

**NCERT Exemplar Solutions of Class 11 Biology – Chapter 20: Locomotion and Movement**  
**LONG ANSWER TYPE QUESTIONS**

**1. Calcium ion concentration in the blood affects muscle contraction. Does it lead to tetany in certain cases? How will you correlate fluctuation in blood calcium with tetany?**

**Enhanced Solution:**

**Yes, calcium ion concentration fluctuations can lead to tetany.**

**Role of calcium in muscle contraction:**

**1. Normal muscle contraction mechanism:**

Ca<sup>2+</sup> ions bind to troponin C on thin filaments

Causes conformational change in tropomyosin

Exposes myosin binding sites on actin

Enables cross-bridge formation and muscle contraction

**2. Calcium regulation:**

**Parathyroid hormone (PTH):** Increases blood Ca<sup>2+</sup> levels

**Calcitonin:** Decreases blood Ca<sup>2+</sup> levels

**Vitamin D:** Enhances Ca<sup>2+</sup> absorption

**TETANY AND CALCIUM CORRELATION:**

**Hypocalcemic tetany (Low blood calcium):**

**Causes:**

**Parathyroid hormone deficiency:** Hypoparathyroidism

**Vitamin D deficiency:** Poor calcium absorption

**Magnesium deficiency:** Required for PTH function

**Alkalosis:** Increases protein-bound calcium

**Mechanism:**

**Increased nerve excitability:** Low Ca<sup>2+</sup> decreases threshold for nerve firing

**Spontaneous depolarization:** Nerves fire without stimulation

**Continuous muscle stimulation:** Results in sustained contractions

**Clinical manifestations:**

**Carpopedal spasm:** Contraction of hands and feet muscles

**Laryngospasm:** Vocal cord muscle spasm

**Chvostek's sign:** Facial muscle twitching when facial nerve tapped

**Trousseau's sign:** Hand spasm when blood pressure cuff inflated

**Physiological explanation:**

**Normal Ca<sup>2+</sup> levels:** 8.5-10.5 mg/dL

**Tetany threshold:** Usually occurs when Ca<sup>2+</sup> < 7 mg/dL

**Membrane stability:** Ca<sup>2+</sup> stabilizes nerve membrane potential

**Reduced calcium:** Makes nerves hyperexcitable

**Treatment:**

**Immediate:** IV calcium gluconate

**Long-term:** Calcium supplements, Vitamin D, treat underlying cause

**2. An elderly woman slipped in the bathroom and had severe pain in her lower back. After X-ray examination doctors told her it is due to a slipped disc. What does that mean? How does it affect our health?**

**Enhanced Solution:**

**WHAT IS A SLIPPED DISC?**

**Definition:** A slipped disc (herniated or prolapsed disc) is a condition where the **intervertebral disc is displaced from its original position** between vertebrae.

**Anatomy of intervertebral disc:**

**Annulus fibrosus:** Tough outer ring of fibrocartilage

**Nucleus pulposus:** Soft, jelly-like inner core

**Function:** Acts as shock absorber and allows spinal flexibility

**Mechanism of injury:**

**Disc degeneration:** Age-related wear and tear weakens annulus fibrosus

**Sudden stress:** Fall, lifting, or twisting motion

**Rupture:** Nucleus pulposus pushes through weakened annulus

**Compression:** Herniated material presses on spinal nerves or spinal cord

**HEALTH EFFECTS:**

**Immediate effects:**

**Severe pain:** Local back pain and radiating pain down legs (sciatica)

**Muscle spasm:** Protective muscle contractions around injury site

**Limited mobility:** Difficulty in bending, sitting, or walking

**Neurological effects:**

**Nerve compression:** Pressure on spinal nerves

**Numbness and tingling:** In legs, feet, or areas served by compressed nerves

**Muscle weakness:** In muscles innervated by affected nerves

**Reflex changes:** Diminished or absent reflexes

**Long-term complications:**

**Chronic pain:** Persistent lower back and leg pain

**Permanent nerve damage:** If left untreated

**Cauda equina syndrome:** Severe compression causing bowel/bladder dysfunction

**Paralysis:** In extreme cases with significant spinal cord compression

**Functional limitations:**

**Activities of daily living:** Difficulty in dressing, bathing, cooking

**Occupational impact:** May affect work performance and attendance

**Social limitations:** Reduced participation in recreational activities

**Sleep disturbances:** Pain interfering with sleep quality

**Treatment options:**

**Conservative:** Rest, physiotherapy, pain medications, epidural injections

**Surgical:** Discectomy, laminectomy in severe cases

**Prevention strategies:**

**Proper lifting techniques:** Bend knees, not back

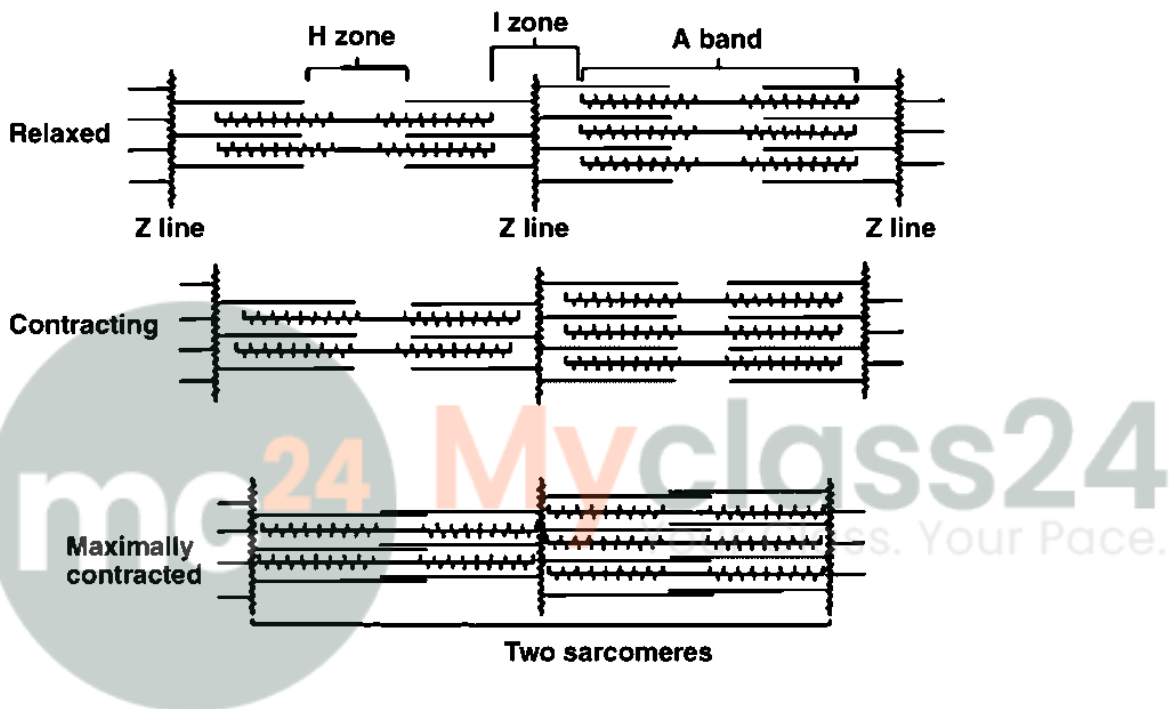
**Regular exercise:** Strengthen core and back muscles

**Weight management:** Reduce stress on spine

**Good posture:** Maintain spinal alignment

**3. Explain sliding filament theory of muscle contraction with neat sketches.**

**Enhanced Solution:**



**SLIDING FILAMENT THEORY:**

The sliding filament theory explains that muscle contraction occurs through the **sliding of thin filaments (actin) over thick filaments (myosin)** without the filaments themselves shortening.

**STRUCTURAL COMPONENTS:**

**Sarcomere structure:**

**I-band:** Contains only actin filaments (light band)

**A-band:** Contains both actin and myosin filaments (dark band)

**H-zone:** Contains only myosin filaments (within A-band)

**Z-line:** Attachment point for actin filaments

**M-line:** Center of myosin filaments

**Protein components:**

**Actin:** Thin filaments with binding sites for myosin

**Myosin:** Thick filaments with globular heads containing ATPase

**Tropomyosin:** Regulatory protein covering myosin binding sites

**Troponin:** Calcium-binding regulatory protein complex

**MECHANISM OF CONTRACTION:**

**Step 1: Excitation-Contraction Coupling**

Neural signal reaches neuromuscular junction

Acetylcholine released, creating action potential

Action potential spreads along sarcolemma and T-tubules

Sarcoplasmic reticulum releases  $\text{Ca}^{2+}$  ions

**Step 2: Calcium Binding**

$\text{Ca}^{2+}$  binds to troponin C

Conformational change moves tropomyosin

Myosin binding sites on actin become exposed

**Step 3: Cross-Bridge Formation**

Myosin heads bind to actin forming cross-bridges

ATP provides energy for myosin head cocking

**Step 4: Power Stroke**

Myosin heads pivot, pulling actin filaments

ADP and  $\text{P}_i$  released during power stroke

Actin slides toward center of sarcomere

**Step 5: Cross-Bridge Breaking**

New ATP binds to myosin head

Cross-bridge breaks, myosin head detaches

ATP hydrolysis re-cocks myosin head

**Step 6: Cycle Continues**

Process repeats as long as  $\text{Ca}^{2+}$  and ATP are available

Multiple cross-bridges work asynchronously

**CHANGES DURING CONTRACTION:**

**I-band:** Shortens (actin slides over myosin)

**A-band:** Length remains constant

**H-zone:** Shortens or disappears

**Sarcomere:** Overall length decreases

**Z-lines:** Move closer together

**RELAXATION:**

$\text{Ca}^{2+}$  pumped back into sarcoplasmic reticulum

Tropomyosin covers myosin binding sites

Cross-bridges cannot form

Muscle returns to resting length

**ENERGY REQUIREMENTS:**

**ATP for contraction:** Cross-bridge cycling,  $\text{Ca}^{2+}$  release

**ATP for relaxation:**  $\text{Ca}^{2+}$  pump operation, cross-bridge breaking

**4. How does a muscle shorten during its contraction and return to its original form during relaxation?**

**Enhanced Solution:**

**MUSCLE CONTRACTION MECHANISM:**

**Phase 1: Neural Activation**

**Signal initiation:** Motor neuron fires action potential

**Neurotransmitter release:** Acetylcholine released at neuromuscular junction

**Membrane depolarization:** Action potential spreads across sarcolemma

**T-tubule conduction:** Signal travels deep into muscle fiber

**Phase 2: Excitation-Contraction Coupling**

**Sarcoplasmic reticulum activation:** T-tubule signal triggers  $\text{Ca}^{2+}$  release

**Calcium flood:**  $\text{Ca}^{2+}$  concentration rises from  $10^{-7}\text{M}$  to  $10^{-5}\text{M}$

**Troponin binding:**  $\text{Ca}^{2+}$  binds to troponin C subunit

**Conformational change:** Troponin complex shifts tropomyosin position

**Phase 3: Cross-Bridge Cycle Initiation**

**Site exposure:** Myosin binding sites on actin become available

**High-energy state:** Myosin heads in cocked position with ADP + Pi

**Initial binding:** Myosin heads attach to actin (weak binding)

**Strong binding:** Release of Pi strengthens cross-bridge

**Phase 4: Power Stroke and Shortening**

**Power stroke:** Myosin heads pivot  $45^\circ$ , pulling actin filaments

**Force generation:** Each myosin head generates  $\sim 5$  piconewtons of force

**ADP release:** Completes the power stroke

**Filament sliding:** Actin slides toward sarcomere center

**Phase 5: ATP Binding and Detachment**

**ATP binding:** New ATP molecule binds to myosin head

**Conformational change:** Myosin head releases from actin

**ATP hydrolysis:**  $\text{ATP} \rightarrow \text{ADP} + \text{Pi}$ , providing energy for re-cocking

**Cycle repetition:** Process continues while  $\text{Ca}^{2+}$  and ATP are present

**MUSCLE SHORTENING CHARACTERISTICS:**

**I-band reduction:** Decreases as actin slides over myosin

**A-band constancy:** Myosin length unchanged

**H-zone narrowing:** Actin filaments overlap more

**Overall shortening:** Sum of all sarcomere contractions

**MUSCLE RELAXATION MECHANISM:**

**Phase 1: Neural Cessation**

**Signal termination:** Motor neuron stops firing

**Acetylcholine breakdown:** Acetylcholinesterase degrades neurotransmitter

**Membrane repolarization:** Sarcolemma returns to resting potential

**Phase 2: Calcium Removal**

**Active transport:**  $\text{Ca}^{2+}$ -ATPase pumps  $\text{Ca}^{2+}$  back into sarcoplasmic reticulum

**Energy requirement:** 1 ATP per 2  $\text{Ca}^{2+}$  ions transported

**Concentration drop:**  $\text{Ca}^{2+}$  levels fall below threshold for troponin binding

**Troponin release:**  $\text{Ca}^{2+}$  dissociates from troponin C

**Phase 3: Regulatory Protein Reset**

**Tropomyosin repositioning:** Returns to blocking position over binding sites

**Myosin binding prevention:** Active sites on actin become covered

**Cross-bridge inhibition:** New cross-bridges cannot form

**Phase 4: Passive Lengthening**

**Elastic recoil:** Elastic elements (titin, connective tissue) restore length

**External forces:** Gravity, antagonist muscles, or external stretch

**Sarcomere restoration:** Returns to original resting length

**Energy independence:** Lengthening requires no ATP

**ENERGY REQUIREMENTS:**

**During contraction:**

**Cross-bridge cycling:** ~1 ATP per cycle per myosin head

**Calcium release:** Minimal ATP for release mechanism

**High demand:** Rapid ATP consumption during intense activity

**During relaxation:**

**Calcium reuptake:** Major ATP consumer during relaxation

**Maintenance metabolism:** Basal ATP for cellular functions

**Recovery processes:** ATP for restoring ion gradients

**REGULATION AND CONTROL:**

**All-or-nothing:** Individual muscle fibers contract maximally

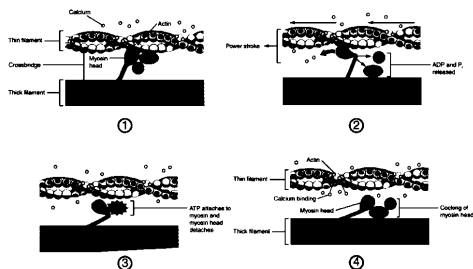
**Graded response:** Recruitment of motor units provides force gradation

**Frequency modulation:** Higher stimulation frequency increases force

**Fatigue factors:** ATP depletion, lactic acid accumulation limit duration

**5. Discuss the role of  $\text{Ca}^{2+}$  ions in muscle contraction. Draw neat sketches to illustrate your answer.**

**Enhanced Solution:**



**ROLE OF  $\text{Ca}^{2+}$  IONS IN MUSCLE CONTRACTION:**

## **CALCIUM STORAGE AND RELEASE:**

### **1. Sarcoplasmic Reticulum (SR):**

**Structure:** Specialized endoplasmic reticulum surrounding myofibrils

**Function:** Primary  $\text{Ca}^{2+}$  storage organelle in muscle cells

**Concentration:** Maintains  $10^{-3}$  M  $\text{Ca}^{2+}$  inside SR vs  $10^{-7}$  M in sarcoplasm

**Release mechanism:** Voltage-gated  $\text{Ca}^{2+}$  channels (ryanodine receptors)

### **2. T-tubule System:**

**Structure:** Invaginations of sarcolemma penetrating muscle fiber

**Function:** Conducts action potential deep into fiber

**Coupling:** Connected to SR through dihydropyridine receptors

**Synchronization:** Ensures simultaneous  $\text{Ca}^{2+}$  release throughout fiber

## **MOLECULAR MECHANISM OF CALCIUM ACTION:**

### **Step 1: Calcium Release**

**Action potential propagation:** Signal travels along T-tubules

**Conformational coupling:** Dihydropyridine receptors activate ryanodine receptors

**Massive release:**  $\text{Ca}^{2+}$  floods from SR into sarcoplasm

**Concentration rise:** From  $10^{-7}$  M to  $10^{-5}$  M (100-fold increase)

### **Step 2: Troponin Binding**

**Troponin C:** Contains high-affinity  $\text{Ca}^{2+}$  binding sites

**Binding sites:** 4  $\text{Ca}^{2+}$  binding sites per troponin molecule

**Cooperativity:**  $\text{Ca}^{2+}$  binding increases affinity for subsequent  $\text{Ca}^{2+}$

**Conformational change:**  $\text{Ca}^{2+}$ -troponin complex alters shape

### **Step 3: Tropomyosin Movement**

**Regulatory position:** Tropomyosin normally blocks myosin binding sites

**$\text{Ca}^{2+}$ -induced shift:** Troponin complex moves tropomyosin

**Site exposure:** Myosin binding sites on actin become accessible

**Cooperative units:** Each tropomyosin regulates 7 actin monomers

### **Step 4: Cross-Bridge Formation**

**Myosin binding:** Strong cross-bridges form with exposed actin sites

**ATPase activation:**  $\text{Ca}^{2+}$  enhances myosin ATPase activity

**Force generation:** Cross-bridge cycling produces contraction force

**Sustained contraction:** Continues while  $\text{Ca}^{2+}$  remains elevated

## **CALCIUM REMOVAL AND RELAXATION:**

### **1. Active Transport:**

**SERCA pumps:** Sarco-endoplasmic reticulum  $\text{Ca}^{2+}$ -ATPase

**Energy requirement:** 1 ATP per 2  $\text{Ca}^{2+}$  ions transported

**Rate:** Can lower  $\text{Ca}^{2+}$  concentration within milliseconds

**Regulation:** Controlled by phospholamban

### **2. Calcium Buffering:**

**Calsequestrin:**  $\text{Ca}^{2+}$  binding protein inside SR

**Parvalbumin:** Cytoplasmic  $\text{Ca}^{2+}$  buffer (in fast-twitch fibers)

**Calmodulin:** Multifunctional  $\text{Ca}^{2+}$  binding protein

**Function:** Prevents excessive free  $\text{Ca}^{2+}$  levels

**CALCIUM SENSITIVITY AND REGULATION:**

**Factors affecting  $\text{Ca}^{2+}$  sensitivity:**

**pH:** Acidosis decreases  $\text{Ca}^{2+}$  sensitivity

**$\text{Mg}^{2+}$ :** Competes with  $\text{Ca}^{2+}$  for binding sites

**Phosphorylation:** Troponin I phosphorylation decreases  $\text{Ca}^{2+}$  sensitivity

**Temperature:** Higher temperature increases sensitivity

**Disease states:**

**Malignant hyperthermia:** Uncontrolled  $\text{Ca}^{2+}$  release

**Muscular dystrophy:** Altered  $\text{Ca}^{2+}$  handling

**Heart failure:** Impaired  $\text{Ca}^{2+}$  cycling

**Tetany:** Low extracellular  $\text{Ca}^{2+}$  affects excitability

**QUANTITATIVE ASPECTS:**

**Calcium dynamics:**

**Resting  $[\text{Ca}^{2+}]$ :** ~100 nM

**Peak  $[\text{Ca}^{2+}]$ :** ~10  $\mu\text{M}$  during contraction

**Release rate:** 0.1-1  $\mu\text{mol/L}$  per millisecond

**Reuptake rate:** 90% removed within 100 ms

**Stoichiometry:**

**Binding ratio:** 4  $\text{Ca}^{2+}$  per troponin complex

**Regulatory units:** 1 troponin per 7 actin monomers

**Cross-bridges:** 1 myosin head per actin monomer (when fully activated)

**6. Differentiate between Pectoral and Pelvic girdle.**

**Enhanced Solution:**

**COMPREHENSIVE COMPARISON OF PECTORAL AND PELVIC GIRDLES:**

Feature	PECTORAL GIRDLE	PELVIC GIRDLE
<b>LOCATION</b>	Shoulder region	Hip region
<b>BONE COMPOSITION</b>	2 bones: Clavicle and Scapula	3 fused bones: Ilium, Ischium, and Pubis
<b>ARTICULATION CAVITY</b>	Glenoid cavity (shallow)	Acetabulum (deep, cup-shaped)
<b>JOINT WITH LIMB</b>	Ball and socket joint with humerus head	Ball and socket joint with femur head
<b>PRIMARY FUNCTION</b>	Forelimb bone articulation	Hindlimb bone articulation

**DETAILED STRUCTURAL DIFFERENCES:**

**PECTORAL GIRDLE:**

**Anatomical features:**

**Clavicle (Collar bone):**

- S-shaped bone
- Articulates with sternum medially
- Provides horizontal support
- Prevents medial displacement of shoulder

**Scapula (Shoulder blade):**

- Triangular flat bone
- Contains glenoid cavity
- Has acromion process and spine
- Highly mobile, not directly attached to axial skeleton

**Functional characteristics:**

**Mobility priority:** Designed for maximum range of motion

**Stability sacrifice:** Less stable due to shallow socket

**Weight bearing:** Not designed for weight bearing

**Range of motion:** 360° rotation possible

**Muscle attachments:** 17 muscles attach to scapula

**PELVIC GIRDLE:**

**Anatomical features:**

**Ilium:**

- Largest, upper portion
- Contains iliac crest and fossa
- Articulates with sacrum posteriorly

**Ischium:**

- Lower, posterior portion
- Forms sitting bone (ischial tuberosity)
- Bears body weight when sitting

**Pubis:**

- Lower, anterior portion
- Forms symphysis pubis joint
- Completes pelvic ring anteriorly

**Functional characteristics:**

**Stability priority:** Designed for maximum stability

**Weight bearing:** Primary weight-bearing structure

**Mobility limitation:** Restricted range of motion

**Deep socket:** Acetabulum covers 40% of femoral head

**Pelvic cavity:** Forms boundaries of pelvic cavity

**COMPARATIVE BIOMECHANICS:**

**Movement patterns:**

**Pectoral:** Multi-directional, reaching, throwing motions

**Pelvic:** Weight-bearing, locomotion, postural support

**Joint stability:**

**Shoulder joint:** Relies on muscles and ligaments for stability

**Hip joint:** Inherently stable due to deep socket and strong ligaments

**Evolutionary adaptation:**

**Pectoral:** Evolved for manipulation and tool use

**Pelvic:** Adapted for bipedal locomotion and childbirth

**CLINICAL SIGNIFICANCE:**

**Common injuries:**

**Shoulder:** Dislocations, rotator cuff tears, impingement

**Hip:** Fractures (especially in elderly), arthritis, labral tears

**Gender differences:**

**Pectoral:** Minimal sexual dimorphism

**Pelvic:** Significant differences for childbirth adaptations

**Age-related changes:**

**Both:** Susceptible to arthritis and degenerative changes

**Hip:** Weight-bearing stress leads to more wear

**Shoulder:** Overuse injuries more common

**FUNCTIONAL INTEGRATION:**

**Kinetic chain relationships:**

**Upper extremity:** Pectoral girdle → arm → forearm → hand

**Lower extremity:** Pelvic girdle → thigh → leg → foot

**Movement coordination:**

**Pectoral:** Works with cervical and thoracic spine

**Pelvic:** Integrates with lumbar spine and core muscles

**Postural significance:**

**Pectoral:** Affects head and neck posture

**Pelvic:** Determines overall body alignment and balance