

ORAL MEDICINE AND
RADIOLOGY- CRRI WORK
DONE
13.04.2020-18.04.2020

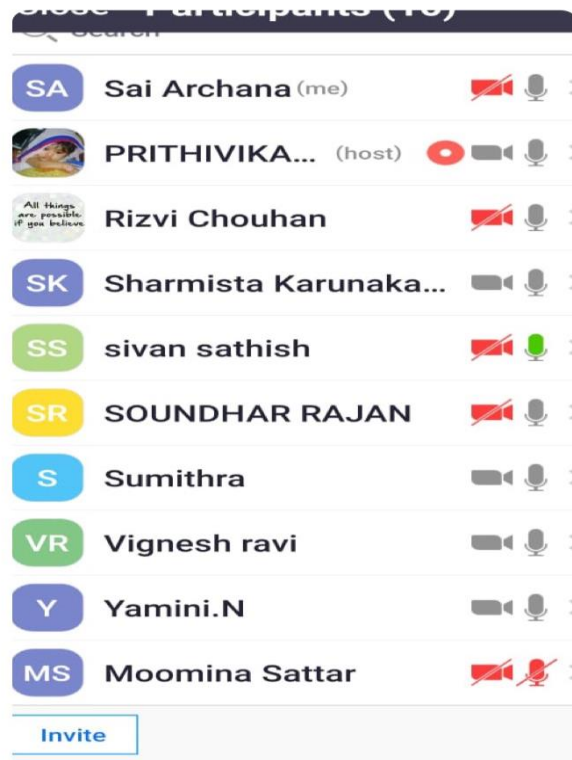
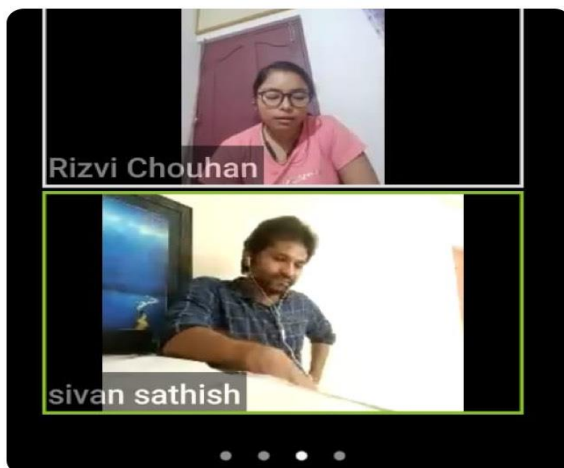
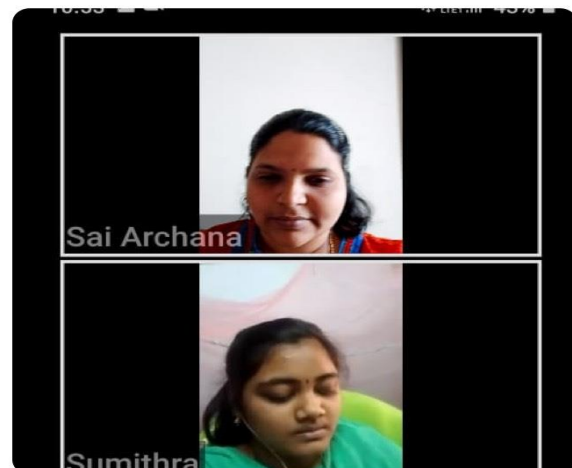
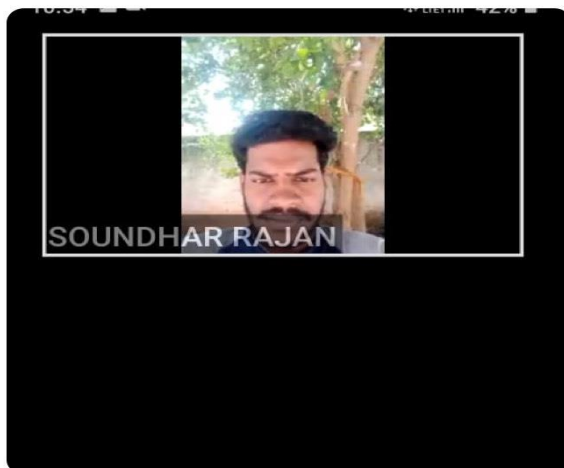
DEPARTMENT OF ORAL MEDICINE AND RADIOLOGY
E-CLASSES FOR CRI BDS

13.4.2020:

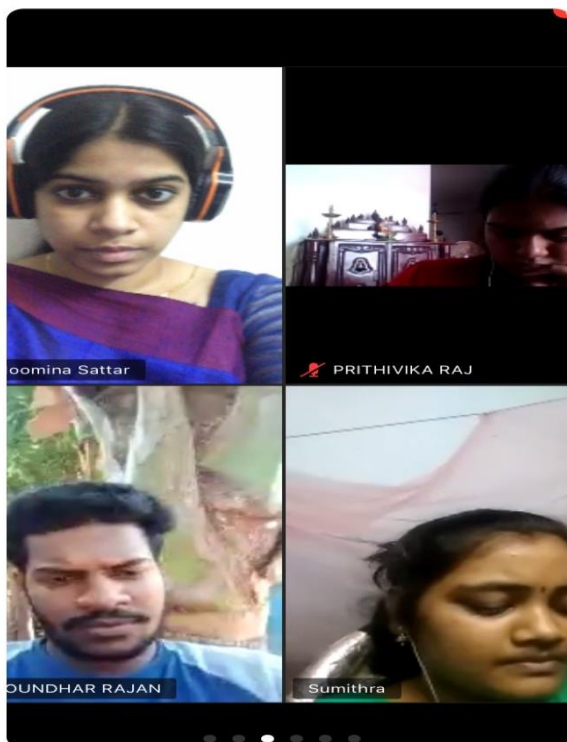
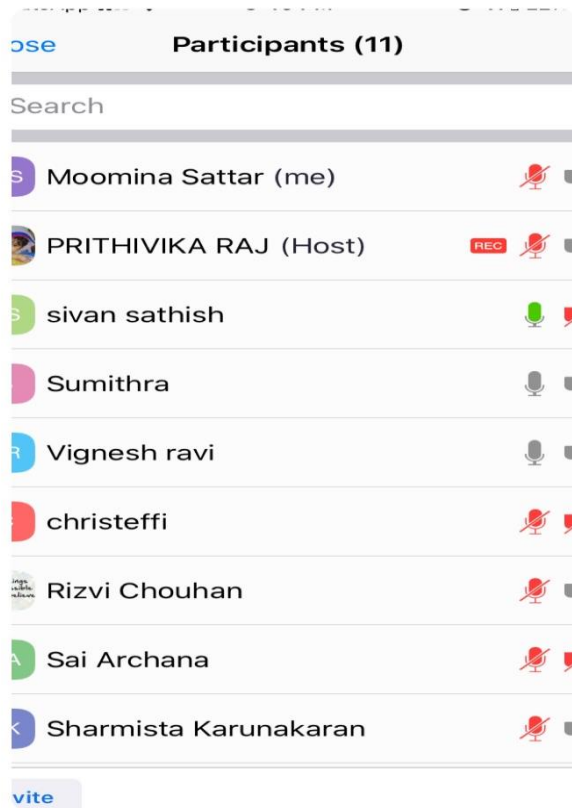
- **NAME OF STAFFS PARTICIPATED: 4**
 1. Dr. SIVAN SATHISH
 2. Dr. CHRISTEFFI MABEL
 3. Dr. SAI ARCHANA
 4. Dr. MOOMINA
- **STUDENTS PARTICIPATED:**
 1. Raj Prithvika
 2. Rizvi Chauhan
 3. Sumithra
 4. Sharmista
 5. Soundhar Rajan
 6. Yamini
- **WORK DONE:**
 - *Discussion on Basic Sciences*
 - *Discussion with PGs on Calcium Homeostasis*
 - *Combined clinical classes with final years.*
 - *NEET Questions discussion with final years*

PICTURES:





AFTERNOON DISCUSSION:



15.4.2020:

- **NAME OF STAFFS PARTICIPATED: 4**

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2. Dr. CHRISTEFFI MABEL
3. Dr. SAI ARCHANA
4. Dr. MOOMINA

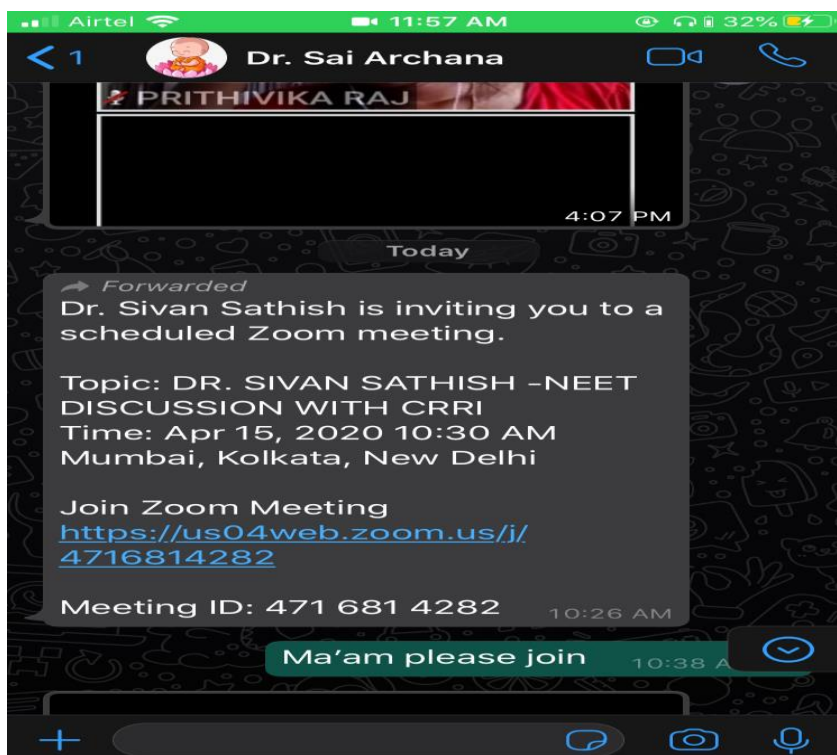
- **STUDENTS PARTICIPATED:**

1. Raj Prithvika
2. Rizvi Chauhan
3. Sumithra
4. Sharmista
5. Soundhar Rajan
6. Yamini

- **WORK DONE:**

- *Dicussion on Basic Sciences*
- *Discussion with PGs on Neurological diseases and dental significance*
- *Combined clinical classes with final years.*
- *NEET Questions discussion with final years.*

PICTURES:



10:42

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104 CHAPTER 4 Hemodynamic Disorders, Thromboembolism, and Shock

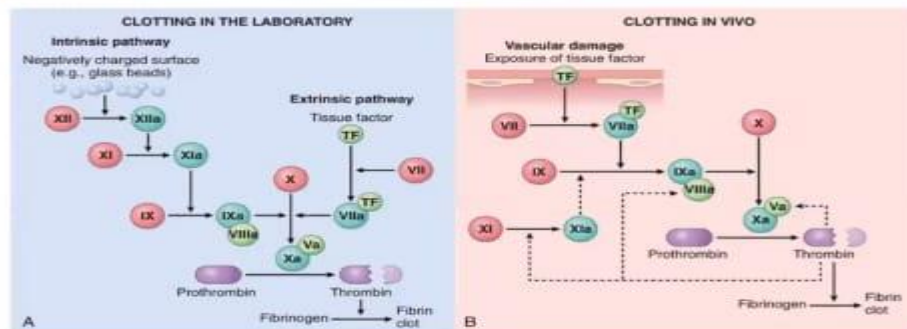


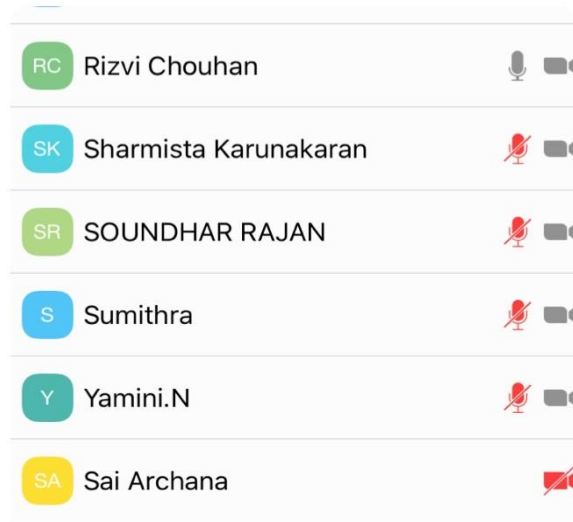
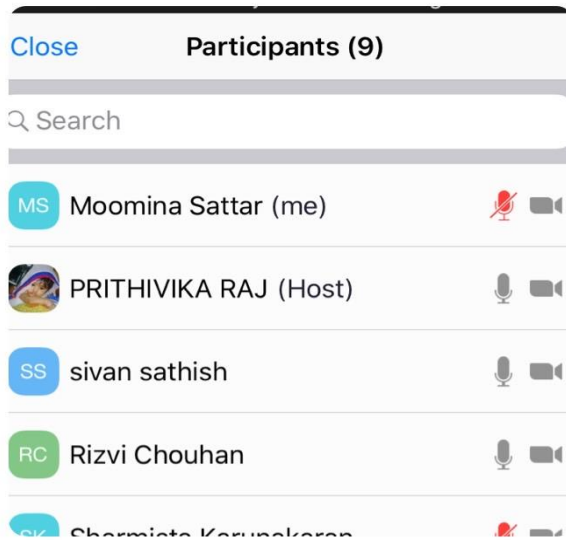
Fig. 4.7 The coagulation cascade in the laboratory and in vivo. (A) Clotting is initiated in the laboratory by adding phospholipids, calcium, and either a negatively charged substance such as glass beads (intrinsic pathway) or a source of tissue factor (extrinsic pathway). (B) In vivo, tissue factor is the major initiator of coagulation, which is amplified by feedback loops involving thrombin (dotted lines). The red polypeptides are inactive factors, the dark green polypeptides are active factors, whereas the light green polypeptides correspond to cofactors.

Based on assays performed in clinical laboratories, the coagulation cascade has traditionally been divided into the *extrinsic and intrinsic pathways* (Figs. 4-7A).

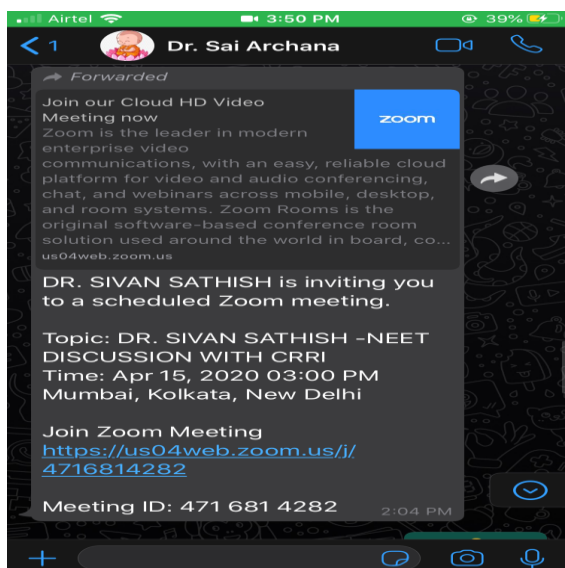
- The *partial thromboplastin time (PTT)* assay screens the function of the proteins in the intrinsic pathway (factors XII, XI, IX, X, VIII, V, II, and fibrinogen). In this assay,

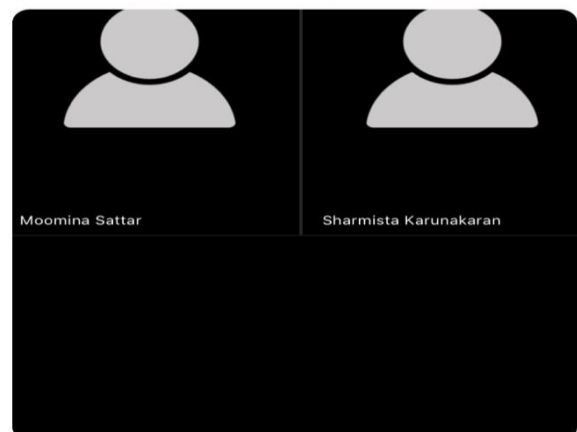
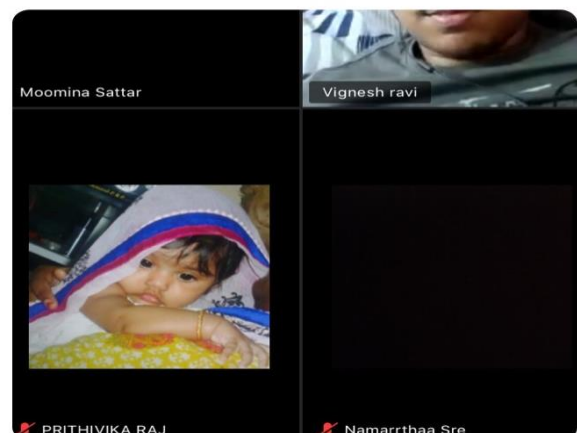
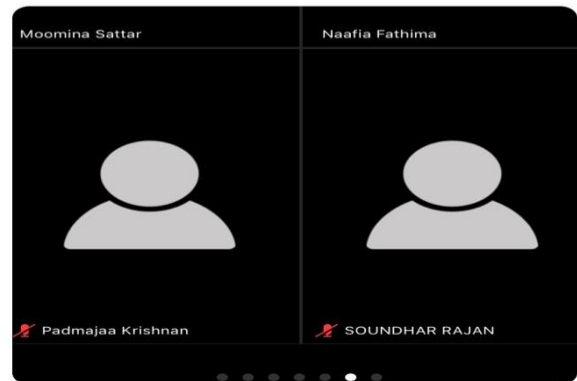
Sharmista Karunakaran's screen





AFTERNOON SESSION:





Participants (14)

Moomina Sattar (me)

Narrthaa Sre (Host)

vi Chouhan

an sathish

afia Fathima

armista Karunakaran

nesh ravi

na Naomi

rthika Kumaresan

Close


Participants (

NF Naafia Fathima


SK Sharmista Karunakarar

VR Vignesh ravi

n Neha Naomi

 Nehrthika Kumaresan

PK Padmajaa Krishnan

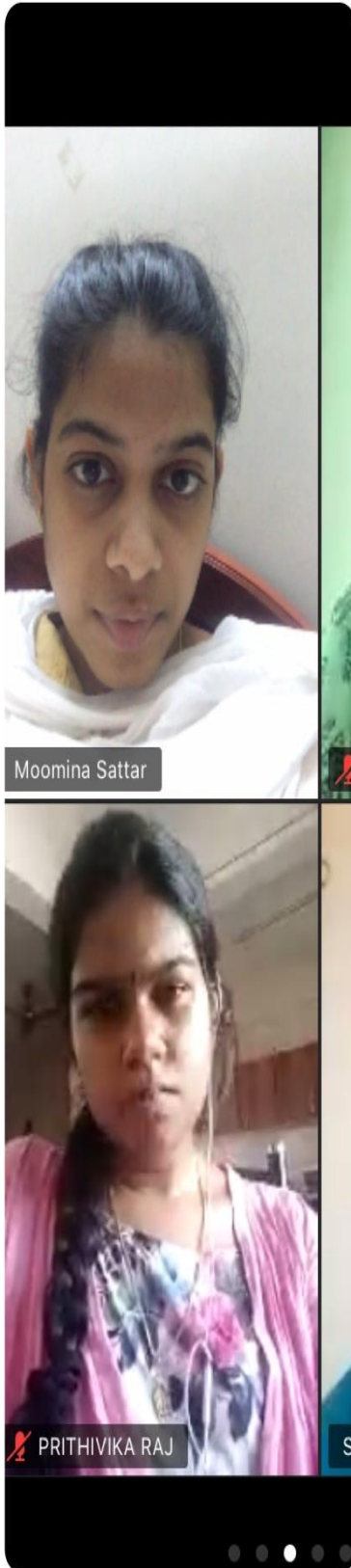
 PRITHIVIKA RAJ

SR SOUNDHAR RAJAN

S Sumithra

Y Yamini.N

Invite



16.4.2020:

- **NAME OF STAFFS PARTICIPATED: 4**

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2. Dr. CHRISTEFFI MABEL
3. Dr. SAI ARCHANA
4. Dr. MOOMINA

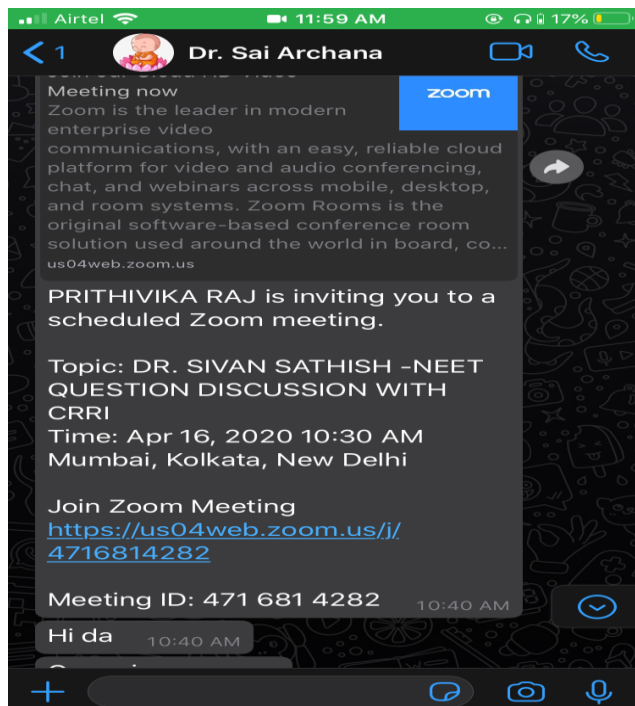
- **STUDENTS PARTICIPATED:**

1. Raj Prithvika
2. Rizvi Chauhan
3. Sumithra
4. Sharmista
5. Soundhar Rajan
6. Yamini


- **WORK DONE:**

- *Dicussion on Basic Sciences*
- *Discussion with PGs on PHYSIOLOGY OF TASTE*
- *Combined clinical classes with final years*
- *NEET Questions discussion with final years*


PICTURES:



rose Participants (17)





nawin Subhaganesh




n

Neha Naomi







Nehrthika Kumaresan




PK

Padmajaa Krishnan






PRITHIVIKA RAJ




All things
re possible
you believe

Rizvi Chouhan




SK

Sharmista Karunaka...




SR

SOUNDHAR RAJAN




S

Sumithra



Y

Yamini.N



Invite



88 SECTION I Cellular and Molecular Basis for Medical Physiology

CLINICAL BOX 4-1

Demyelinating Diseases

Normal conduction of action potentials relies on the insulating properties of **myelin**. Thus, defects in myelin can have major adverse neurologic consequences. One example is **multiple sclerosis (MS)**, an autoimmune disease that affects over 3 million people worldwide, usually striking between the ages of 20 and 50 and affecting women about twice as often as men. The cause of MS appears to include both genetic and environmental factors. It is most common among whites living in countries with temperate climates including Europe, southern Canada, northern United States, and southeastern Australia. Environmental triggers include early exposure to viruses such as Epstein-Barr virus and those that cause measles, herpes, chickenpox, or influenza. In MS, antibodies and white blood cells in the immune system attack myelin, causing inflammation and injury to the sheath and eventually the nerves that it surrounds. Loss of myelin leads to leakage of K^+ through voltage-gated channels, hyperpolarization, and failure to conduct action potentials. Initial presentation commonly includes reports of **paraparesis** (weakness in lower extremities) that may be accompanied by mild spasticity and hyperreflexia; **paresthesia**; numbness; urinary incontinence; and heat intolerance. Clinical assessment often reports **optic neuritis**,

characterized by blurred vision, a change in color perception, visual field defect (**central scotoma**), and pain with eye movements; **dysarthria**; and **dysphagia**. Symptoms are often exacerbated by increased body temperature or ambient temperature. Progression of the disease is quite variable. In the most common form called **relapsing-remitting MS**, transient episodes appear suddenly, last a few weeks or months, and then gradually disappear. Subsequent episodes can appear years later, and eventually full recovery does not occur. A steadily worsening course with only minor periods of remission (**secondary-progressive MS**) develops later in many individuals. Others have a progressive form of the disease in which there are no periods of remission (**primary-progressive MS**). Diagnosing MS is very difficult and generally is delayed until multiple episodes occur with deficits separated in time and space. **Nerve conduction tests** can detect slowed conduction in motor and sensory pathways. Cerebral spinal fluid analysis can detect the presence of **oligoclonal bands** indicative of an abnormal immune reaction against myelin. The most definitive assessment is **magnetic resonance imaging (MRI)** to visualize multiple scarred (sclerotic) areas or plaques in the brain. These plaques often appear in the periventricular regions of the cerebral hemispheres.

THERAPEUTIC HIGHLIGHTS

Although there is no cure for MS, **corticosteroids** (eg, **prednisone**) are the most common treatment used to reduce the inflammation that is accentuated during a relapse. Some drug treatments are designed to modify the course of the disease. For example, daily injections of **β -interferons** suppress the immune response to reduce the severity and slow the progression of the disease. **Glatiramer acetate** may block the immune system's attack on the myelin. **Natalizumab** interferes with the ability of potentially damaging immune cells to move from the

bloodstream to the CNS. A clinical trial using B cell-depleting therapy with **rituximab**, an anti-CD20 monoclonal antibody, showed that the progression of the disease was slowed in patients younger than 51 years in whom the primary-progressive form of MS was diagnosed. Another clinical trial has shown that oral administration of **fingolimod** slowed the progression of the relapsing-remitting form of MS. This immunosuppressive drug acts by sequestering lymphocytes in the lymph nodes, thereby limiting their access to the CNS.

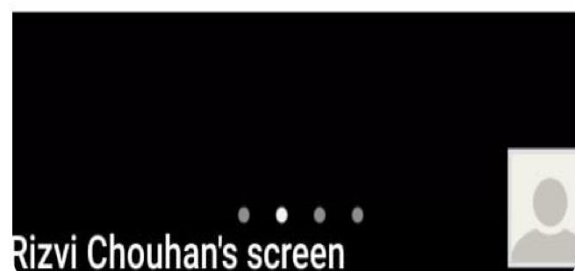
or attached to the side of the axon (eg, cutaneous neurons). Its location makes no difference as far as the receptor function of the dendritic zone and the transmission function of the axon are concerned.

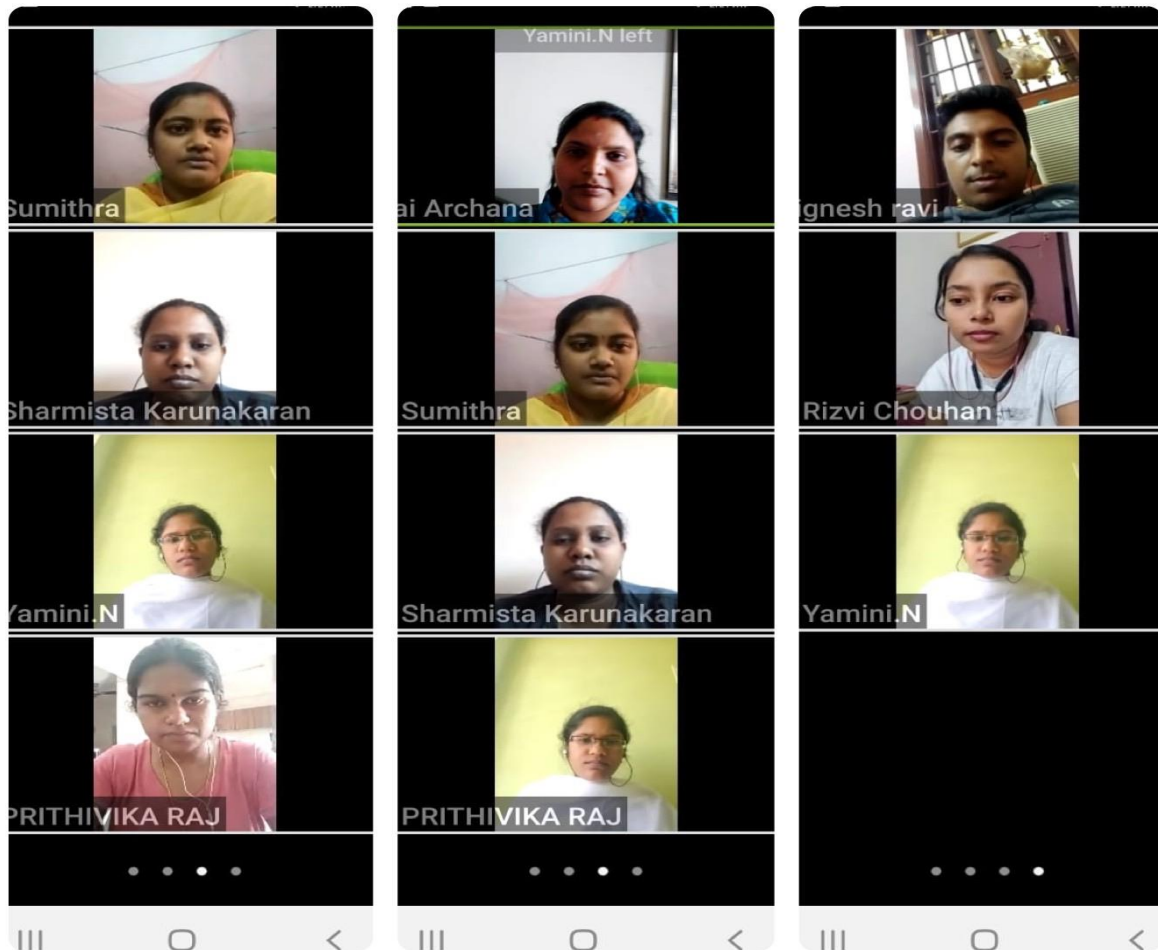
The axons of many neurons are myelinated, that is, they acquire a sheath of **myelin**, a protein-lipid complex that is wrapped around the axon (Figure 4-18). In the peripheral nervous system, myelin forms when a Schwann cell wraps its membrane around an axon up to 100 times. The myelin is then compacted when the extracellular portions of a membrane protein called protein zero (P_0) lock to the extracellular portions of P_0 in the apposing membrane. Various mutations

in the gene for P_0 cause peripheral neuropathies, 29 different mutations have been described that cause symptoms ranging from mild to severe. The myelin sheath envelops the axon except at its ending and at the **nodes of Ranvier**, periodic 1- μ m constrictions that are about 1 mm apart (Figure 4-2). The insulating function of myelin is discussed later in this chapter. Not all neurons are myelinated; some are **unmyelinated**, that is, simply surrounded by Schwann cells without the wrapping of the Schwann cell membrane that produces myelin around the axon.

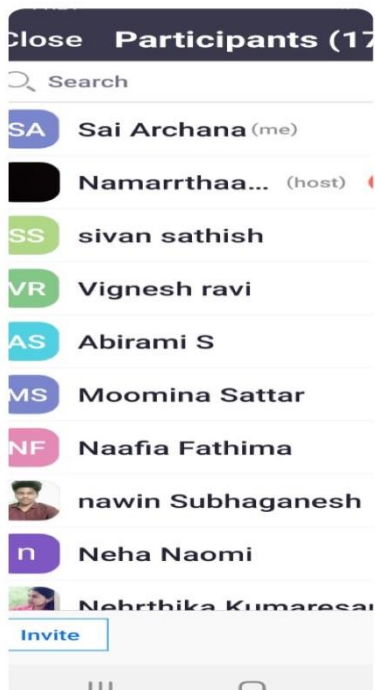
In the CNS of mammals, most neurons are myelinated, but the cells that form the myelin are **oligodendrocytes**

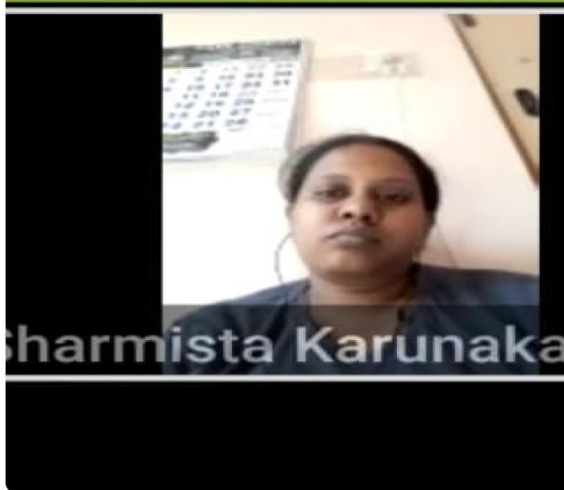
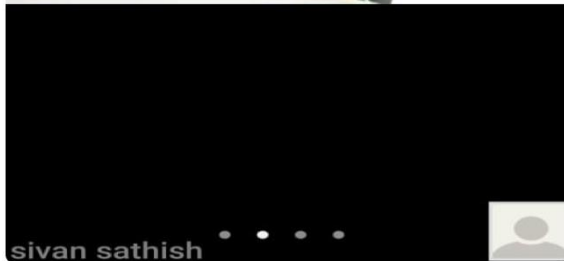
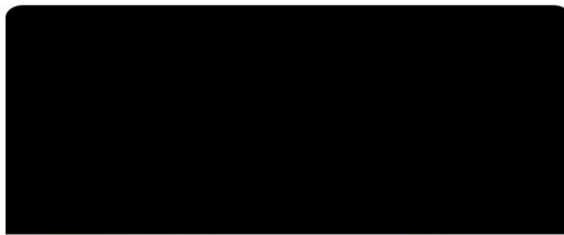
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AFTERNOON SESSION:





Excitable Tissue: Nerve

OBJECTIVES

after studying this chapter, you should be able to:

- Name the various types of glia and their functions.
- Name the parts of a neuron and their functions.
- Describe the chemical nature of myelin, and summarize the differences in the ways in which unmyelinated and myelinated neurons conduct impulses.
- Describe orthograde and retrograde axonal transport.
- Describe the changes in ionic channels that underlie the action potential.
- List the various nerve fiber types found in the mammalian nervous system.
- Describe the function of neurotrophins.

INTRODUCTION

The human central nervous system (CNS) contains about 10^{11} (100 billion) neurons. It also contains 2–10 times this number of **glial cells**. The CNS is a complex organ; it has been calculated that 40% of the human genes participate, at least to a degree, in its formation. The neurons, the basic building blocks of the nervous system, have evolved from primitive neuroeffector cells that respond to various stimuli by contracting. In more complex animals, contraction has become the specialized function of muscle cells, whereas integration and transmission of nerve impulses are the functions of neurons.

functional unit that is required for normal brain function, including synaptic activity, extracellular fluid homeostasis, energy metabolism, and neural protection. Disturbances in the interaction of these elements are the pathophysiological basis for many neurologic disorders (e.g., cerebral ischemia, seizures, neurodegenerative diseases, and cerebral edema). This chapter describes the cellular components of the CNS and the excitability of neurons, which involves the generation of action potentials that enable neurons to integrate and transmit information.

Your microphone is unmuted

CELLULAR ELEMENTS IN THE CNS

GLIAL CELLS

For many years following their discovery, glial cells (or glia) were viewed as CNS connective tissue. **Axonal sprouting** occurs from the proximal stump, growing toward the nerve ending. This results from **growth-promoting factors** secreted by Schwann cells that attract axons toward the distal stump. Adhesion molecules of the immunoglobulin superfamily (e.g., the neuron-glia cell adhesion molecule or Ng2/CD110) promote axon growth along cell membranes and extracellular matrices. Inhibitory molecules in the perineurium ensure that the regenerating axons grow in a correct trajectory. Denervated distal stumps are able to upregulate production of **neurotrophins** that promote growth. Once the regenerated axon reaches its target, a new functional connection (e.g., neuromuscular junction) is formed, allowing for considerable, although not full, recovery. For example, fine motor control may be permanently

There are two major types of glial cells in the vertebrate nervous system: **microglia** and **macroglia**. Microglia are immune system cells; they are scavenger cells that resemble tissue macrophages and remove debris resulting from injury, infection, and disease (e.g., multiple sclerosis [MS], AIDS-related dementia, Parkinson disease, and Alzheimer disease). Macroglia arise from macrophages outside of the nervous system and are physiologically and embryologically unrelated to other neural cell types.

There are three types of macroglia: oligodendrocytes, Schwann cells, and astrocytes (Figure 4-1). Oligodendrocytes

85



Rizvi Chouhan's screen

CLINICAL BOX 4-3

Axonal Regeneration

Peripheral nerve damage is often reversible. Although the axon will degenerate distal to the damage, connective elements of the so-called **distal stump** often survive. **Axonal sprouting** occurs from the proximal stump, growing toward the nerve ending. This results from **growth-promoting factors** secreted by Schwann cells that attract axons toward the distal stump. Adhesion molecules of the immunoglobulin superfamily (e.g., the neuron-glia cell adhesion molecule or Ng2/CD110) promote axon growth along cell membranes and extracellular matrices. Inhibitory molecules in the perineurium ensure that the regenerating axons grow in a correct trajectory. Denervated distal stumps are able to upregulate production of **neurotrophins** that promote growth. Once the regenerated axon reaches its target, a new functional connection (e.g., neuromuscular junction) is formed, allowing for considerable, although not full, recovery. For example, fine motor control may be permanently

impaired because some motor neurons are guided to an inappropriate motor fiber. Nonetheless, recovery of peripheral nerves from damage far surpasses that of central nerve pathways. The proximal stump of a damaged axon in the CNS will form short sprouts, but distant stump recovery is rare, and the damaged axons are unlikely to form new synapses. This is in part because CNS neurons do not have the growth-promoting chemicals needed for regeneration. In fact, CNS myelin is a potent inhibitor of axonal growth. In addition, after a CNS injury, **astrocytic proliferation**, **activation of microglia**, **scar formation**, **inflammation**, and **invasion of immune cells** create an inappropriate environment for regeneration. Thus, treatment of brain and spinal cord injuries focuses on rehabilitation rather than reversing the nerve damage. New research is aiming to identify ways to initiate and maintain axonal growth, to direct regenerating axons to reconnect with their target neurons, and to reconstitute original neuronal circuitry.

THERAPEUTIC HIGHLIGHTS

There is evidence showing that the use of **nonsteroidal anti-inflammatory drugs (NSAIDs)** such as ibuprofen can overcome the factors that inhibit axonal growth following injury. This effect is thought to be mediated by the ability of NSAIDs to inhibit **IKK α** , a small GTPase protein that normally prevents repair of neural pathways and axons. Growth cone collapse in response to myelin-associated

inhibition after nerve injury is prevented by drugs (such as **perlestin kinase**) that interfere with signal transduction via **tyrosine**, **G-protein**. Experimental drugs that inhibit the **phosphoinositide 3-kinase (PI3K) pathway** or the **inositol triphosphate (IP3) receptor** have also been shown to promote regeneration after nerve injury.

- Nerve fibers are divided into different categories (A, B, and C) based on axonal diameter, conduction velocity, and function. A numerical classification (Ia, Ib, II, III, and IV) is also used for sensory afferent fibers.
- Neurotrophins such as NGF are carried by retrograde transport to the neuronal cell body, where they foster the production of proteins associated with neuronal development, growth, and survival and suppress neuronal apoptosis.

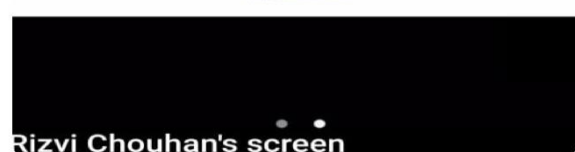
MULTIPLE-CHOICE QUESTIONS

For all questions, select the single best answer unless otherwise directed.

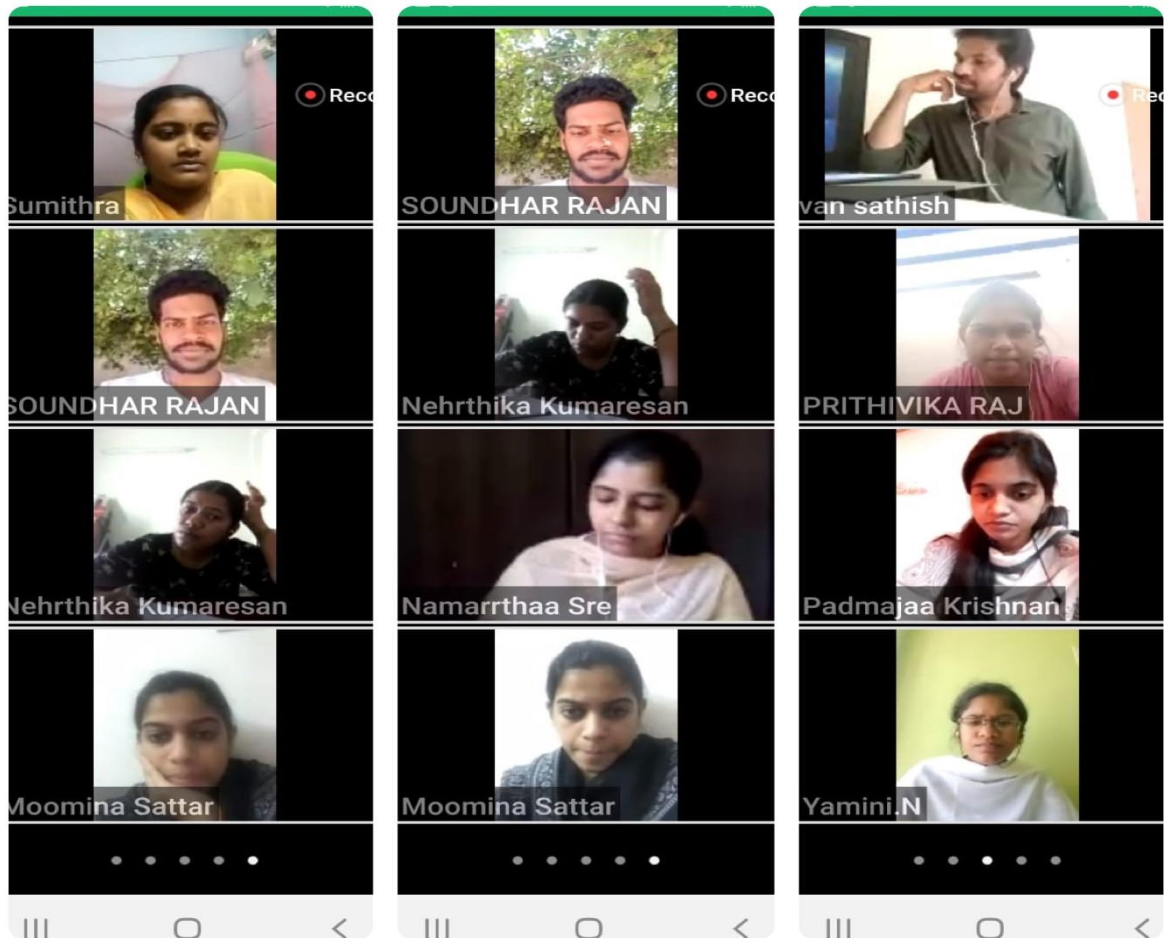
1. Which of the following statements about glia is true?
 - A. Microglia arise from macrophages outside of the nervous system and are physiologically and embryologically similar to other neural cell types.
 - B. Glia do not undergo proliferation.
 - C. Perineuronal astrocytes produce substances that are toxic to neurons to help maintain the appropriate concentration

- of ions and neurotransmitters by taking up K^+ and the neurotransmitters glutamate and GABA.
- D. Oligodendrocytes and Schwann cells are involved in myelin formation around axons in the peripheral and central nervous systems, respectively.
- E. Macroglia are scavenger cells that resemble tissue macrophages and remove debris resulting from injury, infection, and disease.
2. Primary erythromyalgia, which may be due to a peripheral nerve sodium channelopathy, was diagnosed in a 13-year-old girl who was experiencing frequent episodes of red, painful, warm extremities. Which part of a neuron has the highest concentration of Na^+ channels per square micrometer of cell membrane?
 - A. dendrites
 - B. cell body near dendrites
 - C. initial segment
 - D. axonal membrane under myelin
 - E. node of Ranvier
3. A 45-year-old woman who works in an office had been experiencing tingling in her index and middle fingers and

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Rizvi Chouhan's screen



17.4.2020:

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4. Dr. MOOMINA

- **STUDENTS PARTICIPATED:**

1. Raj Prithvika
2. Rizvi Chauhan
3. Sumithra
4. Sharmista
5. Soundhar Rajan
6. Yamini

- **WORK DONE:**

- *Discussion on Basic Sciences*
- *Discussion with PGs on MUCOEPIDERMOID CARCINOMA*
- *Combined clinical classes with final years*
- *NEET Questions discussion with final years*

PICTURES:



Participants (11)

arch

Moomina Sattar (me)

PRITHIVIKA RAJ (Host)

christeffi

Rizvi Chouhan

sivan sathish

Sai Archana

Sharmista Karunakaran

SOUNDHAR RAJAN

Sumithra

Close Participants (10)

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PRITHIVIKA RAJ (host)

Rizvi Chouhan

C christeffi

MS Moomina Sattar

SK Sharmista Karunaka.


SR SOUNDHAR RAJAN

S Sumithra

VR Vignesh ravi

Y Yamini.N

[Invite](#)



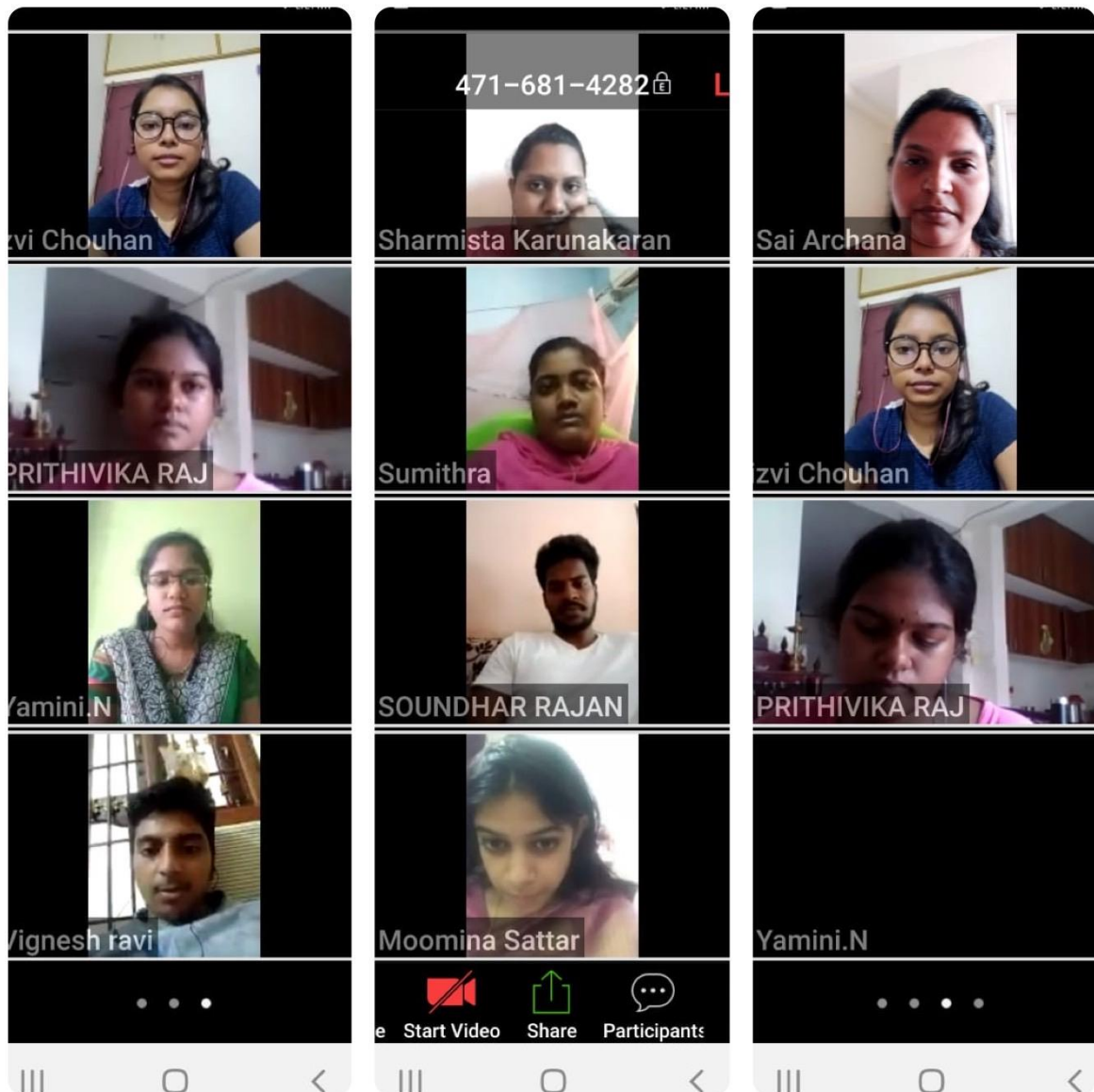
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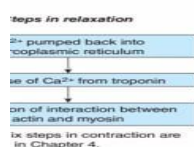
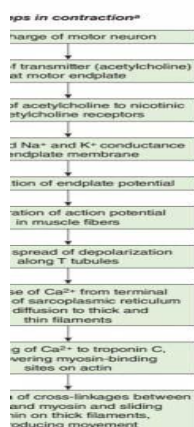
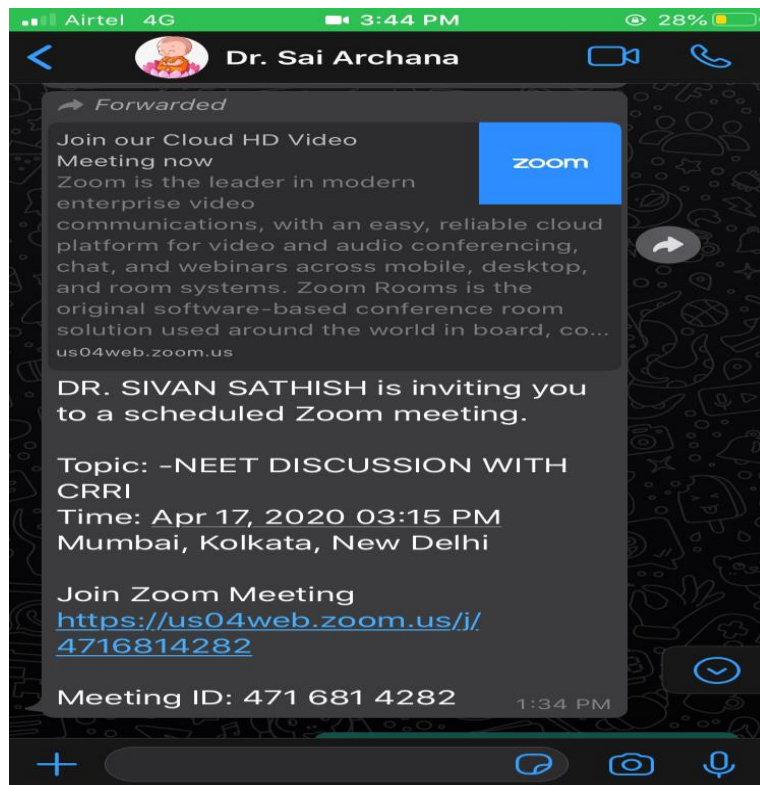
Recording

Sumithra

Moomina Sattar

sivan sathish





*Steps in contraction are in Chapter 4.

Information that leads to muscle

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channel with Ca²⁺ as its natural ligand. In skeletal muscle, Ca²⁺ entry from the extracellular fluid (ECF) by this route is not required for Ca²⁺ release. Instead, the DHPR that serves as the voltage sensor unlocks release of Ca²⁺ from the nearby sarcoplasmic reticulum via physical interaction with the RyR. The released Ca²⁺ is quickly amplified through calcium-induced calcium release. Ca²⁺ concentration is reduced in the muscle cell by the sarcoplasmic or endoplasmic reticulum Ca²⁺-ATPase (SERCA). The SERCA pump uses energy from ATP hydrolysis to remove Ca²⁺ from the cytosol back into the terminal cisterns, where it is stored until released by the next action potential. Once the Ca²⁺ concentration outside the sarcoplasmic reticulum has been lowered sufficiently, chemical interaction between myosin and actin ceases and the muscle relaxes. Note that ATP provides the energy for both contraction (at the myosin head) and relaxation (via SERCA). If transport of Ca²⁺ into the sarcoplasmic reticulum is inhibited, relaxation does not occur even though there are no more action potentials; the resulting sustained contraction is called a **contracture**. Alterations in the excitable response in muscle underscore many different pathologies (**Clinical Box 5-2**).

TYPES OF CONTRACTION

Muscular contraction involves shortening of the contractile elements, but because muscles have elastic and viscous elements in series with the contractile mechanism, it is possible for contraction to occur without an appreciable decrease in the length of the whole muscle (**Figure 5-8**). Such a contraction is called **isometric** ("same measure" or length). Contraction against a constant load with a decrease in muscle length is **isotonic** ("same tension"). Note that because work is the product of force times distance, isotonic contractions do work, whereas isometric contractions do not. In other situations, muscle can do negative work while lengthening against a constant weight.

SUMMATION OF CONTRACTIONS

The electrical response of a muscle fiber to repeated stimulation is like that of nerve. The fiber is electrically refractory only during the rising phase and part of the falling phase of the spike potential. At this time, the contraction initiated by the first stimulus is just beginning. However, because the contractile mechanism does not have a refractory period, repeated stimulation before relaxation has occurred produces additional activation of the contractile elements and a response that is added to the contraction already present. This phenomenon is known as **summation of contractions**. The tension developed during summation is considerably greater than that during the single muscle twitch. With rapidly repeated stimulation, activation of the contractile mechanism occurs repeatedly before any relaxation has occurred, and the individual responses fuse into one continuous contraction. Such a response is called a **tetanus** (**tetanic contraction**). It is a **complete tetanus** when no relaxation occurs between stimuli and

Rizvi Chouhan's screen



18.4.2020:

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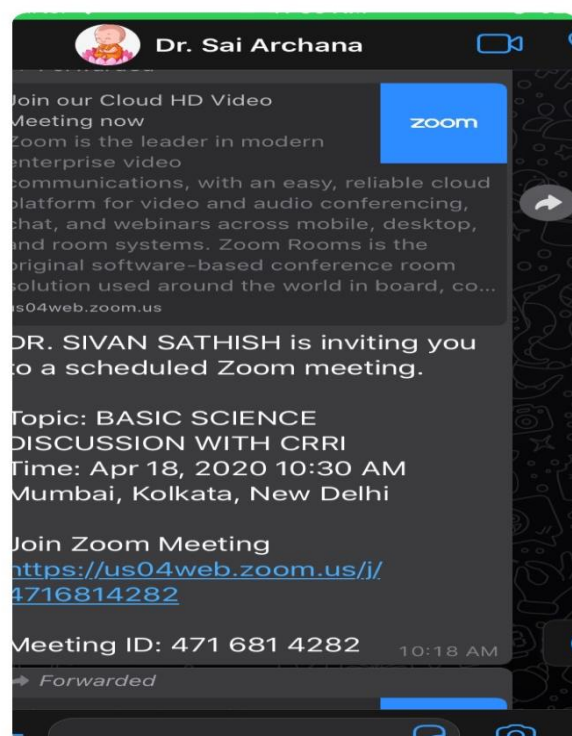
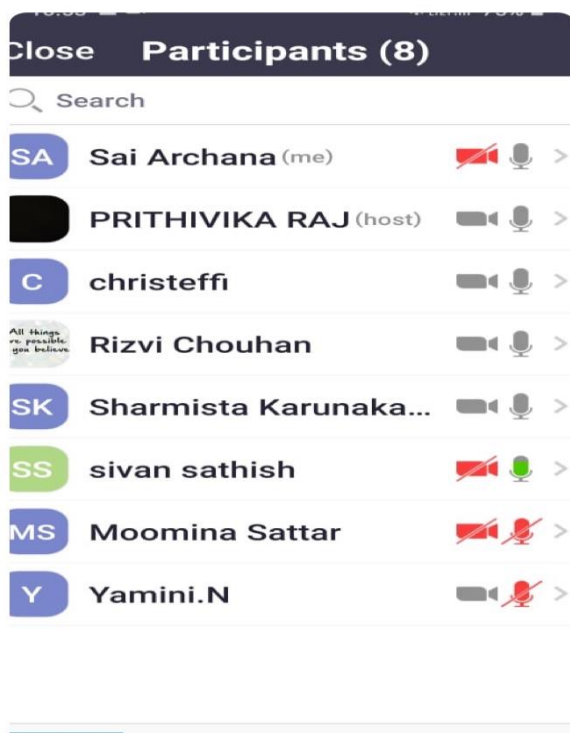
• **STUDENTS PARTICIPATED:**

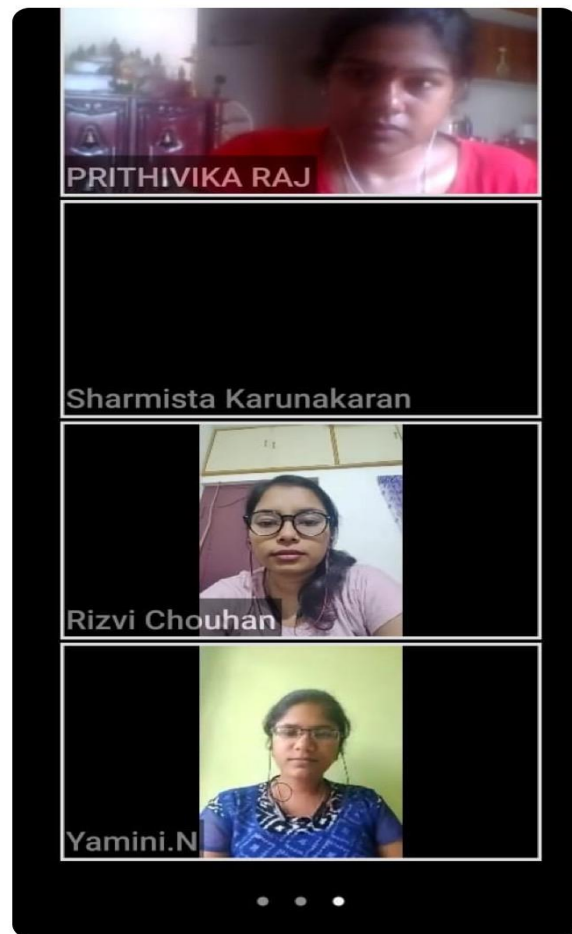
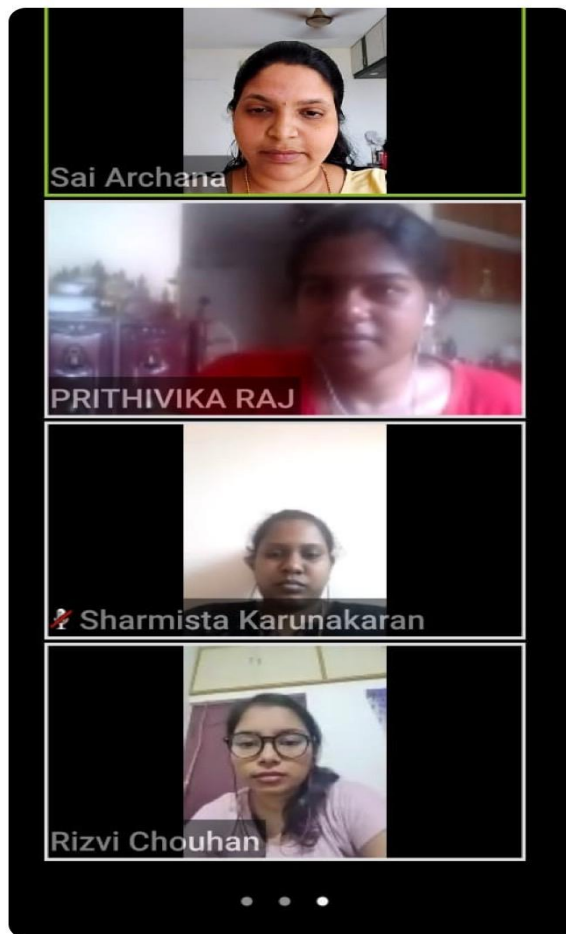
1. Raj Prithvika
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4. Sharmista
5. Soundhar Rajan
6. Yamini

• **WORK DONE:**

- *Dicussion on Basic Sciences*
- *Discussion with PGs on DIABETES MELLITUS AND ITS ORAL COMPLICATIONS*
- *Combined clinical classes with final years*
- *NEET Questions discussion with final years*

PICTURES:





AFTERNOON SESSION:

Dr. Sai Archana
online

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DR. SIVAN SATHISH is inviting you to a scheduled Zoom meeting.

Topic: BASIC SCIENCE DISCUSSION WITH CRRI
Time: Apr 18, 2020 03:15 PM
Mumbai, Kolkata, New Delhi

Join Zoom Meeting
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Meeting ID: 471 681 4282 1:35 PM

Forwarded
Christtefy mam's session

Close Participants (12)

Search

SA	Sai Archana (me)		
	Namarrthaa Sre (host)		
MS	Moomina Sattar		
	PRITHIVIKA RAJ		
SS	sivan sathish		
SR	SOUNDHAR RAJAN		
C	chrieteffi		
	Rizvi Chouhan		
S	Sumithra		
VR	Vignesh ravi		

15:44 52%



PRITHIVIKA RAJ

Sumithra

Rizvi Chouhan

Moomina Sattar

Participants (12)

Moomina Sattar



PRITHIVIKA RAJ



sivan sathish



SOUNDHAR RAJAN



chrieteffi



Rizvi Chouhan



Sumithra



Vignesh ravi



Yamini.N



Sharmista Karunaka...



Prithivika Raj



Vignesh Ravi



Prithivika Raj



Soundhar Rajan



Sumithra



Chrieteffi



Rizvi Chouhan



Yamini.N

