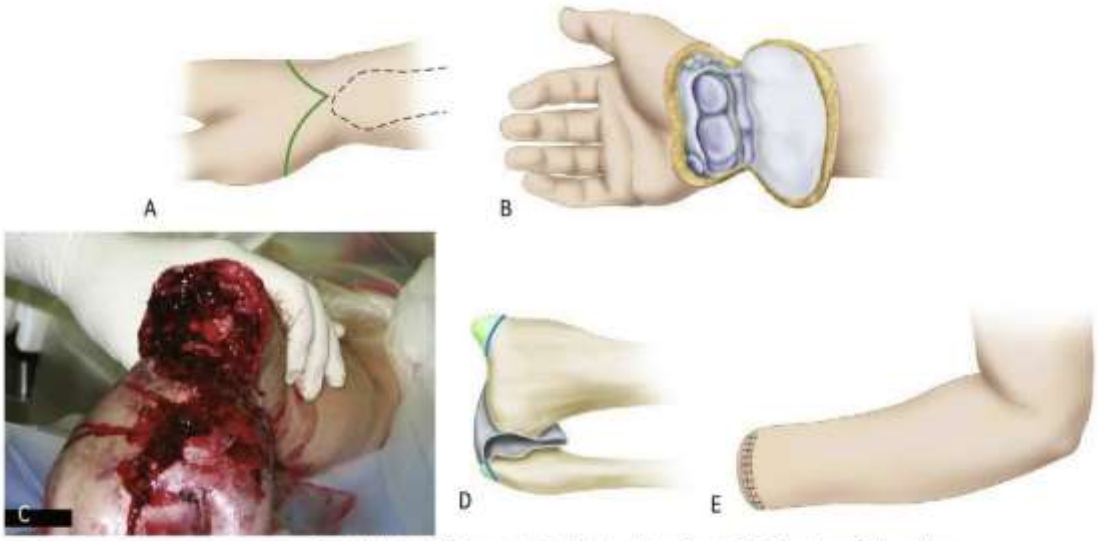
Forequarter amputation through anterior approach. A, Incision. **B,** Resection of clavicle. **C,** Lifting pectoral lid. **D,** Sectioning of vessels and nerves after incision through axillary fascia and insertion of pectoralis minor, costocoracoid membrane, and subclavius. **E,** Sectioning of supporting muscles of scapula. **F,** Completed amputation.



Amputation through arm at supracondylar level. A, Skin incision and bone level. **B,** Anterior muscles are divided transversely, triceps and fascial flap is constructed, and bone is sectioned. **C**, Completed amputation.



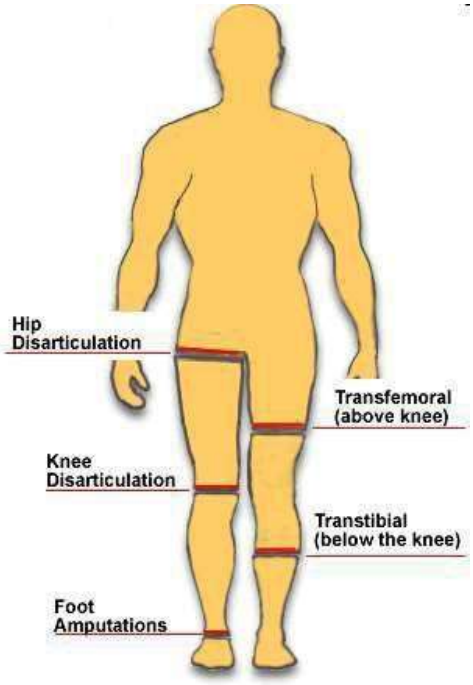
Amputation through distal forearm. A, Skin incision and bone level. **B,** Flaps are reflected, and bones and soft structures are divided. **C,** Completed amputation.

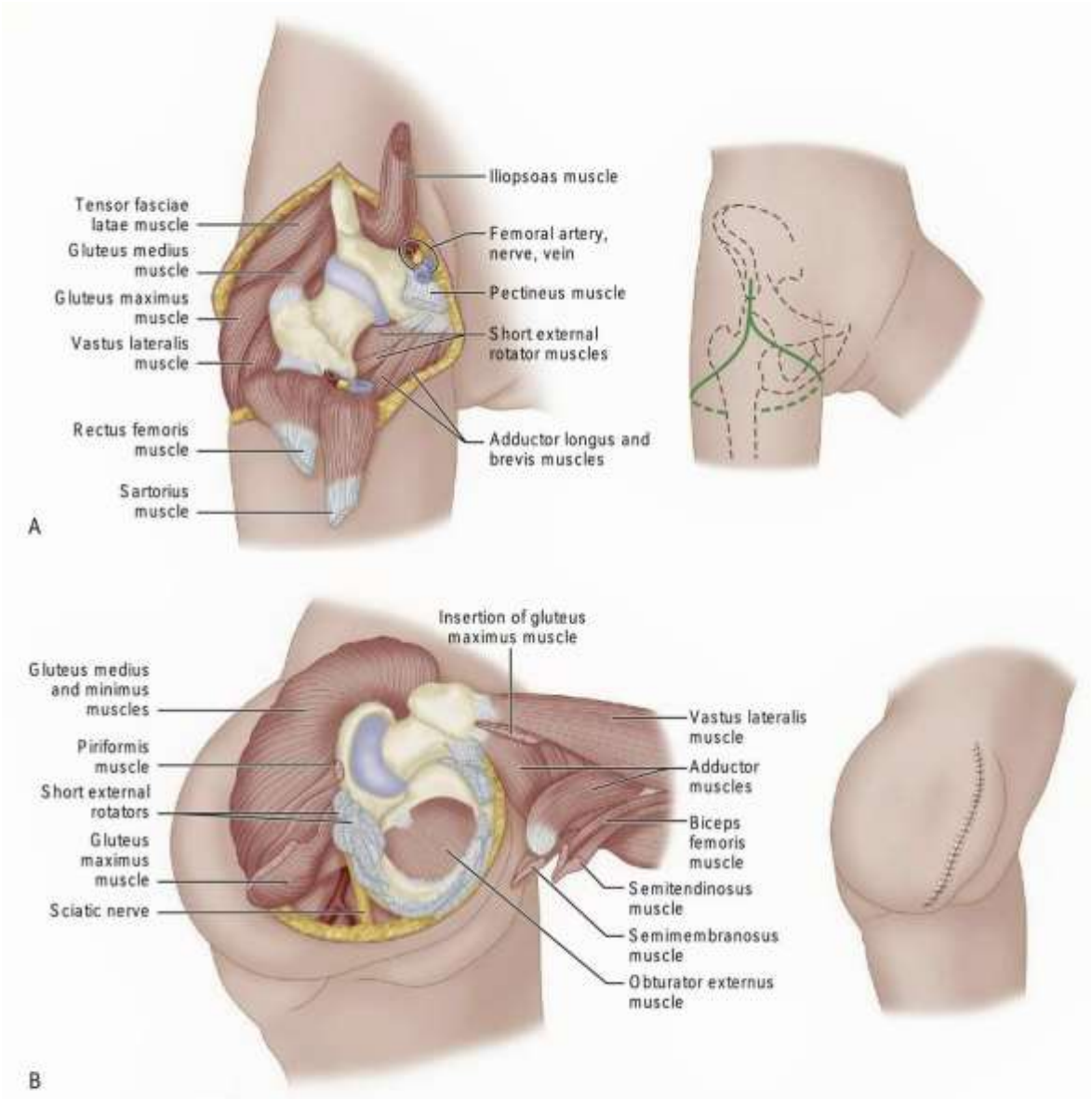


Disarticulation of the wrist. **A,** Skin incision. **B** and **C,** Reflection of the palmar flap and section of wrist joint capsule. **D,** Resection of tips of radial and ulnar styloids with preservation of the triangular ligament and underlying joint space. **E,** Completed amputation.

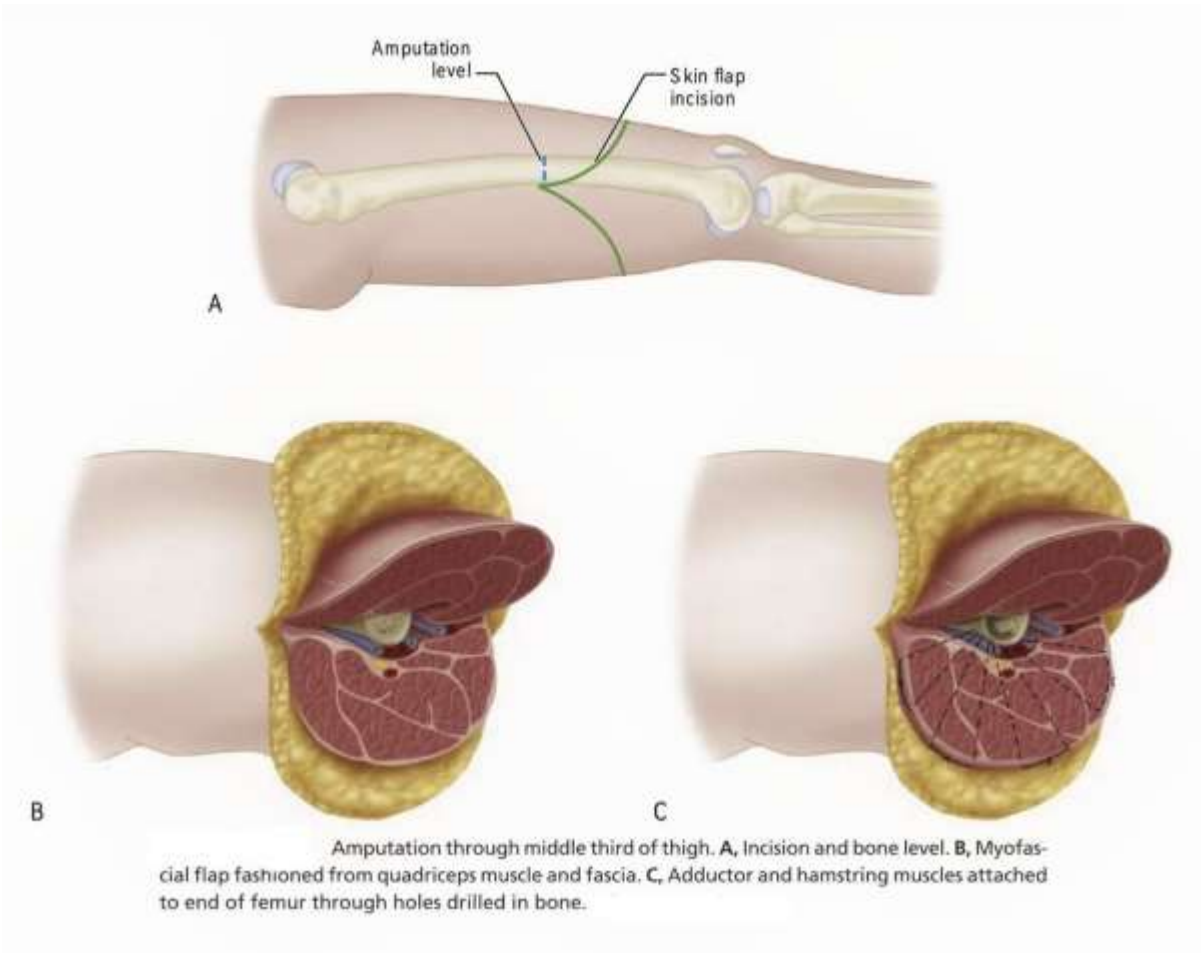
III. LOWER LIMBS AMPUTATION

- Foot Amputations
- Transtibial Amputations (below the knee): Amputation occurs at any level from the knee to the ankle
- Knee Disarticulation: Amputation occurs at the level of the knee joint
- Transfemoral Amputations (above knee): Amputation occurs at any level from the hip to knee joint
- Hip Disarticulation: Amputation is at the hip joint with the entire thigh and lower portion of the leg being removed.

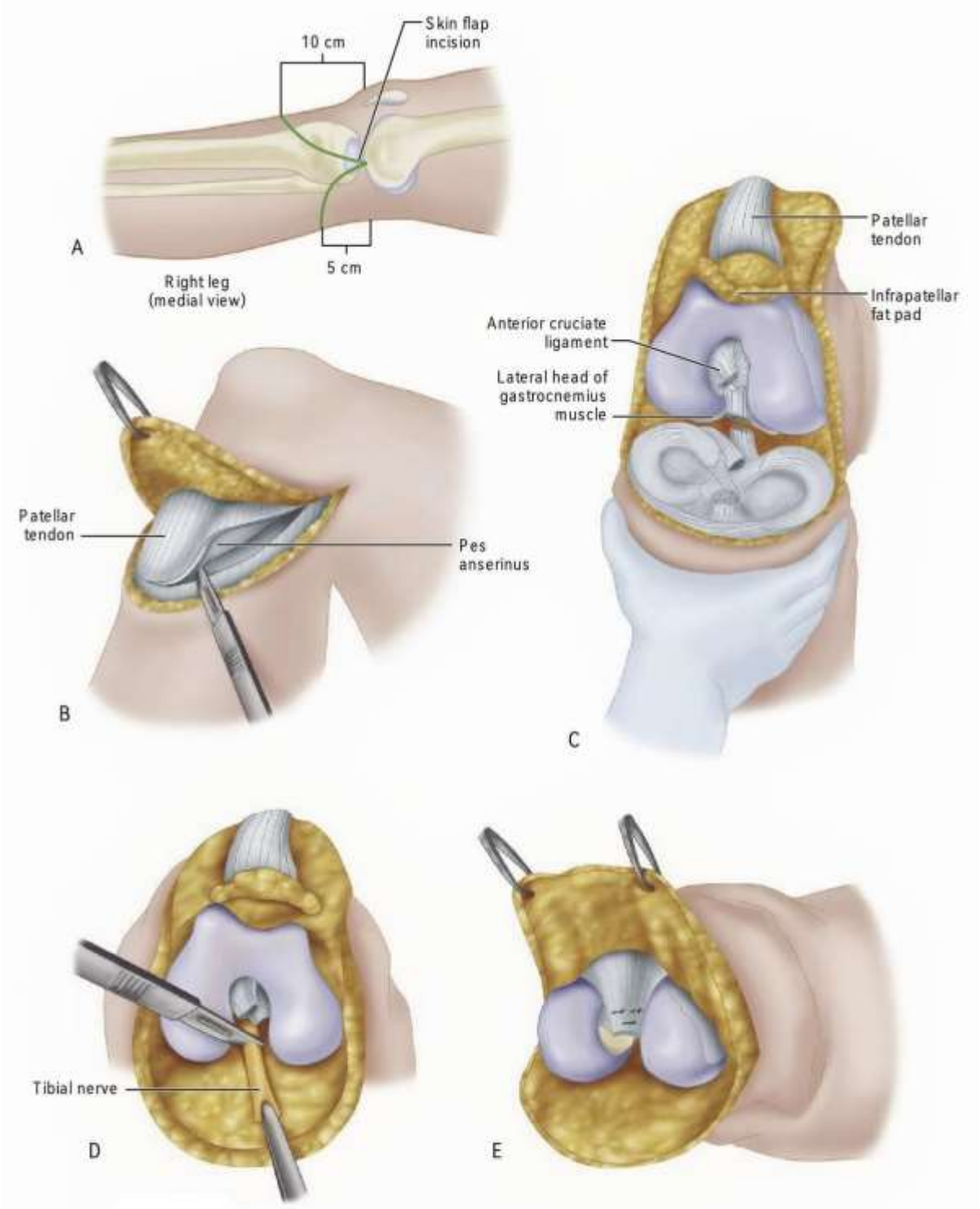




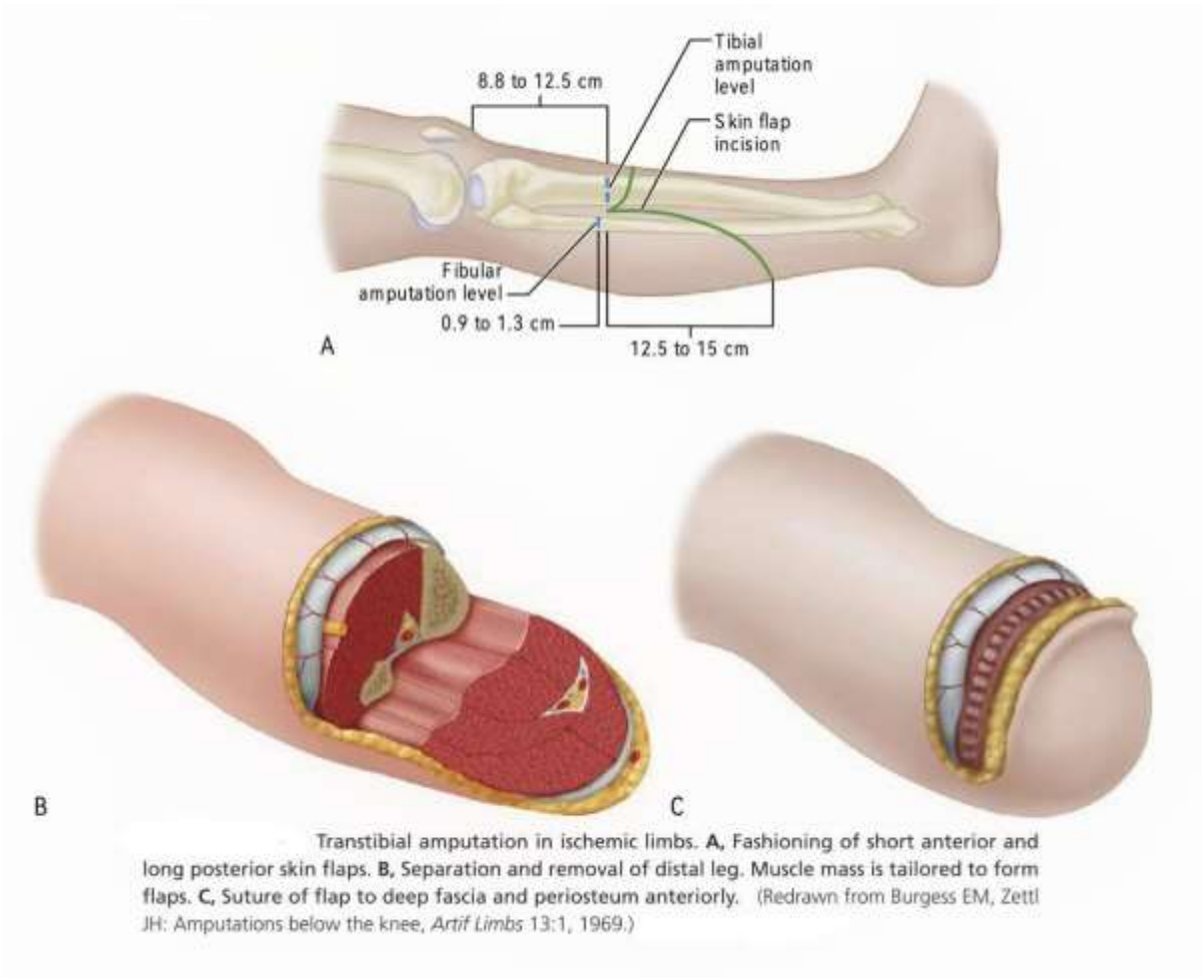
Boyd disarticulation of hip. **A,** Femoral vessels and nerve have been ligated, and sartorius, rectus femoris, pectineus, and iliopsoas muscles have been detached. *Inset,* Line of skin incision. **B,** Gluteal muscles have been separated from insertions, sciatic nerve and short external rotators have been divided, and hamstring muscles have been detached from ischial tuberosity. Inset, Final closure of stump.



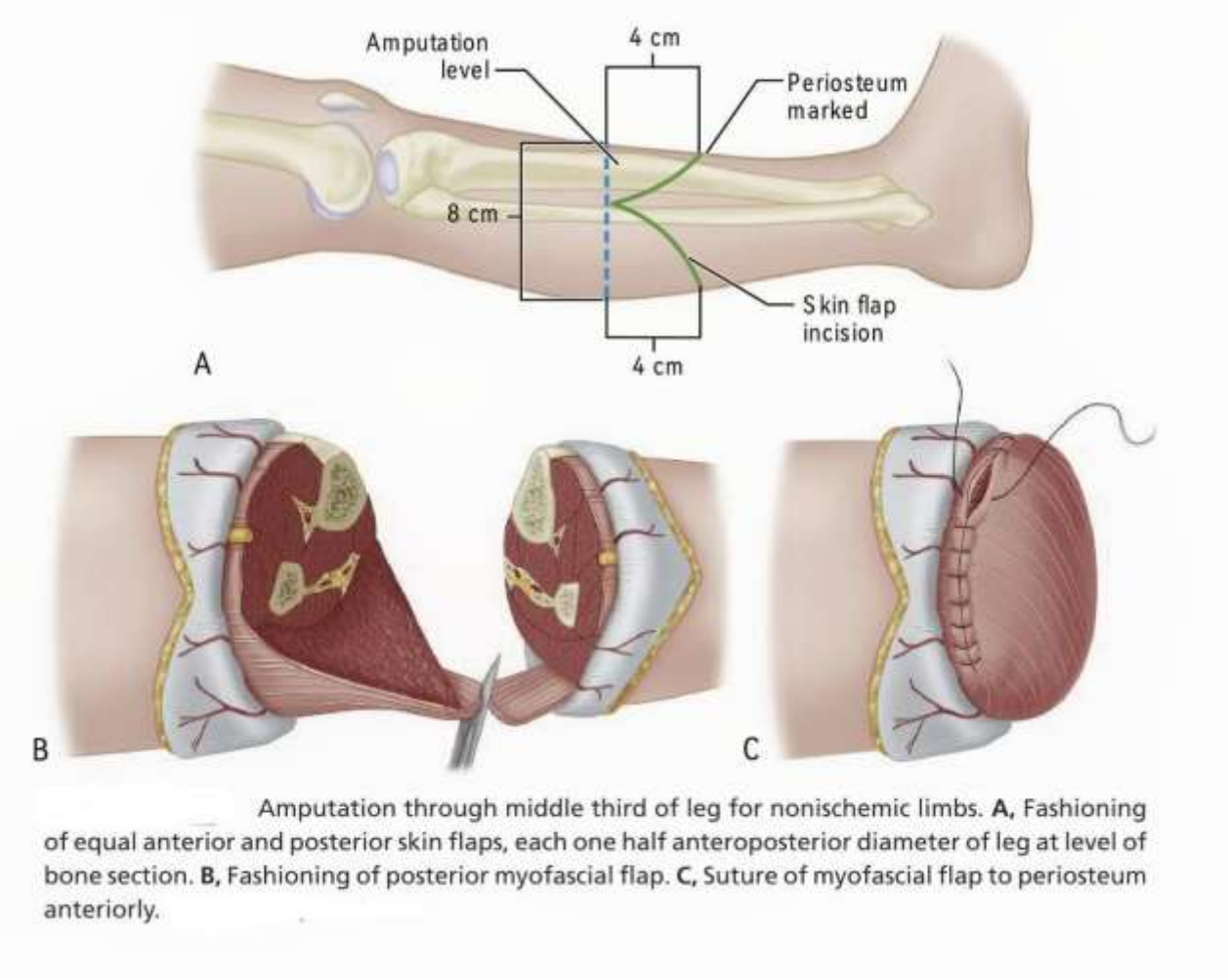
Amputation through middle third of thigh. **A,** Incision and bone level. **B,** Myofascial flap fashioned from quadriceps muscle and fascia. **C,** Adductor and hamstring muscles attached to end of femur through holes drilled in bone.



Disarticulation of knee joint. **A,** Skin incision. **B,** Anterior flap elevated, including insertion of patellar tendon and pes anserinus. **C,** Cruciate ligaments and posterior capsule divided. **D,** Tibial nerve divided high. **E,** Patellar tendon sutured to cruciate ligaments.



Transtibial amputation in ischemic limbs. **A,** Fashioning of short anterior and long posterior skin flaps. **B,** Separation and removal of distal leg. Muscle mass is tailored to form flaps. **C,** Suture of flap to deep fascia and periosteum anteriorly. (Redrawn from Burgess EM, Zettl JH: Amputations below the knee, *Artif Limbs* 13:1, 1969.)



Amputation through middle third of leg for nonischemic limbs. **A,** Fashioning of equal anterior and posterior skin flaps, each one half anteroposterior diameter of leg at level of bone section. **B,** Fashioning of posterior myofascial flap. **C,** Suture of myofascial flap to periosteum anteriorly.

IV. INDICATION

- Peripheral vascular disease (with or without diabetes) is the commonest indication for amputation. Gangrene due to atherosclerosis, embolism, TAO, etc.
- Trauma is leading indication in young patients. To save life in crush injuries
- Burns: thermal, chemical or electric may necessitate amputation.
- Frostbite
- Infections. acute/chronic infection which are unresponsive to antibiotics and surgical debridement. Most worrisome of these are infections causes by gas forming organ isms which typically result from battle field injuries; farm injuries, severe motor vehicle accidents and gunshot wounds.
- Neoplasm: Osteosarcoma, Marjolijn's ulcer, melanomas
- Severe deformity: congenital or acquired
- Principe in amputation:
- Adequate blood supply of the flap should be maintained.
- Tourniquet should not be used if amputation is done for vascular diseases.
- Proximal part of the flap contains muscle component but distal part should contain only skin and deep fascia.

- Nerve should be pulled down and cut using a sharp knife and allowed to retract into the soft tissue otherwise neuromas may develop.

- In crush injury/entrapment injury/sepsis guillotine amputation is done. Later skin is pulled down by using skin traction, eventually to have better skin coverage.
- Bone should be cut with beveling and all sharp margins should be rounded.
- Post-operatively active exercise should be given to the proximal joint so that the prosthesis can be fitted properly.
- Myodesis: muscle sutured to the bone via drill holes, establish resting tension and provides better limb control, avoid contracture
- Myoplasty: muscle of opposing compartment sutured to each other under tension and can achieve function as similar to myodesis.

V. GOALS OF AMPUTATION

- To get rid of all necrotic, infected & painful tissue.
- To have a wound that heals successfully.
- To have an appropriate remnant stump that is able to accommodate a prosthetic.

VI. PRE-OPERATIVE ASSESMENT

- Ascertain indication
- Site of amputation
- General medical condition
- Rehabilitation potential
- Counselling
- Consent
- Optimization
- Assessment of the affected limb, unaffected limb &the patient as a whole is conducted thoroughly.
- Assessment of physical, social & psychological status of the patient should be made.

VII. IDEAL STUMP

- It should be of optimum length
- The end of stump should be smooth & rounded
- It should be firm
- The opposing group of muscles should be sutured together over the end of the bone.
- The muscles are sutured in such a way that they will be converted into fibrous tissue & serve as an effective cushion.
- Vascularity of the flaps should be normal
- There should be no projecting spur of bone.
- The stump shouldn’t be under tension.
- The position of the scar should be avoided of pressure n should be transverse to avoid pulling up between 2 bones in ap scar.

- In case of Upper limb, the scar can be terminal, but in Lower limb a posterior scar is desirable to avoid pressure of weight of artificial limb.

VIII. COMPLICATION

1. Early complication:

- Haematoma
  - It is identified by pain, swelling over the stump underneath the flap.
  - It is aspirated using a wide bore needle.
  - Haematoma may delay healing; may precipitate infection or flap necrosis due to pressure.
  - After aspiration, pressure dressing is needed.
  - If haematoma reforms after 2-3 aspirations, it should be drained by opening the wound on one corner and inserting haemostat into the wound.
  - Infection of the Stump
  - It may cause abscess formation, delay in wound healing, flap necrosis, giving way of the wound.
  - Removing few or all sutures to relieve pressure and draining the pus underneath is needed.
  - Infection may also lead to poor scar, adherent scar which causes difficulty in placing the prosthesis.

2. Late complication

- Flap Necrosis
  - It is a common complication.
  - Main causes for flap necrosis are poor blood supply, infection, haematoma underneath, inadequate length of the flap causing stretching of flap.
  - Small area of necrosis can be excised.
  - Wider area needs laying opening of the wound or revision of the stump or higher level amputation.
  - Anaemia, poor nutrition, nutritional deficiencies, diabetes mellitus, immunosuppression, smoking, old age are other factors causing flap necrosis.
- Stump Neuroma
  - It can occur due to proliferation of the nerve fibrils beyond the point of nerve division and is usually due to failure of cutting of the nerve more proximal to the level of division of the bone.
  - It causes pain and tenderness over the stump. It is usually relieved by analgesics, re-assurance and prosthesis.
  - Occasionally, it may require re-exploration of the wound, excision of end neuroma and also cutting nerve more proximally.
- Stump Pain after Amputation
  - It is a common problem.
  - Causes are-infection, poor blood supply, causalgia, stump neuroma, phantom pain/limb, deep vein thrombosis, adherent scar, formation of spurs and osteophytes at amputated bone end.

- Scar adhesion to bone is prevented by keeping adequate length of deep fascia underneath intact.
- Spurs and osteophytes are confirmed by X-ray and needs removal using bone nibbler after appropriate skin incision.

➢ Phantom Limb
- It is typical awareness of sensation that as if amputated part is still present partly or in toto; often such part may be painful or disturbing or hyperaesthetic.
- Exact cause is not known, but it is probably due to presence of severe pain at the amputated part just prior to amputation making brain area for that part in alert situation causing phantom limb.
- Reassurance, prosthesis, analgesics help to control the condition.
- It is said that it can be prevented by proper pain control for 24 hours prior to amputation; but it is often difficult.
- It is common in upper limb.

➢ Ulceration over the stump
- It is not uncommon. It is due to necrosis, infection, lengthy bone stump pressing on the summit of the flap, prosthesis, nutritional deficiencies, diabetes mellitus, ischemic.
- Ulcer may be small/large; superficial/deep
- Callous chronic ulcer at the end of the stump is called as Douglas ulcer.
- Small ulcer is later treated by regular dressings and suturing.
- Large ulcer needs flap to cover the defect.
- Osteomyelitis of the stump should be ruled out in chronic stump ulcer. Ring sequestrum may be typical in such situation.
- Revision amputation is needed for the stump.

➢ Contracture of the Joint
- Contracture of the joint proximal to the amputated stump is common.
- It is mainly due improper positioning after amputation due to pain, poor exercise and occasionally due to inflammation of surrounding soft tissues.
- Contracture interferes with proper fitting and functioning of the prosthesis and delays rehabilitation.
- Proper positioning, passive stretching and exercises, strengthening exercises with help to correct it.
- Rarely needs surgical release of the contracture.

➢ Other complication
- Scar hypertrophy,
- skin thickening,
- hyperkeratosis,
- papilloma,
- Eczema,
- Lymphoedema,
- boils,
- bursae over bony point
- Spur,
- osteophyte formation,

- causalgia,
- jactitation of the stump,
- stump aneurysm,
- stump fracture

IX. REHABILITATION

- Residual Limb Shrinkage and Shaping
- Limb Desensitization
- Maintain joint range of motion
- Strengthen residual limb
- Maximize Self reliance
- Patient education: Future goals and prosthetic options

X. PSYCHOLOGICAL STRESS

Up to 2/3 of amputees will manifest postoperative psychiatric symptoms

- Depression
- Anxiety
- Crying spells
- Insomnia
- Loss of appetite
- Suicidal ideation.

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CLAVICLE FRACTURE

CHEA Huy, HENG Veasna, SONG Kimhai

I. INTRODUCTION

Fractures of the clavicle are quite common, falls on the outstretched hand and direct trauma are the likeliest causes of fractures of the clavicle.

Many clavicle fractures can be treated by wearing a sling to keep the arm and shoulder from moving while the bone heals. With some clavicle fractures, however, the pieces of bone move far out of place when the injury occurs. For these more complicated fractures, surgery may be needed to realign the clavicle bone.

Clavicle fractures are fairly common and occur in people of all ages. Most fractures occur in the middleportion, or shaft, of the bone.

Clavicle fractures vary. The bone can crack just slightly or break into many pieces (comminuted fracture). Thebroken pieces of bone may line up straight or may be far out of place (displaced fracture).

II. CAUSE

Clavicle fractures are most often caused by a Falls on the outstretched hand and direct trauma to the shoulder or an accident. In a baby, a clavicle fracture can occur during the passage through the birth canal.

III. SYMPTOMS

A clavicle fracture can be very painful and may make it hard to move your arm. Other signs and symptoms of a fracture may include:

- Inability to lift the arm because of pain
- A grinding sensation when you try to raise the arm
- A deformity or bump over the break
- Bruising, swelling, and/or tenderness over the collarbone
- Difficulty breathing or diminished breath sounds on the affected side may indicate a pulmonary injury, such as a pneumothorax

IV. IMAGING STUDIES

Clavicle Radiography or Shoulder Radiography: An anteroposterior (AP) view and a 45° cephalic tilt view are standard for the initial radiographic evaluation.

Chest radiographs: This study may be necessary to evaluate for pneumothorax, hemothorax, and rib fractures and is especially helpful in polytrauma or in patients who are comatose.

Ultrasonography: can accurately diagnose clavicle fractures in children.

V. TREATMENT

Nonsurgical Treatment: The vast majority of clavicle fractures heal with nonoperative management, which includes the use of a simple shoulder sling.

- Indication:
  - < 2cm shortening and displacement
  - < 1cm displacement of the superior shoulder suspensory complex
  - closed and no neurovascular injury
  - low demand patient
- Nonsurgical treatment may include:
  - Arm support (arm sling)
  - figure-of-8 strap to elevate and extend shoulder to bring distal fragment to the proximal fragment

Surgical Treatment: Open reduction and internal fixation.

This is the procedure most often used to treat clavicle fractures. During the procedure, the bone fragments are first repositioned (reduced) into their normal alignment. The pieces of bone are then held in place with special metal hardware.

- Indication:
  - Absolute:
    - open fractures
    - displaced fracture with skin tenting
    - subclavian artery or vein injury
    - floating shoulder (clavicle and scapular neck fracture)
  - Relative and controversial indications
    - displaced with > 2cm shortening
    - bilateral displaced clavicle fractures
    - closed head injury
    - seizure disorder
    - polytrauma patient
- Methods of internal fixation include:
  - Plates and screws
  - Intramedullary fixation

VI. COMPLICATION

Nonoperative:

- nonunion (10-15%)
- malunion
- poor cosmesis
- decreased shoulder strength and endurance

Surgical treatment:

- Infection
- Bleeding

- Problems with wound healing
- Pain
- Blood clots
- neurovascular injury
- Reaction to anesthesia
- hardware failure or migration
- Lung injury
- Numbness below the clavicle
- Hardware irritation

VII. FOLLOW-UP

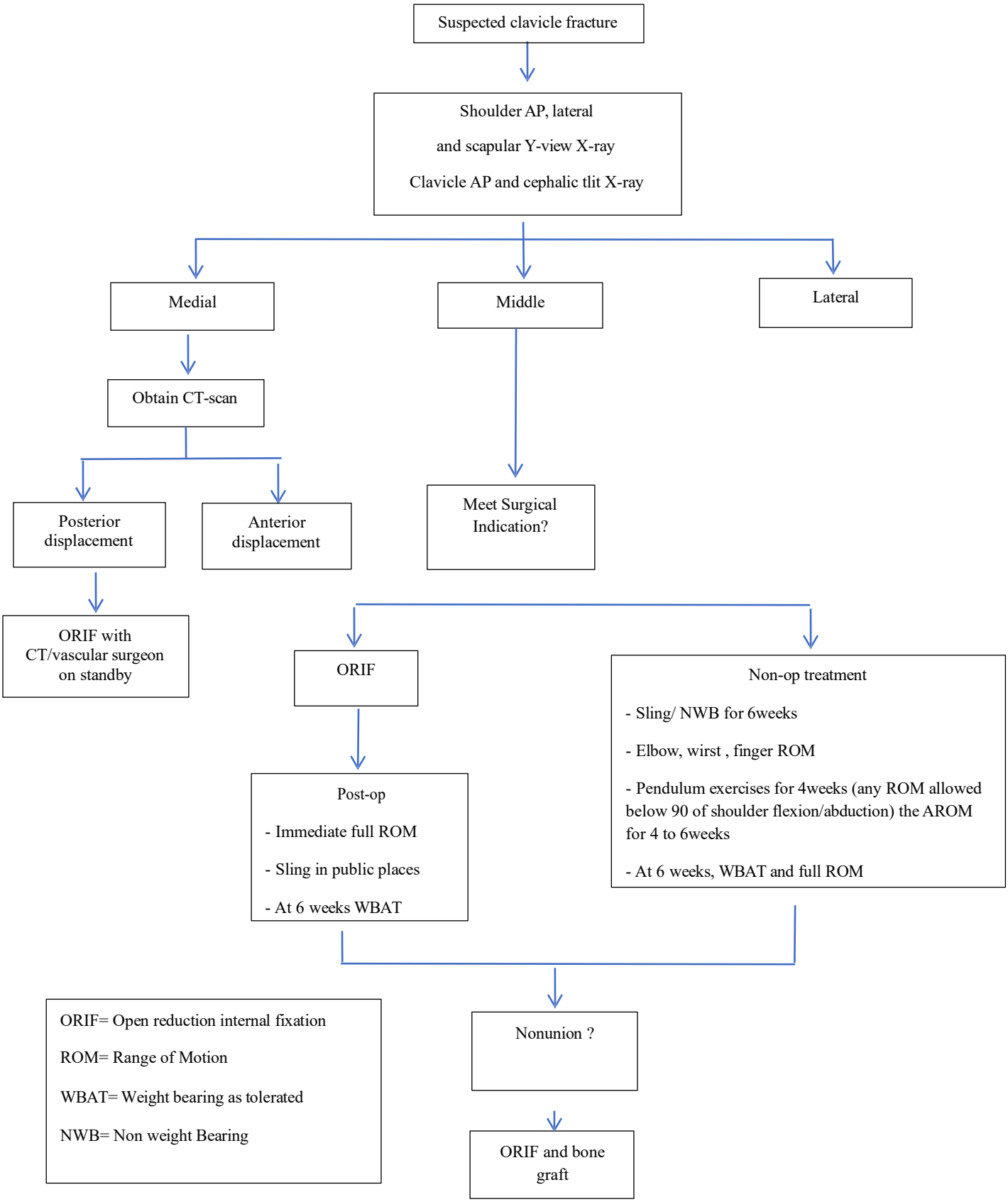
Clinical Follow-up: rehabilitation

- gentle passive range of motion exercises at 2 weeks
- strengthening exercises begin at 6 weeks
- return to sports at 3-6 months

Imaging Follow-up

- Shoulder x-ray (anteroposterior radiography) should be obtaining after 3weeks of nonoperative or surgical treatment to check for secondary displacement or complication.

VIII. ALGORITM



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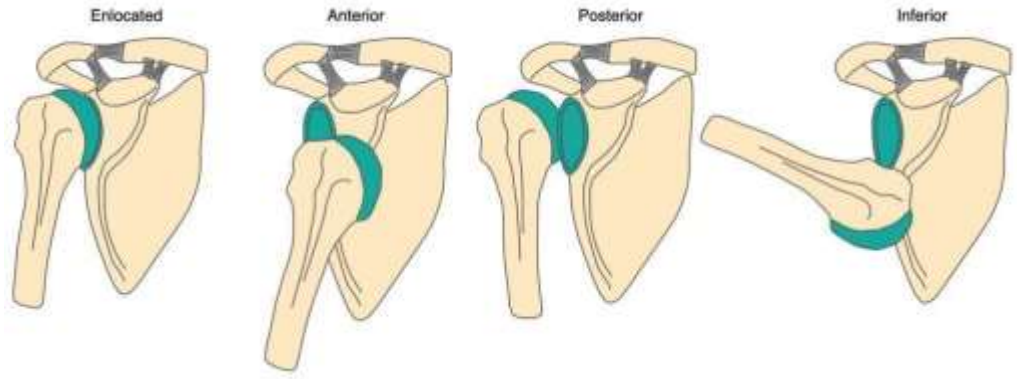
SHOULDER DISLOCATION

YIN Rith, ICH Khuy

II. DEFINITION

A dislocated shoulder is a condition in which the head of the humerus is detached from the shoulder joint. There are 3 different types of shoulder dislocation:

1. Anterior: the head of the humerus is moved toward in front of the socket(glenoid) 95% (most common).
2. Posterior: the head of the humerus is moved behind and above the socket (rarely).
3. Inferior: the head of the humerus is push down and out of the socket toward the armpit (rare)



III. AETIOLOGY

Shoulder dislocation happen when too much stress put on the shoulder such as:

1. Direct: fallen to the shoulder (rarely)
2. Indirect: fallen to the elbow or hand (most common)

IV. DIAGNOSTIC

1. **Sign clinic**
   - Intense hurt
   - Swelling
   - Difficultly to movement total functional
   - Difficultly to abduction and external rotation
   - Effacement of pectoral zone
2. **Imagery**: X-ray shoulder in AP view and lateral view.

V. TREATMENT

1. **Orthopedic**
   - Recent dislocation
   - Urgently to reduction with sedation or general anesthesia

- Immobilization with arm sling around 3weeks

2. **Surgery**
   - This may be need to open reduction if the patient had dislocated in the past or more than 2weeks
   - Dislocation with fracture associated some part surround the head of humerus bone
   - Fail to reduction
   - Recurring dislocation
   - Immobilization with arm sling around 3weeks

VI. COMPLICATION

- Vascular: axillar artery
- Nerve: axillar, circumfixed
- Tearing of the muscles
- Instability, recurrent dislocation

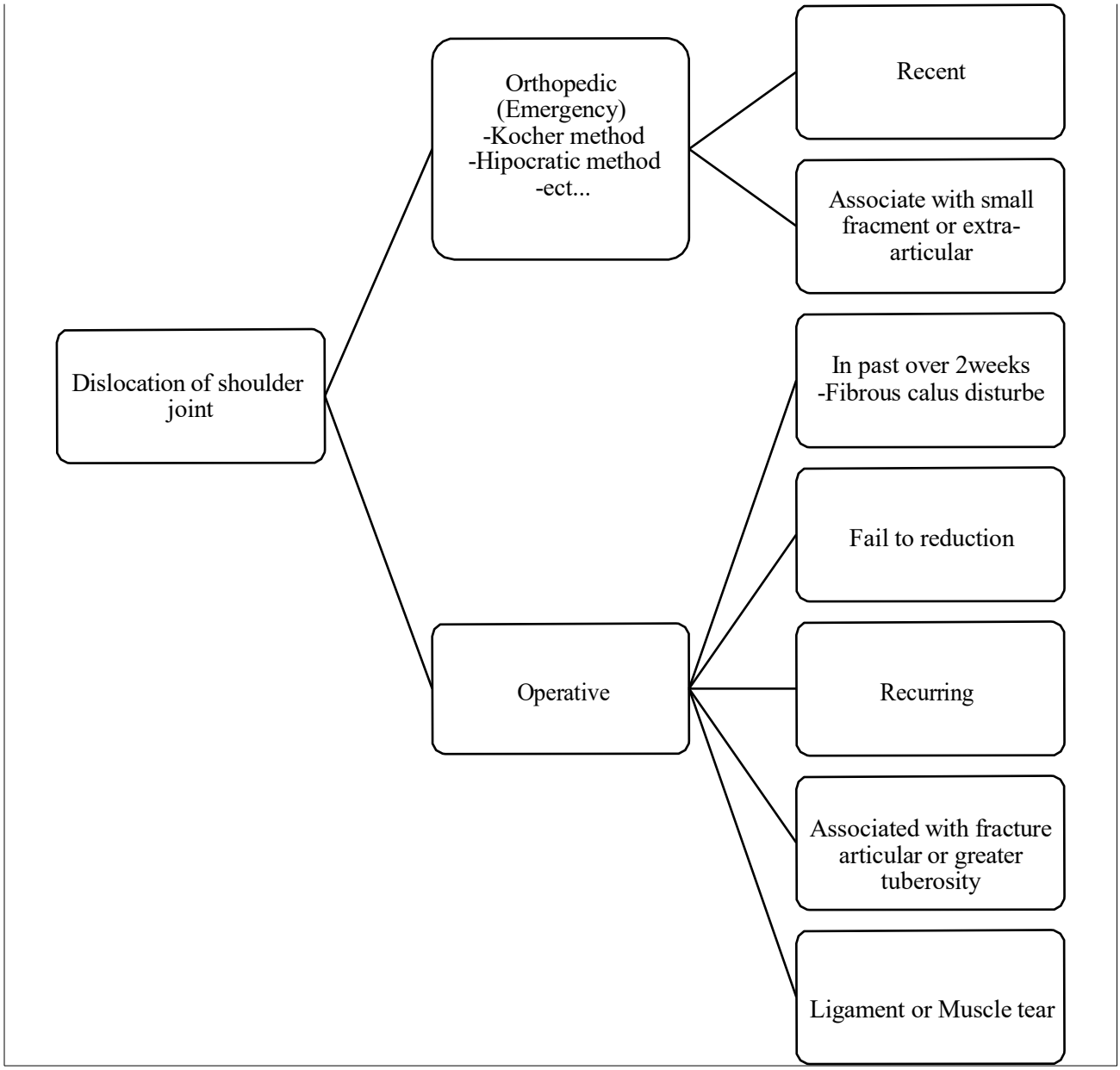
VII. PREVENTION

- Avoid to fallen down to shoulder injury
- Wearing arm sling for 3 weeks after traumatism

VIII. PROGNOSTIC

- Recurrent dislocation
- Glenohumeral necrosis
- Tendon injury
- Dislocation of the shoulder joint – Radiographic Analysis of Osseous Abnormalities by Bruno Vaned Berg
- Intermountainhealthcare.org

IX. ALGORITHM



X. REFERENCES

Mark S.Cohen,MD Rush university,by American shoulder and elbow surgeons(ASES)

https://www.orthobullets.com/video

ELBOW DISLOCATION

YIN Rith, ICH Khuy

I. GENERAL

Elbow dislocations are the most common major joint dislocation second to the shoulder

II. AETIOLOGY:

- axial loading
- supination/external rotation of the forearm
- valgus posterolateral force

III. ANATOMY:

Static and dynamic stabilizers confer stability to the elbow

- Static stabilizers: ulnohumeral oint, anterior bundle of the MCL, LCL complex , radiocapitellar joint
- Dynamic stabilizers : anconeus , brachialis , triceps

IV. CLASSIFICATION (SIMPLE VS COMPLEX)

- Simple : elbow dislocation with no associated fracture
- Complex : elbow dislocation with associated fracture

V. CLINICAL EXAMINATION

Physical exam:

- Inspection : pain , swelling , he status of the skin - evaluate for open injuries
- Palpation: compartment syndrome? neurovascular status? status of wrist and shoulder?

VI. PARACLINIC

- X-ray : AP and lateral view
- CT scan :

Indications

- ➢ suspicion of complex injury pattern
- ➢ useful to identify associated periarticular fractures

VII. TREATMENT

1. Non operative:

Closed reduction and splinting at least 90° for 5-10 days, early therapy

Indications

- ➢ acute simple stable dislocations
- ➢ recurrent instability after simple dislocations is rare (<1-2% of dislocations)

2. Operative:

ORIF (coronoid, radial head, olecranon), LCL repair, +/- MCL repair

Indications

- acute complex elbow dislocations
- persistent instability after reduction

Open reduction, capsular release, and dynamic hinged elbow fixator

Indications:

- chronic dislocations

Postoperative:

- hinged external fixator indicated in chronic dislocation to protect the reconstruction and allow early range of motion

Techniques:

Closed reduction with splinting

Assess post reduction stability

- elbow is often unstable in extension
- elbow is often unstable to valgus stress
  - ➢ test by stressing elbow with forearm in pronation to lock the lateral side

Place post-reduction posterior mold splint in flexion and appropriate forearm rotation

- splint in at least 90° of elbow flexion
- if LCL is disrupted - elbow will be more stable in **pronation**
- if MCL is disrupted - elbow will be more stable in **supination**

Obtain post-reduction radiographs

- if joint is concentric, immobilize (5-10 days) and start early therapy
- obtain repeat radiographs at 3-5 days and 10-14 days to confirm reduction

Rehabilitation:

- initial
  - ➢ immobilize for 5-10 days
  - ➢ immobilization for >3 weeks results in poor final ROM outcomes
- early
  - ➢ supervised (therapist) active and active assist range-of-motion exercises within stable arc
  - ➢ extension block brace is used for 3-4 weeks
  - ➢ proceed with light duty use 2 weeks from injury
- late rehabilitation
  - ➢ extension block is decreased such that by 6-8 weeks after the injury full stable extension is achieved

ORIF of coronoid, radial head, repair of LCL +/- MCL

Approach depends on the pathology

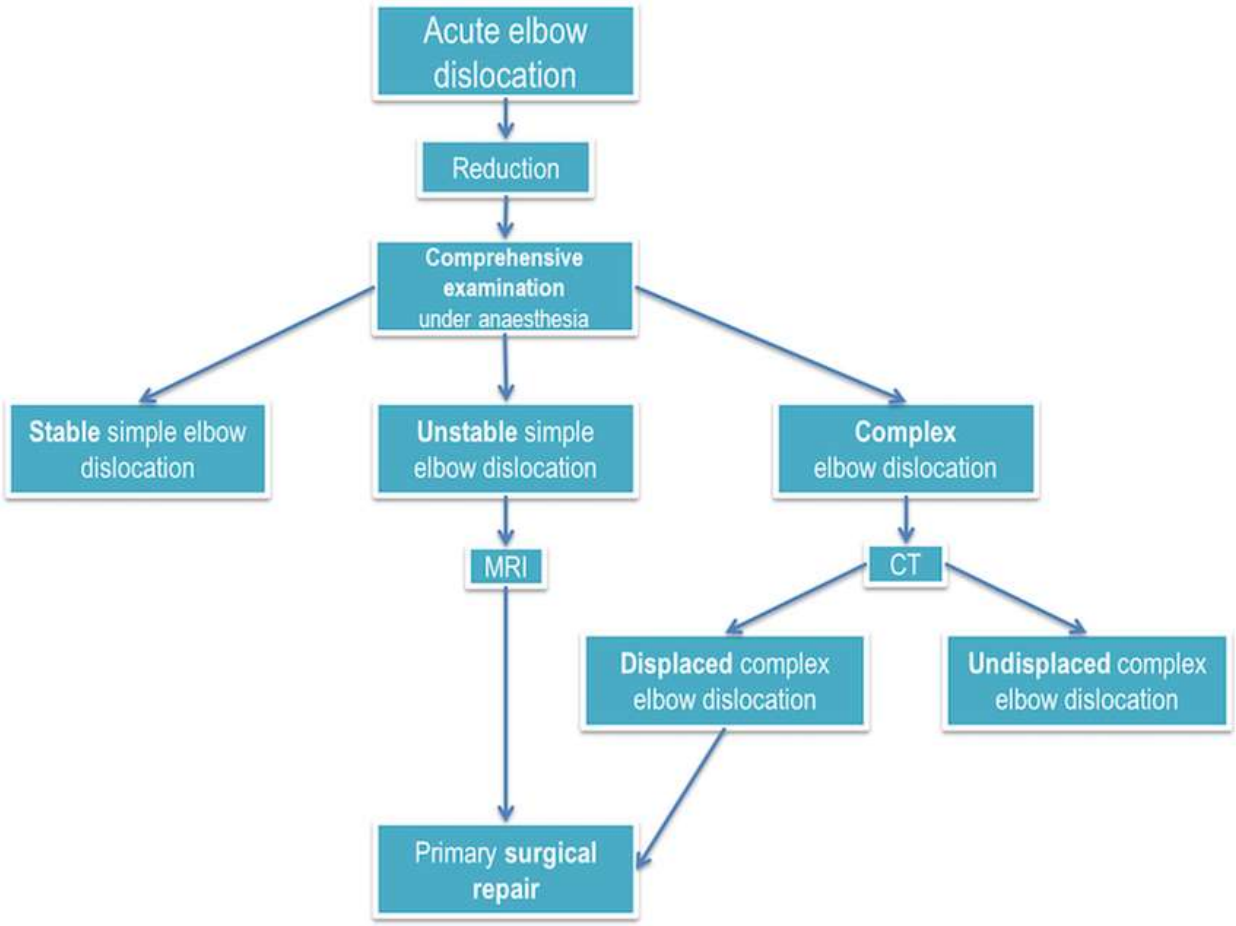
- Kocher approach (ECU/anconeus)
  - ➢ used to address the LCL complex, common extensor tendon origin, coronoid, capitellum, and/or radial head fractures

- when approaching joint (ie, for radial head fractures) during deep dissection, make incision slightly anterior to midline of the radial head to protect the posterior fibers of the LCL complex
- take care with retractor placement to avoid injury to the PIN

- medial approach
  - used to address the MCL, flexor/pronator mass origin, and/or comminuted coronoid fractures
  - identify and protect the ulnar nerve
- posterior approach

VIII. COMPLICATION:

- Early stiffness
- Varus posteromedial instability
- Neurovascular injuries
- Compartment syndrome
- Damage to articular surface
- Recurrent instability



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FEMORAL SHAFT FRACTURE

YIN Rith, ICH Khuy

I. GENERALITY

Femoral shaft fractures are high energy injuries to the femur that are associated with life- threatening injuries (pulmonary, cerebral) and ipsilateral femoral neck fractures.

Diagnosis is made radiographically with radiographs of the femur as well as the hip to rule out ipsilateral femoral neck fractures.

Treatment generally involves intramedullary nailing which is associated with >95% union rates.

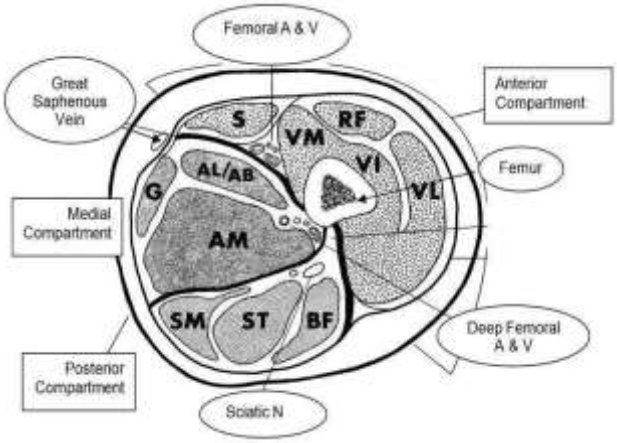
II. EPIDEMIOLOGY:

- Common: 37.1 per 100,000 population annually
- Mostly by high-energy mechanism traumatic

III. ANATOMY:

- Osteology
  - largest and strongest bone in the body
  - femur has an anterior bow
  - lineal aspera
    - rough crest of bone running down middle third of posterior fem ur
    - attachment site for various muscles and fascia
      - acts as a compressive strut to accommodate anterior bow to fem ur

- Muscles
  - 3 compartments of the thigh
    - anterior
      - sartorius
      - quadriceps
    - posterior
      - biceps femoris
      - semitendinosus
      - semimembranosus
    - adductor
      - gracilize
      - adductor longus
      - adductor brevis
      - adductor magnus



- Biomechanics
  - musculature acts as a deforming force
    after fracture

- proximal fragment
  - abducted
    - gluteus medius and minimus abduct as they insert on greater trochanter
  - flexed
    - iliopsoas flexes fragment as it inserts on lesser trochanter
- distal segment
  - varus
    - adductors inserting on medial aspect of distal femur
    - extension gastrocnemius attaches on distal aspect ofposterior femur

IV. CAUSES:

- Traumatic
  - high-energy
    - most common in younger population
    - often a result of high-speed motor vehicle accidents
  - low-energy
    - more common in elderly
    - often a result of a fall from standing
    - gunshot
- Fracture patterns
  - Transverse: pure bending moment
  - Spiral: rotational moment
  - Oblique: uneven bending moment
  - Segmental :4-point bending moment
  - Comminuted: high-speed crush or torsion mechanism

V. DIAGNOSIS

Clinical presentation:

- Symptoms
  - pain in thigh
- Physical exam
  - inspection
    - tense, swollen thigh

- blood loss in closed femoral shaft fractures is 1000-1500ml
- blood loss in open fractures may be double that of closed fractures
- affected leg often shortened
- tenderness about thigh

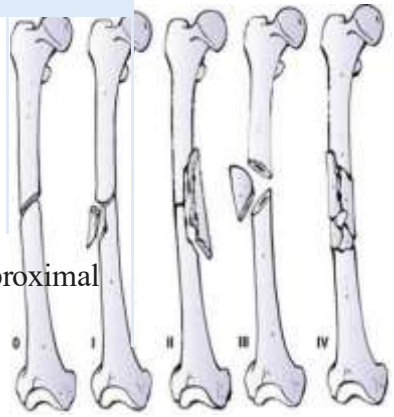
- motion: examination for ipsilateral femoral neck fracture often difficult secondary to pain from fracture
- neurovascular: must record and document distal neurovascular status

- Imaging: Radiographs, CT in case suspicion to femoral neck fracture,

VI. CLASSIFICATION

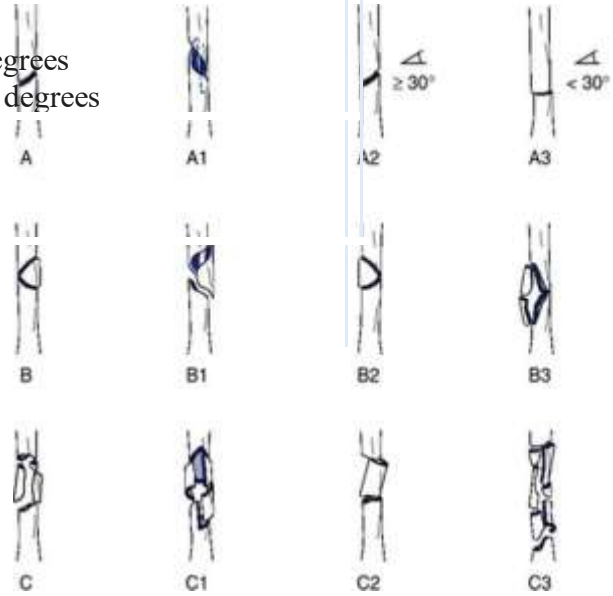
Winquist and Hansen Classification

Type 0	No comminution
Type I	Insignificant amount of comminution
Type II	Greater than 50% cortical contact
Type III	Less than 50% cortical contact
	Segmental fracture with no contact between proximal and distal fragment



AO/OTA Classification

32A - Simple	A1 - Spiral A2 - Oblique, angle > 30 degrees A3 - Transverse, angle < 30 degrees
32B - Wedge	B1 - Spiral wedge B2 - Bending wedge B3 - Fragmented wedge
32C - Complex	C1 - Spiral C2 - Segmental C3 - Irregular

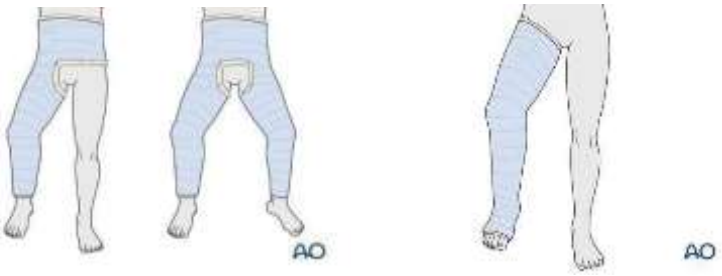


VII. TREATMENT

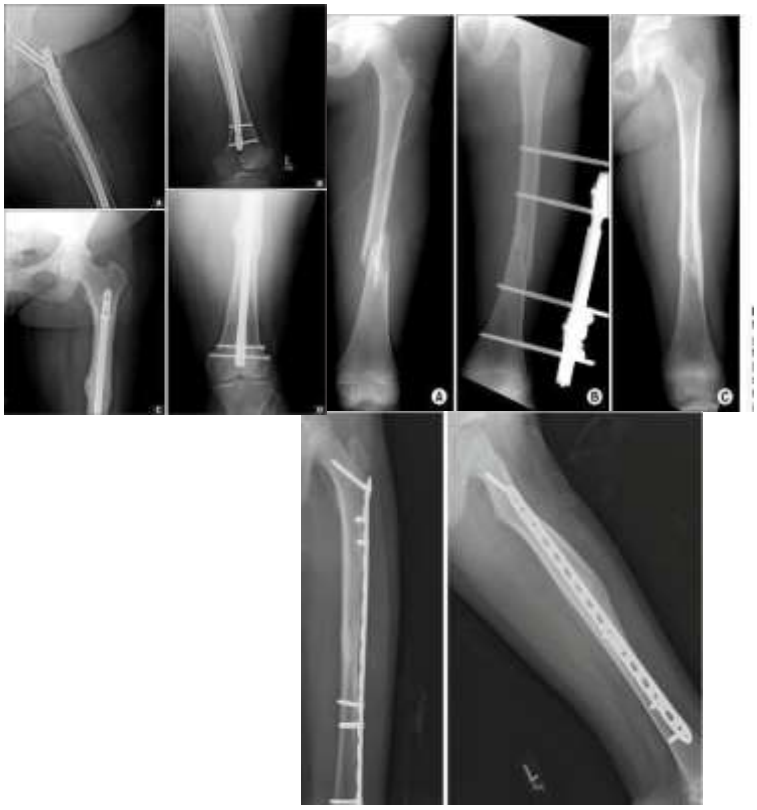
- Nonoperative

long leg cast or hip spica cast

- indications
  - nondisplaced femoral shaft fractures in patients with multiple medical comorbidities
  - pediatric patients



- Operative
  - antegrade intramedullary nail
  - retrograde intramedullary nail
  - external fixation with conversion to intramedullary nail within 2-3 weeks
  - open reduction internal fixation with plate



VIII. COMPLICATION

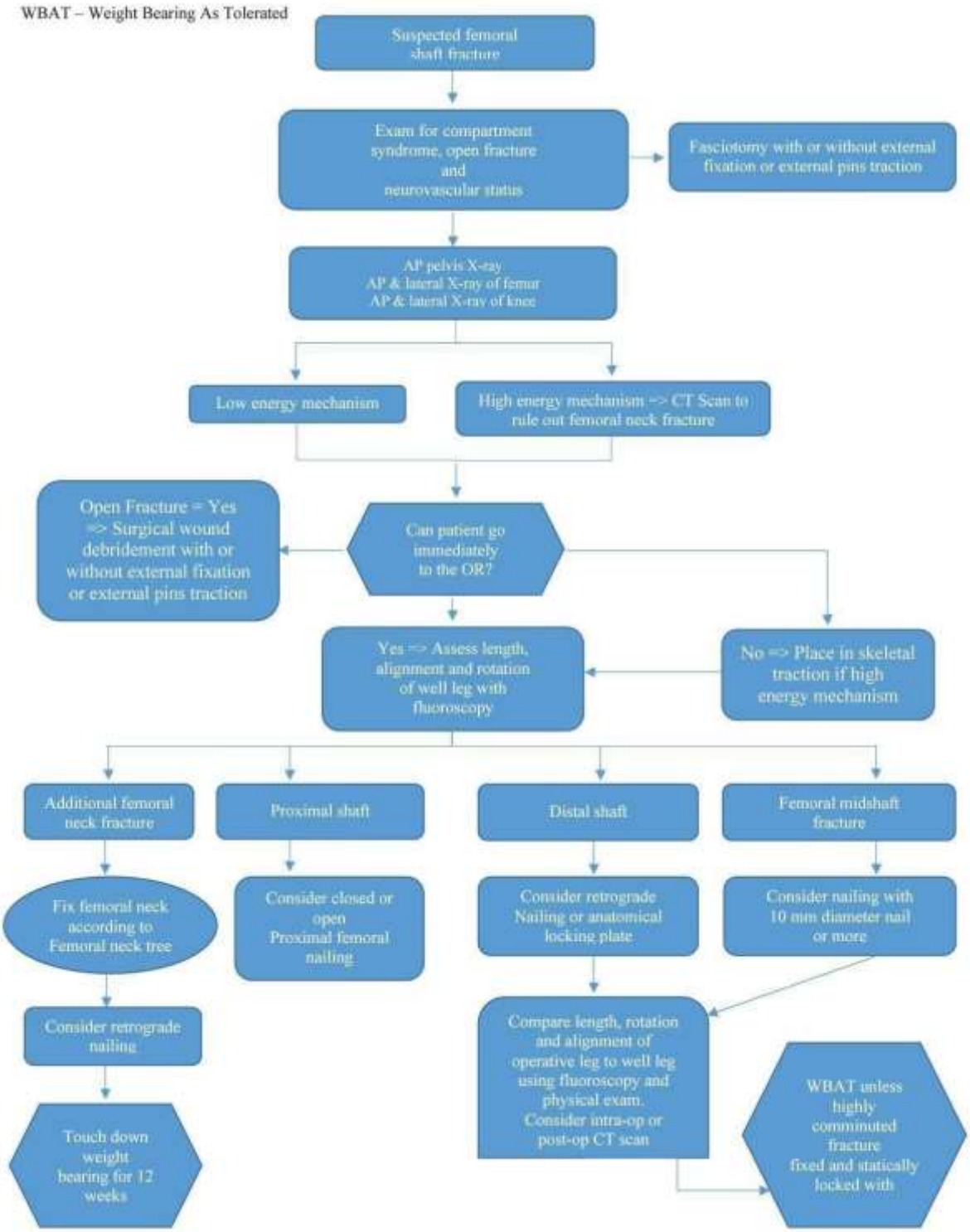
- Heterotopic ossification
- Pudendal nerve injury
- Femoral artery or nerve injury
- Malunion and rotational malalignment
- Delayed union
- Nonunion
- Weakness
- Iatrogenic fracture etiologies
- Mechanical axis deviation (MAD)
- Anterior cortical penetration

IX. PROGNOSTIC

The prognosis is good with isolated femoral fracture and most patient have a good outcome. About 30% required hardware removal.

In elderly patient it maybe associated with another medical condition but will have favorable fracture healing outcomes.

X. ALGORITHMS



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2. AO Principles of Fracture Management by Richard E. Buckley.
3. Lee C EMJ 2005; PMID: 16113195 Emerg Med J. 2005 Sep;22(9):660-3.
4. Femoral shaft fracture : Decision making in orthopedic trauma.

FOREARM FRACTURE

CHEA Huy, HENG Veasna, SONG Kimhai

I. INTRODUCTION

The forearm consists of two relatively parallel bones that connect two joints: elbow and wrist. Besides, the two bones themselves form joints that help in supination and pronation.

The forearm fractures are one of the common fractures seen in both children and adults. These fractures are relatively complex than other long bone fractures. The spectrum of such fractures includes isolated radius and ulna fractures, combined fractures, Galeazzi and Monteggia fractures

II. CAUSE

The forearm fractures may result from both low energy and high energy trauma. The most common mechanism of injury for such injuries is axial loading applied to the forearm, which is a fall onto an outstretched hand. the other common mode of injuries:

- Motor vehicle accident.
- Athletic injuries
- Falls from height

III. SYMPTOMS

- Pain and swelling
- Loss of forearm and hand function
- Physical exam
    - Inspection
      - gross deformity
      - open injuries
      - check for tense forearm compartments
    - vascular
      - assess radial and ulnar pulses
    - neurological
      - document median, radial, and ulnar nerve function
    - provocative tests
      - pain with passive stretch of fingers
      - alert to impending or present compartment syndrome

IV. IMAGING STUDIES

<u>Forearm Radiography:</u>

- An anteroposterior (AP) and lateral views of the forearm
- oblique forearm views: ipsilateral AP and lateral of the wrist and elbow
    - to evaluate for associated fractures or dislocation
    - radial head must be aligned with the capitulum on all views

V. TREATMENT

Nonsurgical Treatment: closed reduction and immobilization

- Indication:
  - completely nondisplaced fractures in patients who are not surgical candidates

Surgical Treatment:

- Closed reduction and external fixation (ExFix)
  - indications
    - severe soft tissue injury (Gustilo IIIB)
- Open reduction internal fixation (ORIF): Plates and screws
  - indications
    - All both bone fractures in surgical candidates
    - Gustilo I, II, and IIIa open fractures may be treated with primary ORIF
- Closed reduction and intramedullary fixation (IMN)
  - indications
    - very poor soft-tissue integrity

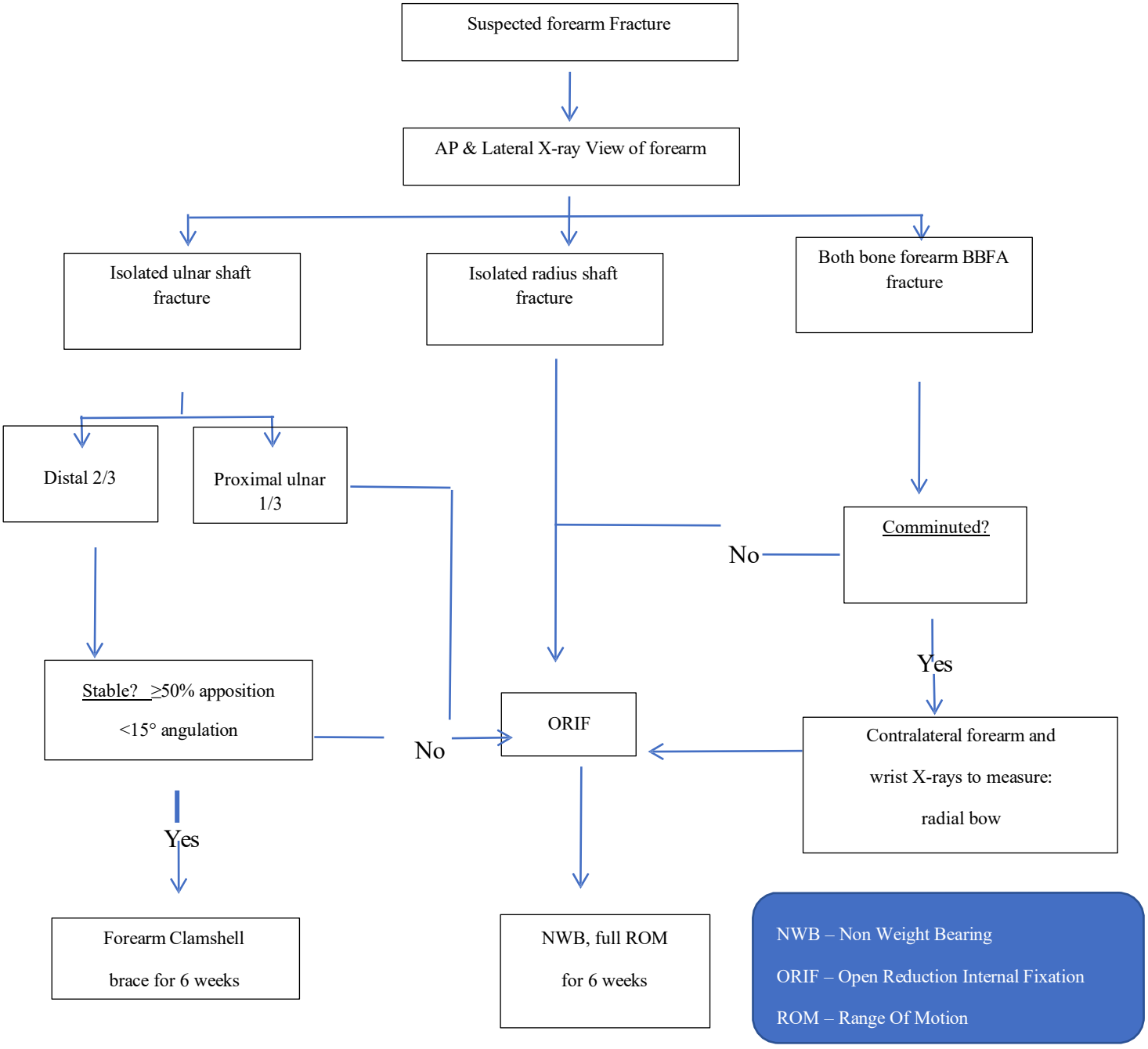
VI. COMPLICATIONS

- Nonunion
- Compartment syndrome
- Malunion
- Nerve Injury
- Infection
- Bleeding
- Problems with wound healing
- Pain
- Hardware failure or migration

VII. POST-OPERATION AND FOLLOW-UP

- Carefully examine the patient for neurological deficits and pulses.
- Surgical fixation of forearm fractures is a stable osteosynthesis of the fracture allowing early passive and active motion. This is crucial to prevent elbow stiffness.
- The rehabilitation regimen should take account of any damage to soft tissues, either as a result of the injury or due to the surgery. It also needs to take account of the security of the fixation.
- Clinical and radiological follow-up should be scheduled at least 3weeks,6 weeks, 12 weeks and 6 months after surgery and continued until a bony healing is confirmed

VIII. ALGORITHM



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2. Orthopedic trauma surgery, second edition, EMIL SCHEMITSCH, MICHAEL MCKEE, Elsevier2010.
3. AO surgery reference

HIP DISLOCATION

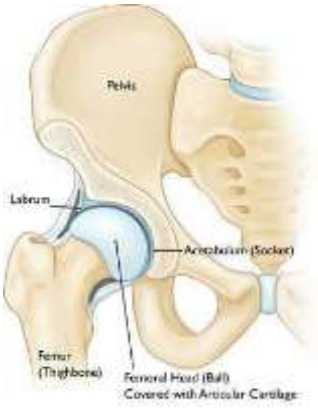
YIN Rith, ICH Khuy

I. GENERALITY:

A traumatic hip dislocation occurs when the head of the thighbone (femur) is forced out of its socket in the hip bone (pelvis). It typically takes a major force to dislocate the hip. Car accidents and falls from significant heights are common causes and, as a result, other injuries like broken bones often occur with the dislocation.
A hip dislocation is a serious medical emergency. Immediate treatment is necessary.

II. ANATOMY:

The hip is a ball-and-socket joint. The socket is formed by the acetabulum, which is part of the large pelvis bone. The ball is the femoral head, which is the upper end of the femur.



Classification:

Thomson and Epstein Classification:

Type I	With or without minor fracture
Type II	With single large fracture of the posterior acetabular rim
Type III	With a comminuted fracture of the posterior rim of the acetabulum with or without a major fragment
Type IV	With fracture of the acetabular rim and floor
Type V	With fracture of the femoral head

Pipkin Classification

Type	
I	Dislocation with fracture of the femoral head caudad to the fovea capitis femoris
II	Dislocation with fracture of the femoral head cephalad to the fovea capitis femoris
III	Type I or type II injury associated with fracture of the femoral neck
IV	Type I or type II injury associated with fracture of the acetabular rim

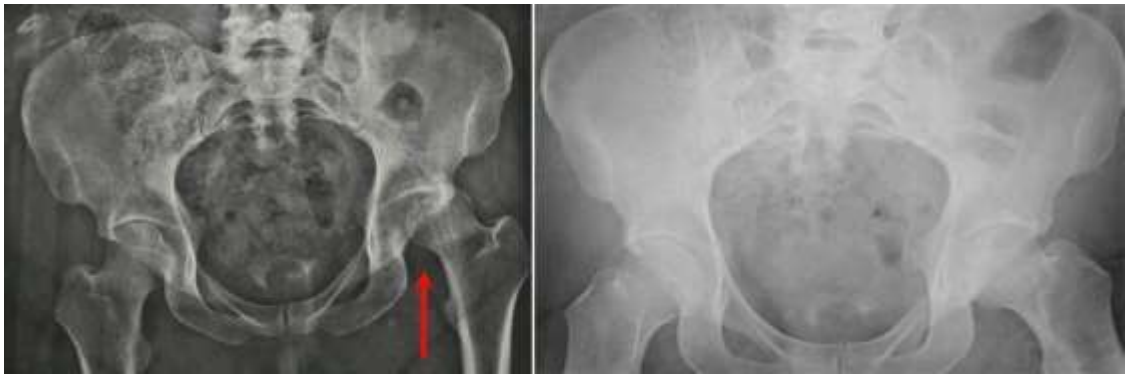
III. DIAGNOSIS:

A hip dislocation is very painful. Patients are unable to move the leg, and, if there is nerve damage, they may not have any feeling in the foot or ankle area.

A hip dislocation is a medical emergency. Call for help immediately. Do not try to move the injured person, and keep them warm with blankets.

When hip dislocation is the only injury, an orthopedic surgeon can often diagnose it simply by looking at the position of the leg. Because hip dislocations often occur with additional injuries, however, your doctor will complete a thorough physical evaluation.

Your doctor will order imaging tests, such as X-rays and likely a CT scan, to show the exact position of the dislocated bones, as well as any additional fractures in the hip or femu

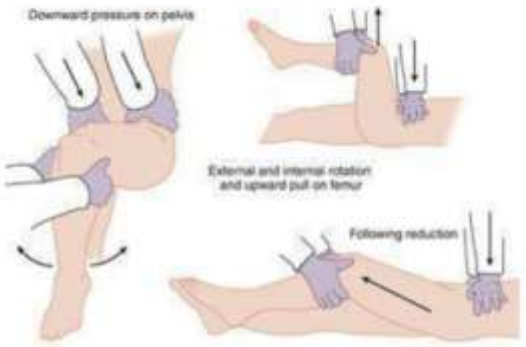


IV. TREATMENT

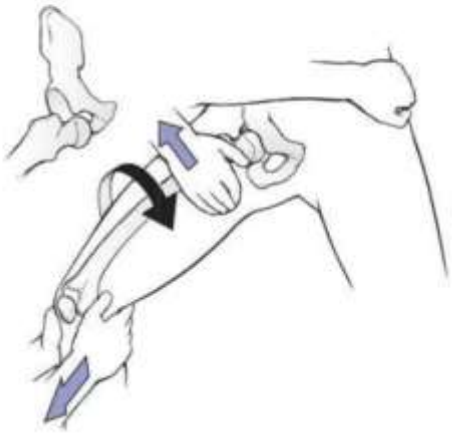
Nonsurgical Treatment

Close reduction:

Posterior Dislocation (Allis Maneuver)



Anterior Dislocation (Walker Modification of Allis Maneuver)



Surgical Treatment

Open reduction:

ORIF: (Plate, Screws)

CRPP: (Screws)

V. COMPLICATION

. Nerve injury

. Osteonecrosis

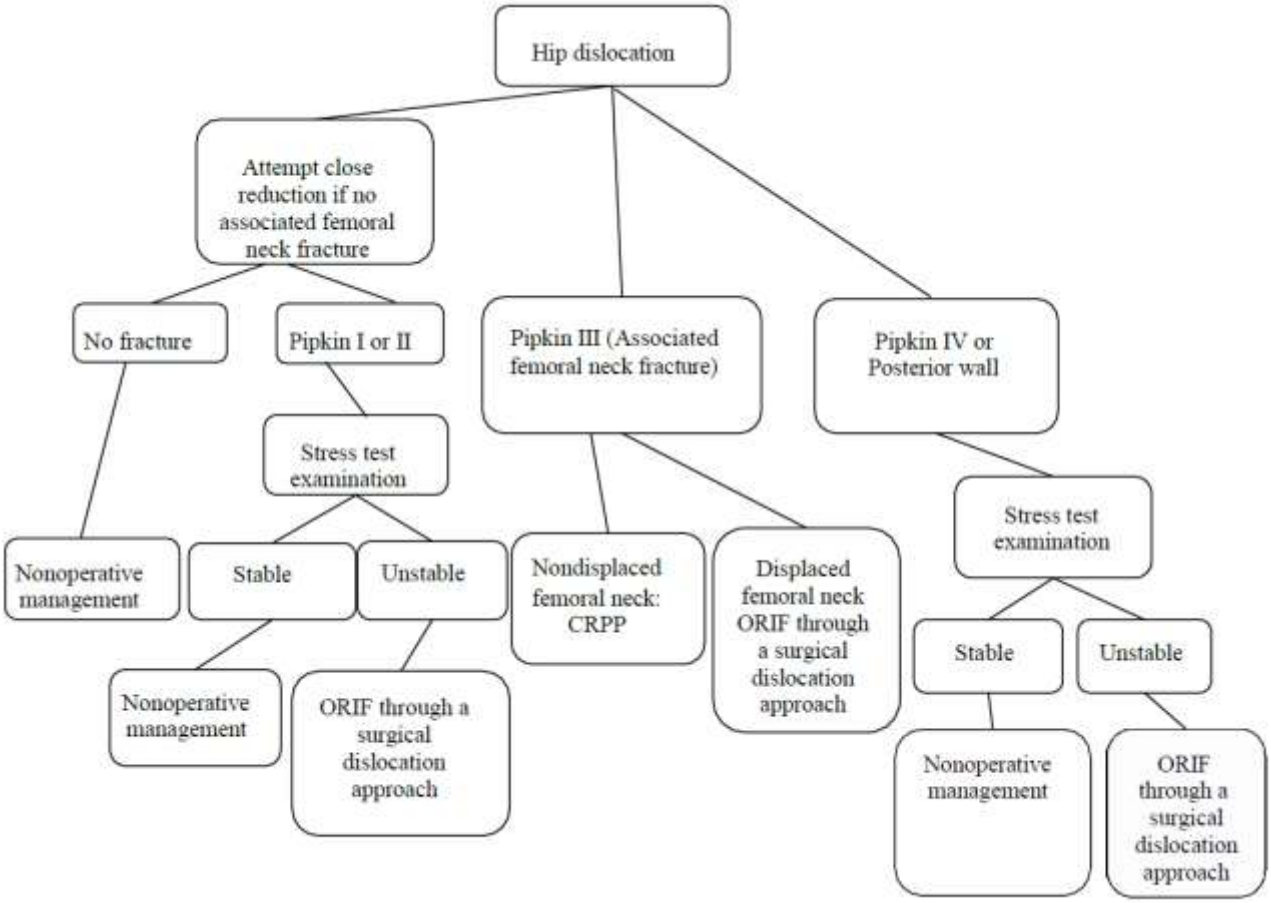
. Arthritis

VI. REFERENCES

1. Hip dislocation https://orthoinfo.aaos.org

2. Rockwood and Green's

VII. ALGORITHM



DIAPHYSEAL HUMERUS FRACTURE

Chea Huy, Heng Veasna, Song Kimhai

I. INTRODUCTION

Diaphyseal humerus Fractures or Humerus shaft fracture typically they are the result of direct trauma. They also occur in those sports where rotational forces are great, particularly baseball or arm wrestling.

Fractures of the middle and distal thirds of the shaft can give rise to injuries of the radial nerve. Vascular injury is associated with humeral shaft fractures in a small percentage of cases.

The upper arm should be examined for swelling, ecchymosis, and deformity. The entire limb is carefully examined for vascular and neurological changes. Evaluation of the function of the radial nerve is especially crucial prior to any reduction

II. CAUSES

Humerus shaft fracture typically they are the result of:

- Motor vehicle accident (direct trauma).
- Pathologic fractures
- They also occur in those sports where rotational forces are great, particularly baseball or arm wrestling.

III. SYMPTOMS

- Pain
- extremity weakness
- swelling
- tenderness over the fracture site
- skin tenting
- limb deformity
- neurovascular exam (examine and document status of radial nerve pre and post-reduction)

wrist and thumb interphalangeal joint extension sensation over the dorsum of the hand

IV. IMAGING STUDIES

Humerus Radiography: An anteroposterior (AP) and lateral view be sure to include joint above and below the site of injury.

V. TREATMENT

Nonsurgical Treatment: immobilization (coaptation splint or hanging arm cast for 7 to 10 days followed by a functional brace)

- Indication:
  - < 20° anterior angulation
  - < 30° varus/valgus angulation
  - < 30° of rotational malalignment
  - < 3 cm shortening

Surgical Treatment:

1. **External fixation (Exfix)**

- indications
  - high energy complex or comminuted fracture
  - open fracture
  - significant soft tissue or bony defect
  - floating elbow
  - hemodynamically unstable polytrauma
  - concomitant vascular injury
- typically utilized as provisional fixation until definitive treatment can be performed, but may be used definitely if needed

2. **Open reduction internal fixation (ORIF) and minimally invasive plate osteosynthesis**

- indications
  - open fracture
  - vascular injury requiring repair
  - brachial plexus injury
  - radial nerve palsy
  - ipsilateral forearm fracture (floating elbow)
  - periprosthetic humeral shaft fractures at the tip of the stem
  - inability to maintain satisfactory close reduction

- progressive nerve deficit after closed manipulation
- segmental fractures

3. Intramedullary nailing (IMN)

- indications
  - pathologic fractures
  - segmental fractures
  - severe osteoporotic bone
  - overlying skin compromise limits open approach
  - polytrauma

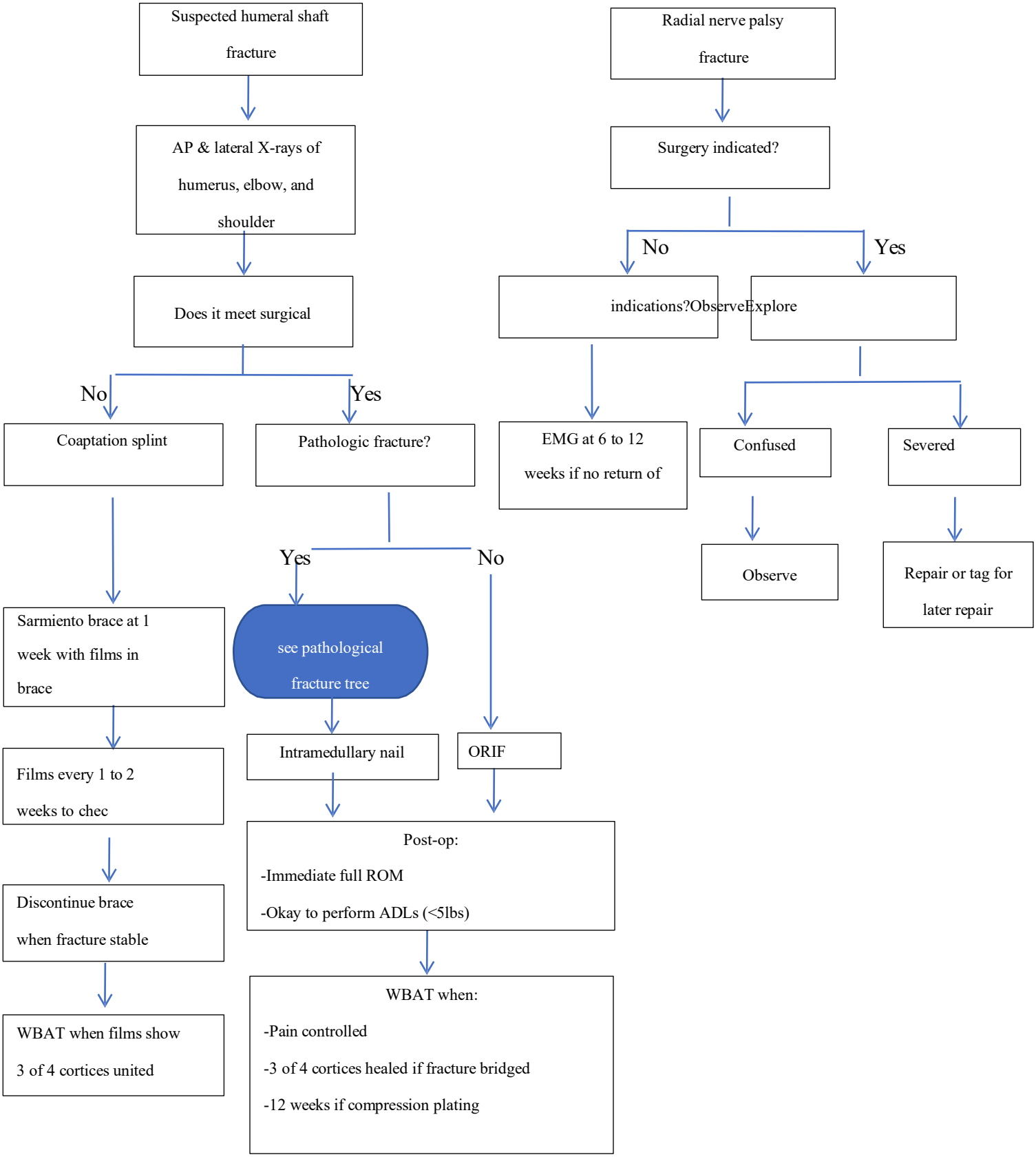
VI. COMPLICATION

- nonunion
- malunion
- Radial nerve palsy
- Infection
- Bleeding
- Problems with wound healing
- Pain
- hardware failure or migration

VII. POST-OPERATION AND FOLLOW-UP

- Carefully examine the patient for neurological deficits and pulses.
- The aim of any surgical fixation of humeral shaft fractures is a stable osteosynthesis of the fracture allowing early passive and active motion. This is crucial to prevent elbow stiffness. return to sports at 3-6 months
- The rehabilitation regimen should take account of any damage to soft tissues, either as a result of the injury or due to the surgery. It also needs to take account of the security of the fixation.
- Clinical and radiological follow-up should be scheduled at least 6 weeks, 12 weeks and 6 months after surgery and continued until a bony healing is confirmed

VIII. ALGORITHM



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LOWER LIMB IMMOBILIZATION

Authors: Chheur Hengnaroth, Sok Chanpheaktra, Ly Kang, Kim Sopharktra, Huot Vutha, Ang Eng Sopheap

I. SARMIENTO CAST

1. Indication
2. Goal of treatment
3. Equipment
4. Position
5. Procedure

II. SHORT LEG CAST

1. Indication
2. Goal of treatment
3. Equipment
4. Position
5. Procedure

III. DORSAL LONG LEG SPLINT

1. Indication
2. Goal of treatment
3. Equipment
4. 1st Position
5. Procedure
6. 2nd Position
7. Procedure

IV. CYLINDER LONG LEG CAST

1. Indication
2. Goal of treatment
3. Equipment
4. Position
5. Procedure

V. DORSAL SHORT LEG SPLINT

1. Indication
2. Goal of treatment
3. Equipment
4. 1st Position
5. Procedure
6. 2nd Position
7. Procedure

References

I. Sarmiento cast (patella tendon bearing)

1. Indication

- Stable open or close diaphyseal fractures of the tibia limited to the distal 2/3 of the tibia shaft
- Primary unstable tibial fractures at the hard callus formation stage

2. Goal

- Stabilization of the lower leg

3. Equipment

EQUIPMENT

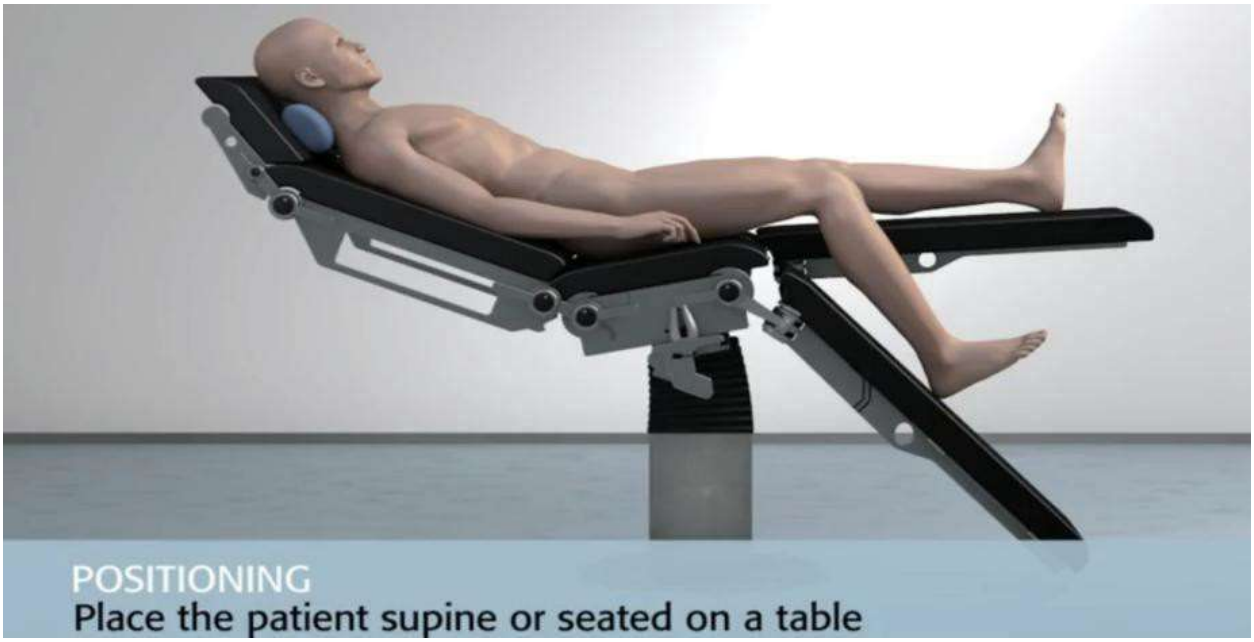
1 Cast padding
2 Plaster of Paris splint 12 cm or 15 cm
3 Plaster of Paris rolls 12 cm or 15 cm
4 Tube bandage 7.5 cm in dispenser box
5 Crepe paper bandage
6 Cut tube bandage
7 Scissors

PERSONNEL

1

4. Positioning

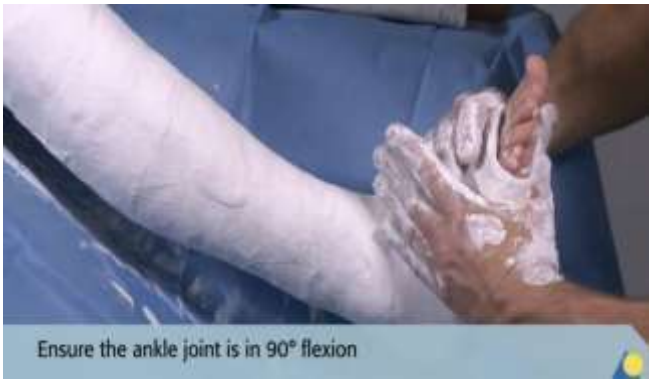
- Patient supine or seat on the table

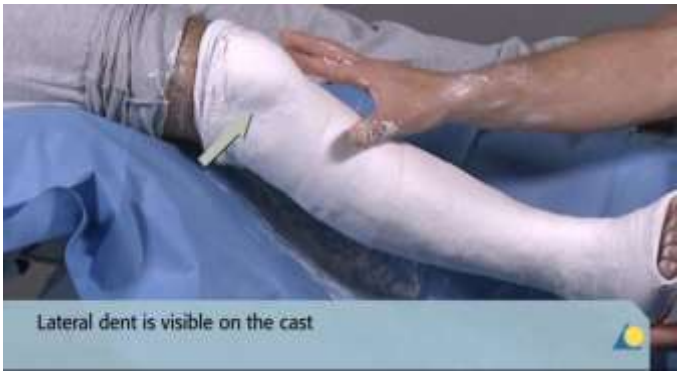


5. Procedure

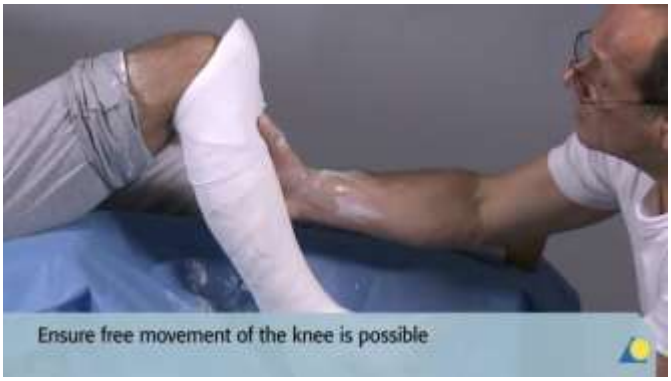
- Apply a tube bandage from the foot to over the knee, apply the padding using the half-overlapping technique
- Ensure the tube bandage extends past the femoral condyles, padding covers the entire patella
- Apply a thick layer of padding over the patella and tibial tuberosity
- Prepare the POP splint and submerge the splint in 20 degree warm water then squeeze out excess water and apply the roll of POP starting distallyn using half-overlapping technique
- Add extra layers of POP over the patella
- Prepare a POP splint to be applied ventrally, and over the patella with fold over the proximal corners
- Stretch and smooth out the splint, pressing the layers together, resulting in a compact splint
- Trim and mold it to the knee
- Prepare U Shape of POP splint and submerge in 20 degree warm water then apply a short POP splint across the tibial condyles for extra support then mold it
- Apply the roll of POP, from distal to proximal
- Mold perfectly to the patella tendon and the tibial condyles, medial and lateral of the tibial tuberosity
- Mold the splint and hold the ankle joint in 90 degree flexion
- Ensure free movement of the knee is possible

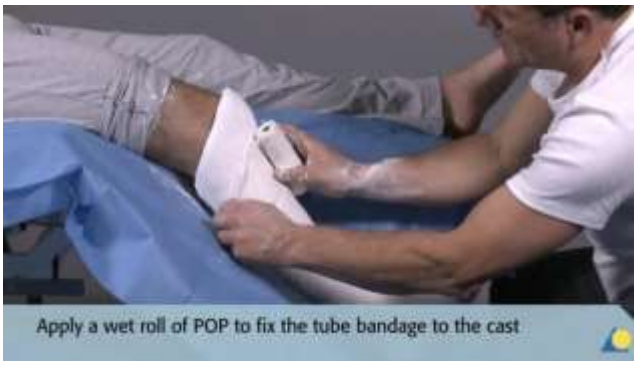

As the POP begins to set, it must be perfectly molded to the patella tendon and the tibial condyles

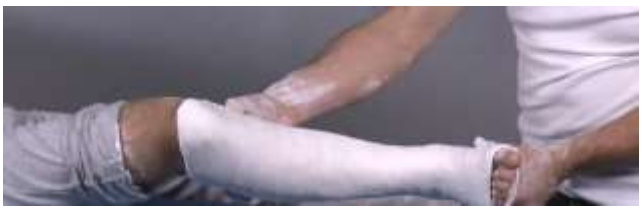

Ensure the ankle joint is in 90° flexion

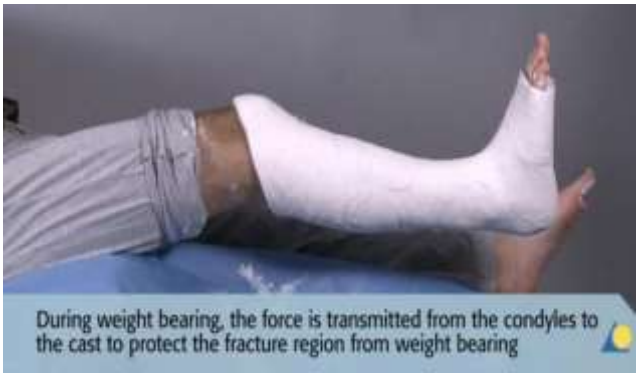

Lateral dent is visible on the cast


Trim the cast with cast scissors or an oscillating saw


Ensure free movement of the knee is possible


Apply a wet roll of POP to fix the tube bandage to the cast





Special things to keep in mind

- Leave the window open on the open wound
- Support at the patella tendon and the tibial condyles
- Free movement of the knee joint
- Free movement of the toes

II. Short leg cast

1. **Indication**
   - Stable open or close metaphyseal distal tibial and fibular fracture
   - Ankle injury (ligamentous injury)
2. **Goal**
   - To prevent rotation of the leg
3. **Equipment**

EQUIPMENT

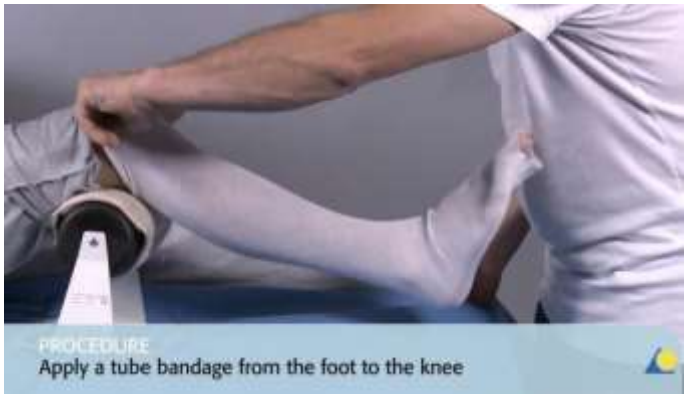
1 Rigid synthetic splint 7.5 cm x 45 cm
2 Elastic foam tape
3 Rigid synthetic splint 7.5 cm x 45 cm
4 Cut tube bandage
5 Broomstick
6 Tube bandage 7.5 cm in dispenser box
7 Semirigid casting tape 7.5 cm
8 Scissors
9 Gloves
10 Elastic bandage

PERSONNEL

1

4. **Positioning**
   - Place the patient supine on the table
   - Knee on support, 45-60 degree flexion
   - Ankle joint 90 degree flexion

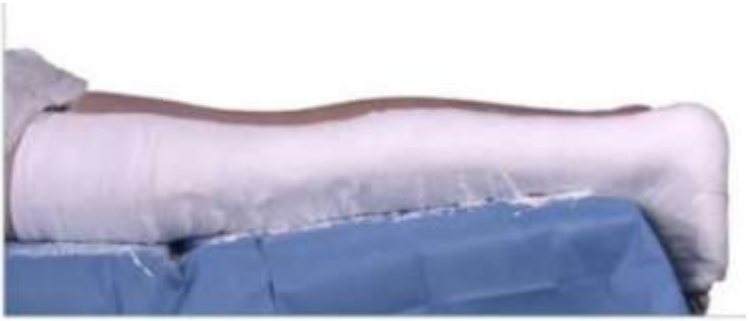
5. **Procedure**
   - Apply tube bandage from foot to knee
   - Pull bandage tight to avoid any wrinkles
   - Apply elastic foam tap to protect the bony prominences: lateral malleolus and medial malleolus, extensor tendons, tibial crest
   - Submerge casting tape in water 20celcus
   - Apply the cast tape around forefoot and leaving the toes free and pass over foot to the ankle joint, the Achilles tendon and over the leg by using half-overlapping technique
   - Half overlapping until proximally and fold back the tube bandage over cast tape proximally and distally and trim it
   - Apply another layer of wet rigid casting tape over the rigid splint
   - Wrap the complete cast with the elastic bandage and mold the cast to the desired position
   - Tap on the cast to check it has set, and remove the wet bandage



Special thing to keep in mind

- Leave the window open on the open wound
- Ensure the fibula head remains free a minimum of 2cm to avoid pressure on the peroneal nerve
- Free flexion of the knee
- Free flexion and extension of the toes, pressure sores
- No disturbance of the contralateral leg

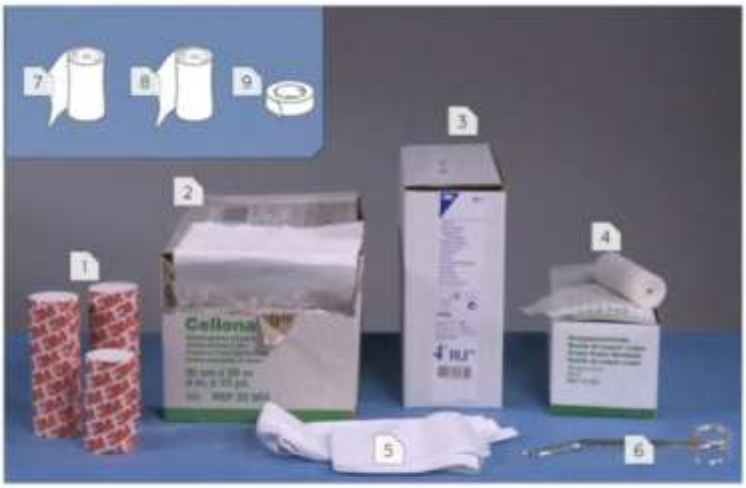
III. Dorsal long leg splint



1. **Indication**
   - Close or open fracture of the tibia and fibula
   - Close or open fracture around the knee

2. **Goal**
   - Stabilization of the knee and lower leg

3. **Equipment**



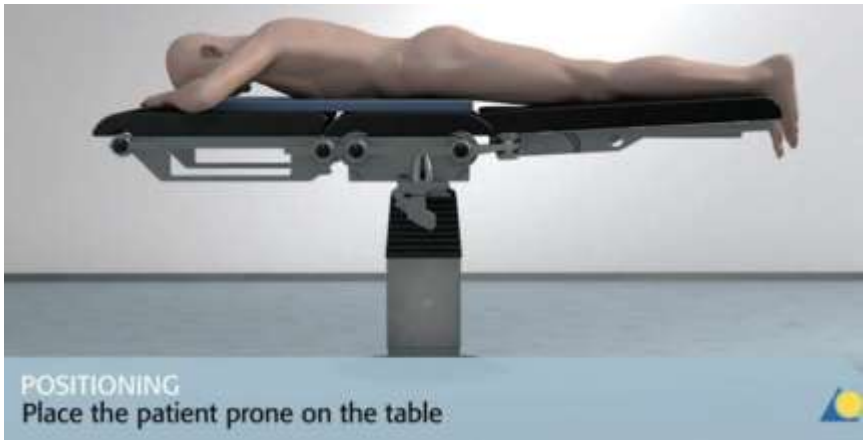
EQUIPMENT

1 Cast padding
2 Plaster of Paris splint 20 cm
3 Tube bandage 10 cm in dispenser box
4 Crepe paper bandage
5 Cut tube bandage
6 Scissors
7 Elastic bandage
8 Gauze bandage
9 Surgical tape or bandage clips

PERSONNEL

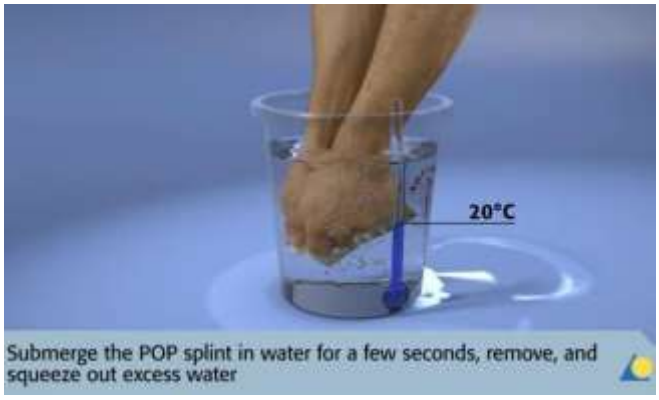
2

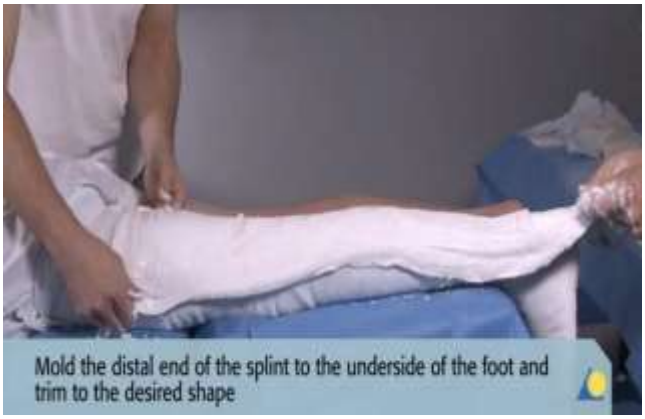
4. **1st Position**
   - Prone position on the table



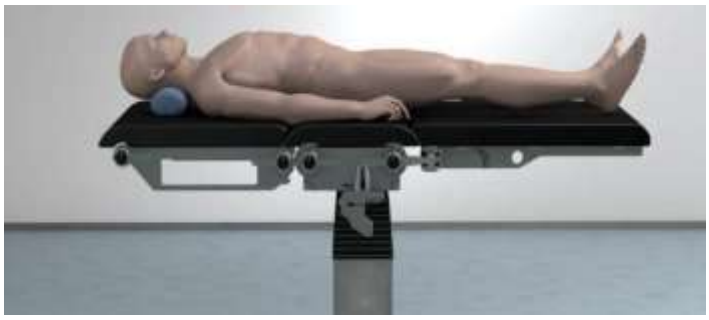
5. **Procedure**
   - Apply a tube bandage up to the hip and pull it tight to avoid any wrinkles
   - Apply padding over the tube bandage, starting from foot and ensure the ankle joint is at 90 degree flexion
   - Wrap the leg using the half-overlapping technique
   - Apply a paper bandage to form a barrier between the dry padding and the wet POP and wrap the leg using the half-overlapping technique
   - Measure the length of POP splint needed, from the toes to the lesser trochanter-greater trochanter

- Submerge the POP splint in warm water 20 degree and stretch and smooth out the splint, pressing the layers together, resulting in a compact splint
- Put the POP splint, mold the distal end of the splint to the underside of the foot and trim to the desire shape
- Apply gauze bandage to the lower leg, from distal to proximal, to hold the splint in place
- The assistant lifts the leg during application of the gauze bandage and wrap the lower leg using the half-overlapping technique to the upper leg
- Ensure the knee is at 10 degree flexion and ankle joint 90 degree flexion
- Mold the splint over the Achilles tendon, both malleoli, the condyles, and there is support for the thigh muscle

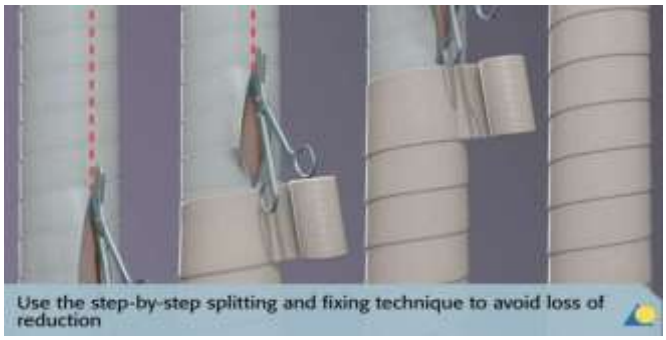


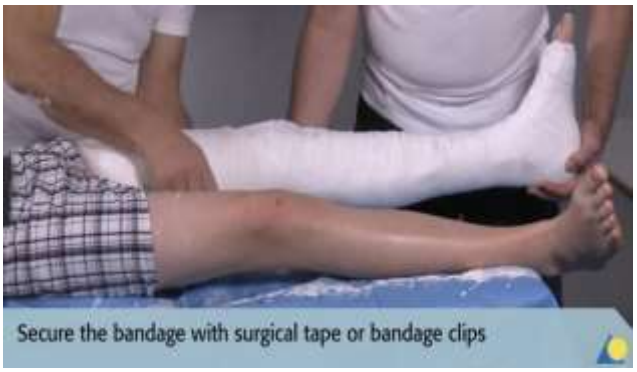


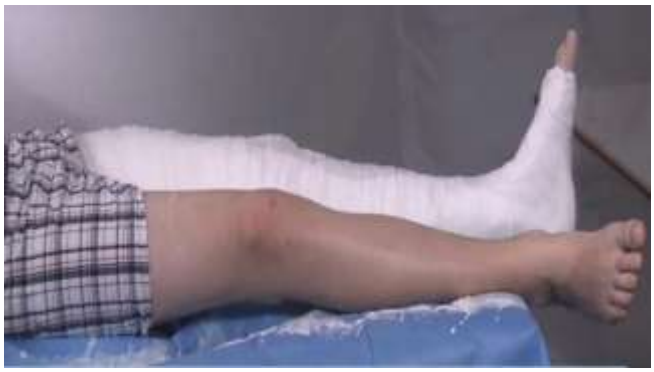
6. **2nd positioning**
   - Patient turn to supine position for splitting the splint



7. **Procedure**
   - Mark the splitting line and split all layer on the ventral side
   - Using the step-by-step splitting and fixing technique to avoid loss of the reduction
   - Apply an elastic bandage distally to close the splint and ensure the knee supported during the splinting and wrapping
   - Secure the bandage with surgical tape or bandage clip







Special things to keep in mind

- Leave the window open on the open wound
- An assistant is necessary to support positioning of the affected limb
- Attention should be paid to keep the ankle joint in a functional position 90 degree
- When possible, free movement of the toes
- Proximal extension: lesser trochanter-greater trochanter

IV. Cylinder long leg cast

1. **Indication**
   - Close or open fracture of the distal femur
   - Close or open fracture of the proximal tibia
   - Close or open fracture of the patella
   - Ligamentous rupture of the knee
2. **Goal**
   - Stabilization of the knee joint
3. **Equipment**

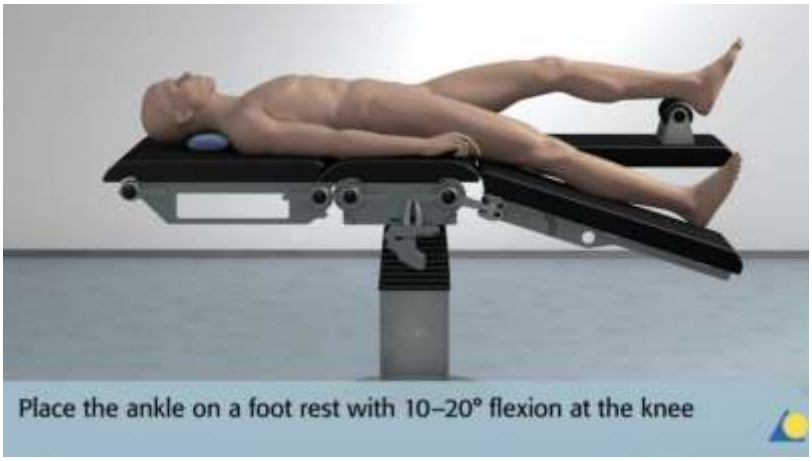
EQUIPMENT

1 Elastic foam tape
2 Rigid synthetic splint 10 cm or 12.5 cm x 90 cm
3 Cut tube bandage
4 Tube bandage 7.5 cm in dispenser box
5 Semirigid casting tape 7.5 cm or 10 cm
6 Scissors
7 Gloves
8 Elastic bandage

PERSONNEL

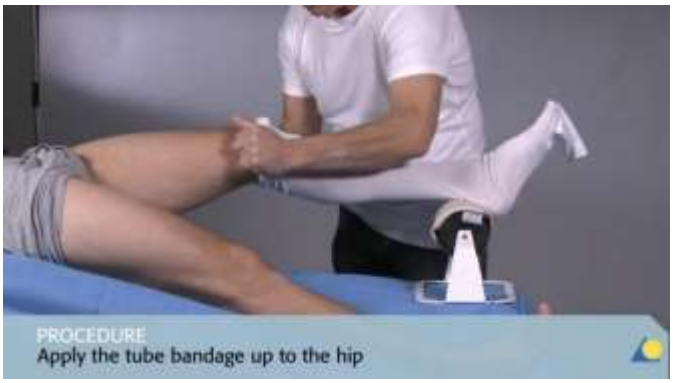
1 or 2

4. **Positioning**
   - Patient supine position
   - Ankle on the foot rest
   - 10-20 degree knee flexion



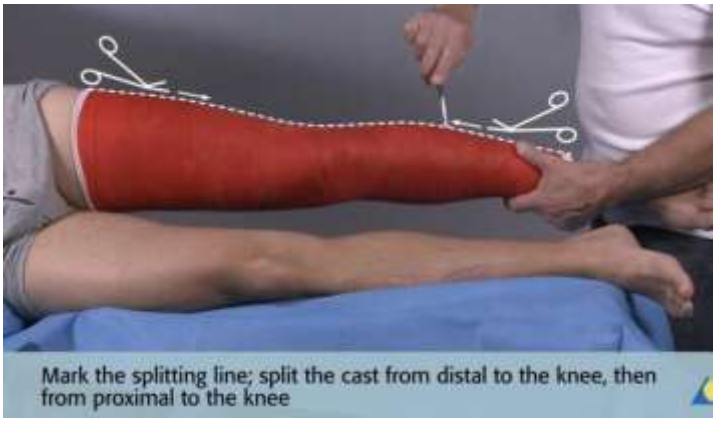
5. **Procedure**
   - Apply the tube bandage up to the hip
   - Pull bandage tight to avoid wrinkles
   - Apply elastic foam tape to protect the bony prominences around the knee
   - Wet cast with 20 degree warm water
   - Apply cast from distal to proximal using half-overlapping technique
   - Ensure the cast tape covers the lesser trochanter and greater trochanter
   - Cut the splint in half, apply the first splint medially and fix in place proximally with cast tape
   - Apply the second splint dorsally, fix both splint in place with casting tape, using the half-overlapping technique (Assistant may be needed to hold the splint in place distally)
   - Fold back the tube bandage over the caste tape proximally and distally and trim the excess
   - Apply second layer of wet cast from distal to proximal using half-overlapping technique
   - Mold the cast to the femoral condyles, the patella and the popliteal fossa
   - Ensure there is no pressure on the patella

- Mark the splitting line, split the cast from distal to the knee, then from proximal to the knee
- Wrap the splint with an elastic bandage, using the half-overlapping technique
- Secure the bandage with surgical tape or bandage clips
- Final assessment: Hip joint and ankle joint have free range of motion



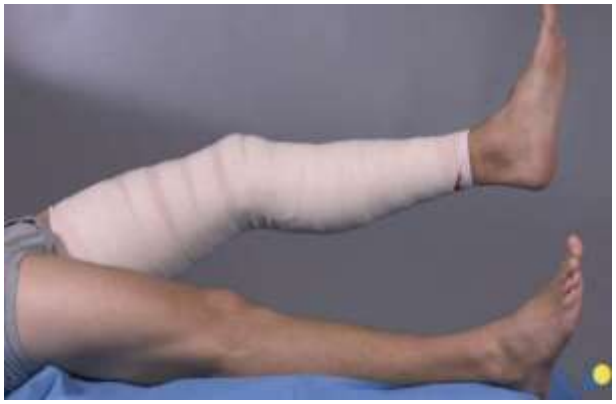

Apply a second layer of wet semirigid casting tape, from distal to proximal, using the half-overlapping technique


Ensure there is no pressure on the patella, as there is no splint in this position


Mark the splitting line; split the cast from distal to the knee, then from proximal to the knee


Secure the bandage with surgical tape or bandage clips

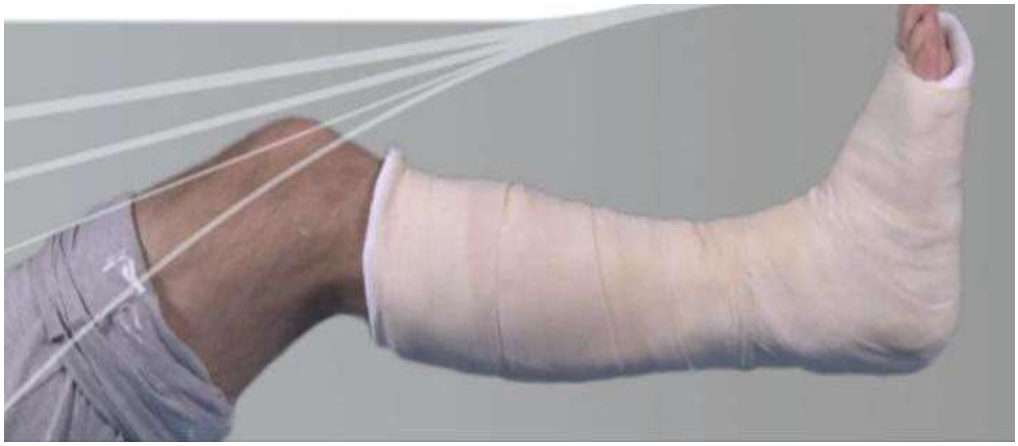

FINAL ASSESSMENT
Hip joint and ankle joint have free range of motion



Special thing to keep in mind

- Leave the window open on the open wound
- Proximal extension: lesser trochanter-greater trochanter
- Distal extension: two fingers proximal of the malleoli, to allow free movement of ankle
- Molding to the femoral condyles, to avoid the cast slipping down the patient's leg
- Pressure sore on the Achilles tendon

V. Dorsal short leg splint



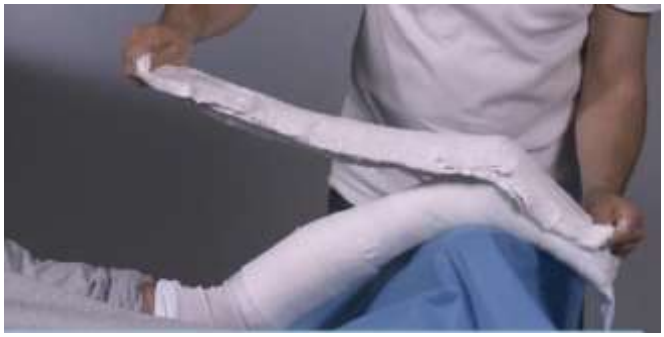
1. **Indication**
   - Close or open fracture of the ankle
   - Close or open fracture of the tarsal bones
   - Ligamentous ruptures

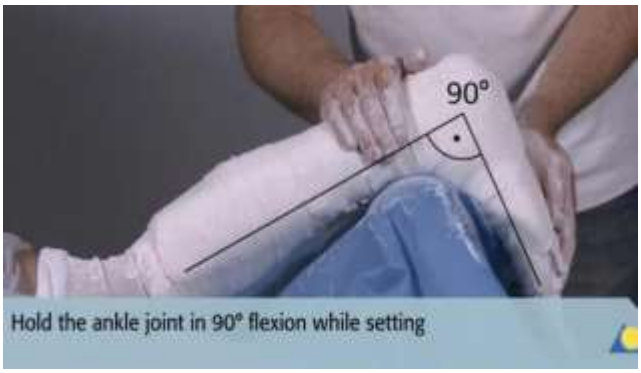
2. **Goal**
   - Stabilization of the ankle joint and foot

3. **Equipment**

4. **1st Positioning**
   - Patient in prone or supine on the table
   - Place the affected leg on a support
   - With the ankle joint in 90 degree flexion

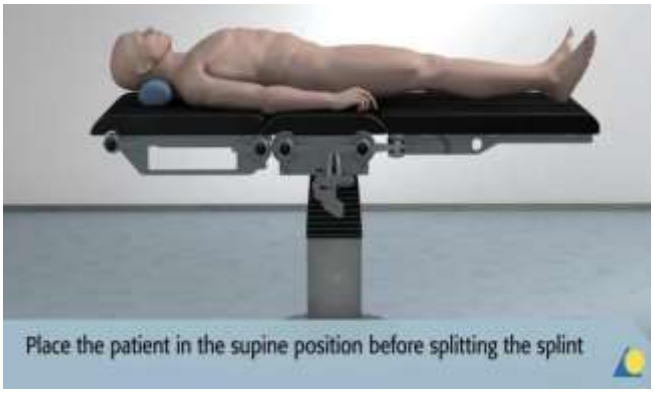
5. **Procedure**
   - Apply a tube bandage from the foot to over the knee, padding over the bandage from distal to proximal using half-overlapping technique
   - Prepare the posterior POP splint and submerge the splint in 20 degree warm water then squeeze out excess water
   - Stretch and smooth out the splint, pressing the layers together, resulting in a compact splint
   - Apply the L-Shaped POP splint, starting at the foot
   - Trim and mold it to the leg
   - Prepare U Shape of POP splint and submerge in 20 degree warm water then apply the U shape POP splint under the heel, and medially and laterally the lower leg and mold it
   - Wrap the splints with a gauze bandage, beginning distally using half-overlapping technique
   - Mold the splint and hold the ankle joint in 90 degree flexion



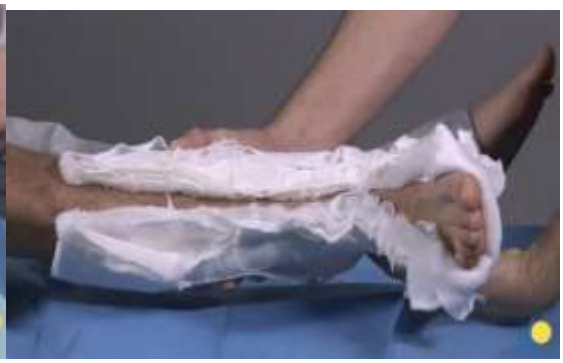


6. 2nd positioning

- Patient turn to supine position for splitting the splint



7. **Procedure**
   - Mark the splitting line and split all layer on the ventral side
   - Using the step-by-step splitting and fixing technique to avoid loss of the reduction
   - Apply an elastic bandage distally to close the splint and ensure the knee supported during the splinting and wrapping
   - Secure the bandage with surgical tape or bandage clip



Special things to keep in mind

- Leave the window open on the open wound
- Ensure the fibula head remains free a minimum of 2 cm to avoid pressure on the peroneal nerve
- Free flexion of the knee
- When possible, free movement of the toes

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OPEN FRACTURE MANAGEMENT

Authors: Kim Sopheaktra, Sok Chan Pheaktra, Ly Kang, Chheur Hengnaroth,

Huot Vutha, Ang Eng Sopheap

I. INTRODUCTION

An open fracture is an injury where the fractured bone and/or fracture hematoma are exposed to the external environment via a traumatic violation of the soft tissue and skin [(1)]. The skin wound may lie at a site distant to the fracture and not directly over it.

II. ETIOLOGY

Open fractures occur secondary to trauma. They most commonly occur as high-energy injuries, but can also be a result of low-velocity trauma when the sharp ends of the fracture fragments pierce through the skin and soft tissue[(2)].

Commonly cause is road traffic accident, and other causes are crushing injury, sport accident, violent etc.

III. CLINICAL EXAMINATION

- History of present illness
  - Time of injury
  - Location of accident
  - Mechanism of injury
  - Associated illness of patient
- Physical examination
  - Inspection
    - Sizes of wound
    - Limb deformity
    - Bleeding on the injury side
    - Soft tissue damage
  - Palpation
    - Limb tenderness (Compartment syndrome)
    - Pulse upper and lower fracture side

IV. PARACLINIC EVALUATION

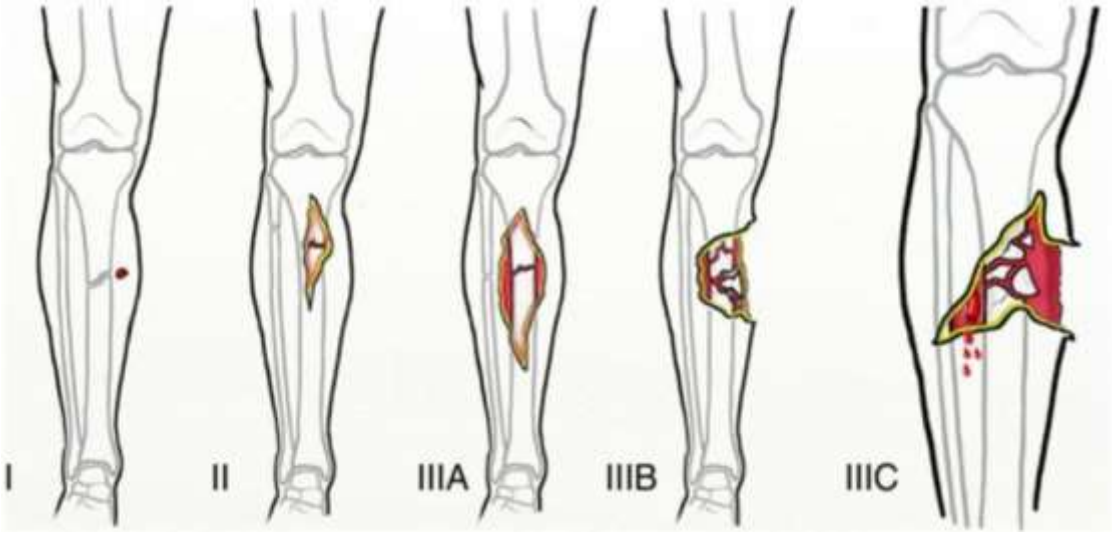
Because patients with open fractures usually sustain significant trauma, an arterial blood gas (ABG), hemoglobin, hematocrit, platelet count, metabolic panel, serum lactate, and toxicology screens are often warranted. Plain radiographs are usually adequate to assess the extent of the fracture. At a minimum, AP and lateral views of the injured bone should be obtained. The joints above and below the injury should also be x-rayed as the fracture could extend into the adjacent joints or involve articular surfaces. Air present on plain radiographs in the muscle, subcutaneous tissue or joint and visualized foreign

bodies indicate an open injury. If the patient is stable, a CT of the ankle or knee joint may be helpful to characterize the orientation of the fracture and aid in reduction and plans for fixation. In the absence of pulses, a CT angiogram can be used to identify vascular injury[3][4]

V. CLASSIFICATION

Common classification use is Gustillo-Anderson which determine degree of soft-tissu injury and severity of bone exposure[5].

Gustilo classification	Description
Type I	An open fracture with a wound <1 cm long and clean.
Type II	An open fracture with a laceration >1 cm long without extensive soft tissue damage, flaps, or avulsions.
Type III	Massive soft tissue damage, compromised vascularity, severe wound contamination, marked fracture instability.
Type IIIA	Adequate soft tissue coverage of fracture despite extensive soft tissue laceration or flaps, or high-energy trauma irrespective of the size of the wound.
Type IIIB	Extensive soft tissue injury loss with periosteal strippingand bone exposure; usually associated with massive contamination.
Type IIIC	Open fracture associated with arterial injury requiring repair.



VI. MANAGEMENT

1. Primary management

- Bleeding control
- Pain control
- Stabilized the fracture limb
  - splint, brace, or traction for temporary stabilization
  - decreases pain, minimizes soft tissue trauma, and prevents disruption of clots
  - dressing
  - remove gross debris from wound, do not remove any bone fragments
  - place sterile saline-soaked dressing on wound
  - little evidence to support aggressive irrigation or irrigation with antiseptic solution in the ED, as this can push debris further into wound[6]
- Anti-tetanus serum and vaccine should be perform
- Antibioproplylaxy for open fracture[7]

Antibioprophylaxis for Open Fractures

Classification	Type	Likely Organism	Antibiotic (IV)	Duration	ATB practically use in KSFH
Open fracture with skin wound < 1cm and clean	I	Gram (+) cocci	1st generation cephalosporin (eg: Cephazolin)	24h	3rd generation cephalosporin (eg: Ceftriaxone)
Open fracture with skin wound > 1 cm without extensive soft tissue damage and flap	II	Gram (+) cocci	1st generation cephalosporin (eg: Cephazolin)	24h -48h	3rd generation cephalosporin (eg: Ceftriaxone)
Open fracture with extensive soft tissue injury or traumatic amputation with adequate tissue coverage	III A	Gram (+) cocci + Gram (-) rod	1st generation cephalosporin (eg: Cephazolin) + Gentamycin	48h –72h	3rd generation cephalosporin (eg: Ceftriaxone) + Gentamycin
Open fracture with extensive soft tissue injury or traumatic amputation with significant soft tissue loss with expose bone that will require skin graft	III B	Gram (+) cocci + Gram (-) rod	1st generation cephalosporin (eg: Cephazolin) + Gentamycin	48h –72h	3rd generation cephalosporin (eg: Ceftriaxone) + Gentamycin + Metronidazole
Open fracture with extensive soft tissue injury or traumatic amputation with associate vascular injury requiring repair for limb preservation	III C	Gram (+) cocci + Gram (-) rod	1st generation cephalosporin (eg: Cephazolin) + Gentamycin	48h –72h	3rd generation cephalosporin (eg: Ceftriaxone) + Gentamycin + Metronidazole

2. Surgical management

- Debridement of the injury zone
- The surgical site should be thoroughly irrigated (several liters of fluid – optimally, a balanced salt solution, such as Ringer-lactate - to reduce the bacterial population)[8]
- Fracture stabilization, Usually by external fixator
- Delayed definitive ORIF

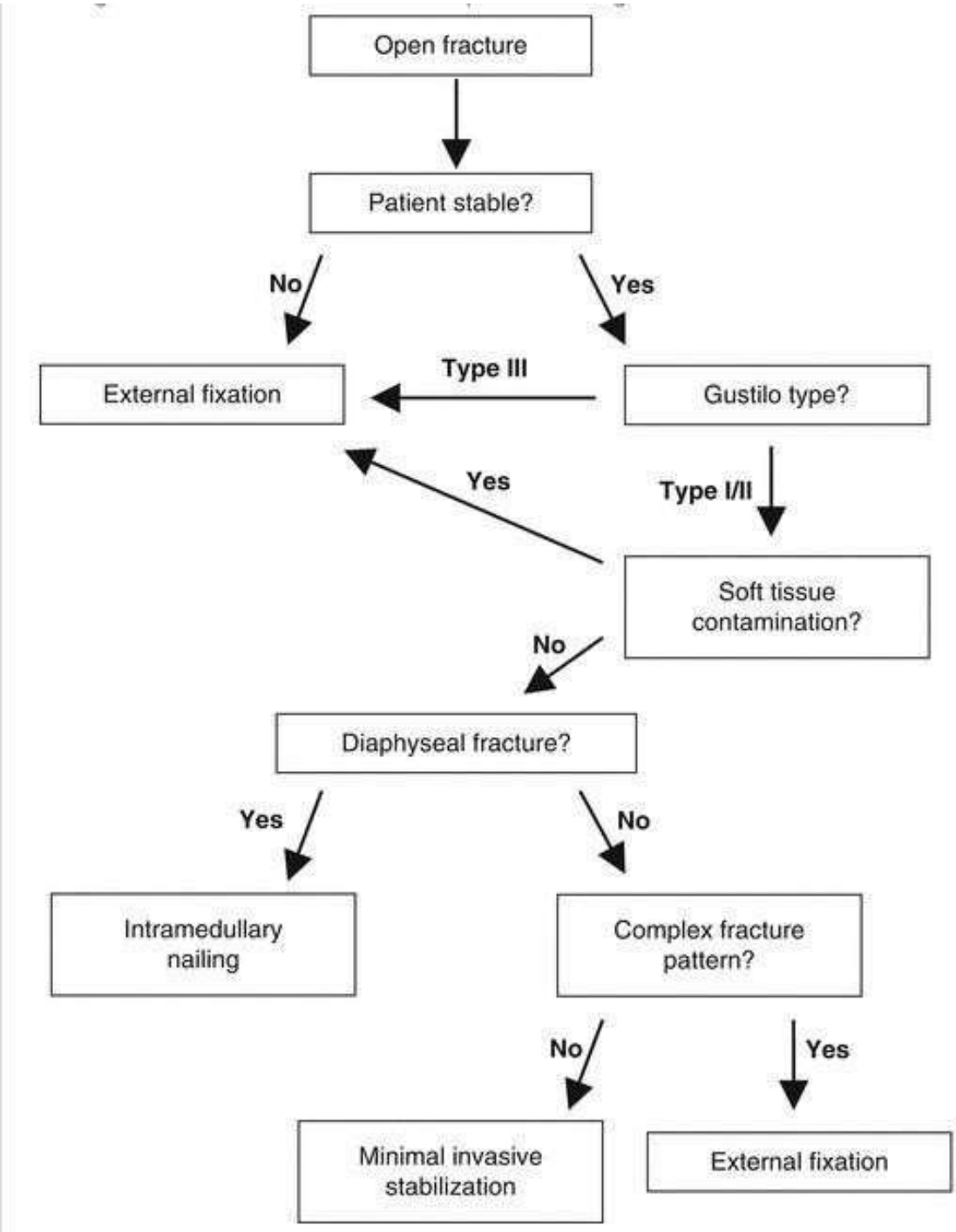
- Soft-tissue care
  - Avoid contamination
  - Avoid desiccation
  - Consider special dressings
  - Cover promptly

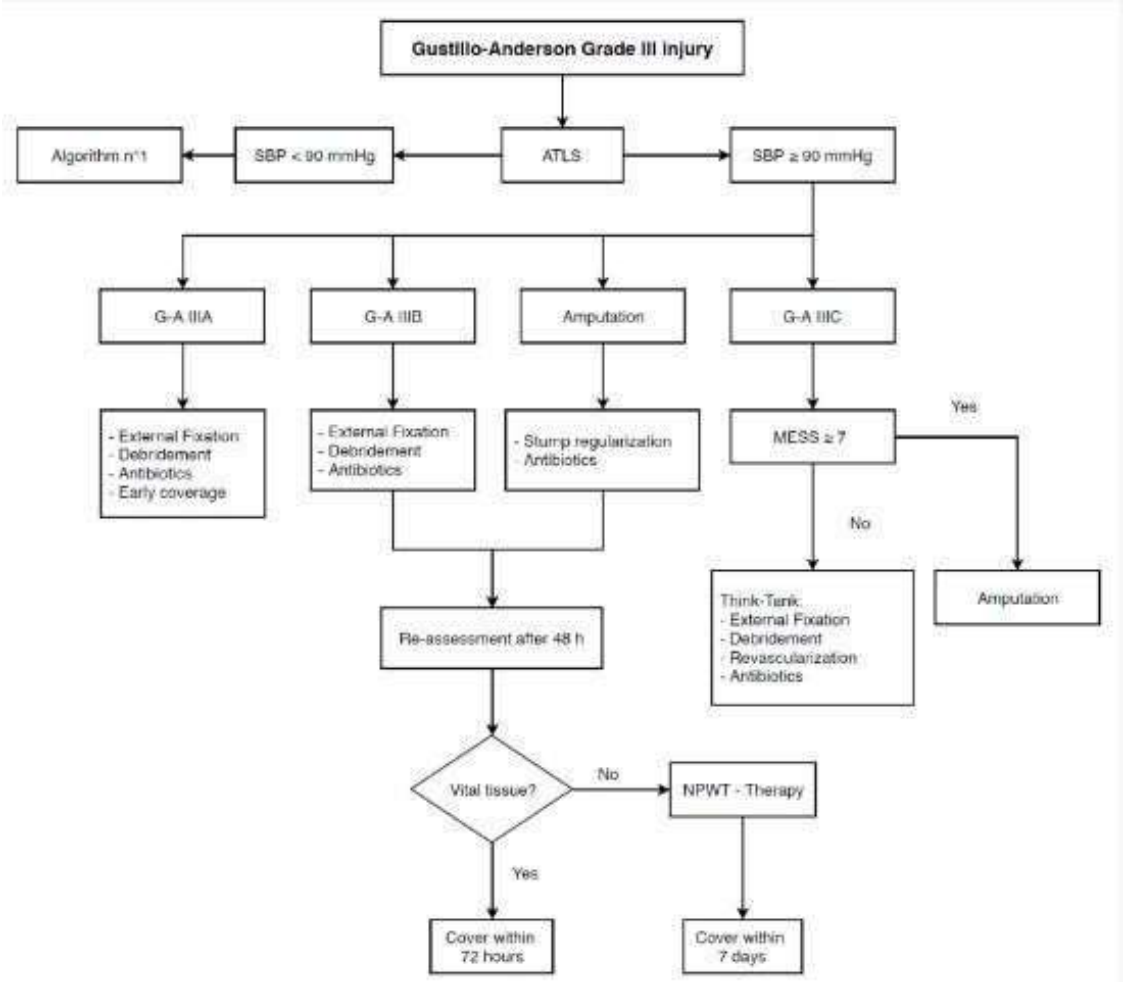
- Soft-tissue coverage in open fractures
  - Only ever after wound excisions are complete
  - Delayed closure of the traumatic wound is safer in all open fractures.
- Second look
  - 48 hours after the original debridement, it is generally advisable to re-inspect the injury zone under anesthesia – so-called "second look".
  - This affords the opportunity:
    - To assess the viability of the soft tissues
    - To conduct any necessary further tissue excision
    - To wash out any accumulated blood clot, tissue fluid coagulum or remaining foreign material

VII. COMPLICATIONS

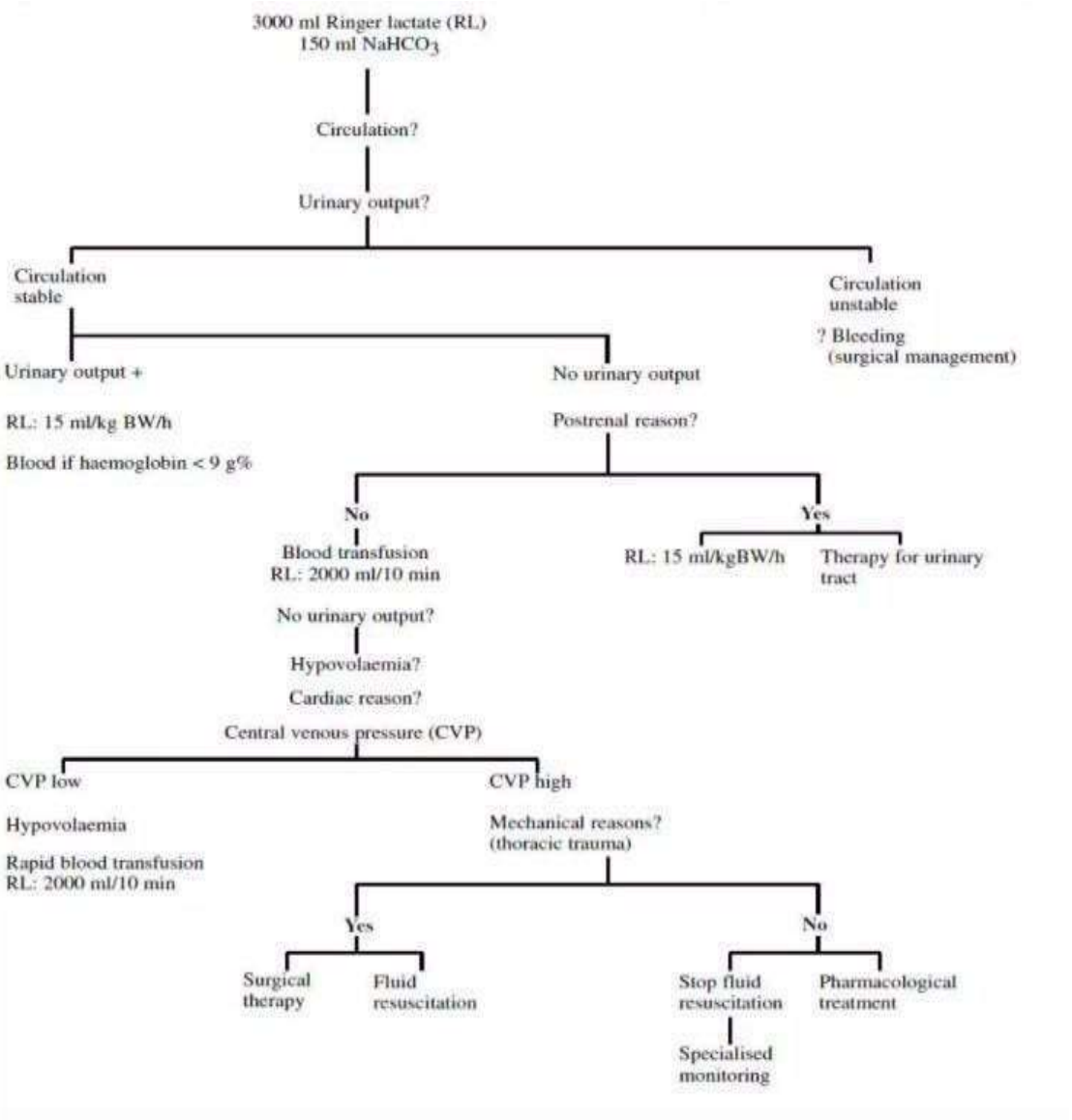
- Surgical site infection
  - Incidence
    - fracture-related infection ranges from <1% in type I open fractures to 30% in type III fractures
- Osteomyelitis
  - Incidence
    - the tibia is the most common site of post-surgical osteomyelitis following surgical treatment of open fractures
    - risk factors include:
      - ❖ blast mechanism of injury
      - ❖ acute surgical amputation
      - ❖ delay in definitive soft tissue coverage greater than 7 days
- Depression
  - Increased risk of developing depression after open tibia fracture
  - Pre-existing depression may worsen outcomes after fracture
- Neurovascular injury
- Compartment syndrome

VIII. ALGORITHM




Gustillo-Anderson Grade III injury
ATLS
SBP < 90 mmHg
Algorithm n°1
SBP ≥ 90 mmHg
G-A IIIA
G-A IIIB
Amputation
G-A IIIC
- External Fixation
- Debridement
- Antibiotics
- Early coverage
- External Fixation
- Debridement
- Antibiotics
- Stump regularization
Antibiotics
MESS ≥ 7
Yes
No
Amputation
Think-Tank
- External Fixation
- Debridement
- Revascularization
- Antibiotics
Re-assessment after 48 h
Vital tissue?
No
NPWT - Therapy
Yes
Cover within 72 hours
Cover within 7 days

Algorith N^{O}1: ATLS for volume replacement in the first 24h after multiple trauma



Mangled Extremity Severity Score (MESS) [9]

Skeletal/soft tissue injury	
	Low energy injury (eg. simple bone fracture) – 1 point
	Medium energy injury (eg. multiple bone fractures) – 2 points
	High energy injury (eg. car accidents) – 3 points
	Very high energy injury (eg. high speed trauma with severe contamination) – 4 points
Limb ischemia	
	Normal perfusion with reduces or even absent pulse – 1ˣpoint
	Absent pulse,paresthesia, diminished capillary refill – 2points
	Cool, paralyzed, insensate limb – 3ˣpoints
Shock	
	Systolic blood pressure > 90 mm Hg: 0 points
	Hypotensive transiently: 1 point
	Hypotensive persistent: 2 points
Age	
	< 30 years: 0 points
	30-50 years: 1 point
	> 50 years: 2 points

ˣThe score is doubled for ischemia > 6 hours

MESS <= 6 – Limb salvageable

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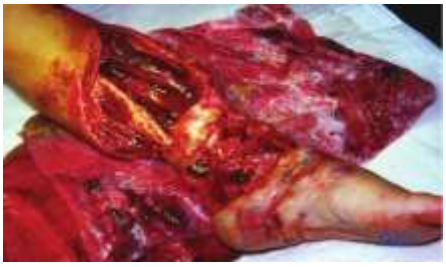
PRINCIPLE OF MANAGEMENT OF OPEN FRACTURES

Authors: YIN Rith, ICH Khuy

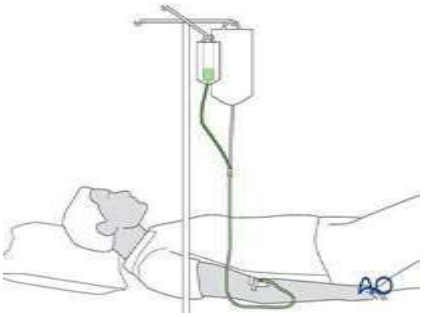
I. INTRODUCTION

1.1 PRINCIPLES OF SURGICAL CARE FOR OPEN FRACTURES



- Prompt diagnosis.
- Appropriate intravenous antibiotics.
- Meticulous injury zone excision (débridement) *
- Fracture stabilization.
- Second look.
- Early soft-tissue cover after soft-tissue recovery

1.2 INTRAVENOUS ANTIBIOTICS FOR OPEN FRACTURES



Antibiotics for open fractures are an adjunct to met iculous wound debridement (see Pearl below).

Bacterial contamination is always present with open fractu res. Bacterial count and infection rate can be significantly redu ced by prompt administration of intravenous antibiotics, in combination with surgical debridement. Most infecting bacteria, except in very dirty wounds, are typical skin flora. A first generation cephalosporin (e.g., cefazolin 1-2 grams/8 hours) is often used, except for patients with penicillin allergy.

For more severe open-fracture wounds, add an aminoglycoside (eg., gentamycin 80 mg/8-12 hours). If "agricultural" contamination is present, high-dose intravenous penicillin is usually added (e.g., 5 million-10 million units/24 hours) and consider metronidazole. They should be started as soon as the open fracture is diagnosed, but continued for only 2-3 days.

1.3 INTRAOPERATIVE WOUND CONTAMINATION

A key principle of safe surgical treatment is to minimize the number of bacteria that might enter the surgical wound. Appropriate preoperative skin decontamination, with washing using antibacterial agents, is a mainstay of this. Similarly, the use of sterile

Type	Description
I	Skin wound less than 1 cm
	Clean
	Simple fracture pattern
II	Skin wound more than 1 cm
	Soft-tissue damage not extensive
	No flaps or avulsions
	Simple fracture pattern
III	High-energy injury involving extensive soft-

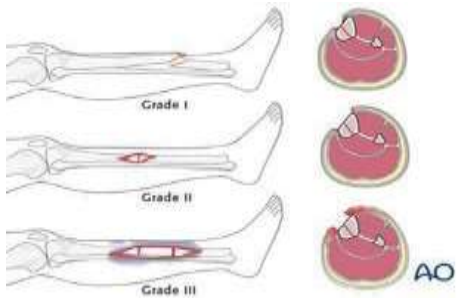
drapes, instruments and implants, and the maintenance of strict aseptic discipline throughout the procedure are also important.

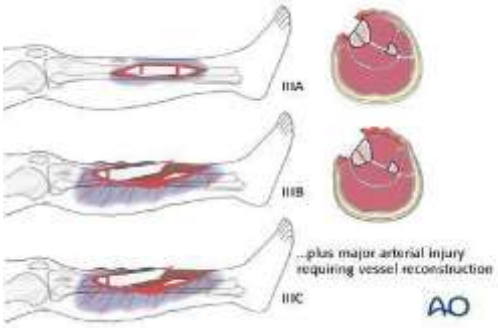
II. CLASSIFICATION OF OPEN FRACTURES

Wound-severity classification
Gustilo and Anderson. (JBJS 1976)
This work largely addressed lower leg injuries, but has some value in other anatomical sites.
The Gustilo – Anderson classification divides soft-tissue wounding of open fractures into three grades – I, II & III.

This illustration summarizes the three basic

grades – I, II & III





Gustilo, Mendoza and Williams. (J.Trauma 1984)

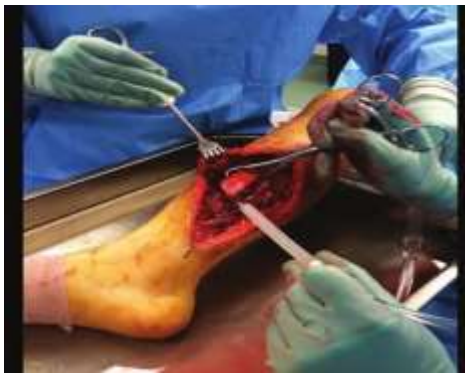
The III grade was later further subdivided into types

IIIA, IIIB & IIIC.

Type	Description
IIIA	Adequate soft-tissue cover of bone despite extensive soft-tissue damage
IIIB	Extensive soft-tissue injury with periosteal stripping and bone exposure
	Major wound contamination
IIIC	Open fracture with arterial injury requiring repair

Gustilo classification of type III open fractures AO

III. DEBRIDEMENT



Debridement of the injury zone in open fra ctures

The injury zone excision must be complete, metic ulous

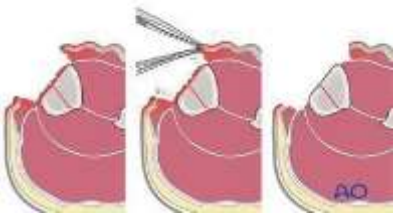
and radical.
Early wound debridement is the most important

component of the care of any open fracture.

Deciding which tissue to remove and which to retain is the essential challenge of wound débridement. This is best learned in the operating room from senior surgeons and by supervised practice. Typical errors are failure to remove enough compromised tissue, or to do so in a way that causes additional injury to retained healthy tissue. Take an organized approach that proceeds in orderly steps through each tissue layer. First, enlarge the traumatic wound for adequate exposure of the whole injury zone. Only minimal non-viable wound margins need to be excised. Explore the depths of the injury zone, and examine it thoroughly. Protect and preserve major blood vessels and nerves, tendon sheaths, healthy periosteum and soft tissue attached to bone**.**

Next, all dead, or questionably viable, tissue is excised systematically from each tissue layer:

- subcutaneous tissues



- deep fascia
- muscle
- bone

At each level, leave only obviously viable tissue.

Any bony fragments devoid of soft-tissue attachment should be removed. Contaminated, or non-viable, bone surfaces will also need excision with hand instruments, such as chisels and rongeurs.

Copious irrigation with a balanced salt solution (such as Ringer-lactate) helps to remove bacteria, bits of dead tissue and blood clot, and improves the surgeon's ability to examine the wound.

The use of pulsed pressure-lavage systems risks driving contamination into the hidden depths of the wound, and is of questionable value.

SECOND LOOK

` Forty-eight hours after the original débridement, it is generally advisable to reinspect the injury zone under anesthesia – so-called "second look".

This affords the opportunity:

- To assess the viability of the soft tissues
- To conduct any necessary further tissue excision
- To wash out any accumulated blood clot, tissue fluid coagulum or remaining foreign material

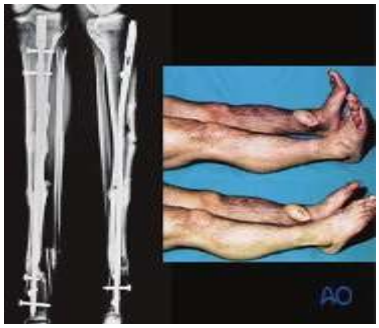
IV. FIXATION OF OPEN FRACTURES

Open fractures need

- surgical stabilization, usually external
- delayed definitive ORIF.

Bony stability in open fractures helps associated soft-tissue wounds to recover, by providing the best possible setting for soft-tissue healing and resistance to infection.

Surgical fixation, external, or internal, is the best way to stabilize an open fracture. This is done only after thorough injury zone débridement.
For lower-grade, open fractures, use fixation that would be appropriate for similar closed injuries. For more severe open fractures, or wounds that need repeated excisions, external fixation is usually preferable.



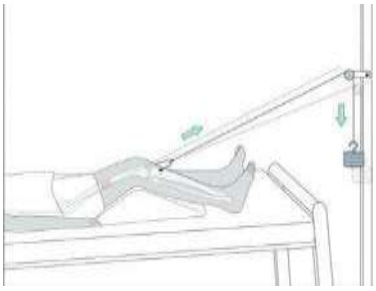
Intramedullary nailing (IMN) is occasionally chosen as fixation for low-grade femoral, or tibial, diaphyseal open fractures.
These illustrations show a severe open segmental tibial fracture, in which, short of primary amputation, IMN, using an unreamed solid nail, was the only realistic alternativ e, despite the risks.
If IMN must be delayed (significant wound contamination, etc.), temporary external fixation can be used for preliminary stabilization.

V. REFERENCE

- **Worlock P, Slack R, Harvey L, Mawhinney R.** (1994) **The prevention of infection in open fractures: an experimental study of the effect of fracture stability**. *Injury:* 25(1):31-8.

TRANS PROXIMAL TIBIAL PIN TRACTION
(PERKIN'S TRACTION)

YIN Rith, ICH Khuy



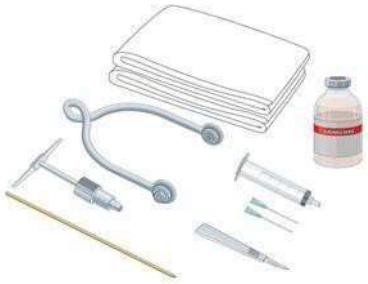
I. INDICATION AND PRINCIPLES

As soon as the decision is made that traction will be the definitive treatment, conversion to skeletal traction should be done. In cases where it is not possible to proceed to early definitive osteosynthesis (polytrauma, soft-tissue problems, patient condition, limited resources), a spanning external fixator is often used. A long leg splint can also be applied.

Temporary, proximal tibial, skeletal traction is reserved for those cases in which it is not possible to place a spanning external fixator, or use a long leg splint.

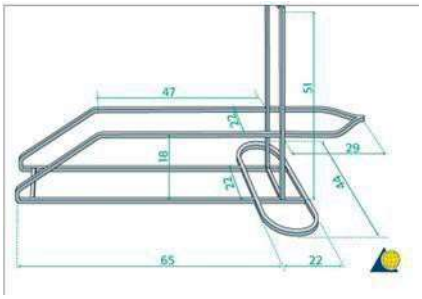
Care should be taken to protect pressure points on the skin.

II. PREPARATION



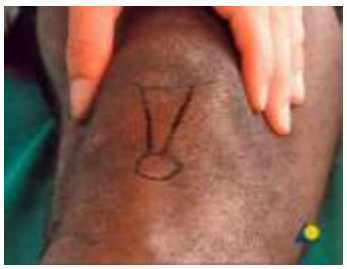
Pack with:

- Sterile towels
- Disinfectant
- Syringe
- Needles
- Local anaesthetic
- Scalpel with pointed blade
- Sharp pointed Steinmann pin, or Denham pin
- Jacobs chuck with T-handle
- Stirrup



This illustration shows the construction of a Braun-type frame, using metal bars.

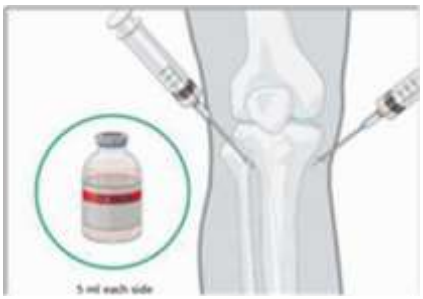
III. SURFACE ANATOMY



Tibial tuberosity/patella/common peroneal nerve
Bend the knee to make identification of the surface anatomy easier.
First, locate the prominence of the tibial tuberosity and circle it with a skin marker.

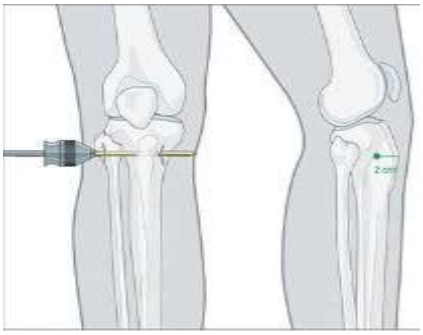
Next, identify the patella, followed by the infrapatellar tendon.

IV. ANESTHESIA



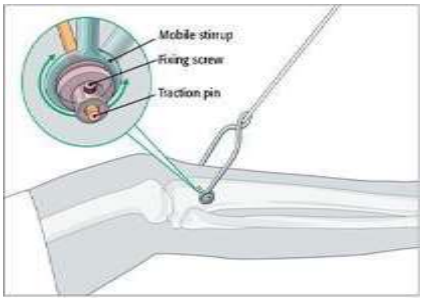
After painting the skin with antiseptic and draping with sterile towels, inject a bolus of local anaesthesia (5 ml of 2% lignocaine) on each side of the tibial tuberosity, into the lateral skin at the proposed site of pin insertion and medially at the anticipated exit point, infiltrating down to the periosteum.

V. PIN INSERTION



At the entry point, a stab incision is made through the skin with a pointed scalpel.
A Steinmann, or preferably a Denham pin, mounted in the T-handle, is inserted manually at a point about 2 cm dorsal to the tibial tuberosity.

As the pin is felt to penetrate the far cortex, check that the exit will coincide with the area of local anaesthetic infiltration. If not, inject additional local anaesthetic.
Once the point of the pin clearly declares its exit site, make a small stab incision in the overlying skin.

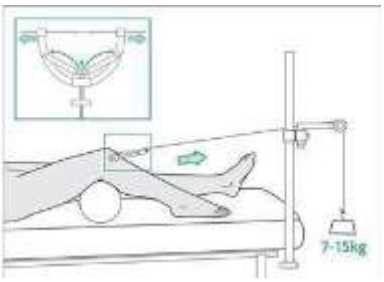


Once the pin is in place, ensure that there is no tension on the skin at the entry and exit points. If there is, then a small relieving incision may be necessary.

It is important that the stirrup be freely mobile around

the traction pin, to prevent rotation of the pin within the bone. Rotating pins loosen quickly and significantly increase the risk of pin track infection.

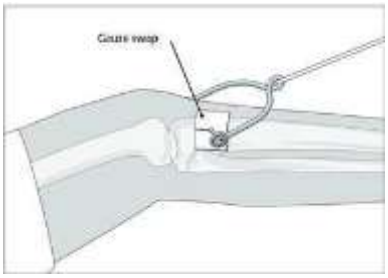
VI. APPLICATION OF SKELETAL TRACTION



After the wire has been inserted, connect it to an appropriate stirrup with 7-15 kg skeletal traction.

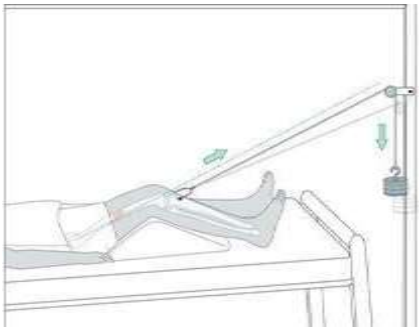
Place a padded bolster in the supracondylar region to allow for knee flexion. There may need to be some counter traction and the foot of the bed may need to be elevated.

VII. PIN CARE



In order to prevent pin track infection, apply a slit gauze swab around the pin and do not remove the crust that develops around the pin on the skin. The gauze swab should only be changed infrequently.

VIII. REDUCTION

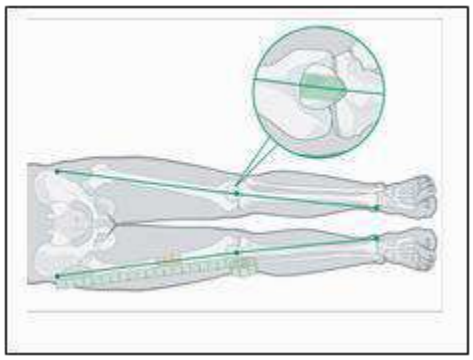


The pull on the femur (weight at the end of the traction) should be enough to correct length and to reduce the fracture.

For maintenance traction 10% of the patient's body weight is usually sufficient.

The pull should always be in line with the femur. For that purpose, the height of the pulley on the Balkan beam must be adjustable. The thigh needs to be supported on a firm triangular foam wedge, or by folded pillows, in order to prevent posterior sag at the fracture site.

IX. CONTROL OF LENGTH AND ROTATION



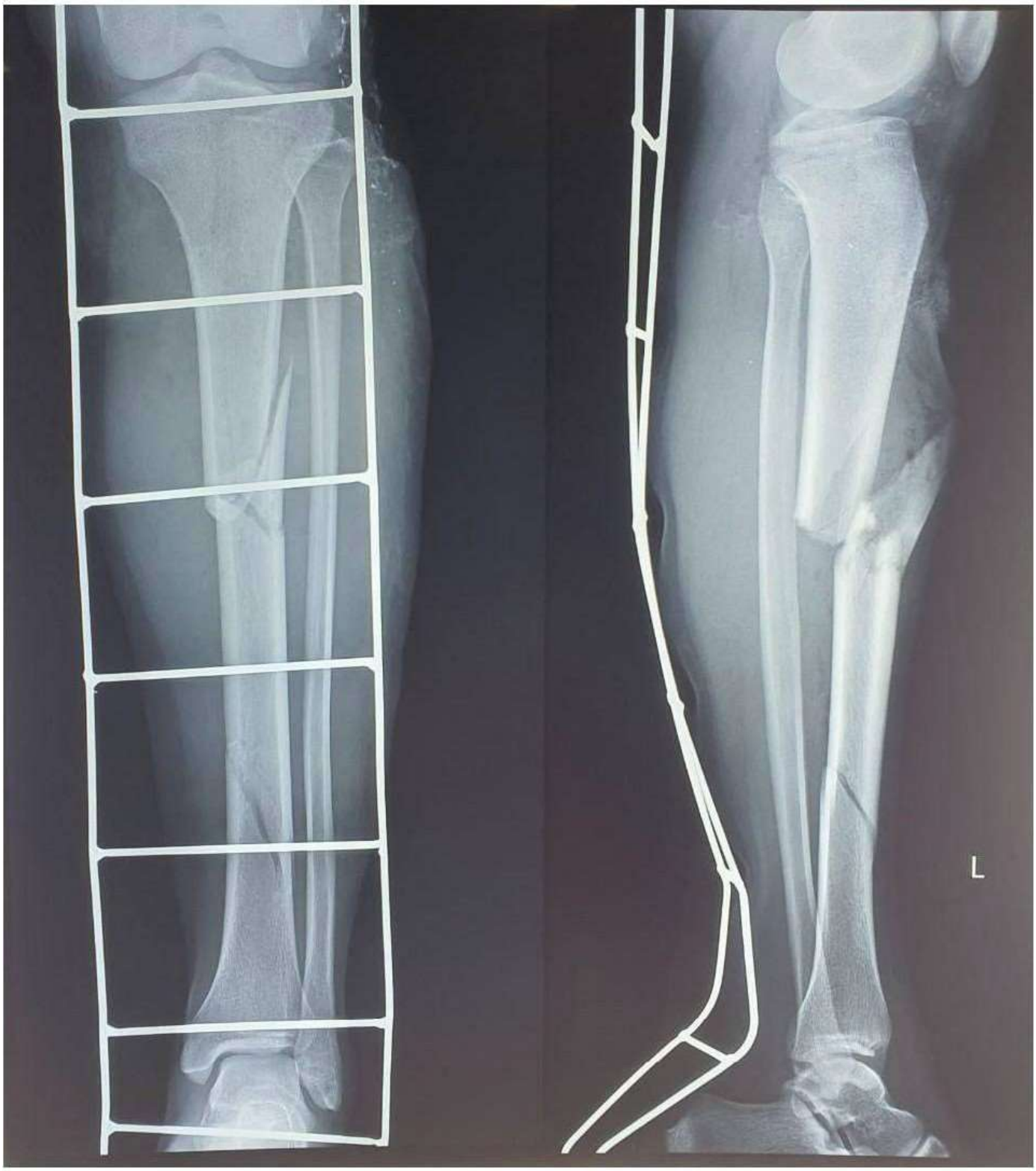
Length and rotation need to be checked daily. Length is measured by comparison to the uninjured leg. Both legs are brought into comparable positions and the distances from the anterior superior iliac spines, over the knee, to the medial malleoli, are measured
and compared.

Adjustment, if required, is done by increasing, or decreasing, the traction weight. Control x-rays need to be taken weekly, if possible, for at least the first 4 weeks.
If the medial/lateral angulation at the fracture site is anatomical, this line will pass over the central third of the patella.

X. REFERENCE

TIBIAL SHAFT FRACTURES MANAGEMENT

Authors: Sok Chan Pheaktra, Kim Sopheaktra, Ly Kang, Chheur Hengnaroth, Huot Vutha, Ang Eng Sopheap



I. SUMMARY

- Diaphyseal tibial fractures are the most common long bone fracture.
- Diagnosis is confirmed by plain radiographs of the tibia and adjacent joints.
- Treatment is generally operative with intramedullary nailing. In rare cases, external fixation or ORIF is more appropriate depending on the location and orientation of the fracture.

II. DEFINITION

A tibial shaft fracture is a break of the larger lower leg bone below the knee joint. This occurs along the long portion of the bone between the knee and ankle joints. These fractures usually result from high energy injuries such as car accidents in younger patients and most often from falls in the elderly patient. The tibia can be broken into many pieces or just crack slightly depending on the quality of bone and the type of injury.

III. EPIDEMIOLOGY

- **Incidence**
  - most common long bone fx
    - make up about 17% of all lower extremity fractures
    - account for 4% of all fractures seen in the Medicare population
  - **Demographics**
    - M > F
    - age bracket: bimodal distribution
      - young patients - high energy mechanisms
      - older patients - falls, lower energy mechanisms
  - **Anatomic location**
    - proximal 1/3 tibia fractures account for 5-10% of tibial shaft fractures

IV. ETIOLOGY

- **Pathophysiology**
  - *mechanism of injury*
    - low energy (fall from standing, twisting, etc
      - result of indirect, torsional injury
        - leads to spiral fracture pattern with fibula fracture at a different level
        - high association of posterior malleolus fractures with spiral distal tibia fractures
      - more likely to be associated with a lower degree of soft tissue injury
    - high energy fracture (MVA, fall from height, athletics, etc)
      - result of direct force
        - leads to wedge or short oblique fracture that may have significant comminution with fibula fracture at same level

- more likely to be associated with severe soft tissue injury
    - Oestern and Tscherne II / III
    - open fractures

- *pathoanatomy*
    - proximal third tibia fractures
        - must rule out extension into tibial plateau on plain films or CT sca
        - high risk for valgus/procurvatum deformity with IM nailing
    - distal third tibia fracture
        - higher rates of ankle injury seen with distal 1/3 tibia fracture and spiral fracture pattern
        - posterior malleolus most common associated ankle injury which, in some cases, may affect syndesmotic stability
    - extension into or adjacent to tibial plafond may require separate/additional fixation and are managed differently than tibial shaft fractures

- *associated conditions*
    - soft tissue injury
        - severity of muscle injury has highest impact on eventual need for amputation
    - compartment syndrome
        - more common in diaphyseal tibial shaft fractures than proximal or distal tibia fractures
        - 8.1% risk in diaphyseal fractures, compared to proximal (1.6%) and distal (1.4%) fractures
        - can occur even in the setting of an open fracture
        - all four compartments must be examined. If patient is unable to participate in examination and concern is high clinically, intracompartmental compartment measurements should be performed
    - bone loss
    - ipsilateral skeletal injury
        - tibial plateau fractures
        - tibial plafond fractures
        - femoral shaft fractures
        - floating knee is an indication for antegrade tibial nailing and retrograde femoral nailing
        - posterior malleolar fracture
        - distal 1/3 and spiral tibial shaft fractures

V. ANATOMY

- *Osteology*
    - tibial shaft is triangular in cross-section
    - proximal medullary canal is centered laterally

- important for start point with IM nailing
- anteromedial tibial crest is composed of dense, cortical bone and rests in a subcutaneous position, making it useful as a landmark
- tibial tubercle sits anterolaterally, approximately 3 cm distal to joint line
  - attachment of patellar tendon
- gerdy's tubercle lies laterally on proximal tibia
  - attachment of iliotibial band
- pes anserinus lies medially on proximal tibia
  - attachment of sartorius, semitendinosus, and gracilis

- *Muscles*

anterior compartment	tibialis anterior extensor digitorum longus (EDL) extensor hallicus longus (EHL)
lateral compartment	peroneus longus peroneus brevis
superficial posterior compartment	gastrocnemius (medial/lateral heads) soleus plantaris
deep posterior compartment	popliteus tibialis posterior flexor digitorum longus (FDL) flexor hallicus longus (FHL)

- *Ligaments*
  - superficial medial collateral ligament (MCL) attaches approximately 5-7 cm distal to joint line deep to the pes anserinus
  - adjacent fibula supports attachments for the lateral collateral ligament complex and long head of biceps femoris

- *Blood Supply*
  - anterior tibial a.
  - peroneal a.
  - posterior tibial a.
  - medial sural a.
  - lateral sural a.

- *Nervous System*
  - superficial peroneal n.
  - deep peroneal n.
  - tibial n.
  - sural n.

- *Biomechanics*

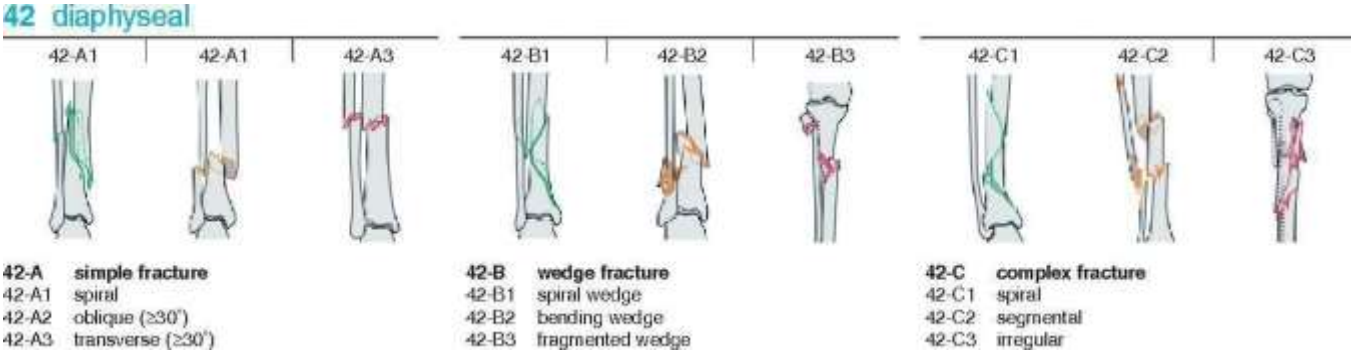
proximal tibiofibular joint	gliding synovial joint

	tibia is responsible for about 80-85% of lower extremity weight-bearing
interosseous membrane	fibrous structure interconnecting tibia/fibula which provides axial stability
tibiofibular syndesmosis	fibula rests in distal tibial incisura and is stabilized by syndesmotic ligaments ∇ anterior inferior tibiofibular ligament (AITFL) ∇ posterior inferior tibiofibular ligament (PITFL) ∇ inferior transverse tibiofibular ligament (ITL) ∇ interosseous ligament (IOL) - continuation of interosseus membrane syndesmotic stability can be affected by distal, spiral tibial shaft fractures

VI. CLASSIFICATION

Fracture classification is primarily descriptive based on pattern and location

OTA Classification	
42A	Simple fracture patterns
42B	Wedge patterns
42C	Complex/comminuted patterns



Oestern and Tscherne Classification of Closed Fracture Soft Tissue Injury	
Grade 0	Injuries from indirect forces with negligible soft-tissue damage
Grade I	Superficial contusion/abrasion, simple fractures

▪ Grade II	▪ Deep abrasions, muscle/skin contusion, direct trauma, impending compartment syndrome
▪ Grade III	▪ Excessive skin contusion, crushed skin or destruction of muscle, subcutaneous degloving, acute compartment syndrome, and rupture of major blood vessel or nerve
Gustilo-Anderson Classification of Open Tibia Fractures	
Type I	Limited periosteal stripping, clean wound < 1 cm
Type II	▪ Minimal periosteal stripping, wound >1 cm in length without extensive soft-tissue injury damage
Type IIIA	▪ Significant soft tissue injury (often evidenced by a segmental fracture or comminution), significant periosteal stripping, wound usually >5cm in length, no flap required.
Type IIIB	▪ Significant periosteal stripping and soft tissue injury, flap required due to inadequa soft tissue coverage (STSG doesn't count). Treat proximal 1/3 fxs with gastrocnemius rotation flap, middle 1/3 fxs with soleus rotation flap, distal 1/3 fxs with free flap.
Type IIIC	▪ Significant soft tissue injury (often evidenced by a segmental fracture or comminution), vascular injury requiring repair to maintain limb viability ▪ For prognostic reasons, severely comminuted, contaminated barnyard injuries, close-range shotgun/high-velocity gunshot injuries, and open fractures presenting over 24 hours from injury have all been included in the grade III group.

VII. PRESENTATION

- Symptoms
  - severe leg pain
  - inability to bear weight
  - deformity
- Physical exam

inspection	deformity / angulation / malrotation contusions blisters open wounds
palpation	check firmness of each compartment to evaluate for compartment syndrome
motion	fracture crepitus noted

- neurovascular

peripheral nerve exam	deep peroneal n.

	superficial peroneal n. sural n. tibial n. saphenous n.
dorsalis pedis and posterior tibial pulses	compare to contralateral side doppler if necessar CT angiography indicated if pulses not dopplerable

VIII. IMAGING

- Radiographs
  - recommended views
    - full-length AP and lateral views of the affected tibia
    - AP, lateral and oblique views of ipsilateral knee and ankle
    - repeat radiographs recommended after splinting or fracture manipulation
- CT
  - Indications
    - intra-articular fracture extension or suspicion of plateau/plafond involvement
    - distal 1/3 or spiral tibia fracture
      - used to exclude posterior malleolar fracture
      - also used to identify nonunion
  - findings
    - high variation in reported incidence of posterior malleolus fracture with distal 1/3 spiral tibia fractures (25-60%)

IX. TREATMENT

- **Nonoperative**: **closed reduction / cast immobilization**

indications	closed, low energy fractures with acceptable alignment ♦ < 5 degrees varus-valgus angulation ♦ < 10 degrees anterior/posterior angulation ♦ > 50% cortical apposition ♦ < 1 cm shortening ♦ < 10 degrees rotational malalignment ♦ certain patients who may be non-ambulatory (ie. paralyzed), or those unfit for surgery
outcomes	angulation and rotational alignment are well maintained with casting, however, shortening is hard to control: ♦ risk of shortening higher with oblique and comminuted fracture patterns ♦ mean shortening is 4 mm

	risk of varus malunion with midshaft tibia fractures and an intact fibula high success rate if acceptable alignment maintained non-union occurs in approximately 1% of patients treated with closed reduction

- **Operative**
  - **I&D + antibiotics**
    - Indications
      - all open tibia fractures require an emergent I&D
        - surgical debridement within 12-24 hours of injury
        - wounds should be irrigated and dressed with saline-soaked gauze in the emergency department before splinting
      - all open tibia fractures require immediate antibiotics
        - should be administered within 3 hours of injury
        - standard abx for open fractures (institution dependent)
          - cephalosporin given continuously for 24 hours after definitive surgery in Grade I, II, and IIIA open fractures
          - aminoglycoside added in Grade IIIB injuries: minimal data to support this
          - penicillin administered in farm injuries: minimal data to support this, theoretically covers Clostridium
        - ∇ tetanus vaccination status should be confirmed and appropriate prophylaxis should be administered if necessary
    - outcomes
      - early antibiotic administration is the most important factor in reducing infection
      - emergent and thorough surgical debridement is also an important factor
      - must remove all devitalized tissue including cortical bone

  - **External fixation**

indications	damage control for polytrauma patients open fractures with soft tissue defects/contamination proximal or distal metaphyseal fractures
techniques	uniplanar, circular, hybrid external fixators all available should be converted to intramedullary nail within 7-21 days, ideally less than 7 days
outcomes	longer time to union and worse functional outcomes with definitive external fixation compared to IM nailing in type III open tibia fracture

	higher incidence of malalignment compared to IM nailing high rate of pin tract infections; avoid intra-articular placement given risk for septic arthritis

- **IM Nailing**

indications	unacceptable alignment with closed reduction and casting soft tissue injury that will not tolerate casting segmental fx comminuted fx ipsilateral limb injury (i.e., floating knee) polytrauma bilateral tibia fx morbid obesity
techniques	reamed vs. unreamed nailing ♦ reamed nailing allows for larger diameter nail suprapatellar vs. infrapatellar nailing provisional reduction techniques (blocking screws, plating, etc) ♦ particularly useful for proximal 1/3 tibial shaft fractures
outcomes	union rates >80% for closed tibia fractures treated with nailing ♦ risks for nonunion: gapping at fracture site, open fracture and transverse fracture pattern shorter immobilization time, earlier time to weight-bearing, and decreased time to union compared to casting decreased malalignment compared to external fixation suprapatellar vs. infrapatellar nailing ♦ improved fracture alignment with suprapatellar nailing reamed vs. unreamed nails ♦ reamed may have higher union rates and lower time to union than unreamed nails in closed fractures (controversial) ♦ reamed nails are safe for use with open fractures, with no evidence of decreased nonunion rates in open fractures ♦ recent studies show no adverse effects of reaming (infection, embolism, nonunion) ♦ reaming with the use of a tourniquet is not associated with thermal necrosis of the tibial shaft, despite prior studies suggesting otherwise ♦ higher rate of locking screw breakage with unreamed nailing

- **open reduction and internal fixation**

indications	proximal tibia fractures with inadequate proximal fixation from IM nailing distal tibia fractures with inadequate distal fixation from IM nail tibia fractures in the setting of adjacent implant/hardware (i.e. prior total knee arthroplasty)
outcomes	compared to IM nailing of tibia fractures: ♦ larger incision ♦ increased risk of wound complications and hardware irritation ♦ similar rates of union in closed fractures ♦ more difficult hardware removal ♦ greater radiation exposure intraoperatively ♦ possibly less angular deformity risk of damage to the superficial peroneal nerve during percutaneous screw insertion ♦ holes 11,12, and 13 (proximally) of a 13 hole plate place nerve at risk

- **augmentation with rhBMP-2**

indications	prior studies have demonstrated some use in open tibial shaft fractures ♦ outcomes (controversial, as recent studies have not fully supported these findings) accelerate early fracture healing decrease rate of hardware failure decrease need for subsequent autologous bone-grafting decrease need for secondary invasive procedures decrease infection rate

- **amputation**

indications	no current scoring system to determine if an amputation should be performed relative indications for amputation include ♦ significant soft tissue trauma ♦ warm ischemia > 6 hrs ♦ severe ipsilateral foot trauma

outcomes	LEAP study ♦ most important predictor of eventual amputation is the severity of ipsilateral extremity soft tissue injury ♦ most important predictor of infection other than early antibiotic administration is transfer to definitive trauma center ♦ study shows no significant difference in functional outcomes between amputation and salvage ♦ loss of plantar sensation is not an absolute indication for amputation □ METALS study ♦ military patients who undergo amputation appear to have better functional outcomes than those who undergo limb salvage

X. TECHNIQUE

- **Closed reduction/cast immobilization**
  - technique
    - long leg casting initially
    - may convert to functional (patellar tendon bearing) brace at around 4 weeks
    - close follow-up with repeat radiographs to ensure no displacement
      - can wedge cast to correct slight deformity
      - monitor for skin irritation
- **Irrigation and debridement**
  - timing
    - within 24 hours of initial injury to decrease risk of infection
  - technique
    - sharp debridement of nonviable soft tissue & bone
    - thorough irrigation of contaminated wound
    - may require multiple debridements
    - immediate closure of open wounds is acceptable if minimal contamination is present and is performed without excessive skin tension
      - if skin cannot be closed, vac-assisted closure should be considered in short-term.
- **External fixation**
  - technique
    - bypass fracture, likely adjacent joint (i.e. open 1/3 tibial shaft fracture with placement of proximal 1/3 tibia and calcaneus/metatarsal pins to span fracture)
    - construct stiffness increased with larger pin diameter, number of pins on each side of fracture, rods closer to bone, and a multiplanar construct
  - complications
    - pin site infections common

- **Intramedullary nailing**
  - approach
    - infrapatellar nailing
      - medial parapatellar
        - most common starting point
        - incision from inferior pole of patella to just above tibial tubercle
        - identify medial edge of patellar tendon, incise
        - insert guidewire as detailed below and ream
        - can lead to valgus malalignment in proximal 1/3 tibial fractures
      - lateral parapatellar
        - helps maintain reduction when nailing proximal 1/3 fractures
        - requires mobile patella
      - patellar tendon splitting
        - gives direct access to start point
        - can damage patellar tendon or lead to patella baja (minimal data to support this)
      - semiextended medial or lateral parapatellar
        - used for proximal and distal tibial fractures
        - skin incision made along medial or lateral border of patella from superior pole of patella to upper 1/3 of patellar tendon
        - knee should be in 5-30 degrees of flexion
        - choice to go medial or lateral is based of mobility of patella in either direction
        - identify starting point and ream as detailed below
    - suprapatellar nailing (transquadriceps tendon)
      - requires special instruments
      - can damage patellofemoral joint
      - easier positioning if additional instrumentation needed
      - more advantageous for proximal or distal 1/3 tibia fractures

  - technique
    - starting point
      - starting guidewire is placed in line with medial aspect of lateral tibial spine on AP radiograph, just below articular margin on lateral view
        - in proximal 1/3 tibia fractures starting point should cheat laterally to avoid classic valgus/procurvatum deformity
      - ensure guidewire is aligned with tibia in coronal and sagittal planes as you insert
      - opening reamer is placed over guidewire and ball-tipped guidewire can then be passed
    - fracture reduction
      - spanning external fixation (ie. traveling traction)
      - clamps
      - femoral distractor

- small fragment unicortical plates/screws
- blocking (poller) screws
    - placed in metaphyseal segment at the concavity of the deformity
    - in proximal 1/3 tibia fractures, posteriorly placed blocking screw in proximal fragment and laterally placed blocking screw in the metaphyseal fragment help direct the nail more centrally, avoiding valgus/procurvatum deformities
    - increase biomechanical stability of bone/implant construct by 25%
- unicortical provisional plate
    - not associated with increased infections, wound complications, and nonunion compared to closed-nailing techniques

- reaming
    - reamed nails superior to unreamed nails in closed fractures
    - ensure fracture is reduced before reaming
    - overream by 1.0-1.5mm to facilitate nail insertion
    - confirm guide wire is appropriately placed prior to reaming
        - should be "center-center" in the coronal and sagittal planes distally at the physeal scar
- nail insertion
    - anterior aspect of nail should be lined up with axis of tibia when inserting nail - typically should line up with 2nd metatarsal in absence of tibial deformity
- locking screws
    - statically lock proximal and distally for rotational stability
        - no indication for dynamic locking acutely
    - number of interlocking screws is controversial
        - two proximal and two distal screws in presence of <50% cortical contact
        - consider 3 interlock screws in short segment of distal or proximal shaft fracture
            - prefer multiplanar screw fixation in these short segments

- **Open reduction and internal fixation**
    - approach
        - lateral vs. medial
            - lateral may have more soft tissue interference but may be preferred in setting of soft tissue/wound issues
    - technique
        - generally, minimally invasive plating is used to preserve soft tissues
            - plate attached to external jig to allow for percutaneous insertion of screws
            - must ensure appropriate contour of plate to avoid malreduction
    - complications
        - higher risk for wound issues, particularly in open fractures
        - neurovascular risk

- superficial peroneal nerve (SPN) commonly at risk laterally

- **Amputation**
  - approach
    - below knee amputation (BKA) vs. above knee amputation (AKA) based on degree of soft tissue damage
      - technique
    - standard BKA vs. ertl/bone block technique
  - complications
    - infection
    - hematoma
    - phantom pain

XI. COMPLICATIONS

- Anterior knee pain
  - incidence
    - >30-50% with IM nailing
  - risk factors
    - infrapatellar nailing with patellar tendon splitting and paratendon approach
      - suprapatellar nailing may have lower rate of anterior knee pain
    - more common if nail left proud proximally
      - lateral radiograph is best radiographic views to evaluate proximal nail position
  - treatment
    - removal of nail
      - pain relief unpredictable with nail removal

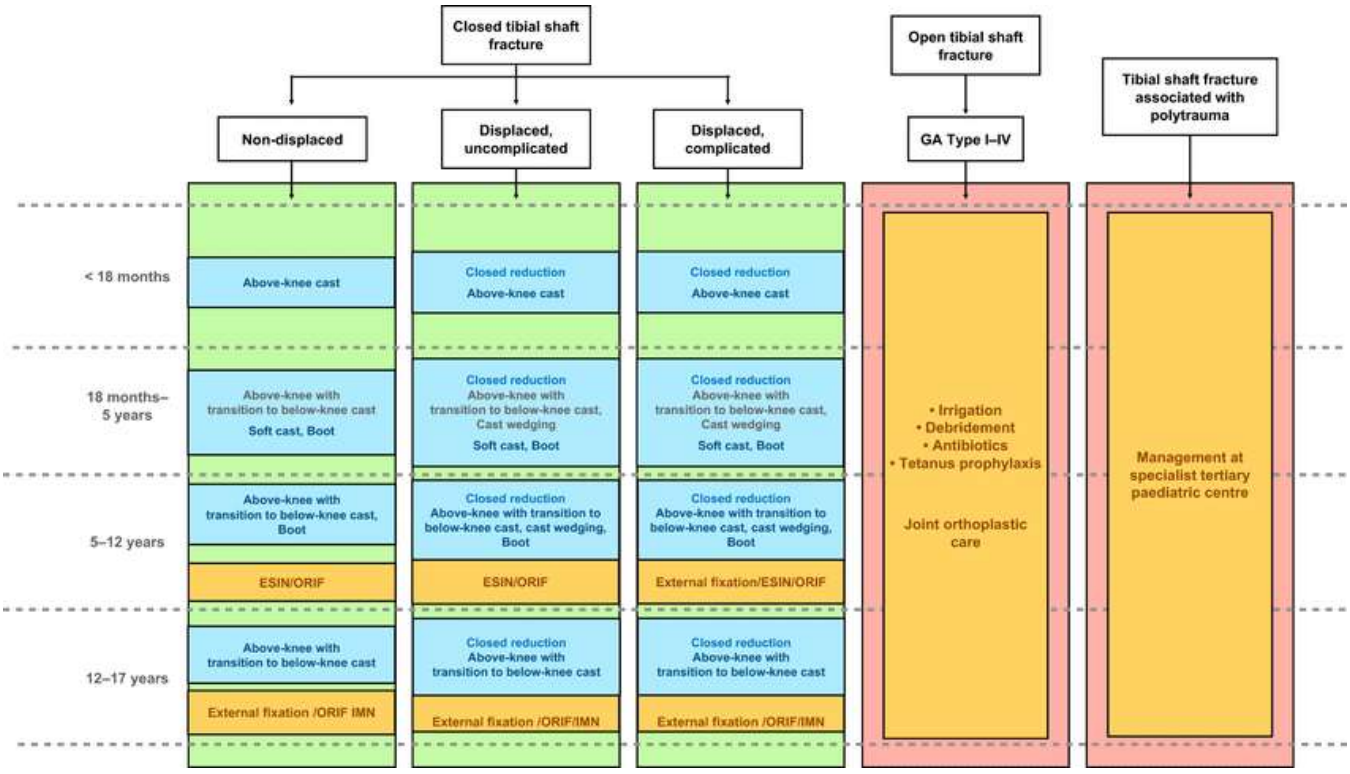
- Malunion
  - incidence
    - all tibial shaft fractures - between 8-10%
    - higher in proximal 1/3 tibia fractures - up to 50%
      - valgus/procurvatum deformity
        - patellar tendon pulls proximal fragment into extension, while hamstring tendons and gastrocnemius pull the distal fragment into flexion (procurvatum)
    - distal 1/3 fractures have a higher rate of valgus malunion with IM nailing compared to plating
  - risk factors
    - definitive management with casting or external fixation
      - most common deformity is varus with nonsurgical management
        - varus malunion may place patient at risk for ipsilateral ankle pain and stiffness
    - starting point too medial with IM nailing
    - poor reduction intraoperatively
  - treatment

- prevention is most important
  - adequate reduction, proper start point when nailing
- if malalignment is noted immediately after surgery, return to operating room is appropriate with removal of nail, reduction and nail reinsertion
- if malunion is appreciated at later followup, eventual nail removal and tibial osteotomy can be considered

- Nonunion (no healing at 9 months)
  - incidence
    - estimated between 2-10%
  - risk factors
    - open fracture
    - cortical contact <50%
    - transverse fracture pattern
  - treatment
    - rule out infection
    - nail dynamization if axially stable
    - exchange nailing if not axially stable
      - reamed exchange nailing most appropriate for aseptic, diaphyseal tibial nonunions
      - oblique tibial shaft fractures have the highest rate of union when treated with exchange nailing
      - consider revision with plating in metaphyseal nonunions
    - posterolateral bone grafting if significant bone loss
    - BMP-7 (OP-1) has been shown equivalent to autograft
      - often used in cases of recalcitrant non-unions
    - compression plating has been shown to have a 92-96% union rate after open tibial fractures initially treated with external fixation
    - fibular osteotomy of tibio-fibular length discrepancy associated with healed or intact fibula

- Malrotation
  - incidence
    - highest after IM nailing of distal 1/3 tibia fractures
      - increases risk of adjacent ankle arthrosis
  - treatment
    - should always assess rotation in operating room
      - obtain perfect lateral fluoroscopic image of knee, then rotate c-arm 105-110 degrees to obtain mortise view of ipsilateral ankle
    - may have reduced risk with adjunctive fibular plating

- Compartment syndrome
  - incidence
    - estimated between 1-9%
      - can occur in both closed and open tibia shaft fractures
  - risk factors
    - high energy injuries
    - significant soft tissue injuries
  - treatment
    - emergent four-compartment fasciotomy
- Nerve injury
  - incidence
    - true incidence unknown
    - believed to be a rare complication
  - risk factors
    - LISS plate application without opening for distal screw fixation near plate holes 11-13 put superficial peroneal nerve at risk of injury due to close proximity
    - saphenous nerve can be injured during placement of locking screws
    - transient peroneal nerve palsy can be seen after closed nailing
      - EHL weakness and 1st dorsal webspace decreased sensation
    - deep peroneal nerve can be injured with overpenetration of posterolaterally-directed proximal external fixator pins
  - treatment
    - usually nonoperatively with variable recovery expected
    - may need AFO if foot drop present
- Infection
  - incidence
    - approximately 5%
  - risk factors
    - open fracture
    - severe soft tissue injury with contamination
    - longer time to definitive soft tissue coverage
  - treatment
    - may require I&D or eventual removal of hardware
    - use of wound vacuum-assisted closure does not decrease risk of infection



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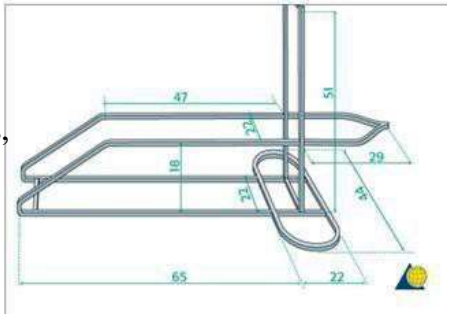
TRANS CALCANEUS PIN TRACTION

YIN Rith, ICH Khuy

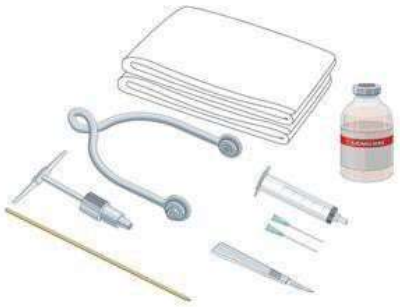


I. INDICATION AND PRINCIPLES

If an external fixator is not available, open fractures, especially if they need repeated wound care, can be treated in the short term with calcaneal pin traction on a Braun frame.

Skeletal traction on a Braun frame is also valuable for maintaining length of unstable multi fragmentary closed tibial fractures (those for which stability cannot be restored with realignment alone.

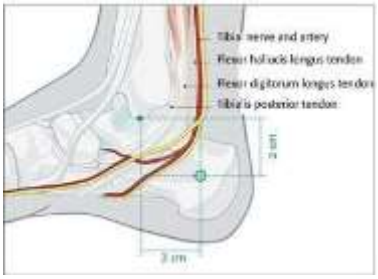
II. PREPARATION



Pack with:

- Sterile towels
- Disinfectant
- Syringe
- Needles
- Local anaesthetic
- Scalpel with pointed blade
- Sharp pointed Steinmann pin, or Denham pin
- Jacobs chuck with T-handle
- Stirrup

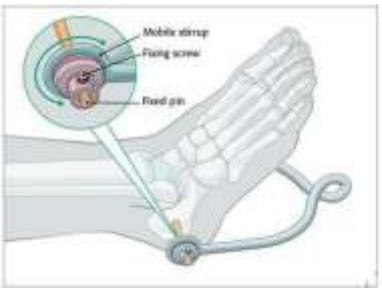
III. CALCANEAL PIN INSERTION



If the patient is not under general or regional anaesthesia, the pin is inserted under local anesthetic (eg, 2% lidocaine, 5 ml

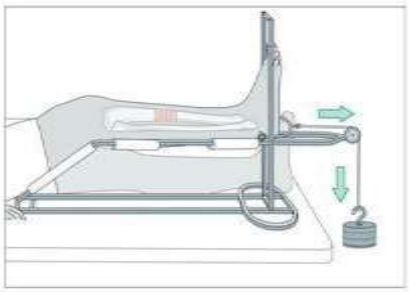
on each side of the calcaneus) 2 cm
below and 2 cm behind the medial malleolus.

It is important that the stirrup is able to rotate around the Steinman pin to prevent rotation of the pin in the bone. Rotating pins loosen quickly. Loose pins significantly increase the risk of pin track infection.



IV. APPLICATION OF TRACTION

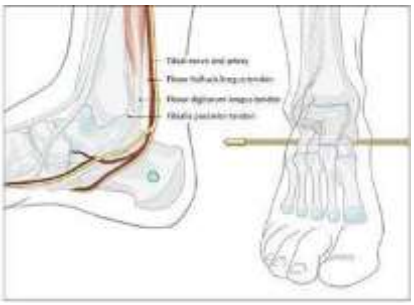
Positioning in Braun frame with 3-4kg traction



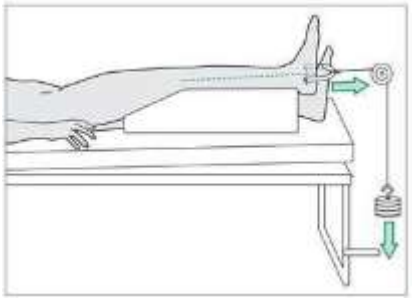
The illustration shows a tibia fracture immobilized with calcaneal traction and supported on a Braun frame.
The frame supports the thigh and the proximal tibial segment. Pressure against the thigh provides counter traction. The traction force and frame with fabric supports maintain fracture alignment.
The non-elastic supports on the Braun frame are arranged to leave a gap, which allows wound care without changing the leg position.

V. AFTERCARE

Apply dressings as needed.



Pin-site care is provided according to the surgeon's routine.
Traction should not be maintained longer than necessary, until local soft-tissue situation permits definitive treatment.
Check frequently for skin pressure from supporting frame or pin/wire clamp.



Note: Watch for pin-site infection. If infection occurs, and definitive internal fixation is not yet possible, pin shave to be

replaced using new insertion points in asafe distance to the infected pin track. Pin-track infections may compromise definitive surgical treatment.

VI. REFERENCE

UPPER EXTREMITY SPLINTS AND CASTS

Autors: Sok Chan Pheaktra, Huot Vutha, Phan Daravuth, Ly Kang, Chheur Hengnaroth, Kim Sopharktra, Im Hakseng, Men Puthi, Chhay Narith, Lim Ratanak, Ang Eng Sopheap

FIGURE-OF-8 SPLINT

I. OVERVIEW

- Figure-of-8 splints are used primarily for fractures about the clavicle
- Figure-of-8 splints are commercially prepared devices intended to create a reduction force on the clavicle.
- No difference in outcome is seen between a figure-of-8 splint and a sling for closed management of clavicle fractures.

II. INDICATIONS FOR USE

- Minimally displaced clavicle shaft fractures
- Medial physeal clavicle fractures

Precautions

- Closed reductions cannot be maintained and should not be attempted.
- Avoid overtightening the splint; excessive tension can result in increased pain, compression of the axillary vessels, and brachial plexus neuropathy.

Pearls

- Reduction is not required for most clavicle fractures.
- Clavicle fractures with more than 1.5 cm of overlap result in long-term disability and should be treated with an open reduction and internal fixation.
- Fractures that tent the skin can erode through the skin and are unlikely to heal without open reduction and internal fixation.

Equipment: Figure-of-8 splint

III. BASIC TECHNIQUE

- Patient positioning: Standing
- Landmarks:
  - Clavicle
  - Acromioclavicular joint
- Steps:
  - Have the patient stand.
  - Fit the patient with a figure-of-8 splint.
  - The figure-of-8 splint should be placed so that the center of the “8” rests on upper back.

IV. DETAILED TECHNIQUE

- Have the patient stand.
- Apply the figure-of-8 splint so that the center of the “8” comes to rest between the shoulder blades on the upper back.
- Adjust the figure-of-8 dressing so that it is as tight as possible while still being comfortable to wear

ARM SLING/ARM SLING AND SWATH

I. OVERVIEW

- “Though simple in form and principle, this sling is rich in security, ease, and comfort.” W.C. Wermuth, MD, 1908
- The arm sling is used for a variety of conditions.
- A swath wrapped around the body is added for shoulder immobilization.

II. INDICATIONS FOR USE

Sling:	▪ Clavicle fractures ▪ Minimally displaced proximal humerus fractures ▪ Acromioclavicular separations ▪ Support for splints and casts of the upper extremity
Sling and swath:	▪ moderately displaced proximal humerus fractures where the humerus does not move as a single unit

III. PRECAUTIONS

- Ensure a proper fit to prevent pressure complications at the back of the neck. It is recommended that a well-padded sling be used or that the neck be padded with cast padding and/or an Army Battle Dressing (ABD) pad.
- Elderly patients and patients with compromised skin (such as persons taking steroids on a long-term basis) should be monitored closely for skin breakdown.

IV. PEARLS

- The adult elbow does not tolerate immobilization well. If possible given the nature of the injury, the patient should be instructed to perform daily elbow, wrist, and hand range-of-motion exercises.
- If a reduction manoeuvre has been performed, obtain postreduction radiographs while the patient is wearing the sling or the sling and swath to ensure maintenance of the reduction.

V. EQUIPMENT

- Arm sling or sling and swath
- Cast padding or ABD pad
- Talcum powder (optional)

VI. IMPROVISATION

- An arm sling and 6-inch elastic bandage can be used if a commercial sling and swath are not available.

VII. BASIC TECHNIQUE

1. Patient positioning:	a. Standing
2. Landmarks:	a. Clavicle b. Acromioclavicular joint c. Acromion
3. Steps:	a. Sling: ▪ Have the patient stand. ▪ Fit the patient with a sling. ▪ The sling should provide support for the weight of the arm. b. Sling and swath: ▪ Have the patient stand. ▪ Place an ABD pad with talcum powder (optional) in the axilla. ▪ Fit the patient with a sling. ▪ Apply the swath.

VIII. DETAILED TECHNIQUE

1. Sling
   - Have the patient stand.
   - Pad the neck strap of the sling to prevent pressure complications at the back of the neck.
   - Apply the sling and adjust the straps.
   - Adjust the sling so it is tight enough to support the weight of the arm.
2. Sling and swath
   - Have the patient stand.
   - Pad the neck strap of the sling to prevent pressure complications at the back of the neck.
   - Place talcum powder on the ABD pad and fold the pad in half, with the talcum side facing out.
   - Place the folded ABD pad in the axilla to absorb perspiration.
   - Apply the sling and adjust the straps so it is loose while providing some support for the weight of the arm.
   - Buckle and adjust the circumferential body strap.
   - If using a sling only, swath the arm to the body using cast padding followed by application of a large elastic bandage.

COAPTATION SPLINT

I. OVERVIEW

- The correct application of either of the methods described below will ensure that the coaptation splint remains secure and does not fall out of place.

II. INDICATIONS FOR USE

- Humeral shaft fracture

III. PRECAUTIONS

- Do not allow one end of the coaptation splint to end at the fracture site; otherwise, the splint terminus will become a fulcrum and cause more displacement.
- An ABD pad can be placed in the axilla after the splint is applied.
  - Use of an ABD pad prevents direct compression of the brachial plexus.
  - An ABD pad absorbs moisture.
- When using the hanging stockinette modification technique, pay clos attention to which side of the splint is padded. Ensure that the well-padded side is facing toward the patient.

IV. PEARLS

- Coaptation splints have a reputation for being made poorly and for sliding down the arm.
- The key to applying a coaptation splint properly is to ensure that the splint always comes above the arm onto the shoulder.
- Use a technique that allows the coaptation splint to be secured around the body to prevent distal displacement. An extra-long elastic or self-adherent bandage is a useful adjunct for a coaptation splint.
- When applying the splint, have the patient turn his or her head to the contralateral side, which prevents the neck from pushing down the splint during application.
- We prefer using a self-adhesive bandage to overwrap the plaster because it acts predictably during application, stays in place well, and looks better than other options.

V. EQUIPMENT

- Stockinette: 4 inches wide, 6 feet long
- Cast padding: 4 inches wide
- Plaster: 4 inches wide
- Elastic or self-adherent bandage: 4 inches wide
- Silk tape: 2 inches wide (optional)
- Bucket of tepid water

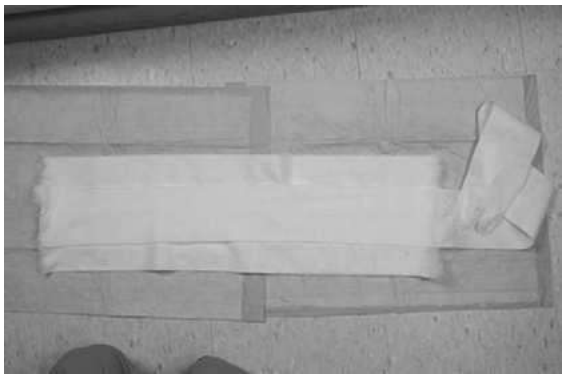
VI. BASIC TECHNIQUE

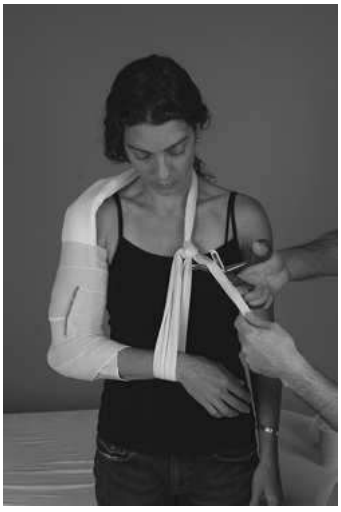
1. Patient positioning:
   - If possible, have the patient stand or sit up with his or her back off the stretcher.
   - The elbow is placed at 90 degrees.
   - If the patient is unable to sit up, move the head of bed as upright as possible.
2. Where to start:
   - As high into the axilla as possible
   - If the fracture is in the proximal third of the humeral diaphysis (right at the level of the axilla on the radiograph), start the splint lower.
3. Where to finish:
   - At the base of the neck
4. Where to mold: Lateral aspect, distal to the fracture site
5. Steps:
   - Measure the length of the splint using plaster.
   - Roll out the cast padding.
   - Roll out the plaster.
   - Cut a 6-foot length of 4-inch stockinette.
   - Position the patient.
   - Prepare the plaster in the usual fashion.
   - Place a splint inside a stockinette.
   - Apply the splint.
   - Definitively secure the splint with an elastic bandage.
   - Complete the stockinette.

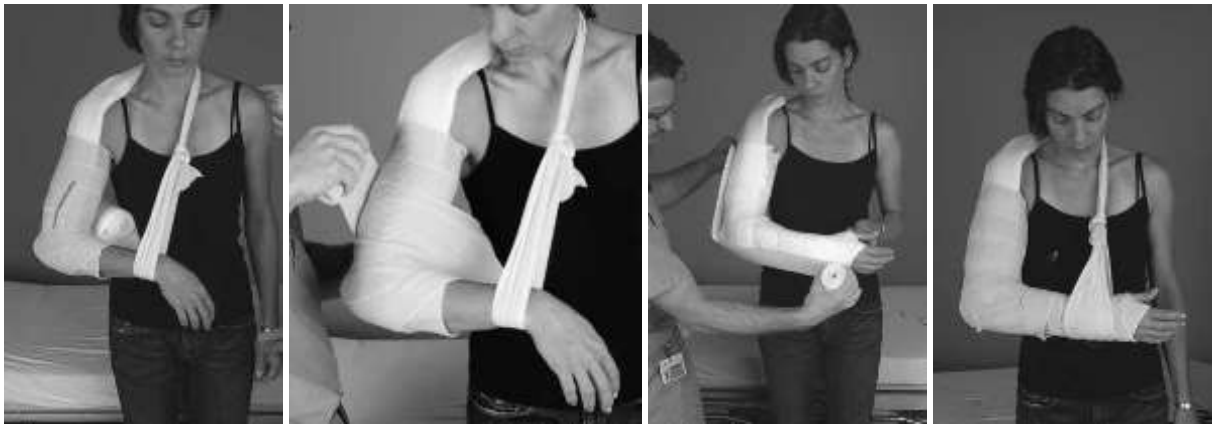
VII. DETAILED TECHNIQUE

1. Measure the length of the splint.
   - Use the contralateral side.
   - Hold one hand in the axilla and wrap the plaster around the elbow until the base of the neck is reached.
   - Mark the length on the plaster by making a small tear on one side.
2. Roll out the plaster.
   - The plaster slab should be 10 to 12 sheets thick.
   - Use the measured length.
3. Roll out the cast padding.
   - Use the usual technique that will allow the cast padding to be folded over.
   - Add at least 6 inches of length so the splint may be folded over.
   - Cut a 6-foot length of 4-inch stockinette.
4. Position the patient.
   - Have the patient sit upright if possible.
   - Place the patient's elbow at 90 degrees.
   - Ensure that the patient's head is facing toward the contralateral side.

5. Prepare the plaster in the usual fashion. Use the usual technique of wetting and laminating, followed by placement in cast padding.
6. Place the splint inside a stockinette.
   - Initially placing the entire length of the stockinette on the surgeon's forearm is helpful.
   - Hold one end of the splint with the arm containing the stockinette.
   - Pull the stockinette to the end of the splint.
   - Do not forget which side of the splint is padded.
7. Apply the splint.
   - Start in the axilla or at an appropriate starting point given the fracture site. Provisionally secure it with cast padding at the middle arm.
   - Loop the splint around the elbow. Again, provisionally secure it with cast padding at the middle arm.
   - Momentarily fold the remaining part of the splint down.
   - Pass the loose end of the stockinette around the neck.
8. Definitively secure the splint with an elastic or self-adherent bandage.
   - Wrap the elastic or self-adherent bandage around the arm and splint.
   - Having an assistant support the arm and/or the splint can be helpful.
9. Apply the mold. Most fractures require a two-point mold, with one hand anterolateral at the fracture site and the other posteromedial at the elbow.
10. Complete the stockinette:
    - Tie a slip knot in the loose end of the stockinette (an overhand knot with a draw-loop).
    - Place the wrist of the affected side inside the slip knot to create a collar-and-cuff type construct.
11. Place a cast padding wedge under the arm to counteract varus displacement of the fracture (optional).
12. A posterior slab may be added to control elbow motion for more distal fractures.



POSTERIOR ELBOW SPLINT

I. Overview

- A posterior elbow splint is inherently weak. Struts must be added to prevent elbow extension in the splint.
- Two techniques are described for stabilization of the posterior elbow splint:
  - External struts with tape
  - Internal struts with plaster

II. Indications for Use

- Fractures about the elbow
- Postoperative/postinjury elbow immobilization
- Elbow dislocations

III. Precautions

- The wrist is usually immobilized to control for pronation and supination about the elbow. Elbow dislocations should be splinted in at least 90 degrees of flexion with the wrist in pronation.
- Ensure that the splint remains proximal to the palmar flexion crease to preserve complete finger range of motion.
- At the antecubital fossa, do not allow edges of cast padding to lay immediately within the fossa borders.
  - Allowing edges of cast padding to lay immediately within the fossa borders will create wrinkling and can lead to skin breakdown in this very fragile area.
  - Span the fossa by having the midpoint of the cast padding roll directly over the elbow flexion crease.
  - By having the midpoint of the cast padding roll directly over the elbow flexion crease, the cast padding will be slightly tented above the fossa and will not be in direct contact, thus reducing the risk of skin breakdown.
- Because the olecranon and ulnar styloid are at risk in this splint, care should be taken to apply additional padding over these areas.

IV. Pearls

- Posterior elbow slab is very weak and does not provide significant immobilization.
  - Some form of strut must be made to prevent flexion/extension
  - The struts can be internal to the splint or external to the splint:
    - Internal struts are made of plaster and applied directly to the posterior slab, providing resistance to both flexion and extension.
    - External struts are made of tape and applied to the splint after it is definitively secured, providing resistance to extension but not flexion.

Have the patient or an assistant hold the hand of the affected side by the fingertips to help with positioning and reduce pain.

V. Equipment

- Cast padding: 3 inches wide
- Plaster: 4 inches wide and 2 inches wide
- Elastic or self-adherent bandage: 4 inches wide
- Bucket of tepid water
- Tape (optional): 2 inches

VI. Basic Technique

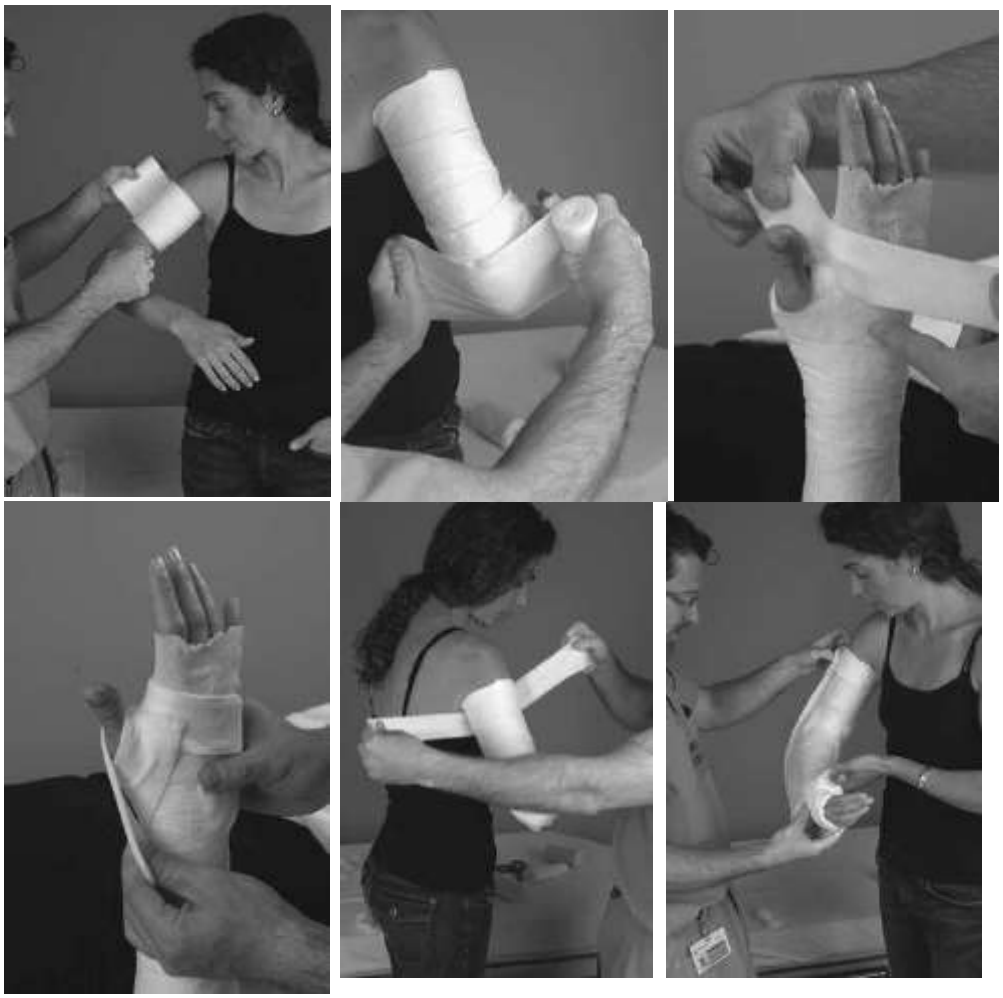
1. Patient positioning:	a. The patient should sit upright with his or her shoulder off the side of the bed. b. The elbow should be in the desired position of flexion and pronation/supination.
2. Where to start:	a. Proximal to the palmar flexion crease
3. Where to finish:	a. Immediately distal to the axillary fold of the arm
4. Where to mold:	a. Slight supracondylar mold above the elbow

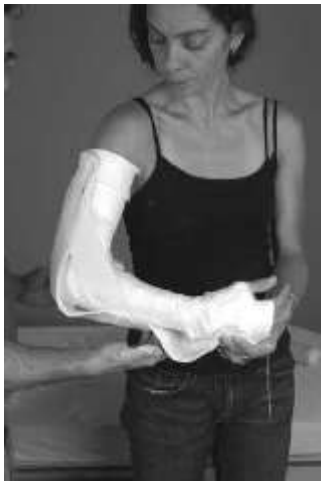
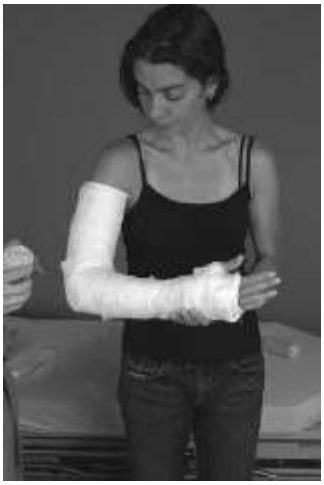
VII. Detailed Technique

1. Measure the length of the splint using plaster.
   - Use the contralateral side.
   - Start proximal to the palmar flexion crease.
   - End immediately below the axillary fold.
2. Roll out the plaster.
   - The posterior slab should be 10 to 12 layers thick and 4 inches wide.
   - Side struts can be 8 to 10 layers thick and 2 inches wide.
3. Position the patient:
   - Standing or sitting upright
   - Arm freely off to the side
   - Elbow bent to desired degree of flexion
4. Wrap the extremity in cast padding.
   - Start at either end.
   - Circumferentially wrap with cast padding, using a standard 50% overlap technique. Two layers of wrapping are sufficient.
   - Carefully tear the cast padding so it conforms around the thumb interspace. Do not go past the palmar flexion crease.
   - Span the fossa with cast padding at the antebrachial fossa (see aforementioned precautions).

- Tear several small strips of cast padding and apply them over the bony prominences of the olecranon and ulnar styloid to provide additional padding.

5. Create three cast padding cuffs:
   - Palmar flexion crease/metacarpal heads: This cuff should form a “V” at the ulnar aspect of the hand to allow for the cascade of the digits.
   - Thumb: This cuff should form a “V” at the base of the thumb.
   - Proximal forearm: This cuff can be circular.
6. Prepare the plaster. Use the usual technique of wetting and laminating.
7. Apply the plaster.
   - Apply the posterior slab first.
     - Position the posterior slab over the ulnar border of the forearm, around the olecranon and the posterior aspect of the arm.
     - Provisionally secure with cast padding at the wrist, forearm, and arm if necessary.
   - Apply side struts (if not using external struts).
     - Start laterally at the mid arm and angle obliquely toward the forearm. Laminate the side strut to the posterior slab.
     - Apply the medial side strut in the same position if additional stability is needed. Laminate the side strut to the posterior slab.
8. Cover the plaster. Wrap cast padding over the top of the plaster to prevent adhesion of the plaster to the elastic or self-adherent bandage.
9. Definitively secure the splint with an elastic or self-adherent bandage
10. Apply molding if necessary.
11. Create an external strut (if no internal struts are used) after the plaster has set. Use the figure-of-8 technique with tape.
    - Take 2-inch silk tape and begin at the midpoint of the posterior aspect of the arm
    - Wrap around to the midpoint of the anterior aspect of the arm.
    - Span the fossa obliquely.
    - Attach tape to the opposite aspect of the forearm.
    - Wrap around the dorsal forearm.
    - Span the fossa obliquely.
    - Attach tape to the lateral aspect of the arm.
    - At the intersection of the figure of 8, wrap with tape.





LONG ARM CAST

I. Indications for Use

- Pediatric supracondylar humerus fractures
- Pediatric forearm fractures
- Pediatric unstable distal radius fractures
- Adult distal radius fractures
- Adult forearm fractures

II. Precautions

- Do not plaster over the thenar eminence.
- Do not extend the plaster beyond the palmar crease. The patient must be able to flex his or her metacarpophalangeal (MCP) joints to at least 70 degrees.
- At the antecubital fossa, do not allow edges of cast padding to lay immediately within the fossa borders.
  - Allowing edges of cast padding to lay immediately within the fossa borders will create wrinkling and can lead to skin breakdown in this very fragile area.
  - Span the fossa by having the mid point of the cast padding roll directly over the elbow flexion crease.
  - By having the mid point of the cast padding roll directly over the elbow flexion crease, the cast padding will be slightly tented above the fossa and will not be in direct contact, thus reducing the risk of skin breakdown.
- Because the olecranon and ulnar styloid are at risk in this splint, care should be taken to apply additional padding over these areas.
- Be prepared to bivalve the cast to prevent compartment syndrome if postreduction swelling occurs.
- Never cast an acute supracondylar fracture or floating elbow injury without bivalving.

III. Pearls

- The easiest method is to create a short arm cast and then continue with the long arm portion.
- Once you start “slinging” plaster more quickly, a long arm cast can be attempted in one step.
- The easiest place to begin is at the wrist. The natural contour of the arm will prevent sliding.
- It is difficult to control elbow flexion and forearm pronation when placing a long arm cast, especially in a child.
  - To partially alleviate this difficulty, it is useful to have older children pretend they are a “drama queen” and have them place the dorsal aspect of their hand on their forehead after the short arm cast has been applied.

- Once the patient is in this position, the long arm portion of the cast can be completed.
- Parents can aid with keeping the arm at 90 degrees of elbow flexion for younger children.

IV. Equipment

- Stockinette: 3 to 4 inches
- Cast padding: 3 to 4 inches
- Plaster: 4 inches
- Elastic or self-adherent bandage: 3 to 4 inches Basic Technique

1. Patient positioning:

- Supine:
  - Have thin patients move their body all the way to the contralateral side of the stretcher.
  - The patient can then rest his or her elbow on the stretcher.
- Upright: Use a bedside table to allow the patient to place his or her arm at a comfortable level if no reduction is required.
  - The elbow should be bent to 90 degrees with the arm upright.
  - The wrist position depends on the type of fracture and the location of the fracture.
    - Distal radius fracture: Wrist in pronation
    - Fracture of both bones of the forearm:
      - Proximal third: wrist in supination
      - Middle third: wrist in neutral
      - Distal third: wrist in pronation

2. Where to start: Palmar flexion crease

3. Where to finish: Upper arm, below the axillary fold

4. Where to mold

- Dependent on fracture
  - Distal radius fracture: Same as sugar tong
  - Fracture of both bones of the forearm:
    - Interosseous mold
    - Ulnar-sided flat mold

5. Steps

- Prepare the stockinette.
- Position the stockinette.
- Wrap the extremity in cast padding.
- Create three cast padding cuffs.
- Fold the stockinette over proximally and distally.
- Prepare the plaster roll in the usual fashion.
- Apply the plaster roll for a short arm cast.
- Reposition the patient.
- Apply the plaster roll to the elbow and arm.
- Split the cast, if necessary.

V. Detailed Technique

1. Prepare the stockinette by cutting it into two pieces:
   - One piece for the proximal portion at the arm
   - One piece for the distal portion at the hand
2. Position the stockinette:
   - Place one stockinette over the upper arm.
   - Cut a thumb hole in the mid portion of the other stockinette and fit
   - this piece over the hand.
   - The stockinette should extend to the proximal interphalangeal (PIP)
   - joints.
3. Wrap the extremity in cast padding.
   - Start at the wrist and circumferentially wrap distally.
     - Use a standard 50% overlap technique.
       - Two layers of padding are sufficient.
   - Carefully tear the cast padding so as to conform around the thumb interspace. Do not go past the palmar flexion crease.
   - Once the hand has been adequately padded, continue wrapping proximally to the proximal forearm.
   - Span the fossa with cast padding at the antebrachial fossa (see aforementioned precautions).
   - Continue proximally to area just below axillary fold.
4. Create three cast padding cuffs.
   - Palmar flexion crease/metacarpal heads: This cuff should form a “V” at the ulnar aspect of the hand to allow for the cascade of the digits.
   - Thumb: Again, form a “V” at the base of the thumb.
   - Proximal arm: This cuff can be circular.
5. Fold the stockinette over proximally and distally. Ensure that MCP motion is completely preserved.
6. Prepare the plaster roll in the usual fashion.
7. Apply the plaster roll for a short arm cast.
   - Start at the wrist and work distally.
   - Use a twisting or pinching motion to get through the thumb interspace. Twist the plaster roll 360 degrees in the interspace.
   - Roll two more times around the hand and through the thumb interspace.
   - Continue the plaster proximally. Use the standard technique of laminating while rolling.
   - Ensure that at least 3 to 5 layers of plaster are applied.
8. Reposition the patient if using the “drama queen” technique.
   - The patient should be supine.
   - The hand is on the forehead with the elbow flexed to 90 degrees.
9. Apply a plaster roll to the elbow and arm. Be vigilant about avoiding patient movement when applying this portion of the cast.
10. Apply a mold, if desired. For a fracture of both bones of the forearm:

- An interosseous mold should be made over the forearm by compressing the anterior and posterior surfaces to make the cast more oval and less cylindrical.
- A straight ulnar border also is applied to prevent the fracture from falling to varus.
  - A straight ulnar border can be applied at the same time as the interosseous mold.
  - Alternatively, after applying an interosseous mold, place the ulnar side of the cast on a hard flat surface to make sure it is flat
- A supracondylar humerus mold also can be applied if desired.

11. Split the cast if necessary

SUGAR-TONG SPLINT

I. Indications for Use

- Distal radius fractures

II. Precautions

- It is crucial that the splint be neither too long nor too short.
  - If the splint is too long and extends beyond the metacarpal heads, finger flexion is significantly impaired and permanent finger stiffness or contractures may ensue.
  - If the splint is too short, the reduction will not hold.
- Do not immobilize the thumb. This error is extremely common. Remember, the thumb must be able to oppose and thus, unlike the fingers, its range of motion begins at the basal joint (carpometacarpal joint).
- Do not mold the splint in such a fashion that the wrist is flexed beyond 10 to 15 degrees; otherwise acute carpal tunnel syndrome may occur.

III. Pearls

- The normal cascade of the fingers slopes downward from the index to the little finger.
  - A 30-degree cut can be made in one end of the plaster slab to incorporate this cascade.
  - A curvilinear cut can be made to free the thenar eminence about the thumb.
- Before measuring the splint, it may be useful to perform a hematoma block, thus allowing the block to become effective while the splint is being measured.
- It is better to measure the splint long rather than short. The splint can always be trimmed or folded over during application if it is too long, but a new splint will need to be made if it is too short.
- The cast padding may be measured 1 to 2 inches long on the volar aspect so that it may be folded over the end of the plaster splint, thus providing a comfortable and safe edge to the splint.
- The sugar-tong splint has a tendency to become very bulky at the elbow.
  - To prevent bulkiness at the elbow, a small cut is made in the plaster at the elbow once it has been applied and provisionally fixed.
  - The free ends are folded over one another to conform to the curvature of the flexed elbow.

IV. Equipment

- Stockinette: 3 inches
- Cast padding: 3 to 4 inches
- Plaster: 3 to 4 inches
- Elastic or self-adherent bandage: 3 to 4 inches

- Portable radiograph machine (optional)

V. Basic Technique

1. Patient positioning:
   - The patient is either supine on the stretcher with the entire shoulder girdle off the side or the patient is sitting or standing.
   - The elbow is bent at 90 degrees.
   - The splint may be applied while the patient is in traction.
2. Where to start:
   - Volar (palmar) aspect, immediately below the palmar crease
   - Where to finish:
     - Dorsal aspect, immediately below the metacarpal heads
   - Where to mold (3-point mold):
     - For a dorsally angulated distal radius fracture:
       - Dorsal aspect of the carpus
       - Volar forearm immediately proximal to the wrist crease
       - Dorsal aspect of the forearm
     - For a volarly angulated distal radius fracture:
       - Volar aspect of the carpus
       - Dorsal forearm immediately proximal to the wrist crease
       - Volar aspect of the forearm
   - Steps
     - Measure the length of the splint using plaster.
     - Roll out the plaster.
     - Roll out the cast padding.
     - Position the patient.
     - Set traction, if necessary.
     - Obtain traction views, if necessary.
     - Perform a reduction maneuver, if necessary.
     - Prepare plaster in the usual fashion.
     - Apply a splint.
     - Definitively secure the splint with an elastic bandage.
     - Perform a three-point mold.

VI. Detailed Technique

1. Measure the length of the splint using plaster.
   - Use the contralateral side.
   - Start the volar (palmar) aspect at the palmar flexion crease.
   - Wrap around the elbow and finish at the metacarpal heads.
2. Roll out the plaster. The plaster slab should be 10 sheets thick.
3. Roll out the cast padding. Use the usual technique that will allow the padding to be folded over.
4. Position the patient:

- The patient should be supine on the stretcher with the shoulder girdle entirely off the side.
- Prepare finger traps if using traction.
- Abduct the shoulder to 90 degrees and flex the elbow to 90 degrees to create a 90-90 position.

5. Set traction, if necessary.
6. Obtain traction views, if necessary (this step may be performed following the reduction maneuver, as desired).
7. Perform a reduction maneuver, if necessary.
8. Prepare plaster in the usual fashion, with wetting and laminating, followed by placement in cast padding.
9. Apply a splint.
   - Begin at the volar aspect. Do not go past the palmar crease.
   - Wrap the splint around the elbow.
   - Provisionally secure the splint at the wrist with cast padding
   - Ensure that the edges of the splint are at the correct length.
     - If the edges are too long, fold them over.
     - If the edges are too short, the splint will need to be remade.
   - Cut a slit in the splint at the elbow to prevent bulk. Fold the edges over one another.
10. Definitively secure the splint with elastic or a self-adherent bandage.
    - Start at the elbow.
    - Ensure that the elastic or self-adherent bandage has only minimal contact with the skin.
    - Wrap the elastic or self-adherent bandage distally. A hole can be cut out for the thumb, if desired.
    - Secure the end with silk tape if needed.
11. Perform a three-point mold, if desired.

SHORT ARM CAST

I. Indications for Use

- Distal radius fracture (nonacute)
- Nonscaphoid carpal fractures
- Distal ulna fracture

II. Precautions

- Do not plaster over the thenar eminence.
- Do not extend the plaster beyond the palmar crease. The patient must be able to flex his or her MCPs to 90 degrees.
- Bony prominences, such as the ulnar styloid, must be well padded. Additional strips of padding may be applied over these potential pressure points.

III. Pearls

The easiest place to begin is at the wrist. The natural contour of the arm will prevent sliding.

IV. Equipment

- Stockinette: 3 inches
- Cast padding: 3 inches
- Plaster: 3 inches
- Elastic or self-adherent bandage: 3 inches

V. Basic Technique

1. Patient positioning:
   - The elbow should be bent to 90 degrees with the arm upright.
   - Supine:
     - Have thin patients move their body all the way to the contralateral side of the stretcher.
     - The patient can then rest his or her elbow on the stretcher.
   - Upright: Use a bedside table to allow the patient to place his or her arm at a comfortable level.
2. Where to start: Palmar flexion crease
3. Where to finish: Mid forearm
4. Where to mold (three-point mold)
   - For a dorsally angulated or displaced distal radius fracture:
     - Dorsal aspect of carpus
     - Volar distal forearm
     - Dorsal aspect of the mid forearm

- For a volarly angulated or displaced distal radius fracture:
  - Volar aspect of the carpus
  - Dorsal distal forearm immediately proximal to the wrist crease
  - Volar aspect of the mid forearm

5. Steps

- Prepare a stockinette.
- Position the stockinette.
- Wrap the extremity in cast padding.
- Create three cast padding cuffs.
- Fold the stockinette over both proximally and distally.
- Prepare the plaster roll in the usual fashion.
- Apply the plaster roll.
- Split the cast, if necessary.

VI. Detailed Technique

1. Prepare a stockinette by cutting it into two pieces:
   - One piece for the proximal portion at the elbow
   - One piece for the distal portion at the hand
2. Position the stockinette:
   - Place one stockinette over the elbow with equal lengths on either side.
   - Cut a thumb hole in the mid portion of the other stockinette and fit this portion over the hand.
   - The stockinette should extend to the PIP joints.
3. Wrap the extremity in cast padding.
   - Start at the wrist and circumferentially wrap distally.
     - Use a standard 50% overlap technique (see Chapter 12).
       - Two layers of padding are sufficient.
   - Carefully tear the cast padding so as to conform around the thumb interspace. Do not go past the palmar flexion crease.
   - Once the hand has been adequately padded, continue wrapping proximally to the proximal forearm.
4. Create three cast padding cuffs:
   - Palmar flexion crease/metacarpal heads: This cuff should form a “V” at the ulnar aspect of the hand to allow for the cascade of the digits.
   - Thumb: Again, form a “V” at the base of the thumb.
   - Proximal forearm: This cuff can be circular.
5. Fold the stockinette over both proximally and distally. Ensure that MCP motion is completely preserved.
6. Prepare the plaster roll in the usual fashion.
7. Apply the plaster roll.
   - Start at the wrist and work distally.
   - Use a twisting motion to get through the thumb interspace.
     - Twist the plaster roll 360 degrees in the interspace.

- Alternatively, if using fiberglass, the fiberglass can be cut to conform to the thumb interspace.
- Roll two more times around the hand and through the thumb interspace. Do not go past the palmar flexion crease.
- Continue the plaster proximally. Use the standard technique of laminating while rolling.
- Ensure that at least three to five layers of plaster are applied.

8. Split the cast, if necessary.

THUMB SPICA CAST

I. Indications for Use

- Scaphoid fracture (nonacute)

II. Precautions

- Do not extend the plaster beyond the palmar crease. The patient must be able to flex his or her MCP joints to 90 degrees.
- Bony prominences, such as the ulnar styloid, must be well padded. Additional strips of padding may be applied over these potential pressure points.

III. Pearls

- The easiest place to begin is at the wrist. The natural contour of the arm will prevent sliding.
- For proximal phalangeal fractures or MCP joint dislocations, a hand based thumb spica cast can be used.
  - A hand-based thumb spica cast begins just distal to the wrist crease and extends to cover the entire thumb.
  - A hand-based thumb spica cast allows wrist motion.

IV. Equipment

- Stockinette: 3 inches
- Cast padding: 3 inches
- Plaster: 3 inches
- Elastic or self-adherent bandage: 3 inches

V. Basic Technique

- Patient positioning:
  - The elbow should be bent to 90 degrees with the arm upright.
  - Supine
    - Have thin patients move their body all the way to the contralateral side of the stretcher.
    - The patient can then rest his or her elbow on the stretcher.
  - Upright: Use a bedside table to allow the patient to place his or her arm at a comfortable level.
- Where to start:
  - Thumb:
    - Distal to the interphalangeal (IP) joint for injuries at or distal to the metaphalangeal (MP) joint

- Proximal to IP joint for injuries proximal to MP joint
- Hand:
  - Proximal to the distal palmar crease
- Where to finish: Upper forearm
- Where to mold:
  - Interosseous mold
- Steps:
  - Prepare a stockinette.
  - Position the stockinette.
  - Wrap the extremity in cast padding
  - Create three cast padding cuffs.
  - Fold the stockinette over both proximally and distally.
  - Prepare the plaster roll in the usual fashion.
  - Apply the plaster roll.
  - Split the cast if necessary.

VI. Detailed Technique

1. Prepare the stockinette by cutting it in two pieces:
   - One piece is for the proximal portion at the elbow
   - One piece is for the distal portion at the hand
2. Position the stockinette
   - Place one stockinette over the elbow with equal lengths on either side.
   - Cut a thumb hole in the mid portion of the other stockinette and fit this portion over the hand.
   - The stockinette should extend to the PIP joints.
3. Wrap the extremity in cast padding.
   - Start at the wrist and circumferentially wrap distally
     - Use a standard 50% overlap technique
     - Two layers of padding are sufficient.
   - Carefully tear the cast padding so as to conform around the thumb interspace. Do not go past the palmar flexion crease.
   - Tear several strips of cast padding to pad the thumb.
   - Once the hand has been adequately padded, continue wrapping proximally to the proximal forearm.
4. Create three cast padding cuffs:
   - Palmar flexion crease/metacarpal heads: This cuff should form a “V” at the ulnar aspect of the hand to allow for the cascade of the digits
   - Thumb: This cuff can be circular distal to the IP joint.
   - Proximal forearm: This cuff can be circular.
5. Fold the stockinette over both proximally and distally. Ensure that MP joint motion is completely preserved.
6. Prepare the plaster roll in the usual fashion.
7. Apply the plaster roll.
   - Start at the wrist and work distally.

- Use a twisting or pinching motion to get through the thumb interspace.
  - Twist the plaster roll 360 degrees in the interspace.
  - Pinch the roll to narrow it at the web space.
- Roll two more times around the hand and through the thumb interspace.
- Roll around the thumb, being careful not to extend beyond the cast padding.
- Continue the plaster proximally
  - Use the standard technique of laminating while rolling
- Ensure that at least three to five layers of plaster are applied.

8. Mold the cast, if desired. An interosseous mold over the forearm should be made by compressing the anterior and posterior surfaces to make the cast more oval and less cylindrical.
9. Split the cast, if necessary

THUMB SPICA SPLINT

I. Indications for Use

- Scaphoid fractures
- Thumb metacarpal fractures
- Thumb carpometacarpal dislocations

II. Precautions

Be careful not to create a mold that is too dramatic, because the indentation in the plaster can lead to skin necrosis and pressure sores.

III. Pearls

- The application of cast padding is the most important aspect of this splint.
- Careful attention should be paid to the area around the thumb to avoid bunching and wrinkles.

IV. Equipment

- Stockinette: 3 inches
- Cast padding: 3 inches
- Plaster: 4 inches
- Elastic or self-adherent bandage: 3 inches

V. Basic Technique

1. Patient positioning:
   - The elbow is bent to 90 degrees with the arm upright.
   - Supine:
     - Have patients who are thin move their body all the way to the contralateral side of the stretcher.
     - The patient can then rest his or her elbow on the stretcher.
   - Upright:
     - Use a bedside table to allow the patient to place his or her arm at a comfortable level.
2. Where to start
   - Distal to the thumb interphalangeal (IP) joint for thumb fractures
   - Just proximal to the IP joint for scaphoid fractures
3. Where to finish: Mid to proximal forearm
4. Where to mold: The thumb should be abducted 30 degrees away from the hand, in its neutral position.
5. Steps:

- Measure the length of the splint using plaster.
- Roll out the plaster.
- Wrap the extremity in cast padding.
- Create three cast padding cuffs.
- Prepare the plaster.
- Apply the plaster.
- Cover the plaster.
- Definitively secure the splint with an elastic or self-adherent bandage.

Detailed Technique

1. Measure the length of the splint using plaster. Start at the thumb and go to the mid to proximal forearm.
2. Roll out the plaster. The plaster slab should be 10 sheets thick.
3. Wrap the extremity in cast padding
   - Start at the wrist and circumferentially wrap distally to the hand.
     - Use a standard 50% overlap technique
     - Two layers of wrapping are sufficient.
   - Carefully tear the cast padding so as to conform around the thumb interspace. Do not go past the palmar flexion crease.
   - Once the hand has been adequately padded, continue wrapping proximally to the mid forearm.
4. Create three cast padding cuffs:
   - Palmar flexion crease/metacarpal heads: The cuff should form a “V” at the ulnar aspect of the hand to allow for the cascade of the digits
   - Thumb: Padding cuff at the tip of the thumb.
   - Proximal forearm: This cuff can be circular.
5. Prepare the plaster. Employ the usual technique of wetting and laminating.
6. Apply the plaster:
   - Position the plaster on the radial border of the thumb and forearm
   - Provisionally secure the plaster at the wrist with cast padding
7. Cover the plaster. Place strips of cast padding over the top of the plaster to protect the elastic bandage.
8. Definitively secure the plaster with an elastic bandage:
   - Begin at the thumb and work distally.
   - An opening for the thumb web space can be cut into the elastic or self-adherent bandage.
   - Work distally to the mid forearm.
   - Secure the end with silk tape.

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DISTAL RADIUS FRACTURE (WRIST FRACTURE)

Authors: Ang Eng Sopheap, Sok Chan Pheaktra, Chheur Hengnaroth, Ly Kang, Kim Sopheaktra, Phan Daravuth, Huot Vutha

I. Definition

A distal radius fracture is a break in the radius bone near the wrist, often caused by a fall onto an outstretched hand. It is common in both older adults and athletes.

II. Etiology

Most common orthopaedic injury with a bimodal distribution

- younger patients - high energy
- older patients - low energy / falls
- 50% intra-articular

III. Diagnostic

A. Clinical argument

- The patient should be evaluated for
  - Age`
  - Dominance hand
  - Occupation
  - Level of activity
  - Quality of bone
  - General medical condition

- Clinical symptoms : like in general trauma, it presents as :
  - Pain
  - Swelling
  - Deformity in displaced fracture / non or minimal deformity in non-displaced fracture
  - Ecchymosis
  - Limitation of wrist movement or impotence
  - Bone crepitus

- Associated injuries
  - distal radio-ulna joint injuries must be evaluated

- radial styloid fracture - indication of higher energy
- soft tissue injuries in 70%
- hand lesions : bones, tendons, nerves and vascular
- other trauma lesions : others bone fracture, head, chest, abdominal …etc. in case of polytraumatic injury

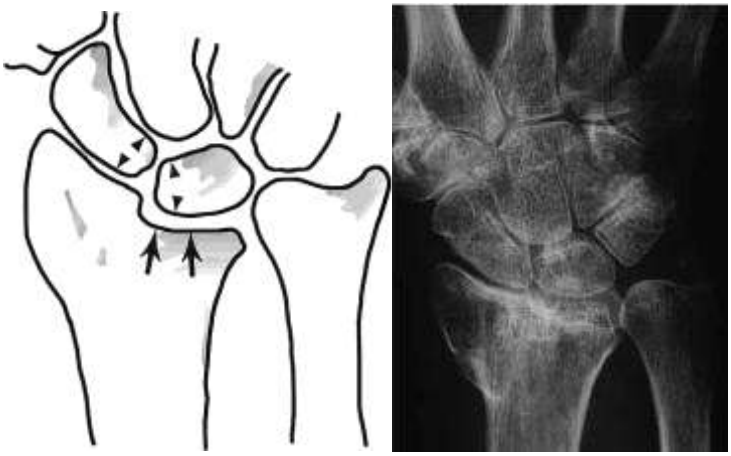
- Osteoporosis
  - high incidence of distal radius fractures in women >50
  - distal radius fractures are a predictor of subsequent fractures: DEXA scan is recommended in woman with a distal radius fracture

B. Classification

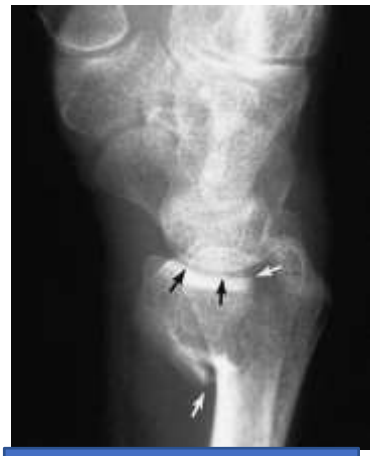
1. AO/ OTA Classification

2. Eponyms: see table for list of commonly used eponyms

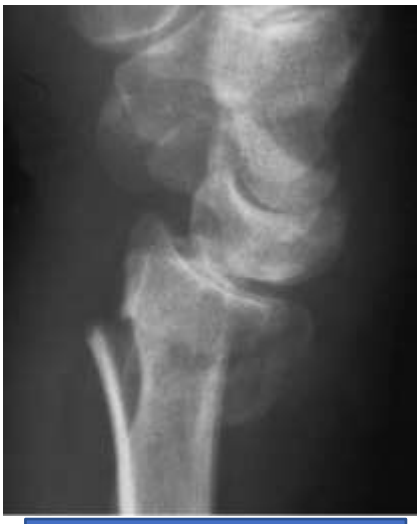
Eponyms	
Die-punch fxs	A depressed fracture of the lunate fossa of the articular surface of the distal radius
Barton's fx	Fx dislocation of radiocarpal joint with intra-articular fx involving the volar or dorsal lip (volar Barton or dorsal Barton fx)
Chauffer's fx	Radial styloid fx
Colles' fx	Low energy, dorsally displaced, extra-articular fx
Smith's fx	Low energy, volar displaced, extra-articular fx



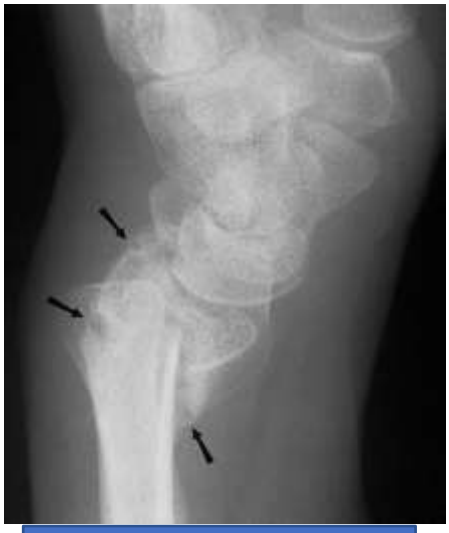
Die-punch fxs



Barton's fracture



Colles's fracture

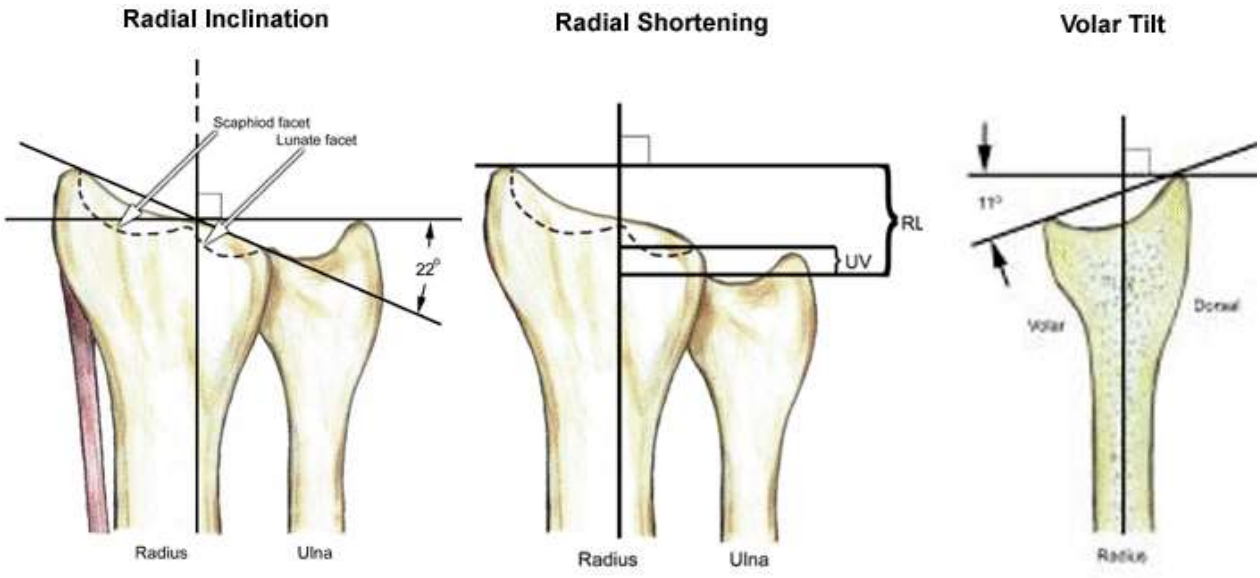


Smith's fracture

C. Imaging

1. Radiographs

View	*Measurement*	*Normal*	*Acceptable criteria*
AP	Radial height	13 mm	<5 mm shortening
	Radial inclination	23 degrees	change <5°
	Articular stepoff	Congruous	<2 mm stepoff
LAT			dorsal angulation <5° or within 20° of contralateral distal radius



2. CT scans

- important to evaluate intra-articular involvement and for surgical planning

3. MRI useful to evaluate for soft tissue injury

- TFCC injuries
- scapholunate ligament injuries (DISI)
- lunotriquetral injuries (VISI)

IV. Treatment

Successful outcomes correlate with

- accuracy of articular reduction
- restoration of anatomic relationships
- early efforts to regain motion of wrist and fingers

A. **Nonoperative**
Closed reduction and cast immobilization

- indications
  - extra-articular
  - <5mm radial shortening
  - dorsal angulation <5° or within 20° of contralateral distal radius
  - technique (see below)

B. **Operative**
Surgical fixation (CRPP, External Fixation, ORIF)

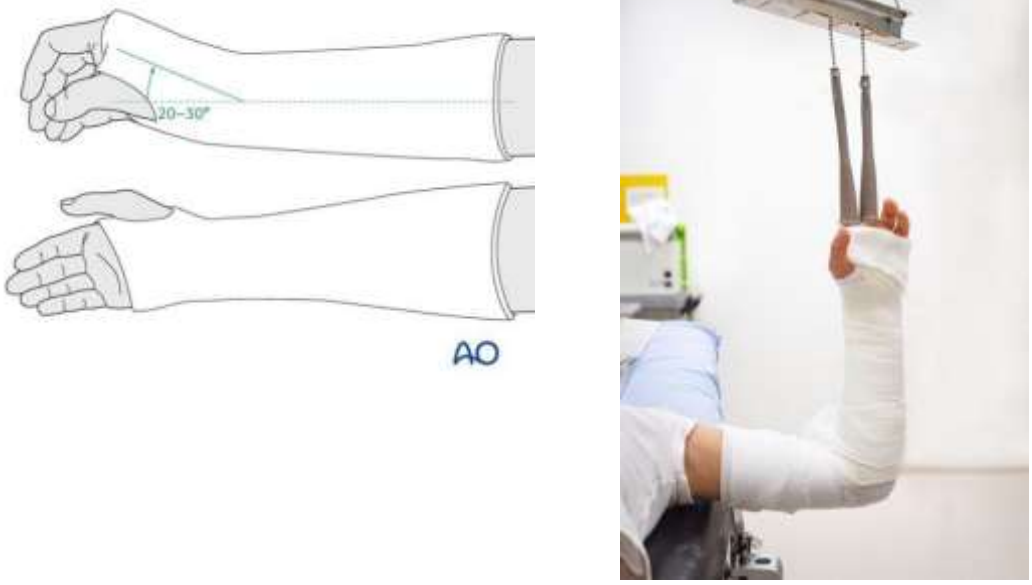
- indications: radiographic findings indicating instability (pre-reduction radiographs best predictor of stability)
  - displaced intra-articular fracture
  - volar or dorsal comminution
  - articular margins fracture
  - severe osteoporosis

- dorsal angulation >5° or >20° of contralateral distal radius
- >5mm radial shortening
- comminuted and displaced extra-articular fractures (Smith's fracture)
- progressive loss of volar tilt and loss of radial length following closed reduction and casting
- associated ulnar styloid fractures do not require fixation

C. Techniques

1. Closed reduction and cast immobilization

- Indications
  - most extra-articular fractures
- Technique
  - rehabilitation : no significant benefit of physical therapy over home exercises for simple distal radius fractures treated with cast immobilization
- Outcomes
  - repeat closed reductions have 50% less than satisfactory results
- Complications
  - acute carpal tunnel syndrome: (see complications below)
  - Extensor pollicis longus rupture: (see complications below)



Short and long cast immobilization for distal radius fracture

2. Percutaneous Pinning

- Indications
  - can maintain sagittal length/alignment in extra-articular fxs with stable volar cortex

- cannot maintain length/alignment when unstable or comminuted volar cortex

- Techniques
  - Kapandji intrafocal technique
  - Rayhack technique with arthroscopically assisted reduction
- Outcomes
  - 82-90% good results if used appropriately

3. **ORIF (open reduction and internal fixation)**

- Indications
  - significant articular displacement (>2mm)
  - dorsal and volar Barton fractures
  - volar comminution
  - metaphyseal-diaphyseal extension
  - associated distal ulnar shaft fractures
  - die-punch fractures

- Technique
  - volar plating
    - volar plating preferred over dorsal plating
    - volar plating associated with irritation of both flexor and extensor tendons
      - rupture of FPL is most common with volar plates
      - associated with plate placement distal to watershed area, the most volar margin of the radius closest to the flexor tendons
    - new volar locking plates offer improved support to subchondral bone
  - dorsal plating
    - dorsal plating historically associated with extensor tendon irritation and rupture
    - dorsal approach indicated for displaced intra-articular distal radius fracture with dorsal comminution
  - other technical considerations
    - can combine with external fixation and PCP
    - bone grafting if complex and comminuted
    - study showed improved results with arthroscopically assisted reduction
    - volar lunate facet fragments may require fragment specific fixation to prevent early post-operative failure

4. **External Fixation**

- Indications
  - alone cannot reliably restore 10 degree palmar tilt
    - therefore usually combined with percutaneous pinning technique or plate fixation
- Technical considerations
  - relies on ligamentotaxis to maintain reduction
  - place radial shaft pins under direct visualization to avoid injury to superficial radial nerve

- nonspanning ex-fix can be useful if large articular fragment
- avoid overdistraction (carpal distraction < 5mm in neutral position) and excessive volar flexion and ulnar deviation
- limit duration to 8 weeks and perform aggressive OT to maintain digital ROM

- Outcomes
  - important adjunct with 80-90% good/excellent results
- Complications
  - malunion/nonunion
  - stiffness and decreased grip strength
  - pin complications (infections, fx through pin site, skin difficulties)
    - pin site care comprising daily showers and dry dressings recommended
  - neurologic (iatrogenic injury to radial sensory nerve, median neuropathy, RSD)

V. Complications

1. Median nerve neuropathy (CTS)
   - most frequent neurologic complication
   - 1-12% in low energy fractures and 30% in high energy fractures
   - prevent by avoiding immobilization in excessive wrist flexion and ulnar deviation (Cotton-Loder Position)
   - treat with acute carpal tunnel release for:
     - progressive paresthesia, weakness in thumb opposition
     - paresthesia do not respond to reduction and last > 24-48 hours

2. Ulnar nerve neuropathy
   - seen with DRUJ (distal radio-ulna joint) injuries

3. Extensor Policis Longus (EPL) Rupture (tendon of 1st finger)
   - nondisplaced distal radial fractures have a higher rate of spontaneous rupture of the extensor pollicis longus tendon
     - extensor mechanism is felt to impinge on the tendon following a nondisplaced fracture and causes either a mechanical attrition of the tendon or a local area of ischemia in the tendon.
   - treat with transfer of extensor indicis proprius to EPL (extensor policis longus)

4. Radiocarpal arthrosis (2-30%)
   - 90% young adults will develop symptomatic arthrosis if articular stepoff > 1-2 mm
   - may be nonsymptomatic

5. Malunion and Nonunion
   - Intra-articular malunion
     - treat with revision at > 6 weeks
   - Extra-articular angulation malunion
     - treat with opening wedge osteotomy with ORIF and bone grafting
   - Radial shortening malunion

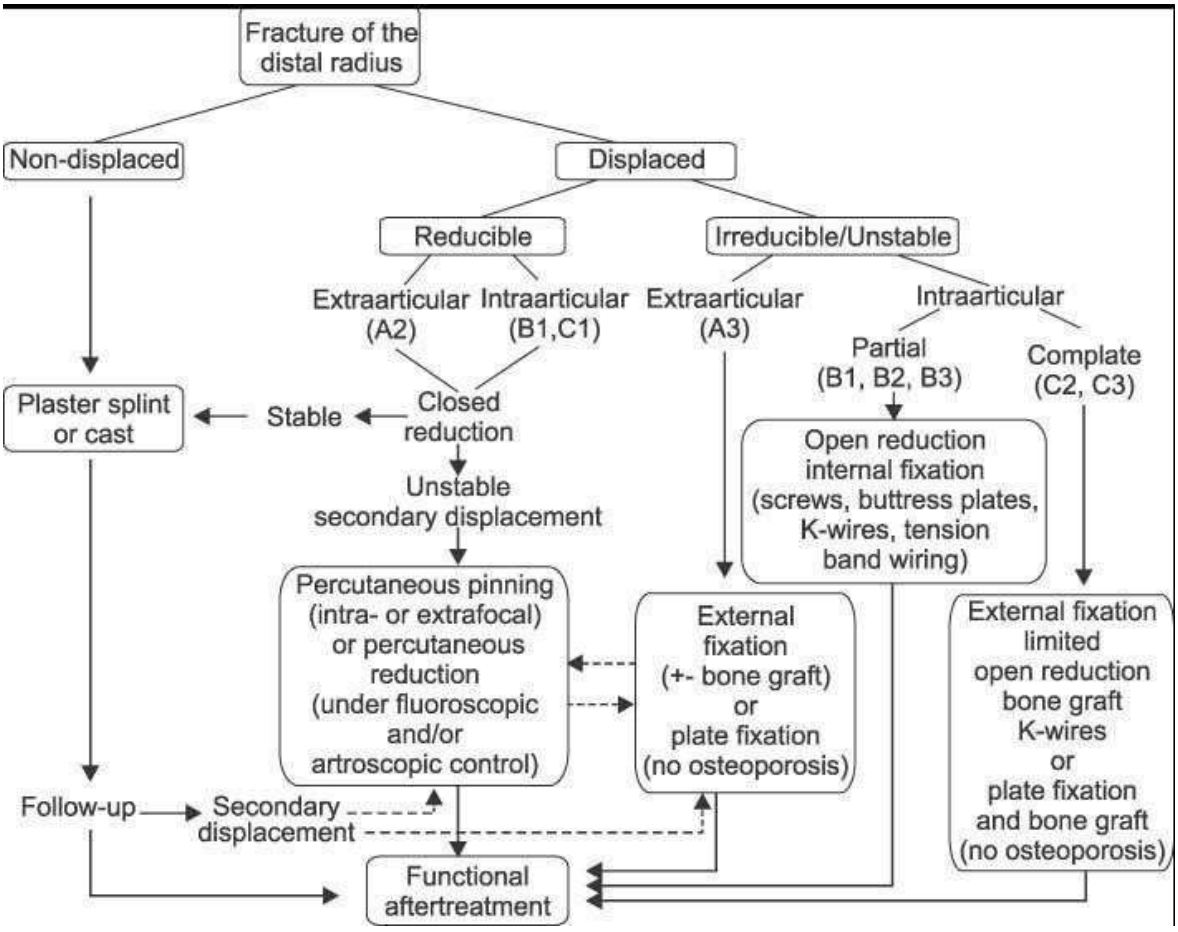
    - radial shortening associated with greatest loss of wrist function and degenerative changes in extra-articular fxs
    - treat with ulnar shortening

6. ECU or EDM entrapment (Extensor Carpi Ulnaris or Extensor Digitis Minimis)
    - entrapment in DRUJ injury

7. Compartment syndrome

8. RSD/CRPS (Reflex Sympathetic Dystrophy/Complex Regional Pain Syndrome)
    - AAOS 2010 clinical practice guidelines recommend vitamin C supplementation to prevent incidence of RSD postoperatively

VI. Algorithm



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CHAPTER VIII
UROLOGICAL SURGERY

1. Bladder stone
2. Bladder trauma
3. Benign prostatic hypertrophy
4. Erectile dysfunction
5. Fournier Gangrene
6. Hematuria 7. Hydrocele
8. Hypospadia
9. Inguinal hernia
10. Kidney stone
11. Male sterilization vasectomy
12. Muscular invasive bladder cancer
13. Nephrostomy catheter care
14. Neurogenic bladder
15. Non-Muscular invasive bladder cancer
16. Paraphimosis
17. Penile cancer
18. Penile fracture
19. Phimosis in Infants and Adults
20. Posterior urethral valve
21. Priapism
22. Prostate cancer
23. Renal cell carcinoma
24. Renal trauma
25. Stress urinary incontinence
26. Suprapubic catheterization
27. Testicular torsion
28. Testicular trauma
29. Transurethral catheterization
30. Undescended testis
31. Ureteral stone
32. Ureteropelvic junction obstruction
33. Urethral stone
34. Urethral stricture
35. Urinary retention
36. Urinary stone disease
37. Urinary tract infection
38. Urine collection
39. Urosepsis
40. Varicocele
41. Vesico-ureteal refiux
42. Vesicovaginal fistula

MANAGEMENT OF BLADDER STONE

HAY VANEL, LAM KORVIN, BOU SOPHEAP, OUK REAKSMEY

I. CASE DEFINITION

The present of Urinary stone in the bladder

II. ETIOLOGY

2.1-Low fluid intake 2.2-
Hypercalciuria
2.3-Primary hyperparathyroidism 2.4-
Hypocitraturia
2.5-High animal protein intake 2.6-
Primary hyperoxaluria

III. DIAGNOSTIC PROCEDURE

3.1- Ultrasound

Ultrasound is the primary imaging technique in children. Its advantages are absence of radiation and no need for anaesthesia. Imaging should include both the fluid-filled bladder with adjoining portion of the ureters, as well as the upper ureter. Color Doppler US shows differences in the ureteral jet [87] and resistive index of the arciform arteries of both kidneys, which are indicative of the grade of obstruction [88]. Nevertheless, US fails to identify stones in > 40% of children and provides limited information on renal function.

3.2- KUB X ray

Kidney-ureter-bladder radiography can help to identify stones and their radiopacity and facilitate follow-up.

3.3-Non-contrast-enhanced computed tomography

Recent low-dose CT protocols have been shown to significantly reduce radiation exposure. In children, only 5% of stones escape detection by NCCT. Sedation or anesthesia is rarely needed with modern high-speed CT equipment.

3.4-Magnetic resonance urography

Magnetic resonance urography (MRU) cannot be used to detect urinary stones. However, it might provide detailed anatomical information about the urinary collecting system, the location of an obstruction or stenosis in the ureter, and renal parenchymal morphology.

3.5-Intravenous urography

The radiation dose for IVU is comparable to that for voiding cysto-urethrography (0.33 mSV) .However, the need for contrast medium injection is a major drawback.

3.6-Diagnostic investigation for recurrent stone former

Nowadays, due to our limited resources on metabolic/genetic tests, etiologic workups are encouraged (optional) and should be done with multidisciplinary team.

Table 1. Basic evaluation of a stone former

Investigation	Rationale for investigation
Medical history and physical Examination	Stone history (Prior stone events, family history) Dietary habits Medication chart
Diagnostic imaging	Ultrasound
Blood analysis	Creatinine Calcium (ionized calcium or total calcium + albumin) Uric acid
Urinalysis	Dipstick test: Leukocytes, erythrocytes, nitrite, Protein, urine pH, specific weight Urine culture

IV. DIFFERENTIAL DIAGNOSIS

The following are some important differentials to be considered in a patient presenting with the above-mentioned features:

- Lower urinary tract infection
- Pyelonephritis
- Renal abscess
- Renal artery aneurysm
- Appendicitis
- Diverticulitis
- Mesenteric ischemia
- Pancreatitis

V. THERAPUETIC APPROACH FOR BLADDER

5.1. Conservative treatment and Indications for active stone removal

Migratory bladder stones in adults may typically be left untreated, especially asymptomatic small stones. Rates of spontaneous stone passage are unknown, but data on ureteric stones suggest stones < 1 cm are likely to pass in the absence of BOO, bladder dysfunction, or long-term catheterisation (see section 3.4.9 Specific stone management of ureteral stones).

Primary and secondary bladder stones are usually symptomatic and are unlikely to pass spontaneously: active treatment of such stones is therefore indicated.

5.2. Medical management of bladder stones

There is a paucity of evidence on chemolitholysis of bladder stones. However, guidance on the medical management of urinary tract stones in section 3.4.9 Specific stone management of ureteral stones, can be applied to urinary stones in all locations.

Uric acid stones can be dissolved by oral urinary alkalinisation when a PH > 6.5 is consistently achieved, typically using alkaline citrate or sodium bicarbonate. Regular monitoring is required during therapy (see section 3.4.4 Chemolysis). Irrigation chemolysis is also possible using a catheter; however, this is time-consuming may cause chemical cystitis and is therefore not commonly employed [734,735].

5.3. Bladder stone interventions

Minimally invasive techniques for the removal of bladder stones have been widely adopted to reduce the risk of complications and shorten hospital stay and convalescence. Bladder stones can be treated with open, laparoscopic, robotic-assisted laparoscopic, endoscopic (transurethral or percutaneous) surgery or ESWL [736].

5.3.1. Suprapubic cystolithotomy

Open suprapubic cystolithotomy is very effective but is associated with a need for catheterisation and longer hospital stay in both adults and children compared to all other stone removal modalities [736]. In children, a non-randomised study found that, if the bladder was closed meticulously in two layers, “tubeless” (drain-less and catheter-less) cystolithotomy was associated with a significantly shorter length of hospital stay compared with traditional cystolithotomy, without significant differences regarding late or intra-operative complications provided that children with prior UTI, recurrent stones, or with previous surgery for anorectal malformation (or other relevant surgery) were excluded [737].

5.3.2. Transurethral cystolithotripsy

In both adults and children, transurethral cystolithotripsy provides high SFRs and appears to be safe, with a very low risk of unplanned procedures and major post-operative and late complications [736].

5.3.3. Percutaneous cystolithotripsy

5.3.4. Extracorporeal shock wave lithotripsy

5.3.5. Laparoscopic cystolithotomy

5.5- General metabolic considerations for patient workup and recurrence prevention

5.5.1-Evaluation of patient risk

All patients should undergo stone analysis using infrared spectroscopy or X-ray diffraction prior to metabolic evaluation [8]. Stone analysis should be performed in recurrent stone formers during each stone episode, even if the initial stone composition is known, because changes in stone content have been reported in recurrent stone formers. When stone analysis is not available, a specific workup of the patient should be performed.

Table 2. High-risk stone formers

General Factor
Early onset of urolithiasis (especially children and teenagers)
Familial stone formation
Brushite-containing stones (calcium hydrogen phosphate; $CaHPO_4 2H_2O$)
Uric acid and urate-containing stones
Infection stones
Solitary kidney (the solitary kidney itself does not particularly increase risk of stone formation, but prevention of stone recurrence is of more importance)
Diseases associated with stone formation
Hyperparathyroidism

Nephrocalcinosis
Gastrointestinal diseases (ie, jejuno-ileal bypass, intestinal resection, Crohn's disease, malabsorptive conditions, enteric hyperoxaluria after urinary diversion) and bariatric surgery
Sarcoidosis
Genetically determined stone formation
Cystinuria (type A, B, AB)
Primary hyperoxaluria (PH)
Renal tubular acidosis (RTA) type I
2,8-Dihydroxyadenine
Xanthinuria
Lesch-Nyhan syndrome
Cystic fibrosis
Drugs associated with stone formation
Anatomical abnormalities associated with stone formation
Medullary sponge kidney (tubular ectasia)
Ureteropelvic junction obstruction
Calyceal diverticulum, calyceal cyst
Ureteral stricture
Vesico-uretero-renal reflux
Horseshoe kidney
Ureterocele

5.5.2--General considerations for recurrence prevention

All stone formers, independent of their individual risk, should follow the preventive measures presented in Table 3.

Table 3. General preventive measures

Fluid intake (drinking advice)	Fluid amount : 2.5-3.0 l/d Circadian drinking (time controlled drinking) Neutral pH beverages Diuresis: 2.0-2.5 l/d Specific weight of urine: < 1.010
Nutritional advice for a balanced diet	Balanced diet Rick in vegetable and fiber Normal calcium content: 1-1.2 g/d Limited NaCl content: 4-5 g/d Limited animal protein content: 0.8-1.0 g/kg/d
Lifestyle advice to normalized General risk factors	BMI: 18-25 kg/m2 Stress limitation measures Adequate physical activity Balancing of excessive fluid loss

Only high-risk stone formers require specific metabolic evaluation, which should be individualized based on different stone types. Specific metabolic evaluation requires collection of two consecutive 24-h urine samples.

VI. COMPLICATION OF BLADDER

Complications include acute renal failure secondary to obstruction, anuria, urinary tract infection with renal obstruction, and sepsis.

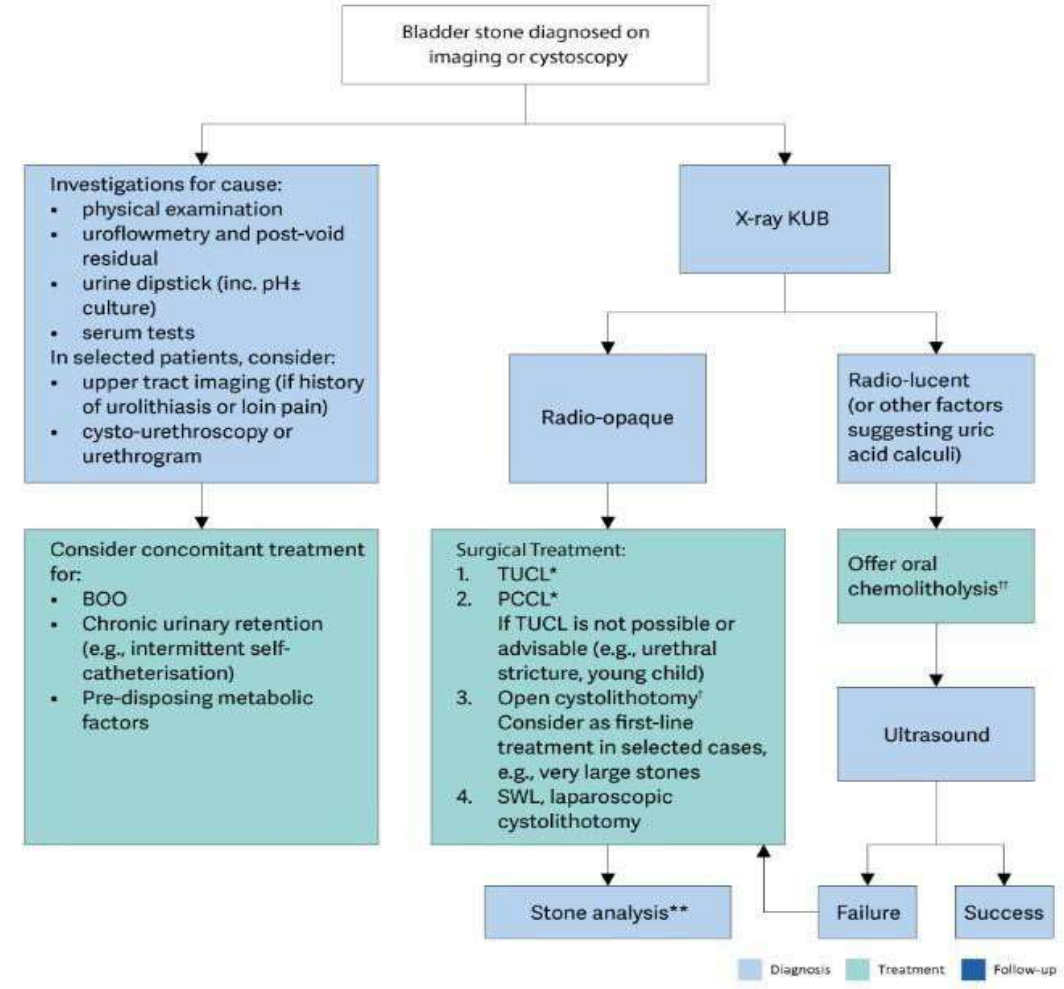
VII. CONCLUSION

After stone passage, every patient should be assigned to a group with low or high risk of stone formation. For correct classification, reliable stone analysis and basic evaluation of every patient are required. Low-risk stone formers may benefit by adopting general preventive measures regarding fluid and nutritional intake, as well as lifestyle improvements. For high-risk stone formers, a specific metabolic evaluation is required to guide individual treatment and prevent stone recurrence.

Follow up for recurrence stone:

- Low risk patient: follow up every 12 months evaluation (Urinalysis, renal function test, KUB ultrasound/X-ray)
- High risk patient: follow up every 6 months evaluation (Urinalysis, renal function test, KUB ultrasound/X-ray with specific tests)

Figure 6.1 Management of Bladder stones



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BLADDER TRAUMA

Dr. HAY VANEL, Dr. OUK REAKSMEY, Prof. BOU SOPHEAP

I. CASE DEFINITION

- Urinary Bladder Rupture is generally caused by a direct blow or penetrating trauma to the urinary bladder.
- The probability of bladder rupture is variable. A urinary bladder that is full is more prone to rupture than an empty one.
- In the past, diagnosis of Bladder Rupture was often missed or delayed.
- Early suspicion of Urinary Bladder Rupture, adequate radiological procedures, and immediate surgical intervention if indicated can successfully treat Bladder Rupture with least complications.
- Prognosis is excellent following early diagnosis with adequate medical and surgical measures.

II. ETIOLOGY

-Blunt trauma: bladder rupture and plevic fracture
-Penetrating trauma: gunshot and stabbing
-Obstetric trauma: forceps delivery
-Gynecologic trauma: hysterectomy
-Urologic trauma: biopsy, TURP, TURBT
-Orthopedic trauma: internal fixation of pelvic fractures
-Spontaneous Rupture: < 2%

III. DIAGNOSTIC PROCEDURE

+Signs and symptoms
    -Hematuria: 98% (microscopic 10%)
    -Suprapubic pain
    -No urine in the Foley Catheter
    -Intraperitoneal bladder rupture: Abdominal distention, Absent bowel sounds,
    -Signs of peritoneal irritation.

+Imagery study
-Laboratory test: UA, CBC, Creatininemia (pre-operation)
-Abdominal ultrasound: area of rupture, hematoma, intraperitoneal effusion
-Retrograde cystography: gold standard
-X-ray bone: Fracture
-Abdominal X-ray: Reflex ileus
-CT Scan with IV contrast: evaluate function and injury of kidney, ureter, bladder and urethra (diagnosis and stage).

Classification of bladder injuries by AAST by CT Scan

	Grade	Description
I.	Hematoma, Laceration	Contusion, Intramural hematoma, Partial thickness
II.	Laceration	Extraperitoneal bladder laceration < 2 cm
III.	Laceration	Extraperitoneal (>2 cm) or intraperitoneal (<2 cm) bladder wall laceration.
IV.	Laceration	Intraperitoneal bladder wall laceration > 2cm
V.	Laceration	Extraperitoneal and Intraperitoneal bladder wall laceration extending into bladder neck or urethral orifice (Trigone)

IV. DIFFERENTIAL DIAGNOSIS

-Urethral Rupture
-Upper urinary tract Traumatism
-Abdominal viscera Perforation
-Pelvic Fracture with hematoma

V. THERAPEUTIC APPROACH

+Conservative management

-Is the standard treatment for an uncomplicated extraperitoneal injury due to blunt or iatrogenic trauma.
-Can also be chosen for uncomplicated intraperitoneal injury after TURB or other operations, only in the absence of peritonitis and ileus.
-Penetrating extraperitoneal bladder injuries (only if minor and isolated) can also be managed conservatively.
-Most extraperitoneal bladder leaks: conservative by bladder drainage per urethral or suprapubic catheter (10 to 14 days)
-Approximately 85% will heal within 7 to 10 days
-Nearly all extraperitoneal bladder injuries heal within 3 weeks.
-Assessed for healing via cystogram

+Surgical management:

+Blunt non-iatrogenic trauma

-Extraperitoneal ruptures: bladder neck involvement, bone fragments in the bladder wall, concomitant rectal or vaginal injury or entrapment of the bladder wall necessitate surgical intervention.
-After surgery, cystogram is obtained 7-10 days.
-Intraperitoneal ruptures: Surgically repaired with a watertight two layer closure with absorbable suture and perivesical drain placement.
-Adequate drainage with a urethral catheter and suprapubic catheter for 10-14days.

+Penetrating non-iatrogenic trauma

-Penetrating bladder injury is managed by emergency exploration, debridement of devitalised bladder wall and primary bladder repair.

-In gunshot wounds, there is a strong association with intestinal and rectal injuries, usually requiring faecal diversion.

-As the penetrating agent (bullet, knife) is not sterile, antibiotic treatment is advised.

+Iatrogenic bladder trauma

-Perforations recognised intra-operatively are primarily closed

-The standard of care for intraperitoneal injuries is surgical exploration and repair.

-For extraperitoneal injuries, exploration is only needed for perforations complicated by symptomatic extravesical collections. It requires drainage of the collection, with or without closure of the perforation.

+Complication of bladder surgery

-Persistent of urinary extravasation

-Wound dehiscence

-Hemorrhage

-Pelvic abscess

-Intra-abdominal infection

-Urinary tract infection

-Low bladder capacity

VI. COMPLICATION

+Hypovolemic shock

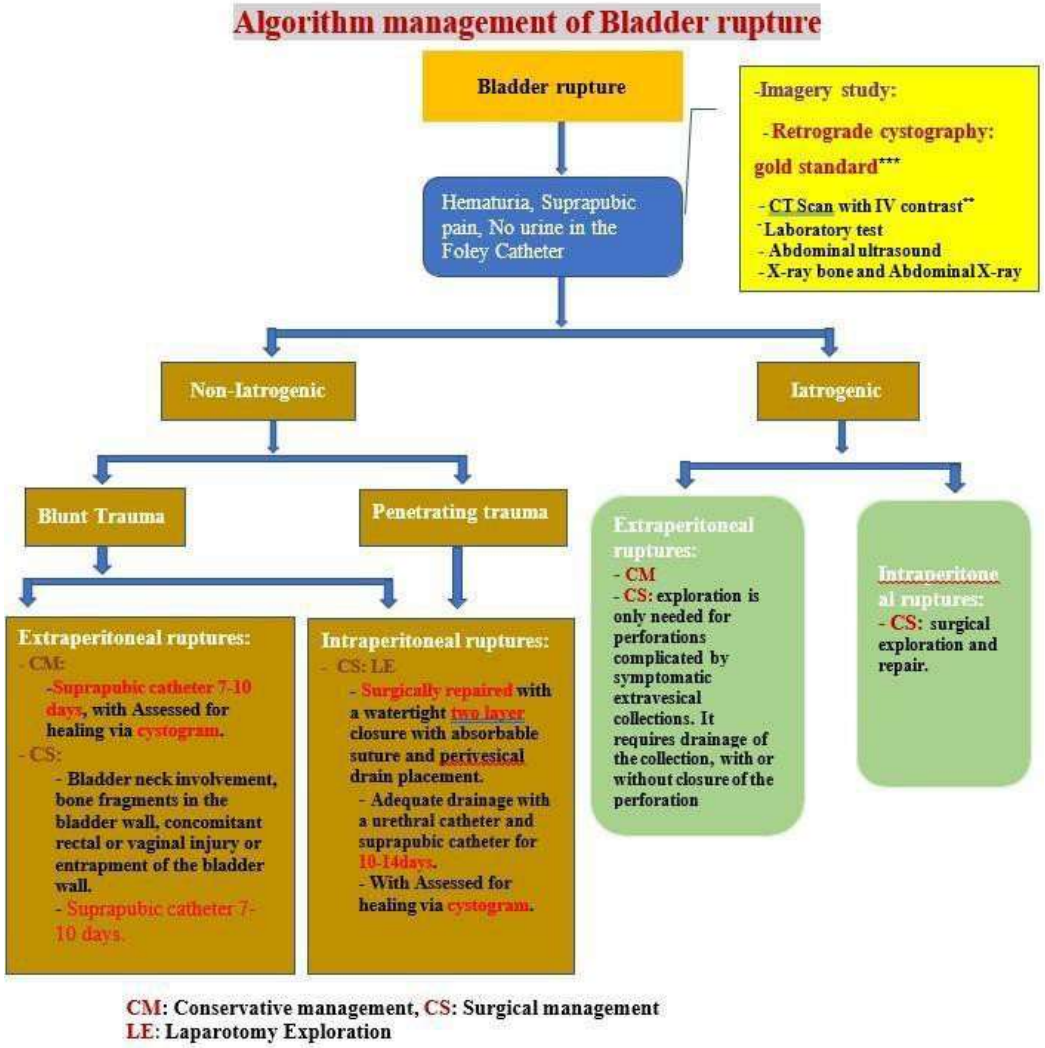
+Uro-hematoma Infection:

-Pelvic cellulitis

-Osteitis,

-Sepsis ...

VII. ALGORITHM



CM: Conservative management, CS: Surgical management
LE: Laparotomy Exploration

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BENIGN PROSTATIC HYPERPLASIA (BPH)

Dr. SIV BUNHENG, Dr. OENG MENGLEANG

I. CASE DEFINITION

Benign prostatic hyperplasia (BPH), also known as benign prostatic hypertrophy, is a histologic diagnosis characterized by proliferation of the cellular elements of the prostate, leading to an enlarged prostate gland.

BPH may lead to urinary retention, impaired kidney function, recurrent urinary tract infections, gross hematuria, and bladder calculi.

II. ETIOLOGY

The causes of BPH are not clear. It mainly occurs in older men. Hormone changes are thought to play a role. Another theory is about the role of dihydrotestosterone (DHT.) This male hormone supports prostate development.

III. DIAGNOSTIC PROCEDURE

3.1. Clinical argument

3.1.1. Predisposing conditions include:

- Men over the age of 50 as the risk for BPH rises with age
- Men whose fathers had BPH
- Men who are overweight or obese
- Men who don't stay active

3.1.2. Signs and symptoms:

a. Storage Symptoms

- Frequency
- Urgency
- Nocturia
- Urge incontinence

b. Voiding Symptoms

- Hesitancy
- Poor flow
- Intermittent flow
- Post-micturition dribble
- Incomplete emptying

3.1.3. Physical Examination

a. abdominal examination (looking for a palpable bladder/loin pain)

b. external genital (meatal stenosis or phimosis)

c. digital rectal examination making a note in particular of the size, shape (how many lobes) and consistency (smooth/hard/nodular) of the prostate (BPH is characterized by a smooth enlarged prostate).

d. Questionnaires (IPSS)

3.2. Technical procedure

3.2.1. Baseline lab:

a. Blood Tests

- including Prostate-specific antigen (PSA), renal function tests, are useful to establish baseline renal function and can help support the diagnosis of renal failure/acute kidney injury in someone with chronic high-pressure retention or acute retention, for example.

b. Urinalysis

- Urine specimen testing can help detect infection, non-visible hematuria, or metabolic disorders (glycosuria). Leucocytes and nitrites are common findings with infection; the presence of proteinuria may point towards nephrological conditions.

3.2.2. Imaging study

- Ultrasound
- Post-void residual volume (PVR)
- Uroflowmetry
- Cystoscopy

IV. DIFFERENTIAL DIAGNOSIS

- Bladder Cancer
- Bladder Stones
- Bladder Trauma
- Interstitial Cystitis
- Neurogenic Bladder
- Prostatitis
- Radiation Cystitis
- Urethral Strictures in Males
- Urinary Tract Infection (UTI) in Males

V. THERAPEUTIC APPROACH

a. Observation

- Weight loss, reducing caffeine intake or reducing fluid intake in the evening, and avoiding constipation to try and reduce risk factors and improve LUTS
- These measures may be trialed in those with mild symptoms (IPSS<7)

b. Medical Therapy

- Alpha 1-adrenoreceptors
- 5 alpha-reductase inhibitors

c. Surgery

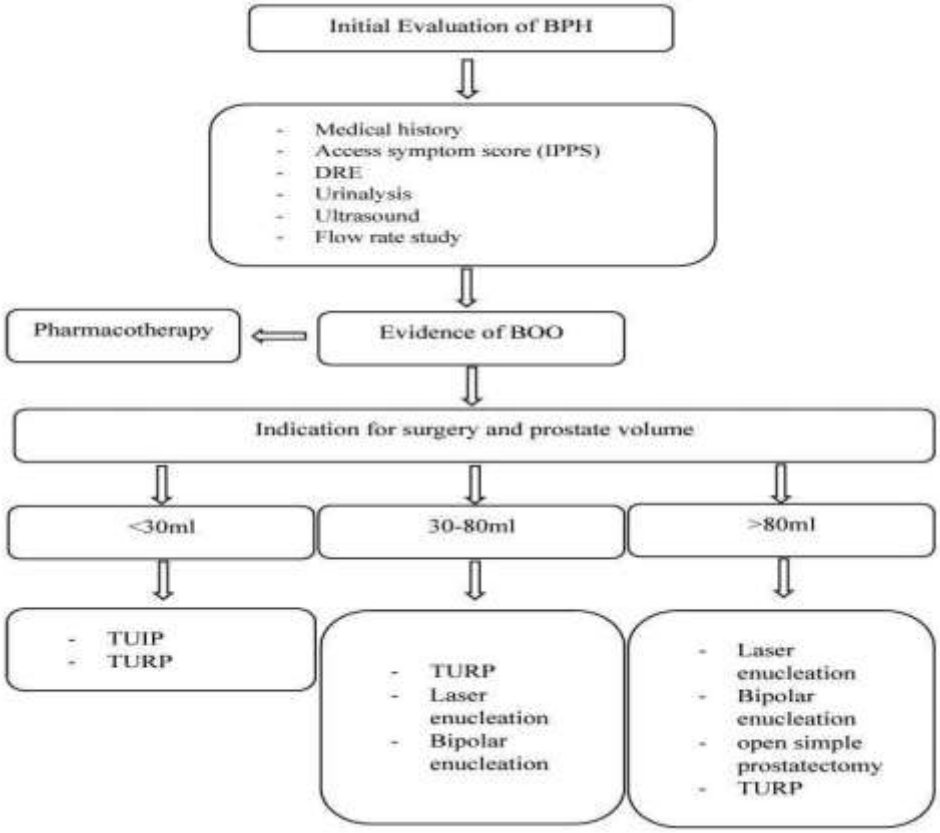
- Indications for surgery in BPH
  - Refractory urinary retention
  - Recurrent urinary infections
  - Hematuria refractory to medical treatment (other causes excluded)
  - Renal insufficiency
  - Bladder stones
  - Increased post-void residual
  - High-pressure chronic retention (absolute indication)

- Surgical management options are outlined below
  - TUIP
  - TURP
  - TUEP
  - Holep
  - Open

VI. COMPLICATION

- Urinary retention
- Chronic retention
- Urinary tract infection (due to incomplete emptying)
- Hematuria
- Bladder calculi

VII. ALGORITHM



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ERECTILE DYSFUNCTION

DR. OUK REAKSMEY, PROF.BOU SOPHEAP

I. CASE DEFINITION

Erectile dysfunction (ED), formerly termed impotence, is defined as the failure to achieve or maintain a rigid penile erection suitable for satisfactory sexual intercourse.[1] While no specific time is part of this definition, some have suggested that the condition needs to persist for six months. ED is a common condition in men who are 40 years and older; prevalence increases with age and other co-morbidities

II. ETIOLOGY

A variety of physical and psychological or emotional issues can cause ED. Physical causes include damage to the nerves, arteries, smooth muscles, and fibrous tissues in the penis. Diseases and disorders that cause damage and can lead to ED include:

- high blood pressure
- diabetes, a complex group of diseases characterized by high blood glucose, also called high blood sugar or hyperglycemia
- atherosclerosis, the buildup of a substance called plaque on the inside of arteries
- heart and blood vessel disease
- chronic kidney disease
- multiple sclerosis, an autoimmune disease that attacks the nerves
- injury from treatments for prostate cancer, including radiation and prostate surgery
- injury to the penis, spinal cord, prostate, bladder, or pelvis
- surgery for bladder cancer
- Peyronie’s disease, a disorder in which scar tissue, called a plaque, forms in the penis

Lifestyle choices, such as smoking, drinking too much alcohol, using illegal drugs, being overweight, and not exercising, can lead to ED. Psychological or emotional issues, such as the following, can also contribute to ED:

- anxiety
- depression
- fear of sexual failure
- guilt
- low self-esteem
- stress

III. DIAGNOSTIC PROCEDURE

Many clinicians often find it challenging and uncomfortable to initiate discussions about their patient's sexual health, a sentiment that is rooted in cultural norms and the potential for embarrassment.

The following phraseology is very acceptable when vocalized to patients in a way that indicates, by intonation, that the questioner is expecting that everything will be fine and normal. If you ask, "How is your sex life? Everything working OK for

you?" men without any sexual problems or issues are likely to respond with a quick "everything is fine" response. If the patient hesitates with their response or indicates that things are "not like they used to be," this should suggest that there is a potential sexual disorder that warrants further inquiries and investigation.

Blood Testing

There are no specific tests required for the initial evaluation of ED. However, many clinicians will order routine blood testing to include a complete blood count (CBC) and electrolytes, baseline renal and liver function tests, HgbA1c to screen for diabetes mellitus, and a lipid profile. Checking a morning testosterone level is recommended by the 2018 AUA Guidelines on Erectile Dysfunction. However, some experts feel it is not necessary unless there are other symptoms suggestive of hypogonadism, such as loss of sexual desire or testicular atrophy on physical examination. If not measured initially, check morning testosterone levels to rule out hypogonadism if patients fail oral PDE-5 ED therapy. Other blood tests that may be requested include LH and prolactin (if hypogonadism is present) and sickle cell testing in patients of African/Caribbean descent. Measure thyroid function (TSH) and prostate-specific antigen in appropriate patients (optionally). We refer patients with abnormal laboratory test results to their primary healthcare providers for further evaluation and treatment.

A standard laboratory workup might include CBC, a comprehensive metabolic panel (CMP, which includes liver and renal function), a lipid profile, TSH level, HgbA1c, and a morning testosterone assay on all new patients presenting for evaluation of their ED.

-Shared Decision Making
-Further Testing (Optional)
-Penile biothesiometry
-Nocturnal tumescence testing (NPT)
-Penile duplex Doppler ultrasound
-Use dynamic infusion cavernosometry and cavernosography
-Pudendal arteriography
-Endothelial cell dysfunction

IV. DIFFERENTIAL DIAGNOSIS

The primary differential diagnoses for ED would be hypogonadism, loss of libido, depression with low mood, and other psychological conditions. This condition may be the first manifestation of diabetes or cardiovascular disease, as well as depression. Differentiating between true erectile dysfunction and other sexual disorders, such as premature ejaculation, is essential and usually easily accomplished by obtaining a good sexual history of the patient.

V. THERAPEUTIC APPROACH

Initial treatment involves improving general health status through **lifestyle modifications**. This treatment not only improves erectile function but reduces cardiovascular risk. Recommended lifestyle modifications would include the following:

- Increased physical activity
- Switching to a Mediterranean diet or nutritional counseling
- Stopping smoking, drugs, and alcohol

- Gaining reasonable control of diabetes, lipids, and cholesterol

-L-arginine
-Eroxon
-Oral phosphodiesterase-5 inhibitors (PDE-5 inhibitors)
-Testosterone supplementation
-External vacuum devices
-Intraurethral prostaglandin E1 (alprostadil)
-Intracavernosal Injections
-Combined therapy with intracavernosal injections (or intraurethral prostaglandin pellets) plus the addition of a PDE-5 inhibitor
-Penile prostheses
-Penile revascularization surgery
-Arterial balloon angioplasty
-Venous ligation surgery
-Low-intensity shockwave therapy

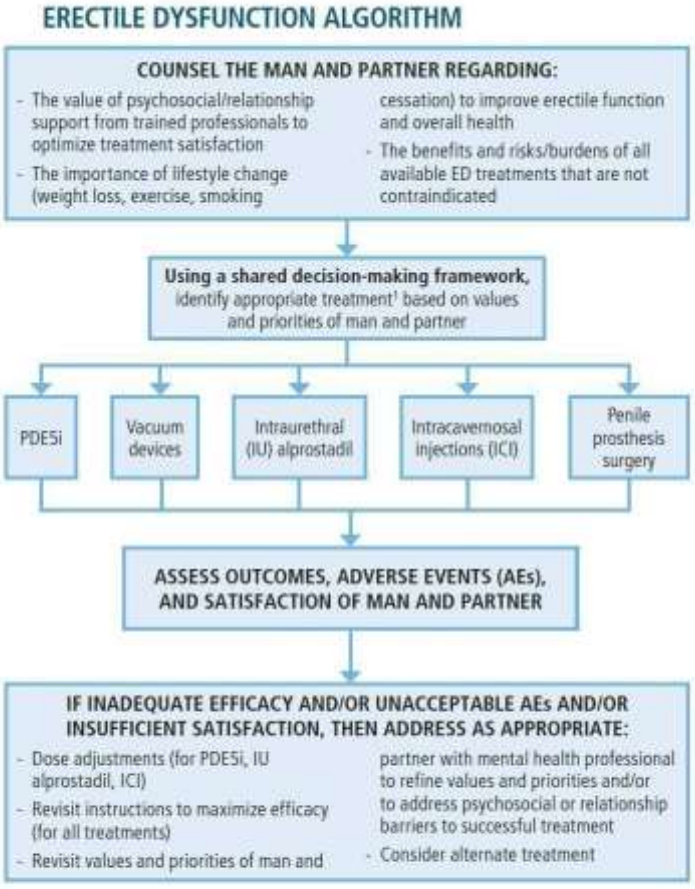
VI. COMPLICATION

Complications of ED can cause a strain on relationships and negatively impact the quality of life of these patients. The cardiovascular pathologies and diabetic complications that may accompany this condition are correlated with other health issues as well.

Priapism from PDE-5 inhibitor medications is relatively uncommon at only about 3% of all priapism cases despite the widespread use of these drugs. Penile injection therapy is involved in about 8.8% of priapism cases and trazodone in about 6%, while second-generation antipsychotic drugs are responsible for 33.8%.

Treatment for drug-induced priapism is intermittent intracavernosal injections of diluted phenylephrine solution, 200 μg at a time, about 5 to 10 minutes apart until detumescence or when a maximum dose of 1 mg of phenylephrine is delivered. If this fails, a surgical shunting procedure will be necessary. Treatment should begin quickly, as permanent corporal fibrosis can occur with delayed therapy.

VII. ALGORITHM



[1]For men with testosterone deficiency, defined as the presence of symptoms and signs and a total testosterone <300 ng/dl, counseling should emphasize that restoration of testosterone levels to theraputic levels is likely to increase efficacy of ED treatments other than prosthesis surgery.

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FOURNIER GANGRENE

HAY VANEL, .OUK READSMEY, BOU SOPHEAP

I. DEFINITION

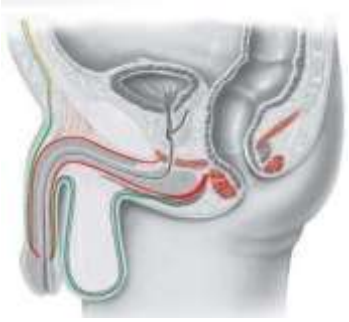
Fournier gangrene is a type of necrotizing fasciitis or gangrene affecting the external genitalia or perineum.

II. EPIDEMIOLOGY

- 5^{th} – 6^{th} decades of life
- Male >> Female (10:1)
- Incidence: 1/7500
- Mortality 3-45%

ANATOMY

- The five fascial plans that can be affected are: Colles' fascia, dartose fascia, Buck's fascia, Scarpa's fascia, and camper's fascia.



- Urogenital causes of Fournier's gangrene lead to initial involvement of the anterior triangle, whereas anorectal causes primarily involve the posterior triangle
- Blood supply to the testis, bladder and rectum originated directly from the aorta and not from the perineal vasculature and for this reason they are rarely affected in Fournier's gangrene.

III. PATHOGENESIS

- The pathogenesis of Fournier's gangrene is characterized by polymicrobial infection with subsequent vascular thrombosis and tissue necrosis, aggravated by poor host defense due to one or more underline systemic disorders.
- Aerobic organisms cause intravascular coagulation by inducing platelet aggregation and complement fixation, while anaerobes produce heparinase.
- Hypoxic tissue leads to the formation of oxygen free radicals. This led to cell membrane disruption, decreases ATP production, and DNA damage, which leads to decreased protein production

- Anaerobic organisms secret various enzyme and toxins. Lecithinase, collagenase, and hyaluronidase cause digestion of the fascia planes. They produce insoluble hydrogen and nitrogen. Leading to the formation of gas in the subcutaneous tissues, clinically palpable as crepitus.
- Endotoxins are released from the cell walls of gram-negative bacteria
- Macrophage activation and subsequent complement activation ensues with release of pro- inflammation cytokines and eventual development of septic shock.

IV. ETIOLOGY

1. Ano-rectal causes:
    - Infection in the perineal glands
    - Colorectal injury, malignancy or diverticulitis
2. Uro-genital causes:
    - Infection in the bulbo-urethral glands
    - Urethral injury
    - Iatrogenic injury
    - Lower urinary tract infection
3. Dermatologic causes:
    - Ulcerative form scrotal pressure
    - Trauma to scrotum or perineum
4. Other less common causes:
    - Consequence of bone marrow malignancy
    - Systemic lupus erythematous
    - Crohn's disease

RISK FACTORS
Diabetes mellitus
- Chronic alcoholism
- Malnutrition
- Obesity
- Liver cirrhosis
- Poor personal hygiene
- Immunosuppressor
- Chemotherapy for malignancy…etc

CAUSATIVE BACTERIA
Polymicrobial infection and synergistic bacteria
- Most common aerobe - E-Coli

- Most common anaerobes – Bacteroids
- Others- Streptococcus, staphylococcus aureus, klebsiella pseudomonas, proteus and clostridium.

V. CLINICAL FEATURE

- Begins with insidious onset of pruritus and discomfort of external genitalia.
- Prodromal symptoms of fever and lethargy, which may be present for 2-7 days before gangrene.
- The hallmark of Fournier gangrenous is out of proportion pain and tenderness in the genitalia.
- Increase genital pain and tenderness with progressive erythema of the overlying skin.
- Dusky appearance of the overlying skin, subcutaneous crepitation, feculent odor.
- Obvious gangrene of a portion of the genitalia, purulent discharge from wounds.
- As gangrene develops, pain subside (nerve necrosis).

VI. INVESTIGATIONS

1. Laboratory
    - Complete blood count (CBC): infection process, red blood cell mass and potential for sepsis-induced thrombocytopenia.
    - Electrolytes
    - BUN/Serum creatinine
    - Blood sugar
    - Arterial blood gas
    - Blood and urine with culture
    - Coagulation profile: sepsis induced coagulopathy
    - Screen HIV, hepatitis or Syphilis
2. Imaging
    - Radiology:
        a) Consider where clinical findings are inconclusive
        b) Presence of gas in soft tissue
    - Ultrasonography
        a) Can be used to detect fluid or gas in soft tissue
        b) Sonography hallmark: present of gas in scrotal tissue
        c) Exclude other conditions
        d) Testicular blood flow
        e) Limitations: Direct pressure on involved tissue cause inconvenience
    - CT Scanning
        a) Can detect smaller amount of soft tissue gas
        b) Defines extent more specifically

- MRI c) Identified underlying cause eg. Small perineal abscess.

a) Yield greater soft tissue detail than does CT scanning. However, it requires greater time
b) Limit the practical usefulness of MRI, especially in patient with critical illness.

VII. DIFFERENTIAL DIAGNOSES

- Acute Epididymitis
- Cellulitis
- Necrotizing fasciitis
- Complicated hydrocele

VIII. TREATMENT

- Medical
  - i. Aggressive resuscitation: systemic toxic manifestation as hypoperfusion or organs failure.
  - ii. Antibiotics with broad-spectrum antibiotics (IV):
    - Empiric regimen might consist of ciprofloxacin or ceftriaxone and **clindamycin** (Clindamycin is useful to treat gram positive and anaerobic spectrum and streptococcal infection).
    - Other possible choices include ampicillin/sulbactam, piperacillin/tazobactam in combination with an +/- **aminoglycoside** and **metronidazole** or clindamycin.
    - **Vancomycin** is used to cover methicillin-resistant staphylococcus aureus (MRSA).
    - **NB:** Refinement of antibiotic regimen according to culture results.
  - iii. In case of sepsis syndrome: IV immunoglobulins to neutralize super antigen as streptotoxine A& B
  - iv. Tetanus prophylaxis +/-: indicated if soft-tissue injury is present
  - v. Antifungal – if tissue stain (potassium hydroxide [KOH] stain) show fungi, as an empiric agent such as amphotericin B or caspofungin.
  - **vi. <u>Treat underline diseases</u>**
- Surgical
  - i. Early emergent aggressive surgical excision:
    - All necrotic tissue must be excised.
    - The skin should be open widely to expose the full extent of the underlying fascia and subcutaneous tissue necrosis.
    - All fascia planes that separate easily: blunt dissection should be considered.
    - Send sample of excision for aerobic and anaerobic culture and a histological assessment: Point of maximal tenderness and include skin and superficial and deep fascia.
    - Repeated aggressive debridement under anesthesia at OT.
    - Vacuum assisted dressing
  - ii. Fecal diversion
    - Anal sphincter involvement

- Colonic or rectal perforation
- Decrease wound contamination
- Facilitate nursing care

iii. Urinary diversion
- Urethral catheter
- Suprapubic catheter

iv. Orchidectomy +/-

- Hyperbaric oxygen therapy

i. Used as an adjuvant to surgical and antimicrobial therapy. HBO is used reduce systemic toxicity, prevent extension of necrotizing infection and inhibit growth of anaerobic bacteria.

ii. Indication:
- Failure of conventional treatment
- Documented clostridial involvement
- Myonecrosis
- Deep tissue involvement

- Reconstruction

i. Primary closure of the skin, if possible
ii. Local skin flap coverage
iii. Split-thickness skin grafts
iv. Muscular flaps, which are used to fil a cavity.

IX. COMPLICATION

i. Systemic complications

- Unresolve sepsis
- Acute respiratory distress syndrome
- Heart failure
- Cardiac arrythmias

ii. Surgical complications

- Wound infection
- Stoma-related complications
- Prolong ileus (7days)
- Eventration or evisceration

iii. Long term complications

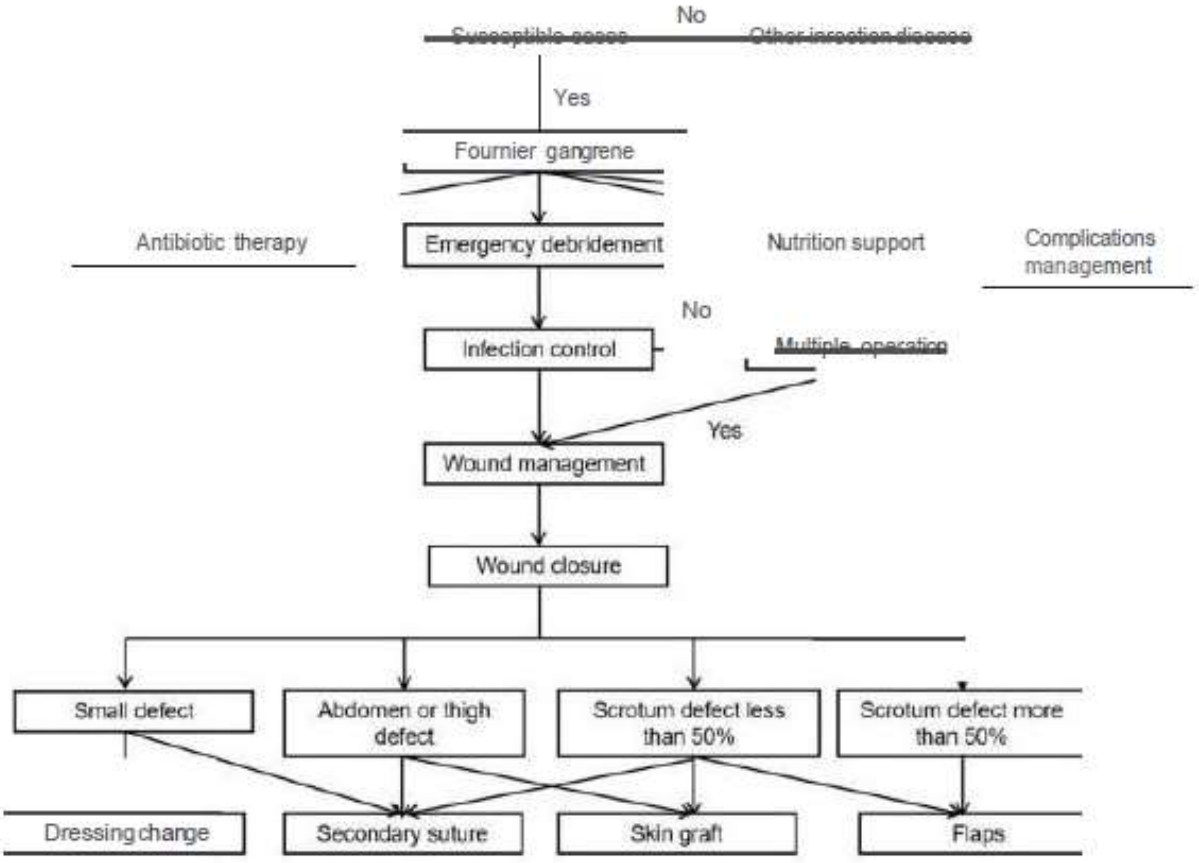
- Pain (50% of patients)
- Impaired sexual function (due to penile deviation/torsion, loss of sensitive of the penile skin or pain during erection)
- Stool incontinence
- Extensive scaring

X. PROGNOSIS

- High risk of morbidity and mortality
- Despite aggressive therapy, the mortality rate nearly 50% because of the aggressive nature of the infection and the presence of underlying comorbidities.
- Delay in diagnosis or treatment increase the mortality rate.
  - A 24h delay in radical debridement increases the mortality rate by 11.5%
  - A 6- day delay is associated with a mortality rate 76%

- Additional factors associated with high mortality include:
  - Anorectal origin
  - Advanced age
  - Shock
  - Sepsis at presentation
  - Renal failure
  - Hepatic dysfunction
- Multiorgan systemic failure secondary to gram-negative sepsis is the most common cause of death.

XI. ALGORITHLVI:



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HEMATURIA

Dr. SRUN SOK AUN, Dr. MAN LIBERTINE

I. DEFINITION

Hematuria is characterized by the presence of blood in the urine during urination. It can be microscopic (visible on the urine strip) or macroscopic (visible to the eye).

II. ETIOLOGY

In the presence of hematuria, we should class the causes of urology and nephrology. The urological causes are dominated by urological cancers, benign prostatic hyperplasia (BPH), urinary tract infection and urinary lithiasis. The nephrological causes are mainly represented by glomerular nephropathy and autosomal dominant polycystic kidney disease.

i. Urological causes

- Cancer (urothelial carcinoma, renal cancer, prostate cancer)
- BPH
- Urinary lithiasis
- Urinary tract infection (bacteria, bilharzia)
- Urinary tract trauma
- Medication-induced hemorrhagic cystitis (endoxan)
-Radiation cystitis
- Renal arteriovenous fistula
- Bladder endometriosis

ii. Nephrological causes

- Non-proliferative glomerulopathy (Berger's disease, Alport syndrome)
- Membrano-proliferative glomerulopathy
- Polycystic kidney disease

III. DIAGNOSTIC APPROACH

i-Clinical arguments

Clinical examination:

- Positive diagnosis (macroscopic hematuria under the eye), search for **critical condition**.
- Positive diagnosis (microscopic hematuria by dipstick)
- Etiological assessment with the chronology of hematuria and search for cancer risk factors
    - Rectal examination…

Critical condition:

- Hemorrhagic shock (hypotension, tachycardia, consciousness disorder)
- Anemia
- Blood clot in the urinary tract (renal colic, urinary retention)
- Timeline of hematuria during urination (3 Glasses of hematuria):
- Initial hematuria : sub vesical cause
- Terminal hematuria : vesical cause

- Total hematuria : not specific

Technical procedure **a.Laboratories tests**

- Blood count, electrolyte, creatininemia
- Urinalysis, Urine culture
- -Red blood cell-leukocyte-minute
- 24h proteinuria

b. Imaging studies

- Ultrasound
- KUB X-ray
- Uro-TDM/IVU
- Cystoscopy +/- biopsy
- Renal biopsy : to be discussed after eliminating urological causes.

IV. DIFFERENTIAL DIAGNOSTIC

i. Red coloring of urine:

- Medications : rifampicin, metronidazole, vit B12, erythromycin
- Blood pigments : myoglobinuria, hemoglobinuria
- Bile pigments
- Heavy metals : lead, mercury
- Food : beetroot, red dragon fruit…

ii. Contamination of urine with blood:

- Urethrorrhagia
- Metrorrhagia
- Hemospermia

V. THERAPEUTIC APPROACH

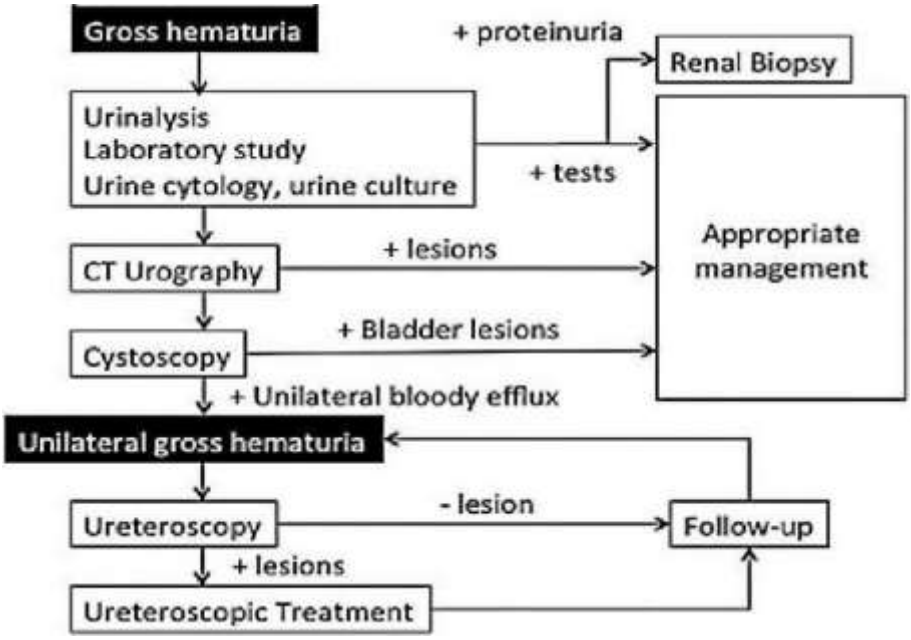
i. Stabilize patient

-Resuscitation the patient

-If urinary clot retention: Evacuation blood clot by 3 ways Urinary Foley catheter /Endoscopic procedure +/- CBI(Continuous bladder irrigation) (annex 1)

ii. Treatment depending on their condition and causes

VI. ALGORITHM



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Annexe : Evacuation blood clot by Urinary catheter /Endoscopic procedure +/- CBI(Continuous bladder irrigation)

-Imaging Studies

HYDROCELE

Dr. HAY VANEL, Dr. OUK REAKSMEY, Prof. BOU SOPHEAP

I. CASE DEFINITION

A hydrocele is an abnormal collection of serous fluid between the two layers of tunica vaginalis of testis. It can either be congenital or acquired.
Congenital hydrocele results from failure of processus vaginalis to obliterate. During development, the testes are formed retroperitoneally in the abdomen and proceed to descend into the scrotum via the inguinal canal in the third gestational week. This descent of the testes into the scrotum is accompanied by a fold of peritoneum of the processus vaginalis. Normally, the proximal portion of processus vaginalis gets obliterated while the distal portion persists as the tunica vaginalis covering the anterior, lateral, and medial aspects of the testes. The tunica vaginalis is a potential space for fluid to accumulate, provided the proximal portion of processus vaginalis remains patent and results in free communication with the peritoneal cavity, leading to congenital hydrocele

II. ETIOLOGY

There are four basic mechanisms by which hydrocele can develop. These are mentioned below:
1. Connection with the peritoneal cavity through a patent processes vaginalis (congenital).
2. Excessive production of fluid (secondary hydrocele).
3. Defective absorption of fluid.
4. Interference with the lymphatic drainage of scrotal structures as in filarial hydroceles. In children, patency of processus vaginalis, allowing peritoneal fluid to flow into the scrotum, is the main cause of hydrocele. However, in adults, filariasis caused by *Wuchereria* bancrofti is the main culprit globally, affecting 120 million people in more than 73 countries. This is not true in the United States, where iatrogenic causes (either trauma or post-herniorrhaphy complications) predominate.

III. DIAGNOSTIC PROCEDURE

Hydroceles can be diagnosed on clinical grounds, as discussed in the history and physical section. However, in the presence of any concomitant medical condition or to exclude other medical or surgical conditions, further studies, including laboratory or imaging, should be considered.

-Laboratory Studies: These are indicated to exclude other surgical or medical conditions that may be in the differential diagnosis.

-Inguinal Hernia: Laboratory tests are usually not indicated, but in the case of an incarcerated inguinal hernia, which can mimic hydrocele, leukocytosis can aid in the differentiation. Negative transillumination and palpable bowel at the deep ring on the digital examination is more consistent with an inguinal hernia.

-Testicular Tumor: Serum alpha-fetoprotein and human chorionic gonadotropin (hCG) levels are indicated if there is suspicion of malignant teratomas or other germ cell tumors.

-Epididymitis/Orchitis: These conditions can lead to secondary or reactive hydroceles. In such cases, urinalysis and urine culture may be useful.

These are helpful in diagnosing and evaluating hydrocele. They can also assess for underlying processes such as epididymitis, testicular torsion, or testicular tumor.

-Ultrasonography: Scrotal pain or failure to delineate the testicular anatomy on palpation is an indication for ultrasonography as it provides excellent detail of testicular parenchyma. During the ultrasonography examination, hydrocele appears as an anechoic or echolucent area surrounding the testis. Ultrasonography could also help with the sizing and characterization of the hydrocele. Spermatoceles, testicular tumors, and testicular atrophy can be easily distinguished via ultrasonography. The patient should be examined in both supine and upright positions as hydrocele has a tendency to reduce into the abdomen based on the position of the patient.

-Duplex Ultrasonography: It provides information regarding testicular blood flow, which will be reduced or absent in hydroceles resulting from testicular torsions. However, in the case of hydroceles secondary to epididymitis, the epididymal flow would be increased. In addition, duplex studies help identify the Valsalva augmented regurgitant flow in varicoceles.

-Plain Abdominal Radiography: In an incarcerated inguinal hernia, one may see gas overlying the groin.

IV. DIFFERENTIAL DIAGNOSIS

- Inguinal hernia
- Epididymal cyst
- Spermatocele
- Testicular tumor
- Scrotal edema
- Varicocele

V. THERAPEUTIC APPROACH

Surgery is the treatment of choice for hydrocele, and it is warranted when hydrocele becomes complicated or symptomatic. For congenital hydroceles, herniotomy is performed, provided they do not resolve spontaneously. On the other hand, acquired hydroceles subside when the primary underlying condition resolves. There are two common surgical approaches available for hydrocelectomy:

1. **Plication:** This technique is suitable for thin-walled hydroceles. As there is minimal dissection, the risk of hematocele or infection is significantly reduced. Lord plication involves the tunica being bunched into a ruff by applying a series of multiple interrupted chromic catgut sutures for the sac to form fibrous tissue.

2. **Excision and Eversion:** This technique is suitable for large thick-walled hydroceles and chyloceles. It involves subtotal excision of the tunica vaginalis and everting the sac behind the testes followed by placing the testes in a newly created pocket between the fascial layers of the scrotum (Jaboulay procedure). Particular consideration is taken not to damage epididymis, testicular vessels, or ductus deferens.

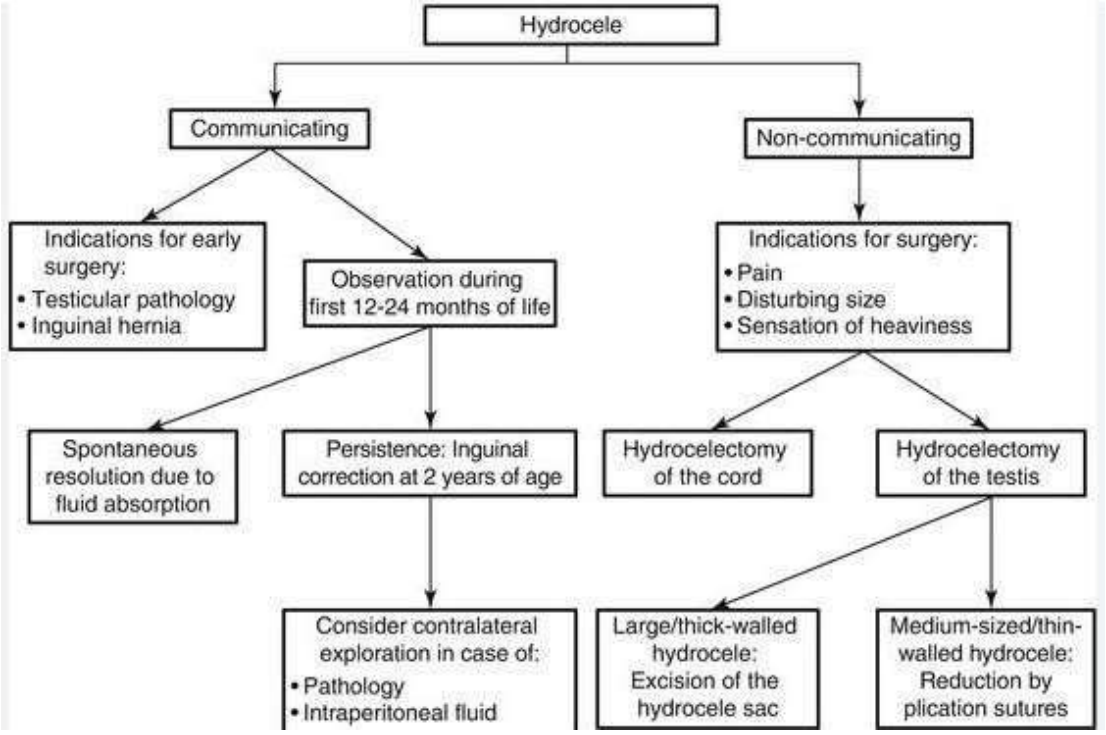
Aspiration

This is another method to treat hydrocele, particularly in patients who cannot tolerate surgery. However, hydrocele fluid almost always reaccumulates within a week or so. In addition, the risk of hematocele and infection after aspiration is high. Aspiration followed by an injection of a sclerosant (tetracycline or doxycycline) has been proven to be effective but painful.

VI. COMPLICATION

- Infection
- Pyocele
- Haematocele
- Atrophy of testes
- Infertility (resulting from the spermatogenesis halt due to increased pressure on the blood supply on the testis from edema)[11]
- Rupture[12][13]
- Hernia of hydrocele (rare)

VII. ALGORITHM



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HYPOSPADIA

Dr. OUK REAKSMEY, Dr. HAY VANEL, Dr. PEN MONYRATH, Prof. BOU SOPHEAP

I. CASE DEFINITION

Hypospadias is an abnormality of anterior urethral and penile development. The urethral opening is ectopically located on the ventral aspect of the penis proximal to the tip of the glans penis, which, in this condition, may be splayed open.

II. ETIOLOGY

Several etiologies for hypospadias have been suggested, including genetic, endocrine, and environmental factors.

Genetic factors

A genetic predisposition has been suggested by a fourfold increase in the incidence of hypospadias among monozygotic twins as compared with singletons. This finding may relate to the demand of two fetuses for human chorionic gonadotropin (HCG) produced by a single placenta, with an inadequate supply during critical periods of urethral development.

A familial trend has been noted with hypospadias. The prevalence of hypospadias in male children of fathers with hypospadias has been reported as 8%, and 14% of brothers of children with hypospadias are also affected. The inheritance is likely polygenic.

Endocrine factors

A decrease in available androgen or an inability to use available androgen appropriately may result in hypospadias. In a 1997 report by Aaronson et al, 66% of boys with mild hypospadias and 40% of those with severe hypospadias were found to have a defect in testicular testosterone biosynthesis.

Mutations in the 5-alpha reductase enzyme, which converts testosterone (T) to the more potent dihydrotestosterone (DHT), have been associated with hypospadias. A 1999 report by Silver et al found that nearly 10% of boys with isolated hypospadias had at least one affected allele with a 5-alpha reductase mutation.

Environmental factors

Endocrine disruption by environmental agents is gaining popularity as a possible etiology for hypospadias and as an explanation for its increasing incidence. Estrogen has been implicated in abnormal penile development in many animal models. Environmental substances with significant estrogenic activity are ubiquitous in industrialized society and are ingested as pesticides on fruits and vegetables, endogenous plant estrogens, in milk from lactating pregnant dairy cows, and in pharmaceuticals such as phthalates. The association of hypospadias with increasing parity, increasing maternal age, and low birth weight noted in some studies may reflect a lifelong exposure to environmental disruptors and a possible cumulative effect.

Combination theory

A growing body of evidence suggests that the development of hypospadias has a two-hit etiology involving a genetic predisposition coupled with fetal exposure to an environmental disruptor.

III. DIAGNOSTIC PROCEDURE

History

It is important to obtain a thorough history and physical examination, including any history of a familial pattern of hypospadias, any past medical history or comorbidity, and a physical assessment focusing on the meatal location, glans configuration, skin coverage, and ventral curvature (chordee).

A history of parental difficulties in conceiving and treatment should also be documented; in-vitro fertilization (IVF) has been associated with a higher incidence of hypospadias.

Physical examination

Although the appearance of hypospadias has been identified with both antenatal fetal ultrasonography (US) and magnetic resonance imaging (MRI), the diagnosis is generally made upon examination of the newborn infant.

A dorsal hood of foreskin and glanular groove are evident, but upon closer inspection, the prepuce is incomplete ventrally and the urethral meatus is noted in an abnormal proximal location. Rarely, the foreskin may be complete, and the hypospadias is revealed at the time of circumcision. If hypospadias is encountered during neonatal circumcision, after the dorsal slit has been performed, the procedure should be halted, and the patient should be referred for urologic evaluation.

Penile curvature may be readily apparent or may be discernible only during erection. Proximal hypospadias is commonly associated with a bifid scrotum and penoscrotal transposition (see the image below), in which the rugated scrotal skin begins lateral to the penis rather than in its normal posterior origin.

IV. DIFFERENTIAL DIAGNOSIS

- Differences (Disorders) of Sex Development (DSDs)
- Genital Anomalies

V. THERAPEUTIC APPROACH

The treatment for hypospadias is surgical repair. Repair is generally performed for functional and cosmetic reasons. The more proximally ectopic the position of the urethral meatus, the more likely the urinary stream is to be deflected downward, which may necessitate urination in a seated position. Any element of ventral curvature (chordee) can further deflect the urinary stream. The abnormal deflection of ejaculate may preclude effective insemination, and significant chordee can preclude vaginal insertion of the penis or can be associated with inherently painful erections.

Minor cases of hypospadias, in which the meatus is located distal to the corona on the glans, may not require surgical repair and may simply be managed with observation. It must be kept in mind, however, that although the most minor forms of hypospadias are insignificant in physiologic terms, they too may merit repair on the basis of the potential long-term psychological stress associated with having abnormal genitalia.

VI. COMPLICATION RENAL FAILURE

Immediate postoperative concerns

Local edema and blood spotting can be expected early after repair and generally do not cause a significant problem.
Postoperative bleeding rarely occurs and is usually controlled with a compressive dressing. Infrequently, reexploration may be required to evacuate a hematoma and to identify and treat the source of bleeding.
Infection is a rare complication of hypospadias repair in the modern era. Skin preparation and perioperative antibiotics are generally used. Patients are often maintained on an antibiotic course until any stents are removed, though this has not clearly been shown to be beneficial.

Long-term issues

Urethrocutaneous fistulization is a major concern in hypospadias repair. The rate of fistula formation is generally less than 10% for most single-stage repairs but rises with the severity of hypospadias, approaching 40% with complex reoperative efforts. Fistulas rarely close spontaneously and are repaired by using a multilayered closure with local skin flaps 6 months after the initial repair. After repair, fistulas recur in approximately 10% of patients.

VII. FOLLOW UP

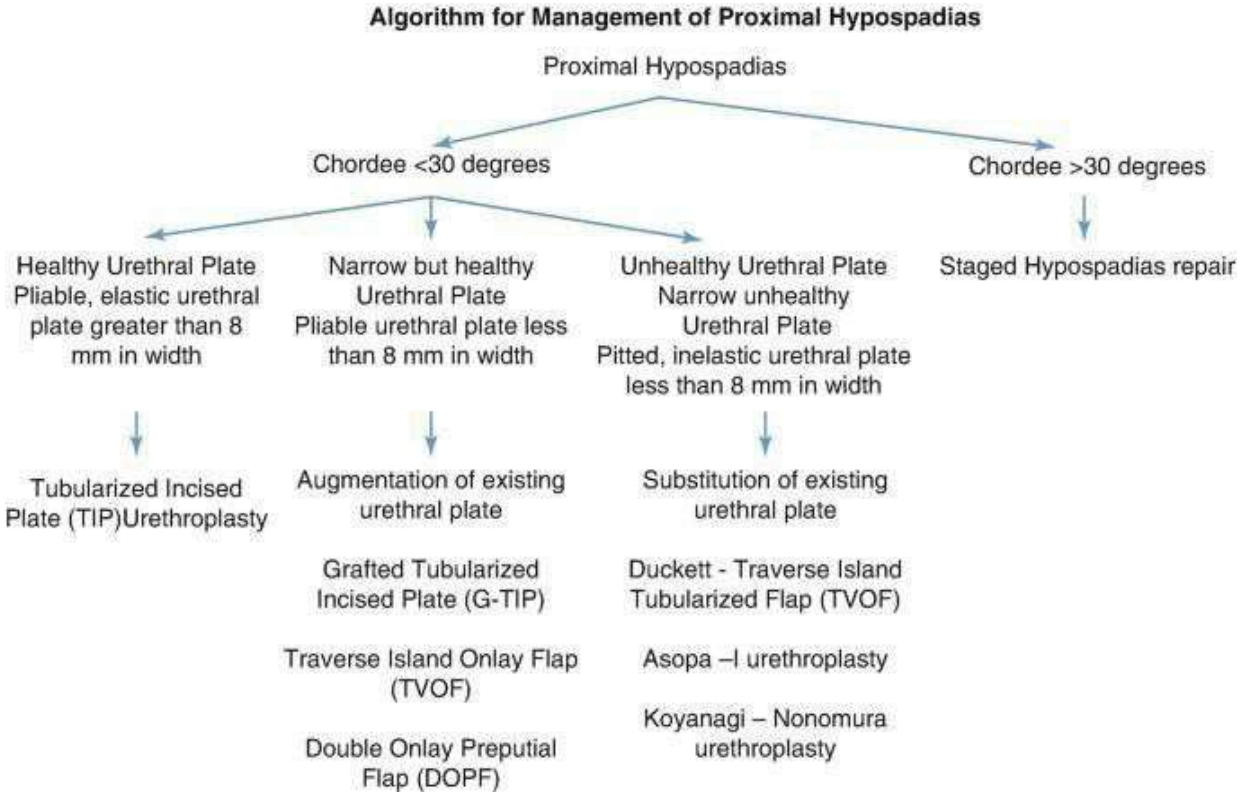
Long-term monitoring

Monitor children for linear growth (height, weight, and head circumfer- ence), renal function, BP, urine analysis (for proteinuria, osmolality), USS, and formal GFR with chromium EDTA. Renography (MAG3 and DMSA) are also performed to assess split renal function and look for evidence of obstruction or reflux. Videourodynamic studies are used to assess and aid in the management of any associated voiding dysfunction.

Prognosis

Thirty-five percent have long-term poor renal function; 20% develop end- stage renal failure. Bladder dysfunction is common despite treatment of outflow obstruction. This includes bladder overactivity, incontinence, and bladder underactivity associated with chronic urinary residuals and poor concentration of urine (with polyuria). From age 16y, care should be transferred to an adult urologist or nephrologist. Problems may arise with retrograde ejaculation, impotence and reduced libido (related to renal impairment), and abnormal prostatic or seminal vesicle secretions, con- tributing to reduced fertility.

VIII. ALGORITHM



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INGUINAL HERNIA

Dr. HAY VANEL, Dr. OUK REAKSMEY, Prof. BOU SOPHEAP

I. 1.INTRODUCTION

Worldwide, there are more than 20 million inguinal hernia repairs performed annually. About 800,000 of these are performed in the United States. These statistics make inguinal hernias one of the most common medical conditions encountered by a general surgeon. The ureter is a retroperitoneal organ, and ureteral involvement with an inguinal hernia is rare. Only about 140 cases of ureteral inguinal hernia were reported through 2009.[1] Involvement of the ureter may be undetected preoperatively, and the general surgeon needs to be keenly aware of a ureteral inguinal hernia because intra-operative iatrogenic damage to the ureter can have serious consequences.

II. ETIOLOGY

Inguinal hernias can be classified as congenital or acquired. The congenital type is related to a patent processus vaginalis, an invagination of the parietal peritoneum, which precedes testicular descent through the inguinal canal during embryogenesis. These are indirect inguinal hernias which protrude through the internal inguinal ring lateral to the epigastric vessels. They are about twice as common as direct inguinal hernias. There is a recent debate that all indirect inguinal hernias result from a processus vaginalis that had never closed. Work by Jiang and Mouravas suggests that adult indirect inguinal hernias may develop after the long-term buildup of pressure on a processus vaginalis that had closed along its entire length except at the neck of the hernia sac. The acquired type of an inguinal hernia is related to a weakening or disruption of the abdominal wall tissues due to several contributing factors, including older age, smoking, increased intraabdominal pressure such as due to a chronic cough or pregnancy, and connective tissue abnormalities. Acquired inguinal hernias are typically direct inguinal hernias where intraabdominal contents protrude through Hesselbach's triangle, medial to the inferior epigastric vessels.

III. DIAGNOSTIC PROCEDURE

3.1. Clinical argument

Though specific symptoms are possible, most patients with a ureteral inguinal hernia present no differently than the typical groin hernia. In either type of ureteral inguinal hernia, obstructive uropathy and urological symptoms may be present, regardless of the length of ureter involved. Flank pain, dysuria, hematuria, acute urinary obstruction, double-phase micturition requiring pressure to initiate or terminate voiding associated with an inguinal hernia may signal the presence of a ureteral inguinal hernia. On laboratory investigation, the clinicians may see an acute kidney injury.

3.2Technical procedure

Specific imaging studies are not routinely obtained in the preoperative workup of an inguinal hernia. However, if concerning signs of symptoms as stated above are noted, these findings should prompt preoperative imaging. An ultrasound may demonstrate hydronephrosis of the ipsilateral kidney. Intravenous pyelography may demonstrate the telltale "curlicue" sign, with the ureter seen in a pathognomonic spiral

or loop-the-loop formation. In patients with unexpected abnormal renal function, Gellett describes the potential for preoperative diagnosis by computed tomography (CT) with confirmation of a ureteral inguinal hernia on delayed, post-contrast, 3- dimensional reconstruction. CT or magnetic resonance imaging (MRI) may show the ureter entering the inguinal canal or extending beyond the bony pelvis. Nephroptosis visible on cross-sectional imaging may accompany ureteral inguinal hernia. This phenomenon is likely due to the loss of the perirenal supportive tissue into the hernia sac, rather than traction of the ureter on the kidney.

IV. DIFFERENTIAL DIAGNOSIS

- Inguinal hernia with bowel, omentum, or extraperitoneal fat
- Femoral hernia
- Hydrocele or varicocele
- Urologic malignancy
- Malignancy of the pelvic organs or retroperitoneal space

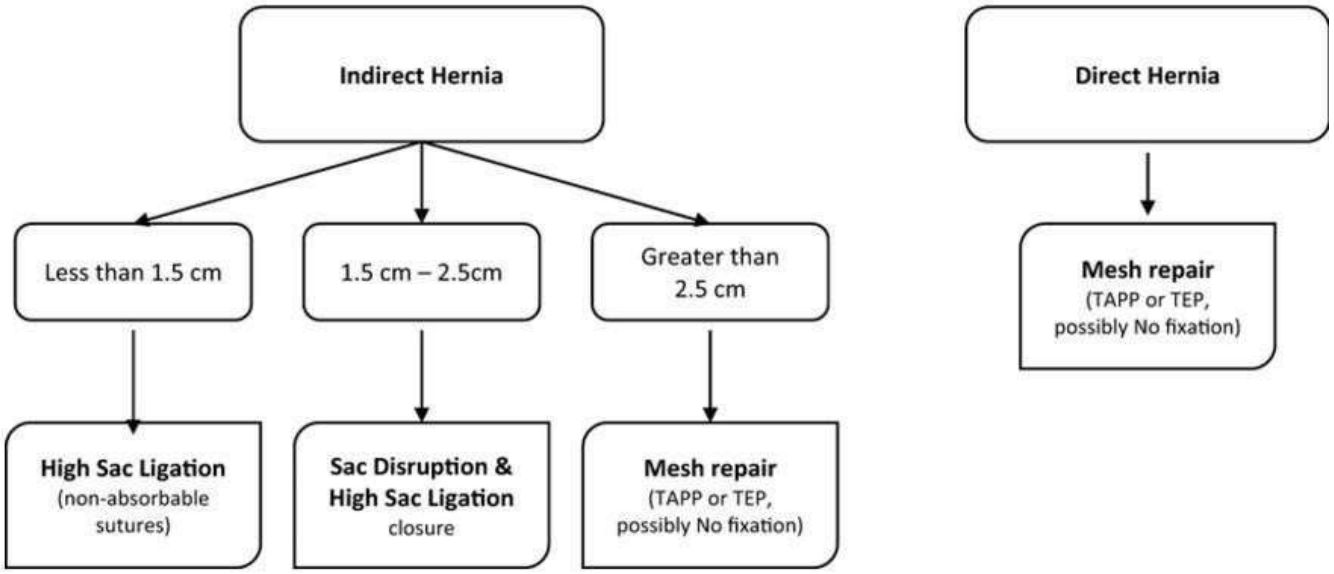
V. THERAPEUTIC APPROACH

A ureteral-inguinal hernia repair should be performed in an open manner, not laparoscopically, if diagnosed preoperatively. Repair of ureteral inguinal hernias may involve a simple reduction of the ureter with the hernia sac. Depending on the length of ureter involved, repair may require resection of the redundant ureter with primary anastomosis, ureteroneocystostomy, psoas hitch, Boari flap, or transureteroureterostomy. The surgeon should resect the necrotic or dilated areas of the ureter. In the case of a complicated repair, postoperative CT imaging should be done to ensure the patency and proper placement of the ureter back in the retroperitoneal space. Ureteral protection with a ureteral stent improves the identification of an involved ureter when it is known preoperatively. In a patient unable to withstand an operation, palliation of obstructive uropathy may also be achieved by placement of a nephrostomy tube or a nephro-ureteral stent.

VI. COMPLICATION RENAL FAILURE

Complications of an inguinal hernia include possible incarceration or strangulation of the hernia. Strangulation is a life-threatening emergency. If not repaired, hernias tend to enlarge over time.

VII. ALGORITHM



Institutional algorithm for repair of inguinal hernias in adolescents

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MANAGEMENT KIDNEY STONE

HAY VANEL, BOU SOPHEAP, OUK REAKSMEY

I. CASE DEFINITION

The presence of Urinary stone in the Kidney

II. ETIOLOGY

2.1-Low fluid intake
2.2-Hypercalciuria
2.3-Primary hyperparathyroidism 2.4-
Hypocitraturia
2.5-High animal protein intake 2.6-
Primary hyperoxaluria

III. DIAGNOSTIC PROCEDURE

3.1- Ultrasound

Ultrasound is the primary imaging technique in children. Its advantages are absence of radiation and no need for anaesthesia. Imaging should include both the fluid-filled bladder with adjoining portion of the ureters, as well as the upper ureter. Colour Doppler US shows differences in the ureteral jet [87] and resistive index of the arciform arteries of both kidneys, which are indicative of the grade of obstruction [88]. Nevertheless, US fails to identify stones in > 40% of children and provides limited information on renal function.

3.2- KUB X ray

Kidney-ureter-bladder radiography can help to identify stones and their radiopacity and facilitate follow-up.

3.3-Non-contrast-enhanced computed tomography

Recent low-dose CT protocols have been shown to significantly reduce radiation exposure. In children, only 5% of stones escape detection by NCCT. Sedation or anaesthesia is rarely needed with modern high-speed CT equipment.

3.4-Magnetic resonance urography

Magnetic resonance urography (MRU) cannot be used to detect urinary stones. However, it might provide detailed anatomical information about the urinary collecting system, the location of an obstruction or stenosis in the ureter, and renal parenchymal morphology.

3.5-Intravenous urography

The radiation dose for IVU is comparable to that for voiding cysto- urethrography (0.33 mSV) .However, the need for contrast medium injection is a major drawback.

3.6-Diagnostic investigation for recurrent stone former

Nowadays, due to our limited resources on metabolic/genetic tests, etiologic workups are encouraged (optional) and should be done with multidisciplinary team.

Table 4. Basic evaluation of a stone former

Investigaion	Rationale for investigation
Medical history and physical Examination	Stone history (Prior stone events, family history) Dietary habits Medication chart
Diagnostic imaging	Ultrasound
Blood analysis	Creatinine Calcium (ionized calcium or total calcium + albumin) Uric acid
Urinalysis	Dipstick test : Leukocytes, erythrocytes, nitrite, Protein, urine pH, specific weight Urine culture

IV. DIFFERENTIAL DIAGNOSIS

The following are some important differentials to be considered in a patient presenting with the above-mentioned features:

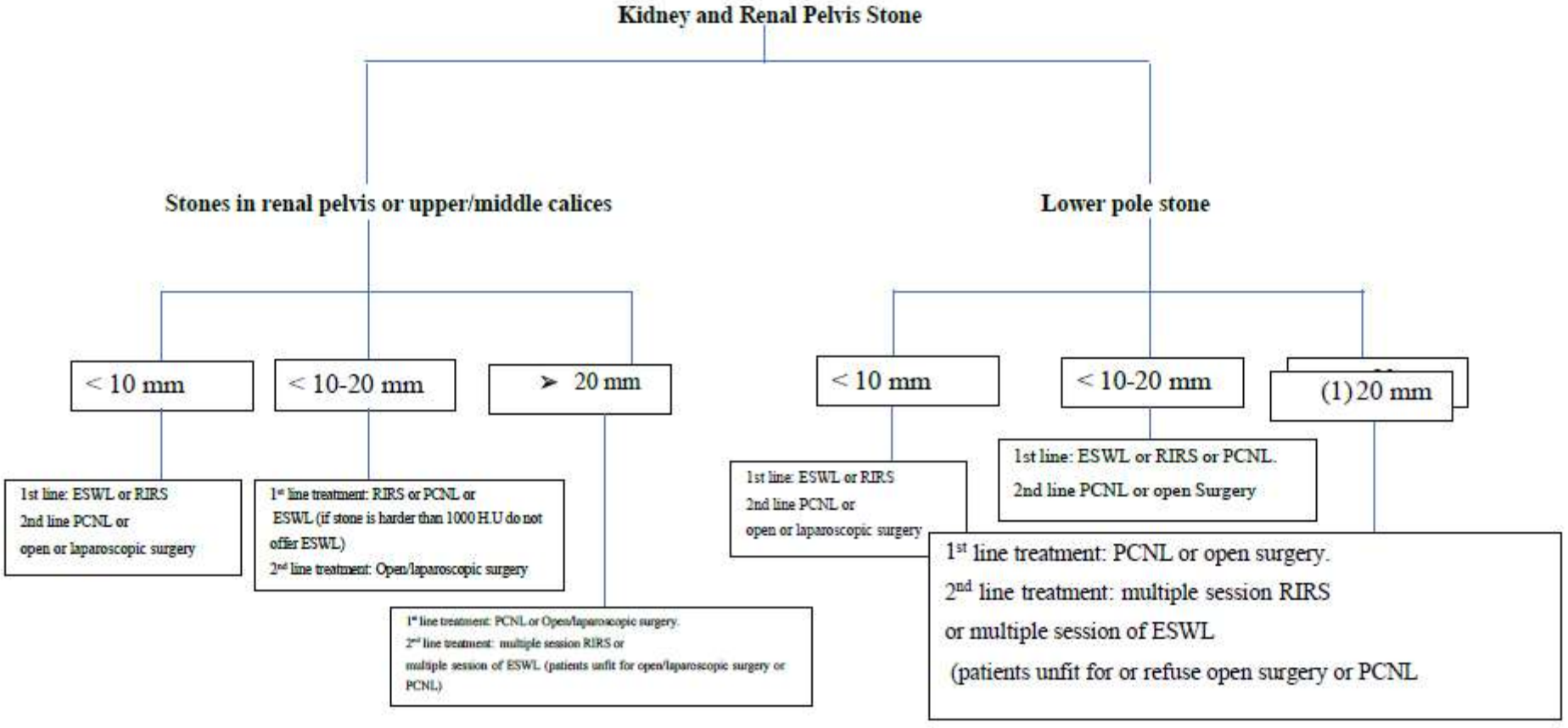
- Lower urinary tract infection
- Pyelonephritis
- Renal abscess
- Renal artery aneurysm
- Appendicitis
- Diverticulitis
- Mesenteric ischemia
- Pancreatitis

V. THERAPUETIC APPROACH

5.1-Kidney stone

Indications for the active removal of renal stones are:

- Symptomatic stones (e.g., pain or haematuria).
- Stones > 15 mm; or stones < 15 mm if observation is not the option of choice.
- Stone that give any complication as follow: Obstruction, UTI,
- Stones in high-risk patients for stone formation,
- Stone that increase in volume.
- Patient preference
- Comorbidity;
- Social situation of the patient (e.g., profession or travelling)


Kidney and Renal Pelvis Stone
Stones in renal pelvis or upper/middle calices
Lower pole stone
< 10 mm
< 10-20 mm
➢ 20 mm
< 10 mm
< 10-20 mm
(1) 20 mm
1st line: ESWL or RIRS
2nd line PCNL or
open or laparoscopic surgery
1st line: ESWL or RIRS
2nd line PCNL or
open or laparoscopic surgery
1st line: ESWL or RIRS or PCNL.
2nd line PCNL or open Surgery
1st line treatment: RIRS or PCNL or
ESWL (if stone is harder than 1000 H.U do not offer ESWL)
2nd line treatment: Open/laparoscopic surgery
1st line treatment: PCNL or Open/laparoscopic surgery.
2nd line treatment: multiple session RIRS or
multiple session of ESWL (patients unfit for open/laparoscopic surgery or PCNL)
1st line treatment: PCNL or open surgery.
2nd line treatment: multiple session RIRS
or multiple session of ESWL
(patients unfit for or refuse open surgery or PCNL

VI. COMPLICATION OF UROLITHIASIS

Complications include acute renal failure secondary to obstruction, anuria, urinary tract infection with renal obstruction, and sepsis.

VII. CONCLUSION

After stone passage, every patient should be assigned to a group with low or high risk of stone formation. For correct classification, reliable stone analysis and basic evaluation of every patient are required. Low-risk stone formers may benefit by adopting general preventive measures regarding fluid and nutritional intake, as well as lifestyle improvements. For high-risk stone formers, a specific metabolic evaluation is required to guide individual treatment and prevent stone recurrence.

Follow up for recurrence stone:

- Low risk patient: follow up every 12 months evaluation (Urinalysis, renal function test, KUB ultrasound/X-ray)
- High risk patient: follow up every 6 months evaluation (Urinalysis, renal function test, KUB ultrasound/X-ray with specific tests)

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MALE STERILIZATION VASECTOMY

DR. KHY SOTHEARA, PROF. BOU SOPHEAP

I. DEFINITION

- Vasectomy is a surgical procedure used in men to disrupt and occlude the vas deferens, which delivers sperm from the testicles. By interrupting sperm transport, this procedure provides permanent sterilization.
- Vasectomy is regarded as the safest method now available for male fertility control.

II. INDICATIONS

Vasectomy may be a good choice for a man who:

- Is in a relationship, and both partners agree that they do not want children or additional children. They do not want to use, or cannot use, other forms of birth control.
- Is in a relationship and a pregnancy would be unsafe for the woman partner because of health problems.
- Is in a relationship, and one or both partners have genetic disorders that they do not want to pass on.
- Does not want to be bothered by having to use other forms of birth control during sexual activity.

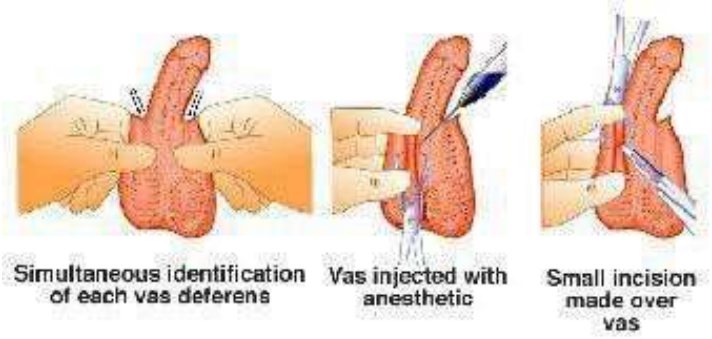
III. PROCEDURE VASECTOMY

Two types of vasectomy under local anesthesia with antibioprophylaxis are available (+/-histology confirmation).

Based on the patient, surgeons decide which type of vasectomy needs to be carried out.

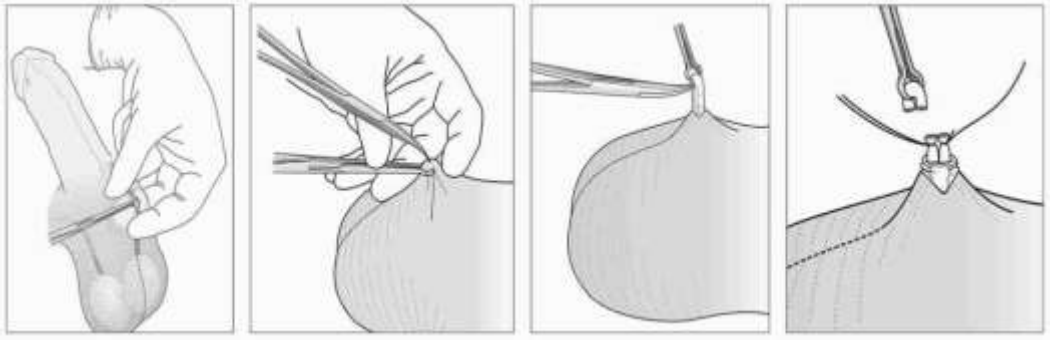
3.1 Conventional vasectomy

For a conventional vasectomy, one or two small cuts are made in the skin of the scrotum to reach the vas deferens. The vas deferens is cut and a small piece may be removed, leaving a short gap between the two ends. Next, the urologist may cut the ends of the vas and then tie the cut ends or put some tissue in between them. These steps are then repeated on the other vas, either through the same cut or through a new one. The scrotal cuts may be closed with dissolvable stitches or allowed to close on their own.



3.2 No-scalpel vasectomy

For a no-scalpel vasectomy, the urologist feels for the vas under the skin of the scrotum and holds it in place with a small clamp. A tiny hole is made in the skin and stretched open so the vas deferens can be gently lifted out. It is then cut, tied or seared, and put back in place.



IV. CONTRAINDICATIONS

There are no absolute contraindications for a vasectomy.

- Vasectomy may not be a good choice for a man who:
  • Is in a relationship with someone who has not decided on whether to have children in the future.
  • Is in an unstable or stressful relationship.
  • Is considering the operation just to please a partner.
  • Wants to have children later by storing sperm or by reversing the vasectomy.
  • Is young and may want to make a different decision in the future.
  • Is single when deciding to have a vasectomy. This includes men who are divorced, widowed, or separated.
  • Have coagulopathy
  • Have previous scrotal surgery
  • Have chronic orchialgia
  • Have testicular pathology such as a malignancy.

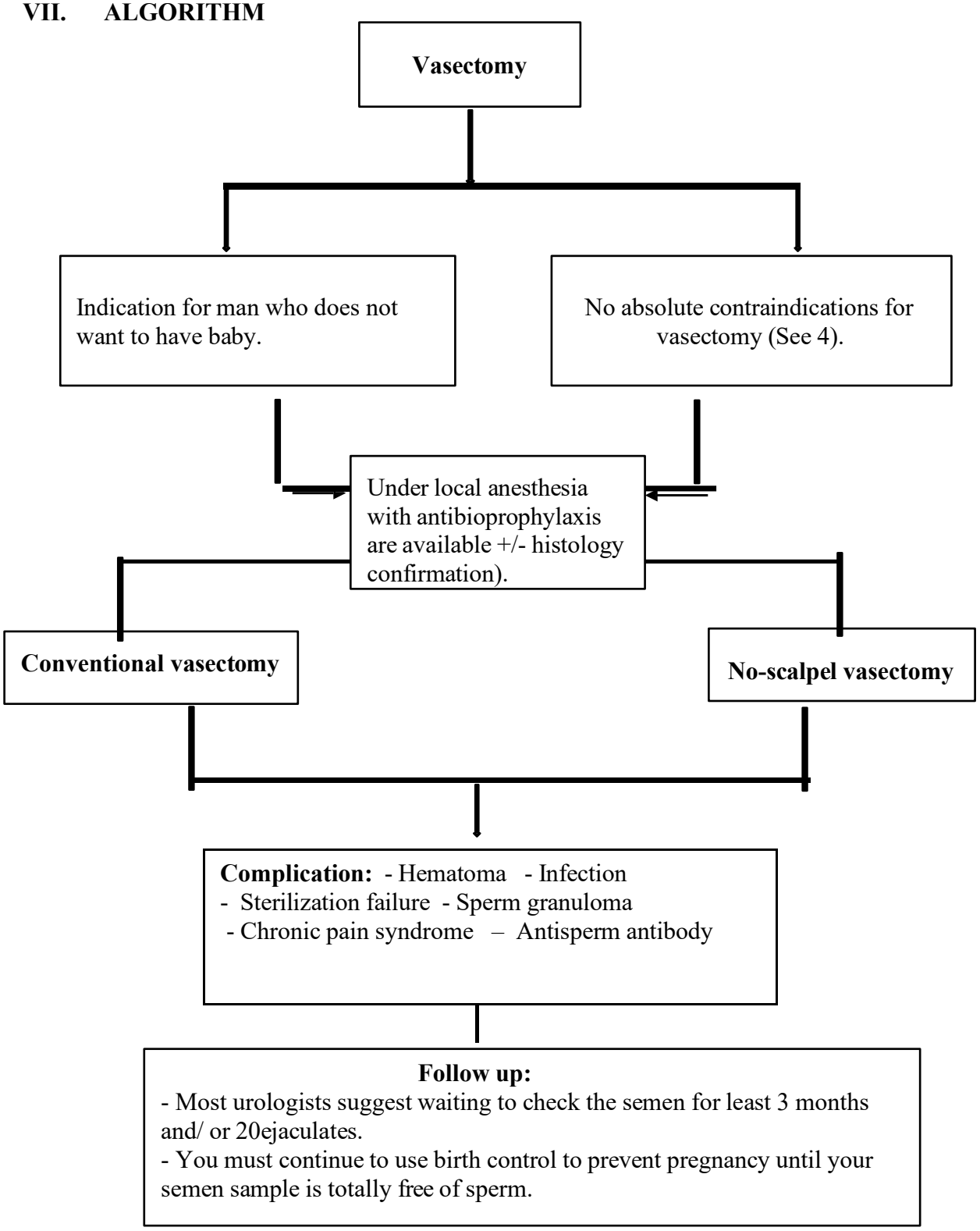
V. MONITORING

- Vasectomy does not affect a man's ability to have an erection or orgasm, or to ejaculate semen. A vasectomy does not prevent the spread of sexually transmitted infections.
- A vasectomy does not increase your risk of prostate cancer or testicular disease.
- Your sperm count gradually decreases after a vasectomy. After about 3 months, sperm are no longer present in the semen.
- Most urologists suggest waiting to check the semen for at least 3 months and/or 20 ejaculates.
- One in 100 men will still have sperm in their ejaculate at that time and may need to wait longer for the sperm to clear). You must continue to use birth control to prevent pregnancy until your semen sample is totally free of sperm.

VI. COMPLICATIONS

- Hematoma
- Infection
- Sterilization failure
- Sperm granulomas
- Post-vasectomy pain syndrome
- Anti-sperm antibodies(autoimmune disease)

VII. ALGORITHM



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MUSCLE INVASIVE BLADDER CANCER

DR. LAM KORVIN, PROF.BOU SOPHEAP, DR.OUK REAKSMEY

I. CASE DEFINITION:

Bladder cancer is the common urologic cancer that has the highest recurrence rate of any malignancy. The most common type is urothelial carcinoma (UC).

Muscle-invasive Bladder Cancer (MIBC), It comprises all the bladder tumors stages that invade the muscle layer of the bladder's wall (T2, T3, T4) and metastatic bladder cancer.

II. ETIOLOGY

- Tobacco usage and 'Second-hand' exposure to tobacco smoke: the most common cause.
- Occupational exposure: Aromatic mines, Polycyclic aromatic hydrocarbons and Chlorinated hydrocarbons (found in paint, dye, metal, and petroleum products): 2nd most important risk factor
- Arsenic exposure (Exa : arsenic-based pesticides)
- Radiation treatment of the pelvis.
- Chemotherapy with cyclophosphamide
- Long-term indwelling catheters: can leads to Squamous cell carcinoma of bladder (x 16- to 20).
- Genetic polymorphisms:
- Schistosomiasis infection: can leads to Squamous cell carcinoma of bladder
- Others: Bladder diverticulum, Bacillus Calmette-Guerin (BCG) treatment for CIS (Rarely),
- Bladder exstrophy.

III. DIAGNOSTIC PROCEDURE

i. Clinical argument:

- Macroscopic hematuria or microscopic: +++
- Others lower urinary symptoms: not specific to bladder's cancer
- Irritative lower urinary tract symptoms (Carcinoma *in situ*)

ii. Technical procedure

a. Laboratory test:
   - Urines dipstick
   - Urinary cytology
   - Complete Blood Count
   - Kidney function tests (BUN, Creatininemia)

b. Imaging study
   - KUB Ultrasound.
   - CT-urography: +++ Used to detect papillary tumors in the urinary tract (filling defects and/or hydronephrosis).
   - CT-Thoraco-abdomino-pelvis: to detect tumor's extension in all patients with confirmed muscle-invasive bladder cancer.
   - MRI: abdomino-pelvis: better than CT for evaluation of local extension of tumor.

c. Cystoscopy+/- biopsy: most important in making diagnosis and possible biopsy for pathology examination.

- Technologies that enhance visualization of tumors during cystoscopy (Photodynamic diagnosis, Narrow-band imaging, IMAGE1 S™)

d. Endoscopic bladder tumor resections/ablation options: is the most important step in diagnosis and definitive treatment choices for all stage of bladder cancer.
-Transurethral resection of bladder tumor (TURBT): +++
   - The most important steps in making diagnosis and treatment of non–muscle-invasive bladder cancer begins with TURBT.
   - It offers the precise pathological staging; grading.
   - Bipolar current more preferable to monopolar current due to less obturator nerve reflex.

- Enbloc Laser enucleation of bladder tumor (Bipolar, Holmium laser, Thulium Fiber Laser, Thulium laser, ,,,,,,,,)
- Fulguration and laser vaporization:
   - For small tumor of Ta low grade (G1) tumors, small papillary recurrences.

IV. DIFFERENTIAL DIAGNOSIS

1. Secondary tumor of bladder from surrounding organs' malignancy (gynecological cancers, rectal cancer, prostate cancer)
2. Benign tumor/diseases of bladder: Pheochromocytoma, hemorrhage cystitis, radiation induced cystitis, acute cystitis,,,,,,
3. Abnormal bleeding conditions of the patients (coagulopathy, usage anticoagulation agents, antiplatelet medication,,,,,,,,,,)
4. Nephrolithiasis
5. Renal Cell carcinoma, Renal transitional Carcinoma
6. Urinary tract trauma/injuries

V. STAGING AND CLASSIFICATION, SEE ANNEX 1

VI. GRADING, AND HISTOLOGIC SUBTYPES: SEE ANNEX2, ANNEX3

iii. Grading: Pathology of muscle-invasive bladder cancer All MIBC cases are high-grade UCs. For this reason, no prognostic information can be provided by grading MIBC.

iv. Identification of morphological subtypes is important for prognostic reasons and treatment decisions.

Currently the following subtypes of UC are used: 1. urothelial carcinoma (>90%). urothelial carcinomas with partial squamous and/or glandular or divergent differentiation; micropapillary UC; nested/microcystic; large nested; microtubular UC; plasmacytoid, signet ring; lymphoepithelioma-like; giant cell, diffuse, undifferentiated; sarcomatoid UC; UCs with other rare differentiations; urothelial carcinomas with partial NE (neuroendocrine differentiation, % to be given); pure neuroendocrine carcinoma (including small and large cell neuroendocrine carcinomas. In the new WHO 2022 all subtypes are considered HG. The majority of subtypes are MIBC.

VII. THERAPEUTIC APPROACH: AFTER MAKING DIAGNOSIS OF MIBC BY CLINICAL INFORMATION, CYTOLOGY, IMAGING, PATHOLOGICAL STAGING, GRADING, SUBTYPE IDENTIFICATION, ADDITIONAL TREATMENT OPTIONS ARE CHOSEN:

a. Counselling of smoking cessation must be done quickly.

b. For non-metastatic MIBC treatments are:

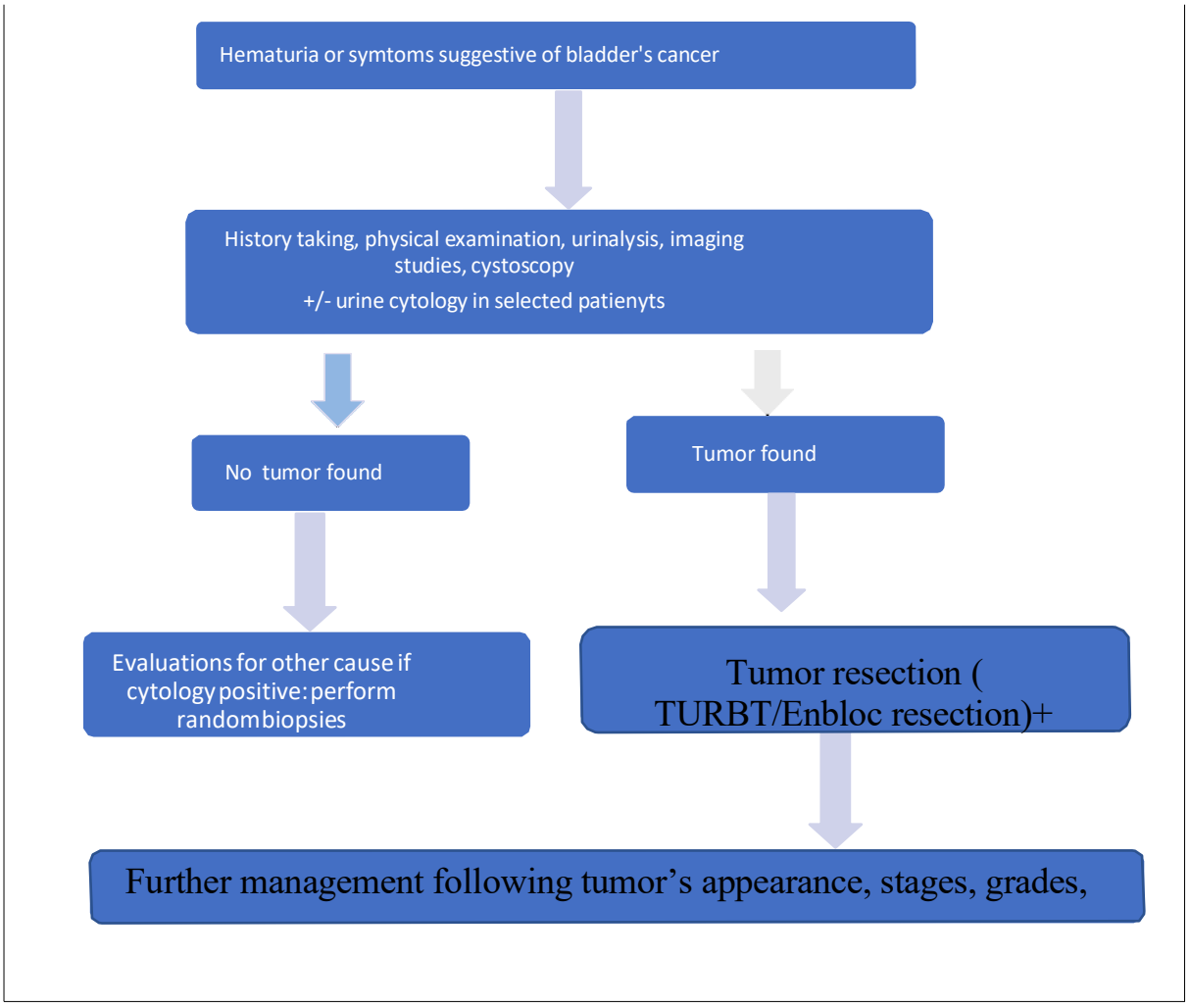
- Radical cystectomy is the criterion standard for the treatment of patients with stage c T2-T4a N0Mo if the patient is fit for surgery:
  - In male patient:
    - Cystoprostatectomy: removal of the bladder, peritoneal covering, perivesical fat, prostate, seminal vesicles, vasa deferentia, and, sometimes, the membranous or entire urethra.
    - Total urethrectomy: if positive urethral margin (concomitantly if found on frozen section exam / as delay surgery if found on final pathology).
    - Pelvic lympadenectomy; standard, or extended
    - Urinary diversion: continence or incontinence
  - In Female: Anterior pelvic exenteration consists of
    - Cystectomy, urethrectomy, hysterectomy, salpingo-oophorectomy, and partial anterior vaginectomy.
    - Vaginal-sparing techniques can be considered in low-stage patients, which will help with the preservation of sexual function.
    - Pelvic lympadenectomy; standard, or extended
    - Urinary diversion: continence or incontinence
- Palliative cystectomy with urinary diversion or urinary diversion only: can be used in unresectable locally-advanced tumors invading the pelvic or abdominal wall (T4b)
  With debilitating symptoms, including bleeding, pain, dysuria and urinary obstruction. If control of the symptoms is not possible by less invasive methods such as radiotherapy.

- Neo-adjuvant chemotherapy: for down staging purpose, control micrometastasis (improve survival and local recurrent): Platinum based Chemotherapy.
- Adjuvant chemotherapy: offer adjuvant Platinum based Chemotherapy.
- to patients with pT3/4 and/or pN+ disease if no neoadjuvant chemotherapy has been given.
- Trimodality bladder-preserving treatment: (TURB, concurrent Chemo-Radiation). Indicated in patient with MIBC stage cT2 No Mo disease who is unfit or refuse radical surgery.
- Radiotherapy: for unfit patient for radical cystectomy or as part of Trimodality bladder preserving treatment or for bleeding control purpose.

c. For metastatic MIBC:

- First line: Immunotherapy as combination therapy (Enfortumab vedotin plus Pembrolizumab) or monotherapy
- Second line treatment: Cysplatine or Caboplatine based chemotherapy.

VIII. DIAGNOSIS AND TREATMENT ALGORITHMS FOR BLADDER':



Flow chart for the management of T2–T4a N0M0 urothelial bladder cancer

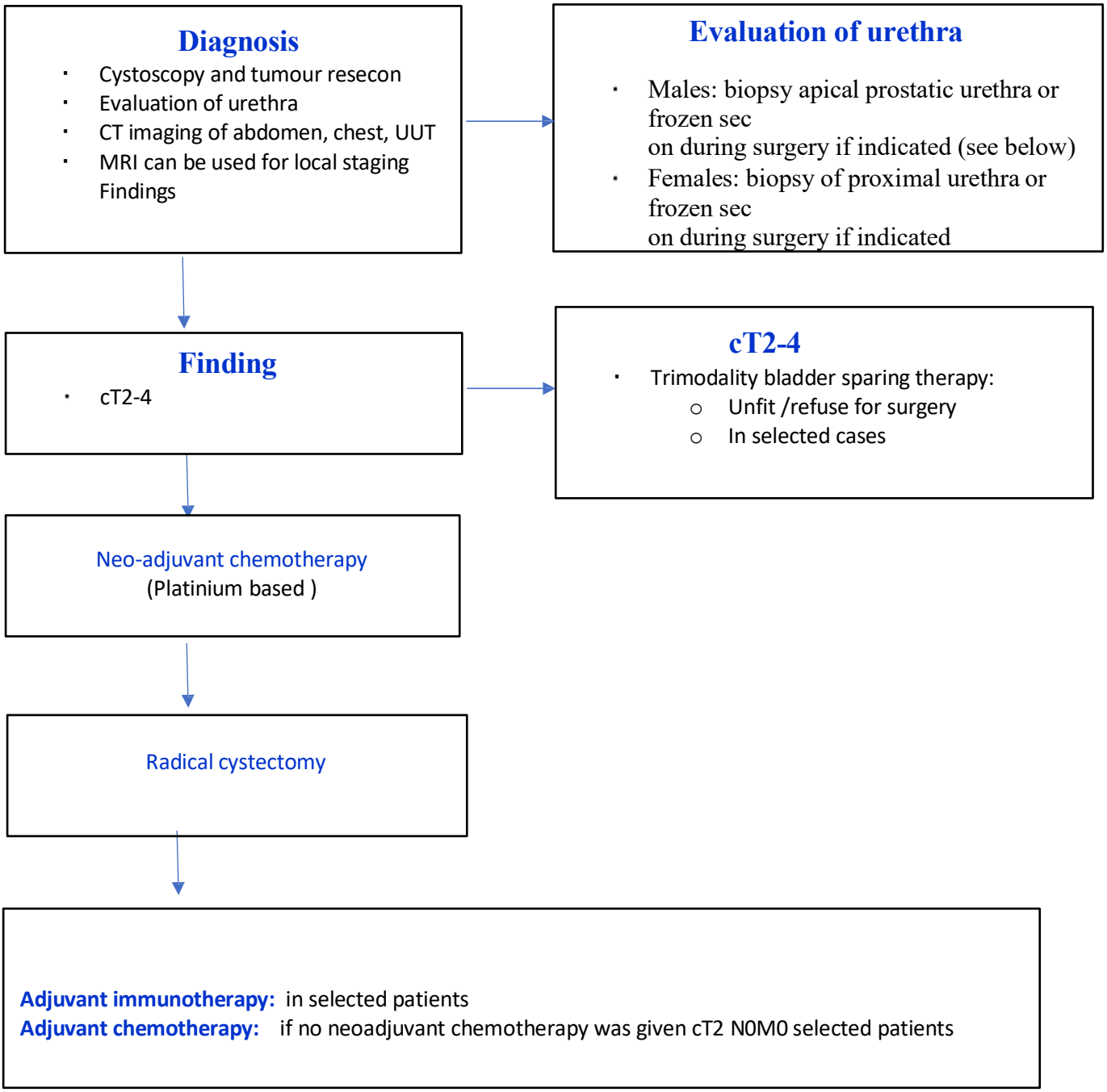
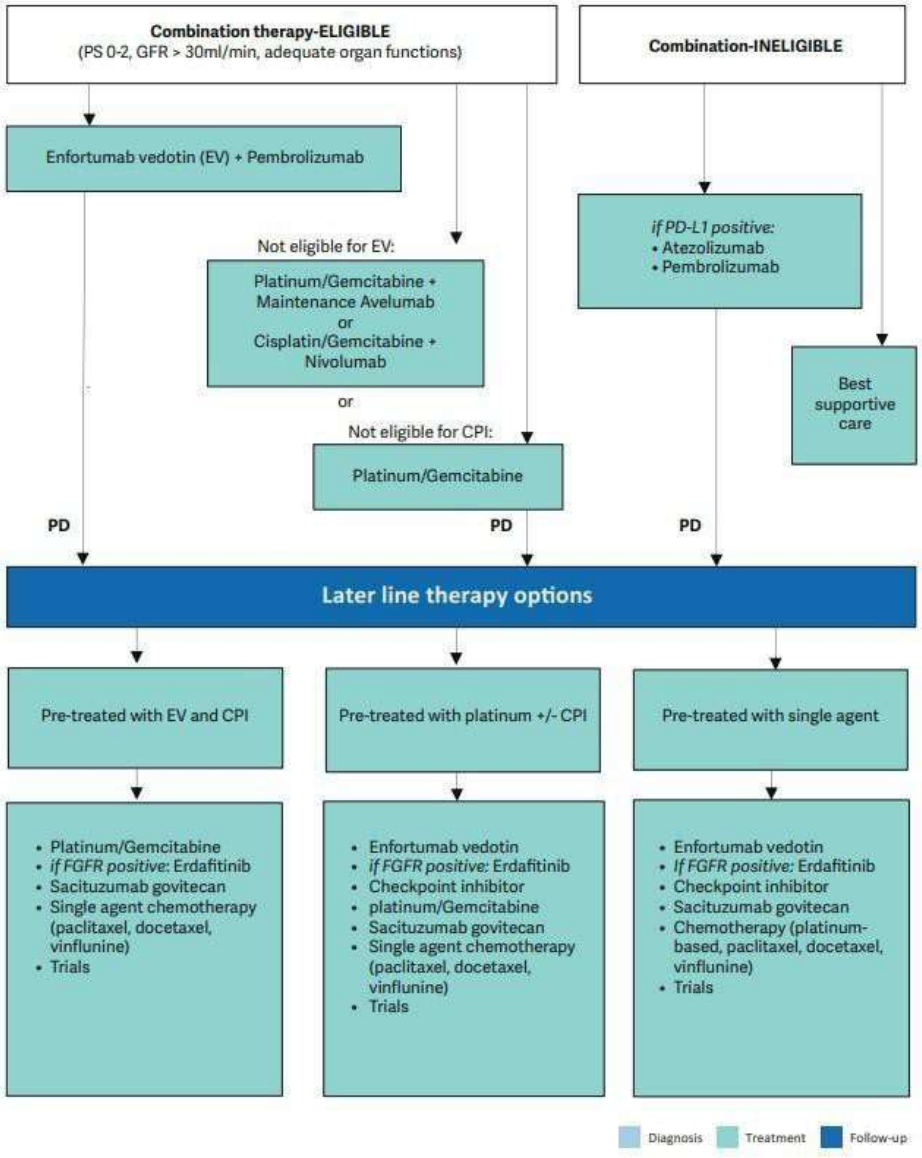


Figure 2: Flow chart for the management of metastatic urothelial cancer*



**EV = enfortumab vedotin; FGFR = fibroblast growth factor receptor; GFR = glomerular filtration rate; PS = performance status; CPI=checkpoint inhibitor; PD-L1= programmed death-ligand 1; PD= programmed death*

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NEPHROSTOMY CATHETER CARE

DR. OEUR SOPAGNA, PFOF.BOU SOPHEAP

I. DESCRIPTION

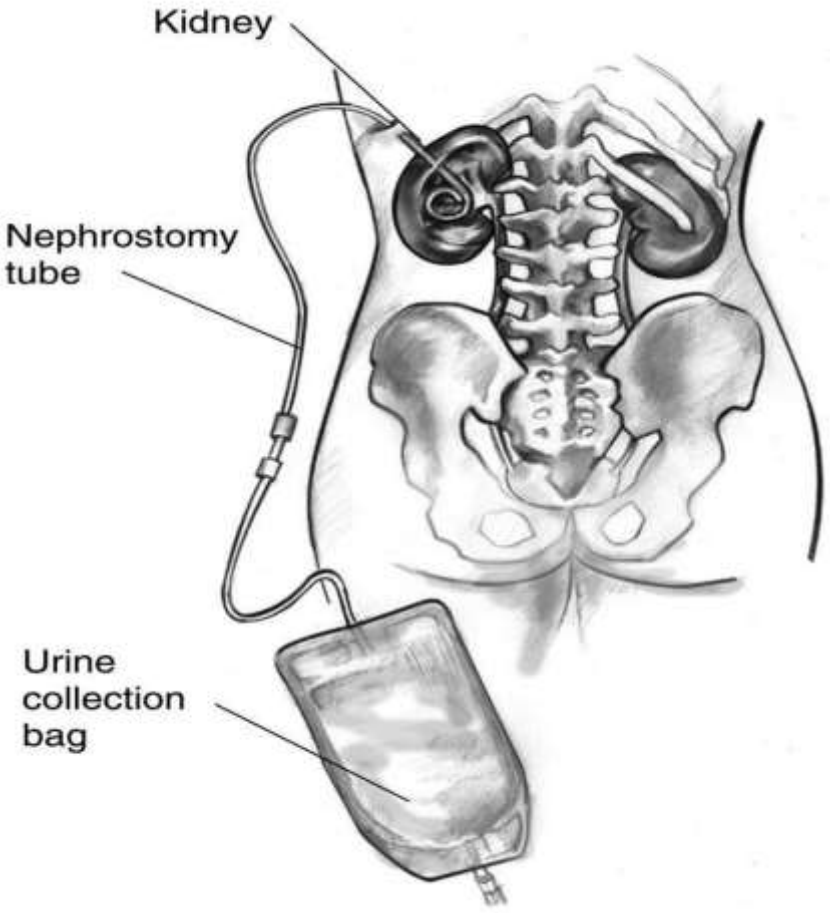
A urologic procedure that involves the insertion of a pigtail catheter (nephrostomy tube) into the dilated renal pelvis.

II. INDICATIONS

- Upper urinary tract obstruction (when ureteral stent cannot be placed)
- Ureteral injuries or fistulas
- Access for urologic procedures (e.g., percutaneous nephrolithotomy, delivery of chemotherapeutic agents)
- Antegrade pyelography

III. PROCEDURE

- Insertion of a catheter into the renal pelvis under ultrasound guidance using the Seldinger technique



- Routine antibiotic prophylaxis within one hour before the procedure is recommended.
- Catheter should be exchanged every 3 months.

IV. CONTRAINDICATIONS

- Uncorrectable coagulopathy

V. COMPLICATIONS

- Damage of adjacent organs
    - Vascular injury and hemorrhage
    - Bowel perforation
    - Pleural complications (e.g., pneumothorax, hemothorax)
- Urosepsis (esp. in patients with pyonephrosis)
- Catheter encrustation or displacement

VI. NEPHROSTOMY PROCEDURE

- Preparation: Before the procedure, you will be asked to empty your bladder. You may also receive medication to help you relax or antibiotics to prevent infection.

- Placement: You'll be positioned on the examination table, usually lying on your stomach or side. The skin over the kidney area is cleaned and sterilized.

- Local anesthesia: The area where the catheter will be inserted is numbed with a local anesthetic to minimize discomfort during the procedure.

- Guided insertion: Using ultrasound or fluoroscopy (real-time X-ray), the healthcare provider guides a thin needle through the skin and into the kidney's collecting system.

- Catheter insertion: Once the needle is in the correct position, a guide wire is inserted through the needle into the kidney. The needle is then removed, and a larger catheter is threaded over the guide wire into the kidney. The catheter is typically soft and flexible to minimize trauma to the surrounding tissues.

- Confirmation: Once the catheter is in place, imaging may be used to confirm its position within the kidney and ensure proper drainage.

- Securing the catheter: The catheter is secured to your skin using tape or a securement device to prevent it from being accidentally dislodged.

- Post-procedure care: After the procedure, you may be monitored for a short time to ensure there are no immediate complications. You'll receive instructions on how to care for the catheter at home, including how to empty the drainage bag and signs of infection to watch for.

- Follow-up: You'll typically have a follow-up appointment with your healthcare provider to monitor the catheter and assess your progress.

VII. NEPHROSTOMY CARE

- Taking care of a nephrostomy catheter involves several steps to ensure cleanliness and prevent infection:

- Hand hygiene: Wash your hands thoroughly with soap and water before and after handling the catheter.

- Cleaning the site: Clean around the catheter site with mild soap and water daily, and pat dry. Avoid using alcohol or hydrogen peroxide, as they can irritate the skin.

- Securing the catheter: Make sure the catheter is securely taped to your skin to prevent it from being accidentally pulled out.

- Emptying the drainage bag: Regularly empty the drainage bag when it's about half full to prevent leakage and infection. Always keep the drainage bag below the level of your kidney to ensure proper drainage.

- Monitoring for signs of infection: Watch for signs of infection such as redness, swelling, warmth, or foul odor around the catheter site. Also, monitor for signs of urinary tract infection such as fever, chills, or cloudy urine.

- Maintaining hygiene during showers: Cover the catheter site with a waterproof dressing or plastic wrap when showering to prevent water from getting into the catheter site.

- Avoiding tugging or pulling: Be careful not to tug or pull on the catheter, as this can cause pain and dislodgement.

- Regular follow-up: Attend all scheduled follow-up appointments with your healthcare provider to monitor the catheter and address any concerns or complications.

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NEUROGENIC BLADDER

Dr. OUK REAKSMEY, Dr. HAY VANEL, Prof. BOU SOPHEAP

I. CASE DEFINITION

Neurogenic bladder is the term for what happens when neurological (nervous system) conditions affect the way the bladder works. There are two major types of bladder control problems linked to neurogenic bladder. Depending on the nerves involved and the nature of the damage, your bladder becomes either overactive (spastic or hyper-reflexive) or underactive (flaccid or hypotonic).

II. ETIOLOGY

These are some possible causes of neurogenic bladder:

- Diabetes
- Infections
- Accidents that cause injury to the brain or spinal cord
- Genetic nerve problems
- Heavy metal poisoning
- Birth defects that affect the spinal cord
- Brain or spinal cord tumors
- Stroke
- Herniated disks
- Multiple sclerosis
- Parkinson disease

III. DIAGNOSTIC PROCEDURE

These are the most common symptoms of neurogenic bladder:

- Urinary tract infection (UTI)
- Kidney stones
- Unable to control urine (urinary incontinence)
- Small amount of urine when urinating
- Urinary frequency and urgency
- Dribbling urine
- Loss of feeling that the bladder is full
- Unable to urinate

The symptoms of neurogenic bladder may look like other conditions.

- **X-rays of the skull and spine.** This imaging test uses invisible energy beams to make images of tissues, bones, and organs.
- **Imaging tests of the bladder and ureters**
- **Ultrasound.** This imaging test uses sound waves to create images of the organs on a computer screen.
- **Cystoscopy.** We put a thin, flexible tube and viewing device in through the urethra to examine the urinary tract. It checks for structure changes or blockages, such as tumors or stones.

- **Tests that are done by filling the bladder, such as urodynamics.** These tests show how much the bladder can hold. They also check to see if it fully empties.

IV. DIFFERENTIAL DIAGNOSIS

- Antenatal Urinary Tract Dilation (Hydronephrosis)
- Myelodysplasia and Neurogenic Bladder Dysfunction
- Pediatric Myelodysplasia
- Pediatric Ureteropelvic Junction Obstruction
- Pediatric Urinary Tract Infection
- Posterior Urethral Valves
- Urethral Anomalies and Urethral Prolapse in Children
- Voiding Dysfunction

V. THERAPEUTIC APPROACH

Treatment for neurogenic bladder depends on the cause. It's aimed at preventing kidney damage and may include:

- Medicines
- Emptying the bladder with a catheter at regular times
- Preventive antibiotics to reduce infection
- A surgical procedure that places an artificial cuff around the neck of the bladder that can be inflated to hold urine and deflated to release it
- Surgical procedures that may enlarge the bladder size or remove a portion of the weak sphincter muscle or create an opening in the belly (stoma) for the urine to drain.
- Botulinum toxin shots (injections) into the bladder muscle. This medicine keeps the bladder from contracting too often.
- Placing an electrical device to stimulate or slow down bladder activity

VI. COMPLICATION

The following are often linked to a neurogenic bladder:

- **Urine leakage.** This often happens when the muscles holding urine in don't get the right message.
- **Urine retention.** This happens if the muscles holding urine in don't get the message that it's time to pass urine.
- **Damage to the tiny blood vessels in the kidney.** This may happen if the bladder becomes too full and urine backs up into the kidneys. This causes extra pressure. It may lead to blood in the urine and kidney failure.
- **Infection of the bladder, ureters, or kidneys.** This often results from urine that is held too long before it’s passed out of the body.

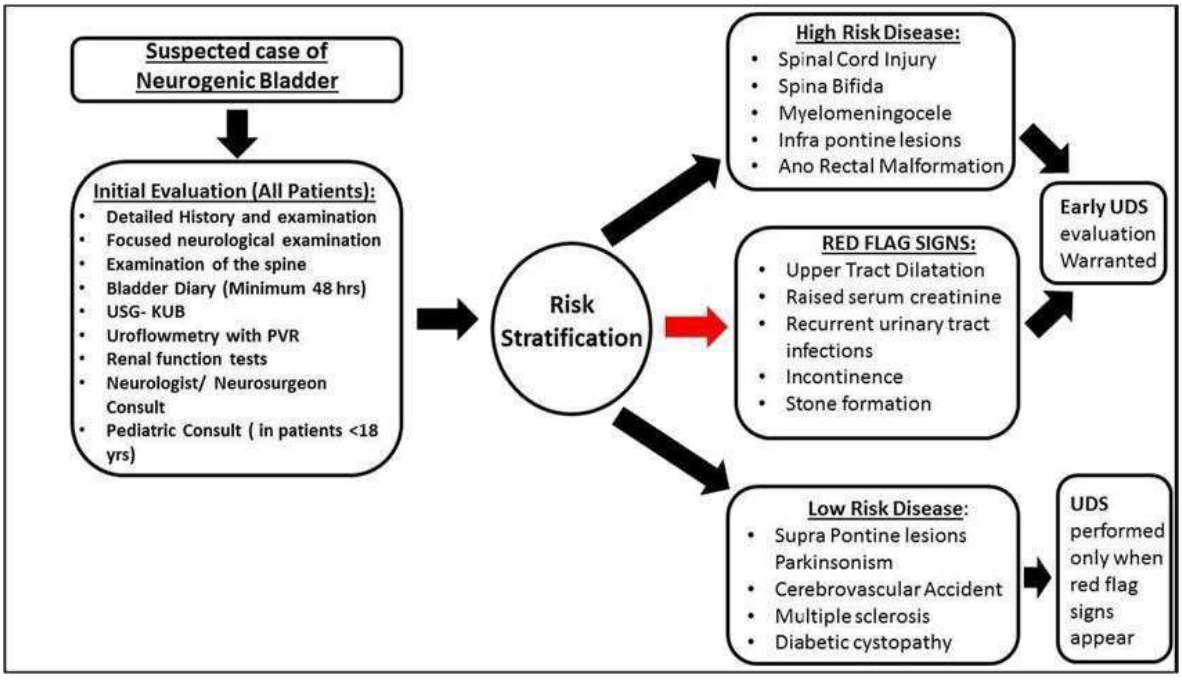
VII. FOLLOW UP

- With neurogenic bladder, the nerves that carry messages back and forth between the bladder and the spinal cord and brain don’t work the way they should.
- Common symptoms include dribbling urine, loss of feeling that the bladder is full, and being unable to control urine (urinary incontinence).
- Damage or changes in the nervous system and infection are some of the causes

of neurogenic bladder.

- Treatment is aimed at preventing kidney damage. It may include medicine, urinary catheters, antibiotics to reduce the chance of infection, and in severe cases, surgery.
- Some complications include urine leakage, inability to pass urine, kidney damage, and kidney or urinary tract infections.

VIII. ALGORITHM



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NON-MUSCLE INVASIVE BLADDER CANCER

DR. LAM KORVIN, PROF.BOU SOPHEAP, DR.OUK REAKSMEY

I. CASE DEFINITION:

Bladder cancer is the common urologic cancer that has the highest recurrence rate of any malignancy. The most common type is urothelial carcinoma (UC).

Non-muscle-invasive Bladder Cancer (NMIBC), It comprises all the bladder tumors stages that not yet invade the muscle layer of the bladder's wall (Ta, T1 and carcinoma *in situ* (CIS)).

II. ETIOLOGY

- Tobacco usage and 'Second-hand' exposure to tobacco smoke: the most common cause.
- Occupational exposure: Aromatic mines, Polycyclic aromatic hydrocarbons and Chlorinated hydrocarbons (found in paint, dye, metal, and petroleum products): 2nd most important risk factor
- Arsenic exposure (Exa : arsenic-based pesticides)
- Radiation treatment of the pelvis.
- Chemotherapy with cyclophosphamide
- Long-term indwelling catheters: can leads to Squamous cell carcinoma of bladder (x 16- to 20).
- Genetic polymorphisms:
- Schistosomiasis infection: can leads to Squamous cell carcinoma of bladder
- Others: Bladder diverticulum, Bacillus Calmette-Guerin (BCG) treatment for CIS (Rarely),
- Bladder exstrophy.

III. DIAGNOSTIC PROCEDURE

Clinical argument:

- Macroscopic hematuria or microscopic: +++
- Others lower urinary symptoms: not specific to bladder's cancer
- Irritative lower urinary tract symptoms (Carcinoma *in situ*)

Technical procedure

d. Laboratory test:
   - Urines dipstick
   - Urinary cytology
   - Complete Blood Count
   - Kidney function tests (BUN, Creatininemia)

e. Imaging study
   - KUB Ultrasound.
   - CT-urography: +++ Used to detect papillary tumors in the urinary tract (filling defects and/or hydronephrosis).

f. Cystoscopy+/- biopsy: most important in making diagnosis and possible biopsy for pathology examination.

- Technologies that enhance visualization of tumors during cystoscopy (Photodynamic diagnosis, Narrow-band imaging, IMAGE1 S™)

g. Endoscopic bladder tumor resections/ablation options: is the most important step in diagnosis and definitive treatment choices for all stage of bladder cancer.
-Transurethral resection of bladder tumor (TURBT): +++
  - The treatment of non–muscle-invasive bladder cancer begins with TURBT.
  - It offers the precise pathological staging; grading.
  - Bipolar current more preferable to monopolar current due to less obturator nerve reflex.

- Enbloc Laser enucleation of bladder tumor (Bipolar, Holmium laser, Thulium Fiber Laser, Thulium laser, ,,,,,,,,)
- Fulguration and laser vaporization:
  - For small tumor of Ta low grade (G1) tumors, small papillary recurrences.

IV. DIFFERENTIAL DIAGNOSIS

h. Secondary tumor of bladder from surrounding organs' malignancy (gynecological cancers, rectal cancer, prostate cancer)
i. Benign tumor/diseases of bladder: Pheochromocytoma, hemorrhage cystitis, radiation induced cystitis, acute cystitis,,,,,,
j. Abnormal bleeding conditions of the patients (coagulopathy, usage anticoagulation agents, antiplatelet medication,,,,,,,,,,)
k. Nephrolithiasis
l. Renal Cell carcinoma, Renal transitional Carcinoma
m. Urinary tract trauma/injuries

V. STAGING AND CLASSIFICATION:

Table 1: TNM Classification 2017(annex1)

T - Primary Tumor	
TX	Primary tumor cannot be assessed
T0	No evidence of primary tumor
Ta	Non-invasive papillary carcinoma
Tis	Carcinoma in situ: 'flat tumour'
T1	Tumour invades subepithelial connective tissue
T2 T2a	Tumour invades muscle Tumour invades superficial muscle (inner half)

T2b	Tumour invades deep muscle (outer half)
T3 T 3	Tumour invades perivesical tissue Microscopically Macroscopically (extravesical mass)
T4 T 4	Tumour invades any of the following: prostate stroma, seminal vesicles, uterus, vagina, pelvicwall, abdominalwall Tumour invades prostate stroma, seminal, vesicles, uterus or vagina Tumour invades pelvic wall or abdominal wall
N – Regional Lymph Nodes	
NX	Regional lymph nodes cannot be assessed
N0	No regional lymph node metastasis
N1	Metastasis in a single lymph node in the true pelvis (hypogastric, obturator, external iliac, or presacral)
N2	Metastasis in multiple regional lymph nodes in the true pelvis (hypogastric, obturator, external ili ac
N3	Metastasis in common iliac lymph node(s)
M - Distant Metastasis	
M0	No distant metastasis
M1a	Non-regional lymph nodes
M1b	Other distant metastases

Carcinoma in situ (CIS): flat, high-grade, non-invasive urothelial carcinoma, and classified into the clinical types

CIS Types	Description
Primary CIS	Isolated CIS with no previous or concurrent papillary tumors and no previous CIS
Secondary CIS	CIS detected during follow-up of patients with a previous tumor that was not CIS
Concurrent CIS	CIS in the presence of any other urothelial tumors in the bladder.

VI. GRADING AND HISTOPATHOLOGICAL UROTHELIAL CARCINOMA'S TYPE, SUBTYPE, LYMPHOVASCULAR INVASION.(ANNEX2)

urinary tumours in 1973 and 2004 [39,40]

WHO 1973
- Urothelial papilloma
- Grade 1: well differentiated
- Grade 2: moderately differentiated
- Grade 3: poorly differentiated

WHO 2004
- Urothelial papilloma
- PUNLMP
- Low-grade papillary urothelial carcinoma
- High-grade papillary urothelial carcinoma

PUNLMP = papillary urothelial neoplasms of low malignant potential.

Types of urothelial carcinomas vs Grade(annex3)

Urothelial carcinoma types	**Grade**
Pure UC(>90% of all cases)	High or low grade
Subtypes - UC with partial (squamous-glandular or trophoblastic) divergent differentiation, - UC with micropapillary divergent differentiation; - UC with plasmacytoid divergent differentiation; - UC with sarcomatoid divergent differentiation; - UC with nested/microcystic divergent differentiation; - UC with microtubular divergent differentiation; - UC with large nested divergent differentiation; - UC with lymphoepithelioma-like divergent differentiation; - UC with giant cell, diffuse, undifferentiated divergent differentiation - Some UCs with other rare differentiations; - UCs with partial neuroendocrine differentiation, % to be given); - Pure neuroendocrine carcinoma (including small and large cell neuroendocrine carcinomas	High grade

Note:
-Most subtypes of urothelial carcinoma (micropapillary,plasmacytoid, sarcomatoid) have a worse prognosis than pure high-grade (HG) urothelial carcinoma.

-The presence of lymphovascular invasion (LVI) in transurethral resection of the bladder (TURB) specimens is associated with worse prognosis.

VII. RISKS STRATIFICATION:

The EAU, AUA/SUO stratifies NIMBC into risk groups based on their probability of progression to muscle-invasive disease. And this stratification will facilitate treatment recommendations. (annex4)

EAU's risk stratification of the patients(annex4)

Risks type	EAU Definition(T,G,CIS ,additional clinical risk factors)	Treatment recommendation
Low risk tumors	-Primary, single, TaT1 LG/G1, tumor < 3 cm, no CIS, age ≤ 70 years -Primary Ta LG/G1 tumor, no CIS with at most ONE of the additional clinical risk factors	Single immediate post TURBT intravesical instillation of chemotherapy
Intermediate-risks tumors	All tumors not meeting the definition of either low- or high-risk tumors	1st option: Single immediate post TURBT intravesical instillation of chemotherapy followed by Induction intravesical instillation of chemotherapy 1 year. 2nd option: Single immediate post TURBT intravesical instillation of chemotherapy followed by 1-year full dose of intravesical instillation BCG.
High-risks tumors	-All T1 HG/G3 without CIS, (except those included in the very high-risk group) -All CIS patients, (except those included in the very high-risk group) -Ta LG/G2 or T1G1, no CIS with all 3 risk factors -Ta HG/G3 or T1 LG, no CIS with at least 2 risk factors -T1G2 no CIS with at least 1 risk factor	1-3 years of full dose of intravesical instillation BCG Or Radical cystectomy AUA
Very high-risk Tumor	**Stage, grade with additional clinical risk factors:** Ta HG/G3 and CIS with all 3 risk factors T1G2 and CIS with at least 2 risk factors T1 HG/G3 and CIS with at least 1 risk factor T1 HG/G3 no CIS with all 3 risk factors	Radical cystectomy should be considered
	BCG refractory tumor	Radical cystectomy is recommended

-Additional clinical risk factors are: age > 70; multiple papillary tumors; and tumors diameter
> 3 cm. (Annex5)

AUA/SUO risk stratification of the patients (annex6)

Note: Management of any particular case that is not fall into any risk stratification above, the must be discussed in Multi-Disciplinary Team.

Note: 2021 EAU NMIBC Risk Calculator (www.nmibc.net)

VIII. THERAPEUTIC APPROACH:

v. Counselling of smoking cessation must be done quickly to:

vi. Treatment options for NMIBC: With pathological staging, grading and risk stratification, additional treatment options are chosen:

- Active Surveillance: Maybe suitable for recurrence low grade(G1)Ta tumors(low grade and non-invasive). (Need more evidence to recommend this option).
  - Adjuvant intravesical treatment:

- Post-operative irrigation: In case intravesical chemotherapy is not feasible, irrigation of the bladder might be considered Prevention of early recurrences. (Optimal volume infused and duration of irrigation remains unknown.)

- Intravesical chemotherapy: Advantage: reduce recurrent and progression of the tumors (by destroying circulating tumor cells after TURB, and by an ablative effect on residual tumor cells at the resection site and on small overlooked tumors.

  Chemotherapy 's drugs: mitomycin C (MMC), epirubicin or pirarubicin, gemcitabine,

  - A single, immediate, post-operative intravesical instillation of chemotherapy (should be considered during the 1st 2 hours after TURBT): (standard and complete for in low-risk patients)
  - *Repeat* adjuvant intravesical chemotherapy instillations (for intermediate risks patients)

- Intravesical bacillus Calmette-Guérin (BCG) immunotherapy
  - (for intermediate, high risks patients and CIS patients)
  - More effective but more side effects and more difficult to tolerate compared to Intravesical chemotherapy.
  - Optimal BCG schedule: 1-3 years depending on risk categories.

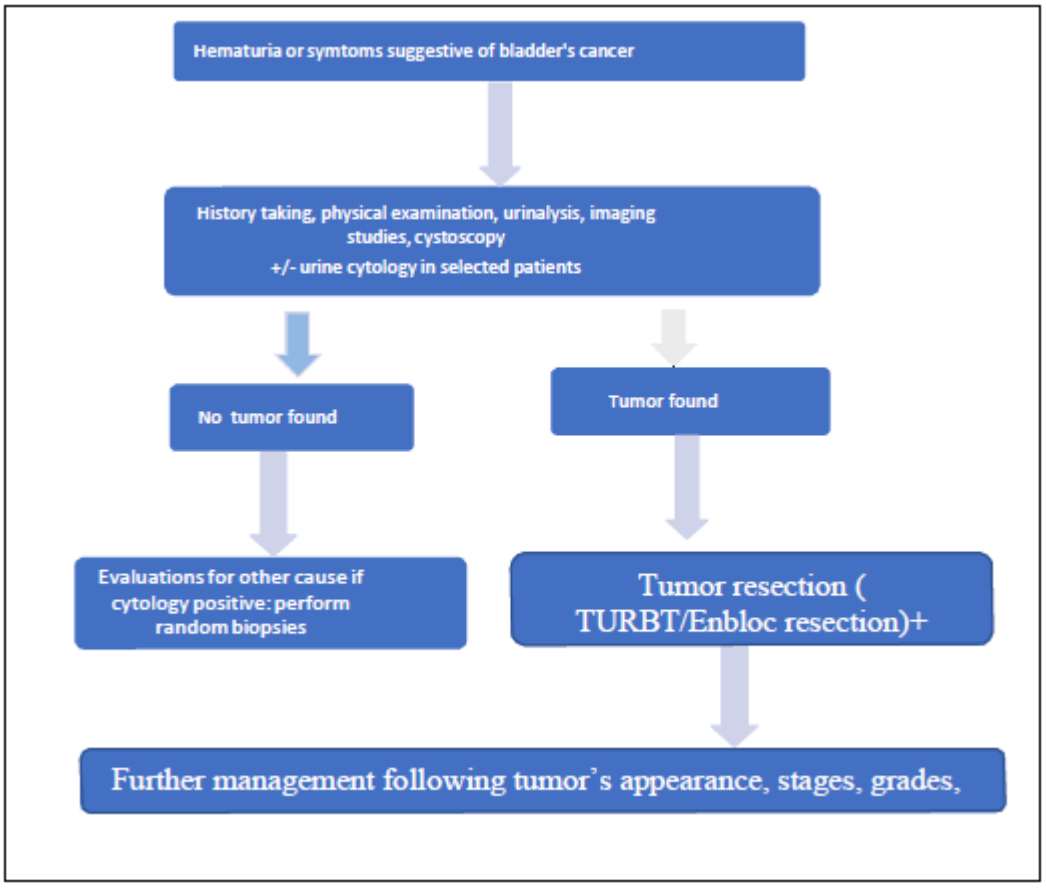
- Intravesical BCG plus chemotherapy: more effective than monotherapy but more toxicity.

- Early radical cystectomy for non-muscle-invasive bladder cancer:
  - In patient who are very high risk of disease

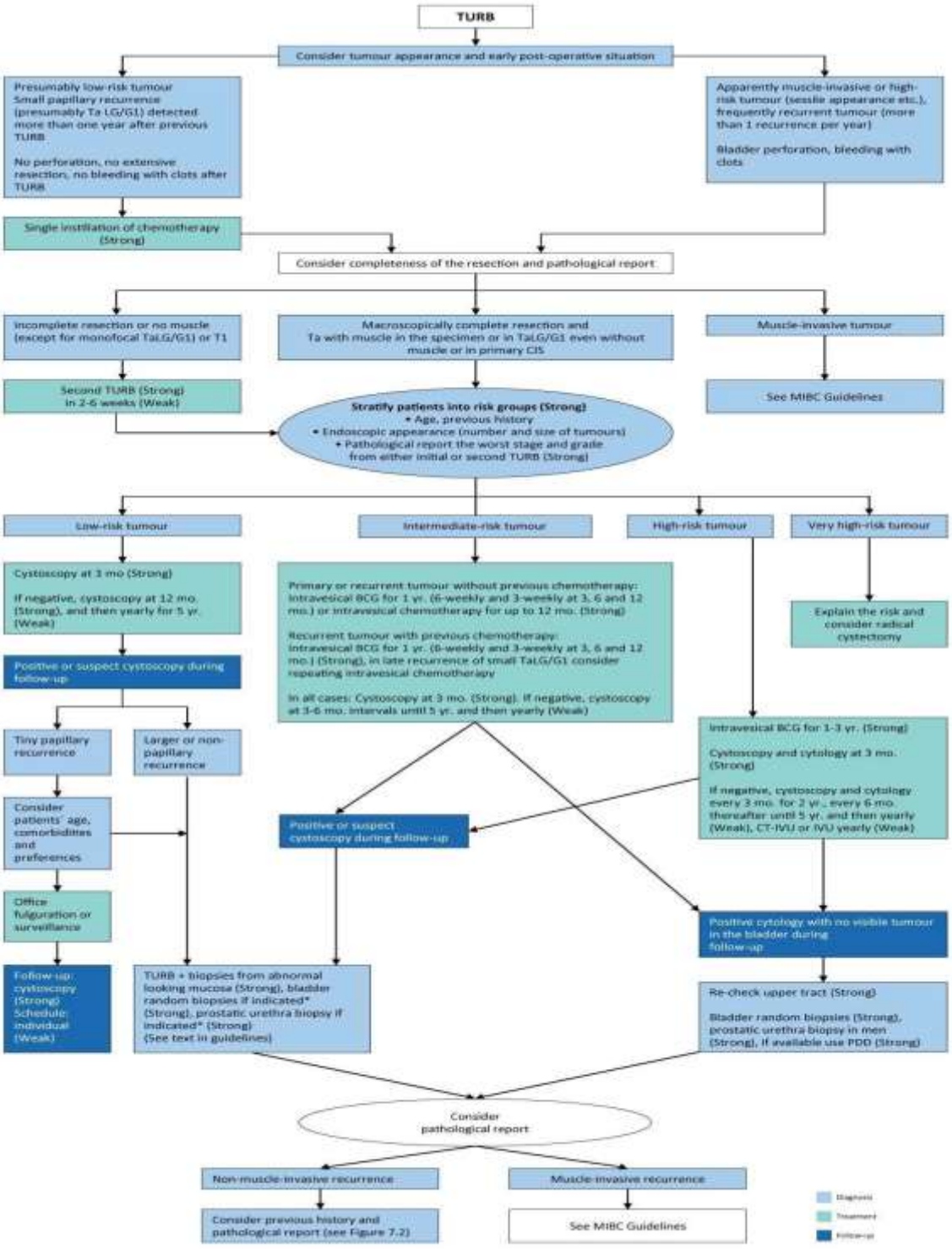
progression
- Patients with BCG-unresponsive tumors.
- BCG relapsing.
- High grade tumors.

- A delay in RC may lead to decreased disease-specific survival.
- The potential benefit of RC must be weighed against its risks, morbidity, and impact on quality of life (QoL) and discussed with patients, in a shared decision- making process.
- In patients in whom RC is performed before progression to MIBC, the 5-year DFS rate exceeds 80%.

IX. DIAGNOSIS AND TREATMENT ALGORITHMS FOR BLADDER':

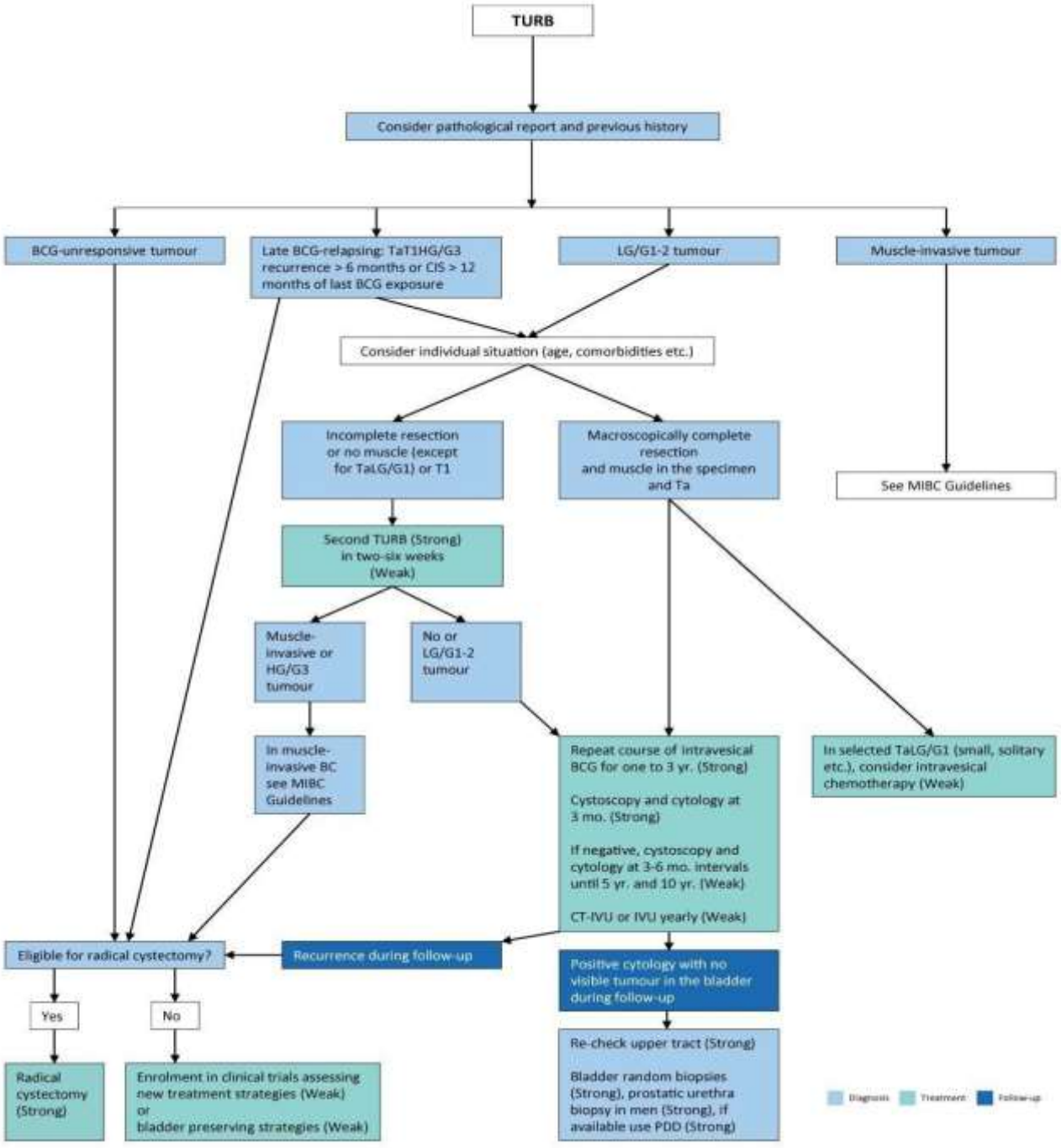


Diagnosis and treatment's algorithms for primary or recurrent bladder tumor(s) without previous BCG



BCG = bacillus Calmette-Guérin; CIS = carcinoma in situ; CT = computed tomography; IVU = intravenous urography; MIBC = muscle-invasive bladder cancer; PDD = photodynamic diagnosis; TURB = transurethral resection of the bladder.

Treatment's algorithms for tumor's recurrence's during or after intravesical BCG



BCG = bacillus Calmette-Guérin; CIS = carcinoma in situ; HG = high-grade; IVU = intravenous urography;LG = low-grade; PDD = photodynamic diagnosis; TURB = transurethral resection of the bladder.

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PARAPHIMOSIS

Dr. KHY Sotheara, PROF.BOU SOPHEAP, DR.OUK REAKSMEY

I. DEFINITION

- Paraphimosis is a true urologic emergency in which the retracted foreskin of an uncircumcised male cannot be returned to its normal anatomic position.
- In uncircumcised children, four months to 12 years old, with foreskin problems, paraphimosis (0.2%) is less common than other penile disorders such as balanitis (5.9%), irritation (3.6%), penile adhesions (1.5%), or phimosis (2.6%).
- In adults, paraphimosis is most commonly found in adolescents. It will occur in about 1% of all adult males over 16 years of age.

II. ETIOLOGY

- Commonly occurs iatrogenically(urinary catheter, cystoscopy, penile examination) Failure to return the retracted foreskin over the glans promptly after the initial retraction can lead to paraphimosis.
- Penile coital trauma and self-inflicted injuries.
- Risk factors:
  - Lack of circumcision
  - Urinary catheterization
  - Dependence on a caregiver for daily hygiene
  - Tight foreskin
  - Phimosis
  - Poor hygiene
  - Bacterial infection
  - Parasitic infections

III. DIAGNOSTIC PROCEDURE

3.1 Clinical argument

3.1.1 History

Parents of patients with physiologic phimosis may bring in the patient after noting an inability to retract the foreskin during routine cleaning or bathing. Parents may also be alarmed by "ballooning" of the prepuce during urination.

3.1.2 Sign and symptom

- Not being able to pull the foreskin back to its normal position
- Swelling of the end of the penis
- Discomfort and pain
- Other symptoms could include: Redness and tenderness, Trouble urinating

3.1.3 Physical examination

- The physical exam should focus on the penis, foreskin, and urethral catheter (if present).
- A pink color to the glans indicates reasonably good blood supply, whereas a dark, dusky, pale, bluish or black color implies possible ischemia or even necrosis.
- Typical paraphimosis symptoms include erythema, pain, and swelling of foreskin and glans due to the constricting ring of the phimotic foreskin.

3.2 Technical procedure

- Baseline lab: CBC+ blood group, PT, PTT, Electrolytes.
- Imaging study: Color-Doppler ultrasound, Angio-scan, MRI.

IV. DIFFERENTIAL DIAGNOSIS

- Acute angioedema
- Allergic contact dermatitis
- Anasarca
- Balanitis
- Balanitis xerotica obliterans
- Cellulitis
- Foreign body tourniquet
- Insect bites
- Penile carcinoma
- Penile fracture
- Penile hematoma

V. THERAPEUTIC APROACH

Three goals manage paraphimosis: manage pain, decrease swelling, manual reduction.

5.1 Medical therapy

- Pain control
- Non-pharmacological therapy
  - Ice packs
  - Manual reduction
- Pharmacological therapy
  - Osmotic agents (50% glucose, 20% Mannitol soaked-gauze,)
  - Injection of hyaluronidase into edematous prepuce (Dundee Technique)

5.2 Surgical therapy

- Minimally invasive: Puncture technique, Blood aspiration.
- Dorsal slit
- Circumcision: Stapler Circumcision, Open Circumcision.

5.3 Type of anesthesia

- Local anesthesia for adolescent: **Lidocaine (gel or injection) without adrenaline**.
- General anesthesia for children.

5.4 Monitoring

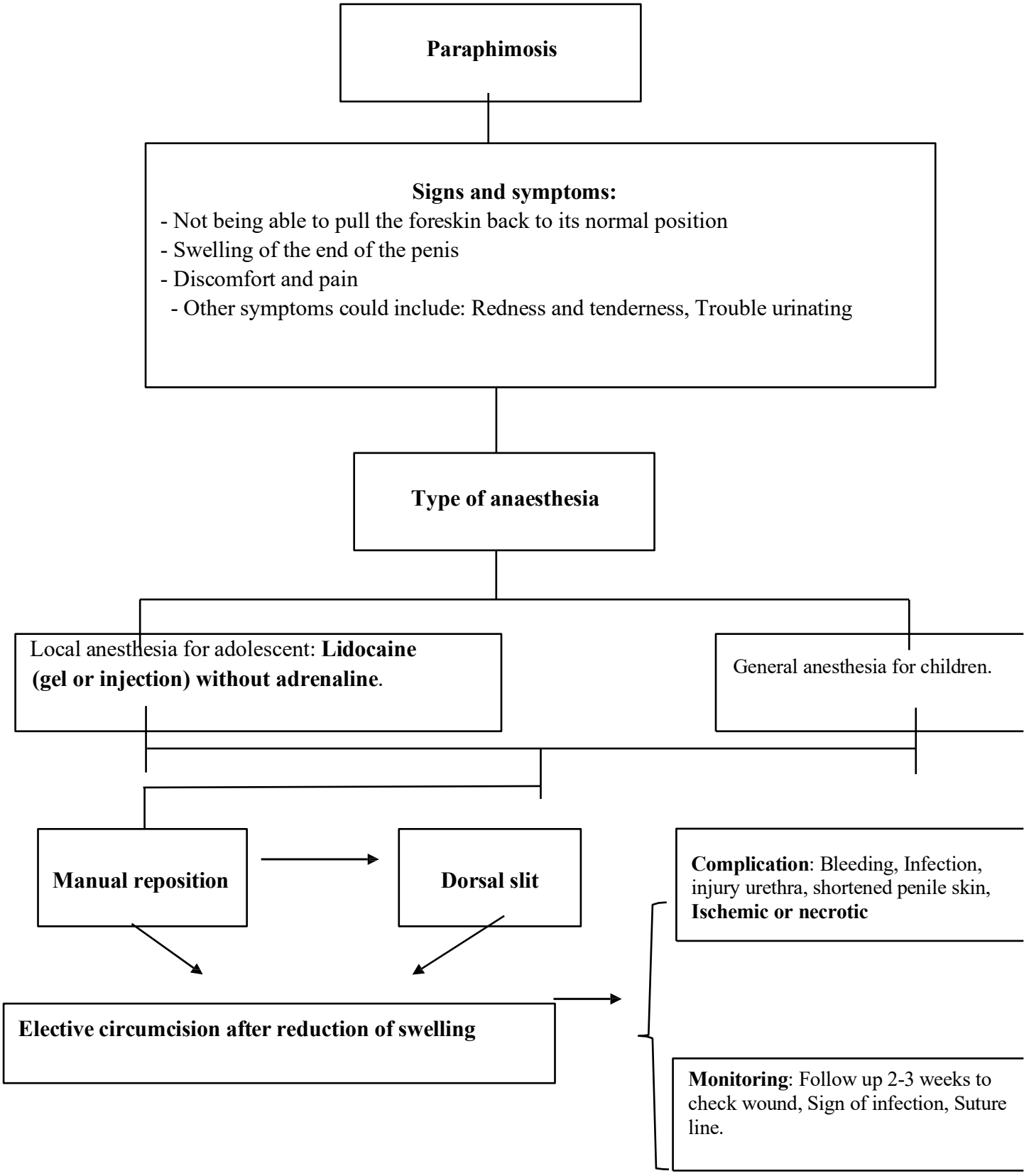
Patients generally undergo follow-up examination in 2-3 weeks to check the wound. Assess the wound for signs of infection and inspect the suture line.

VI. COMPLICATION

- Complications that can occur with paraphimosis include pain, infection, and inflammation of the glans penis.
- Operative complications include bleeding, infection, injury to the urethra, and shortened penile skin.

- If the condition is not relieved in a sufficiently prompt timeframe, **the distal penis can become ischemic or necrotic.**

VII. ALGORITHM



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PENILE CANCER

Dr. HAY VANEL, Dr. OUK REAKSMEY, Prof. BOU SOPHEAP

I. CASE DEFINITION

Penile carcinoma is usually a SCC and there are several recognised subtypes of penile SCC with different clinical features and natural history (see Table 1). Penile SCC usually arises from the epithelium of the inner prepuce or the glans.

II. ETIOLOGY

-Phimosis
-Chronic penile inflammation (balanoposthitis related to phimosis), lichen sclerosus
-Sporalene and ultraviolet A phototherapy for various dermatological conditions such as psoriasis
-Smoking
-HPV infection, condylomata acuminate
-Rural areas, low socio-economic status, unmarried
-Multiple sexual partners, early age of first intercourse

III. DIAGNOSTIC PROCEDURE

Penile cancer can be cured in over 80% of cases if diagnosed early, but is a life-threatening disease when lymphatic metastasis occurs. Local treatment can be mutilating, and devastating for the patient's psychological well-being.

3.1. Primary lesion

Penile carcinoma is usually a clinically obvious lesion but it may be hidden under a phimosis [24]. Physical examination should include palpation of the penis to assess the extent of local invasion and palpation of both groins to assess the lymph node status. Ultrasound (US) can provide information about infiltration of the corpora [70, 71]. Magnetic resonance imaging (MRI) with an artificially induced erection can be used to exclude corporal invasion but is very unpleasant for the patient [72, 73]. The sensitivity and specificity of MRI in predicting corporal or urethral invasion was reported as 82.1% and 73.6%, and 62.5% and 82.1%, respectively [74]. Penile Doppler US has been reported to have a higher staging accuracy than an MRI in detecting corporal infiltration [75].

3.2. Regional lymph nodes

Careful palpation of both groins for enlarged inguinal lymph nodes must be part of the initial physical examination of patients suspected of having penile cancer

5.4 Guidelines for the diagnosis and staging of penile cancer

Recommendations	Strength rating
Primary tumour	
Perform a physical examination, record morphology, extent and invasion of penile structures.	Strong
Obtain a penile Doppler ultrasound or MRI with artificial erection in cases with intended organ-sparing surgery.	Weak
Inguinal lymph nodes	
Perform a physical examination of both groins, record the number, laterality and characteristics of inguinal nodes and: • If nodes are not palpable, offer invasive lymph node staging in intermediate- and high-risk patients; • If nodes are palpable, stage with a pelvic computed tomography (CT) or positron emission tomography (PET)/CT.	Strong
Distant metastases	
In N+ patients, obtain an abdominopelvic CT scan and chest X-ray/thoracic CT for systemic staging. Alternatively, stage with a PET/CT scan.	Strong
In patients with systemic disease or with relevant symptoms, obtain a bone scan.	

Confirmation of diagnosis should be made by biopsy, which may be combined with definitive treatment. A biopsy is not necessary prior to surgical removal of obvious penile abnormalities.

CT of the groins, pelvis abdomen and thorax should be performed.
Staging is by CT, but this is unreliable with regard to inguinal lymph nodes , especially in the presence of infection. Staging investigations should not hold up treatment of the primary tumour.

IV. GRADING AND TNM CLASSIFICATION

The TNM classification for penile cancer includes tumour grade, due to its prognostic relevance (Table 9). Tumour grading in penile cancer has been shown to be highly observer-dependent and can be problematic, especially in heterogeneous tumours. Grading should use the categories specified by the WHO for penile cancer (Table 7)

Table 7: Grading recommendations for penile SCC

Feature	Grade 1	Grade 2	Grade 3
Cytological atypia	Mild	Moderate	Anaplasia
Keratinisation	Usually abundant	Less prominent	May be present
Intercellular bridges	Prominent	Occasional	Few
Mitotic activity	Rare	Increased	Abundant
Tumour margin	Pushing/well	Infiltrative/ill defined	Infiltrative/ill defir

Table 9: 2016 TNM clinical and pathological classification of penile cancer [51]

Clinical classification	
T - Primary Tumour	
TX	Primary tumour cannot be assessed
T0	No evidence of primary tumour
Tis	Carcinoma *in situ*
Ta	Non-invasive verrucous carcinoma*
T1	Tumour invades subepithelial connective tissue
T1a	Tumour invades subepithelial connective tissue without lymphovascular invasion and is not poorly differentiated
T1b	Tumour invades subepithelial connective tissue with lymphovascular invasion or is poorly differentiated
T2	Tumour invades corpus spongiosum with or without invasion of the urethra
T3	Tumour invades corpus cavernosum with or without invasion of the urethra
T4	Tumour invades other adjacent structures
N - Regional Lymph Nodes	
NX	Regional lymph nodes cannot be assessed
N0	No palpable or visibly enlarged inguinal lymph nodes
N1	Palpable mobile unilateral inguinal lymph node
N2	Palpable mobile multiple or bilateral inguinal lymph nodes
N3	Fixed inguinal nodal mass *or* pelvic lymphadenopathy, unilateral or bilateral
M - Distant Metastasis	
M0	No distant metastasis
M1	Distant metastasis
Pathological classification	
The pT categories correspond to the clinical T categories. The pN categories are based upon biopsy or surgical excision	
pN - Regional Lymph Nodes	
pNX	Regional lymph nodes cannot be assessed
pN0	No regional lymph node metastasis
pN1	Metastasis in one or two inguinal lymph nodes
pN2	Metastasis in more than two unilateral inguinal nodes or bilateral inguinal lymph nodes
pN3	Metastasis in pelvic lymph node(s), unilateral *or* bilateral extranodal or extension of regional lymph node metastasis
pM - Distant Metastasis	
pM1	Distant metastasis microscopically confirmed
G - Histopathological Grading	
GX	Grade of differentiation cannot be assessed
G1	Well differentiated
G2	Moderately differentiated
G3	Poorly differentiated
G4	Undifferentiated

**Verrucous carcinoma not associated with destructive invasion.*

V. THERAPEUTIC APPROACH

5.1 Treatment of the primary tumor

The aims of the treatment of the primary tumour are complete tumour removal with as much organ preservation as possible, without compromising oncological control. Local recurrence has little influence on long-term survival, so organ preservation strategies are justified [87]. There are no randomised controlled trials (RCTs) or observational comparative studies for any of the treatment options for localised penile cancer. Penile preservation appears to be superior in functional and cosmetic outcomes to partial or total penectomy, and is considered to be the primary treatment method for localised penile cancer. However, there are no RCTs comparing organ-preserving and ablative treatment strategies. Histological diagnosis with local staging must be obtained before using non-surgical treatments. With surgical treatment, negative surgical margins must be obtained. Treatment of the primary tumour and of the regional nodes can be staged. Local treatment modalities for small and

localised penile cancer include excisional surgery, external beam radiotherapy (EBRT), brachytherapy and laser ablation. Patients should be counselled about all relevant treatment options.

Surgery provides the mainstay of treatment, with consideration for maintenance of the cosmetic appearance and function of the penis where possible. Glansectomy (with creation of a neo-glans using split skin grafts) and partial amputation with skin grafting should be employed where possible. In men with locally advanced disease in whom radical amputation is essential, consideration should be given to referral for formation of a neo-penis at a specialist centre (Leicester or University College Hospital London).

Radiotherapy should be offered where surgery is contra-indicated or the patient is extremely averse to surgery, but in such cases the patient should be warned of the inferior cosmetic results of radiotherapy in the long-term.

Treatment strategy is as follows:

a) T1, N0: tumour limited to the glans or prepuce: local electron beam irradiation.

b) T2, N0: tumour invading the corpora or deep invasion of the shaft: irradiation of the whole shaft of the penis.

c) T3, N0: tumour invading the urethra or prostate gland: may be considered for radical radiotherapy, however large volume disease may be most appropriately managed with palliative radiotherapy.

d) T4, inoperable nodal disease: consider palliative radiotherapy.

5.2 Management of regional lymph nodes

The development of lymphatic metastases in penile cancer follows the route of anatomical drainage. The inguinal lymph nodes, followed by the pelvic lymph nodes, provide the regional drainage system of penis. The superficial and deep inguinal lymph nodes are the first regional node group to be affected, which can be uni- or bilateral

-Clinical assessment and imaging of inguinal lymph nodes are unreliable. About half of enlarged nodes will be due to infection rather than metastatic tumour. Aspiration cytology should be used to confirm metastases.

-If the nodes are clinically involved, treatment is with bilateral lymph node dissection, assuming no evidence of widespread nodal or metastatic disease. After such a procedure (and histological confirmation of nodal involvement) consideration should be given to prophylactic iliac node dissection.

-Radiotherapy is reserved for incompletely resected disease. Block dissection with post-operative radiotherapy frequently causes lymphoedema. Prophylactic post-operative radiotherapy to iliac nodes should also be considered after resection of metastatic inguinal lymphadenopathy especially when multiple nodes OFFICIAL 6 are involved or there is extracapsular spread. A dose of 45 Gy over 5 weeks is required.

-Concurrent chemotherapy using combinations such as Cisplatin + 5FU may be considered to imptove tumour control; this has not been addressed by a RCT and may increase lower limb and scrotal oedema. A boost of 20Gy in 10 fractions over 2 weeks should be considered if there is residual disease.

-If nodes are not obviously involved with tumour, bilateral prophylactic inguinal node dissection is relevant in certain cases. Patients can be stratified into low, medium or high risk depending on the primary histology:

a) low risk – Cis, ptag1/2 and pt1g1 –node dissection not recommended.

b) medium risk – T1G2 –node dissection or sentinel node biopsy may be advisable in patients with vascular and lymphatic invasion, or an infiltrating growth pattern.
c) high risk – pt2 or above, or any G3 tumours - node dissection indicated.
-Patients should be counselled regarding the risks and benefits of lymph node dissection, as the procedure is associated with significant morbidity. Dynamic sentinel node biopsy is in development in the Supra Regional centre.
-Prophylactic inguinal node irradiation remains unproven, doses of 45-50 Gy over 6 weeks are recommended where this is to be considered. Despite using lower doses than for primary disease, a risk of lymphodema remains.

5.3 Palliative radiotherapy

Megavoltage irradiation to encompass gross disease to 20Gy in 5 fractions over 1 week; repeated depending upon response and tolerance to treatment.

5.4 Carcinoma-in-situ (cis)

Glans resurfacing may be appropriate in widespread cis; non- surgical approaches using 5 F-U cream or imiquimod should be considered initially.

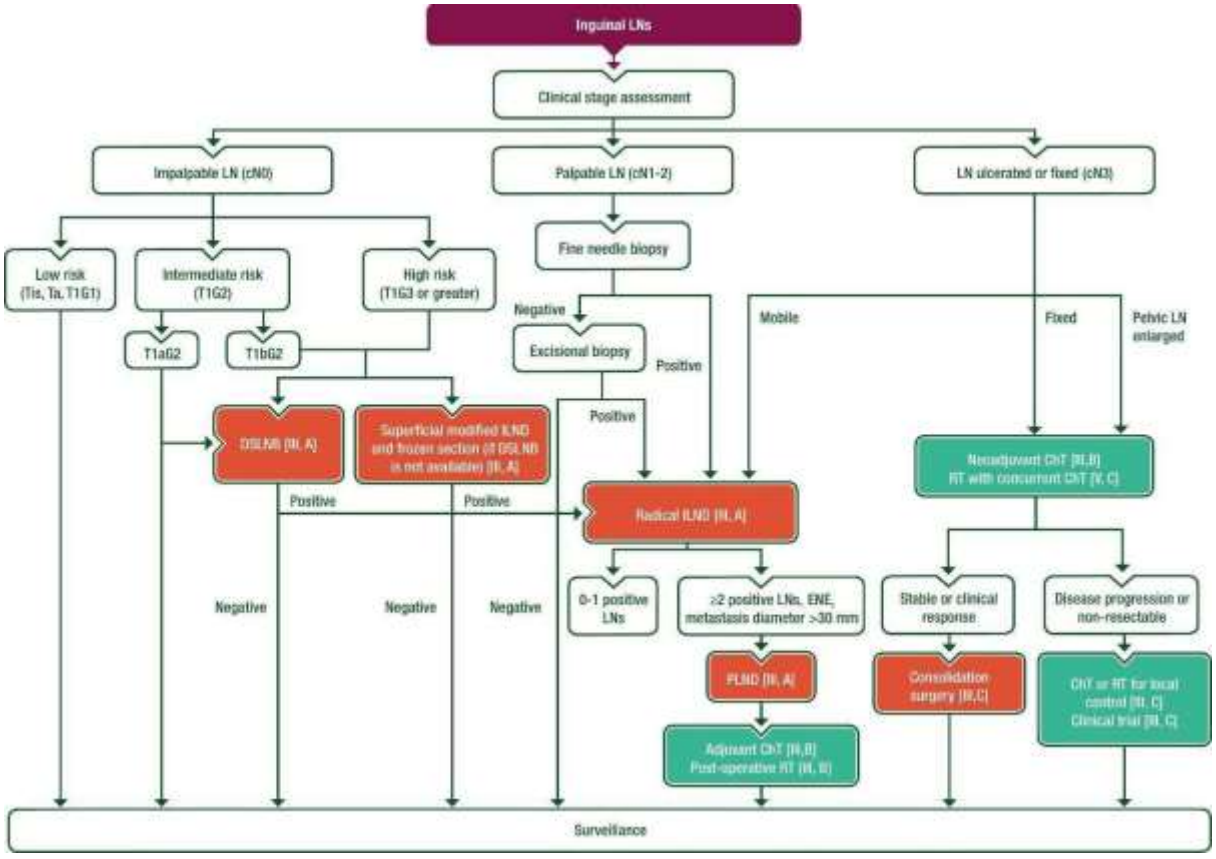
5.5 Management of patients with advanced inoperable disease

Radiotherapy and/or chemotherapy using schedules such as Cisplatin + 5 Fluorouracil. Mitomycin C + 5 Fluorouracil may be considered as an alternative if renal function is impaired.

5.6 Metastatic disease

Metastatic disease is normally treated with chemotherapy. Cisplatin + 5 Fluorouracil is the most commonly employed schedule. Mitomycin C + 5 Fluorouracil may be considered as an alternative if renal function is impaired. Palliative radiotherapy may be required for metastatic sites such as bone pelvic or para-aortic lymphadenopathy.

VI. ALGORITHM



VII. REFERENCES

The format in which to cite the EAU Guidelines will vary depending on the style guide of the journal in which the citation appears. Accordingly, the number of authors or whether, for instance, to include the publisher, location, or an ISBN number may vary.

The compilation of the complete Guidelines should be referenced as: EAU Guidelines. Edn. presented at the EAU Annual Congress Amsterdam 2022. ISBN 978-94-92671-16-5.

If a publisher and/or location is required, include: EAU Guidelines Office, Arnhem, The Netherlands. http://uroweb.org/guidelines/compilations-of-all-guidelines/

References to individual guidelines should be structured in the following way: Contributors' names. Title of resource. Publication type. ISBN. Publisher and publisher location, year

PENILE FRACTURE

Dr. HAY VANEL, Dr. OUK REAKSMEY, Prof. BOU SOPHEAP

I. CASE DEFINITION

Penile fracture is uncommon, but it is essential to promptly identify this specific urogenital injury. The majority of such injuries occur from direct blunt penile trauma during sexual intercourse. Severe blunt trauma to an erect penis results in markedly increased cavernosal pressure that, if severe enough, results in the rupture of the tunica albuginea, which is termed a penile fracture.

II. ETIOLOGY

A **penile fracture** is typically the result of direct trauma to the **penis** during sexual intercourse. In one study, 57.2% of patients with confirmed **penile fractures** reported such direct blunt trauma to the erect **penis** during intercourse. The erect **penis** may slip from the vagina and be thrust directly into the perineum or pelvic **bone**. This active thrusting results in markedly increased pressure inside the blood-filled corpus cavernosa, where the tension causes a rupture of the tunica albuginea. The tearing causes an acute loss of the erection together with immediate swelling, bruising, angulation, hematoma formation, and pain.

Additionally, there may be an associated tear of the **penile** urethra and urinary retention. The urethra is involved in about 20% of cases, and the corpora spongiosum in up to 30%.The most commonly associated sexual positions are "female superior" or "rear entry," however, one study noted that meta-analyses showed no particular sexual position had an increased risk.

Masturbation injuries and falls landing on an erect **penis** are other notable causes of **penile fractures**. Bending or angulation of the **penis** while attempting vigorous vaginal penetration, rolling over during sleep, and anal intercourse, are other reported causes. Forcefully bending the erect **penis** downwards to cause rapid detumescence (called taqaandan) is a common practice in many Middle Eastern, North African, and Central Asian cultures, which can also lead to **penile fractures**.

III. DIAGNOSTIC PROCEDURE

3.1. Clinical argument

Penile fractures often occur during intercourse. Typical historical findings associated with **penile fracture** include:[6]

- Angulation of the **penis**, typically away from the side of the injury
- An erect **penis** at the time of the trauma
- Bruising and significant swelling of the **penis** and surrounding area
- Immediate detumescence
- Pain in the genitals
- A "pop" or "shaping" sound (patient-reported)
- Trauma to the genitals

A physical exam should be comprehensive and may include the following findings:

- Angulated **penis**
- Ecchymotic shaft (an "eggplant" deformity)
- Flaccid **penis** or asymmetric erection
- Significant **penile** shaft swelling
- Tenderness of the **penis**

3.2Technical procedure

The diagnosis of **penile fractures** is typically made clinically by direct examination. The **penis** will often demonstrate a classic "eggplant deformity" and will tend to deviate away from the side of the rupture. The presence of a possible urethral injury may not be readily apparent.

Blood at the urethral meatus, hematuria, and difficulty voiding should prompt an assessment for a urethral injury. The American Urological Association guidelines recommend provocative testing with the intent to rule out a urethral injury if there is a suspicion that this may be the case. This testing could either be an intraoperative cystoscopy or a retrograde urethrogram. The European Association of Urology guidelines are aligned with these recommendations but suggest intraoperative cystoscopy over preoperative retrograde urethrography since retrograde urethrograms have a higher false positive rate and may delay access to the operating room.

The workup surrounding **penile fractures** should include preoperative laboratory evaluation, and other studies to rule out concomitant urethral injury may be warranted. Suspicion of a **penile fracture**, based on history alone, should warrant a thorough evaluation to rule out related injuries, including dorsal **penile** vein and nerve damage, while simultaneously diagnosing the **penile fracture**. In addition to clinical suspicion for a tunica rupture, multiple imaging modalities are available which can be useful to identify **penile fractures** and diagnose urethral injuries.

Ultrasound is readily available in most emergency centers, can be performed quickly, and is generally recommended; however, there is some controversy over its true clinical utility as the actual test is very operator-dependent, and successful identification of a **penile** corporal injury requires specific expertise. The ultrasound may show irregular defects at the site of a cavernosal rupture. However, if there is a significant hematoma (which is common in these cases), it may increase the difficulty of diagnosing a tunica rupture exclusively by ultrasound.

Although CT is widely available and has been demonstrated to be helpful in the identification of the exact location and size of injury to aid in surgical repair, it exposes the patient to radiation (particularly to the genitals) and incurs extra cost. Additionally, the test rarely affects the surgery and delays the patient's arrival at the operating room.

MRI, although not the quickest, cheapest, or most readily available test, has been shown to assist in the diagnosis and perioperative management of **penile fractures**. One study demonstrated 100% sensitivity along with 77.8% specificity for the identification of tunica ruptures in the **penis** by MRI.

Cavernosography can be useful and definitive; however, like MRI and CT

imaging, it is generally reserved for those unusual cases where the clinical presentation and physical examination are not adequate for a diagnosis, and a more detailed examination is required.

IV. DIFFERENTIAL DIAGNOSIS

- Anasarca
- Coagulation disorders
- Dorsal vein rupture
- Dependent **penile** edema
- Paraphimosis
- Pelvic trauma
- **Penile** cellulitis
- **Penile** contusion
- Priapism
- Thrombocytopenia
- Urethral tear

V. THERAPEUTIC APPROACH

Treatment of penile fractures should be a prompt operative repair of the defect. Preoperative antibiotics should be given.

An initial linear incision is acceptable for the opening of the skin, but many urologists prefer a circumcising subcoronal incision that allows degloving of the **penile** shaft skin. This provides optimal surgical exposure and avoids leaving a longitudinal suture line along the ventral **penile** shaft. Access to injuries at the base of the **penis** may also be accomplished by a midline penoscrotal incision which avoids a full degloving of the shaft.

After opening the skin, the hematoma should be evacuated as completely as possible. The hematoma should be removed, allowing direct and complete visualization of the tunica for any tears or defects. These are typically seen on the ventral surface of the tunica, starting at its juncture with the urethra and extending axially.

The urethra should also be carefully examined even if the preoperative evaluation did not suggest a urethral injury. If there is any doubt, an intraoperative flexible cystoscopy should be performed.

The placement of a Foley catheter at the time of surgery is recommended even in cases where no urethral injury is found or suspected. This stabilizes the corpora and serves as a useful surgical landmark. The catheter is usually removed the day after surgery in patients without urethral damage.

An absorbable suture (polyglactin or polydioxanone, size 00 is the most frequently selected) is used to repair the tunica defect. Interrupted sutures are typically used for strength. Lateral sutures at the edges of the defect are optional. Some surgeons will bury the knots from the tunica repair to further minimize postoperative scarring.

A solution of indigo carmine and normal saline can be injected directly into the

corpora cavernosa or through the glans into the corpora spongiosum to evaluate the integrity of the repair and to inspect the area for any unrecognized injuries or leaks. A tourniquet should be placed around the base of the **penis** immediately before such intraoperative test injections.
Buck's fascia should also undergo repair if it appears damaged. A torn Buck's fascia can result in a scrotal hematoma. Such a hematoma should be surgically evacuated and drained.

This surgical repair should be performed promptly, as delays cause increased extravasation, poorer healing, increased fibrosis, and more complications. Multiple studies have proven a significant change in functional outcomes with delayed repairs of **penile fractures**. One study showed that a delay of approximately 8 hours resulted in substantial increases in erectile dysfunction postoperatively.

The one exception to the standard recommendation to perform urgent surgical repair of a **penile fracture** may be those cases of corporal tearing that follow the administration of clostridium histolyticum collagenase plaque injections for treating Peyronie disease. In such cases, the recommendation is to treat the injury conservatively if the urethra has not been injured, as the integrity of the tunica has been compromised by the collagenase.

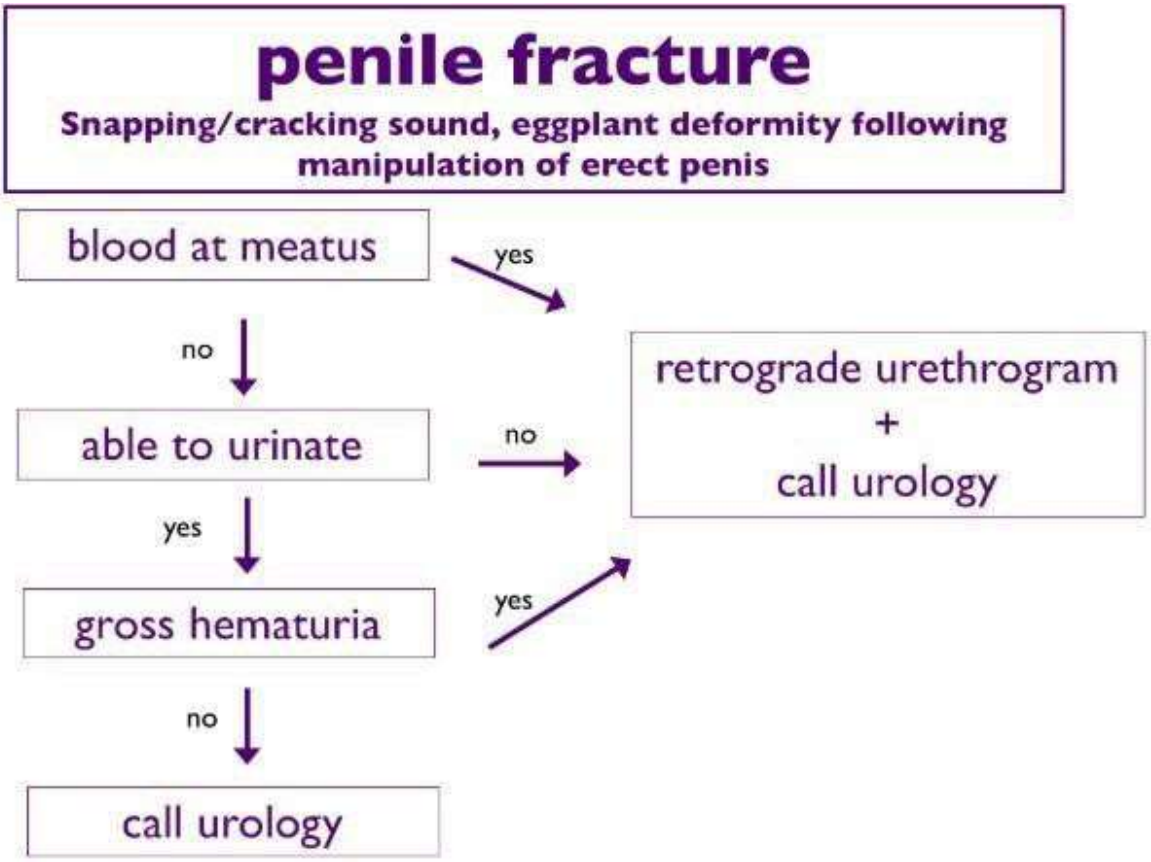
Urethral tears should generally be repaired primarily with smaller, absorbable sutures in a tension-free manner over a Foley catheter. The catheter should remain in place for at least two weeks, and a periurethral urethrogram should be performed before Foley removal.

Postoperatively, patients should receive routine postsurgical care instructions, including incisional care and information regarding indications to return to the emergency department. Patients should be instructed to refrain from intercourse during the postoperative period as well.

VI. COMPLICATION RENAL FAILURE

- Abscess formation
- Curvature of the **penis**
- Erectile dysfunction
- Extravasation
- Fibrosis and plaque formation
- Fistula formation (arteriovenous, corporourethral, urethrocutaneous, etc)
- Hematuria
- Nodule formation at the injury site
- Painful erections
- Painful intercourse
- Urethral stricture
- Urinary retention
- Urinoma formation
- Weak urinary stream

VII. ALGORITHM



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PHIMOSIS IN INFANTS AND ADULTS

Bou Sopeap, Pen Monyrath, Hay Vanel

I. INTRODUCTION

Phimosis is a condition frequently encountered in pediatric and adult urology. The incidence of physiological phimosis is high in newborns, with approximately 96% of male infants born with a non-retractable foreskin. As development progresses, spontaneous resolution occurs in the majority of cases by adolescence. In contrast, phimosis in adults, particularly pathological phimosis, can lead to significant discomfort, infections, and sexual dysfunction. This article explores the natural history, clinical implications, and management of phimosis across these two distinct age groups. It is important to distinguish between physiological phimosis, which is common in young children, and pathological phimosis, which may occur later in life due to infection, scarring, or other medical conditions. While physiological phimosis typically resolves without intervention, pathological phimosis often requires treatment.

II. ETIOLOGY AND PATHOPHYSIOLOGY

Infants:

Phimosis in infants is considered a normal physiological condition. At birth, the foreskin is adhered to the glans penis by epithelial connections, and complete retractability is rare. Over time, these adhesions gradually break down due to normal penile growth and erections. By the age of 3, approximately 50-70% of boys are able to retract their foreskin, and this number increases to 90% by age 10-12. This process is benign and typically does not require intervention unless complications such as balanoposthitis (inflammation of the glans and foreskin) arise.

Adults:

In adults, phimosis is most commonly pathological, meaning it results from a disease process rather than normal development. The primary causes include:

i. Chronic inflammation: Recurrent infections such as balanitis or sexually transmitted infections can lead to fibrosis and scarring of the foreskin.
ii. Lichen sclerosus (balanitis xerotica obliterans): A chronic skin condition leading to sclerotic changes in the foreskin, causing narrowing and non-retractability.
iii. Trauma or forceful retraction: Injuries to the foreskin can result in scar tissue formation, further contributing to phimosis.

- Commonly seen in infants and young children, it is a normal developmental condition where the foreskin is adherent to the glans. Most cases resolve naturally with age.

Pathological Phimosis:

- Occurs in older children and adults due to scarring, infection, or inflammation that causes the foreskin to become fibrotic and non-retractile.

Etiology:

1. Physiological Phimosis:

- Present at birth and resolves spontaneously in most children by age 3-5 years.
- Adhesions between the inner foreskin and the glans are naturally present in newborns and gradually separate over time.

2. Pathological Phimosis:

- **Infections:** Recurrent episodes of balanitis (inflammation of the glans) or balanoposthitis (inflammation of both the glans and foreskin) can lead to scarring and fibrosis.
- **Balanitis Xerotica Obliterans (BXO):** A chronic inflammatory condition that causes white patches and fibrosis of the foreskin and glans.
- **Trauma:** Forced or premature retraction of the foreskin in children can lead to scarring and the development of pathological phimosis.
- **Diabetes mellitus:** Adults with poorly controlled diabetes are at higher risk for recurrent infections that can cause phimosis.
- **Poor hygiene:** Inadequate cleaning under the foreskin can lead to infection and inflammation, contributing to pathological phimosis.

Pathophysiology:

- Physiological Phimosis: In newborns, the foreskin is normally adherent to the glans and becomes more retractable over time as natural desquamation of epithelial cells occurs and the adhesions break down. By age 3-5, most boys will have a retractable foreskin.

- Pathological Phimosis: Chronic inflammation, infection, or trauma leads to fibrosis and thickening of the foreskin, forming a non-retractile fibrotic ring. This fibrosis prevents the foreskin from retracting over the glans and can result in complications such as difficulty urinating or pain during erection.

III. CLINICAL PRESENTATION

Infants:

In infants and young boys, physiological phimosis is generally asymptomatic. Parents may notice an inability to retract the foreskin during hygiene routines, but unless there are signs of infection or discomfort, this is not concerning. Complications in rare cases include balanitis or urinary tract infections, which may prompt a clinical evaluation.

Adults:

Adult men with phimosis often present with symptoms that may affect sexual, urinary, and general well-being. These symptoms include:

i. Painful erections
ii. Difficulty with hygiene, leading to foul odor and infections

iii. Dysuria (painful urination)
iv. Paraphimosis (trapped foreskin behind the glans, causing swelling)
v. Sexual dysfunction due to pain or difficulty during intercourse

1. Inability to retract the foreskin: The hallmark of phimosis. In children, it is often asymptomatic, while in adults, it can cause significant discomfort.

2. Ballooning of the foreskin during urination: In both children and adults, the foreskin may balloon when urinating, as urine gets trapped beneath the non-retractile foreskin.

3. Pain or discomfort during urination: Especially if there is associated infection or inflammation.

4. Recurrent infections: Patients may experience repeated episodes of balanitis or urinary tract infections due to poor hygiene under the foreskin.

5. Pain during erection (in adolescents and adults): Tight foreskin may cause pain, particularly during sexual activity.

6. Paraphimosis: A complication where the retracted foreskin becomes stuck behind the glans and cannot return to its normal position, leading to swelling and pain.

IV. DIAGNOSIS

Diagnosis of phimosis is primarily clinical, based on physical examination. In infants, observation of retractability over time is usually sufficient, while in adults, further investigation may be necessary to determine underlying causes, particularly in cases of chronic infections or suspected dermatological conditions like lichen sclerosus.

Classification: Phimosis can be classified as:

i. Physiological (Developmental): Normal in infants and children.
ii. Pathological: Typically in adults, due to scarring or disease.

The Kikiro's classification of phimosis grades the severity from grade 0 (completely retractable foreskin) to grade 5 (absolutely no retraction).

1. Clinical History:

- Obtain a history of symptoms, including any episodes of infection, trauma, or difficulty urinating.
- In adults, ask about discomfort during sexual activity or erections.

2. Physical Examination:

- Inspect the foreskin and attempt gentle retraction to assess the degree of phimosis.
- Look for signs of infection, such as redness, swelling, or discharge, and check for any scarring or fibrosis of the foreskin.

3. Additional Investigations:

- Not routinely required unless there is suspicion of underlying systemic conditions

., diabetes).

- **Blood** glucose **levels** should be checked in adults with recurrent infections or in cases associated with diabetes.
- **Urine analysis** to rule out urinary tract infections.

Kikiros Classification of Phimosis

The Kikiros classification is used to categorize the degree of phimosis, which refers to the inability to retract the foreskin over the glans penis. It helps in clinical decision-making regarding the management of phimosis, particularly in children. **The classification ranges from grade 0 to grade 5:**

1. Grade 0: Fully retractable foreskin with no scarring.

2. Grade 1: Full retraction with a ring of tightness, but without causing pain or difficulty.

3. Grade 2: Partial retraction with some restriction in movement due to a fibrotic ring, allowing partial exposure of the glans.

4. Grade 3: The foreskin can only retract enough to reveal the external urethral meatus (opening), but no further.

5. Grade 4: No retraction of the foreskin is possible, and the preputial opening is very narrow, allowing only the passage of urine.

V. MANAGEMENT

Infants:

The management of physiological phimosis in infants is generally conservative:

i. Observation: In most cases, no treatment is required, and parents should be advised not to forcefully retract the foreskin.
ii. Topical corticosteroids: For persistent cases or mild symptomatic phimosis, topical corticosteroids such as 0.05% betamethasone can help loosen the foreskin and allow gradual retraction.
iii. Surgical intervention: Circumcision is rarely indicated in infants unless there are recurrent infections, urinary obstruction, or significant discomfort. Other surgical options, such as preputioplasty (foreskin-preserving surgery), may be considered. The surgical is done under general anesthesia.

Adults:

In adults, the treatment depends on the underlying cause and severity:

i. Topical treatments: Corticosteroids may be effective in early stages of pathological phimosis, especially in conditions like lichen sclerosus.
ii. Antibiotics or antifungals: In cases where infections contribute to phimosis, appropriate antimicrobial therapy is essential.
iii. Circumcision: This is the most definitive treatment for pathological phimosis. It provides permanent resolution by removing the foreskin and eliminating the possibility of future narrowing. Most procedure of adult could be done by local anesthesia including penile block.
iv. Dorsal slit or preputioplasty: These are alternative procedures to circumcision, especially for patients who wish to preserve the foreskin. They involve making incisions to relieve tightness without complete removal.

1. Conservative Management (especially for Physiological Phimosis in children):

- Observation: Physiological phimosis in children often resolves spontaneously by age 3-5. No treatment is needed unless the child experiences infections or other

complications.

- Topical corticosteroids: Application of a corticosteroid cream (e.g., betamethasone 0.05%) to the foreskin for 4-6 weeks can soften the skin, reduce inflammation, and promote gentle retraction.

- Gentle stretching: After using corticosteroid cream, parents may be advised to gently retract the foreskin regularly to promote loosening.

2. Medical Treatment:

- Antibiotic or antifungal creams: Used to treat any underlying infection, particularly in cases of recurrent balanitis or balanoposthitis.

3. Surgical Management:

- Circumcision: This is the definitive treatment for pathological phimosis. The foreskin is completely removed, eliminating the problem.

- Preputioplasty: A less invasive alternative to circumcision, where a small incision is made to widen the foreskin, allowing it to retract more easily. This procedure preserves the foreskin and may be preferred by some patients.

- Dorsal slit: A temporary solution in cases of acute paraphimosis where the foreskin is trapped behind the glans and cannot be repositioned.

4. Management of paraphimosis:

- Paraphimosis is a medical emergency requiring immediate manual reduction. If manual reduction fails, a dorsal slit or circumcision may be necessary.

Management Summary:

- Grade 0-1: No treatment necessary, regular hygiene.
- Grade 2-3: Conservative treatment with topical steroids (betamethasone) and regular foreskin stretching.
- Grade 4-5: Surgical intervention is often required, ranging from preputioplasty to circumcision.

VI. COMPLICATIONS

If left untreated, pathological phimosis can lead to recurrent infections, pain, and potentially more serious complications like paraphimosis or an increased risk of penile cancer (though this is rare).

Untreated pathological phimosis in adults can lead to significant complications, including:

i. Paraphimosis: A medical emergency where the retracted foreskin cannot return to its original position, leading to glans ischemia.
ii. Recurrent infections: Both balanitis and urinary tract infections may recur due to poor hygiene or trapped secretions under the foreskin.
iii. Sexual dysfunction: Pain during intercourse and psychological distress are common among adult males with severe phimosis.

In infants, complications are rare, but repeated infections or difficulty in urination may prompt early intervention.

VII. PROGNOSIS

The prognosis for infants with physiological phimosis is excellent, with most cases resolving spontaneously without the need for medical or surgical intervention. In contrast, the prognosis for adults depends on timely intervention. Pathological phimosis responds well to treatment but may lead to irreversible complications if left untreated.

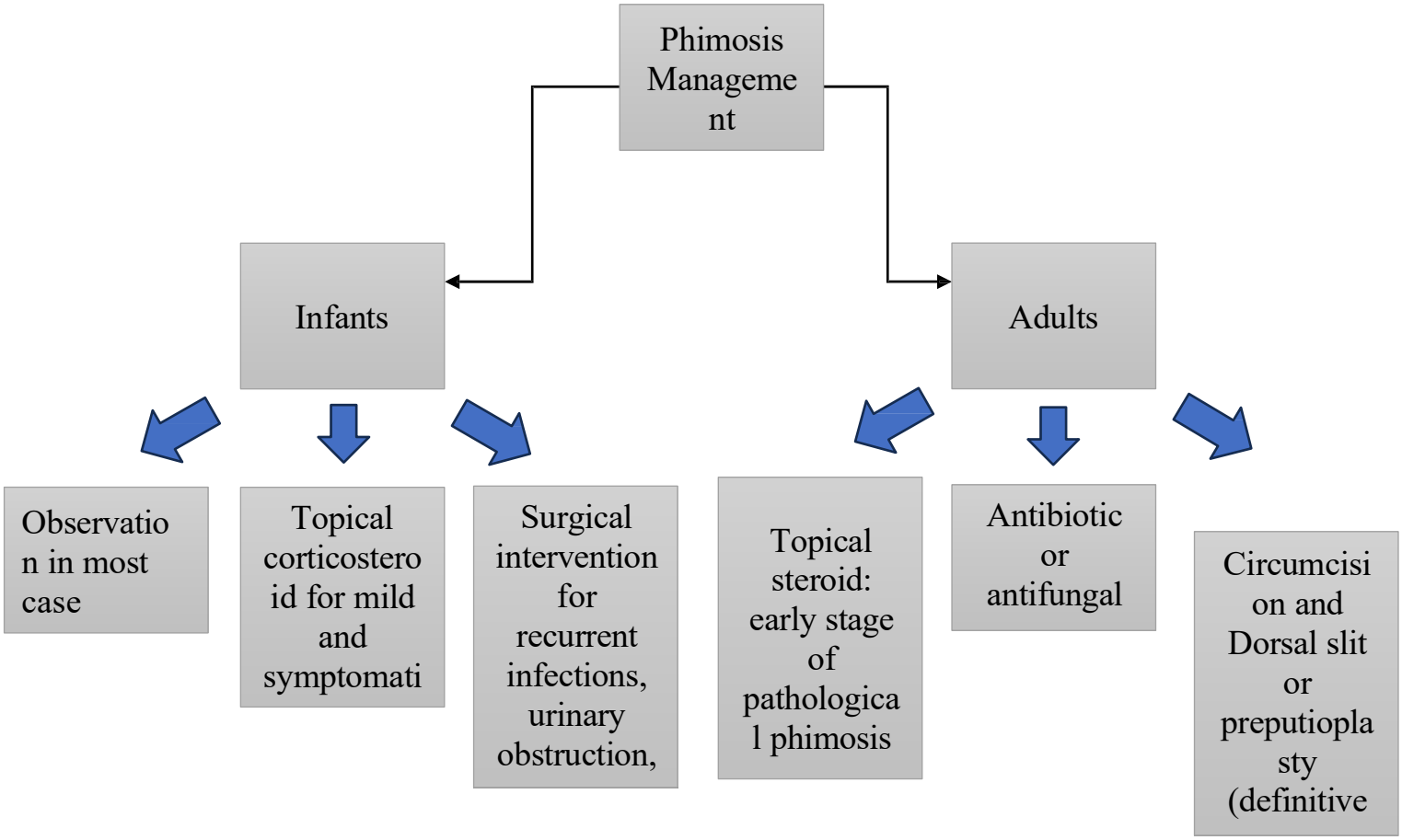
- Physiological Phimosis: The prognosis is excellent. Most cases resolve naturally by the time the child reaches 3-5 years of age.

- Pathological Phimosis: With appropriate treatment, the prognosis is generally good. Topical steroids and surgical intervention (if needed) usually result in resolution of symptoms.

VIII. CONCLUSION

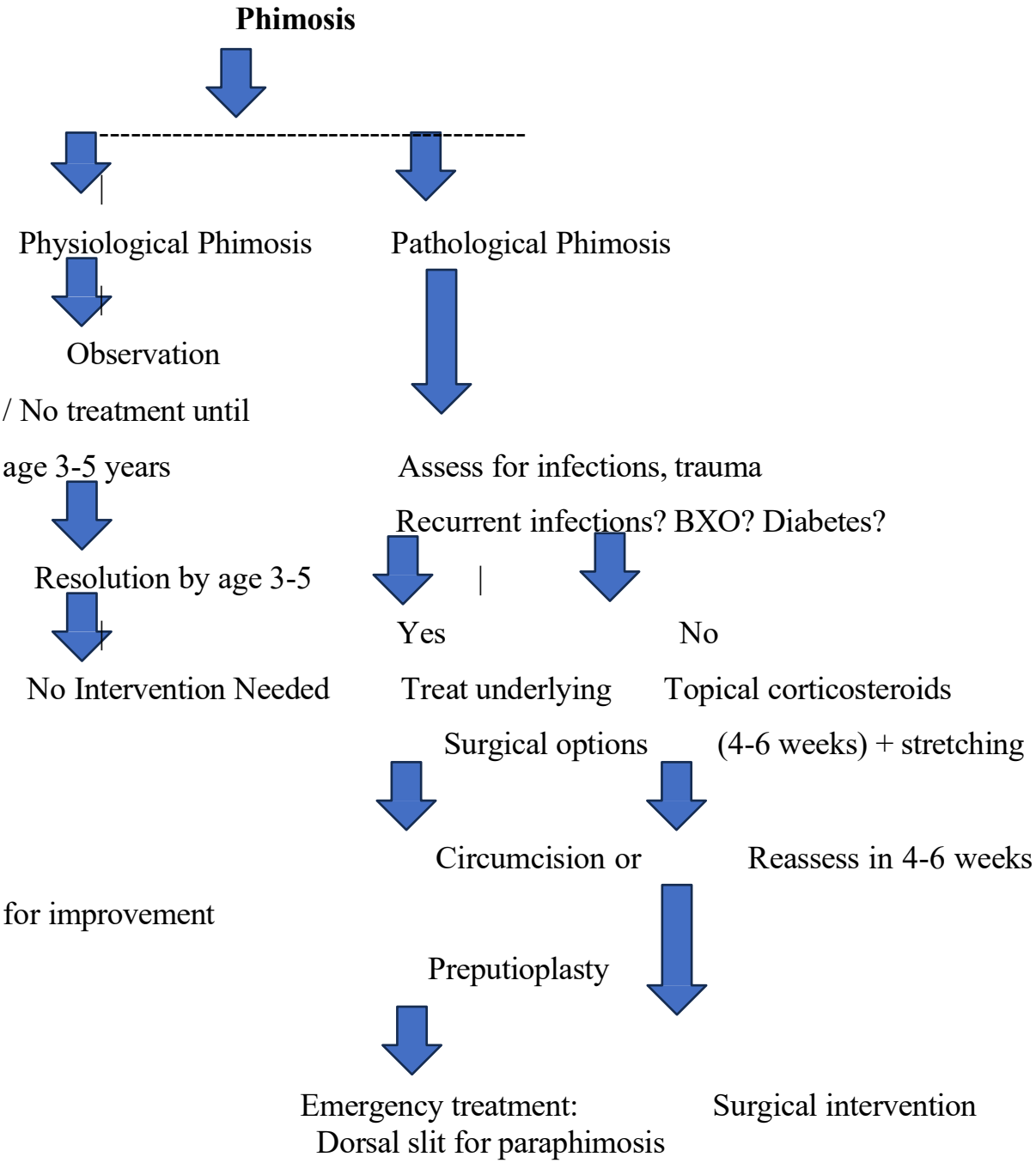
Phimosis presents differently in infants and adults, and the management strategies must be tailored to each group. While the majority of infant cases are physiological and self-resolving, adult phimosis is typically pathological, requiring more proactive treatment to prevent complications. A proper understanding of the etiology, clinical presentation, and treatment options ensures that patients receive appropriate care at all stages of life.

Phimosis is a common condition that presents differently in children and adults. Physiological phimosis in children is generally harmless and resolves with time, while pathological phimosis can cause significant symptoms and complications in older children and adults. Early diagnosis and appropriate management—whether conservative or surgical—are essential for preventing complications and improving quality of life. Surgical options, such as circumcision or preputioplasty, provide definitive resolution in most cases.

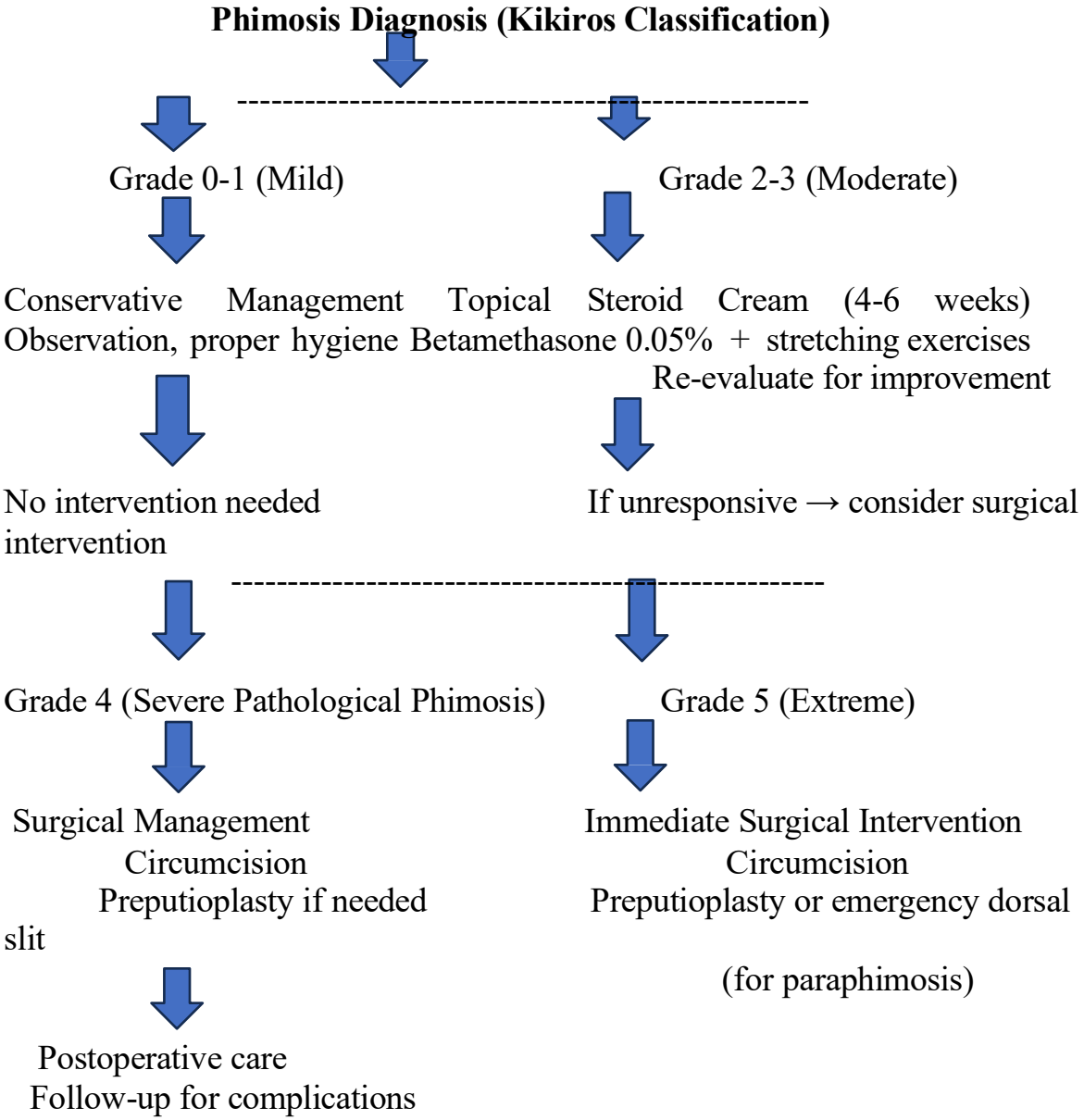


1. **ALGORITHM No: 1**

Algorithm for phimosis management No. 2



Algorithm for Management of Phimosis Using Kikiros Classification No. 3



2. **Annex:** Kikiros classification of phimosis severity: grade 0 = full retractability; grade 1 = full retraction but tight behind glans; grade 2 = partial exposure of glans; grade 3

= partial retraction, meatus just visible; grade 4 = slight retraction, but some distance between tip and glans, i.e., neither meatus nor glans can be exposed; grade 5 = absolutely no retraction.

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POSTERIOR URETHRAL VALVES (PUV)

Dr. OUK REAKSMEY, Dr. HAY VANEL, Dr. PEN MONYRATH, Prof. BOU SOPHEAP

I. CASE DEFINITION

PUV are derived from an abnormal congenital membrane arising from the verumontanum and attaching obliquely to the anterior urethra (beyond the external urethral sphincter), resulting in lower urinary tract obstruction. An alternative term is COPUM or congenital obstructive posterior urethral membrane. Urethral instrumentation or spontaneous partial rup- ture of the membrane is thought to cause the classical appearance of two valve-like folds in the prostatic urethra.

II. ETIOLOGY

PUV may arise through an abnormal insertion of the Wolffian ducts into the urogenital sinus during fetal development.

III.IAGNOSTIC PROCEDURE

Presentation

Prenatal USS: the majority are diagnosed prenatally, with 60% identified on USS at 20 weeks. They account for 1% of cases of antenatal hydro- nephrosis. Features include: bilateral hydroureteronephrosis, dilated and thick-walled bladder, dilated posterior urethra (keyhole sign), thick-walled bladder, oligohydramnios (reduced amniotic fluid), and renal dysplasia. Early diagnosis is associated with poor prognosis.

Newborn and infants: respiratory distress secondary to pulmonary hypo- plasia, palpable abdominal mass (hydronephrotic kidneys or distended bladder), ascites, UTI sepsis, electrolyte abnormalities (renal impairment), failure to thrive.

Older children: milder cases may present later with recurrent UTI, poor urinary stream, incomplete bladder emptying, poor growth and incontinence. There is a risk of renal failure, VUR, and voiding dysfunction (over- or underactive bladder), also described as ‘valve bladder syndrome’.

Associated features: ‘pop-off valve syndrome’ is seen in 20%. It describes mechanisms by which high urinary tract pressure is dissipated to allow normal renal development. It includes leaking of urine from a small blad- der or renal pelvis rupture (urinary ascites), unilateral reflux into a non- functioning kidney (VUR with renal dysplasia or VURD), and formation of bladder diverticuli.

IV. DIFFERENTIAL DIAGNOSIS

- Anterior urethral valves
- Urethral stricture disease
- Detrusor sphincter dyssynergy
- Diurnal urinary incontinence
- Pediatric renal insufficiency

V. THERAPEUTIC APPROACH

Commence prophylactic antibiotics immediately (trimethoprim 2mg/kg daily) and drain the bladder with a paediatric feeding tube or suprapubic catheter if this proves difficult. Check serum electrolyes and arrange for urgent post-natal renal tract USS and MCUG.

Definitive treatment is with cystoscopy and transurethral ablation of the valve. The most important incision is made at the 12 o'clock position with either cold knife or electrocautery. Complications of surgery include urethral strictures. A temporary cutaneous vesicostomy is indicated (com- municating stoma between the bladder dome and suprapubic abdominal wall, allowing free drainage of urine) when the urethra is too small for the resectoscope. Alternatives are ureterostomy drainage with valve ablation performed at a later stage. Any underlying bladder dysfunction should be diagnosed and treated.

VI. COMPLICATION RENAL FAILURE

Pulmonary hypoplasia secondary to intrauterine renal dysfunction and oligohydramnios is the primary cause of patient death.Other complications of PUV are generally secondary to chronic bladder changes, leading to elevated detrusor pressures. This, in turn, leads to progressive renal damage, infection, and incontinence.

Renal insufficiency

Historically, of patients with adequate pulmonary function, approximately 25% died of renal insufficiency in the first year of life, 25% died later in childhood, and 50% survived to adulthood with varying degrees of renal function. Today, with the advent of better techniques in the treatment of pediatric renal insufficiency, most of these children can be expected to survive.

The goal of treatment is to preserve the maximal obtainable renal function for each patient. This entails aggressive treatment of infections and bladder dysfunction.

Certain risk factors for progression of PUV have been identified. Elevated nadir creatinine, defined as greater than 1 mg/dL, measured during the first year of life has been identified as a risk factor for development of future renal insufficiency. Additionally, bladder dysfunction with poor compliance, elevated leak point pressures, and the need for CIC have been identified as predictive of eventual renal deterioration.

Vesicoureteral reflux

VUR (see the image below) is commonly associated with PUVs and is present in as many as one third of patients. In most children, VUR is believed to be due to an abnormal insertion of the ureter into the bladder. When associated with PUV, reflux is generally secondary to elevated intravesical pressures. Therefore, treatment of VUR in patients with PUVs involves reducing intravesical pressures by using anticholinergics, timed voiding, double voiding, CIC, and, at times, bladder augmentation.

Note irregular trabeculated bladder and high-grade vesicoureteral reflux.

Urinary tract infections

Recurrent UTIs are common in patients with PUVs. Elevated intravesical pressures predispose patients to infection, possibly by altering urothelial blood flow.

Additionally, patients with PUV may have elevated postvoid residual urine volumes, leading to stasis of urine. Dilated upper urinary tracts, with or without VUR, further elevate UTI risk.

UTI management is directed at lowering bladder pressures (anticholinergic medication), lowering postvoid residual urine volume (via CIC), and, at times, administering prophylactic antibiotics.

Urinary incontinence

The same factors that lead to VUR and UTI also lead to urinary incontinence. Correct management of bladder function depends on adequate bladder evaluation with urodynamic studies. Lowering bladder pressure, improving bladder compliance, and minimizing postvoid residual urine volume contribute to attainment of urinary continence. In some, bladder augmentation may be needed.

VII. FOLLOW UP

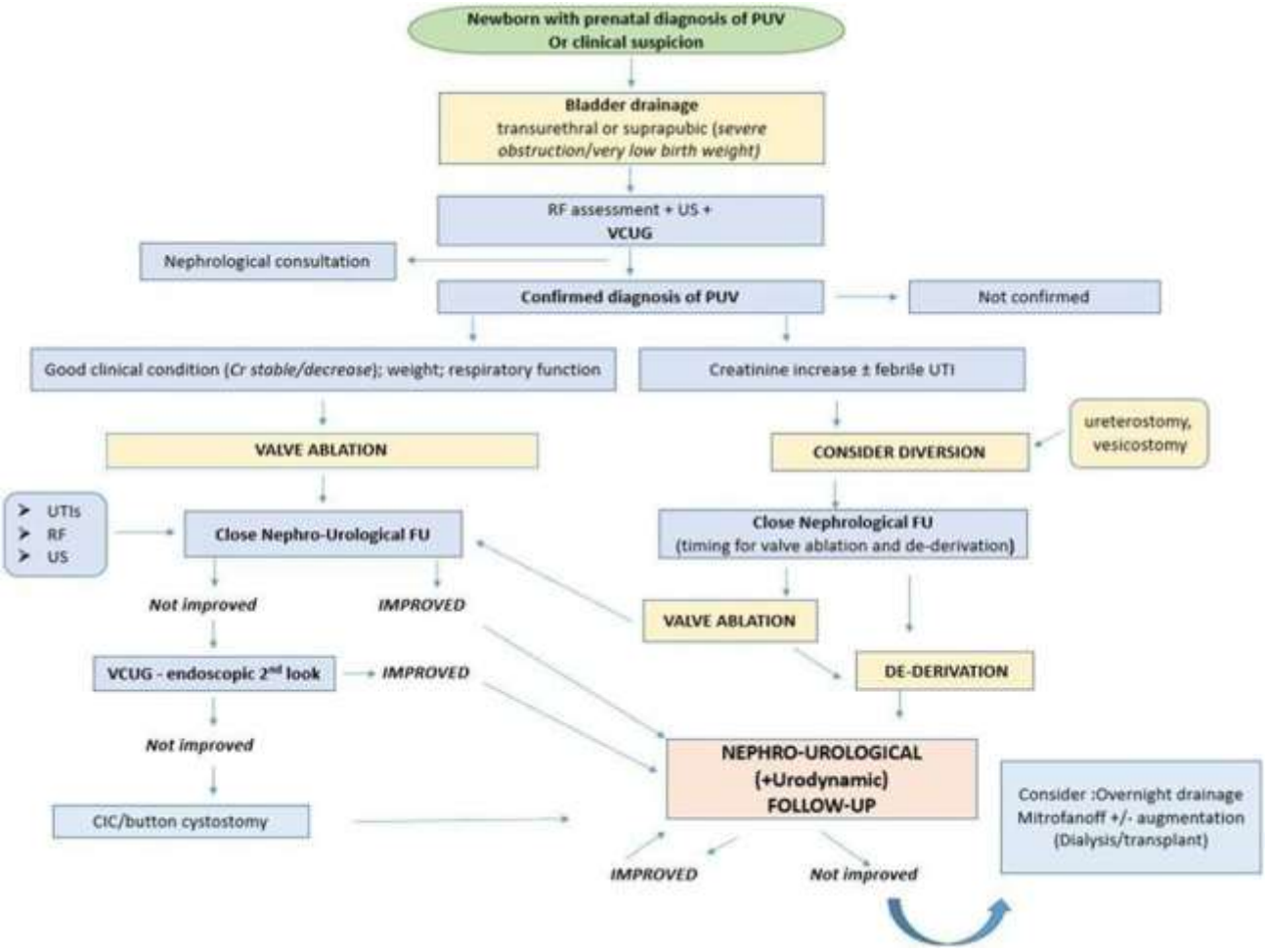
Long-term monitoring

Monitor children for linear growth (height, weight, and head circumfer- ence), renal function, BP, urine analysis (for proteinuria, osmolality), USS, and formal GFR with chromium EDTA. Renography (MAG3 and DMSA) are also performed to assess split renal function and look for evidence of obstruction or reflux. Videourodynamic studies are used to assess and aid in the management of any associated voiding dysfunction.

Prognosis

Thirty-five percent have long-term poor renal function; 20% develop end-stage renal failure. Bladder dysfunction is common despite treatment of outflow obstruction. This includes bladder overactivity, incontinence, and bladder underactivity associated with chronic urinary residuals and poor concentration of urine (with polyuria). From age 16y, care should be transferred to an adult urologist or nephrologist. Problems may arise with retrograde ejaculation, impotence and reduced libido (related to renal impairment), and abnormal prostatic or seminal vesicle secretions, con- tributing to reduced fertility.

VIII. ALGORITHM



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PRIAPISM

Dr. Sotheara Khy, Prof.Sopheap Bou

I. DEFINITION

Priapism is a pathological condition representing a true disorder of penile erection that persists more than 4 hours and is beyond, or is unrelated to sexual interest or stimulation. Erections lasting up to 4 hours are defined by consensus as 'prolonged'. Priapism may occur at all ages.

Pathophysiology and Classification

Ischemic priapism (low flow or veno-occlusive) : most common > 95%

- Imbalance in the vasoconstrictive and vasorelaxatory mechanisms governing penile erection.
- Persistent rigid erection and Penile pain
- Cavernous blood gases : pO2 <30 mmHg, pCO2 >60 mmHg, and pH<7.25): blood is hypoxic and dark in color (hypoxia, hypercapnia and acidosis)
- Color duplex ultrasonography: minimal or absent blood flow.
- Emergency

Histological Changes

- 12H: corpora specimens show interstitial edema, progressing to destruction of sinusoidal endothelium
- 24H: exposure of the basement membrane and thrombocyte adherence
- 48H: thrombus can be found in the sinusoidal spaces and smooth muscle necrosis

Arterial priapism (high flow or non-ischemia)

- A nonsexual, persistent erection caused by unregulated cavernous arterial inflow.
- Corpora are tumescent but not rigid and the penis is not painful.
- History of blunt trauma to the penis or an iatrogenic needle injury is the commonly.
- Cavernous blood gases : Blood is oxygenated and red (Similar to normal arterial blood : pO2 >90 mmHg, pCO2 <40 mmHg, and pH of 7.40)
- Color duplex ultrasonography: blood flow is normal to high in velocity.
- Nonischemic priapism does not require emergent treatment.

Stuttering (recurrent or intermittent) priapism

- Recurrent unwanted and painful erections in men with sickle cell disease.
- Recurrent priapism is a form of ischemic priapism, which starts off with erections of short duration. The onset is usually during sleep with persistence upon waking.
- Dysregulation of nitric oxide and phosophodiesterase-5 has been put forward as a possible mechanism.
- Cavernous blood gases : blood is hypoxic and dark in color (pO2 <30 mmHg, pCO2 >60 mmHg, and pH<7.25).
- Patients seek medical help when the discomfort interferes with daily life or when they develop a prolonged episode of ischemic priapism requiring

emergency medical intervention.

II. EPIDEMIOLOGY

Epidemiological data are rarely. The study carried out between 2007 and 2008 in the emergency services of the United States reports 4175 cases of priapism and concluded with an incidence of 6.5 cases per 100,000 inhabitants per year. The average age of the patients was 36.4 years. Depending on the ethnic origin of the populations, 10 to 20% were Sickle cell disease. In an older study, the cause of priapism was most often unknown, associated with taking a drug or stupefacient in 21% of cases and secondary to perineal or penile trauma in 12% of cases.

III. ETIOLOGY

Hematologic

- Sickle cell disease
- Thalassemia
- Granulocytic leukemia
- Myeloid leukemia
- Lymphocytic leukemia
- Multiple myeloma
- Hemoglobin Olmsted variant
- Fat emboli associated with hyperalimentation
- Hemodialysis
- Glucose-6-phosphate dehydrogenase deficiency

Genitourinary

- Straddle injury
- Coital injury
- Pelvic trauma
- Kick to penis or perineum
- Arteriovenous or arterio-cavernous bypass surgery
- Urinary retention

Recreational drugs : Alcohol, cocaine, crack cocaine, marijuana

Neurologic

- Syphilis
- Spinal cord injury
- Caudal equina compression
- Autonomic neuropathy
- Lumbar disk herniation
- Spinal stenosis
- Cerebral vascular accident
- Brain tumor
- Spinal anesthesia
- Cauda equina syndrome

Neoplastic : Sarcoma, Secondaries, Myeloma, Lymphoma, Penis, Prostate, urethra, testis, bladder, rectum, lung, kidney cancer

Hormones : Gonadotropin-releasing hormone, testosterone

Infectious (Toxin-mediated) causes : Scorpion sting, spider bite, rabies, malaria, prostatitis, urethritis, Mumps, Syphilis

Metabolic condition : Amyloidosis, fabry disease, gout, diabetes, nephrotic syndrome, renal failure, Hemodialysis

ED pharmacotherapy : Oral sildenafil, Intraurethral alprostadil, Intracavernous agents, Papaverine, phentolamine, oral phosphodiesterase type 5 inhibitors

Pharmacologic causes

- Alpha adrenergic receptor antagonists: Prazosin, terazosin, doxazosin, tamsulosin
- Antianxiety agent: Hydroxyzine
- Anticoagulants : Heparin, warfarin
- Antihypertensives : Hydralazine, guanethidine, propranolol
- Antidepressants and antipsychotics : Trazodone, bupropion, fluoxetine, sertraline, lithium, clozapine, risperidone, olanzapine, chlorpromazine, thioridazine, phenothiazines
- Attention-deficit/hyperactivity disorder agents : Methylphenidate, atomoxetine

IV. PREVENTION

If you have recurrent or stuttering priapism, to prevent future episodes your doctor might recommend :

- Treatment for an underlying medical condition : sickle cell anemia, that might have caused priapism
- Use of oral or injectable phenylephrine
- Hormone-blocking medications-only for adult men
- Use of oral medications used to manage erectile dysfunction

V. DIAGNOSIS AND TREATMENT ALGORITHM

The three objectives of treatment are :

- Obtain detumescence
- Avoid immediate or distant recurrence
- Avoid the consequences o erectile function.

Ischemia priapism or recurrent (stuttering)

- Duration < 4hours :

- **1st : observation** :

+ **For ischemic or recurrent (stuttering) priapism lasting up to 4 hours**, Observation or treatment are both acceptable options for management, depending on clinician or patient preference. However, delays in treatment predispose the patient to tissue injury placing the patient at risk for the development of erectile dysfunction. Therefore, prompt treatment of all episodes of ischemic or stuttering priapism are encouraged.

+ **Ischemic or recurrent (stuttering) priapism lasting > 4 hours** is an emergency treatment.

- **1st : Aspiration ± irrigation :** Irrigation/flushing of the

cavernosa with normal saline or phenylephrine diluted with normal saline to a concentration of 100 to 500 micrograms/mL may be used in conjunction with aspiration. If phenylephrine is unavailable, other sympathomimetics may be used with similar success. **Plus**

- **Intracavernosal injection of sympathomimetic agent:**
  + **Primary options**:

- Phenylephrine is diluted in normal saline to a concentration of 100-500 µg/mL. Usually 200 µg are given every 3-5 minutes directly into the corpus cavernosum. Maximum dosage is 1 mg within 1 hour, before deciding that the treatment will not be successful. A lower concentration or volume is applicable for children and patients with severe cardiovascular disease. During and following intracavernous injection of any sympathomimetic, the patient should be monitored for known adverse effects (e.g., acute hypertension, headache, reflex bradycardia, tachycardia, palpitations, and cardiac arrhythmia). In all patients undergoing aspiration with irrigation, especially patients with high cardiovascular risk, blood pressure and ECG monitoring are recommended.

  + Secondary options :

- Ephedrine : 50-100 mg/dose. OR
- Adrenaline (epinephrine) (10-20mcg/dose) : Intracavernosal adrenaline (dosage of 2 mL of 1/100,000 adrenaline solution up to five times over a 20-minute period), has been used in patients with ischemic priapism due to an intracavernosal injection of vasoactive agents. Success rate of over 50% after a single injection, with an overall success rate of 95% with repeated injections is achieved. OR
- Noradrenaline (Norepinephrine) : 10-20mcg/dose. OR
- Metaraminol : 2-4mg/dose.
- Phenylephrine is the preferred sympathomimetic agent because it has a lower risk of cardiovascular adverse effects than other agents. However, if phenylephrine is unavailable, other alpha adrenergic agonists may be used with similar success.

- **Duration >4 hours :**
- **1st aspiration ± irrigation plus Intracavernosal injection of sympathomimetic agent**
- **2nd Penile shunt surgery :**

DISTAL SHUNTS	
Percutaneous Distal Shunts	
Winter (Corporo Glanular) Shunt	Large biopsy needle is inserted through glans into corpora cavernosum several times creating multiple fistulae
Ebbehoj (Corporo Glanular) Shunt	#11 blade scalpel is percutaneously passed multiple times through glans into corpus cavernosum creating openings in the tunica albugenia resulting in larger fistulae
T-Shunt (Corporo Glanular) Shunt	Modified Ebbehoj using #10 blade scalpel and turning scalpel 90 degrees when pulling out creating T-shaped openings in tunica albugenia.
Open Distal Shunt	
Al-Ghorab	A 1 cm incision is made distal to coronal sulcus with excision of 5x5 cone segment of distal tunica albuginea from each corporal body
Burnett "Snake" Maneuver	Modification of Al-Ghorab shunt. A Hegar dilator is used to evacuate ischemic blood through a distal tunical window.
PROXIMAL SHUNTS	
Open Proximal Shunt	
Quackels or Sacher (Corporo-Spongiosal) Shunt	In lithotomy position, bulbocavernosus muscle is dissected from corpus spongiosum and 1cm staggered ellipses of tissue are incised/excised from spongiosal/corporal bodies.
Corporo-Saphenous Vein or Superficial/ Deep Dorsal Vein Shunts	
Grayhack Shunt	The saphenous vein is ligated and anastomosed with corpora cavernosa
Barry Shunt	The superficial or deep dorsal vein is ligated and anastomosed to the corpora cavernosa.

Types of surgical shunt procedures for the treatment of ischaemic priapism
Helen R. Levey, DO, MPH

- **Penile Prosthesis :**

  + Currently there is no defined indication for implantation of a penile prosthesis in patients with priapism.
  + In priapism of extreme prolonged duration, exceeding 72 hours, or in patients with multiple episodes of recurrent
  refractory ischemic priapism, definitive treatment that
  includes placement of a penile prosthesis may be an appropriate first-line therapy to treat the priapism and allow resumption of sexual activity.
  + In such cases of prolonged priapic duration (>72 hours) complete erectile dysfunction may occur.

Non ischemic priapism

- **1st observation :**

  + The initial management of non-ischemic priapism should be observation.
  + Spontaneous resolution is seen in 62% of cases, although erectile dysfunction of some form is seen in approximately 30% of patients.

- **Patient preference for intervention : counselling and cavernosal artery embolization :**

  + For patients who request such treatment following

discussion, selective arterial embolization is recommended.
+ Both non-permanent (i.e., autologous clot, absorbable gels) and permanent (i.e., coils, ethanol, polyvinyl alcohol particles, and acrylic glue) embolization materials are available for use. All achieve a 75% resolution rate. However, non- permanent agents are preferred over permanent agents because they are associated with a lower incidence of subsequent erectile dysfunction.

- **Surgery :** Surgical management of non-ischemic priapism should be considered only as a last resort. Usually this involves direct surgical ligation of cavernosal sinusoidal fistulae or pseudoaneurysms. This should be performed with intraoperative color duplex ultrasonography.

Recurrent (stuttering) priapism

- **Treatment should focus on preventing future episodes**, whereas management of each episode should follow that for ischemic priapism.
- Anti-androgens, 5-alpha-reductase inhibitors, and gonadotrophin-releasing hormone agonists for prevention, and sympathomimetic intra-cavernous injection therapies for immediate patient self- administration, have shown to be successful medical management options for some patients with stuttering priapism.

- **1st treatment of any underlying condition medical condition should be appropriately managed.**
- **Adjunct gonadotrophin-releasing hormone agonist or anti-androgen :**

· Leuprorelin: 7.5 mg intramuscularly once a month OR
· Flutamide: 250 mg orally three times daily OR
· Ketoconazole: 200 mg orally once daily

If adjunct with Prednisone : potentially effective treatment for pre-pubertal men or those desiring fertility.While ketoconazole is wellknown as an antifungal agent, one of its side effects is that it reduces testosterone levels.

- Hormonal agents should not be used in patients who have not achieved full sexual maturation and adult stature.
- Ketoconazole may cause severe liver injury and adrenal insufficiency. If used, liver and adrenal function should be monitored before and during treatment.

- The treatment can be stopped after a few months to see whether priapism recurs and, if it does, treatment can be restarted. However, there is minimal information regarding the efficacy and safety of most of these agents and none have been investigated in controlled clinical studies.
- **Intracavernosal self-injection of phenylephrine or other sympathomimetic agent**

VI. COMPLICATION

- Penile fibrosis
- Penile ischemia
- Shunt procedure-related complications
- Erectile dysfunction

VII. PROGNOSIS

Ischemic priapism

- The most common complication of priapism is complete erectile dysfunction > 59%.
- Patients treated within 12 to 24 hours will have a more favorable response than those with delayed treatment.
- Patients with prolonged priapism >36 hours and recurrent episodes are more likely to have erectile dysfunction owing to impaired corporal smooth muscle function and fibrosis.
- Corporal ischemia lasting >24 hours results in varying degrees of irreversible penile fibrosis with endothelial and smooth muscle cell destruction. If left untreated, ischemic priapism results in global penile fibrosis with significant impairments in erectile function.
- For priapism episodes refractory to medical therapy and requiring surgical shunting, the success rate of these procedures ranges between 50% and 65%.
- For those patients refractory to all treatment strategies or who have irreversible erectile dysfunction, a penile prosthesis is the only management option available.
- Prompt recognition and treatment of ischaemic and recurrent priapism are essential for optimal outcomes.

Non-ischaemic priapism

- Spontaneous resolution of untreated non-ischemic priapism is reported in up to 62% of cases.
- In cases treated by embolisation, data suggest that outcome is procedure-dependent.
- Resolution of non-ischemic priapism is reported in 78% of patients treated with permanent embolisation, although 39% of patients have subsequent erectile dysfunction.
- By contrast, temporary embolisation shows a 74% resolution rate with only 5% reporting erectile dysfunction.

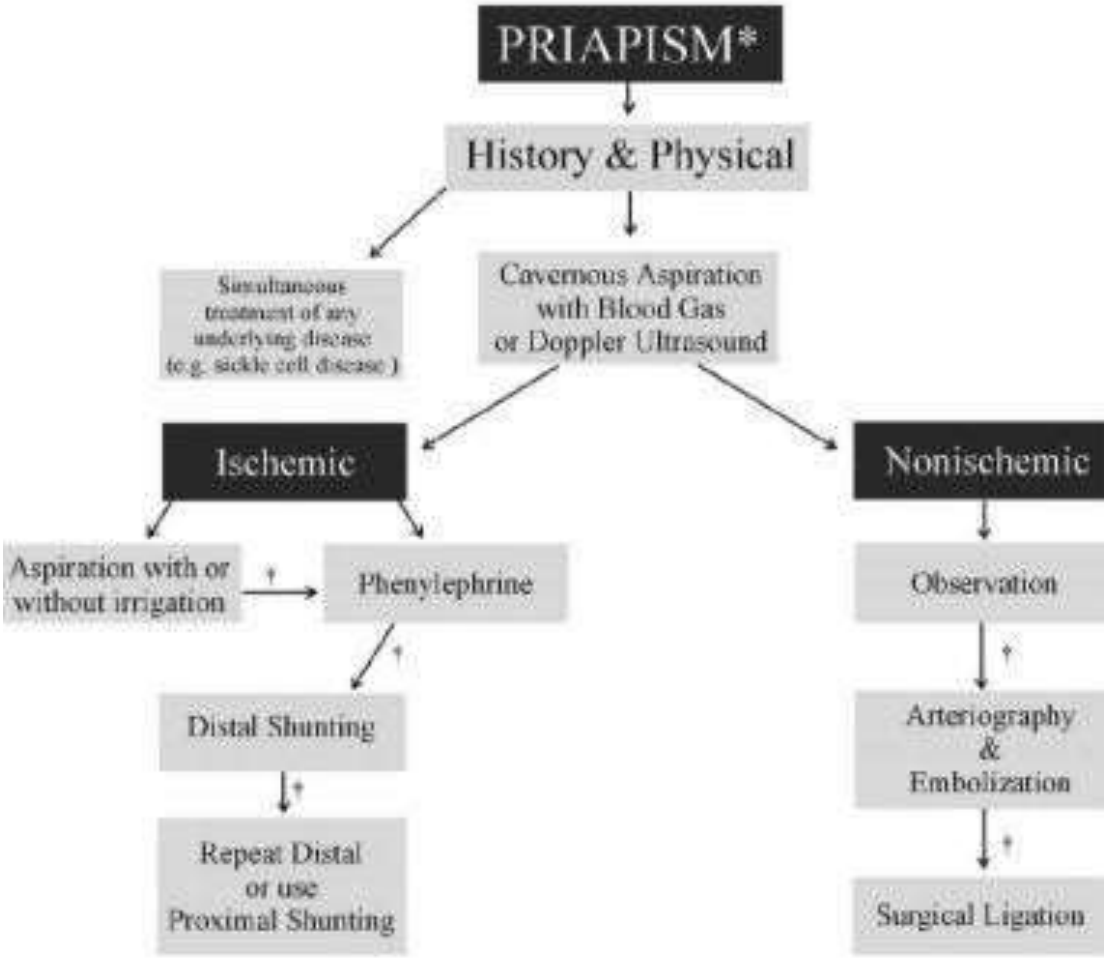
Stuttering priapism

- The frequency and duration with which the priapic episodes occur will

largely determine the amount and extent of damage to the penis and likelihood of erectile dysfunction.

- Often, penile vascular dysfunction may actually be a consequence of significant fibrosis resulting from repeated and prolonged episodes of priapism, rather than the result of a surgical or shunt procedure itself.
- In patients who have undergone extended durations of ischemic priapism, some authorities suggest that immediate placement of a penile implant may be more beneficial than proceeding with a surgical shunt.
- Significant fibrosis, which is commonly found in these patients, makes surgery more difficult with higher complication rates.
- This type of priapism should be followed and treated as for ischemic priapism with the goal of preventing future stuttering episodes.

VIII. ALGORITHM



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PROSTATE CANCER

HAY VANEL, OUK REAKSMEY, BOU SOPHEAP

I. INTRODUCTION

- PCa : most common male cancer in developed countries, second only to lung cancer worldwide, most common cancer of male GU tract, second leading cause of death in men from cancer.
- PCa can be slow growing, making the diagnosis and staging of this cancer of great medical and public interest.
- Many PCa will not need to have treatment quickly. Most PCa grow slowly. Some will die from other coexisting diseases rather than from PCa.

II. CLINICAL PRESENTATION

- Prostatism 70%
- Retention of urine 23%
- Back pain 14 %
- Haematuria 5%
- Post prostatic symptoms 5 %
- Renal failure 4 %
- Weight loss
- Altered bowel habits 3%
- Anaemia 1%

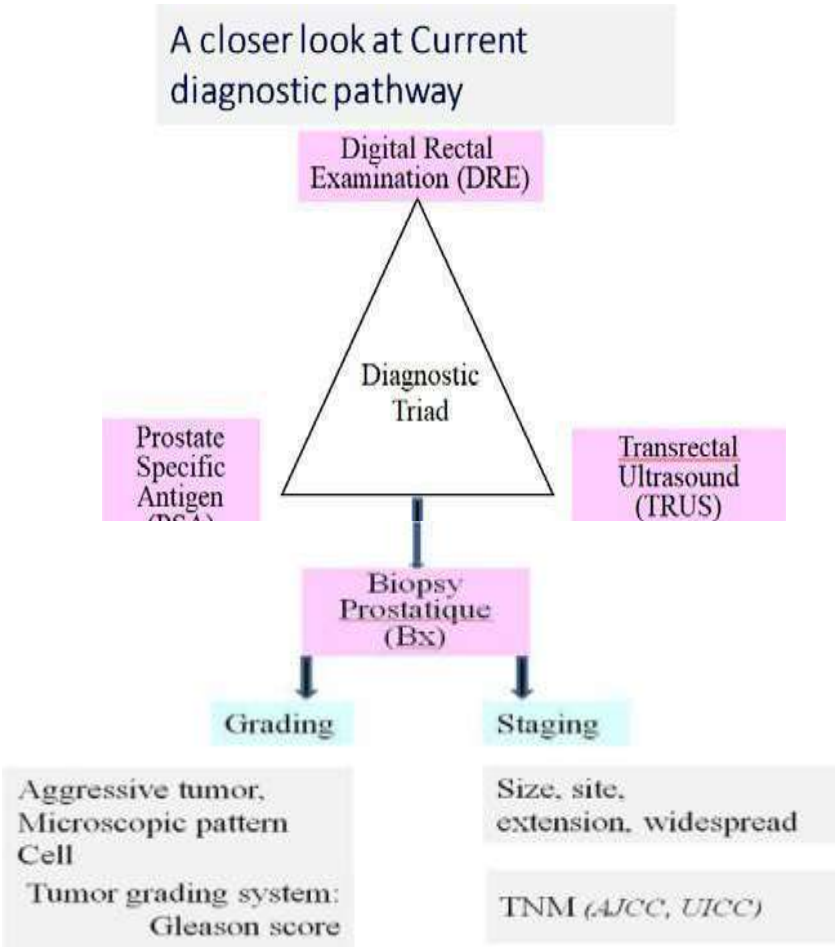
III. DIAGNOSIS

- Detailed history
- Thorough examination
- Appropriate investigation

Clinical Diagnostic:

- Digital Rectal Examination DRE (first choice)

- PSA (history of family PCa, age < 60 ans before endorectal procedure/ wait until 2 weeks after endorectal procedure)
- Echo abdominal or transrectal ultrasound/IRM
- Biopsy



Biopsy Protocol:

- Systematic 18g trucut needle biopsies are taken, including any palpable or sonographic target lesion.
- Biopsy guided by transurectal ultrasound; digital guided
- Transurectal ultrasound first choice 10-12 biopsies, palpable 6 biopsies.

Antibioprophylaxis:

- Risk of acute prostatitis 10 % without antibiotic
- Give, Ex. Ciprofloxacine500mg 1 h before procedure

NB: Repeat biopsy after previously negative biopsy :

- Rising and/or persistently elevated PSA
- Suspicious DRE, 5-30% PCa risk
- Atypical small acinar proliferation (i.e. atypical glands suspicious for cancer), 31-40% PCa risk on repeat biopsy
- Extensive (multiple biopsy sites, i.e. > 3) high-grade prostatic intraepithelial neoplasia (HGPIN), ~30% PCa risk
- A few atypical glands immediately adjacent to high-grade prostatic intraepithelial neoplasia (i.e. PINATYP),~50% PCa risk
- Intraductal carcinoma as a solitary finding, > 90% risk of associated high-grade PCa
- Positive multiparametric MRI (mpMRI) findings

Prostate Specific Antigen (PSA):

- It is a glycoproteine
- It liquefies semen
- It is prostate specific but NOT prostate cancer specific
- Normal (0-4.0 ng/ml)

Free PSA first choice, but big prostate total PSA Free PSA / Total PSA ratio: Les than 7 % = Probability of neoplasia 93
% 7-15 % = Probability of neoplasia
48 %
16-25 % = Probability of neoplasia 27 %
>25% = Probability of neoplasia 7 % PSA-Density , PSA velocity

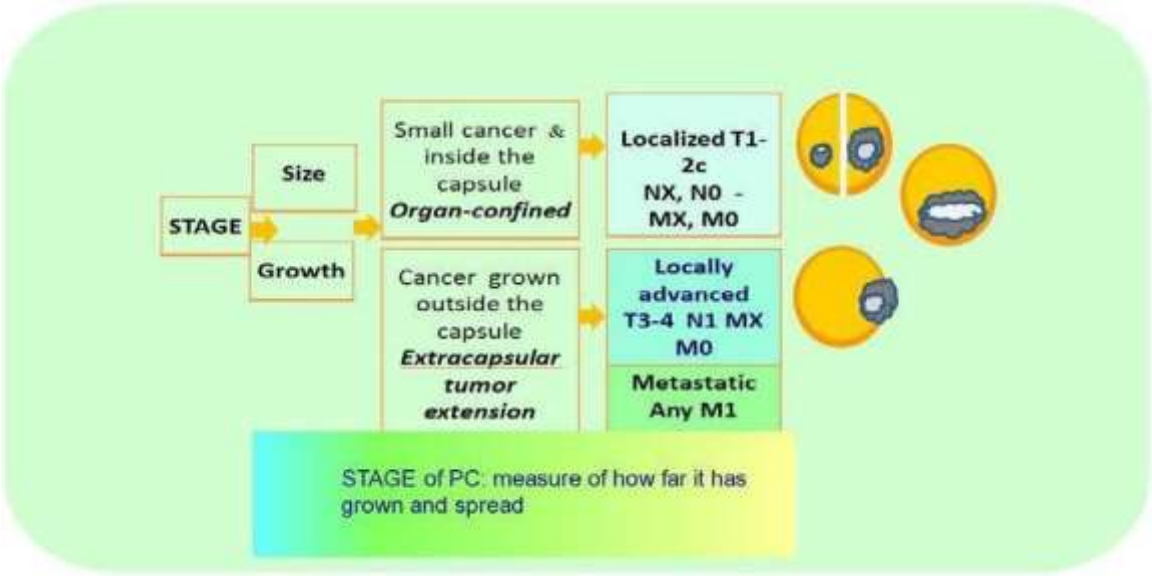
- **PSA** may be elevated in :
  - BPH
  - Prostatitis
  - Drugs
  - Manipulation –DRE, Catherization, ejaculation

TNM staging:

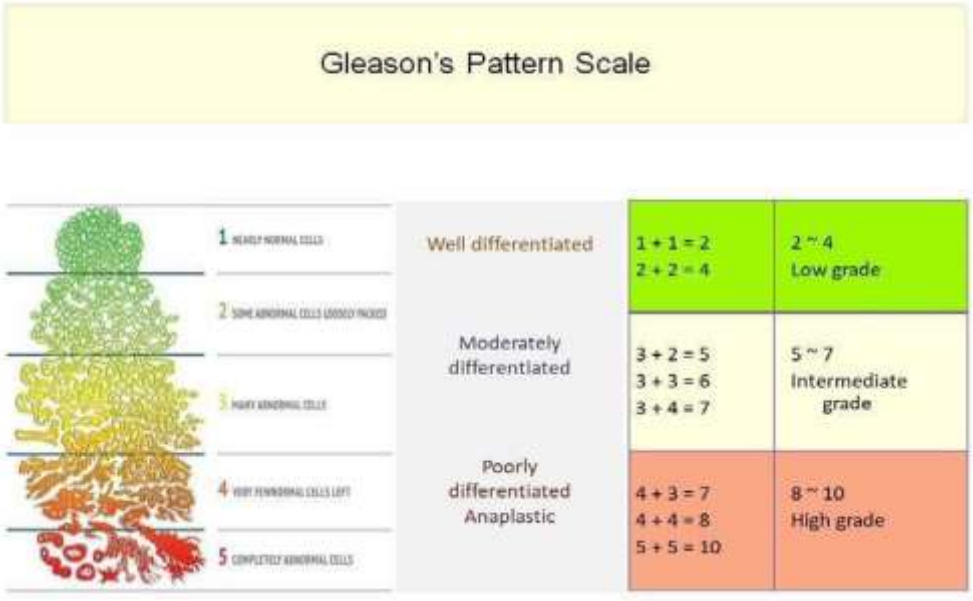
- For T category = Clinic examination, CTU,IRM, Endoscopy and BIOPSY
- For Nodal status N = Clinical examination + CT
- For Metastase M = Clinical examination or CT ,Skelatal survey ,Bone Scan, PSA

2017 TNM classification

T - Primary Tumour	
TX	Primary tumour cannot be assessed
T0	No evidence of primary tumour
T1	Clinically inapparent tumour that is not palpable
	T1a Tumour incidental histological finding in 5% or less of tissue resected
	T1b Tumour incidental histological finding in more than 5% of tissue resected
	T1c Tumour identified by needle biopsy (e.g. because of elevated prostate-specific antigen [PSA])
T2	Tumour that is palpable and confined within the prostate
	T2a Tumour involves one half of one lobe or less
	T2b Tumour involves more than half of one lobe, but not both lobes
	T2c Tumour involves both lobes
T3	Tumour extends through the prostatic capsule*
	T3a Extracapsular extension (unilateral or bilateral) including microscopic bladder neck involvement
	T3b Tumour invades seminal vesicle(s)
T4	Tumour is fixed or invades adjacent structures other than seminal vesicles: external sphincter, rectum, levator muscles, and/or pelvic wall
N - Regional Lymph Nodes[1]	
NX	Regional lymph nodes cannot be assessed
N0	No regional lymph node metastasis
N1	Regional lymph node metastasis
M - Distant Metastasis[2]	
M0	No distant metastasis
M1	Distant metastasis
	M1a Non-regional lymph node(s)
	M1b Bone(s)
	M1c Other site(s)



Gleason score and International Society of Urological Pathology 2014 grade:



Gleason score	ISUP grade
2-6	1
7 (3+4)	2
7 (4+3)	3
8 (4+4 or 3+5 or 5+3)	4
9-10	5

Risk groups for biochemical recurrence of localised and locally advanced prostate cancer :

Definition			
Low-risk	**Intermediate-risk**	**High-risk**	
PSA < 10 ng/mL and GS < 7 (ISUP Grade 1) and cT1-2a	PSA 10-20 ng/mL or GS 7 (ISUP Grade 2/3) or cT2b	PSA > 20 ng/mL or GS > 7 (ISUP Grade 4/5) or cT2c	any PSA any GS cT3-4 or cN+ Any ISUP Grade
Localised			**Locally advanced**

GS = Gleason score; ISUP = International Society for Urologcal Pathology; PSA = prostate-specific antigen.

IV. MANAGEMENT OF PCA

The choice of treatment should be decided on many risk groupe factors and survival rate (age and comorbidities) for each individual patient:
Deferred treatment / watchful waiting/active surveillance:

	Active surveillance	Watchful waiting
Treatment intent	Curative	Palliative
Follow-up	Predefined schedule	Patient-specific
Assessment/markers used	DRE, PSA, re-biopsy, mpMRI	Not predefined
Life expectancy	> 10 years	< 10 years
Aim	Minimise treatment-related toxicity without compromising survival	Minimise treatment-related toxicity
Comments	Low-risk patients	Can apply to patients with all stages

DRE = digital rectal examination; PSA = prostate-specific antigen; mpMRI = multiparametric magnetic resonance imaging.

- Active surveillance(AS): PSA testing every 3 months,DRE 6- monthly, and 2-yearly repeat biopsy to assess for upgrading
- Watchful waiting(WW): most men with localized PC on WW are seen 6 months for clinical history, examination, including a DRE and a serum PSA.

Localized tumor without any metastases Mo :

Deferred traitement (follow up and Monitor)

- Radical Surgery(RP): (Radical prostatectomy- open/laparoscopic)
  - Local Obstructive Symptoms = TURP
  - Radiotherapy(external beam or interstitial)
  - No endocrine or Hormonal traitement (not for Mo disease)

*RP: indicated for the traitement (with curative intent) of fit men with localized PC
whose life expectancy exceeds 10 years.
*External radiotherapy: Indication for Pca N0M0 with survival rate >10 years Locally Advanced : Hormonotherapy
Surgical castration Medical castration
Radiotheapy (external beam or interstitial) Metastastic disease at diagnostic M+ :
Systemic traitement should be given for a systemic disease already spread into the systems Hormonal traitement :

- Treatment to lower testicular androgen levels:
- Chemical Castration :

LHRH agonists – available in 1 month, 3 months, 6 months, and once months (side effect: the potential flare response that can occur the first 3 weeks of traitement)

LHRH antagonist : avaible in a 1-month depot

- Surgical castration: Bilateral orchidectomy
- Treatment to lower androgen levels from the adrenal glands **:**

Abiraterone (Zytiga)

- Anti-androgne therapy: androcur 50mg 4 or 6 tablet per day .

Alternative traitements:

- TURP
- Cystotomy definitive

Follow up and Monitor :

- Objectively : - Primary tumour DRE, USG,PSA ,
  Bony and soft tissue metastases
- Sujectively :
  - Activity
  -Analgesics reguirement
  -Body Weight
  -symptoms

* After external radiotherapy: PSA every 6 monthly duration 3 years after every year.

*After Radical prostatectomy: first PSA at 2 months,PSA every 6 monthly duration 3 years after every year.

* Hormone therapy: PSA at 3 months after semester

Castration Resistant Prostate Cancer (CRPC) :

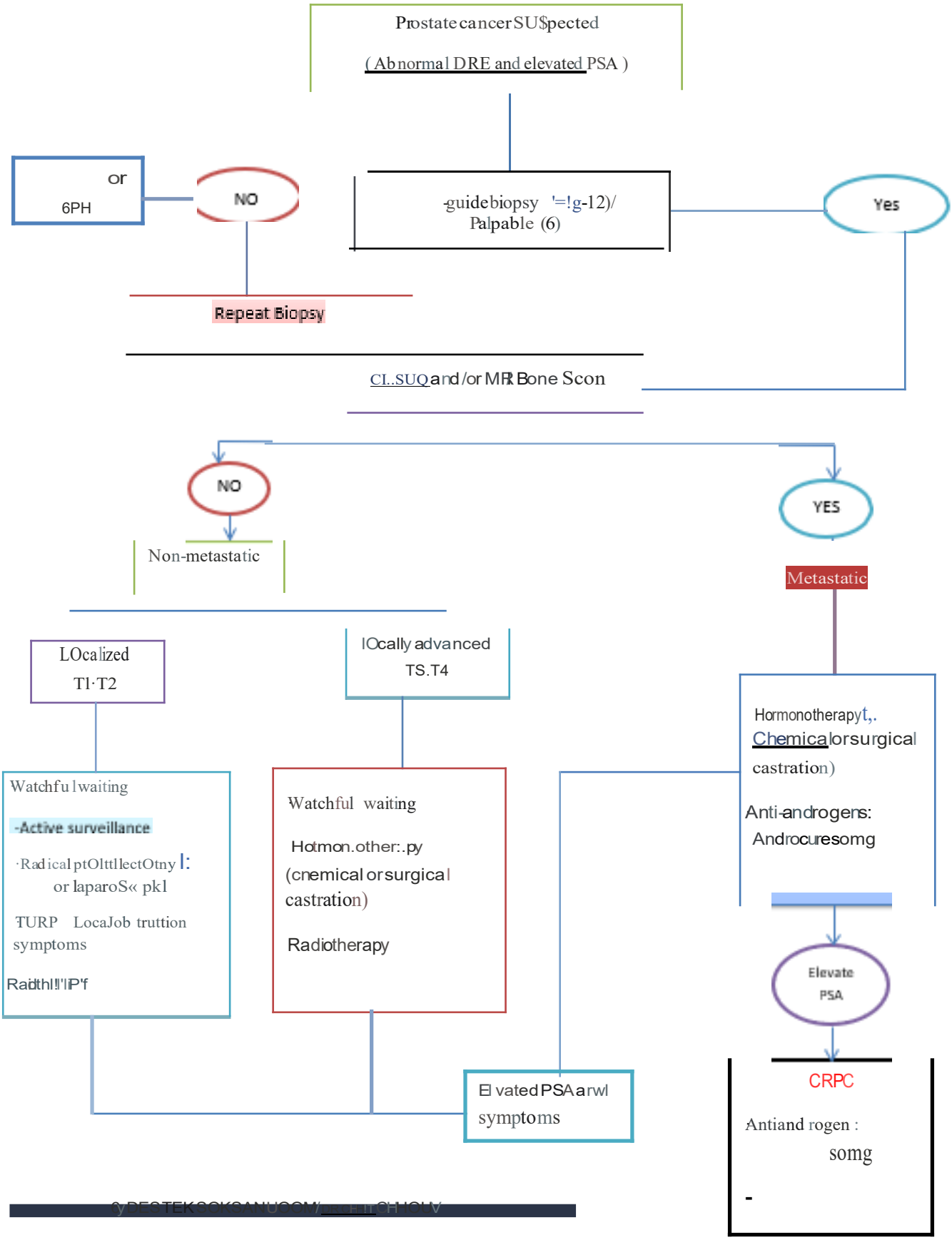
Castrate serum testosterone < 50 ng/dL or 1.7 nmol/L plus either;

a. Biochemical progression: Three consecutive rises in PSA one week apart resulting in two 50% increases over the nadir, and a PSA > 2 ng/mL or,
b. Radiological progression: The appearance of new lesions: either two or more new bone lesions on bone scan or a soft tissue lesion using RECIST (Response Evaluation Criteria in Solid Tumours) [822]. Symptomatic progression alone must be questioned and subject to further investigation. It is not sufficient to diagnose CRPC.

Management of CRPC:

Anti-androgen therapy: Androcure 50mg 4 or 6 tablet per day
Chimiotherapy: Docetaxel, Cabazitaxel

ALGORITHM GUIDELINE MANAGEMENT OF PROSTATE CANCER FOR CAMBODIA-CHINA FRIENDSHIP PREAH KOSSAMAK HOSPITAL



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RENAL CELL CARCINOMA

HAY VANEL, OUK REAKSMEY , BOU SOPHEAP

I. INTRODUCTION

Renal cell carcinomas (RCCs), which originate within the renal cortex, are responsible for 80 to 85 % of all primary renal neoplasms. Transitional cell carcinomas of the renal pelvis are the next most common (approximately 8 %). Other parenchymal epithelial tumors, such as oncocytomas, collecting duct tumors, and renal sarcomas, occur infrequently. Nephroblastoma or Wilms tumor is common in children (5 to 6 % of all primary renal tumors), while renal medullary carcinoma is a rare form of RCC seen in sickle cell disease.

II. EPIDEMIOLOGY

Renal cell carcinoma represents around 3% of all cancers, with the highest incidence occurring in Western Countries. Generally, during the last two decades until recently, there has been an annual increase of about 2% in incidence both worldwide and in Europe leading to approximately 99,200 new RCC cases and 39,100 kidney cancer-related deaths within the European Union in 2018. In Europe, overall mortality rates for RCC increased until the early 1990s, with rates generally stabilizing or declining thereafter.

III. AETIOLOGY

- Smoking is the most well-established risk factor for RCC; it is implicated in 20% to 30% of RCC in men, and 10% to 20% in women.
- Obesity and hypertension are also known risk factors, although it can be difficult to establish if hypertension is a cause or a consequence of RCC in individual cases.
- Renal transplantation and dialysis have also been linked to RCC development.
- Exposure to pelvic radiation is a weak risk factor.
- A positive family history increases the risk of RCC fourfold.

IV. PATHOLOGY

Several distinct subtypes of RCC have been identified, including the following:

- Clear cell (75 to 85 percent of tumors)
- Papillary (chromophilic; 10 to 15 percent)
- Chromophobe (5 to 10 percent)
- Oncocytic (3 to 7 percent)
- Collecting duct (Bellini duct; very rare)

V. DIAGNOSTIC EVALUATION

1) Symptoms

- Many renal masses remain asymptomatic until the late disease stages.
- More than 50% of RCCs are detected incidentally by non-invasive imaging investigating various non-specific symptoms and other abdominal diseases.
- The classic triad of RCC (flank pain, hematuria, and a palpable abdominal renal mass) occurs in at most 9 percent of patients; when present, it strongly suggests locally advanced disease.

- Paraneoplastic syndromes are found in approximately 30% of patients with symptomatic RCCs
- Some symptomatic patients present with symptoms caused by metastatic disease, such as bone pain or persistent cough.

2) Physical examination

Physical examination has a limited role in RCC diagnosis. However, the following findings should prompt radiological examinations:

- palpable abdominal mass
- palpable cervical lymphadenopathy
- non-reducing varicocele and bilateral lower extremity oedema, which suggests venous involvement.

3) Laboratory findings

Commonly assessed laboratory parameters are serum creatinine, glomerular filtration rate (GFR), complete cell blood count, erythrocyte sedimentation rate, liver function study, alkaline phosphatase, lactate dehydrogenase (LDH), serum corrected calcium, coagulation study, and urinalysis.

4) Imaging

- **Ultrasound** is an appropriately sensitive initial imaging for determining if cystic renal lesions are more likely to be benign, especially in hereditary syndromes prone to cystic disease (e.g., von Hippel Lindau). However, it is not possible to assess complex cystic masses and/or solid renal masses with ultrasound alone.
- **Spiral or contrast-enhanced CT** is the modality of choice. The patient should undergo CT before and after injection of iodinated contrast. Abdominal CT provides information on:
  - Function and morphology of the contralateral kidney
  - Primary tumour extension;
  - Venous involvement;
  - Enlargement of locoregional lns;
  - Condition of the adrenal glands and other solid organs
- **Magnetic resonance imaging (MRI)** may be useful when ultrasonography and/or CT are inconclusive or if iodinated contrast cannot be administered because of allergy or poor renal function.
- **Angiography:** Catheter-based renal angiography is rarely necessary. If preoperative mapping of the vasculature is required prior to possible nephron-sparing surgery, either CT or MR angiography is preferable.
- **Radiographic investigations to evaluate RCC metastases**
  - Chest CT is accurate for chest staging.
  - Bone scan, brain CT, or MRI may be used in the presence of specific clinical or laboratory signs and symptoms

5) Renal tumor biopsy

- Percutaneous renal tumor biopsy can reveal histology of radiologically indeterminate renal masses and can be considered in patients who are candidates for active surveillance of small masses, to obtain histology before ablative treatments, and to select the most suitable medical and surgical treatment strategy in the setting of metastatic disease.
- Renal biopsy is not indicated for comorbid and frail patients who can be considered only for conservative management (watchful waiting) regardless of biopsy results.

VI. STAGING AND CLASSIFICATION SYSTEMS

The Tumour Node Metastasis (TNM) classification system is recommended for clinical and scientific use, but requires continuous re-assessment.

T - Primary Tumour

TX Primary tumour cannot be assessed
T0 No evidence of primary tumour
T1 Tumour < 7 cm or less in greatest dimension, limited to the kidney
  T1a Tumour < 4 cm or less
  T1b Tumour > 4 cm but < 7 cm
T2 Tumour > 7 cm in greatest dimension, limited to the kidney
  T2a Tumour > 7 cm but < 10 cm
  T2b Tumours > 10 cm, limited to the kidney
T3 Tumour extends into major veins or perinephric tissues but not into the ipsilateral adrenal gland and not beyond Gerota fascia
  T3a Tumour grossly extends into the renal vein or its segmental (muscle-containing) branches, or tumour invades perirenal and/or renal sinus fat (peripelvic fat), but not beyond Gerota fascia
  T3b Tumour grossly extends into the vena cava below diaphragm
  T3c Tumour grossly extends into vena cava above the diaphragm or invades the wall of the vena cava
T4 Tumour invades beyond Gerota fascia (including contiguous extension into the ipsilateral adrenal gland)

N - Regional Lymph Nodes

NX Regional lymph nodes cannot be assessed
N0 No regional lymph node metastasis
N1 Metastasis in regional lymph node(s)

M - Distant Metastasis

M0 No distant metastasis
M1 Distant metastasis

pTNM stage grouping

Stage I	T1	N0	M0
Stage II	T2	N0	M0
Stage III	T3	N0	M0
	T1, T2, T3	N1	M0
Stage IV	T4	Any N	M0
	Any T	Any N	M1

VII. PROGNOSTIC FACTORS

Prognostic factors can be classified into: anatomical, histological, clinical, and molecular.

- **Anatomical factors:** The anatomic extent of disease is the most consistent factor that influences prognosis in patients with renal cell carcinoma (RCC)
  - Stage I/II — Patients with stage I RCC have a five-year survival rate over 90 percent in most contemporary series. The survival rate may be slightly lower for patients with stage II disease, with reported five-year survival rates ranging from 75 to 95 percent.
  - Stage III — The reported five-year survival rate for patients with stage III RCC who undergo nephrectomy ranges from 59 to 70 percent.
  - Stage IV — While the median survival for patients with stage IV disease was a little over one year when cytokines were the predominant systemic therapies,
  - analyses from the International Metastatic Renal Cell Carcinoma Database Consortium (IMDC).
- **Histological factors**
  - Histological factors include tumour grade, RCC subtype, sarcomatoid features, microvascular invasion, tumour necrosis, and invasion of the collecting system.
  - Multiple systems are used to grade renal cell carcinoma (RCC), of which Fuhrman's grade is the most widely used. In one report, the five-year survival
  - rates based upon tumor grade were 89, 65, and 46 percent for tumors of
  - histologic grade 1, 2, and 3 to 4, respectively.
- **Clinical factors**
  - In addition to the anatomic extent of disease, clinical factors can influence survival. Adverse prognostic signs include a poor performance status, the presence of symptoms and/or paraneoplastic syndromes (eg, anemia, hypercalcemia, thrombocytosis, fever, weight loss), and obesity.
- **Molecular factors:** Although none of these factors currently has a clinical application for patient care, some markers have shown promise as prognostic markers in patients with clear cell RCC. Examples of markers that are potentially associated with a worse prognosis for patients with clear cell RCC include:
  - Human B7 homolog 1 (B7H1) and 4 (B7H4) expression
  - Low levels of carbonic anhydrase IX (CAIX)
  - High levels of the proliferation marker Ki-67
  - Higher levels of hypoxia-inducible factor (HIF)-1 alpha expression
  - Expression of the U3 small nucleolar ribonucleoprotein (IMP3)
  - Deletion of chromosome 9p
  - Mutations of tumor suppressor genes on chromosome 3p21

VIII. DISEASE MANAGEMENT

RCC can be classified as:

- Localized disease – This includes stage I, II, and III
- Advanced disease – This includes tumor invading beyond Gerota's fascia or extending into the ipsilateral adrenal gland (T4) and metastatic disease (M1).

A. Treatment of localised RCC

- For patients with localized, resectable renal cell carcinoma (RCC), we recommend surgery as the primary treatment approach.

For patients with a primary tumor <7 cm, we recommend a partial nephrectomy(either open or laparoscopic) rather than radical nephrectomy when it is technically feasible.

- For patients with a primary tumor >7 cm, we perform a radical nephrectomy. Radical nephrectomy has been the most widely used approach and remains the preferred procedure when there is evidence of invasion into the adrenal, renal vein, or perinephric fat.
- Patients with a solitary kidney, those with multiple and/or bilateral renal tumors, and those with baseline renal dysfunction should undergo a partial nephrectomy whenever possible.
- Patients with RCC should undergo a lymph node dissection at the time of radical nephrectomy.
  - For patients with suspected retroperitoneal lymph node involvement and those at high risk for nodal involvement, we recommend an extended lymphadenectomy at the time of radical nephrectomy.
  - For patients in whom retroperitoneal lymph node involvement is not suspected and those at low risk for nodal involvement, we suggest a limited lymph node dissection concentrated around the renal hilum.
- For patients with involvement of the inferior vena cava (IVC), we recommend surgery rather than medical therapy. These patients should undergo thrombectomy at the time of radical nephrectomy.
- For patients with RCC that directly extends to the ipsilateral adrenal gland and those at risk for invasion of the adrenal gland, we recommend surgery rather than medical therapy.
- For elderly patients and those with significant comorbid disease, ablative techniques (cryoablation, radiofrequency ablation) are an alternative.
- Active surveillance may be an option for patients with small asymptomatic lesions (defined as tumor size <4 cm), particularly for patients with significant comorbidity or a short life expectancy.

- Following complete resection of a localized RCC, we do not suggest adjuvant therapy outside of a clinical trial, as this approach confers no clear overall survival benefit and increases toxicity.

B. Advanced or metastatic clear cell RCC

- **Immunotherapy** — Immunotherapy is an important option for the management of patients with advanced clear cell RCC, both as initial therapy or as subsequent therapy after molecularly targeted therapy.
  - **Checkpoint inhibitor immunotherapy** — Checkpoint inhibition targeting either the programmed cell death receptor 1 (PD-1) pathway and/or cytotoxic T lymphocyte-associated antigen 4 (CTLA-4) has represented an important advance in the treatment of multiple malignancies, including clear cell RCC. The combination of nivolumab (an anti-PD-1 antibody) and ipilimumab (an anti-CTLA-4 antibody) has an established role in the treatment of intermediate- and poor-risk patients.
  - **Interleukin 2** — Immunotherapy with high-dose bolus IL-2 can activate an immune response against RCC that results in tumor regression in a minority of patients.
  - **Interferon alfa** — The use of interferon alfa (IFNa) has largely been replaced by molecularly targeted agents and checkpoint inhibitor immunotherapy.
- **Molecularly targeted therapy**

- **Antiangiogenic (VEGF pathway)** — Two different approaches have clinical activity in blocking the vascular endothelial growth factor (VEGF) pathway. For patients who are ineligible for immunotherapy-based combinations, we offer antiangiogenic therapy with inhibitors of the vascular endothelial growth factor (VEGF) pathway.

- **mTOR inhibitors** — The mechanistic (mammalian) target of rapamycin (mTOR) pathway is downstream of the phosphoinositide 3-kinase and Akt pathway that is regulated by the phosphatase and tensin homolog (PTEN) tumor suppressor gene.

- **Combined antiangiogenic plus checkpoint inhibitor therapy**
  - Combinations of immunotherapy plus antiangiogenic therapy are active in patients with advanced or metastatic RCC.
- **Chemotherapy and hormonal therapy —** Both chemotherapy and progestational agents had only very limited activity in early studies prior to the development of immunotherapy and molecularly targeted therapy.
- **Radiation therapy —** Although RCC has been characterized as a radioresistant tumor, conventional and stereotactic RT are frequently useful to treat a single or limited number of metastases. Examples of situations where RT is useful include:
  - Painful bone metastases
  - Brain metastases
  - Painful recurrences in the renal bed
- **Cytoreductive nephrectomy**
  - Removal of the primary tumor (cytoreductive or debulking nephrectomy) may be indicated prior to initiating systemic therapy in select patients (eg, good performance status, 75 percent debulking possible, no symptomatic metastatic disease).
  - Randomized clinical trials demonstrated that patients who undergo a cytoreductive nephrectomy prior to IFNa immunotherapy had improved survival compared with those with an intact primary tumor.
- **Metastasectomy** — Surgical resection of a single or limited number of metastases is a reasonable option for carefully selected patients. Resection of metastatic disease (metastasectomy) has been performed in several situations:
  - Patients with stage IV disease at presentation, where metastasectomy is performed with nephrectomy
  - Patients who develop metastatic disease following nephrectomy
  - Patients who have persistent disease despite systemic therapy

C. Non-clear-cell metastatic RCC

- The specific treatment approach to patients with advanced-stage non-clear cell RCC is based on histologic subtype.
- Although many advances have been made in the treatment of non-clear cell RCC, there are limited high-quality data to help inform management due to the infrequency of these tumors.
- For patients with advanced papillary RCC, some contributors offer checkpoint inhibitor immunotherapy, while others offer a vascular endothelial growth factor (VEGF) tyrosine kinase inhibitor (TKI) as initial therapy.
- For patients with treatment-naïve advanced or metastatic RCC with sarcomatoid features, we recommend immunotherapy-based regimens rather than VEGF inhibitors as initial therapy.
- For patients with advanced or metastatic chromophobe RCC, available evidence is limited due to the rarity of these tumors. Some contributors offer initial treatment with targeted therapy such as a mammalian target of rapamycin (mTOR) inhibitor (eg,

everolimus) or a VEGF inhibitor (eg, sunitinib).

- For patients with advanced or metastatic collecting duct or renal medullary carcinoma, we suggest cytotoxic chemotherapy as initial therapy rather than VEGF inhibitors.
- For patients with advanced or metastatic translocation RCC, we suggest VEGF inhibitors with sunitinib as initial therapy rather than immunotherapy or cytokine therapy.
- For patients with advanced or metastatic unclassified RCC, some contributors offer initial therapy with immunotherapy (eg, pembrolizumab or nivolumab plus ipilimumab), while other contributors offer VEGF inhibitors (eg, sunitinib or cabozantinib).

D. Recurrent RCC

- Locally recurrent disease can occur either after nephrectomy, PN, or after ablative therapy.
- Patients can benefit from a complete surgical resection of local recurrent disease.
- In cases where complete surgical removal is not feasible due to advanced tumour growth and pain, palliative treatments including radiation treatment can be considered.

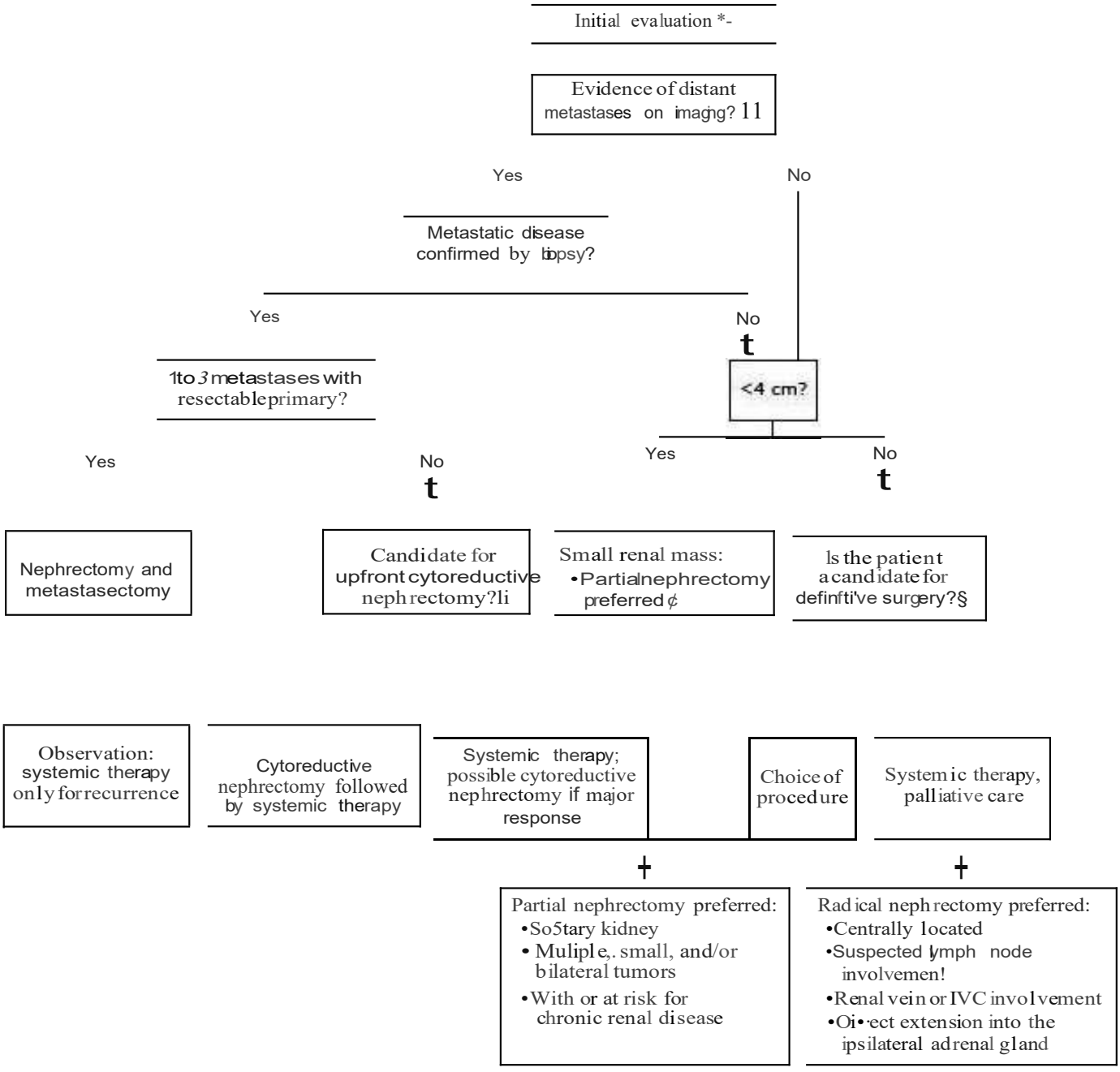
E. Surveillance following surgery for RCC

- The aim of surveillance is to detect either local recurrence or metastatic disease while the patient is still surgically curable. Surveillance after treatment for RCC allows the urologist to assess:
  - Postoperative complications
  - Renal function
  - Local recurrence
  - Recurrence in the contralateral kidney
  - Development of metastases.
- AUA guidelines — The AUA recommends specific guidelines for surveillance after surgical management of RCC
  - Stage I or T1N0/X (partial or radical nephrectomy)
    - History and physical examination at months 6, 12, 24, and 36 and further follow-up at the discretion of the treating clinician.
    - Serum blood urea nitrogen (BUN) or creatinine and urine analysis are recommended and other tests as clinically indicated.
    - Abdominal imaging:
      - After partial nephrectomy – Baseline abdominal computed tomography (CT)/magnetic resonance imaging (MRI) at month 6 and then abdominal CT/MRI/or ultrasound (US) at 12, 24, and 36 months.
      - After radical nephrectomy – Baseline abdominal CT, MRI at month 6 then imaging as clinically indicated.
    - Chest imaging – Chest radiograph or CT annually for three years, then as clinically indicated
    - Central nervous system (CNS) imaging, pelvic imaging, and bone imaging as clinically indicated.
  - PT2-4N0/X or pTanyN1
    - History and physical examination every six months until five years and further follow-up at the discretion of the treating clinician.

- BUN or creatinine and urinalysis; other tests as clinically indicated.
- Abdominal imaging – CT/MRI recommended at month 6, after which CT/MRI/US use acceptable for abdominal imaging every six months until five years, with further follow-up at the discretion of the treating clinician.
- Chest imaging – Chest CT initially at month 6 followed by chest radiograph or CT every six months until five years, and further follow-up at the discretion of the treating clinician.

- CNS imaging, pelvic imaging, and bone imaging as clinically indicated.

Initial evaluation and treatment of renal cell carcinoma



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TESTICULAR TORSION

HAY VANEL , OUK REAKSMEY , BOU SOPHEAP

I. INTRODUCTION

Immediate surgical exploration is indicated for patients with testicular torsion. For reliable salvage of the testicle, surgical repair must occur within 6 hours of symptom onset. If treatment is delayed, the patient may experience decreased fertility or may require orchiectomy.

II. INVESTIGATIONS AND PROCEDURE SPECIFIC REQUIREMENTS

+ Check urinalysis and urine culture.
+ History and physical examination finding are strongly suggestive :

Diagnosis	Features on history	Features on exam	Management
Testicular torsion Peak neonates, adolescents, 13-16years	**Sudden onset** unilateral testicular pain, swelling Pain usually constant **Associated Nausea/vomiting**	Discoloration, swollen of hemiscrotum. Cremasteric reflex and Prehn sign : absent.	**Early surgery** is vital – delay in exploration and detorsion.
Torsion hydatid Peak 11years (prepubertal boys)	More gradual onset testicular pain (1-2days) At time of rapid testicular growth No nausea or vomiting	Focally tender upper pole of testis : Blue dot sign. Reactive hydrocele	Analgesia, rest. Surgical exploration : excise torted areas.
Testicular rupture	Scrotal trauma: sports injury. Delayed onset of scrotal pain and swelling	Tender, swollen testis Bruising,Oedema Haematoma or haematocele	Surgical review in all testicular trauma.

+ Imaging studies should **NOT** be performed : the patient should be kept fasted and a surgical referral. But some time the Doppler ultrasound may be performed to determine the direction of testicular torsion and guide manual detorsion.

Doppler sonography

Diagnosis	Appearance on ultrasonography
Normal testis	Homogenous echogenicity surrounded by thin bright line (the tunica albuginea)
Testicular torsion	Absent or decreased blood flow
Epididymitis/orchitis	Increased blood flow

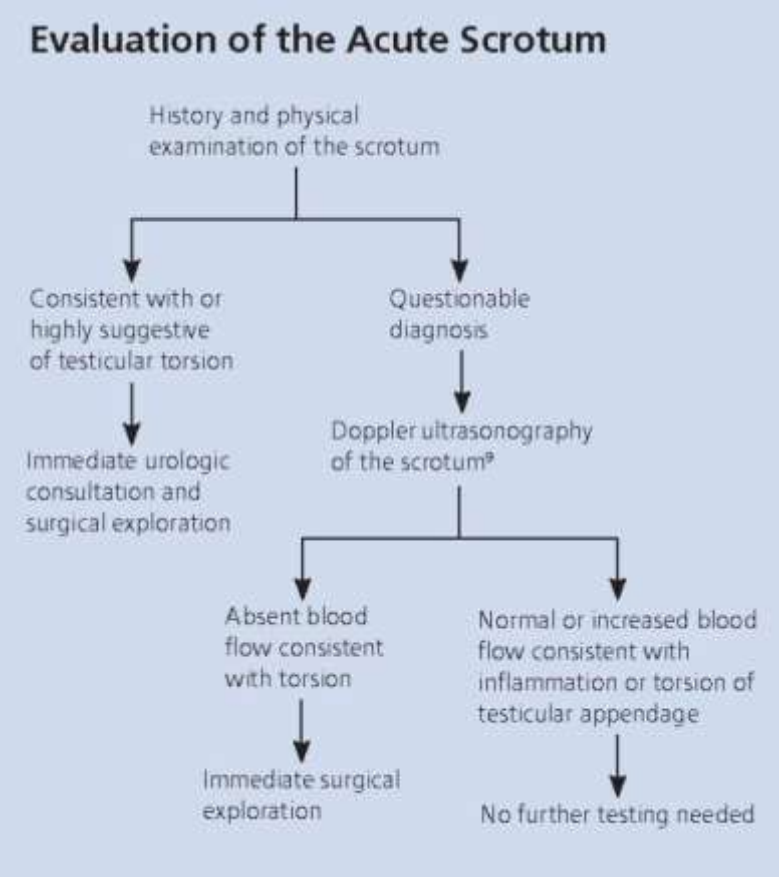
+ Appropriate radiology and laboratory facilities should be available.
+ Non-operative management should be made only by a senior surgical decision.
+ When viable, fixation of the affected testes and the contralateral testes is required (orchiopexy) :

- Midline raphe incision or bilateral transverse scrotal incisions can be made. to deliver the testicle for examination : if the testis is necrotic, perform an orchiectomy. If testis is still in good condition : fix gonad to the scrotal wall with 3-4 non absorbable sutures.
- Testicular prosthesis is usually delayed for 6 months through an inguinal incision.

III. FOLLOW UP

+ Follow up to assess the testis at around 6 months : fertility and testis development).
+ Testicular prosthesis insertion should be discussed and offered after completion of puberty.
+ Local psychology services should be available to the patient if required.

IV. ALGORITHM



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TESTICULAR TRAUMA

HAY VANEL, OUK REAKSMEY, BOU SOPHEAP

Genitourinary (GU) injury is present in approximately 10% of cases of abdominal trauma of those GU injuries, up to 67% involve the external genitalia. GU trauma is more common in malesTesticular trauma is uncommon and rarely necessitates surgical intervention.

Injuries are divided into either blunt or penetrating Initial evaluation and management

+ The first priority is stabilisation of the patient and treatment of associated life-threatening injuries.
+ In any penetrating trauma, tetanus vaccination should be considered.
+ US of the scrotum is the recommended for testicular trauma.
+ Management of testicular trauma based on the classification of scrotum injury of AAST :

Grade	Description
I	Contusion/hematoma
II	Subclinical laceration of tunica albuginea
III	Laceration of tunica albuginea with less than 50% parenchymal loss.
IV	Major laceration of tunica albuginea with 50% or greater than parenchymal loss.
V	Total parenchyma destruction or avulsion

Blunt Scrotal Trauma

+ May result in testicular dislocation, haematocoele, testicular rupture and/or scrotal haematoma.
+ Dislocation of the testicle is rare. Treat by manual replacement and secondary orchidopexy. If manual reposition cannot be performed, immediate orchidopexy is indicated.
+ If haematocele is smaller than three times the size of the contralateral testis-conservative management.

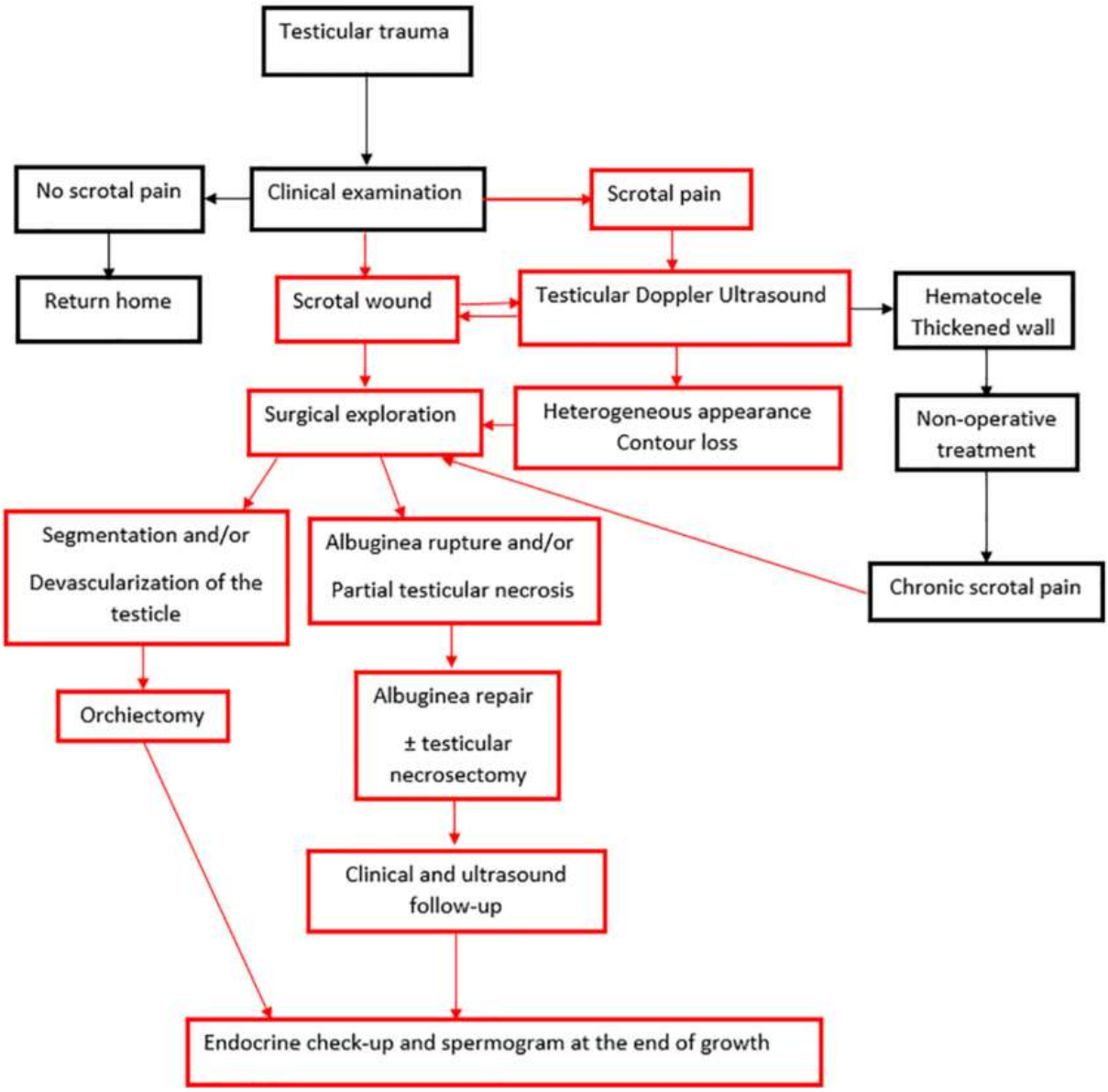
+ If large haematocele - explore.
+ If testicular rupture suspected, explore, evacuate clot and any necrotic testicular tubules and close the tunica albuginea.

Penetrating Scrotal Trauma

+ Surgical exploration with conservative debridement of nonviable tissue.
+ Primary reconstruction of testis and scrotum can be performed in most cases.
+ In complete disruption of the spermatic cord, realignment without vaso-vasostomy may be considered.

+ In extensive destruction of the tunica albuginea, mobilization of a free tunica vaginalis flap can be performed for testicular closure.
+ If reconstruction cannot be achieved, orchiectomy is indicated.

ALGORITHM



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3. EAU

URETHRAL CATHETER CARE

DR. OEUR SOPAGNA, PROF.BOU SOPHEAP, DR.OUK REAKSMEY

I. SUMMARY

Transurethral Catheterization is a type of bladder catheterization procedure involving the insertion of a flexible catheter through the urethra into the bladder.

II. INDICATIONS

2.1-Diagnostic

- Diseases requiring sterile sample collection for urinalysis and/ or urine culture, e.g., UTI
- Diseases requiring measurement of postvoid residual volume, e.g., overflow incontinence
- Patients who require measurement of urinary output, e.g., critically ill patients

2.2-Therapeutic

- Conditions requiring complete or intermittent bladder drainage, e.g., urinary retention, urinary obstruction, neurogenic bladder
- Patients with impaired voiding and/or mobility, e.g., those with paralysis, injury, or receiving end-of-life care
- Bladder access required for treatment, e.g., bladder irrigation for bladder tamponade, intravesical chemotherapy for bladder cancer

III. CONTRAINDICATIONS

- Absolute: none
- Relative
    - Alternative equally effective, less invasive procedures (e.g., clean catch urine, condom catheter) available
    - Acute bacterial prostatitis
    - Known or suspected urethral injury

IV. TECHNICAL BACKGROUND

4.1 Types of transurethral catheters

- Foley catheter
    - A thin, flexible, sterile tube used for continuous drainage
    - Held in the bladder by a water-filled balloon
    - Three-way Foley catheter: a large-gauge Foley catheter with three channels, allowing for bladder irrigation
- Straight urinary catheter: a flexible catheter used for intermittent drainage that is removed after use.
- Coude catheter
    - A thin, flexible catheter with a semirigid curved tip used for both intermittent and continuous drainage
    - Most commonly used if there is difficulty inserting a flexible straight tip catheter (e.g., because of prostatic enlargement)

4.2 Transurethral catheter selection

General catheter recommendations for different patient groups are shown below; catheter size may vary based on patient anatomy.

- Adults: 14–16 Fr straight urinary catheter or Foley catheter

- Patients with prostatic enlargement: 14–18 Fr coude catheter or 18–22 Fr Foley catheter
- Patients with gross hematuria: ≥ 20 Fr three-way Foley catheter

V. LANDMARKS AND POSITIONING

5.1-Landmarks

- Penis
  - Urethral length: ~ 20 cm
  - The urethra curves in an S-shape and passes through the prostate into the bladder.
- Vulva
  - The urethral meatus is located between the labia minora, directly superior to the vagina and inferior to the clitoris.
  - Rarely, locating the urethral meatus via palpation may be necessary.

5.2-Positioning

- Patients with a penis: Place the patient supine and hold the penis taut and upright.
- Patients with a vulva: Place the patient in the frog-leg position.

VI. EQUIPMENT CHECKLIST

The following equipment is included in most prepackaged catheterization kits. Become familiar with the equipment available.

- Sterile gloves
- Sterile drape
- Antiseptic and applicator forceps
- Cotton swabs
- Lubricating jelly and/or viscous lidocaine
- Transurethral catheter
- Syringe containing water or air
- Collection bag or drainage system

VII. PREPARATION

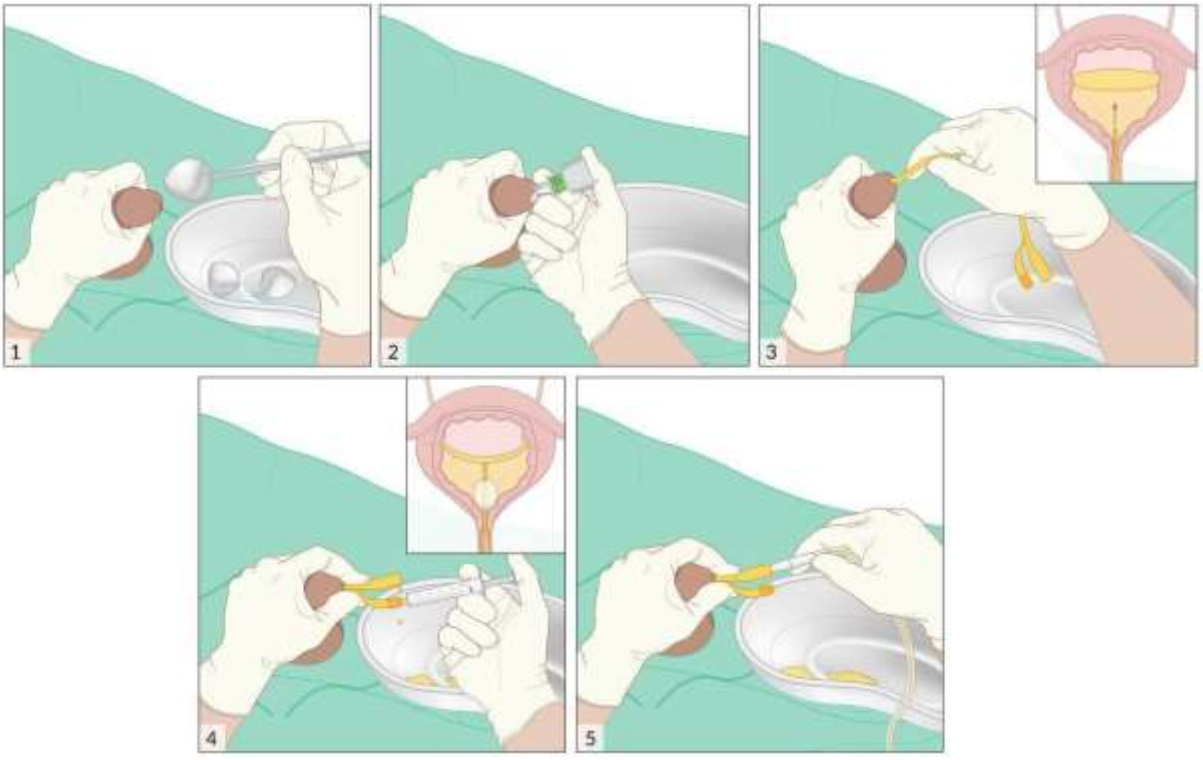
- Gather equipment at the bedside.
- Ensure that the patient is in a comfortable position and that the urethral meatus is easily accessible.
- Put on PPE and place the sterile drape.
- Lubricate the catheter with viscous lidocaine and/or lubricating jelly

VIII. PROCEDURE/APPLICATION

Transurethral catheterization of the penis

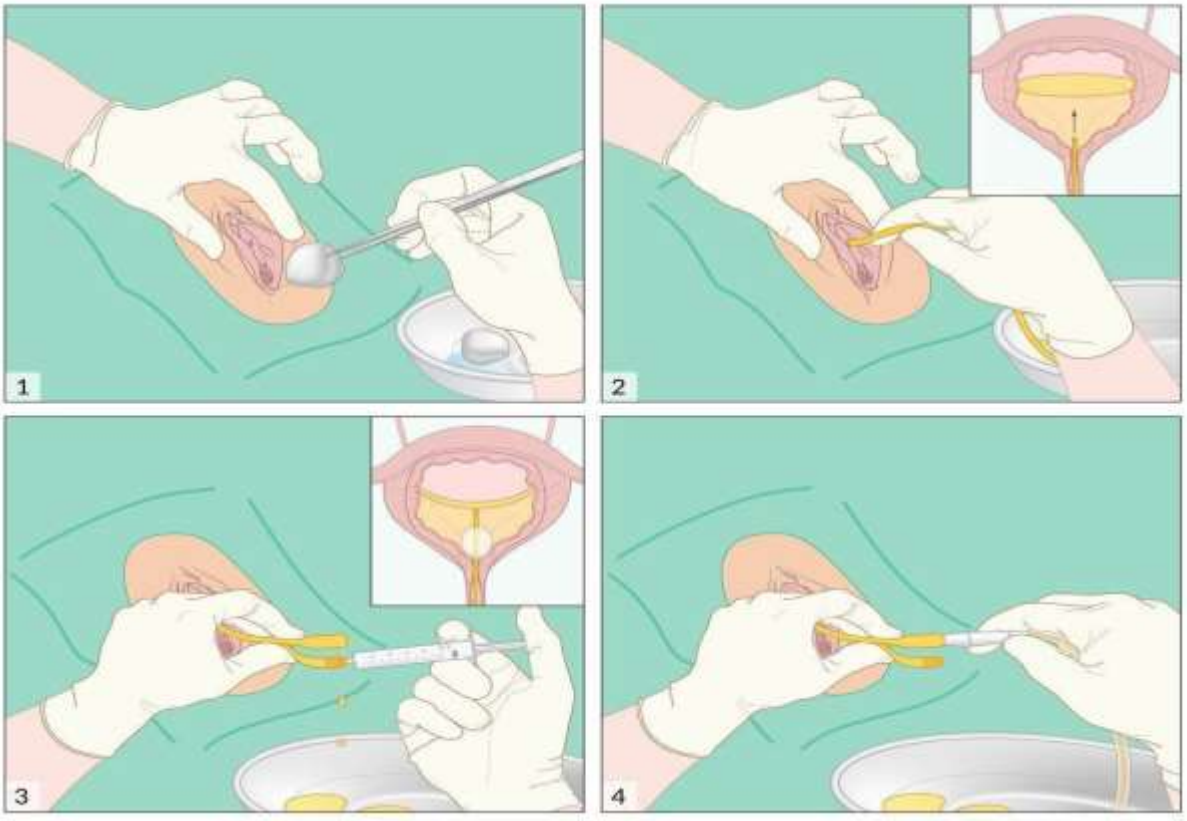
1. Uncircumcised or partially circumcised penis: Retract the foreskin with the nondominant hand.
2. Hold the penis taut and upright.

3. Cleanse the urethral meatus with antiseptic, moving outwards in a circular motion.
4. Inject 5–10 mL of viscous lidocaine into the urethra and allow time for the anesthetic to take effect.
5. Insert the entire length of the catheter into the urethra.
6. Inflate the catheter balloon using a syringe filled with the recommended volume of water or air.
7. Withdraw the catheter slowly until resistance is met.
8. Connect the catheter to a collection bag or drainage system.
9. Reduce the foreskin.
10. Attach the catheter to the patient's thigh using tape or a catheter securement device.



Transurethral catheterization of the vulva

1. Use the nondominant hand to spread the labia.
2. Cleanse the urethral meatus with antiseptic, moving outwards in a circular motion.
3. Pass the catheter into the urethra and slowly advance.
4. Once urine return is noted, advance the catheter multiple centimeters further.
5. Inflate the catheter balloon using a syringe filled with the recommended volume of water or air.
6. Withdraw the catheter slowly until resistance is met.
7. Connect the catheter to a collection bag or drainage system.
8. Attach the catheter to the patient's thigh using tape or a catheter securement device.



IX. POSTPROCEDURAL CHECKLIST

- Urine flowing into the drainage system (i.e., catheter and drainage system clamps open)
- Sterile urine samples obtained and sent for laboratory studies if needed
- Bladder irrigation initiated if necessary
- Patient and/or family educated about catheter care

X. COMPLICATIONS

- Urethral injury
- Hematuria
- Paraphimosis (if the foreskin is not reduced)
- Catheter malfunction (e.g., catheter obstruction)
- Catheter-associated UTI
- Bladder injury
- Prostate injury
- Post-obstructive diuresis
- Electrolyte imbalance after bladder irrigation

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VESICO-URETERAL REFLUX

Dr. OUK REAKSMEY, Dr. HAY VANEL, Dr. PEN MONYRATH, Prof. BOU SOPHEAP

I. CASE DEFINITION

Vesicoureteral reflux (VUR), or the retrograde flow of urine from the bladder into the ureter, is an anatomic and functional disorder that can result in substantial morbidity, both from acute infection and from the sequelae of reflux nephropathy.

II. ETIOLOGY

The cause of the defect in primary reflux is unknown.

The existence of a strong genetic component is indicated by the high rate of reflux in relatives of patients with reflux, but the mechanism of transmission is not clear. Some investigators have favored a polygenic mode of inheritance, whereas others have suggested autosomal or sex-linked transmission with variable penetrance.

III. DIAGNOSTIC PROCEDURE

History

Most children with vesicoureteral reflux (VUR) present in two distinct groups, as follows:

The first group presents with hydronephrosis, often identified antenatally via ultrasonography (US); these children typically progress through evaluation and treatment in the absence of clinical illness

The second group presents with clinical urinary tract infection (UTI)

Even for experienced pediatricians, the diagnosis of UTI in children can be difficult. Children often present with nonspecific signs and symptoms. Infection in infants can manifest as failure to thrive, with or without fever. Other features include vomiting, diarrhea, anorexia, and lethargy.

Older children may report voiding symptoms or abdominal pain. Pyelonephritis in young children is more likely to manifest with vague abdominal discomfort rather than with the classic flank pain and tenderness observed in adults. The presence of fever, while highly suggestive of pyelonephritis, is not reliable enough to lead to the diagnosis.

Physical examination

As with the history, few findings on physical examination suggest VUR or UTI. Fever, flank or abdominal tenderness, or an enlarged palpable kidney may be present. In the absence of reliable historical or physical findings, diagnosis depends on laboratory testing and imaging, as well as family history.

Work up

Diagnosis of urinary tract infection (UTI) depends on obtaining accurate urine culture findings. The criterion standard for obtaining urine specimens remains the suprapubic aspiration. Any growth in such a sample should be considered significant. In practice, however, this procedure is rarely done. Urethral catheterization provides substantially better specificity; more than 1000 colony-forming units (CFU)/mL is considered significant for these samples.

Imaging is the basis of diagnosis and management of vesicoureteral reflux (VUR). The standard imaging tests include renal and bladder ultrasonography (US) and voiding cystourethrography (VCUG), though numerous studies are available. Imaging after the first UTI is indicated in all children younger than 5 years, children of any age with febrile UTI, and boys of any age with UTI. In addition, children with

antenatally identified hydronephrosis should be evaluated postnatally. US performed during the first 3 days of life may have a high rate of false-negative results because of relative dehydration during the neonatal period.
All children with a history of febrile UTI should undergo kidney and bladder US. This allows assessment of the upper tracts for obstruction, renal anomalies and scarring, and other drainage patterns. It does not, however, effectively evaluate for or rule out VUR, and US should not be considered an accurate screening test for findings that would be identified on VCUG. A study by Öztürk et al suggested that preoperative US measurements (specifically, the detrusor-to-ureteral orifice distance and the ratio of the detrusor-to-ureteral orifice distance to the distal ureteral diameter) may be reliable predictors of whether endoscopic subureteric injection therapy will be successful.

Although the 2011/2016 AAP guidelines recommended that US alone should be the initial screening test for children after UTI, the Society for Pediatric Urology continued to recommend that both US and cystography be performed.

Although the traditional approach in children with UTI has been evaluation for VUR with VCUG or radionuclide cystography (RNC), some authorities have advocated a "top-down" approach for children with UTI. In this algorithm, a child with a history of febrile UTI undergoes a dimercaptosuccinic acid (DMSA) renal scan to assess for evidence of kidney involvement, kidney scarring, or both. Negative DMSA scan findings suggest that clinically significant VUR is unlikely, rendering VCUG unnecessary. However, if DMSA scan findings are positive, VCUG is recommended. The merits of alternative approaches to children with UTI are still discussed.

IV. DIFFERENTIAL DIAGNOSIS

- Antenatal Urinary Tract Dilation (Hydronephrosis)
- Myelodysplasia and Neurogenic Bladder Dysfunction
- Pediatric Myelodysplasia
- Pediatric Ureteropelvic Junction Obstruction
- Pediatric Urinary Tract Infection
- Posterior Urethral Valves
- Urethral Anomalies and Urethral Prolapse in Children
- Voiding Dysfunction

V. THERAPEUTIC APPROACH

Controversy persists over the optimal management of vesicoureteral reflux (VUR), specifically with respect to the timing, technique, and benefits of surgical correction. Guidelines have been published by the American Urological Association (AUA).
Guidelines have also been developed by the European Association of Urology (EAU) and the European Association of Paediatric Urology (ESPU).
Febrile urinary tract infection (UTI) with signs of pyelonephritis in children with VUR requires admission and also treatment with parenteral antibiotics to prevent renal damage. This is particularly true in children who are dehydrated, unable to retain oral intake, or in a toxic state.

The need for inpatient admission should be based on the clinical assessment at the time of presentation. Many patients with febrile UTI can be managed as outpatients. Children who are severely dehydrated or in a septic state, as well as those

for whom there are social concerns regarding whether home caregivers can be relied on to care for the ill child properly and completely, should be admitted. Hospitalization after open antireflux surgery typically lasts 24-72 hours. It is increasingly common for children to be discharged home the morning after surgery, and some centers are performing these procedures on an outpatient basis. Generally, children are discharged once they tolerate a regular age-appropriate diet, their pain is managed with oral pain medication, and they are active at an age-appropriate level. Endoscopic antireflux surgery is generally performed as an outpatient procedure.

VI. COMPLICATION

Obstruction after open antireflux surgery

Most cases of postoperative upper tract obstruction are mild, produce no symptoms, and spontaneously resolve. These cases are due to edema at the ureteroneocystostomy site, blood clots, or mucus causing mechanical obstruction. Cases of severe obstruction often have a delayed presentation (1-2 week or longer) and may be associated with flank or abdominal pain, nausea, and vomiting.

US reveals dilation on the affected side, though this can be difficult to assess in patients who had significant dilation preoperatively.

High-grade obstruction is usually due to ischemia of the implanted ureteral segment with resulting fibrosis and stricture. This is a rare complication. Occasionally, patients may present with intermittent obstruction due to kinking of the reimplanted ureter with bladder filling.

Treatment for high-grade obstruction is surgical revision of the obstructed system. Percutaneous nephrostomy for temporary drainage may be required if the patient is symptomatic or in a toxic state.

Persistent vesicoureteral reflux after antireflux surgery

Open surgery

Modern series consistently report success rates greater than 95% for antireflux surgery. When VUR persists postoperatively, initial observation with continued antibiotic prophylaxis is indicated. Reoperation is generally reserved for patients with persisted febrile UTI despite prophylaxis.

A very high percentage of patients in whom surgery has failed have voiding dysfunction. Urodynamic evaluation should be considered in these patients, especially if reoperation is considered. Even so, a substantial majority of patients with reflux at the first postoperative study have complete resolution at the 1-year follow-up point.

Endoscopic surgery

Initial management is often repeat injection. Many investigators report routinely injecting as many as three separate times. Patients in whom multiple injections fail should be reevaluated and treated for causes of secondary VUR. Patients with persistent VUR and indications for surgical correction should proceed to open surgery.

New contralateral vesicoureteral reflux after unilateral antireflux surgery

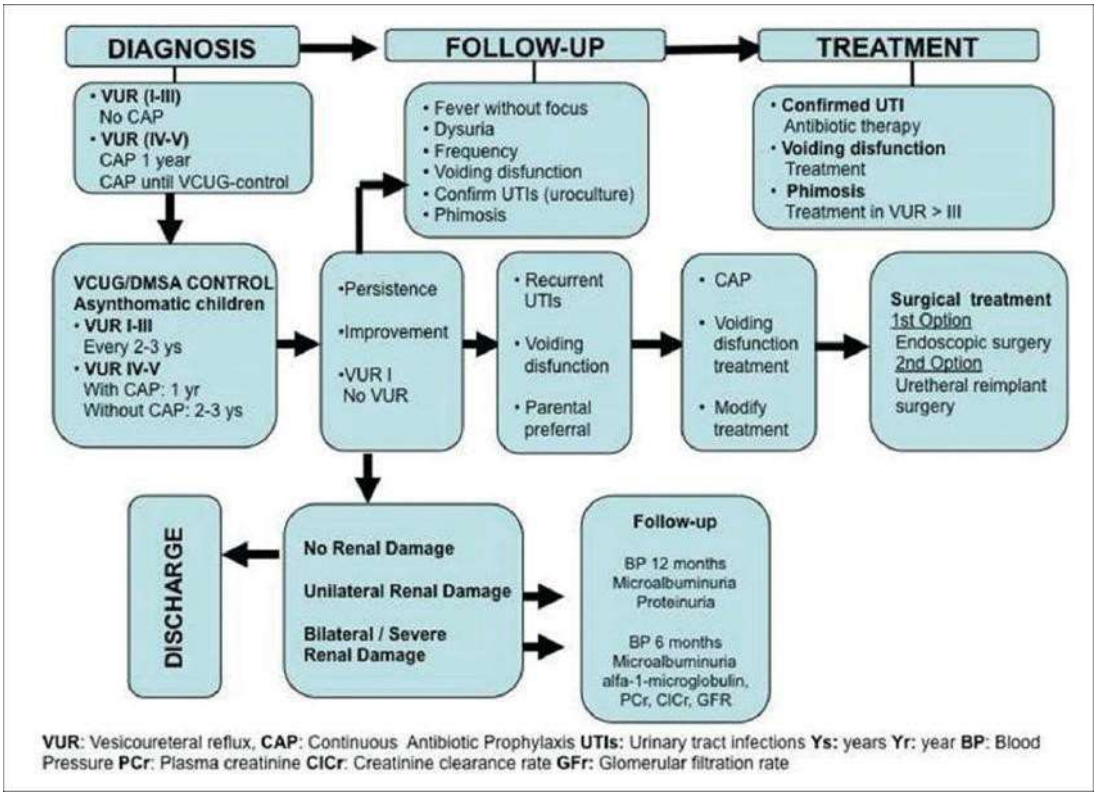
New onset of VUR in a renal unit that had no VUR on preoperative imaging occurs in 10-32% of patients after open correction and 7-14% of patients after endoscopic correction. In general, the new VUR is thought to be of low grade and may be more likely to resolve spontaneously.

VII. FOLLOW UP

Children whose VUR is being managed medically are regularly seen on an annual basis. Routine evaluation includes urinalysis and urine culture, appropriate imaging, and blood pressure measurement. Parents must understand the need for proper evaluation and urine culture if they suspect UTI. In some cases, parents are taught to perform urinalysis at home. Positive home urinalysis results should prompt formal testing at a physician's office.

After surgical correction of VUR, patients are seen in the clinic 2-6 weeks after discharge with renal US or renal scintigraphy to exclude upper-tract obstruction. Patients continue taking prophylactic antibiotics until a second return visit 3-6 months postoperatively, at which time VCUG or nuclear cystography is performed.

VIII. ALGORITHM



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VESICO-VAGINAL FISTULA

HAY VANEL, BOU SOPHEAP, OUK REAKSMEY

I. CASE DEFINITION

Fistula is an abnormal epithelialised tract between 2 epithelialised surfaces VVF common in developing countries due to birth trauma; uncommon in developed world – typically iatrogenic

II. ETIOLOGY

Congenital
Acquired
-Iatrogenic
-Surgical:
-Hysterectomy*
-Anteriorcolporraphy
-Colposuspension
-Subtrigonal phenol Radiotherapy
-Non-iatrogenic
-Advancedpelvicmalignancy
-Tuberculosis
-Obstructed labour
-Foreign body erosion *

Hysterectomy accounts for ~ 90% of iatrogenic causes. Bladder injury complicates 0.5-1% of all hysterectomies. Incidence of fistula 0.1%. Fistula 3 x more common with abdominal than with vaginal hysterectomy. NB. In the setting of a difficult hysterectomy, ureteric injury is the least likely cause of urinary fistula.

III. DIAGNOSTIC PROCEDURE

Persistent dribbling incontinence 'Serous' discharge and failure to progress after gynae op Occasionally normal voiding and small loss per vaginum

History
Gynae (malignancy, RT, surgery, endometriosis, cervical Rx) Obstetric (obstructed labour, caesarian) Urology (malignancy, RT, surgery, neurogenic bladder) Examination
-Fluid for U+E
-Speculum vaginal examination (Cusco)
-Flexible cystoscopy
-Three pad dye test occasionally helpful for occult cases. [Methylene blue instilled into bladder. Staining of upper/mid pads suggests VVF, staining of lower pad SUI. Attempts to use IV dye to identify ureteric involvement innacurate and does not obviate requirement for RPG]
-EUA, vaginoscopy, cystoscopy and bilateral RPG prior to contemplating repair (biopsy of the fistula edge mandatory in all patients with previous or suspected malignancy) Vesico-vaginal Fistula Tom Walton January 2011 2
-CT urogram with delayed images or VCUG for complex/occult cases

IV. THERAPUETIC APPROACH

i- Conservative

Prolonged catheter drainage

Appropriate for surgically unfit patients May occasionally suffice for patients with small nonepithelialised uncomplicated (no RT, malignancy, ischaemia TB) fistula following hysterectomy (give Abx: quote 10% cure rate) Unlikely to heal if remain open after 3 weeks of catheter drainage

De-epithelialisation by curretage, silver nitrate, transvesical diathermy (Bugbee) and metal screws all tried followed by catheter drainage. Generally poor results (<10%)with established fistula Nephrostomy for urinoma, obstruction , ureteric fistulae

(ii) Surgical

Standard surgical principles important: tension-free well vascularised anastomosis with avoidance of overlapping suture lines

Remember SNAP:

S – eradicate sepsis

N – ensure adequate nutrition (?pre-op topical oestrogen) A – define anatomy

P – determine surgical plan if unexpected problems Timing of surgery

Iatrogenic 2-3 weeks*

Obstetric injury 3-6 months

Radiation fistula 12 months + (allows tissue healing/angiogenesis following obliterative endarteritis)

* Traditional teaching recommended a delayed period for all fistulas. However early repair at 2-3 weeks believed to be equivalent to delayed repair, and reduces psychological and therefore medico-legal 'complications'

However best chances of repair = first chance. Therefore:

< 72 hrs immediate repair

> 72 hrs 6-8 wks delayed uncomplicated

6 months baby/infected

12 months radiotherapy

Transvaginal and abdominal approaches described. In experienced hands minimally invasive TV approach a/w equivalent success rates (82-100%); depends on surgeons preference Vaginal repair

Labial stitches

Ring retractor

Weighted Simms speculum

Interposition with Martius fad pad

Problems with supply of blood/proliferation Difficult to get Martius high (but dual supply - can divide below and rotate from above) Vesico-vaginal Fistula Tom Walton January 2011 3 Alternative coverage with gracilis, gracilis-based myocutaneous flap, labial or gluteal flaps

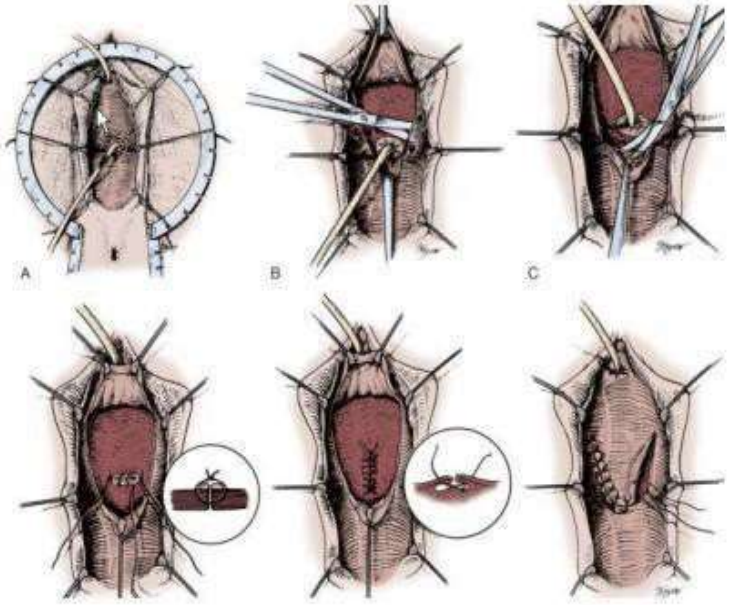
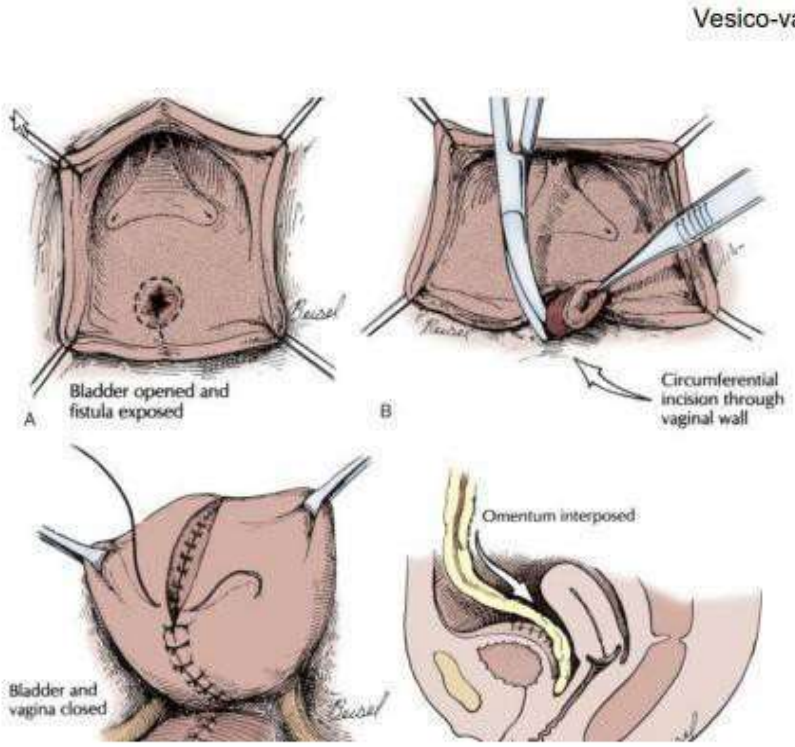


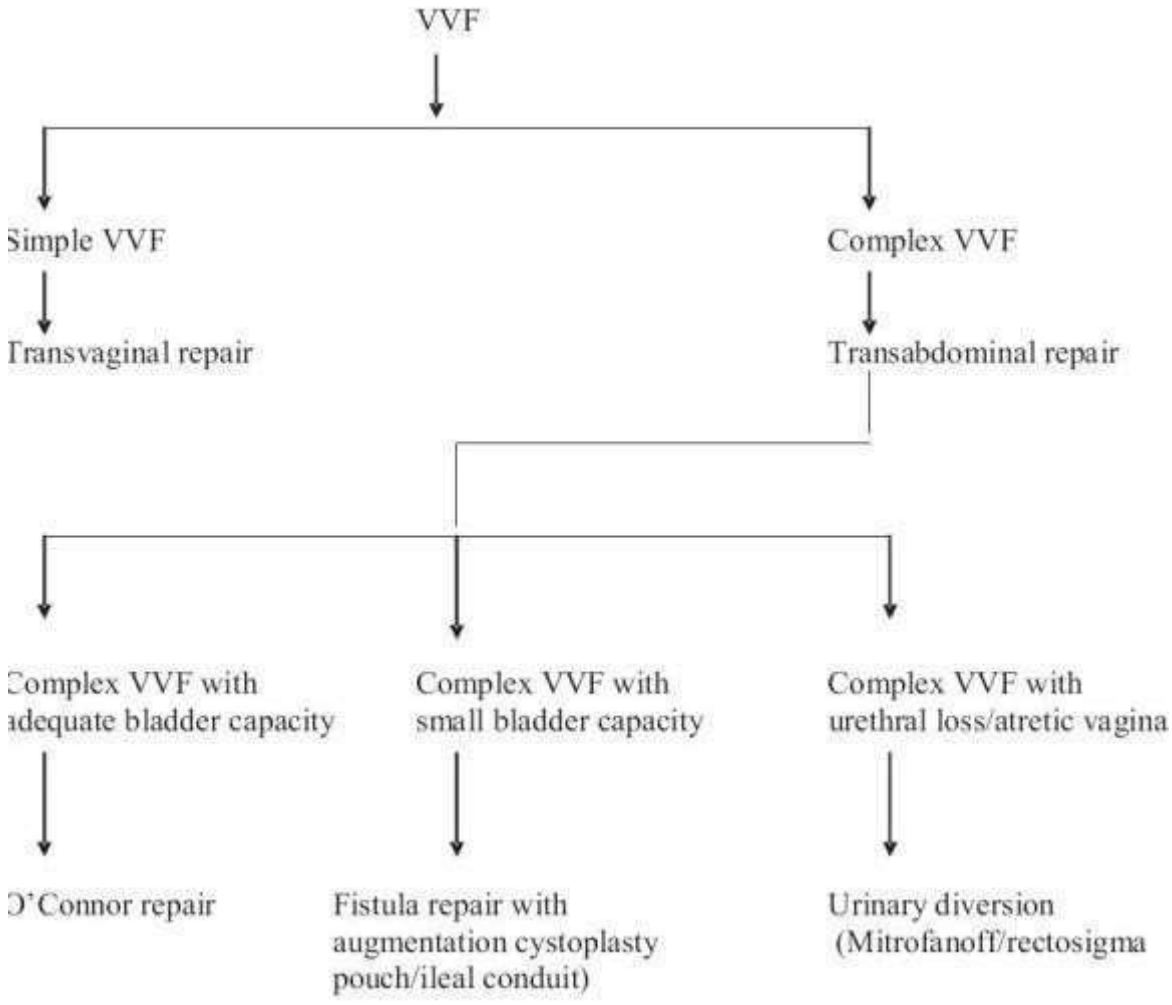
Figure 7-12 Technique of vaginal repair of a post-hysterectomy VVF. **A,** Retraction including ring retractor, vaginal speculum, and Foley catheter in the VVF track. A Foley catheter is seen in the VVF track providing traction on the vaginal cuff. **B,** Mobilization of anterior vaginal wall flap. Lateral flaps are developed as well, thereby isolating the VVF track. **C,** Mobilization of posterior vaginal wall flap. **D,** Initial layer of closure is performed without excising the edges of the fistula track. **E,** The perivesical fascia is closed with Lembert-type sutures. This line of closure is perpendicular to the initial suture line. **F,** The vaginal wall flaps are advanced to avoid overlapping suture lines. *(From Ganabathi K, Siris L, Zimmern P, Leach GE: Vesicovaginal fistulas: Reconstructive techniques. In McAninch J, ed: Traumatic and Reconstructive Urology. Philadelphia, WB Saunders, 1996:317.)*



V. COMPLICATION

Complications include recurrence, vaginal shortening/stenosis and ureteric injury

VI. ALGORITHMS



VII. CONCLUSION

After stone passage, every patient should be assigned to a group with low or high risk of stone formation. For correct classification, reliable stone analysis and basic evaluation of every patient are required. Low-risk stone formers may benefit by adopting general preventive measures regarding fluid and nutritional intake, as well as lifestyle improvements. For high-risk stone formers, a specific metabolic evaluation is required to guide individual treatment and prevent stone recurrence.

Follow up for recurrence stone:

Low risk patient: follow up every 12 months evaluation (Urinalysis, renal function test, KUB ultrasound/X-ray)

High risk patient: follow up every 6 months evaluation (Urinalysis, renal function test, KUB ultrasound/X-ray with specific tests)

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RENAL TRAUMA

HAY VANEL, OUK REAKSMEY, BOU SOPHEAP

I. ZNTRODUCTION

Renal trauma can cause injury to the parenchyma or renal vessels, causing bleeding, or injury to the collecting system with urine extravasation. Among genitourinary (GU) trac injuries, which are rare, the kidneys are most commonly injured.

The management of traumatic renal has evolved with time, with an increasing emphasis on nonsurgical management, particularly for blunt renal injuries. This change came about from the recognition that urgent surgical exploration of renal injuries frequently led to nephrectomy and that angioembolization to treat bleeding is highly successful for renal salvage. While nonoperative management of low-grade blunt renal injuries is the standard of care, nonoperative management of high-grade blunt injuries and penetrating renal injuries is controversial.

II. EPIDEMIOLOGY

Renal trauma is present in to up 5% of all trauma cases. It is most common in young males and has an overall population incidence of 4.9 per 100,000.

III. ETIOLOGY

- **Blunt injuries**: Rapid deceleration (eg, motor vehicle crash, fall from heights); direct blow to the flank (eg, pedestrian struck, sports injury)
- **Penetrating injuries:** Penetrating (eg, gunshot wounds, stab wounds)
- **Iatrogenic** (eg, endourologic procedures, extracorporeal shock- wave lithotripsy, renal biopsy, percutaneous renal procedures)

IV. DIAGNOSIS

1. History

- **Detailed** history of the trauma from the patient or from the witnesses and emergency personnel is an essential part of patient evaluation.
- **Preexisting** renal disease or abnormalities (eg, ureteropelvic junction obstruction, renal cysts, kidney stones, past surgery), renal anomalies, and solitary kidneys should be documented.

2. Physical Examination

- Ecchymosis in the flank or upper quadrants of the abdomen is often noted.
- Most important indicator of renal trauma is gross or microscopic hematuria but the absence of hematuria, although rare, does not exclude renal injury and it is absent in 5% of patients.

- Retroperitoneal bleeding may cause abdominal distention, ileus, and nausea and vomiting.

3. Laboratory Studies

- The complete blood count to obtain hematocrit level and platelet count
- The prothrombin time and activated partial thromboplastin time
- Serum creatinine
- Urinalysis to diagnose hematuria
- Blood grouping and cross match.

4. Imaging: Criteria For Radiographic Assessment

The goals of imaging are to grade the renal injury, document pre- existing renal pathology, demonstrate presence of the contralateral kidney and identify injuries to other organs. In patients who have not had any imaging the indications for renal imaging are:

- visible haematuria
- non-visible haematuria and one episode of hypotension
- a history of rapid deceleration injury and/or significant associated injuries
- penetrating trauma
- clinical signs suggesting renal trauma e.g. flank pain, abrasions, fractured ribs, abdominal distension and/or a mass and tenderness

a. Computed tomography*

Computed tomography is the imaging modality of choice in stable patients. It is quick, widely available, and can accurately identify grade of renal injury, establish the presence of the contralateral kidney and demonstrate concurrent injuries to other organs.

b. Ultrasonography (US)

In the primary survey of a critically injured patient, FAST (Focused Assessment Sonography in Trauma) is used to identify hemoperitoneum as cause of haemorrhage and hypovolemia. However, it is not routinely used for the assessment of solid organ injury as it is insensitive, operator dependant, does not define the injury well, and inferior to CT. **It is an option for follow- up**.

c. Intravenous pyelography (IVP)

Intravenous pyelography has been superseded by cross-sectional imaging and should only be performed when CT is not available. **One-shot intra-operative IVP** can be used to confirm the presence of a functioning contralateral kidney in patients too unstable to have had pre-operative imaging. The technique consists of a bolus intravenous injection of 2 mL/kg of radiographic contrast followed by a single plain film taken after ten minutes.

d. Magnetic resonance imaging (MRI)

The diagnostic accuracy of MRI in renal trauma is similar to that of CT. However, the logistical challenges of MRI make this modality impractical in acute trauma.

V. CLASSIFICATION

The most commonly used classification system is that of the **AAST**. It is validated and predicts morbidity and the need for intervention. This remains the most useful of urological trauma classifications.

Grade*	Description of injury
1	Contusion or non-expanding sub-capsular haematoma
	No laceration
2	Non-expanding peri-renal haematoma
	Cortical laceration < 1 cm deep without extravasation
3	Cortical laceration > 1 cm without urinary extravasation
4	Parenchymal laceration: through corticomedullary junction into collecting system Or Vascular: segmental renal artery or vein injury with contained haematoma, or partial vessel laceration, or vessel thrombosis
5	Parenchymal: shattered kidney Or Vascular: renal pedicle or avulsion

**Advance one grade for bilateral injuries up to grade 3.*

VI. DISEASE MANAGEMENT

a. Non-Operative Management

This includes patients who are initially hemodynamically stable (systolic blood pressure >90 mmHg) identified with renal trauma on imaging, as well as those who are stabilized in the operating room through the management of other injuries and identified during trauma laparotomy as having a nonexpanding perirenal hematoma. Stability is defined as a lack of clinical signs of shock, and stable serial hematocrit values during monitoring.

Blunt renal injuries:

- **Grade 1 - 3 injuries** are managed non-operatively.
- **Grade 4 injuries** are also mostly treated conservatively, but the requirement for subsequent intervention is higher.
- **Grade 5 injuries** often present with haemodynamic instability and major associated injuries. There is thus a higher rate of exploration and nephrectomy.
- Persistent urinary extravasation from an otherwise viable kidney after blunt trauma often responds to stent placement and/or percutaneous drainage.

Penetrating renal injuries:

Penetrating abdominal wounds have traditionally been managed surgically. However, selective non-operative management of penetrating abdominal wounds is now accepted following detailed assessment in stable patients.

- **Low-**grade **(Grade I,II)** can be managed non-operatively in stable patients and all of the following:
  - Absence of major blood loss
  - Absence of major renal parenchymal injury
  - Absence of renal vascular injury

- Absence of associated intra-abdominal injury
- Grade **3 or higher** lesions due to stab wounds in stable patients can be managed expectantly, but warrant closer observation as the clinical course is more unpredictable and associated with a higher rate of delayed intervention.
- Overall, non-operative management of penetrating injuries in selected stable patients is associated with a successful outcome in up to 50% of stab wounds and up to 40% of gunshot wounds.

Selective angioembolization:

- Selective angioembolisation (AE) has a key role in the non- operative management of blunt renal trauma in haemodynamically stable patients.
- Accepted CT findings indicating the need for AE are **active extravasation of contrast, arteriovenous fistula and pseudo-aneurysm.**
- Angioembolisation has been utilised in the non-operative management of all grades of renal injury; however, it is likely to be most beneficial in the setting of high-grade renal trauma (AAST > 3).

Repeat imaging (early)

- For patients who are managed conservatively, the **American Urological Association Urotrauma guidelines** recommend a repeat contrast-enhanced CT scan at 48 to 72 hours if clinical signs that suggest complications.
- Clinical signs during the period of observation that suggest a missed renal injury include **progressively worsening flank pain, fever, persistent blood loss, abdominal distention, ileus, or hemodynamic instability**.
- Repeat imaging can be safely omitted for patients with **Grade 1-3 injuries** as long as they remain clinically well.

b. Surgical Management

Renal exploration may be necessary at the time of the initial trauma presentation because of hemodynamic instability (eg, **expanding/pulsatile zone II hematoma, grade V renal injury**) or **penetrating injury with active bleeding**, or subsequently during the course of conservative management.

Indications for renal exploration

Absolute	*Relative*
▪ Hemodynamically unstable from renal bleeding ▪ Renal vascular injury in solitary kidney ▪ Pulsatile or expanding flank mass	▪ Urinary extravasation ▪ Laparotomy for other injury ▪ Major renal injury ▪ Nonviable renal tissue

VII. COMPLICATIONS

- **Early (< 1 month) complications** include bleeding, infection, perinephric abscess, sepsis, urinary fistula, hypertension, urinary extravasation and urinoma.

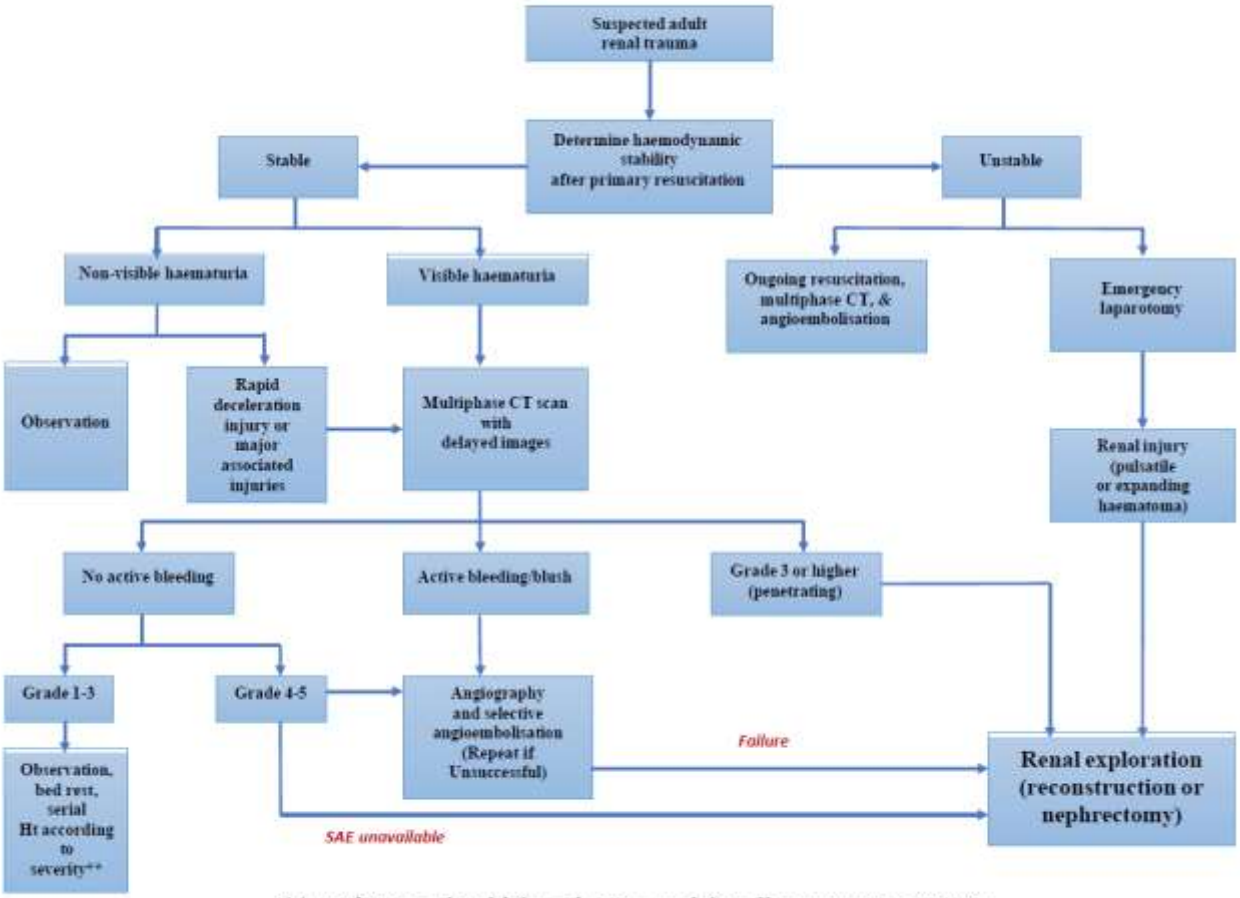
- **Delayed complications** include bleeding, hydronephrosis, calculus formation, chronic pyelonephritis, hypertension, arteriovenous fistulae (AVF) and pseudo-aneurysms.

VIII. FOLLOW-UP

Follow-up approximately three months after major renal injury with:

- physical examination
- urinalysis
- blood pressure measurement;
- renal function tests.
- Ultrasound can be used to define the post-injury anatomy

Management of renal trauma algorithms



**Antibiotics should for administered for all penetrating injuries

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STRESS URINARY INCONTINENCE

DR. OUK REAKSMEY, PROF BOU SOPHEAP

I. CASE DEFINITION

Urinary incontinence (UI) is the complaint of any involuntary leakage of urine. It results from a failure to store urine during the filling phase of the bladder due to dysfunction of the bladder smooth muscle (detrusor), urethral sphincter, or anatomical abnormalities (congenital or acquired). Urine loss is either urethral or extraurethral (i.e. due to ectopic ureter or vesicovaginal fistula).

II. ETIOLOGY

2.1- Specifi c risk factors for female SUI

- Childbirth (increased risk with vaginal delivery, forceps delivery).
- Ageing.
- Oestrogen withdrawal.
- Previous pelvic surgery.
- Obesity.

2.1.2- Specifi c risk factors for male SUI

External urethral sphincter damage (from pelvic fracture, prostatectomy, pelvic surgery, or radiotherapy).

2.1.3- Other risk factors

Neurological disorders causing sphincter weakness (SCI, multiple sclerosis, spina bifi da).

III. DIAGNOSTIC PROCEDURE

3.1- Women

- Stress test: a leakage of urine from the urethra on cough denotes a positive test.
- Pad test: number and weight of pads used to estimate urine loss.
- Pelvic exam: check for pelvic organ prolapse (POP). Elevation of an existing anterior wall prolapse will unmask any occult sphincter incompetence in those who are continent as a result of obstruction caused by the prolapse. Assess oestrogen status and requirement for topical oestrogen treatments.
- Q-tip test: although not performed routinely, the Q-tip angle is a measure of urethral mobility in women. With the patient in lithotomy position and the bladder comfortably full, a well lubricated sterile cotton-tipped applicator is gently inserted through the urethra into the bladder. Once in the bladder, the applicator is withdrawn to the point of resistance which is at the level of the bladder neck. The resting angle from the horizontal is recorded. The patient is then asked to strain and the degree of rotation is assessed. Hypermobility

is defined as a resting or straining angle of greater than 30* from the horizontal.

- Urethral pressure profi le (selected cases only): microtransducers are mounted in a catheter that is placed into the bladder, then slowly withdrawn, measuring intraluminal urethral pressures. A measure of urethral closure pressure can be obtained.
- Urodynamics: recommended for women before SUI surgery if:[1]
  - There is suspicion of concomitant detrusor overactivity.
  - History of previous surgery for SUI or anterior compartment prolapse.
  - Symptoms of voiding dysfunction.

3.2-Men

- Abdominal exam to detect a palpable bladder.
- External genitalia exam to assess for penile abnormalities.
- DRE.
- Flow rate and PVR.
- Consider imaging of upper tracts if evidence of BOO.

IV. DIFFERENTIAL DIAGNOSIS

The estimated prevalence for the types of urinary incontinence are as follows:[3]

- Stress urinary incontinence – 24% to 45% in women over 30 years
- Urge urinary incontinence – 9% in women 40 to 44 years; 31% in women over 75 years; 42% in men over 75 years
- Mixed urinary incontinence – 20% to 30% of those with chronic incontinence
- Overflow urinary incontinence – 5% of those with chronic incontinence
- Functional urinary incontinence – Uncertain

The mnemonic DIAPPERS can be used as an aid to develop a differential diagnosis for reversible causes of urinary incontinence:[3]

- Delirium, dementia, or other cognitive impairments
- Infection (urinary tract infection)
- Atrophic vaginitis or urethritis
- Pharmaceuticals or substances (e.g., diuretics, caffeine, alcohol)
- Psychological disorder
- Excessive urine output (e.g., diabetes, diabetes insipidus)
- Reduced mobility or reversible urinary retention
- Stool impaction

Other conditions to consider include:

- Neurologic conditions such as spinal cord injuries, cauda equina syndrome, multiple sclerosis, cerebral vascular accidents, normal pressure hydrocephalus, spinal stenosis

- Renal or ureteral calculi
- Intraabdominal or pelvic mass
- Anatomic abnormalities such as urogenital fistulas, diverticula, and ectopic ureters (though these are less common)[16]

V. THERAPEUTIC APPROACH

5.1- Conservative treatment

- Pelvic fl oor muscle training (PFMT): for a minimum of 3 months is the fi rst-line treatment, performing at least eight contractions, three times per day. PMFT improve symptoms in 30% of women with mild SUI.
- Lifestyle modifi cation: weight loss, stop smoking, avoid constipation, modify fl uid intake.
- Biofeedback: the technique by which information on ability and strength of pelvic fl oor muscle contraction is presented back to the patient as a visual, auditory, or tactile signal. Patients may also be helped by the perineometer which measures pelvic fl oor contraction.
- Medication: duloxetine inhibits the reuptake of both serotonin and noradrenaline. It is given orally 20–40mg twice daily and acts to increase sphincteric muscle activity during bladder fi lling. Recommended as an alternative to surgery rather than fi rst-line treatment due to adverse effects.[1]
- Extracorporal magnetic innervation: involves sitting the patient in a chair and using a pulsed magnetic fi eld to stimulate the nerves of the sphincter and pelvic fl oor. Possible benefi t in mixed incontinence.
- High frequency electrical stimulation: produces contraction of the pelvic fl oor (35–50Hz). No proven therapeutic benefi t in SUI.

5.2- Surgical treatment

- Urethral bulking agents
- Retropubic suspension
- Suburethral slings
- Artifi cial urinary sphincters

VI. COMPLICATION

Complications related to urinary incontinence include:[10][14][21]

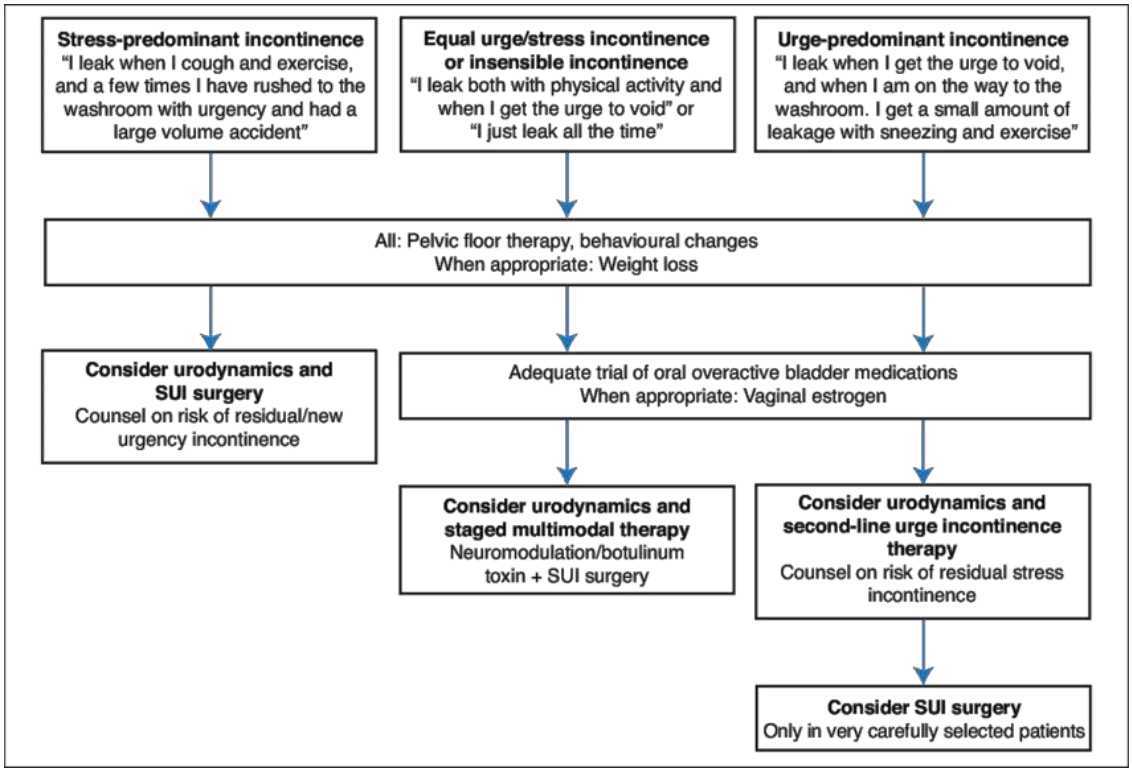
- Urinary tract infections
- Renal dysfunction secondary to obstructive uropathy
- Cellulitis
- Pressure ulcers
- Medication side effects
    - Alpha-adrenergic agonists side effects:[14] dry mouth, restlessness,

hypertension, insomnia

- Duloxetine:[22] dry mouth, nausea, fatigue, constipation, hyperhidrosis
- Antimuscarinic side effects:[17] dry mouth, constipation, blurred vision, dry eyes, fatigue, difficulty in micturition, palpitations
- Mirabegron:[23] urinary tract infections, hypertension, dry mouth
- OnabotulinumtoxinA injection:[22] urinary tract infections, urinary retention
- Alpha-adrenergic antagonists:[22] hypotension, dizziness, fatigue, sedation

- Trauma and infection due to catheterization
- Worsening of urinary incontinence after surgical intervention
- Increased risk of falls and subsequent fractures
- Decreased physical activity
- Sexual dysfunction
- Depression
- Social isolation
- Increased caregiver burden

VII. ALGORITHM



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UNDESCENDED TESTIS

Dr. HAY VANEL, Dr. OUK REAKSMEY, Prof. BOU SOPHEAP

I. 1.CASE DEFINITION

Terms such as undescended testis, retentio testis, cryptorchidism, and maldescended testis describe a testis that is not normally located at the bottom of the scrotum

II. ETIOLOGY

- intrauterine growth restriction (IUGR),
- prematurity – incidence in premature infants 30%,
- first- or second-born boys,
- perinatal asphyxia,
- Cesarean section,
- toxemia of pregnancy,
- congenital subluxation of hip,
- seasonal (especially winter).

III. DIAGNOSTIC PROCEDURE

Medical history

The interview should include the data on the course and duration of pregnancy, medication used and exposure to environmental toxins, as well as birth weight, position of testes at birth, other defects and diseases of the child and family history .

Physical examination

Palpation is a basic technique to examine UDT. It allows differentiation between palpable and nonpalpable, retractile and gliding testes [7]. It is mandatory to assess the appearance of external genitalia to exclude DSD. A patient should be examined in both supine and standing (older boys) position in a warm room, with warm hands.

Gonads should be carefully examined for size, turgor, any palpable paratesticular anomalies, and the presence of hernia or hydrocele .

Imaging

Accurate assessment of the position of the UDT and its volume compared with the contralateral, healthy testis gives the surgeon a basic knowledge in cryptorchid boys. Different imaging techniques have been evaluated for the assessment of UDT [68, 69]:

- ultrasonography (US) – good to assess the size of inguinal testes, less reliable for abdominal testes,

- computed tomography (CT) – may be helpful for bilateral impalpable testes; performed under general anesthesia in young children,
- magnetic resonance imaging (MRI) – may be helpful for bilateral impalpable testes; performed under general anesthesia in young children; the least invasive, most expensive,
- venography, angiography – invasive, difficult to perform, high rate of complications; not useful in children.

IV. DIFFERENTIAL DIAGNOSIS

- Retractile testis
- Anorchia
- Intra-abdominal testis
- Vanishing testis syndrome or nubbin testicle resulting from perinatal torsion

V. THERAPEUTIC APPROACH

There are two basic modes of treatment of UDT used for many years and accepted all over the world: hormonal and surgical. They can be used alone or as complementary methods [1–9]. The main goal of UDT treatment is to pull the testis down to the scrotum. This should be done for the following reasons:

- to prevent the impairment of spermatogenesis,
- to prevent, or at least decrease, the risk of TGCN,
- to facilitate future examination of the testicle (palpation, US),
- to correct the inguinal hernia frequently accompanying UDT,
- to minimize the risk of torsion of the testis, which is increased in infants with UDT due to the greater mobility of the inguinal testis and patent processus vaginalis. Diagnosis and surgery are often delayed, with a high rate of orchiectomies .

Hormonal treatment

The hormonal treatment of UDT is based on the hypothesis of deficiency of the hypothalamic-pituitary-testicular axis at the end of gestation or shortly after birth, and hence the lack of the ‘mini-puberty’. Hormonal therapy is usually carried out using hCG,

gonadotropin releasing hormone (GnRH, luteinizing hormone releasing hormone – LHRH) or a combination of both. It can be administered as a neoadjuvant therapy prior to the orchiopexy or as a supplementary treatment after early surgery for UDT .

The first method of hormonal therapy was hCG administration, advocated in boys with UDT in the 1950s, with some of the treatment series dated as early as the 1930s .The hCG is produced by the syncytiotrophoblast and stimulates testicular Leydig cells to produce testosterone. As androgen takes part in the process of testicular descent it seems justified to stimulate its production. Evidence for the beneficial role of the hormonal therapy to improve testis position has been reported . Treatment with hCG is still used; however, in the 1990s and 2000s critical studies and metaanalyses of UDT treatment with hCG and its adverse effect on future reproductive function in adults appeared .

GnRH therapy was first administered in boys with UDT in the 1970s . GnRH is produced by the hypothalamus and stimulates the anterior pituitary gland to secrete LH and follicle-stimulating hormone (FSH). FSH stimulates the proliferation and differentiation of spermatogonia. GnRH therapy may improve germ cell number, maturation and later semen parameters in boys with UDT. The combined administration of GnRH and hCG in boys younger than 1 year can be beneficial for spermatogonial transformation and proliferation, with a success rate of about 20%.

The dose of hormonal therapy is usually as follows :

- GnRH – 3 × 400 μg/day (i.e. 3 × daily one puff of 200 μg into each nostril) over 4 weeks as nasal spray,
- hCG – 50 IU/kg body weight in intramuscular injection twice a week for 3–5 weeks (total dosage of 6,000–9,000 IU).

Surgical treatment (orchiopexy)

Nowadays, the surgical therapy for the palpable UDT is orchiopexy with creation of a subdartos pouch . Fixation is achieved by the scarring of the everted tunica vaginalis to the surrounding tissues. The Bianchi single high scrotal incision is an optional technique for orchiopexy in boys with UDT situated distal to the external inguinal ring [. Theretroperitoneal dissection is however crucial for the success of any surgical procedure .

When the testis is non-palpable, diagnostic laparoscopy through an umbilical port is the procedure of choice. If the testicular vessels exit through the internal ring, an inguinal incision allows one to locate the testis (orchiopexy) or its remnants (removal and histopathologic examination).

Timing of orchiopexy

In the 1950s, orchiopexy was recommended in boys aged 10–15 years ,in the 1970s in 5–6- year-old boys . During the 1970s and early 1980s the age of orchiopexy declined to 2 years of age . Currently orchiopexy is recommended between 6 and 12–18 months . The main goal of this timing of orchiopexy is to prevent the impairment of spermatogenic function and decrease the risk of TGCT in adult life. Although many researchers have reported the beneficial role of early orchiopexy in preventing these problems, there is still a need for large prospective studies providing more clinical evidence .

Life and clinical practice verify all the recommendations, and numerous studies show that the mean age of boys with UDT at the time of surgery is well above this recommended age and has not decreased significantly during the last decade. However, the risk of poor sperm count is probably independent of the age of surgery, but it is correlated with the number of gonocytes and spermatogonia .

VI. COMPLICATION RENAL FAILURE

Intraoperative (rare):

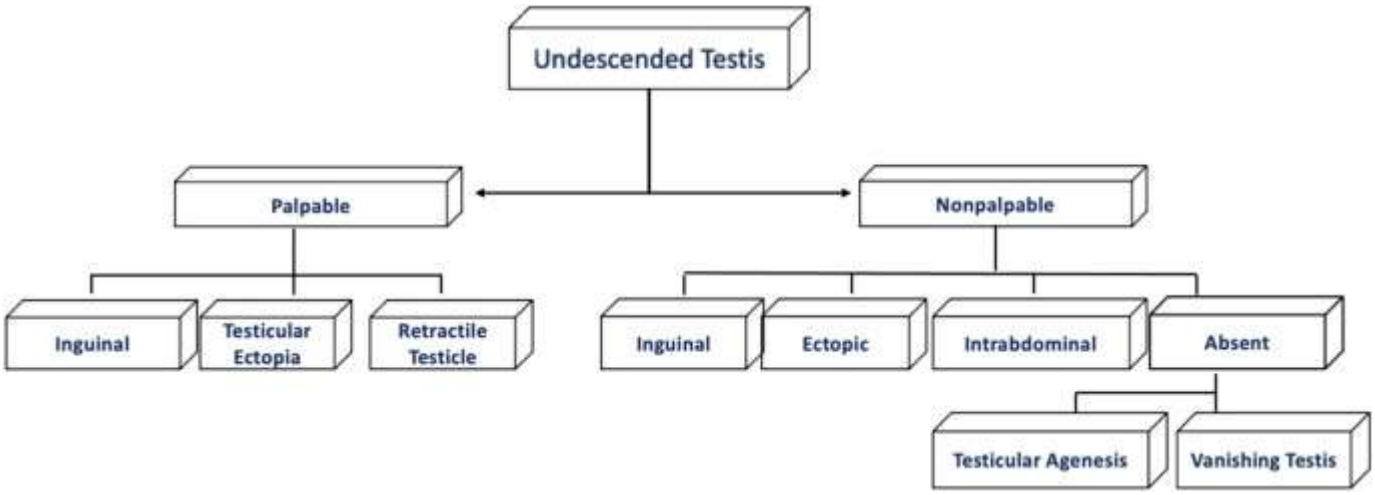
- ilioinguinal nerve injury,
- damage to the vas deferens. Postoperative early:
- hematoma formation,
- wound infection. Postoperative late:
- testicular atrophy,
- testicular retraction (ascent, acquired UDT),
- postoperative torsion (either iatrogenic or spontaneous).

VII. TESTICULAR BIOPSY

Biopsy of UDT for CIS detection is generally not recommended in childhood. Intraoperative testicular biopsy in children is controversial and at present reserved for use in patients with ambiguous genitalia, chromosomal disorders or as part of clinical studies

It is difficult to estimate the risk-to-benefit ratio of this procedure, especially taking into consideration the potential risk of worsening the function of an already compromised testis. Nevertheless, recent reports show that testicular biopsy in prepubertal boys can predict future sperm count and identify preinvasive CIS without causing damage resulting in presence of antisperm antibodies or testicular microlithiasis in adulthood . It has been suggested that testicular biopsy at the time of orchiopexy is a risk factor for overt TGCT in postpuberty . However, no correlation between the biopsy and subsequent TGCT was found in a large study of 830 boys with UDT undergoing routine biopsy . On the other hand, alternative diagnostic methods such as reliable blood tests have not been found yet, and semen analysis cannot be performed in boys

VIII. 8.ALGORITHM



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MANAGEMENT URETERAL STONE

HAY VANEL, BOU SOPHEAP, OUK REAKSMEY

I. CASE DEFINITION

The present of Urinary stone in the ureter

II. ETIOLOGY

2.1-Low fluid intake 2.2-Hypercalciuria
2.3-Primary hyperparathyroidism 2.4-Hypocitraturia
2.5-High animal protein intake 2.6-Primary hyperoxaluria

III. DIAGNOSTIC PROCEDURE

3.1- Utrasound

Ultrasound is the primary imaging technique in children. Its advantages are absence of radiation and no need for anaesthesia. Imaging should include both the fluid-filled bladder with adjoining portion of the ureters, as well as the upper ureter. Colour Doppler US shows differences in the ureteral jet [87] and resistive index of the arciform arteries of both kidneys, which are indicative of the grade of obstruction [88]. Nevertheless, US fails to identify stones in > 40% of children and provides limited information on renal function.

3.2- KUB X ray

Kidney-ureter-bladder radiography can help to identify stones and their radiopacity and facilitate follow-up.

3.3-Non-contrast-enhanced computed tomography

Recent low-dose CT protocols have been shown to significantly reduce radiation exposure. In children, only 5% of stones escape detection by NCCT. Sedation or anaesthesia is rarely needed with modern high-speed CT equipment.

3.4-Magnetic resonance urography

Magnetic resonance urography (MRU) cannot be used to detect urinary stones. However, it might provide detailed anatomical information about the urinary collecting system, the location of an obstruction or stenosis in the ureter, and renal parenchymal morphology.

3.5-Intravenous urography

The radiation dose for IVU is comparable to that for voiding cysto-urethrography (0.33 mSV)
.However, the need for contrast medium injection is a major drawback. 3.6-Diagnostic investigation for recurrent stone former

Now day, due to our limited resources on metabolic/genetic tests, etiologic workups are encouraged (optional) and should be done with

multidisciplinary team.

Table 1. Basic evaluation of a stone former

Investigaion	Rationale for investigation
Medical history and physical Examination	Stone history (Prior stone events, family history) Dietary habits Medication chart
Diagnostic imaging	Ultrasound
Blood analysis	Creatinine Calcium (ionized calcium or total calcium + albumin) Uric acid
Urinalysis	Dipstick test : Leukocytes, erythrocytes, nitrite, Protein, urine pH, specific weight Urine culture

IV. DIFFERENTIAL DIAGNOSIS

Following are some important differentials to be considered in a patient presenting with the above-mentioned features:

- Lower urinary tract infection
- Pyelonephritis
- Renal abscess
- Renal artery aneurysm
- Appendicitis
- Diverticulitis
- Mesenteric ischemia
- Pancreatitis

V. THERAPUETIC APPROACH FOR URETERAL STONE

5.1- Conservative treatment(observation)

- Spontaneous stone passage according to stone size.
- 80% to 90% of stones up to 4 mm pass within 4 to 6 weeks.
- Based on expert opinion: Patients with uncomplicated ureteral stones ≤6 mm should be offered observation.
- Spontaneous stone expulsion decreases with increasing stone size and that there are differences between individual patients.

5.2- Pharmacological treatment, medical explusive therapy(MET)

- Based on expert opinion: Patients with uncomplicated ureteral stones >6 mm - ≤10 mm should be offered MET with α-blockers(Tansulosine 0.4mg, Doxazocine…).
- Offer α-blockers as medical expulsive therapy as one of the treatment options for (distal) ureteral stones > 5 mm.
- MET: only be used in informed patients if active stone removal is not indicated.

- Treatment should be discontinued if complications develop (infection, refractory pain, deterioration of renal function).
- In most patients, if observation with or without MET is not successful after four to six weeks and/or the patient/clinician decide to intervene sooner based on a shared decision making approach, the clinicians should offer definitive stone treatment.

5.3-Indications for active removal of ureteral stones

3-1. Indications for active removal of ureteral stones are:

- stones with a low likelihood of spontaneous passage (More than 10mm);
- persistent pain despite adequate analgesic medication;
- persistent obstruction;
- renal insufficiency (renal failure, bilateral obstruction, or single kidney).
- 3-2.Note: Clinicians should offer reimaging to patients prior to surgery if passage of stones is suspected or if stone movement will change management.

Choice	Lombar Stone (LU)	Iliaque Stone(IU)	Pelvis Stone (PU)
	(1) URS ± Flexible/SWL	(1) URS± Flexible	(1) URS
	(2) cœlioscopy /NLPC anterograde /open surgery	(2) cœlioscopy 3. Flush + SWL/NLPC	(2) open surgery 3- JJ + SWL later
		4/ Open surgery	

5.4- Treatment for Pregnant Patients with Ureteral or Renal Stones

- In pregnant patients, clinicians should coordinate pharmacological and surgical intervention with the obstetrician.
- In pregnant patients with ureteral stone(s) and well controlled symptoms, clinicians should offer observation as first-line therapy.
- In pregnant patients with ureteral stones, clinicians may offer URS to patients who fail observation. Ureteral stent and nephrostomy tube are alternative options with frequent stent or tube changes usually being necessary.

5.5- General metabolic considerations for patient workup and recurrence prevention

5.5.1-Evaluation of patient risk

All patients should undergo stone analysis using infrared spectroscopy or X-ray diffraction prior to metabolic evaluation [8]. Stone analysis should be performed in recurrent stone formers during each stone episode, even if the initial stone composition is known, because changes in stone content have been reported in recurrent stone formers. When stone analysis is not available, a specific workup of the patient should be performed.

General Factor
Early onset of urolithiasis (especially children and teenagers)
Familial stone formation
Brushite-containing stones (calcium hydrogen phosphate; $CaHPO_4 2H_2O$)
Uric acid and urate-containing stones
Infection stones
Solitary kidney (the solitary kidney itself does not particularly increase risk of stone formation, but prevention of stone recurrence is of more importance)
Diseases associated with stone formation
Hyperparathyroidism
Nephrocalcinosis
Gastrointestinal diseases (ie, jejuno-ileal bypass, intestinal resection, Crohn's disease, malabsorptive conditions, enteric hyperoxaluria after urinary diversion) and bariatric surgery
Sarcoidosis
Genetically determined stone formation
Cystinuria (type A, B, AB)
Primary hyperoxaluria (PH)
Renal tubular acidosis (RTA) type I
2,8-Dihydroxyadenine
Xanthinuria
Lesch-Nyhan syndrome
Cystic fibrosis
Drugs associated with stone formation
Anatomical abnormalities associated with stone formation
Medullary sponge kidney (tubular ectasia)
Ureteropelvic junction obstruction
Calyceal diverticulum, calyceal cyst
Ureteral stricture
Vesico-uretero-renal reflux
Horseshoe kidney
Ureterocele

5.5.2--General considerations for recurrence prevention

All stone formers, independent of their individual risk, should follow the preventive measures presented in Table 3.

Table 3. General preventive measures

Fluid intake (drinking advice)	Fluid amount : 2.5-3.0 l/d Circadian drinking (time controlled
Nutritional advice for a balanced diet	Balanced diet Rick in vegetable and fiber Normal calcium content: 1-1.2 g/d Limited NaCl content: 4-
Lifestyle advice to normalized General risk factors	BMI: 18-25 kg/m2 Stress limitation measures

Only high-risk stone formers require specific metabolic evaluation, which should be individualized based on different stone types. Specific metabolic evaluation requires collection of two consecutive 24-h urine samples.

VI. COMPLICATION OF UROLITHIASIS

Complications include acute renal failure secondary to obstruction, anuria, urinary tract infection with renal obstruction, and sepsis.

VII. CONCLUSION

After stone passage, every patient should be assigned to a group with low or high risk of stone formation. For correct classification, reliable stone analysis and basic evaluation of every patient are required. Low-risk stone formers may benefit by adopting general preventive measures regarding fluid and nutritional intake, as well as lifestyle improvements. For high- risk stone formers, a specific metabolic evaluation is required to guide individual treatment and prevent stone recurrence.

VIII. FOLLOW UP FOR RECURRENCE STONE:

- Low risk patient: follow up every 12 months evaluation (Urinalysis, renal function test, KUB ultrasound/X-ray)
- High risk patient: follow up every 6 months evaluation (Urinalysis, renal function test, KUB ultrasound/X-ray with specific tests)

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URETEROPELVIC JUNCTION OBSTRUCTION

Dr. HAY VANEL, Dr. OUK REAKSMEY, Prof. BOU SOPHEAP

I. CASE DEFINITION

Ureteropelvic junction obstruction (UPJO), or pelviureteric junction obstruction, is defined as a blockage or obstruction of urine flow from the kidney into the proximal upper ureter. This obstruction can lead to an increase in back- pressure on the kidney, hydronephrosis, and progressive damage to the kidney function. It is therefore important to understand how to diagnose and treat this condition.

II. ETIOLOGY

- scarring of ureteric valves
- ureteric hypoplasia
- Enterococcus sp.
- Pseudomonas aeruginosa
- Burkholderia pseudomallei (melioidosis)
- Candida sp.

III. DIAGNOSTIC PROCEDURE

3.1. Clinical argumen

Most hydronephroses are diagnosed antenatally using ultrasonographic scans at 18–20 wk. Prior to the advent of ultrasonographic scanning, the most common presenta- tion of UPJO was pain, especially with excessive drinking. Urinary tract infections that may have progressed to pyonephrosis were sometimes seen with an end-stage kidney, especially in the elderly. In children, infection in UPJO is rare unless there is coincident reflux. Some children may present with an abdominal mass or hematuria following a minor trauma. Finally, some hydronephroses only come to light as an incidental finding when investi- gating for a cause of abdominal pain

A special diagnostic dilemma has arisen in the last 20 yr with the finding of unilateral or bilateral hydronephrosis in the fetus in an otherwise normal pregnancy, which is now the most common presentation. Providing there is no evidence of oligohydramnios, the pregnancy is allowed to continue to term, and the baby's condition is investigated further after delivery.

3.2. Imaging study

- CBC, creatinine, BUN
- Ultrasonography of bladder and kidneys
- Radiography: supine abdominal X-Ray, IVP (intravenous pyelography)
- Diuretic renography
- URO SCAN
- Voiding/micturating cystourethrogram

IV. THERAPEUTIC APPROACH

The aims of treatment are to prevent deterioration of renal function and relieve pain (if present). The difficulty lies in determining which kidneys need surgical treatment. The natural history of UPJO is not clearly defined, and using the investigative modalities previously described, it is not possible to fully agree on a treatment algorithm.open reconstructive surgery has been considered to be the gold standard for the treatment of UPJO

Other procedures have been aimed at reducing the size of the scar (laparoscopic procedures performing the same reconstruction) or avoiding a scar altogether (endoscopic procedures). The question, then, is how much deterioration in outcomes is acceptable to achieve these goals. It is important to remember that with all reconstructions, the first operation is the easiest, and subsequent operations will be hampered by the effects of a failed first one.

Surgical options include the following procedures:

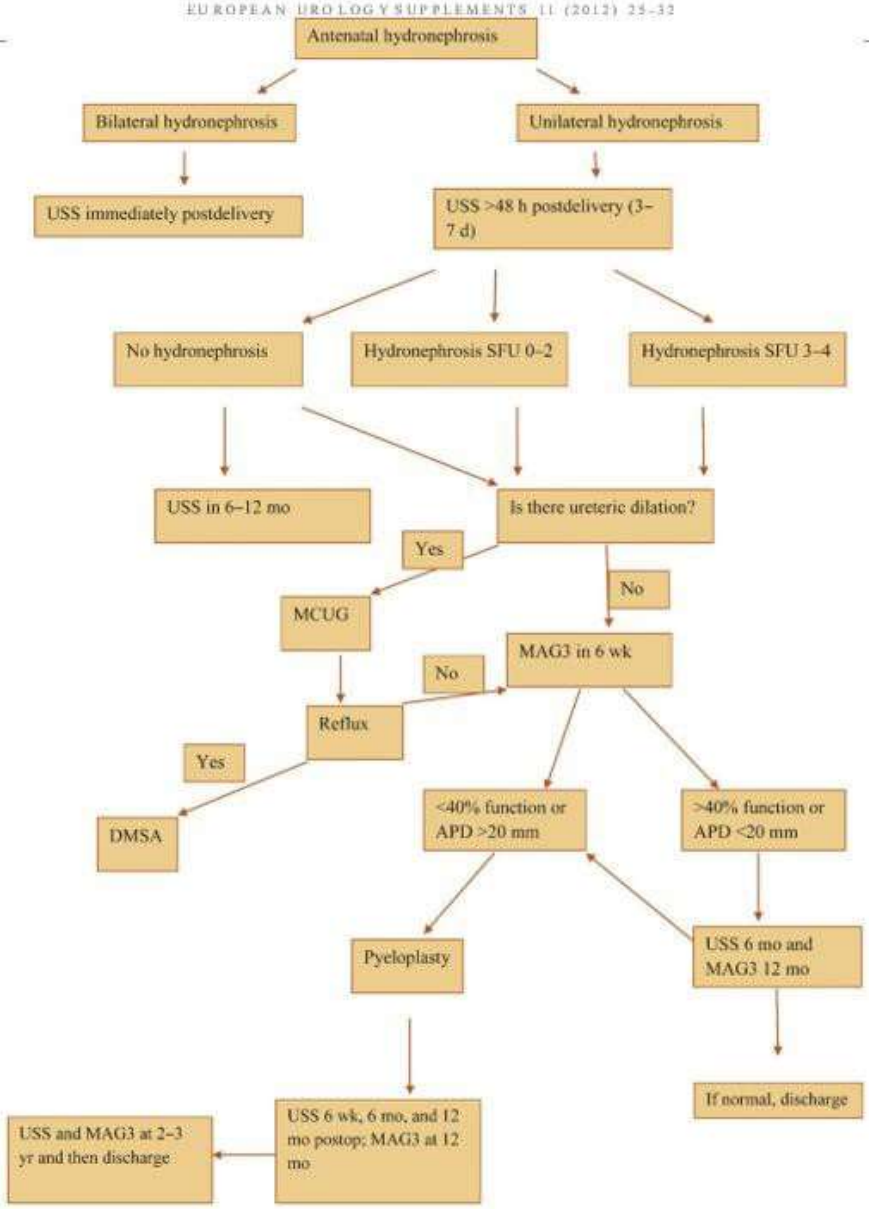
- Pyeloplasty, the gold standard treatment of a UPJO, may be a dismembered Anderson-Hynes, Culp, or Foley Y-V pyeloplasty. This treatment can be used in long strictures, in severe hydronephrosis, or in the presence of crossing vessels. Open pyeloplasty can be approached through a lumbotomy incision, an incision above the 12th rib, or an anterior abdominal wall incision [35]. The success rate is ≥95%, and the procedure has stood the test of time. Laparoscopic pyeloplasty (retro-peritoneal or intraperitoneal) is technically challenging in children and has a ≤95% success rate in the best hands. Pyeloplasty also can be performed robotically.

Endopyelotomy has an approximately 80% success rate but only in the absence of a crossing vessel. The JJ stent must stay in situ for 6 wk postoperatively.

V. COMPLICATION RENAL FAILURE

Complications of pyeloplasty include urinary tract infec- tions, pyelonephritis, urinary extravasation and leakage, recurrent UPJO, and stricture formation. Minor urinary extravasation can be treated conservatively initially for 10–14 d. If this treatment fails or if the extravasation is large, a JJ stent or nephrostomy tube is inserted. Of recurrent UPJO and/or strictures, 2–5% will need to be treated with further surgery, beit redo pyeloplasty, endopyelotomy, or ureterocalicostomy Complications of endopyelotomy include significant intraoperative bleeding if the endoscopic incision is made inadvertently into a major polar vessel (treated immediately with arteriography and embolization if there is hypotension), postoperative infection, and recurrence of obstruction.

VI. ALGORITHM



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URETHRAL STONE

HAY VANEL, BOU SOPHEAP, OUK REAKSMEY

I. CASE DEFINITION

The present of Urinary stone in the urethra

II. ETIOLOGY

2.1-Low fluid intake 2.2-Hypercalciuria
2.3-Primary hyperparathyroidism 2.4-Hypocitraturia
2.5-High animal protein intake 2.6-Primary hyperoxaluria

III. DIAGNOSTIC PROCEDURE

3.1- Utrasound

Ultrasound is the primary imaging technique in children. Its advantages are absence of radiation and no need for anaesthesia. Imaging should include both the fluid-filled bladder with adjoining portion of the ureters, as well as the upper ureter. Colour Doppler US shows differences in the ureteral jet [87] and resistive index of the arciform arteries of both kidneys, which are indicative of the grade of obstruction [88]. Nevertheless, US fails to identify stones in > 40% of children and provides limited information on renal function.

3.2- KUB X ray

Kidney-ureter-bladder radiography can help to identify stones and their radiopacity and facilitate follow-up.

3.3-Non-contrast-enhanced computed tomography

Recent low-dose CT protocols have been shown to significantly reduce radiation exposure. In children, only 5% of stones escape detection by NCCT. Sedation or anaesthesia is rarely needed with modern high-speed CT equipment.

3.4-Magnetic resonance urography

Magnetic resonance urography (MRU) cannot be used to detect urinary stones. However, it might provide detailed anatomical information about the urinary collecting system, the location of an obstruction or stenosis in the ureter, and renal parenchymal morphology.

3.5-Intravenous urography

The radiation dose for IVU is comparable to that for voiding cysto-urethrography (0.33 mSV). However, the need for contrast medium injection is a major drawback.
3.6-Diagnostic investigation for recurrent stone former

Now day, due to our limited resources on metabolic/genetic tests, etiologic

workups are encouraged (optional) and should be done with multidisciplinary team.

Table 4. Basic evaluation of a stone former

Investigaion	Rationale for investigation
Medical history and physical Examination	Stone history (Prior stone events, family history) Dietary habits Medication chart
Diagnostic imaging	Ultrasound
Blood analysis	Creatinine Calcium (ionized calcium or total calcium + albumin) Uric acid
Urinalysis	Dipstick test : Leukocytes, erythrocytes, nitrite, Protein, urine pH, specific weight Urine culture

IV. DIFFERENTIAL DIAGNOSIS

Following are some important differentials to be considered in a patient presenting with the above-mentioned features:

- Lower urinary tract infection
- Pyelonephritis
- Renal abscess
- Renal artery aneurysm
- Appendicitis
- Diverticulitis
- Mesenteric ischemia
- Pancreatitis

V. THERAPUETIC APPROACH FOR URETERAL STONE

5.1- Conservative treatment(observation)

- Spontaneous stone passage according to stone size. 5.2-Indications for active removal of urethral stones Depend on localization of the stone

Prostatic:

- Metal bougie (Dislodge stone to bladder)
- Open Surgery (Cystolithotomy) Membranous and Bulbo :
- Cystoscopy by bire basket

-Open surgery Perineal incision Penile: gentle milking (No surgery)

Fossa navicularis or Meatus: Gentle milking or Meatotomy

5.3- General metabolic considerations for patient workup and recurrence prevention 5.3.1-Evaluation of patient risk

All patients should undergo stone analysis using infrared spectroscopy or X-ray diffraction prior to metabolic evaluation [8]. Stone analysis should be performed in recurrent stone formers during each stone episode, even if the initial stone composition is known, because changes in stone content have been reported in recurrent stone formers. When stone analysis is not available, a

specific workup of the patient should be performed.

Table 5. High-risk stone formers

General Factor
Early onset of urolithiasis (especially children and teenagers)
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Uric acid and urate-containing stones
Infection stones
Solitary kidney (the solitary kidney itself does not particularly increase risk of stone formation, but prevention of stone recurrence is of more importance)
Diseases associated with stone formation
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Nephrocalcinosis
Gastrointestinal diseases (ie, jejuno-ileal bypass, intestinal resection, Crohn's disease, malabsorptive conditions, enteric hyperoxaluria after urinary diversion) and bariatric surgery
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Genetically determined stone formation
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Ureteropelvic junction obstruction
Calyceal diverticulum, calyceal cyst
Ureteral stricture
Vesico-uretero-renal reflux
Horseshoe kidney
Ureterocele

5.3.2--General considerations for recurrence prevention

All stone formers, independent of their individual risk, should follow the preventive measures presented in Table 3.

Table 6. General preventive measures

Fluid intake (drinking advice)	Fluid amount : 2.5-3.0 l/d Circadian drinking (time controlled drinking) Neutral pH beverages Diuresis: 2.0-2.5 l/d Specific weight of urine: < 1.010
Nutritional advice for a balanced diet	Balanced diet Rick in vegetable and fiber Normal calcium content: 1-1.2 g/d Limited NaCl content: 4-5 g/d Limited animal protein content: 0.8-1.0 g/kg/d
Lifestyle advice to normalized General risk factors	BMI: 18-25 kg/m2 Stress limitation measures Adequate physical activity Balancing of excessive fluid loss

Only high-risk stone formers require specific metabolic evaluation, which should be individualized based on different stone types. Specific metabolic evaluation requires collection of two consecutive 24-h urine samples.

VI. COMPLICATION OF UROLITHIASIS

Complications include acute renal failure secondary to obstruction, anuria, urinary tract infection with renal obstruction, and sepsis.

VII. CONCLUSION

After stone passage, every patient should be assigned to a group with low or high risk of stone formation. For correct classification, reliable stone analysis and basic evaluation of every patient are required. Low-risk stone formers may benefit by adopting general preventive measures regarding fluid and nutritional intake, as well as lifestyle improvements. For high- risk stone formers, a specific metabolic evaluation is required to guide individual treatment and prevent stone recurrence.

Follow up for recurrence stone:

- Low risk patient: follow up every 12 months evaluation (Urinalysis, renal function test, KUB ultrasound/X-ray)
- High risk patient: follow up every 6 months evaluation (Urinalysis, renal function test, KUB ultrasound/X-ray with specific tests)

VIII. ALGORITHM

IX. REFERENCES

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URETHRAL STRICTURE

(PROF. BOU SOPHEAP)

I. INTRODUCTION

The reduction of urethral lumen caliber, cause resistance to the antegrade flow of urine or impossible to pass urine.

It results from inflammatory, ischemic, or traumatic process that can lead scaring tissue formation then contraction.

II. EPIDEMIOLOGY

i. Urethral stricture is a relatively common condition in men, with a prevalence of 229-627 per 100,000 males (0.6% of the at-risk population), typically affecting older men. The incidence increases markedly after age 55, with data from Medicare and Medicaid Services confirming higher rates in those over 65 (9.0/100,000) compared to under 65 (5.8/100,000).

ii. The most common causes are iatrogenic trauma, often from improper catheterization (32% of cases), and idiopathic factors (30%). Infectious strictures, usually from gonococcal or nongonococcal urethritis, account for nearly 10%

III. ETIOLOGY

i. Trauma
   a. anterior urethra: penile fracture/sexual intercourse, penetrating wound…,
   b. posterior urethra: pelvic fracture (4-19%, unstable fracture, diastasis joints, openbook fx), straddle trauma…), rare in women

ii. Inflammatory or infection: UTI, STD infection

iii. Iatrogenic: (TUR, Prolonged catheterization, Cystoscopy, Hypospadias repair, post radiation of radical prostatectomy)

iv. Congenital stricture

v. Malignancy

IV. PHYSIOPATHOLOGY

vi. Anterior urethral damage seems to be caused by a blunt force impact to the perineum, which crushes the urethral tissues. The early lesions are frequently overlooked by the patient, and the urethral injury emerges years later as a stricture. The stricture is the outcome of scarring caused by ischaemia at the injury site.

vii. It is also common to see strictures caused by iatrogenic reasons, such as previous urethral instrumentation with scopes or urethral catheters.

viii. A congenital stricture is caused by inadequate integration of the anterior and posterior urethra, is short in length, and is not accompanied by an inflammatory process. This is a very rare cause.

V. DIAGNOSIS

ix. History taking (causes orientations)

x. Signs and symptoms:

a. Obstructive symptoms/ urinary retention

b. Scarring tissue feeling,

c. Tender enlarges mass along the urethral

d. Urethral fistula or peri-urethral abscess

e. Enlarged bladder

f. Urethral injury:

- blood at urethral meatus
- butterfly perineal hematoma;
- High riding prostate on PE

VI. INVESTIGATION

xi. UA, urine culture, urethral swab test, CBC, renal function tests, +/- pelvic X- ray

xii. Retrograde / antegrade cysto-urethrogram: Gold standard (localization and length of stenose)

xiii. Ultrasound of male urethra

xiv. Uroflowmetry

xv. Rigid/flexible cysto-urethroscopy (LA)

VII. DIFFERENTIAL DIAGNOSIS

xvi. Mechanical Obstruction

a. Benign or malignant prostatic obstruction

b. Bladder and urethral stone

c. Foreign body

xvii. Neurogenic Obstruction

a. Neurogenic bladder

VIII. MANAGEMENT

xviii. No medical therapy for urethral stricture

xix. Urinary catheter should be avoided if urethra injury suspected

xx. Surgical indication: severe voiding symptoms; bladder stone; increase post voiding symptoms; or repeated UTI.

xxi. Urine should be sterile before procedure

xxii. Patients should be informed about benefits and risks of procedure and post op care:

bleeding, infection, recurrence stricture, impotence and fistula formation

8.1. Emergency treatment for urethral injury:

i. resuscitation of shock and bleeding…
ii. cystostomy/SPC: unstable patients, then will investigate or repair in 3-6 months later.
iii. Immediate realignment:
    a. opened realignment (if surgery indicated for abdominal or ORIF ofpelvic bone…), or
    b. endoscopic realignment in case of stable pts for less than 48H

8.2. Urethral reconstruction

i. Principles:
    a. Resection of scarring tissue
    b. Tension free anastomosis
    c. Suitable foley catheter 16 Fr, silicone, will remove in one month.
    d. Orthopedic fracture stabilization of pelvic bone is a very important successful factor

ii. Excision of scarring tissue with primary anastomosis(urethrorrhaphy): ≤ 2 cm
    a. Free graft repair (Buccal mucosa...): success rate 84.3% ≥ 2cm
    b. Pedicle skin flaps: equivalent to graft but some complications later

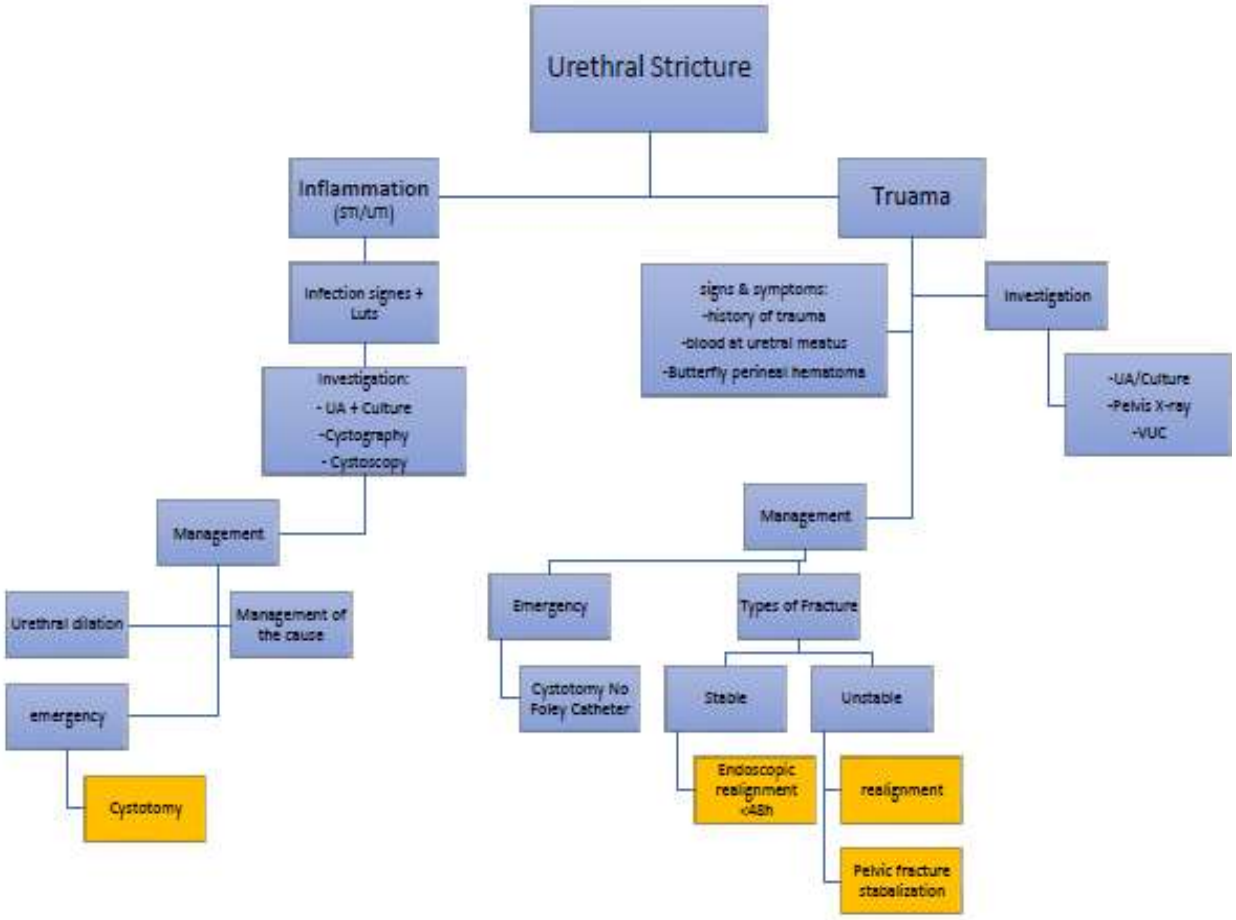
VIII.3. Alternative Treatment

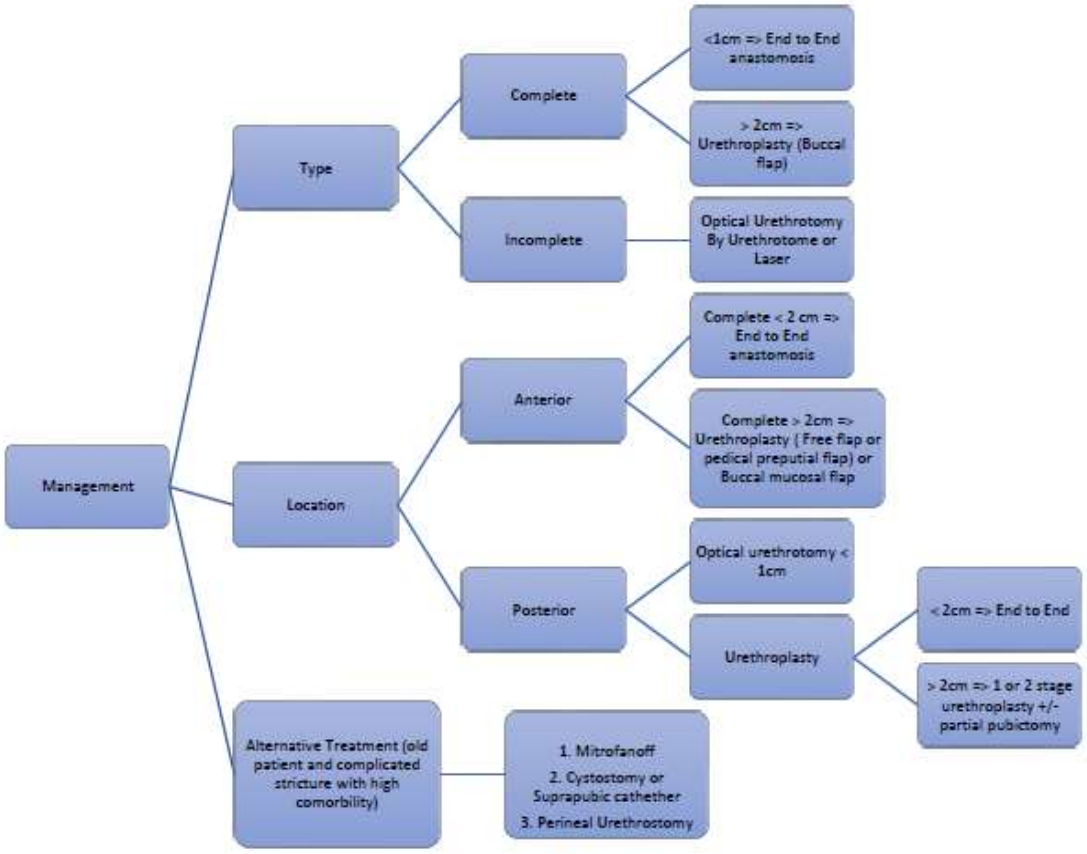
i. Urethral dilation: Inflammatory stenosis
ii. Internal Urethrotomy (cold knife/laser): No significant difference in efficacy to urethral dilation as initial treatment ≤ 1cm
iii. Mitrofanoff Procedure
iv. Old patients with severe comorbidity risk or complicated stricture:
    a. Permanent urethral stent
    b. Perineal Urethrostomy
    c. Cystostomy or Suprapubic catheter

IX. COMPLICATION

xxiii. Acute urinary retention
xxiv. Vesicoureteral reflux
xxv. Voiding dysfunction
xxvi. UTI
xxvii. Urethrocutaneous fistula
xxviii. Bladder diverticulum

X. ALGORITHMS





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URINARY RETENTION (UR)

Dr. UNG ROTHKANGCHHAKRITH, Dr. HENG SOVANDARA

I. CASE DEFINITION

Urinary retention is a condition in which you cannot empty all the urine from the bladder. Urinary retention can be acute or chronic.

II. ETIOLOGY

Many different conditions and other factors can cause urinary retention, including:

- Blockage in urinary tract (bladder stone)
- UTIs or injury
- Nerve damage (spinal cord injury)
- Prostate issue (BPH,prostatitis,or prostate cancer)
- Medication that affects the nervous system
- Severe constipation that compresses the urethra or bladder
- Cystocele (one cause in women)
- Pelvic floor (injuries following childbirth or other physical traumas)

III. DIAGNOSTIC PROCEDURE

Clinical examination and paraclinical, can help find the cause of urinary retention.

3-1 Clinical examination:

- Difficulty starting to urinate
- Weak urine steam
- Dribbling after urination
- Feeling like the bladder is still full after urination
- Pain and discomfort in the lower abdomen or back
- UTIS

Investigation

- Urines sample (Urinalysis, urine culture)
- Blood tests
- Post-void residual volume (PVR)
- Uroflowmetry

- Urodynamic
- Abdominal ultrasound
- Cystoscopy
- CT scan

IV. THERAPEUTIC APPROACH

The treatment options for urinary retention depend on the condition's underlying cause. Acute urinary retention is a medical emergency that requires immediate treatment, such as catheterization to drain the bladder (Foley or suprapubic). How to inserted or catheterization into the bladder for drainage of urine?

a. Pain relief
   - Oral analgesia
   - Local anaesthetic lubrication (e.g. Instillagel)

b. Technique: Obtain equipment needed for catheterisation
   - Sterile catheterisation pack
   - Cleaning fluid
   - Syringe and sterile water for non-prefilled catheters
   - Lubricating jelly
   - Catheter (Foley or suprapubic).
   - Catheter bag/Drainage system

4.1 Indications ***for Foley Catheter Placement*:**

- During and post-surgery
- Monitoring renal function during critical illness
- Acute urinary retention
- Chronic urinary retention (if symptomatic and/ or renal problems)
- To irrigate the bladder if hematuria is a concern
- For investigations, such as Urodynamics
- To instill medication into a bladder
- Where it is assessed as 'in the patient's best interest' to use a
- catheter, such as end of life care
- Neurological bladder dysfunctions
- Damaged skin (open sacral or perianal wound in an incontinent patient) Incontinence alone is not an indication for catheterization.

Catheters come in sizes that measure the outside circumference in millimeters (mm). which is called the French (Fr) size.

	Catheter size	10 fr	12 fr	14 fr	16 fr	18 fr	20 fr	22 fr	24 fr
	Plastic ring colo	Black	White	Green	Orange	Red	Yellow	Purple	Blue

4.1.1 Contraindications to **Foley Catheter** Placement:

- Unexplained bleeding
- History of infection
- Risk of urethral damage
- False passages
- Risk of damage to internal and external sphincters
- Urethral surgery
- Gender reassignment surgery

4.2 Indications for ***Suprapubic catheters/SPT:***

- Urinary retention
- Inability to pass a urethral catheter due to an obstruction (such as a stricture, enlarged prostate or tumor)
- Trauma to the pelvis or urinary tract
- The patient's inability to tolerate a urethral catheter
- Following pelvic or urinary tract surgery
- To minimize the risk of urethral trauma
- A needs for long-term catheterizations
- Sexually active patients

The supra-pubic catheter is usually inserted in theatre and the first change is approximately **6 weeks** post insertion.

4.2.1 Contraindications to ***Suprapubic catheters/SPT:***

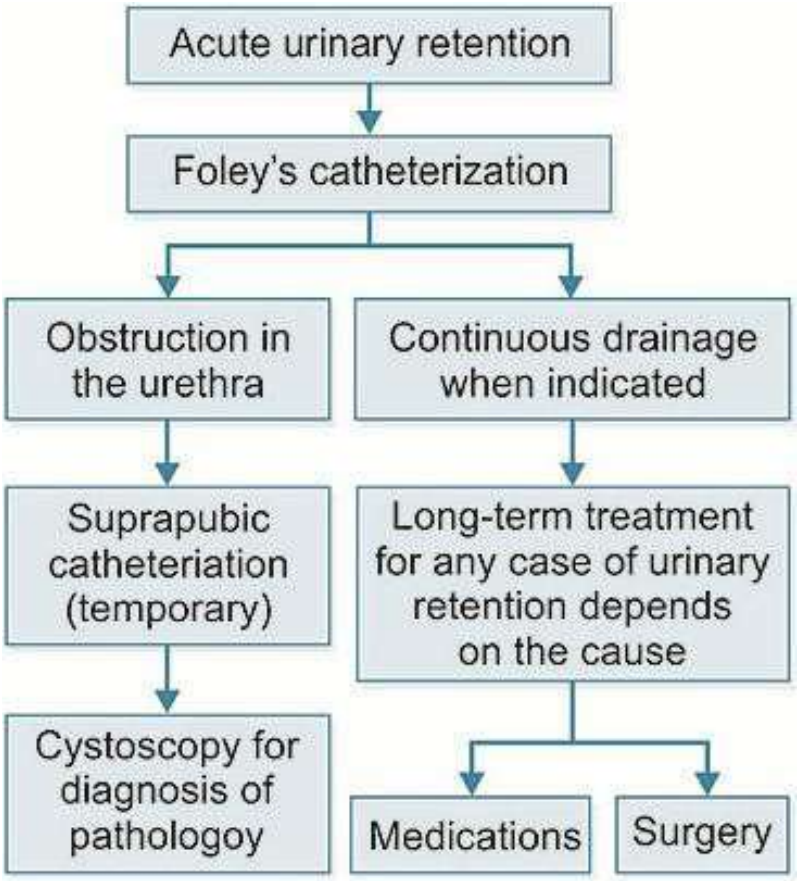
- Unexplained bleeding
- Previous lower abdominal surgery
- History of bladder tumor
- Blood clotting disorders and anti-coagulation therapy
- Ascites
- Suspicion of ovarian cyst
- Very obese patients
- Absence of an easily palpable or ultrasound localized distended urinary

bladder

V. CATHETER CARE:

- Urinary drainage bags should be positioned below the level of the bladder, and should not be in contact with the floor.
- Maintain a closed system to prevent infection.
- Drainage bags must be changed at least every five to ten days.
- They should also be changed if they become discolored, smell offensive or become damaged.
- Catheters and drainage bags must always be situated in such a way that will prevent the backflow of urine into the bladder.
- Do not allow the drainage bag to fill beyond two thirds full. The urinary drainage bag should be emptied frequently enough to maintain urine flow and prevent reflux.
- A clean pair of non-sterile gloves and a single-use apron should be worn and hand hygiene carried out on their removal, in line with aseptic technique.
- Gently washing the urethral meatus with unperfumed soap and water during the daily bathing or showering routine is best practice. Overzealous meatal cleansing may increase the risk of infection.
- Do not add antiseptic or antimicrobial solutions to urinary drainage bag.

VI. ALGORITHM



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URINARY STONE DISEASE

HAY VANEL, BOU SOPHEAP, OUK REAKSMEY

I. CASE DEFINITION

The present of Urinary stone in the urinary system

II. ETIOLOGY

2.1-Low fluid intake

2.2-Hypercalciuria

2.3-Primary hyperparathyroidism

2.4-Hypocitraturia

2.5-High animal protein intake

2.6-Primary hyperoxaluria

III. DIAGNOSTIC PROCEDURE

3.1- Utrasound

Ultrasound is the primary imaging technique in children. Its advantages are absence of radiation and no need for anaesthesia. Imaging should include both the fluid-filled bladder with adjoining portion of the ureters, as well as the upper ureter. Colour Doppler US shows differences in the ureteral jet [87] and resistive index of the arciform arteries of both kidneys, which are indicative of the grade of obstruction [88]. Nevertheless, US fails to identify stones in > 40% of children and provides limited information on renal function.

3.2- KUB X ray

Kidney-ureter-bladder radiography can help to identify stones and their radiopacity and facilitate follow-up.

3.3-Non-contrast-enhanced computed tomography

Recent low-dose CT protocols have been shown to significantly reduce radiation exposure. In children, only 5% of stones escape detection by NCCT. Sedation or anaesthesia is rarely needed with modern high-speed CT equipment.

3.4-Magnetic resonance urography

Magnetic resonance urography (MRU) cannot be used to detect urinary stones. However, it might provide detailed anatomical information about the urinary collecting

system, the location of an obstruction or stenosis in the ureter, and renal parenchymal morphology.

3.5-Intravenous urography

The radiation dose for IVU is comparable to that for voiding cysto-urethrography (0.33 mSV) However, the need for contrast medium injection is a major drawback.

3.6-Diagnostic investigation for recurrent stone former

Now day, due to our limited resources on metabolic/genetic tests, etiologic workups are encouraged (optional) and should be done with multidisciplinary team.

Table 7. Basic evaluation of a stone former

Investigaion	Rationale for investigation
Medical history and physical Examination	Stone history (Prior stone events, family history) Dietary habits Medication chart
Diagnostic imaging	Ultrasound
Blood analysis	Creatinine Calcium (ionized calcium or total calcium + albumin) Uric acid
Urinalysis	Dipstick test : Leukocytes, erythrocytes, nitrite, Protein, urine pH, specific weight Urine culture

IV. DIFFERENTIAL DIAGNOSIS

Following are some important differentials to be considered in a patient presenting with the above-mentioned features:

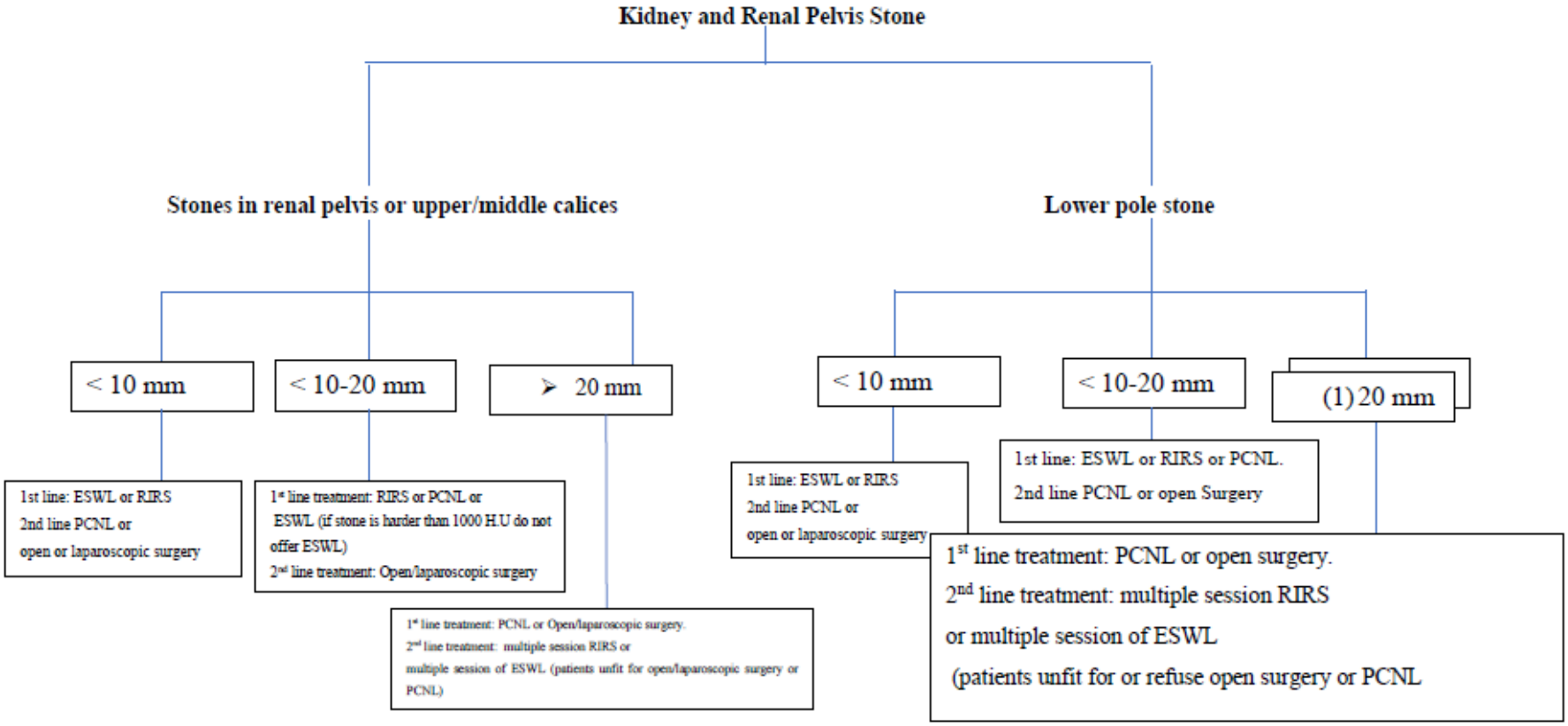
- Lower urinary tract infection
- Pyelonephritis
- Renal abscess
- Renal artery aneurysm
- Appendicitis
- Diverticulitis
- Mesenteric ischemia
- Pancreatitis

V. THERAPUETIC APPROACH

5.1-Kidney stone

Indications for the active removal of renal stones are:

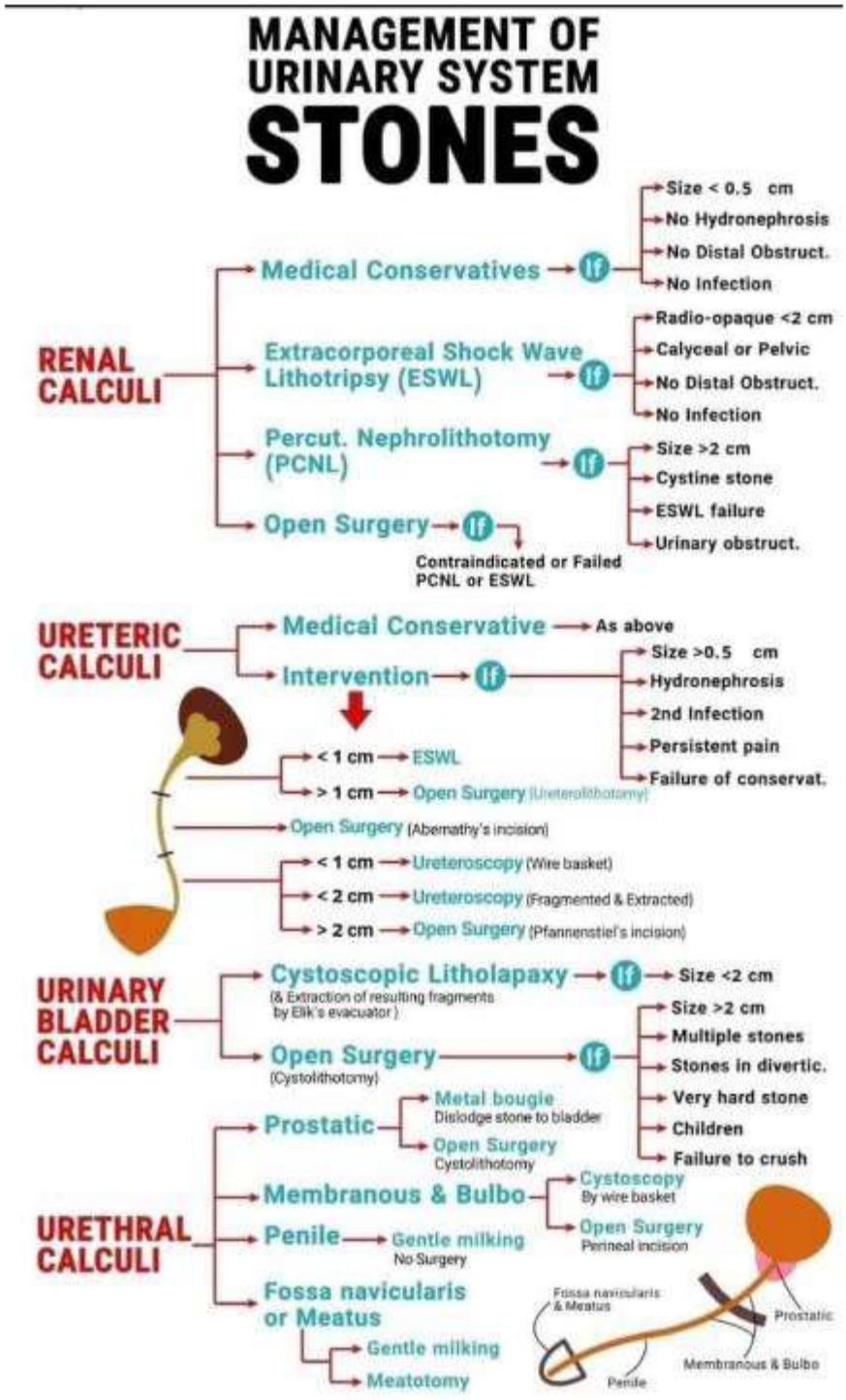
- Symptomatic stones (e.g., pain or haematuria).
- Stones > 15 mm; or stones < 15 mm if observation is not the option of choice.
- Stone that give any complication as follow: Obstruction, UTI,
- Stones in high-risk patients for stone formation,
- Stone that increase in volume.
- Patient preference
- Comorbidity;
- Social situation of the patient (e.g., profession or travelling)


Kidney and Renal Pelvis Stone
Stones in renal pelvis or upper/middle calices
Lower pole stone
< 10 mm
< 10-20 mm
➢ 20 mm
< 10 mm
< 10-20 mm
(1) 20 mm
1st line: ESWL or RIRS
2nd line PCNL or
open or laparoscopic surgery
1st line: ESWL or RIRS
2nd line PCNL or
open or laparoscopic surgery
1st line: ESWL or RIRS or PCNL.
2nd line PCNL or open Surgery
1st line treatment: RIRS or PCNL or
ESWL (if stone is harder than 1000 H.U do not offer ESWL)
2nd line treatment: Open/laparoscopic surgery
1st line treatment: PCNL or Open/laparoscopic surgery.
2nd line treatment: multiple session RIRS or
multiple session of ESWL (patients unfit for open/laparoscopic surgery or PCNL)
1st line treatment: PCNL or open surgery.
2nd line treatment: multiple session RIRS
or multiple session of ESWL
(patients unfit for or refuse open surgery or PCNL

VI. COMPLICATION OF UROLITHIASIS

Complications include acute renal failure secondary to obstruction, anuria, urinary tract infection with renal obstruction, and sepsis

VII. ALGORITHMS



VIII. CONCLUSION

After stone passage, every patient should be assigned to a group with low or high risk of stone formation. For correct classification, reliable stone analysis and basic evaluation of every patient are required. Low-risk stone formers may benefit by adopting general preventive measures regarding fluid and nutritional intake, as well as lifestyle improvements. For high- risk stone formers, a specific metabolic evaluation is required to guide individual treatment and prevent stone recurrence. Follow up for recurrence stone:

- Low risk patient: follow up every 12 months evaluation (Urinalysis, renal function test, KUB ultrasound/X-ray)
- High risk patient: follow up every 6 months evaluation (Urinalysis, renal function test, KUB ultrasound/X-ray with specific tests)

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URINE COLLECTIONS

DR. OEUR SOPAGNA, PROF.BOU SOPHEAP

I. DESCRIPTION

Urine collection refers to the process of collecting urine samples for medical testing or analysis. There are various methods for urine collection, depending on the specific requirements of the test and the patient's condition. Here are some common methods:

- Midstream clean-catch: This method involves collecting urine midstream to minimize contamination from the genital area. The patient is instructed to clean the genital area, then urinate a small amount into the toilet before collecting a sample midstream in a sterile container.

- 24-hour urine collection: In this method, the patient collects all urine produced over a 24-hour period. A container with preservative may be provided to store the urine during this time. This method is often used to measure substances that are excreted in small amounts over time or to assess kidney function.

- Random urine collection: A urine sample is collected at any time without specific timing or preparation. This method is commonly used for routine urinalysis or screening tests.

- Catheterized urine collection: In some cases, particularly if a patient is unable to urinate voluntarily or if a sterile sample is required, a catheter may be inserted into the bladder to collect urine.

- Pediatric urine collection: Specialized collection bags or pads may be used for infants and young children who are not yet toilet trained.

- Timed urine collection: Similar to 24-hour urine collection, this method involves collecting urine over a specific period shorter than 24 hours, such as 2 or 4 hours. It may be used for specific tests or to monitor changes in urine composition over a shorter time frame.

II. URINE COLLECTIONS METHODS:

A. Midstream Clean-catch

a) Indication: is a routine check-up
   - Urinary tract infection (UTI)
   - Other urinary tract-related issues, or as part of preoperative testing

b) Procedure

how to perform a midstream clean-catch urine collection:

1. **Wash Hands**: Wash your hands thoroughly with soap and water to reduce the risk of contaminating the sample.
2. **Prepare Materials**: Gather a sterile container for collecting the urine sample, as well as any wipes or swabs provided for cleaning the genital area.
3. **Clean Genital Area**: Using the provided wipes or swabs, clean the genital area from front to back to remove any bacteria or contaminants. For females, it's especially important to wipe from front to back to prevent contamination from the anus.
4. **Start Urinating**: Begin urinating into the toilet as you normally would.
5. **Stop Midstream**: After a few seconds of urinating, stop midstream by pausing briefly. This helps flush out any contaminants near the urethral opening.
6. **Collect Sample**: While still holding the container, carefully position it under the urine stream to collect the midstream urine. Be sure not to touch the inside of the container or the rim to maintain sterility.
7. **Finish Urinating**: Once you've collected enough urine in the container, finish urinating into the toilet.
8. **Cap the Container**: Securely cap the container to prevent any leakage or contamination.
9. **Label the Sample**: Label the container with your name, date, and any other required information as specified by your healthcare provider or the laboratory.
10. **Store and Transport**: Store the urine sample as instructed by your healthcare provider. If you're transporting it to a laboratory, make sure it's kept at the appropriate temperature and delivered in a timely manner to maintain sample integrity.

B. 24-hour urine collection

Collecting a 24-hour urine sample can provide valuable information about various aspects of kidney function, electrolyte balance, and other metabolic processes. Here's a basic guide on how to perform a 24-hour urine collection:

1. Preparation:
   - Inform your healthcare provider about any medications or supplements you are taking, as some may need to be temporarily stopped before the collection.
   - Obtain a large, clean container from your healthcare provider or pharmacy for collecting the urine.
2. Start of Collection:
   - Discard your first morning urine into the toilet. This time is typically not included in the 24-hour collection.
   - Note the time and begin collecting all urine passed for the next 24 hours. Make sure to empty your bladder completely each time you urinate and collect all subsequent urine in the provided container.
3. Storage:
   - Store the collection container in a cool place like the refrigerator during the 24- hour period. This helps prevent bacterial growth and maintain sample integrity.
4. Hygiene:
   - Keep the collection container tightly closed when not in use to minimize contamination.
   - Wash your hands thoroughly before and after collecting urine to prevent contamination.

5. Continuation:
    - Continue collecting urine at the same time the next day until exactly 24 hours have passed since the start time of the collection.
6. Final Collection:
    - At the end of the 24-hour period, urinate one last time into the container to ensure that the final sample is included.
7. Completion:
    - Record the end time of the collection on the container or in a log provided by your healthcare provider.
    - Return the container to your healthcare provider or laboratory as instructed.

      Make sure to follow any specific handling and transportation instructions they provide.

C. Random urine collection:

To perform a random urine collection:

1. **Prepare the Collection Container**: Ensure you have a clean, sterile container for collecting the urine. You can obtain these containers from a pharmacy or your healthcare provider.
2. **Wash Hands**: Before starting the collection process, wash your hands thoroughly with soap and water to prevent contamination.
3. **Collect the Urine**: When you're ready to collect the urine, simply urinate into the collection container. Make sure to catch a sufficient amount of urine for the intended testing or analysis.
4. **Label the Container**: After collecting the urine, label the container with your name, the date, and any other relevant information requested by the testing facility or healthcare provider.
5. **Transport or Store Properly**: If the urine sample needs to be transported to a lab for analysis, follow any specific instructions provided. Some samples may need to be refrigerated or kept at a certain temperature during transport.
6. **Submit the Sample**: If the urine sample is for medical testing or analysis, submit it to the appropriate healthcare provider, laboratory, or testing facility as instructed.

D. Catheterized urine collection:

There are several reasons why catheterized urine collection might be necessary:

1. **Sterile Sample**: Catheterization ensures that the urine sample is collected in a sterile manner, minimizing the risk of contamination from external sources.
2. **Inability to Void**: In some cases, individuals may have difficulty urinating voluntarily, such as patients with urinary retention or neurological disorders. Catheterization allows for the collection of urine when natural voiding is not possible.
3. **Accurate Measurement**: Catheterization can provide an accurate measurement of urine output in critically ill patients, those undergoing surgery, or individuals in intensive care settings.
4. **Diagnostic Testing**: Certain diagnostic tests require uncontaminated urine samples, such as urine culture for detecting urinary tract infections (UTIs). Catheterization ensures that the sample is free from external contaminants.
5. **Monitoring**: Catheterized urine collection may be necessary for continuous monitoring of urine output in patients with certain medical conditions, such as kidney disease or fluid imbalances.

E. Pediatric urine collection:

Pediatric urine collection can be a sensitive procedure, but it's crucial for diagnostic purposes. Here's a general guide:

1. Explain the Procedure: If the child is old enough to understand, explain the procedure in simple terms. Assure them it won't be painful but may feel a bit uncomfortable.
2. Prepare the Environment: Choose a clean and private area. Make sure you have all necessary supplies ready, including a sterile urine collection container.
3. Clean the Area: Clean the genital area with warm water and soap. For girls, it's important to wipe from front to back to avoid contamination. For uncircumcised boys, retract the foreskin gently to clean.
4. Collecting the Sample: There are different methods:
    - Midstream Clean Catch: Have the child urinate a little into the toilet,

then catch the rest in the sterile container midstream.

- Bag Collection: For infants, a urine collection bag with adhesive edges can be placed over the genital area to collect urine. However, this method may lead to contamination.
- Suprapubic Aspiration: In rare cases where a sterile sample is absolutely necessary, a healthcare provider may use a needle to collect urine directly from the bladder through the abdomen.

5. Handling the Sample: Label the container with the child's name, date, and time of collection. Transport it promptly to the lab or healthcare provider as per instructions to ensure accurate results.
6. Comfort and Support: Throughout the process, offer reassurance and support to the child. Encourage them to relax and cooperate.

F. Timed urine collection:

Here's a basic overview of how timed urine collection typically works:

1. **Preparation** : The patient is given instructions on how to prepare for the collection process. This might include avoiding certain foods, drinks, or medications that could affect the results.
2. **Start Time**: The patient begins the collection process by emptying their bladder and noting the exact time that the collection period starts. This usually occurs in the morning upon waking.
3. **Collection Container**: A clean, sterile container is provided for the patient to collect all urine voided during the designated time period. The container is typically kept on ice or refrigerated to prevent the breakdown of certain substances in the urine.
4. **Record Keeping**: The patient keeps track of the timing of each voiding, making sure to collect all urine during the specified time frame.
5. **End Time**: After the designated time period (e.g., 24 hours), the patient empties their bladder one final time and notes the exact end time of the collection period.
6. **Return of Sample**: The collected urine sample is then returned to the laboratory or healthcare provider for analysis.

III. REFERENCE

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   This textbook provides a comprehensive overview of urinalysis techniques, including urine collection methods and their importance in diagnosing various diseases.

2. **Clinical Laboratory Urinalysis and Body Fluids** by Susan King Strasinger and Marjorie Schaub Di Lorenzo, 7th Edition (2014): This book covers the principles and techniques of urinalysis and other body fluid analyses, including detailed information on urine collection methods.

3. **Clinical Diagnostic Tests: How to Avoid Errors in Ordering Tests and Interpreting Results** by Michael Laposata, 3rd Edition (2016): While not exclusively focused on urine collection methods, this book provides valuable insights into diagnostic testing procedures, including considerations for specimen collection and handling.

4. **Tietz Textbook of Clinical Chemistry and Molecular Diagnostics** edited by Nader Rifai, Andrea Rita Horvath, and Carl T. Wittwer, 6th Edition (2018): This comprehensive textbook covers all aspects of clinical chemistry and molecular diagnostics, including chapters on urine testing and specimen collection methods.

5. **Henry's Clinical Diagnosis and Management by Laboratory Methods** edited by focusing on laboratory diagnostic methods, this book also includes information on specimen collection techniques, including urine collection.

UROSEPSIS

DR. OUK REAKSMEY, DR. UNG SARAN, PROF BOU SOPHEAP

I. DEFINITION OF UROSEPSIS

Urosepsis is caused by the invasion, from a focus in the urinary tract, of pathogenic or commensal microorganisms, or their constituents into the body, prompting a complex response by the synthesis of endogenous mediators responsible for the clinical phenomena (Dinarello 1984; Van Amersfoort et al. 2003). Progress of sepsis to severe sepsis and septic shock correlates with an increased risk of death.

II. ETIOLOGIE OF UROSEPSIS

Urosepsis is caused by Gram-negative bacteria (e.g., *Escherichia coli,* 52%; other *Enterobacteriaceae* spp., 22%, *Pseudomonas aeruginosa*, 4%), Gram- positive bacteria (e.g., *Enterococcus* spp., 5%, *Staphylococcus aureus*, 10%), and other pathogenic bacteria in nosocomial urosepsis (1% of all cases) (multidrug resistant bacteria, e.g., *Pseudomonas aeruginosa).*

Erythrocyte sedimentation rate increased (normal range: females 1 – 25 mm/h; males 0 – 17 mm/h)
C-reactive protein (CRP) increased (normal range, 0.1 – ≤ 8.2 mg/l, depends on the method used)
Leukocyte counts (> 12×10^9/l or < 4×10^9/l) with toxic granulation, and immature neutrophils (bands) > 10 %
Thrombocytopenia (< 80×10^9/l)
Hyperbilirubinemia (normal range, < 1 mg/100 ml)
Increased creatinine level (normal range, < 1.5 mg/100 ml)
Proteinuria
Initially respiratory alkalosis, later on metabolic acidosis
Hypoxemia
Biomarkers of sepsis (cytokines, procalcitonin) and of blood coagulation (D-dimer, protein C, protein S, anti-thrombin) may be determined and provide further hints

Figure 2. Laboratory finding in urosepsis

III. CLINICAL STAGE OF UROSEPSIS

3.1- Hyperdynamic early stage

- Precapillary sphincters shut the capillary bed, the blood

- rushes via precapillary arterial-venous shunts; gas exchange
- and removal of metabolites, e.g., lactate, cease
- Hyperventilation induces respiratory alkalosis
- The patient is warm
- Cardiac output normal or increased (up to 10–20 l/min)
- Peripheral vascular resistance and arterial-venous oxygen
- gradient reduced
- Central venous pressure normal or increased
- Patient appears as seriously ill, is pale, and sweating profusely
- Pulse is frequent and soft
- Hypotension
- Nausea, emesis, diarrhea
- Agitation, confusion, disturbance of orientation

3.2- Intermediate stage

- Accumulation of lactate results in metabolic acidosis
- Increasing myocardial depression
- Due to endothelial injury and increased vascular permeability,effusion of plasma into renal, hepatic, pulmonary interstitial space, increasing organ dysfunction followed by organ failure (shock kidney, shock liver, shock lung [ARDS])
- Due to activation of the complement and coagulatory cascades and increased adherence of cellular elements (neutrophils,thrombocytes, endothelial cells), disseminated intravascular coagulation (DIC) with consumption coagulopathy leading to hemorrhages, organ hypoxia, organ failure, and mostly lethal septic shock

3.3- Hypodynamic late stage

- Patient's skin cold and cyanotic
- Reduced cardiac output
- Peripheral vascular resistance increased due to vasoconstriction; central venous pressure reduced

IV. MANAGEMENT OF UROSEPSIS

Patients should immediately be transferred to the ICU

– Volume replacement: infusion of 1–2 l of electrolyte solution over 1–2 h; goal: central venous pressure (CVP) 8–12 mmHg, mean arterial blood pressure & 65 mmHg, but e 90 mmHg Blood transfusion in case of central venous oxygenation <70% and of hematocrit <30; optimal: fresh erythrocyte concentrates; goal: hemoglobin value & 7– e 10 g/100 ml whole blood, hematocrit >30 In case of hypalbuminemia (<2 g/100ml), the additional infusion of albumin solutions has been

suggested but is still controversial

- Controlled and assisted ventilation: tidal volume, 6 ml/kg body weight; goal: arterial oxygen saturation & 93%, central venous oxygen saturation & 70%. If <70%, administration of dobutamine (initially 2.5 μg/kg/min, after 30 min each, increase by 2.5 μg/kg/min; maximum, 20 μg/kg/min)
- Administration of vasopressors: if mean arterial pressure (MAP) <65 mmHg, give dopamine, 1–3 μg/kg/min, or noradrenaline (norepinephrine), 0.1–1.0 μg/kg/min, as a continuous i.v. infusion
- Control of urine excretion; goal: >30 ml/h; if necessary, give furosemide in order to inhibit tubular re-resorption (therapeutic value not evidence- based). Tight control of blood glucose; goal: 80-110 mg/100 ml; exact stabilization with intensive insulin therapy (anti-apoptotic effect) (Evans 2001; Russell 2006; Van den Berghe et al. 2001)
- Antimicrobial therapy: if possible, targeted (pathogen identified, sensitivity determined), otherwise calculated, or initially untargeted (wide- spectrum): reserve beta- lactam antibiotics i.v., e.g., cefotaxime, 3×2–4 g/day, or ceftazidime, 3×1–2 g/ day, or ceftriaxone, 2×2 g at day 1, then 1×2 g/day, plus aminoglycoside i.v., e.g., gentamicin, 1×240–320 mg/day, by infusion. Monitor blood levels of aminoglycoside, trough concentration should be <1–2 μg/ml, and creatinine levels, three to seven times/week (Bodmann and Vogel 2001; Gilbert et al. 2006)
- After stabilization of cardiovascular function and start of antimicrobial therapy, removal of the infectious focus is mandatory. Abscesses have to be drained, and pyonephrosis has to be treated either by intraureteral JJ catheters or percutaneous nephrostomy. A Foley catheter should be inserted in any case
- Supportive measures: for patients in septic shock and/or those with proved adrenocortical insufficiency (serum cortisol level <15 μg/100 ml; corticotropin test:within 30–60 min after i.m. or i.v. injection of 250 μg of adrenocorticotropin hormone, increase of serum cortisol level <9 μg/100 ml), the i.v./i.m. administration of hydrocortisone (4×50 mg/day), or equivalent, is indicated (Cooper and Stewart 2003; Hamrahian et al. 2004; Rhen and Cidlowski 2005; Russell 2006)
- In order to inhibit imminent disseminated intravascular coagulation (reduced levels of plasma protein C) in cases of severe sepsis, recombinant human activated protein C (drotrecogin alpha-activated) with a dose of 24 μg/kg/h as a continuous i.v. infusion for 96 h is recommended (Bernard et al. 2001; Dellinger 2003;Matthay 2001; Opal et al. 2003). The drug is approved for patients with an Apache II score of & 25, but should not be used in patients with severe sepsis who are at low risk for death, such as those with single-organ failure or an Apache II score <25 (Abraham et al. 2005; Parrillo 2005; Russell 2006). The substance has antithrombotic, anti-apoptotic, antiinflammatory, and pro-fibrinolytic properties. Potential adverse effect is hemorrhagic diathesis[1]
- -Emergency in urology-Urosepsis

URINARY TRACT INFECTION (UTI)

Dr. HAY VANEL, Dr. OUK REAKSMEY, Prof. BOU SOPHEAP

I. CASE DEFINITION

- Uncomplicated lower urinary tract infection (UTI): cystitis
- Uncomplicated upper UTI: pyelonephritis +/- urosepsis
- Complicated UTI (lower or upper caused by abnormal anatomy)

  +Including recurrent UTI (occurrence of UTI >3 times/year)
- Special types: may be STD related: prostatitis, epididymitis, orchitis, urethritis

II. ETIOLOGY

2.1. In normal host:

- Escherichia coli (80 %) and other Enterobacteriaceae (Klebsiella pneumoniae, Enterobacter sp.…)
- Staphylococcus saprophyticus 5-15%
- Enterococcus sp.

2.2. In immunocompromised host: as above, plus:

- Pseudomonas aeruginosa
- Burkholderia pseudomallei (melioidosis)
- Candida sp.

III. DIAGNOSTIC PROCEDURE

3.1. Clinical argument

3.1.1. Predisposing conditions include:

- Women are always anatomically more predisposed!
- Underlying urological structural abnormality, nephrolithiasis, benign prostatic hyperplasia
- Underlying immune depressed status: HIV, diabetes, immunosuppressive medication, pregnancy, elderly…
- Recent hospitalization or urologic tract manipulation (e.g. Foley catheter)

3.1.2. Signs and symptoms:

a. Lower UTI symptoms: dysuria, urinary frequency and urgency, sometimes with suprapubic pain/pressure, rarely with hematuria (no fever)

b. Upper UTI symptoms

- Fever (sometimes with chills) with or without signs of shock
- Flank pain/kidney percussion pain
- Lower tract symptoms (may or may not be clearly present)
- Severe pain (colicky) with radiation into the groin (if renal calculus)

c. UTI in older adults: mostly asymptomatic, but can present with frequency, dysuria, hesitancy, and incontinence

- Abdominal pain
- Altered mental status (AMS)
- Shock/sepsis of unknown source

3.1.3. Technical procedure

3.2.1. Baseline lab:

a. Urine analysis (UA, dipstick):

- WBC > 2+, with or without positive Nitrite (if positive is strongly indicated)
- If dipstick is negative but UTI symptoms: do microscopic examination and urine culture (depends on presence of WBC on microscopy).

b. Microscopic examination of the urine:

- Pyuria: clean-catch mid-stream urine specimen: ≥ 10 WBC/mm3
- Asymptomatic bacteriuria: presence of a significant number of bacteria in the urine without symptoms
- Sterile pyuria: presence of significant number of WBC in urine with repeatedly negative culture (without antibiotic)
- RBC: can be present in UTI. If gross/persistent hematuria rule out stones, tumors, vasculitis, glomerulonephritis, renal tuberculosis (≥ 5 RBC/mm3)
- Epithelial cells: moderate or many epithelial cells indicate a bad sampling method
- Casts: consider other causes (e.g. glomerulonephritis)

c. Urine culture (if complicated UTI) =>See SOP of urine sampling(nurse)

- Urine culture dome only if position distick or urine microscopic≥10WBC/mm3
- Midstream clean catches (if necessary through catherization).
- ≥105 cfu/ml (colony forming unit)
- Can be false negative if exposure to natibiotics

Note: - clinical symptoms should be evaluated carefully or repeat a sample by clean catheter if suspicion of contamination. The contaminated samples often grow multi-drug resistant bacteria for which unnecessary antibiotics are being prescribed.

A high epithelial cell counts suggest bad sampling (= chance to find the true bacteria causing UTI is less) => reject sample and need to repeat sample before making decision to treat.

3.2.1. Imaging study

- CBC, creatinine, BUN
- Ultrasonography of bladder and kidneys
- Radiography: supine abdominal X-Ray, IVP (intravenous pyelography)
- Blood culture (follow the sepsis guideline)

IV. DIFFERENTIAL DIAGNOSIS

4.1 For presumed diagnosis of cystitis:

- Neurological bladder
- Bladder cancer
- Mucosa irritation by foreign body, chemical substance
- Women: vaginitis/cervicitis due to Candida, STD (Trichomonas vaginalis, gonorrhea/Chlamydia/herpes), other gynecological pathology
- Men: urethritis/balanitis due to Candida or STD (idem)

4.2 For presumed diagnosis of pyelonephritis:

- Obstructive uropathy (stone, co5m8pression)
- Glomerulonephritis

4.3 For presumed diagnosis of complicated UTI:

- Uninfected stone
- Cancer
- TB
- Prostate hyperplasia

4.4 For presumed diagnosis of STD:

- Cancer
- Stone or foreign body
- TB

V. THERAPEUTIC APPROACH

5.1 Stabilize patient abundant drinking (2-2.5 liters extra) to increase urine flow

* In “renal colic”, no abundance water intake within the first 12 hours

5.2 pirical treatment

5.2.1. Uncomplicated lower UTI: cystitis

- First choice: nitrofurantoin 100 mg PO q8h for 7 d
- Contraindication if creatinine clearance < 40 ml/min
- Second choice
  - Ciprofloxacin 500 mg PO q12h for 3 d
  - Amoxicillin-clavulanic acid 500/125 mg PO q8h for 5 d

5.2.2. Uncomplicated upper UTI (pyelonephritis +/- urosepsis):

- Perform urine and blood cultures
- Ceftriaxone 2 g IV qd +/- amikacin 20 mg/kg IV qd (see CPG on Sepsis)
- Adapt antibiotic therapy with the result of urine/blood culture and antibiogram. If result negative, stop ceftriaxone and amikacin, and switch to ciprofloxacin 500 mg PO bid
- Total duration of treatment is 10-14 d

5.2.3. Complicated UTI

- Ceftriaxone 2 g IV qd
  - If sepsis: add amikacin 20 mg/kg IV qd (see CPG sepsis).
  - If sepsis and renal failure (Cr Cl< 30ml/min): switch to meropenem (adapted dose).
  - If abscess or hydronephrosis with/without stone: use meropenem 1 g IV q8h.
    - Amikacin has reduced penetration efficacy in abscess due to low pH. Beware that amikacin can be nephro- toxic.
    - Discuss **with AB team**
    - Discuss with surgeon for necessity of drainage or decompression
    - Adapt all antibiotic therapy with the result of urine/blood culture and antibiogram. If result negative, stop IV empiric therapy, and switch to ciprofloxacin 500 mg PO bid.
    - Total treatment duration depends on clinical evolution: 2 weeks; longer for abscesses (4-6 weeks)

5.2.4. Recurrent UTI:

- Episodic treatment of acute-recurrent UTI (nitrofurantoin)
- If permanent catheter in place (Foley or suprapubic):
  - Most patients have bacterial colonization of urinary tract, often with very resistant bacteria.
  - Try to remove or replace the catheter. If impossible, consider bladder irrigation with betadine solution 1%

(flash 200ml of betadine solution once or twice daily).

- Treat with antibiotic only in case of fever and/or sepsis signs.

- AB prophylaxis is not recommended because of increased risk of resistance.

5.2.5. Special situations:

a. Urethritis: ceftriaxone 250 mg IM single dose (for gonorrhea) +/- doxycycline 100 mg PO q12h for 7 d (for Chlamydia)

For pregnancy or breast feeding: ceftriaxone 250 mg IM single dose + erythromycin 500 mg PO q6h for 7d

b. Acute Prostatitis, Epididymitis, Orchitis:

- Ciprofloxacin 750 mg PO q12h *plus* doxycycline 100 mg PO q12h for 4w
- Ceftriaxone 2 g IV qd (then switch to ciprofloxacin PO) + doxycycline 100 mg PO q12h + amikacin 20 mg/kg IV qd for sepsis cases. The duration is 4 weeks except for amikacin (stop/adapt according to culture result)

* Note: - Should think of melioidosis, mumps

- The sexual partner should be treated as well in case of STD

c. UTI in pregnancy

Asymptomatic bacteriuria/cystitis is treated with a 10-day course based on sensitivity testing.

- Nitrofurantoin 100 mg PO q8h 1st and 2nd trimester
- Amoxicillin-clavulanic acid 500/125 mg PO q8h in 3rd trimester

d. If urine culture positive with urine WBC < 20 cells/ml, suggest to repeat urine microscopy before deciding to treat according to previous antibiogram (discuss with AB team).

5.3 Directed treatment 5.4Monitoring

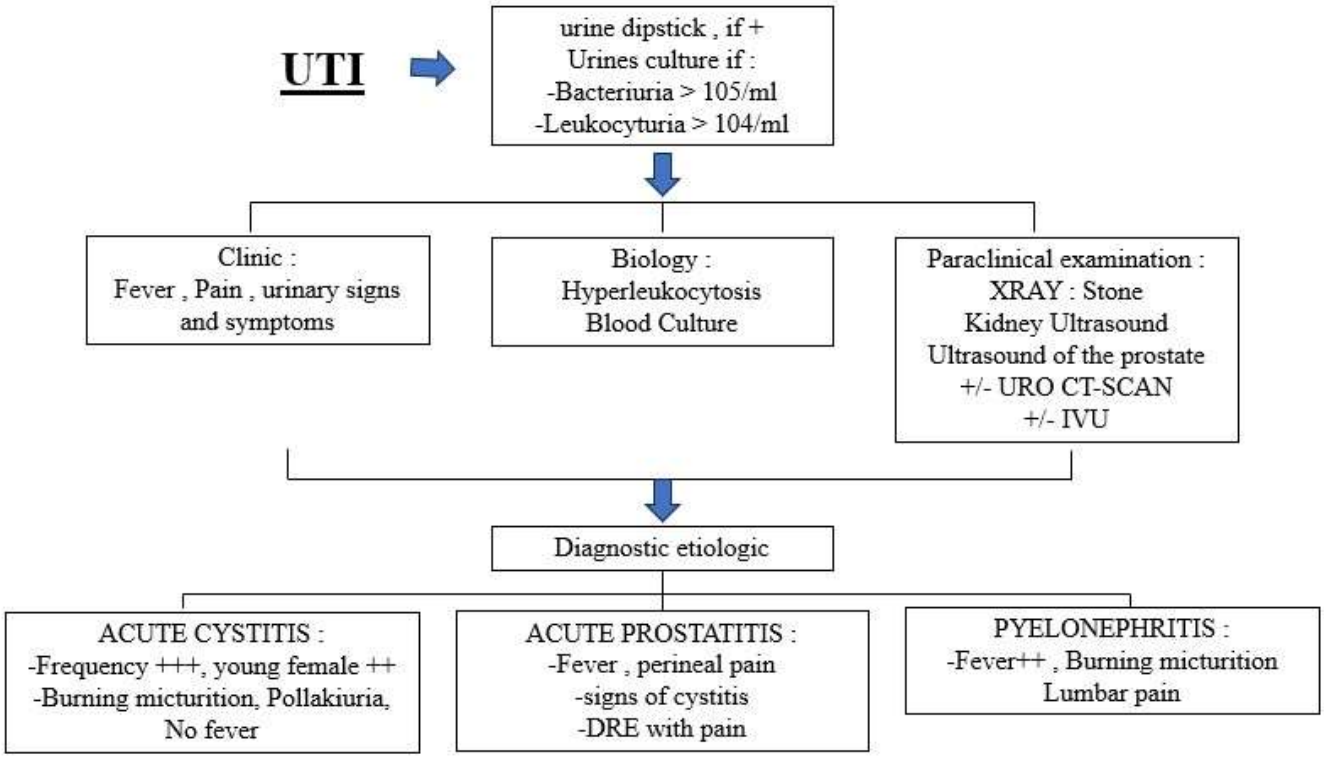
5.4.1 Clinical monitoring necessary for complicated UTI/hospitalized patients: creatinine, CBC

5.4.2 Laboratory monitoring If no improvement after 72 hours: repeat blood and urine culture +/- abdominal ultrasound

VI. COMPLICATION RENAL FAILURE

- Sepsis
- Urinary stricture
- Urine incontinence

VII. ALGORITHM



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VARICOCELE

Dr HENG SOVANDARA, DR UNG ROTHKANGCHHAKRITH

I. DEFINITION

Varicocele is the dilatation and tortuosity of the pampiniform plexus of spermatic cord found in about 15% of male adolescents with a marked left side predominance.

II. ETIOLOGY

Varicocele elevated scrotal temperatures, consequently, affect spermatogenesis and loss of testicular volume over time in some patient. Responsible factors are:

- 8-10cm longer left testicle veinous increased hydrostatic pressure in upright position.
- Entry of left testicular veinous intro renal vein at 90̊
- Nutcracker phenomenon due to passage of left testicular vein between superior mesenteric artery and aorta
- Congenital absence of valve in left vein in 40%
- Intrinsic ectasia of plexus due to cremaster atrophy
- Loaded left colon (Constipation chronic)

III. DIAGNOSTIC PROCEDURE

i. Clinical argument:

- Constant dragging pain in Testis aggravated by standing and relieved by lying down
- Swelling in scrotum
- Failure of affected testis to grow
- Asymptomatic: detected during medical examination or evaluation of infertile male
- Impaired sperm quality
- Examine in warm room, standing and lying position, with or without Valsalva Maneuver (Standing, tell patient to take a deep breath, hold it and bear down, similar to the pressure during a bowel movement)
- Painless compressible mass with feeling of “Bag of worms”
- Varicocele grading system

Grade	Size	Definition
0	Subclinical	Detected only on USS
1	Small	Palpable only with Valsalva maneuver
2	Moderate	Palpable without Valsalva
3	Large	Visible through the scrotal skin

ii. Technical procedure

a. Laboratory test:

- Semen analysis: Varicoceles are associated with low or absent sperm counts, reduced sperm motility, and abnormal morphology, either alone or in combination (Oligo-astheno- teratozoospermie syndrome)
- Serum testosterone level (Infertility rule out purpose)

b. Imaging study:

- Scrotal Color Doppler Ultrasound: for diagnostic (venous diameter >3.5mm with patient supine).
- Venography: is the **"Gold standard"** but is reserved for patients considering embolization or for varicocele recurring after treatment.

IV. DIFFERENTIAL DIAGNOSTIC

- Inguino-scrotal hernia
- Testicle tumor
- Epididymo-orchitis
- Testicle torsion
- Spermatocele
- Encysted hydrocele of cord

V. THERAPEUTIC APPROACH

iii. Observation remains the approach of choice for adolescent males who are asymptomatic with normal size of testis

iv. Treatment of varicocele should be indicated when:

a. Symptomatic (Pain, heaviness, aesthetic discomfort)

b. Adolescents: Pain, bilateral large varicoceles, varicocele in a solitary testis, persistent delayed testicular growth by more than 20% (as compared with non-affected side)

c. Impaired sperm quality (couple has known infertility)

v. Treatment options are outlined below:

a. Surgical ligation of spermatic veins:

1. Inguinal (Ivanissevich) approach (First choice): the inguinal canal is incised to access the spermatic cord and the external spermatic veins are tied off as they exit the internal ring.

2. Laparoscopic: internal spermatic veins are occluded high in the retroperitoneum.

3. Subinguinal (Marmar) approach: external spermatic veins are accessed and ligated via a small transverse incision below the external ring. With microscopic assistance, this technique is reported to have superior outcomes to other approaches.

4. High retroperitoneal (Palomo) approach: a muscle-splitting incision is made near the anterior superior iliac spine and the internal spermatic veins are ligated at that level.

b. Embolization (Future): interventional radiological technique where the femoral vein is used to access the spermatic veins for venography and embolization (with coils or other sclerosing agents), with success rates of 83%.

VI. COMPLICATION

vi. Complication of treatment:

a. 20% chance of recurrence

b. 5% Hydrocele formation- due to ligation of lymphatics

c. Testicular infarction

d. Migration of coil to pulmonary artery-usually not fatal

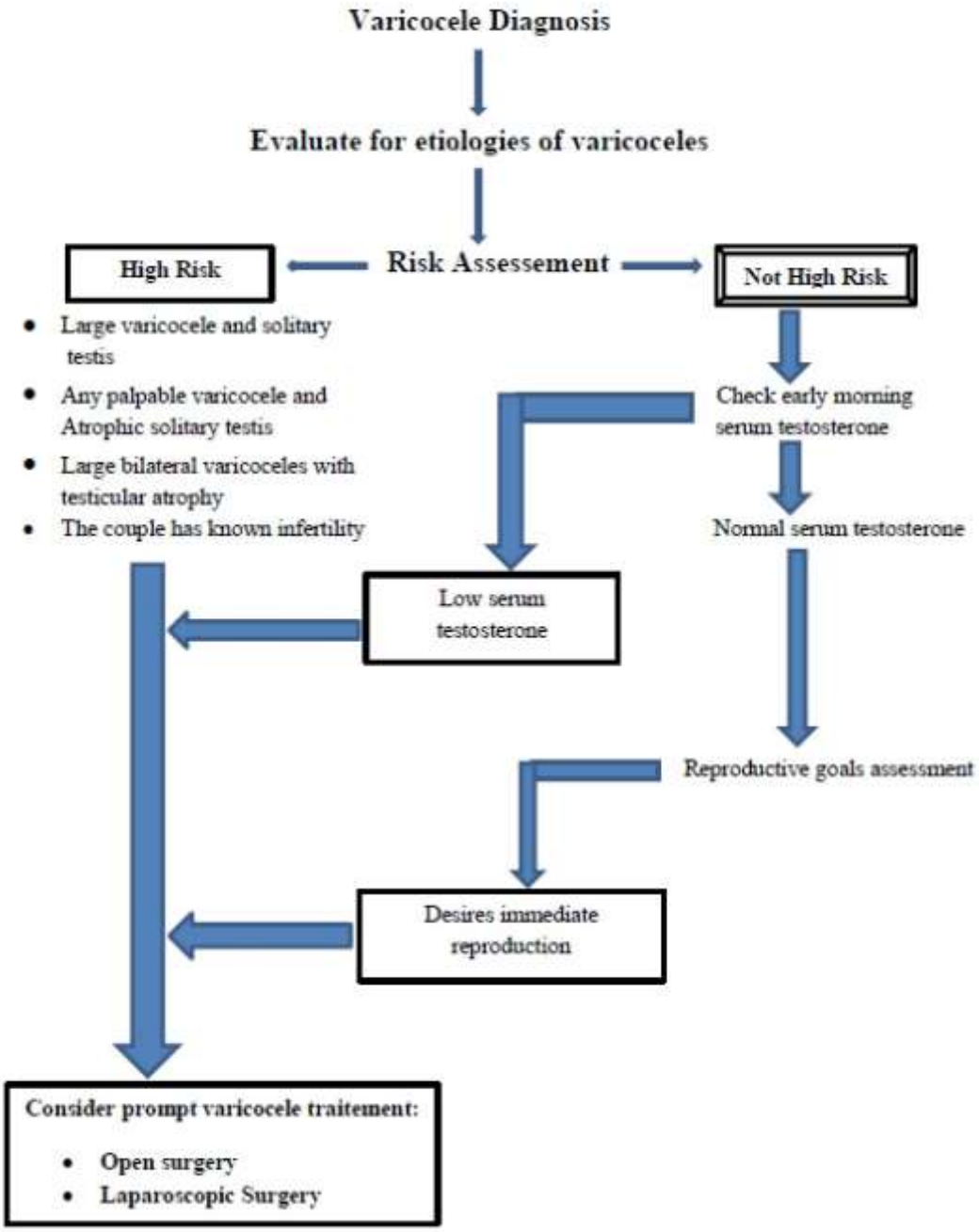
e. Infection and haemorrhage

vii. Complication of untreated varicocele

a. Male infertility

b. Testicular atrophy

VII. ALGORITHM



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VESICO-URETERAL REFLUX

Dr. OUK REAKSMEY, Dr. HAY VANEL, Dr. PEN MONYRATH,
Prof. BOU SOPHEAP

I. CASE DEFINITION

Vesicoureteral reflux (VUR), or the retrograde flow of urine from the bladder into the ureter, is an anatomic and functional disorder that can result in substantial morbidity, both from acute infection and from the sequelae of reflux nephropathy.

II. ETIOLOGY

The cause of the defect in primary reflux is unknown.

The existence of a strong genetic component is indicated by the high rate of reflux in relatives of patients with reflux, but the mechanism of transmission is not clear. Some investigators have favored a polygenic mode of inheritance, whereas others have suggested autosomal or sex-linked transmission with variable penetrance.

III. DIAGNOSTIC PROCEDURE

History

Most children with vesicoureteral reflux (VUR) present in two distinct groups, as follows:

The first group presents with hydronephrosis, often identified antenatally via ultrasonography (US); these children typically progress through evaluation and treatment in the absence of clinical illness

The second group presents with clinical urinary tract infection (UTI)

Even for experienced pediatricians, the diagnosis of UTI in children can be difficult. Children often present with nonspecific signs and symptoms. Infection in infants can manifest as failure to thrive, with or without fever. Other features include vomiting, diarrhea, anorexia, and lethargy.

Older children may report voiding symptoms or abdominal

pain. Pyelonephritis in young children is more likely to manifest with vague abdominal discomfort rather than with the classic flank pain and tenderness observed in adults. The presence of fever, while highly suggestive of pyelonephritis, is not reliable enough to lead to the diagnosis.

Physical examination

As with the history, few findings on physical examination suggest VUR or UTI. Fever, flank or abdominal tenderness, or an enlarged palpable kidney may be present. In the absence of reliable historical or physical findings, diagnosis depends on laboratory testing and imaging, as well as family history.

Work up

Diagnosis of urinary tract infection (UTI) depends on obtaining accurate urine culture findings. The criterion standard for obtaining urine specimens remains the suprapubic aspiration. Any growth in such a sample should be considered significant. In practice, however, this procedure is rarely done. Urethral catheterization provides substantially better specificity; more than 1000 colony-forming units (CFU)/mL is considered significant for these samples.

Imaging is the basis of diagnosis and management of vesicoureteral reflux (VUR). The standard imaging tests include renal and bladder ultrasonography (US) and voiding cystourethrography (VCUG), though numerous studies are available. Imaging after the first UTI is indicated in all children younger than 5 years, children of any age with febrile UTI, and boys of any age with UTI. In addition, children with antenatally identified hydronephrosis should be evaluated postnatally. US performed during the first 3 days of life may have a high rate of false-negative results because of relative dehydration during the neonatal period.

All children with a history of febrile UTI should undergo kidney and bladder US. This allows assessment of the upper tracts for obstruction, renal anomalies and scarring, and other drainage patterns. It does not, however, effectively evaluate for or rule out VUR, and US should not be considered an accurate screening test for findings that would be identified on VCUG. A study by Öztürk et al suggested that preoperative US measurements (specifically, the detrusor-to-ureteral orifice distance and the ratio of the detrusor-to-ureteral orifice distance to the distal ureteral diameter) may be reliable predictors of whether endoscopic subureteric injection therapy will be successful.

Although the 2011/2016 AAP guidelines recommended that US alone should be the initial screening test for children after UTI, the Society for Pediatric Urology continued to recommend that both US and cystography be performed.

Although the traditional approach in children with UTI has been evaluation for VUR with VCUG or radionuclide cystography (RNC), some authorities have advocated a "top-down" approach for children with UTI. In this algorithm, a child with a history of febrile UTI undergoes a dimercaptosuccinic acid (DMSA) renal scan to assess for evidence of kidney involvement, kidney scarring, or both. Negative DMSA scan findings suggest that clinically significant VUR is unlikely, rendering VCUG unnecessary. However, if DMSA scan findings are positive, VCUG is recommended. The merits of alternative approaches to children with UTI are still discussed.

IV. DIFFERENTIAL DIAGNOSIS

- Antenatal Urinary Tract Dilation (Hydronephrosis)
- Myelodysplasia and Neurogenic Bladder Dysfunction

- Pediatric Myelodysplasia
- Pediatric Ureteropelvic Junction Obstruction
- Pediatric Urinary Tract Infection
- Posterior Urethral Valves
- Urethral Anomalies and Urethral Prolapse in Children
- Voiding Dysfunction

V. THERAPEUTIC APPROACH

Controversy persists over the optimal management of vesicoureteral reflux (VUR), specifically with respect to the timing, technique, and benefits of surgical correction. Guidelines have been published by the American Urological Association (AUA).

Guidelines have also been developed by the European Association of Urology (EAU) and the European Association of Paediatric Urology (ESPU).

Febrile urinary tract infection (UTI) with signs of pyelonephritis in children with VUR requires admission and also treatment with parenteral antibiotics to prevent renal damage. This is particularly true in children who are dehydrated, unable to retain oral intake, or in a toxic state.

The need for inpatient admission should be based on the clinical assessment at the time of presentation. Many patients with febrile UTI can be managed as outpatients. Children who are severely dehydrated or in a septic state, as well as those for whom there are social concerns regarding whether home caregivers can be relied on to care for the ill child properly and completely, should be admitted. Hospitalization after open antireflux surgery typically lasts 24-72 hours. It is increasingly common for children to be discharged home the morning after surgery, and some centers are performing these procedures on an outpatient basis. Generally, children are discharged once they tolerate a regular age-appropriate diet, their pain is managed with oral pain medication, and they are active at an age-appropriate level. Endoscopic antireflux surgery is generally performed as an outpatient procedure.

VI. COMPLICATION

Obstruction after open antireflux surgery

Most cases of postoperative upper tract obstruction are mild, produce no symptoms, and spontaneously resolve. These cases are due to edema at the ureteroneocystostomy site, blood clots, or mucus causing mechanical obstruction. Cases of severe obstruction often have a delayed presentation (1-2 week or longer) and may be associated with flank or abdominal pain, nausea, and vomiting.

US reveals dilation on the affected side, though this can be difficult to assess in patients who had significant dilation preoperatively.

High-grade obstruction is usually due to ischemia of the implanted ureteral segment

with resulting fibrosis and stricture. This is a rare complication. Occasionally, patients may present with intermittent obstruction due to kinking of the reimplanted ureter with bladder filling.

Treatment for high-grade obstruction is surgical revision of the obstructed system. Percutaneous nephrostomy for temporary drainage may be required if the patient is symptomatic or in a toxic state.

Persistent vesicoureteral reflux after antireflux surgery

Open surgery

Modern series consistently report success rates greater than 95% for antireflux surgery. When VUR persists postoperatively, initial observation with continued antibiotic prophylaxis is indicated. Reoperation is generally reserved for patients with persisted febrile UTI despite prophylaxis.

A very high percentage of patients in whom surgery has failed have voiding dysfunction. Urodynamic evaluation should be considered in these patients, especially if reoperation is considered. Even so, a substantial majority of patients with reflux at the first postoperative study have complete resolution at the 1-year follow-up point.
Endoscopic surgery

Initial management is often repeat injection. Many investigators report routinely injecting as many as three separate times. Patients in whom multiple injections fail should be reevaluated and treated for causes of secondary VUR. Patients with persistent VUR and indications for surgical correction should proceed to open surgery.

New **contralateral vesicoureteral reflux after unilateral antireflux surgery**

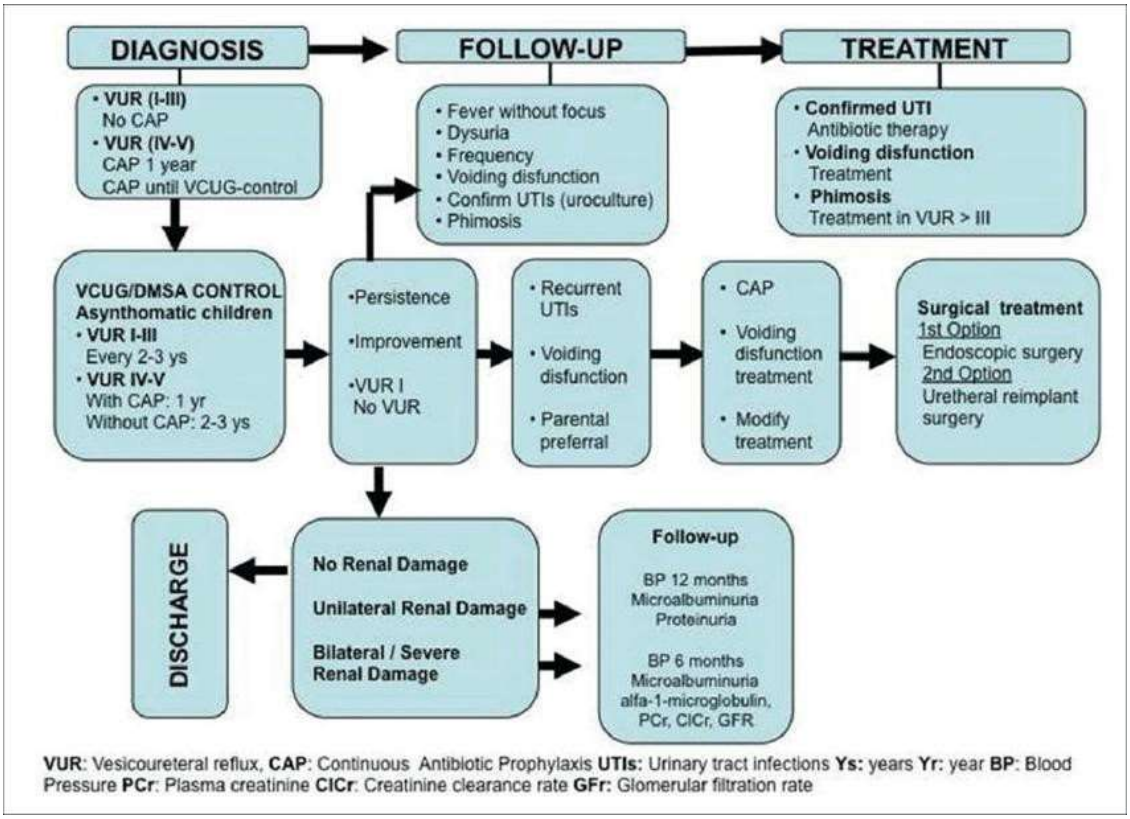
New onset of VUR in a renal unit that had no VUR on preoperative imaging occurs in 10-32% of patients after open correction and 7-14% of patients after endoscopic correction. In general, the new VUR is thought to be of low grade and may be more likely to resolve spontaneously.

VII. FOLLOW UP

Children whose VUR is being managed medically are regularly seen on an annual basis. Routine evaluation includes urinalysis and urine culture, appropriate imaging, and blood pressure measurement. Parents must understand the need for proper evaluation and urine culture if they suspect UTI. In some cases, parents are taught to perform urinalysis at home. Positive home urinalysis results should prompt formal testing at a physician's office.

After surgical correction of VUR, patients are seen in the clinic 2-6 weeks after discharge with renal US or renal scintigraphy to exclude upper-tract obstruction. Patients continue taking prophylactic antibiotics until a second return visit 3-6 months postoperatively, at which time VCUG or nuclear cystography is performed.

VIII. ALGORITHM



VUR: Vesicoureteral reflux, **CAP**: Continuous Antibiotic Prophylaxis **UTIs:** Urinary tract infections **Ys:** years **Yr:** year **BP**: Blood Pressure **PCr**: Plasma creatinine **ClCr**: Creatinine clearance rate **GFr:** Glomerular filtration rate

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VESICO-VAGINAL FISTULA

HAY VANEL, BOU SOPHEAP, OUK REAKSMEY

I. CASE DEFINITION

Fistula is an abnormal epithelialised tract between 2 epithelialised surfaces VVF common in developing countries due to birth trauma; uncommon in developed world – typically iatrogenic

II. ETIOLOGY

Congenital Acquired

- Iatrogenic
- Surgical:
- Hysterectomy*
- Anteriorcolporraphy
- Colposuspension
- Subtrigonal phenol Radiotherapy
- Non-iatrogenic
- Advancedpelvicmalignancy

  Tuberculosis
- Obstructed labour
- Foreign body erosion *

Hysterectomy accounts for ~ 90% of iatrogenic causes. Bladder injury complicates 0.5-1% of all hysterectomies. Incidence of fistula 0.1%. Fistula 3 x more common with abdominal than with vaginal hysterectomy. NB. In the setting of a difficult hysterectomy, ureteric injury is the least likely cause of urinary fistula.

III. DIAGNOSTIC PROCEDURE

Persistent dribbling incontinence 'Serous' discharge and failure to progress after gynae op Occasionally normal voiding and small loss per vaginum

History

Gynae (malignancy, RT, surgery, endometriosis, cervicalRx) Obstetric (obstructed labour, caesarian) Urology (malignancy, RT, surgery ,neurogenic bladder)

Examination

- Fluid for U+E

- Speculum vaginal examination (Cusco)
- Flexible cystoscopy
- Three pad dye test occasionally helpful for occult cases. [Methylene blue instilled into bladder. Staining of upper/mid pads suggests VVF, staining of lower pad SUI. Attempts to use IV dye to identify ureteric involvement innacurate and does not obviate requirement for RPG]
- EUA, vaginoscopy, cystoscopy and bilateral RPG prior to contemplating repair (biopsy of the fistula edge mandatory in all patients with previous or suspected malignancy) Vesico-vaginal Fistula Tom Walton January 2011 2
- CT urogram with delayed images or VCUG for complex/occult cases

IV. THERAPUETIC APPROACH

i- Conservative

Prolonged catheter drainage

Appropriate for surgically unfit patients May occasionally suffice for patients with small nonepithelialised uncomplicated (no RT, malignancy, ischaemia TB) fistula following hysterectomy (give Abx: quote 10% cure rate) Unlikely to heal if remain open after 3 weeks of catheter drainage

De-epithelialisation by curretage, silver nitrate, transvesical diathermy (Bugbee) and metal screws all tried followed by catheter drainage. Generally poor results (<10%)with established fistula Nephrostomy for urinoma, obstruction , ureteric fistulae

(ii) Surgical

Standard surgical principles important: tension-free well vascularised anastomosis with avoidance of overlapping suture lines

Remember SNAP:

S – eradicate sepsis

N – ensure adequate nutrition (?pre-op topical oestrogen)

A – define anatomy

P – determine surgical plan if unexpected problems

Timing of surgery

Iatrogenic 2-3 weeks* Obstetric injury 3-6 months

Radiation fistula 12 months + (allows tissue healing/angiogenesis following obliterative endarteritis)

* Traditional teaching recommended a delayed period for all fistulas. However early repair at 2-3 weeks believed to be equivalent to delayed repair, and reduces psychological and therefore medico-legal ‘complications’

However best chances of repair = first chance. Therefore:

< 72 hrs immediate repair

> 72 hrs 6-8 wks delayed uncomplicated 6 months baby/infected 12 months radiotherapy

Transvaginal and abdominal approaches described. In experienced hands minimally invasive TV approach a/w equivalent success rates (82-100%); depends on surgeons preference Vaginal repair

Labial stitches Ring retractor

Weighted Simms speculum
Interposition with Martius fad pad

Problems with supply of blood/proliferation Difficult to get Martius high (but dual supply - can divide below and rotate from above) Vesico-vaginal Fistula Tom Walton January 2011 3 Alternative coverage with gracilis, gracilis-based myocutaneous flap, labial or gluteal flaps

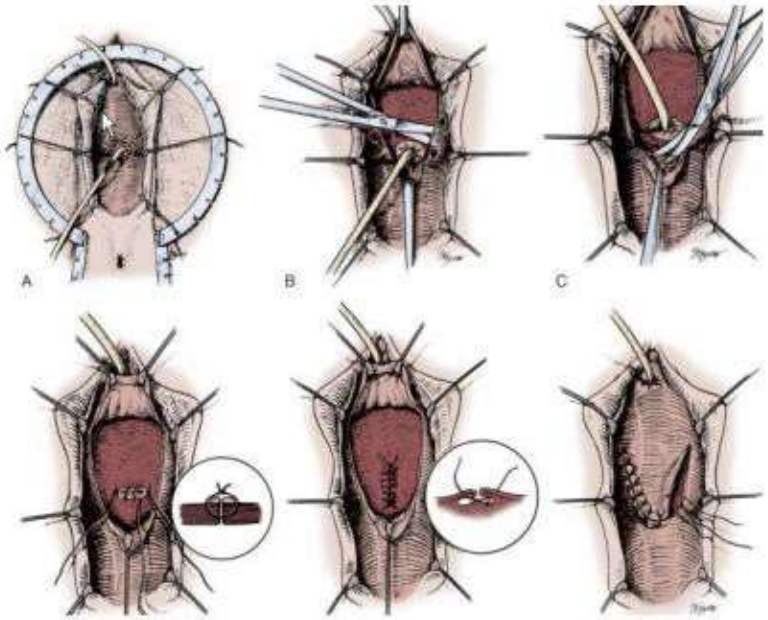
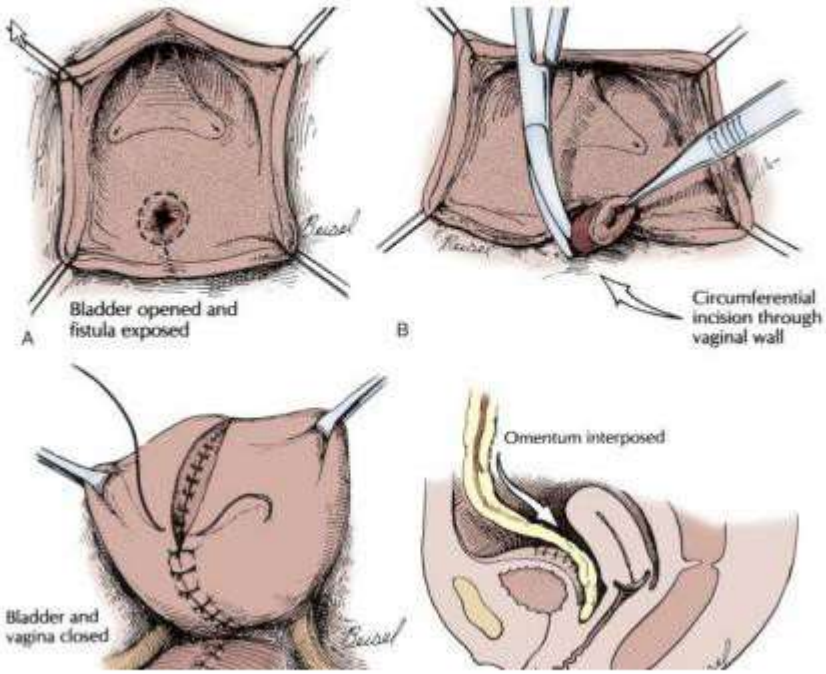


Figure 12-12 Technique of vaginal repair of a post-hysterectomy VVF. **A,** Retraction including ring retractor, vaginal speculum, and Foley catheter in the VVF track. A Foley catheter is seen in the VVF track providing traction on the vaginal cuff. **B,** Mobilization of anterior vaginal wall flap. Lateral flaps are developed as well, thereby isolating the VVF track. **C,** Mobilization of posterior vaginal wall flap. **D,** Initial layer of closure is performed without excising the edges of the fistula track. **E,** The perivesical fascia is closed with Lembert-type sutures. This line of closure is perpendicular to the initial suture line. **F,** The vaginal wall flaps are advanced to avoid overlapping suture lines. *(From Ganabathi K, Siris L, Zimmern P, Leach GE: Vesicovaginal fistulas: Reconstructive techniques. In McAninch J, ed: Traumatic and Reconstructive Urology. Philadelphia, WB Saunders, 1996:317.)*

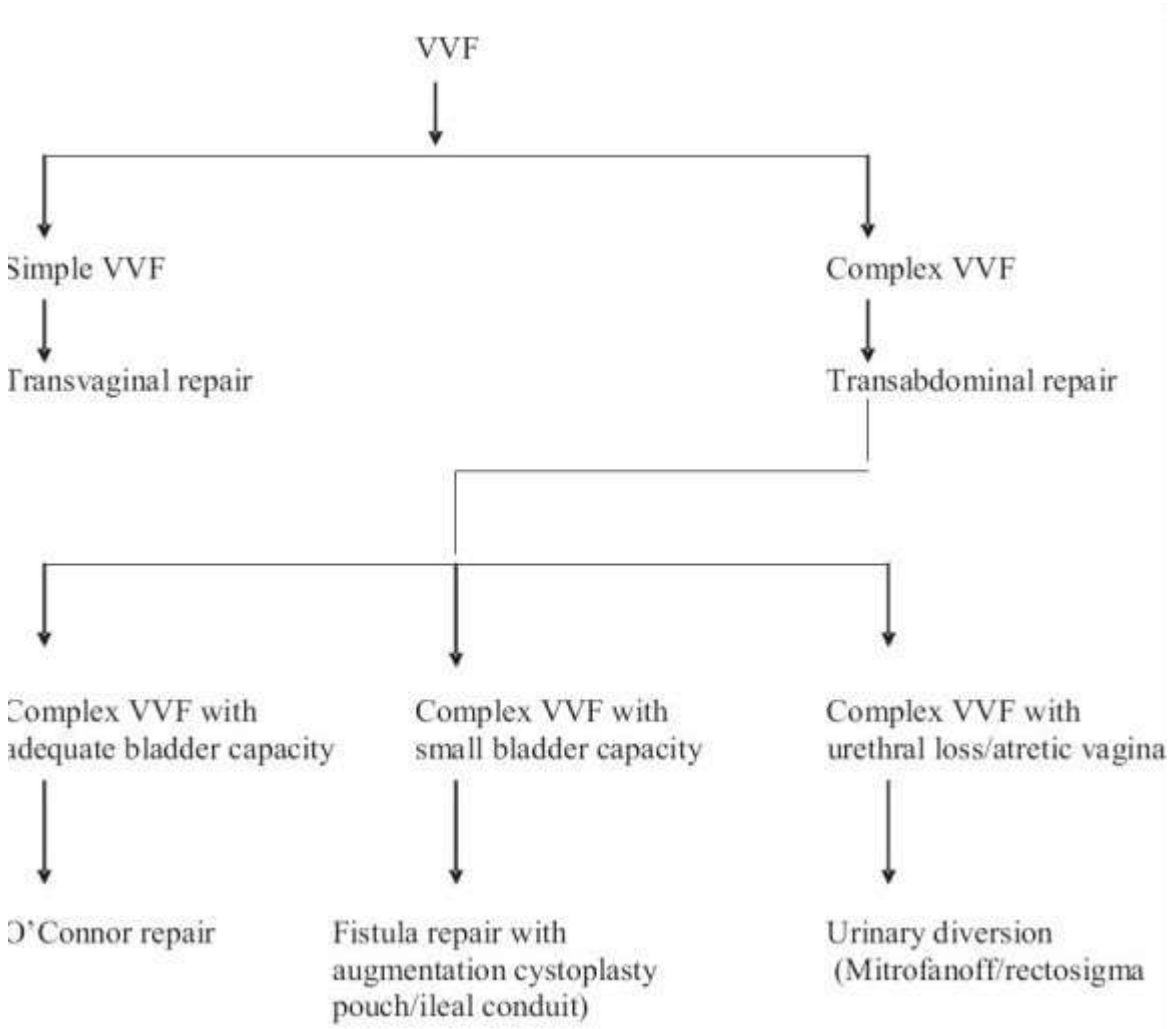
Vesico-vaginal Fistula



V. COMPLICATION

Complications include recurrence, vaginal shortening/stenosis and ureteric injury

VI. ALGORITHMS



VII. CONCLUSION

After stone passage, every patient should be assigned to a group with low or high risk of stone formation. For correct classification, reliable stone analysis and basic evaluation of every patient are required. Low-risk stone formers may benefit by adopting general preventive measures regarding fluid and nutritional intake, as well as lifestyle improvements. For high-risk stone formers, a specific metabolic evaluation is required to guide individual treatment and prevent stone recurrence.

Follow up for recurrence stone:

Low risk patient: follow up every 12 months evaluation (Urinalysis, renal function test, KUB ultrasound/X-ray)

High risk patient: follow up every 6 months evaluation (Urinalysis, renal function test, KUB ultrasound/X-ray with specific tests)

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SUPRAPUBIC CATHETERIZATION

DR. OEUR SOPAGNA, PROF.BOU SOPHEAP, DR.OUK REAKSMEY

1. DESCRIPTION

Suprapubic catheterization is a surgical procedure that involves insertion of a catheter through the abdominal wall with placement in the bladder.

2. INDICATIONS

- Method of choice if transurethral catheterization is difficult (e.g., urethral stricture, large prostate) or contraindicated (e.g., suspected urethral trauma, recent urethral surgery, acute bacterial prostatitis)
- Often preferred for chronic bladder catheterization

3. PROCEDURE

- Percutaneous urinary catheter insertion above the pubic symphysis
- May be used intermittently or left in place to continuously drain urine

4. ADVANTAGES

- Higher level of comfort and easier to change than a transurethral catheter
- Prevents urethral trauma and stricture formation
- Decreased incidence of catheter-associated bacteriuria

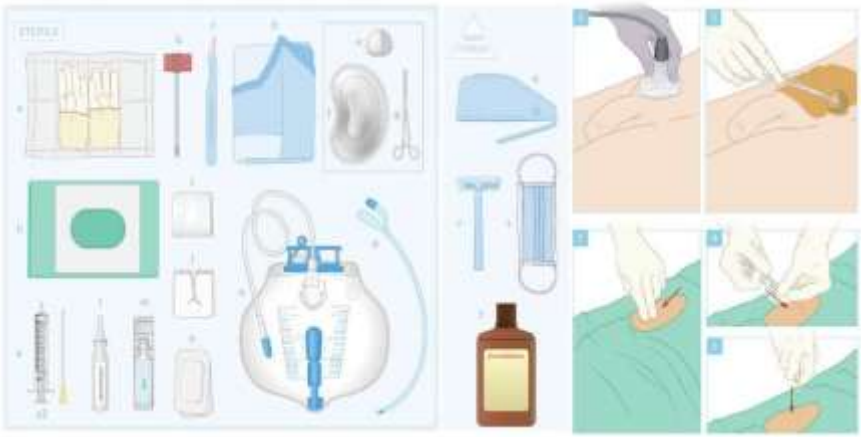
5. CONTRAINDICATIONS

- Empty or poorly localized bladder
- Prior bladder irradiation or surgery
- Bladder cancer
- Uncorrectable coagulopathy

6. COMPLICATIONS

- Infection
- Peritoneal perforation
- Injury to surrounding organs

7. PREPARATION



Sterile equipment (left):

(a) gloves

(b) trocar

(c) incision scalpel

(d) scrubs

(e–g) equipment for disinfection: surgical swab, kidney dish, hemostat

(h) incise drape

(i) compress

(j) slit compress

(k) two syringes (one with a long hypodermic needle for local anesthesia and one syringe for blocking)

(l) local anesthetic

(m) aqua

(n) patches

(o) catheter bag (sterile packed to ensure a closed, sterile system)

(p) balloon catheter Non-sterile equipment (center):

(q) surgical cap

(r) isposable razor

(s) surgical mask

(t) disinfectant

8. PROCEDURE/APPLICATION

Procedure:

Preparation (not shown): The patient is placed in a supine position (upper body slightly lower). If the patient is hirsute, the abdominal wall is shaved.

(1) sonography to check the bladder fill level and to rule out prevesical location of the intestine

(2) disinfection of the abdominal wall (under sterile conditions)

(3) after suitable covering: checking/finding of the puncture area (centered, approx. two fingerbreadths above the symphysis)

(4) superficial infiltration with local anesthetic

(5) deep infiltration with local anesthetic (strictly vertical towards the bladder)

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សមាសភាពក្រុមការងារបច្ចេកទេសមគ្គុទេសក៍ព្យាបាលគ្លីនិកសល្យសាស្ត្រ

Technical working group of Clinical Practice Guidelines for Surgery

១	ឯកឧត្តមសាស្ត្រ.	**ឆ្បឹម**	**តារា**	រដ្ឋលេខាធិការក្រសួងសុខាភិបាល	ប្រធាន
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៣	លោកសាស្ត្រចារ្យ	**ស៊ុន**	**សារិន**	នាយករងមន្ទីរពេទ្យព្រះអង្គឌួង	អនុប្រធាន
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៥	លោកវេជ្ជ.ឯកទេស	**ប៉ូ**	**រដ្ឋា**	ប្រធានការិ.បច្ចេកទេសមន្ទីរពេទ្យព្រះអង្គឌួង	សមាជិក
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៣៧	លោកវេជ្ជ.ឯកទេស	**ហេង**	**សុតា**	មន្ទីរពេទ្យកុមារជោតិ	សមាជិក
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៤១	លោកវេជ្ជ.ឯកទេស	**រស់**	**ប្រសើរ**	មន្ទីរពេទ្យកាល់ម៉ែត	សមាជិក
៤២	លោកវេជ្ជ.ឯកទេស	**ព្រាប**	**ឆ្បី**	មន្ទីរពេទ្យព្រះសីហនុ មណ្ឌលនៃក្តីសង្ឃឹម	សមាជិក
៤៣	លោកវេជ្ជ.ឯកទេស	**យិន**	**ស៊ីណាត**	ប្រធានមន្ទីរពេទ្យបង្អែកខេត្តកំពង់ចាម	សមាជិក
៤៤	លោកវេជ្ជ.ឯកទេស	**គង់**	**ភុឌ្ឍី**	ប្រធានផ្នែកវះកាត់នៃមន្ទីរពេទ្យគន្ធបុប្ផា	សមាជិក
៤៥	លោកវេជ្ជ.ឯកទេស	**គង់**	**ពិសិទ្ធ**	សមាគមគ្រូពេទ្យភ្នែកកម្ពុជា	សមាជិក
៤៦	លោកវេជ្ជ.ឯកទេស	**ឃាង**	**យ៉ាណា**	ទីប្រឹក្សាបច្ចេកទេសមន្ទីរពេទ្យមិត្តភាពខ្មែរ-សូវៀត	សមាជិក
៤៧	អង្គការដៃគូអភិវឌ្ឍ			WHO, USAID, UNICEF, JICA, KOIC,RHAC, DFAT/ACCESS, KHANA, OIC, HI, Caritas-CCAMH, ect…	សមាជិក

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