DNB
Master Solutions in Orthopedics
Volume - 1



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Paper 1 | DNB Orthopedics (Oct 2024)

Question 1.

a. **Blood supply of adult and paediatric bone**

b. **Methods of bone formation**

c. **Diagram of physio-epiphyseal complex and which layer is affected in physeal # and rickets, scurvy.**

Answer 1a.

Feature	Pediatric Bone	Adult Bone
Primary Blood Supply	Nutrient artery and epiphyseal arteries	Nutrient artery predominantly supplies the diaphysis
Epiphyseal Blood Supply	Separate blood supply to epiphyses via epiphyseal arteries; essential for growth	Epiphyseal blood supply diminishes as the growth plates close
Metaphyseal Blood Supply	Rich metaphyseal blood supply; vital for bone growth and remodeling	Metaphyseal blood supply remains, but is less prominent compared to pediatric bones
Role of Growth Plates	Growth plates receive independent blood supply, which is critical for longitudinal bone growth	No separate blood supply to growth plates, as they are fused
Periosteal Blood Supply	Thin periosteum with less contribution to blood supply compared to adults	Thicker periosteum with more significant contribution to blood supply
Healing	Faster healing due to abundant blood supply and active bone growth	Slower healing compared to pediatric bones due to less active bone growth and reduced blood supply
Bone Density	Lower bone density, higher metabolic activity due to rapid growth	Higher bone density, lower metabolic activity in mature bones
Bone Marrow	More red marrow, with hematopoietic activity in long bones	Gradual replacement of red marrow with yellow marrow in long bones
Vascular Network Complexity	More complex vascular network to support growth and development	Less complex vascular network as bones are fully formed

Answer 1b.

Methods of bone formation

1. Intramembranous Ossification

- This process involves the direct transformation of mesenchymal connective tissue into bone. It does not involve a cartilage precursor.
- Occurs primarily in flat bones such as the skull, face, mandible, and clavicle.
- Steps:
  1. Mesenchymal cells aggregate in the area where bone will form.
  2. Mesenchymal cells differentiate into

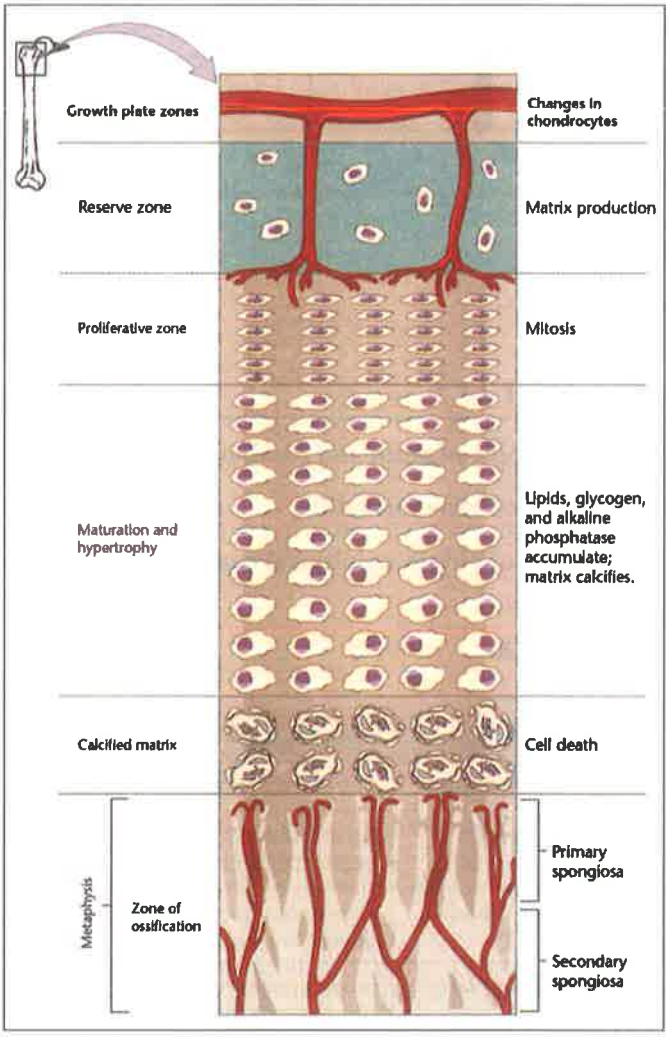
osteoblasts, which start secreting osteoid (the unmineralized bone matrix).

3. Osteoid becomes calcified, trapping some osteoblasts, which become osteocytes.
4. Bone spicules form, and these spicules join together to form trabeculae, which create a woven network of bone.
5. The trabecular network continues to thicken, and eventually, it is remodeled into compact bone.

2. Endochondral Ossification

- This process involves the replacement of a pre-existing hyaline cartilage model with bone. Most of the bones in the body are formed through this method.
- Occurs in long bones (such as the femur, tibia, and humerus), short bones, and most other bones in the body.
- Steps:
  1. Formation of cartilage model: Mesenchymal cells differentiate into chondrocytes, forming a hyaline cartilage model of the bone.
  2. Cartilage calcification: Chondrocytes within the cartilage model hypertrophy, and the surrounding cartilage matrix begins to calcify.
  3. Primary ossification center: Blood vessels invade the calcified cartilage, bringing osteoblasts that form the primary ossification center in the center of the diaphysis.
  4. Bone formation in diaphysis: Osteoblasts replace the calcified cartilage with bone tissue, gradually forming the bone collar around the diaphysis.
  5. Secondary ossification centers: After birth, secondary ossification centers form in the epiphyses, following a similar pattern of cartilage calcification and bone formation.
  6. Formation of growth plates: Between the diaphysis and epiphysis, the growth plate remains cartilaginous, allowing for continued bone growth during childhood.
  7. Epiphyseal closure: Once growth is complete, the growth plates ossify, and the bone reaches its final length.

Answer 1c.



Zone affected in scurvy and rickets- Zone of Provisional Calcification (in Maturation and Hypertrophy)

Question 2.

Frax and frax plus score, evidence based treatment of osteoporosis

Answer 2.

FRAX and FRAX Plus are tools used to assess fracture risk, particularly in people at risk of developing osteoporosis or fragility fractures. They provide an estimate of a person's 10-year probability of sustaining a major osteoporotic fracture.

1. FRAX Score (Fracture Risk Assessment Tool)

The FRAX score is a widely used, evidence-based

algorithm that calculates the 10-year risk of a major osteoporotic fracture. It was developed by the World Health Organization (WHO) and is often used by clinicians to guide decisions about treatment for osteoporosis and fracture prevention.

Key Features:

- Purpose: Estimates the 10-year risk of major osteoporotic fractures or hip fractures.
- Inputs:
  - Age
  - Sex
  - Weight and height (to calculate BMI)
  - Previous fracture history
  - Parent history of hip fracture
  - Current smoking status
  - Glucocorticoid (steroid) use
  - Rheumatoid arthritis
  - Secondary osteoporosis (conditions like Type I diabetes, untreated hyperthyroidism, etc.)
  - Alcohol intake (3 or more units/day)
  - Bone Mineral Density (BMD) at the femoral neck (optional input, but improves accuracy)

Calculation:

- The FRAX tool is available as an online calculator or in some healthcare software systems.
- It provides two key outputs:
  1. 10-year probability of a major osteoporotic fracture (spine, forearm, hip, or shoulder).
  2. 10-year probability of a hip fracture.

Use in Clinical Practice:

- The FRAX score helps guide decisions on whether to initiate pharmacological treatment for osteoporosis.
- National guidelines often recommend using FRAX in combination with other clinical judgment factors, especially in patients who do not yet have osteoporosis but may be at risk.

2. FRAX Plus (or FRAX Adjusted for Additional Risk Factors)

The FRAX Plus tool expands upon the standard FRAX by incorporating additional risk factors that can influence fracture risk. Some of these factors are not included in the original FRAX algorithm but may be important in certain patients.

Additional Risk Factors in FRAX Plus:

- Provides additional insight into bone microarchitecture and quality, which can affect fracture risk independent of Bone Mineral Density (BMD).
- A history of frequent falls is an important predictor of fractures, particularly hip fractures.
- Chronic deficiencies in calcium and vitamin D can increase fracture risk.
- Diabetes is associated with altered bone quality, which can raise fracture risk.
- Certain medications, like anti-epileptics, may increase the risk of fractures.

Use in Clinical Practice:

- FRAX Plus gives a more comprehensive risk assessment by integrating more detailed clinical information beyond what is included in the standard FRAX tool.
- It may be used for patients with complex medical histories where standard FRAX might underestimate or not fully capture the fracture risk.

Feature	FRAX	FRAX Plus
Risk Factors	Limited to age, sex, BMI, fracture history, glucocorticoid use, smoking, etc.	Includes all FRAX factors plus additional ones like falls history, TBS, diabetes, etc.
Bone Mineral Density (BMD)	Optional input to improve accuracy	Can include TBS as an additional factor for bone quality
Additional Clinical Factors	Limited set of basic risk factors	Incorporates a broader range of risk factors for more nuanced risk calculation

Purpose	Basic fracture risk assessment for treatment guidance	Advanced fracture risk assessment, especially in complex cases
Clinical Use	Commonly used for general osteoporosis risk	Used for individuals with more complex risk profiles or specific concerns

Evidence based treatment of osteoporosis:

1. Lifestyle Modifications

While not a cure for osteoporosis, lifestyle changes are crucial for preventing further bone loss and enhancing the effectiveness of medical treatments.

- Calcium and Vitamin D Supplementation:
  - Calcium: Adequate calcium intake is essential for maintaining bone health. Adults with osteoporosis should aim for 1,000–1,200 mg/day of calcium, obtained through diet or supplements if needed.
    - Evidence: Studies show calcium supplementation reduces bone loss and, in combination with other therapies, can reduce fracture risk.
  - Vitamin D: Necessary for calcium absorption, with recommended intake ranging from 800 to 1,000 IU/day.
    - Evidence: Clinical trials indicate that vitamin D supplements reduce the risk of falls and fractures, especially in the elderly.
- Physical Activity and Exercise:
  - Weight-bearing and muscle-strengthening exercises help increase bone mass, improve balance, and reduce the risk of falls.
    - Evidence: Regular exercise has been shown to improve bone mineral density (BMD) and reduce fracture risk, particularly in postmenopausal women.
- Smoking Cessation and Alcohol Moderation:
  - Smoking and excessive alcohol consumption are associated with increased bone loss and higher fracture risk.
    - Evidence: Studies show that quitting smoking and limiting alcohol intake can slow the progression of bone loss.

2. Pharmacological Treatments

Pharmacological agents are used to prevent bone loss, increase bone strength, and reduce fracture risk in patients with osteoporosis. These therapies are supported by extensive clinical trials and guidelines from organizations such as the National Osteoporosis Foundation (NOF) and the American College of Physicians (ACP).

A. Antiresorptive Medications

These medications slow down the rate at which bone is broken down (resorbed), helping to maintain or increase bone density.

- Bisphosphonates (e.g., alendronate, risedronate, ibandronate, zoledronic acid):
  - Inhibit osteoclast activity, reducing bone resorption.
  - Evidence: Multiple randomized controlled trials (RCTs) have shown that bisphosphonates increase BMD and reduce the risk of vertebral, hip, and other fractures by 30–50%.
  - Typically first-line therapy for postmenopausal women and men at high fracture risk.
- Denosumab (Prolia):
  - A monoclonal antibody that inhibits RANK ligand, preventing the formation of osteoclasts.
  - Evidence: Large RCTs have demonstrated that denosumab increases BMD and reduces vertebral, nonvertebral, and hip fractures.
  - Often used in patients intolerant to bisphosphonates or at high risk of fractures.
- Selective Estrogen Receptor Modulators (SERMs) (e.g., raloxifene):
  - Mimics estrogen's protective effect on bone by inhibiting bone resorption.
  - Evidence: Clinical trials show raloxifene reduces the risk of vertebral fractures, but not hip fractures.
  - Used in postmenopausal women, particularly those who also seek breast cancer prevention benefits.
- Hormone Replacement Therapy (HRT):
  - Estrogen therapy helps maintain bone density by reducing bone resorption.
  - Evidence: HRT reduces fracture risk, but

due to potential risks such as cardiovascular events and cancer, it is generally reserved for younger postmenopausal women or women at significant risk of fractures.

B. Anabolic Agents

These medications stimulate bone formation, enhancing bone mass and strength.

- Teriparatide (Forteo):
  - A recombinant form of parathyroid hormone (PTH) that stimulates osteoblasts, promoting new bone formation.
  - Evidence: Clinical trials have shown teriparatide significantly increases BMD and reduces the risk of both vertebral and nonvertebral fractures.
  - Recommended for patients with severe osteoporosis or those who have failed other therapies.
- Abaloparatide (Tymlos):
  - Similar to teriparatide, it stimulates bone formation.
  - Evidence: Studies show it reduces the risk of vertebral and nonvertebral fractures in high-risk populations.
  - Another anabolic option for patients at high fracture risk.
- Romosozumab (Evenity):
  - A monoclonal antibody that inhibits sclerostin, a regulator of bone formation, promoting bone growth and reducing bone resorption.
  - Evidence: Clinical trials have shown that romosozumab significantly reduces the risk of vertebral and nonvertebral fractures.
  - Used in high-risk patients, often following bisphosphonate or denosumab therapy.

3. Combined or Sequential Therapy

- Sequential Treatment: Starting with anabolic agents (e.g., teriparatide or romosozumab) followed by antiresorptive drugs (e.g., bisphosphonates or denosumab) has shown greater long-term benefits in maintaining bone density and reducing fractures.
- Combination Therapy: In some cases, combining two agents, such as teriparatide with denosumab, may be used in patients with very high fracture risk, but this approach is not routinely recommended due to limited evidence of additive benefits.

4. Fall Prevention

Preventing falls is crucial in reducing the risk of fractures, particularly in elderly patients with osteoporosis. Evidence-based strategies include:

- Home safety interventions: Removing trip hazards, improving lighting, and using assistive devices.
- Balance training: Tai Chi and similar exercises have been shown to improve balance and reduce falls.
- Medication review: Reducing or discontinuing medications that may increase the risk of falls, such as sedatives or blood pressure medications.

Question 3.

Pathogenesis, clinical features, lab findings, xray features of Vit D dependent, vit D resistant rickets, hyphophatasia.

Answer 3.

Feature	Vitamin D–Dependent Rickets (VDDR)	Vitamin D–Resistant Rickets (Hypophosphatemic Rickets)	Hypophosphatasia
Pathogenesis	Caused by mutations affecting vitamin D metabolism	Caused by impaired renal phosphate reabsorption due to mutations (e.g., PHEX gene)	Genetic disorder caused by mutations in the ALPL gene, which encodes alkaline phosphatase
	Two types: VDDR Type 1 (deficiency in 1α-hydroxylase enzyme), and VDDR Type 2 (resistance to calcitriol)	Most common form: X-linked hypophosphatemia (XLH)	Low alkaline phosphatase (ALP) activity leads to impaired bone mineralization
	VDDR Type 1: Inadequate conversion of 25(OH)D to $1,25(OH)_2D_3$ (active form)	Phosphate wasting results in hypophosphatemia despite normal or high calcitriol	Leads to defective bone, teeth, and muscle development, with varying severity

	VDDR Type 2: Mutation in Vitamin D receptor (VDR), causing tissue resistance to active vitamin D	Impaired bone mineralization due to low phosphate levels	Severity ranges from perinatal (fatal) to adult forms
Clinical Features	Presents in infancy or early childhood	Presents in early childhood, often after 1 year of age	Varies by severity: Perinatal (severe, life-threatening), infantile, childhood, adult
	Growth retardation, delayed walking, bone pain	Growth retardation, short stature	Early loss of deciduous teeth, muscle weakness, failure to thrive (severe forms)
	Delayed closure of fontanelles, frontal bossing	Bowing of legs (genu varum), dental abnormalities (delayed tooth eruption, abscesses)	Skeletal deformities (short limbs, large head), fractures, muscle weakness
	Bowing of legs (genu varum), widening of wrists and ankles	Widening of wrists, bowing of long bones	Poor bone healing, early tooth loss, craniosynostosis (in severe cases)
	Rachitic rosary, Harrison's groove	Bone pain, dental abnormalities	Fractures or pseudo-fractures (Looser zones) common in adult forms
Laboratory Findings			
Serum Calcium	Low to normal	Normal or slightly low	Normal
Serum Phosphate	Low	Low	Normal
PTH	Elevated (secondary hyperparathyroidism)	Normal or slightly elevated	Normal
25(OH)D	Normal (Type 2) or low (Type 1)	Normal	Normal
1,25 $(OH)_2D$ (Calcitriol)	Low (Type 1); High (Type 2)	Normal or high	Normal
ALP	Elevated	- Alkaline Phosphatase: Elevated	Low (a hallmark of the disease)
Urinary Calcium	Low	Normal or low	Normal
X-ray Features	Widened growth plates (metaphyseal widening)	Widened growth plates	Widened growth plates (metaphyseal dysplasia)
	Cupping and fraying of metaphyses (especially at wrists and knees)	Cupping and fraying of metaphyses	Cupping and fraying of metaphyses
	Generalized osteopenia	Generalized osteopenia	Generalized osteopenia, thin bones
	Looser's zones (pseudo-fractures)	Looser's zones (pseudo-fractures)	Looser's zones (pseudo-fractures) common, especially in adults
	Delayed bone age	Delayed bone age	Delayed bone age
	Severe bone deformities (bowing of legs, skull abnormalities)	Bowing of long bones	Shortened, deformed bones, poorly mineralized

Question 4.

Define allograft, types of allograft, processing, character, of graft. Enlist stages of graft uptake.

An allograft is a tissue graft obtained from a donor of the same species as the recipient but with a different genetic makeup.

Answer 4.

Types of Allografts:

1. Fresh Allograft: Collected and transplanted immediately without preservation. Rarely used due to infection risk and short shelf life.
2. Fresh-Frozen Allograft: Collected, sterilized, and stored at ultra-low temperatures (usually -80°C).

Preserves biomechanical properties, but immunogenic cells may still be present.

3. Freeze-Dried (Lyophilized) Allograft: Freeze-dried to remove moisture, making it shelf-stable at room temperature. Easily stored and shipped, but has reduced mechanical properties compared to fresh-frozen grafts.
4. Irradiated Allograft: Subjected to gamma irradiation to sterilize the tissue and reduce immunogenicity. May cause weakening of the graft's mechanical properties due to radiation exposure.
5. Demineralized Bone Matrix (DBM): Bone graft that has been demineralized to expose bone morphogenetic proteins (BMPs), which promote osteoinduction. Available in powder, gel, or putty forms, commonly used for bone healing.
6. Composite Grafts: A combination of different types of tissues (e.g., bone and tendon or bone and cartilage) used in joint reconstructions or for complex defects.

Processing of Allografts: It involves several steps to ensure the graft is safe, sterile, and functional while maintaining its biological properties. The main steps in processing include:

1. Donor Screening: Comprehensive screening of the donor for infections, diseases, and suitability for donation.
2. Aseptic Harvesting: Tissue is harvested under sterile conditions to minimize contamination.
3. Sterilization: Gamma irradiation or ethylene oxide gas may be used to sterilize the graft and reduce immunogenicity. Some methods may impact the mechanical properties of the graft.
4. Freeze-Drying (Lyophilization): Water is removed under low temperature and pressure, making the graft stable for long-term storage.
5. Freezing: Grafts may be stored in a fresh-frozen state at ultra-low temperatures (-80°C or lower) to preserve tissue viability.
6. Demineralization: The mineral content of bone is removed, exposing osteoinductive proteins (like BMPs), which help in bone regeneration.
7. Packaging and Storage: After sterilization, the allograft is packaged in sterile containers and stored until needed.

Characteristics of Allograft:

- Osteoconductive: Provides a scaffold for new bone growth by supporting the migration and attachment of osteoblasts.
- Osteoinductive: In some grafts (e.g., DBM), the ability to stimulate the differentiation of mesenchymal stem cells into osteoblasts.
- Non-Immunogenic: Processing methods (e.g., freeze-drying, irradiation) aim to remove or reduce cellular components to lower immune rejection risk.
- Mechanical Properties: The graft retains varying degrees of its original strength, depending on processing techniques (e.g., fresh-frozen vs. irradiated).
- Biocompatibility: Allografts are biologically compatible, but less so than autografts (tissue from the same individual).

Stages of Graft Uptake (Graft Incorporation):

The process of graft uptake consists of several stages, which involve cellular and molecular interactions between the graft and the recipient's body.

1. Inflammation (Days 1–7):
   - After implantation, the host's immune system recognizes the graft as foreign, resulting in an inflammatory response.
   - Neutrophils and macrophages infiltrate the graft area to clear necrotic tissues and any residual immunogenic cells. Cytokines and growth factors are released to promote healing.
2. Vascularization (Days 7–21):
   - Angiogenesis (new blood vessel formation) occurs within the graft.
   - Vascularization is essential for nutrient delivery and removal of waste products, enabling graft survival and integration.
3. Osteoinduction & Osteoconduction (Weeks 2–6):
   - Osteoblasts migrate to the graft site and begin to form new bone.
   - Osteoinductive properties (e.g., in DBM) stimulate the differentiation of mesenchymal stem cells into osteoblasts, enhancing bone regeneration.
   - Osteoconduction occurs as the graft provides a scaffold for new bone growth.
4. Remodeling (Weeks to Months):
   - New bone begins to remodel and integrate with the host bone.
   - Osteoclasts resorb necrotic bone from the graft,

while osteoblasts lay down new bone. The graft gradually becomes indistinguishable from native bone, fully integrating over several months to a year, depending on the type and size of the graft.

5. Graft Incorporation:
   - Complete integration of the graft into the recipient's bone structure with restoration of mechanical function.
   - Long-term incorporation varies by graft type, recipient's health, and surgical technique.

Question 5.

Etiopathogenesis of Downs syndrome, musculoskeletal features of downs, Name any 4 HLA B 27 positive rheumatic diseases, Extracble nuclear antigen test.

Answer 5.

Down's syndrome is a genetic disorder caused by the presence of an extra copy of chromosome 21. The etiopathogenesis involves:

- Non-Disjunction (95% of cases):
  - Occurs when chromosome 21 fails to separate properly during meiosis, resulting in a gamete with an extra copy of chromosome 21. After fertilization, the zygote has three copies of chromosome 21 instead of two (trisomy 21).
- Robertsonian Translocation (3-4% of cases):
  - Part of chromosome 21 becomes attached (translocated) to another chromosome, typically chromosome 14. A parent may carry a balanced translocation without showing symptoms but can pass an unbalanced translocation to the child.
- Mosaicism (1-2% of cases):
  - A rare form where some cells have the normal 46 chromosomes while others have an extra chromosome 21. This occurs due to a post-zygotic error during mitosis, resulting in a mixture of normal and trisomic cells.

Musculoskeletal Features of Down's Syndrome:

Individuals with Down's syndrome often have distinctive musculoskeletal characteristics, including:

- Hypotonia: Common in infants, causing floppiness and delayed motor development.
- Joint Laxity: Increased flexibility due to ligamentous laxity, especially in the hips, knees, and elbows.
- Atlantoaxial instability (instability between the first and second cervical vertebrae) may lead to neurological complications.
- Short Stature: Growth retardation and short stature are common throughout life.
- Pes Planus: Low or absent arches in the feet, which can lead to foot pain and difficulty walking.
- Scoliosis: Lateral curvature of the spine may develop over time.

Four common HLA-B27 positive rheumatic diseases include:

1. Ankylosing Spondylitis (AS): A chronic inflammatory disease primarily affecting the spine and sacroiliac joints, leading to pain and stiffness.
2. Reactive Arthritis: Arthritis that develops as a reaction to an infection, commonly gastrointestinal or urogenital infections.
3. Psoriatic Arthritis: A type of inflammatory arthritis associated with psoriasis, affecting joints and connective tissue.
4. Enteropathic Arthritis: Arthritis associated with inflammatory bowel diseases such as Crohn's disease and ulcerative colitis.

Extractable Nuclear Antigen (ENA) Test:

The Extractable Nuclear Antigen (ENA) test is a blood test used to detect specific autoantibodies that target proteins in the cell nucleus, helping in the diagnosis of autoimmune diseases. The ENA panel typically includes tests for antibodies against the following:

1. Anti-Sm (Anti-Smith): Associated with systemic lupus erythematosus (SLE).
2. Anti-RNP (Ribonucleoprotein): Found in mixed connective tissue disease and SLE.
3. Anti-SS-A (Ro) and Anti-SS-B (La): Associated with Sjögren's syndrome and systemic lupus erythematosus.
4. Anti-Scl-70: Seen in systemic sclerosis (scleroderma).
5. Anti-Jo: For Dermatomyositis.

Question 6.

Eysipelas, cellulitis, pyomyositis, Necrotising fascitis, Gas gangrene- affected tissue, pathogenesis, organism, clinical features, treatment

Answer 6.

Condition	Affected Tissue	Pathogenesis	Common Organisms	Clinical Features	Treatment
Erysipelas	Upper dermis and superficial lymphatics	• Infection spreads via skin breaks (e.g., cuts, insect bites).	• Streptococcus pyogenes (Group A strep)	• Rapidly spreading erythematous, raised, and sharply demarcated rash • Affected area is tender, swollen, and warm • Fever, chills, malaise may be present	• Antibiotics: Penicillin (IV or oral), amoxicillin • IV antibiotics for severe cases • Elevation of the limb • Symptomatic treatment (e.g., antipyretics)
Cellulitis	Deeper dermis and subcutaneous tissue	• Bacterial entry through cuts, abrasions, or insect bites • Bacteria spread within the subcutaneous tissue	• Staphylococcus aureus (including MRSA) • Streptococcus pyogenes	• Ill-defined, erythematous, swollen area of skin • Warm, painful, and tender skin • Fever, chills, fatigue • Localized lymphadenopathy	• Antibiotics: Cephalexin, amoxicillin-clavulanate, clindamycin, or vancomycin (for MRSA) • Elevation • Treat underlying skin breaks
Pyomyositis	Skeletal muscles	• Bacterial infection causing muscle abscess • Typically secondary to trauma or hematogenous spread	• Staphylococcus aureus (most common) • Streptococcus spp. • Other Gram-negative organisms may be involved	• Fever, localized muscle pain, swelling, and tenderness • Warm, erythematous overlying skin • Pus formation within muscles • May progress to sepsis	• Antibiotics: Empiric therapy targeting staph and strep (e.g., cefazolin or vancomycin) • Drainage of abscess • Supportive care
Necrotizing Fasciitis	Fascia, subcutaneous tissue, and muscle	• Rapidly spreading infection with toxins causing extensive tissue necrosis • May begin with minor trauma or surgical wounds • High mortality rate	• Streptococcus pyogenes (Group A strep) • Clostridium perfringens • Mixed infections (Gram-positive, Gram-negative, anaerobes)	• Severe, rapidly progressing pain • Skin overlying may appear pale, dusky, or erythematous • Blisters, bullae, crepitus • Systemic toxicity (fever, hypotension, shock)	• Emergency surgical debridement to remove necrotic tissue • Broad-spectrum antibiotics: Vancomycin + Piperacillin-tazobactam • IV fluids

Gas Gangrene	Muscle and subcutaneous tissue	• Clostridial infection produces gas and toxins within tissues, leading to necrosis • Follows trauma or surgery	• Clostridium perfringens • Other Clostridium species	• Sudden onset of severe pain at site of injury • Tense swelling, skin discoloration (bronze/ purple), and crepitus • Tachycardia, fever, hypotension • Shock may occur rapidly	• Surgical debridement (emergent) • Antibiotics: Penicillin + clindamycin • Hyperbaric oxygen therapy (adjunctive) • Amputation may be required

Question 7.

Etiopathogenesis of crush syndrome, clinical features and management.

Answer 7.

A crush injury occurs when heavy physical pressure is applied to the muscles, resulting in significant damage. Crush syndrome, also known as rhabdomyolysis, is a severe condition that arises from major muscle injuries, where cellular integrity is compromised, causing muscle contents to leak into the bloodstream.

Crush injuries can be life-threatening, with up to 80% of victims dying from severe head trauma or asphyxiation. Among those who survive and make it to the hospital, about 10% recover without complications, while another 10% develop crush syndrome, which is marked by serious metabolic disturbances. During this process, muscle cells release substances like myoglobin, which eventually breaks down into metmyoglobin and acid hematin in the bloodstream. Other substances like potassium, magnesium, phosphate, acids, and enzymes such as creatine phosphokinase (CKMM) and lactate dehydrogenase (LDH) are also released. While normally essential for cellular function, these substances become toxic in large amounts when they enter the bloodstream.

Compression of muscles also leads to local ischemia as both micro and macrocirculation is blocked. This ischemia causes sodium, calcium, and fluids to flood into the muscles, leading to swelling and increased muscle tension. The depletion of CK and ATP, along with activation of the nitric oxide system, worsens muscle vasodilation and hypotension. Upon rescue and relief of pressure, the return of blood flow to the ischemic muscles disrupts the sodium-potassium-ATPase mechanism, causing the release of myoglobin breakdown products, lactic acid, uric acid, and muscle enzymes into the blood. Myoglobin is filtered through the kidneys but can clog the distal tubules when it exceeds renal thresholds. Its breakdown products can also cause vasoconstriction of the afferent arterioles, worsening kidney damage.

Lactic acid levels rise in proportion to the duration of muscle ischemia, but serum creatine phosphokinase (CK) is the main indicator of muscle injury. The degree of oliguria and kidney damage doesn't necessarily match the extent of muscle injury. Affected muscles may become swollen, hard, cold, and necrotic, and kidneys can show signs of edema and increased volume. Elevated potassium levels can cause cardiac rhythm issues, and patients may go into shock due to lung edema, heightening the risk of acute respiratory distress syndrome (ARDS). Though crush injuries are less common than head or chest injuries—since extended pressure often results in death—up to 10% of chest trauma cases involve crush injuries.

Crush syndrome is typically diagnosed when serum creatine kinase (CKMM) levels rise above 1000 IU/l. Levels increase within 2 to 12 hours after the injury, peak in 1 to 3 days, and decline after 3 to 5 days. Other helpful tests include measurements of serum aldolase, myoglobin, and its breakdown products, which are highly sensitive indicators. Serum lactic acid, AST, ALT, LDH, uric acid, and potassium levels also rise, and hypocalcemia and stress-related hyperglycemia may be seen. Urinalysis can detect myoglobin products, and blood gas analysis, hemograms, and ECGs provide further information. Intracompartmental pressure monitoring is vital, with pressures over 30 mm Hg suggesting the need for a fasciotomy. Doppler studies help assess limb ischemia, and body weight is monitored.

Patients may initially appear stable after rescue but rapidly deteriorate into shock. Common symptoms include petechiae, blisters, bruised muscles, superficial injuries, muscle pain, paralysis, sensory loss, fever, cardiac arrhythmias, pneumonia, dark-colored urine (like tea or cola), reduced urine output, and kidney failure. Those rescued late may also experience nausea, vomiting, agitation, and delirium.

Managing crush injuries involves several key steps: rescue, resuscitation, syndrome recognition, treatment, and rehabilitation. Early fluid resuscitation, ideally within the first 6 hours, is critical. Saline is commonly used, with bicarbonate or lactate to counteract metabolic acidosis. Monitoring includes checking central venous pressure (CVP), blood pressure (BP), lung function, and urine output. Blood transfusions may be necessary. Maintaining kidney function is essential. In established crush syndrome, urine output should be at least 300 ml/hr, requiring about 12 liters of fluid per day. Mannitol is often used to promote diuresis. Dialysis is crucial in managing severe cases, particularly if there is anuria, fluid overload, high serum creatinine, elevated BUN, bicarbonate, or potassium levels above 7 meq/l. Patients may need dialysis up to three times daily for 15 days, and prophylactic dialysis may be necessary for those at risk of hyperkalemia. Administered at high pressures, this treatment increases plasma oxygen levels, improves tissue viability, reduces swelling, aids wound healing, and limits anaerobic bacterial growth. A typical regimen is 2.5 atmospheres for 1.5 hours, twice daily for a week. Broad-spectrum, non-nephrotoxic antibiotics are often required. Procedures like laparotomy or thoracotomy may be needed for extensive debridement of dead muscle tissue, with suturing either primary or secondary. Fasciotomy should be performed early to prevent the need for amputation if compartment pressure rises. Fractures should be stabilized, and conservative amputations may be required. Tetanus immunization is recommended, and rehabilitation is important to manage muscle contractures and other long-term complications.

Question 8.

Etiopathogenesis of DVT and Pulm. Embolism. Clinical features. Current evidence based prophylaxis - type of drug, mechanism of action, ADR, post op usage.

Answer 8.

- Deep venous thrombosis (DVT) is a common condition that affects about 1 person per 1,000 population per year.
- It is associated with many medical conditions but is a relatively common complication of trauma.
- About 20% of multiply injured patients die as a result of a DVT.
- It is also one of the most significant complications after THA & TKA, possibly resulting in life-threatening PE.
- In early reports of total hip arthroplasty without routine prophylaxis, venous thrombosis occurred after total hip replacement in 50% of patients, and fatal pulmonary emboli occurred in 2%.
- The overall prevalence of DVT after TKA without any form of mechanical or pharmaceutical prophylaxis has been reported to range from 40% to 84%.

Risk Factors for Venous Thromboembolic Disease

Clinical Risk Factors	Hemostatic Abnormalities (Hypercoagulable States)
Advanced age	Antithrombin III deficiency
Fracture of pelvis, hip, femur, or tibia	Protein C deficiency
	Protein S deficiency
Paralysis or prolonged immobility	Dysfibrinogenemia
Prior venous thromboembolic disease	Lupus anticoagulant and antiphospholipid antibodies
Operation involving abdomen, pelvis, or lower extremities	Myeloproliferative disorder
	Heparin-induced thrombocytopenia
Obesity	
Congestive heart failure	Disorders of plasminogen and plasminogen activation
Myocardial infarction	
Stroke	

ETIOLOGY

- Venous thrombi are mainly composed of fibrin and erythrocytes together with platelets and leucocytes.
- They usually form in venous sinuses in the deep veins of the calf and in veins damaged by trauma.
- Damage to the endothelium of the veins leads to platelet adhesion, leucocyte accumulation, and the activation of the intrinsic and extrinsic pathways.
- Cytokines and tumor necrosis factor may be released, causing distant venous damage leading to coagulation.
- Venous stasis during surgery or as a result of trauma may contribute by slowing the clearance of coagulative factors.
- This is particularly important in lower limb surgery because the normal pumping action of the calf muscles may be slowed or abolished.

- There is a clear association between DVT and PE.
- About 50% of patients with a proven DVT develop PE, and about 70% of patients with confirmed PE have asymptomatic DVTs.
- Pulmonary emboli can take the form of multiple small emboli or a single large, often fatal embolus.
- Most significant PEs arise from the proximal veins.

DIAGNOSIS

- The clinical signs of DVT include leg pain and swelling, edema, altered temperature, dilated superficial veins, and erythema.
- Tests include assessment of leg circumference and Homan's sign, which is calf pain caused by forced dorsiflexion of the ankle with the knee flexed.
- Clinical examination is unreliable in detecting DVT because most clots occur without signs or symptoms.
- Comparison with venography has shown that these clinical signs and tests are inaccurate, and the diagnosis of DVT requires the use of venography, ultrasound, or impedance plethysmography.
- Venography remains the standard investigation.
    1. Contrast material is injected into the superficial veins of the foot and x-rays are used to delineate areas of inadequate venous filling.
    2. It requires expert radiological assessment and is inconvenient if multiple studies are required.
    3. Venography carries the risk of anaphylactic reaction to the contrast media and a small risk of inducing DVT
- Duplex ultrasound seems to be useful, especially as a screening test, because of its minimal morbidity, low cost, and repeatability with minimal patient discomfort, but its accuracy depends on the experience of the ultrasound technologist.
- Duplex ultrasound is particularly useful for visualization of proximal vein DVT and is less effective for calf vein DVT.
- An alternative technique is impedance plethysmography, which has been shown to have a high accuracy in the diagnosis of thigh DVT in particular.
- These tests remain the standard diagnostic tests for proving the presence of a DVT.
- The diagnosis of PE is also difficult if the surgeon relies on the clinical criteria of chest pain and breathlessness because these symptoms are caused by many different medical conditions.
- Diagnosis is best undertaken by angiography or by a perfusion lung scan.
- Investigations should also include an ECG to exclude cardiac problems, a chest x-ray, and arterial blood gases.

Prophylaxis for deep vein thrombosis (DVT) typically involves early mobilization, mechanical methods, antiplatelet medications, and heparin. Prolonged immobility is a known risk factor for DVT, so it is generally agreed that patients should begin moving as soon as their condition allows. Active exercises of the lower limbs help reduce venous stasis and the formation of blood clots. Mechanical methods of DVT prevention include graduated elastic compression stockings (GECS), intermittent pneumatic compression, and mechanical foot pumps. These methods, particularly mechanical compression boots and foot pumps, are beneficial as they pose little risk to patients. However, their effectiveness is limited by patient compliance and the short duration of hospital stays. Combining GECS with pharmacological prophylaxis is more effective than using medication alone. Aspirin, although commonly used, has not proven effective in preventing DVT after total knee arthroplasty (TKA), with reported DVT rates between 59% and 73%, and it also increases the risk of bleeding during surgery.

- Warfarin is often used for prophylaxis, starting either the evening before or just after surgery, with dosages adjusted based on daily prothrombin time. The international normalized ratio (INR) standardizes anticoagulant effects, with a target range of 2.0 to 3.0 for warfarin therapy. Warfarin's advantages include oral administration and low cost, but its downsides are numerous, including drug interactions, the need for regular monitoring, delayed onset of action, and the risk of bleeding complications.
- Low Molecular Weight Heparin (LMWH) is another widely used option, with enoxaparin being the most common, though ardeparin, logiparin, dalteparin, and nadroparin have also been studied. These drugs differ from unfractionated heparin in that they have less antithrombin activity and do not significantly prolong activated partial thromboplastin time.

LMWH can be administered subcutaneously once or twice a day without the need for regular monitoring, though periodic checks of platelet count are recommended due to the potential for heparin-induced thrombocytopenia. The main advantages of LMWH are its standardized dosage and the lack of routine lab monitoring, while disadvantages include higher cost, subcutaneous administration, and an increased risk of bleeding. Patient compliance after hospital discharge can also be a concern. If blood is observed during the placement of a needle or catheter, the use of LMWH should be delayed for 24 hours. Administering LMWH twice a day may increase the risk of hematoma formation, so the first dose should be delayed for 24 hours post-surgery, with proper hemostasis ensured. If continuous postoperative epidural use is necessary, the first dose of LMWH should be given two hours after catheter removal. Single-dose LMWH can usually be started 6 to 8 hours after surgery, with the second dose more than 24 hours later. Indwelling catheters should be removed 10 to 12 hours after the last LMWH dose, and the next dose should not be given until at least two hours after catheter removal. For patients on warfarin, the epidural catheter should be removed when the INR is below 1.5, and if a preoperative dose of warfarin was administered, the INR should be checked before inserting the catheter.

- Fondaparinux, a factor Xa inhibitor administered via a single daily injection, is the newest anticoagulant used for preventing thromboembolism in patients undergoing total hip, total knee, or hip fracture surgeries. Studies comparing fondaparinux with enoxaparin have shown similar or better effectiveness in preventing DVT, with comparable rates of major bleeding. Like LMWH, fondaparinux carries a risk of bleeding complications and thrombocytopenia, and it should not be used in patients with renal insufficiency
- For patients undergoing elective hip or knee replacement surgery, the American College of Chest Physicians recommends using one of three anticoagulants: LMWH, fondaparinux, or a vitamin K antagonist with an INR target of 2.5 (range 2.0 to 3.0). Thromboprophylaxis should continue for at least 10 days following hip or knee surgery or hip fracture repair. However, continuing prophylaxis after hospital discharge poses a challenge. If anticoagulants are needed post-discharge, preparation for monitoring their effects is essential. This involves frequent clinical evaluations for wound complications and regular laboratory checks for patients on warfarin. High-risk patients, especially those with a prior history of thromboembolism, may need to stay on LMWH or warfarin for up to six weeks after surgery.

Contraindications to Pharmacologic DVT Prophylaxis

1. Uncorrected bleeding disorders
2. Major hemorrhage
3. Intracranial bleed
4. Pelvic hemorrhage
5. Esophageal varices
6. Peptic ulcer
7. Spinal cord injury
8. Anemia
9. Allergy
10. Heparin-associated thrombocytopenia
11. Aspirin-induced asthma
12. Severe liver impairment
13. Severe renal impairment

TREATMENT

- Despite early mobilization, mechanical and pharmacological prophylaxis, and careful clinical monitoring, some patients develop deep vein thrombosis and pulmonary embolism and require full therapeutic anticoagulation.
- Respiratory support, serial arterial blood gas determinations, and repeat lung scans may be indicated.
- Heparin is administered to provide initial anticoagulation.
- Warfarin is then introduced, and the dose adjusted according to the international normalized ratio (INR), which should be within the range of 2.0 to 3.0.
- Warfarin treatment is continued for at least 3 months.
- Two thirds of patients who die of pulmonary emboli do so within 30 minutes.

- Patients who survive long enough to have the diagnosis established usually survive if appropriate treatment is started, provided that they do not have certain other comorbidities.
- If additional pulmonary emboli develop despite adequate anticoagulation, or if bleeding complications occur because of anticoagulation, it may be necessary to place a filter in the inferior vena cava.
- Such a filter may be indicated preoperatively in patients deemed to be at excessively high risk.
- Thrombolytic enzymes, such as urokinase and streptokinase, have been used to dissolve emboli, but these can produce massive hemorrhage from the surgical wound.

Question 9.

EMG, NCV- and it's interpretation, explain it with example of iatrogenic radial n.injury during surgery of humerus #

Answer 9.

Electrodiagnostic studies are helpful in determining the early phases of recovery as well as in detecting nerve injury, compression, or both.

After nerve damage, electromyography (EMG) is carried out at least four weeks later. Early EMG testing could result in false-negative results because muscle fibrillations take 4-6 weeks to manifest. The presence of fibrillations in the muscle is indicative of denervation. Motor unit potentials (MUPs) indicate the presence of reinnervation.

Studies on nerve conduction velocity are especially helpful in locating potential secondary compression sites. It is best done at 2 weeks. Axonal regeneration may be hindered and reinnervation may be limited if the nerve is compressed at an entrapment site. An avulsion injury can be diagnosed by motor denervation and intact normal distal sensory nerve conduction.

Terms related to NCV:

SNAP and CMAP- The recorded response resulting from the depolarization of a muscle or sensory nerve is known as a compound muscle action potential (CMAP) or sensory nerve action potential (SNAP). Axon loss, which lowers the distal signal, frequently causes a decline in these values. But in the case of a demyelinating conduction block, where the signal is unable to pass through the demyelinated segment, the amplitude of the SNAP and CMAP can also decrease. In this case, a smaller CMAP amplitude will be produced by stimulating the nerve closer to the conduction block site than by stimulating the nerve farther away from the block.

Velocity and Latency- Conduction velocity, expressed in metres per second, is the signal speed over a measured segment. When confined to a particular point of compression entrapment, such as the elbow in cubital tunnel syndrome, a decrease in velocity may signify a demyelinating process. Alternatively, it may indicate a peripheral demyelinating disease without entrapment, such as chronic inflammatory demyelinating polyneuropathy or Charcot Marie Tooth. In a similar vein, latency is the measurement of the milliseconds it takes a signal from a stimulus point to the target electrode. Unlike SNAP and CMAP amplitudes, which are correlated with the number of axons within the nerve, velocity and latency are determined by the fastest-conducting fibres in a nerve.

Terms related to EMG:

Insertional and spontaneous activity- A brief burst of insertional activity is a normal, physiological reaction to the insertion of a needle electrode into a muscle. This insertional activity increases with acute denervation of muscles. The insertional activity will decrease as fat and fibrous connective tissue replace muscle that has been chronically denervated. A normal muscle is silent at rest after the initial insertional activity. However, positive sharp waves (PSWs) and fibrillation potentials are continuous, spontaneous activity seen in acutely denervated muscle. The length of time it takes to develop this spontaneous activity is correlated with the distance between the motor end plates (MEPs) and the nerve injury because of the active process of Wallerian degeneration. PSWs and fibrillations can gradually vanish under various circumstances.

Fibrillations and Positive Sharp Waves- Fibrillations and PSWs in axonal injuries with minimal axonal loss may go away in a few months as collateral growing from nearby, uninjured axons reinnervates the denervated MEPs. Fibrillations and PSWs may eventually go away in axonal injuries with higher axon loss because the muscle is successfully reinnervated by developing axons. If receptive MEPs are no longer present and reinnervation does not

happen promptly, they might also vanish. It is significant to remember that, even though PSWs and fibrillations are recorded as a range of 0–4+, the numerical scale is ordinal and, as a result, does not represent a linear quantitative value but rather a qualitative severity of denervation. A low chance of surgical reinnervation is indicated by decreased or absent insertional activity.

Motor unit action potential or motor unit potential and morphology- Based on their recruitment and morphology, motor unit action potentials (MUPs), which are connected MEPs with intact muscle activity, are qualitatively described. Additional MUPs are not recruited when an acutely partially denervated muscle is activated; as a result, the available MUPs fire more quickly in an attempt to produce the desired amount of force in the muscle fibres innervated by that intact motor unit. Thus, this compensation will sustain normal clinical function up to a certain point. This gradual increase in MUPs shows up on an EMG as a reduction in the recruitment of new MUPs and a faster firing rate of the MUPs that are already there. Crucially, recruitment is physiologic and serves as a proxy for the number of axons that are innervating target muscles, in contrast to activation, which can be lowered by any process involving the central nervous system. Reinnervation in recovering muscle results in MUPs that are longer in duration, have a greater amplitude, and may exhibit more polyphasia. These alterations are all the consequence of nearby, unharmed axons or developing axons from the initial, damaged axons collaterally sprouting. Reinnervated MUPs eventually lose their polyphasic morphology but retain their larger amplitude and, slightly, longer duration morphology. Therefore, muscle that has been successfully reinnervated is indicated by chronic MUP morphology.

Electrodiagnostic Finding	Interpretation
Decreased SNAP and CMAP	Represent axonal loss and are quantitative.
Decreased conduction velocity	Severely reduced velocity represents demyelination. Mild slowing of conduction velocities can be seen in primary axon loss.
Presence of fibrillations and PSW	Represents acutely denervated muscle.
Reduced or absent insertional activity	Suggests a poor potential for surgical reinnervation.
MUPs	Represent innervated MEPs within muscle.
Polyphasic MUPs	Represent early reinnervation
Chronic MUPs	Represent longer standing reinnervated muscle from collateral sprouting and nascent axons.
Decreased recruitment	Represents a loss of innervated MEPs with severe implying prominent loss and mild implying less prominent loss.

H reflexes can be abnormal even in cases of mild neuropathies and are a sensitive test for polyneuropathies. H reflexes involve both proximal and distal fibre conduction. Therefore, even when studies of distal function are unremarkable, these studies may be abnormal because they define proximal nerve injury. Acute inflammatory demyelinating polyneuropathy, or Guillain-Barré syndrome, is characterised by absent H reflexes. H reflexes may be abnormal in plexopathies and radiculopathies, as well as in asymptomatic patients with potential neuropathic dysfunction. H reflexes in the calf and forearm flexor muscles can both be abnormal in cases of S1 radiculopathies and C6 or C7 root injuries, respectively. Damage to the anterior or posterior roots can impact H reflexes. Hence, even in cases where needle EMG is unrevealing due to sparing of the anterior roots, examination of these reflexes can be crucial in the electrodiagnostic evaluation of radiculopathies by confirming nerve injury. Their usefulness in identifying an S1 radiculopathy may be improved by eliciting the H reflex through direct stimulation of the S1 root.

F waves are common, low-amplitude responses that vary in latency, configuration, and amplitude by nature. They are created when motor neurons experience antidromic activation, or "backfiring." F waves are more noticeable during high-intensity stimulation than H reflexes.

The most often reported F wave parameters are latencies. F-wave latencies correlate directly with limb length, age, and height, though not as much with age. The range of conduction velocities in the axons during the recorded F waves can be determined by comparing the minimal and maximal latencies in a sequence of F waves. The percentage of detectable F responses that endure after a sequence of stimuli is known as F-wave persistence, and it is associated with the antidromic excitability of a specific motor neuron pool. F-wave latencies may be prolonged in proximal nerve or root injury. The percentage of repeater waves is increased in neurogenic injury, especially with atrophy. F waves have been abnormal in greater than 90% of nerves in patients with acute and chronic demyelinating polyneuropathies. Prolonged F-wave latencies are a

sensitive abnormality in polyneuropathies and may be present even when more distal motor nerve conductions are unremarkable.

Findings in an iatrogenic radial nerve injury:

NCV Findings:

- Sensory Nerve Conduction Study: A reduced amplitude or absence of sensory nerve action potentials (SNAPs) along the radial nerve in the affected arm would indicate axonal injury.
- Motor Nerve Conduction Study: Delayed or absent motor response from stimulating the radial nerve, typically at the level of the elbow or spiral groove, can indicate localized nerve damage (conduction block) or axonal loss.

EMG Findings:

- Resting Activity: Fibrillations and positive sharp waves may be seen in the radial nerve-innervated muscles (e.g., extensor carpi radialis, extensor digitorum) if there is axonal degeneration.
- Voluntary Activity: Reduced motor unit recruitment or absence of motor units in muscles innervated by the radial nerve may be observed during attempts to extend the wrist and fingers.

Question 10.

a. Biomechanics of hip with diagram.

b. Use of stick with diagram

Answer 10a.

The biomechanics of the hip refers to the study of the forces, movements, and mechanical functions of the hip joint, which is a ball-and-socket joint formed by the head of the femur and the acetabulum of the pelvis. This area of biomechanics examines how the hip joint facilitates movements such as walking, running, sitting, and standing, and how it bears and distributes loads throughout these activities.

Key aspects of hip biomechanics include the range of motion (flexion, extension, abduction, adduction, rotation), muscle activation (particularly the roles of muscles like the gluteals, hip flexors, and adductors), joint stability, and how forces are transmitted through the joint during weight-bearing activities. Understanding hip biomechanics is crucial for analyzing normal movement patterns, diagnosing injuries, and developing rehabilitation strategies.

When standing on one leg, several key processes occur. The center of gravity shifts away from the leg that is bearing the body's weight. The hip of the weight-bearing leg is influenced by the fact that the non-weight-bearing leg is still part of the body mass. This creates a downward force that generates rotational movement around the center of the femoral head. To counteract this rotation, the hip abductors—such as the tensor fascia latae, gluteus medius and minimus, piriformis, and obturator internus—work together to produce a force that stabilizes the femoral head. This creates a moment around its center. However, the lever arm of the hip abductors is shorter compared to the lever arm of the body's weight. As a result, the abductors need to exert a force several times greater than body weight—usually around three times the body's weight, corresponding to a lever ratio of about 2.5.

The intensity and distribution of forces on the hip joint are influenced by several factors. Mechanically, two important parameters are the body-weight lever arm, which is related to pelvis width, and the abductor lever arm, which is influenced by the neck-shaft angle and neck length.

An increase in the abductor lever arm reduces the force needed by the abductor muscles to keep the pelvis level, which in turn decreases the reaction forces on the hip joint. On the other hand, when standing on one leg, a wider pelvis increases the body-weight lever arm, resulting in higher joint contact forces. Other factors that intricately affect the magnitude and distribution of hip contact forces include the orientation of the acetabulum and femoral neck, the shape of the femoral head, the elasticity of cartilage and bone, and slight incongruities in the joint caused by acetabular deformation under increased load. While sliding movements dominate in an ideal ball-and-socket joint, nonspherical shapes also allow rolling movements. Cartilage elasticity, which optimizes load transfer in the hip joint, is compromised during arthroplasty procedures. Studies show that the load on the hip increases significantly when acetabular anteversion is reduced. A similar effect occurs when the neck-shaft angle or femoral anteversion is decreased. People with short femoral necks and wide pelvises require greater forces in the hip. Due to their higher lever arm ratio, these individuals need stronger abductor muscle