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Dental College & Research Institute

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:

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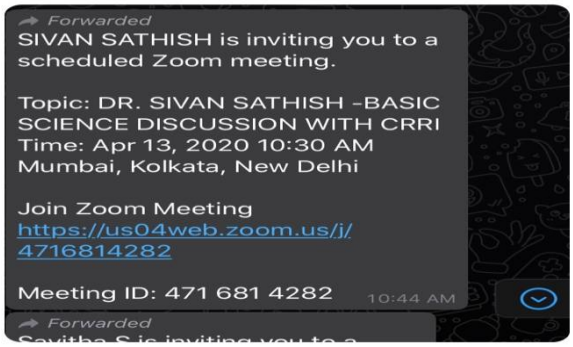
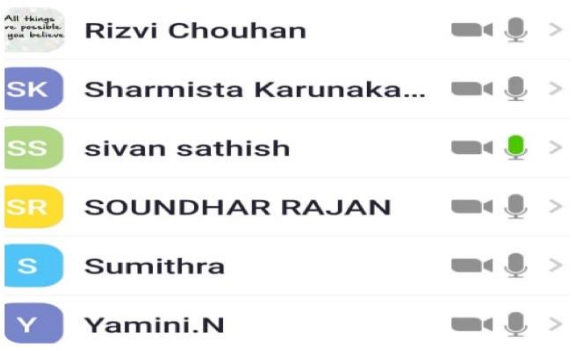
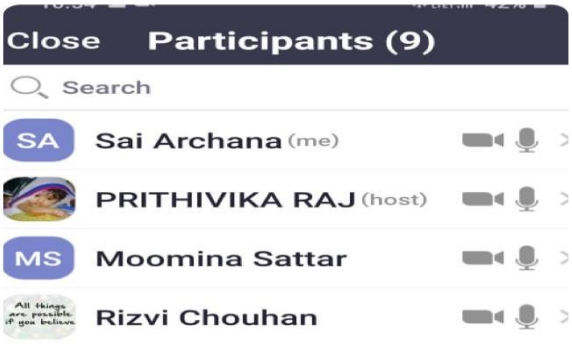
    1. Raj Prithvika
    2. Rizvi Chauhan
    3. Sumithra
    4. Sharmista
    5. Soundhar Rajan
    6. Yamini
- **WORK DONE:**
    - ***Dicussion on Basic Sciences***
    - ***Discussion with PGs on Calcium Homeostasis***
    - ***Combined clinical classes with final years.***
    - ***NEET Questions discussion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

PICTURES:







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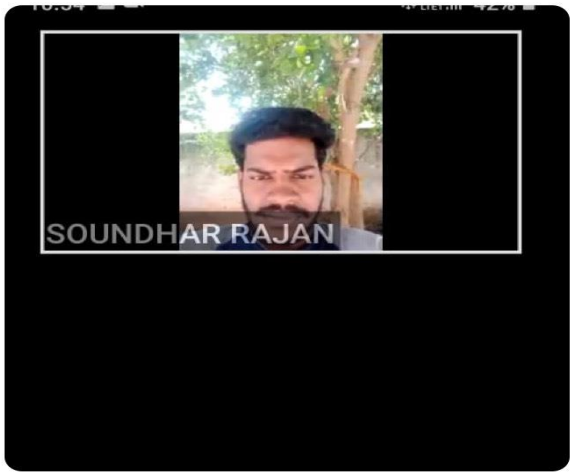

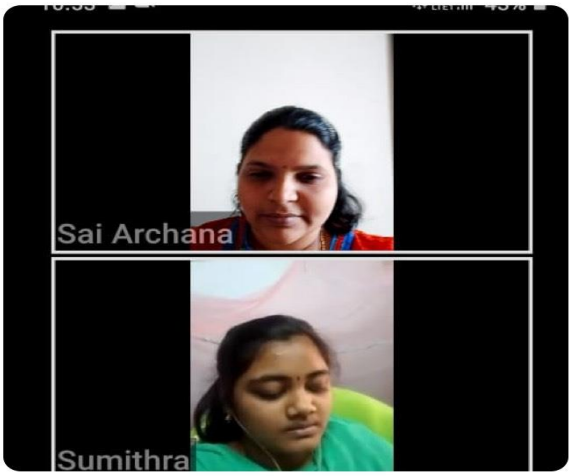

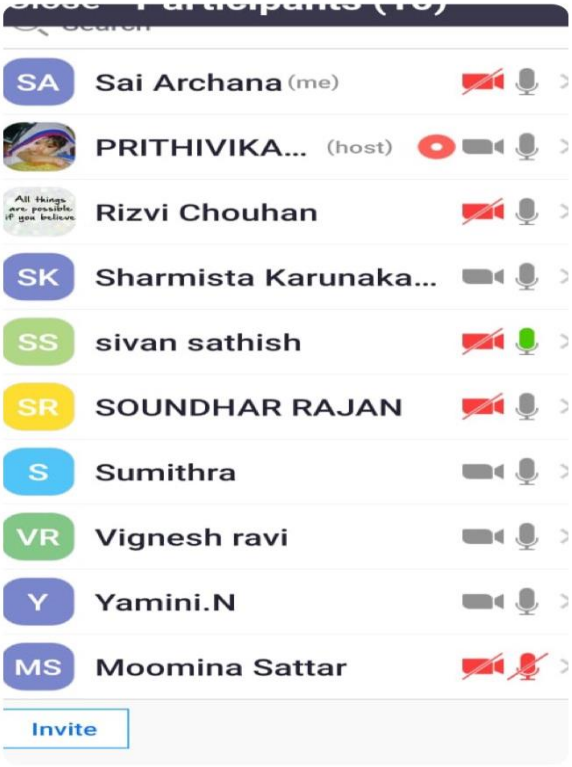

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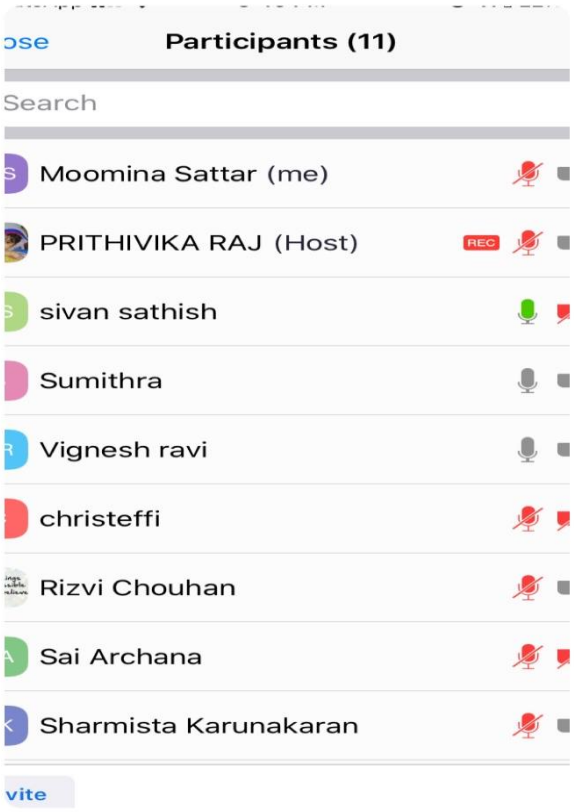

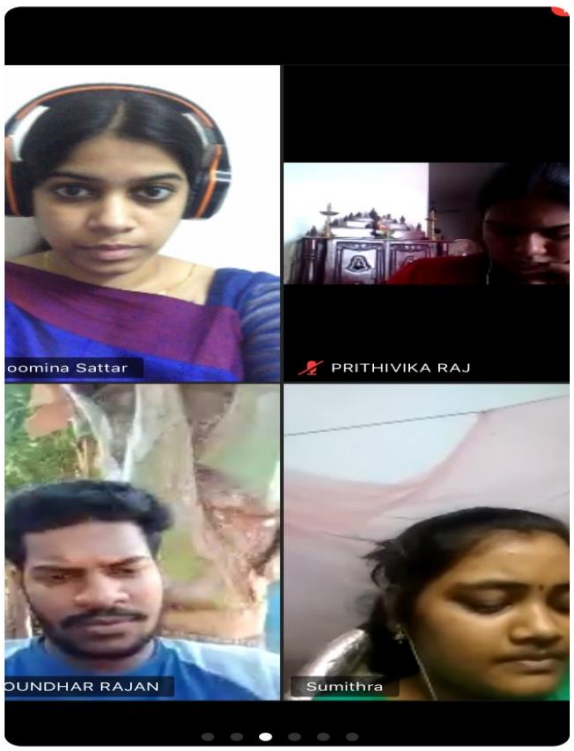

15.4.2020:

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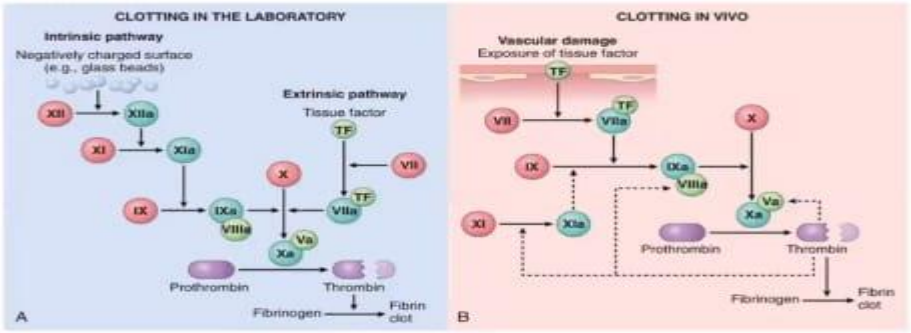
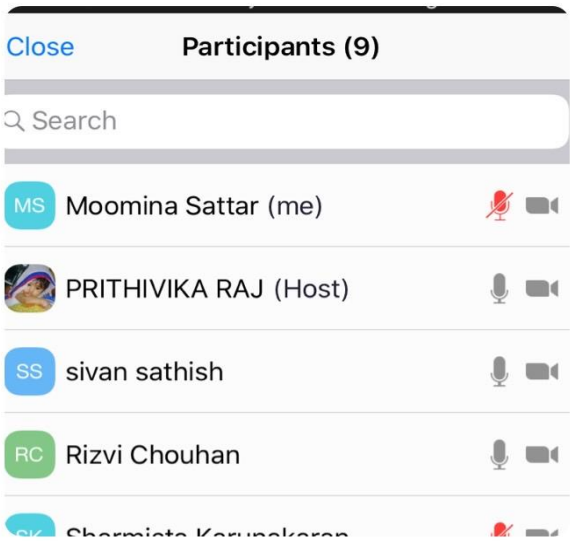


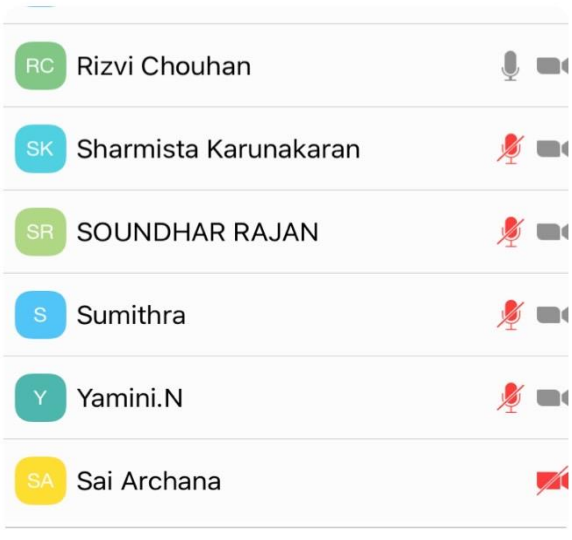
Fig. 4.7 The coagulation cascade in the laboratory and in vivo. (A) Clotting is initiated in the laboratory by adding phospholipids, calcium, and either a negative-charged substance such as glass beads (intrinsic pathway) or a source of tissue factor (extrinsic pathway). (B) In vivo, tissue factor is 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

- The *partial thromboplastin time* (PTT) assay screens the function of the proteins in the intrinsic pathway (factors

Sharmista Karunakaran's screen





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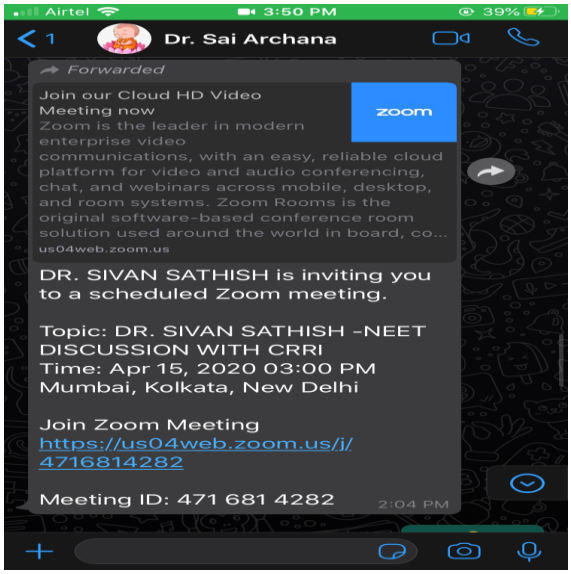

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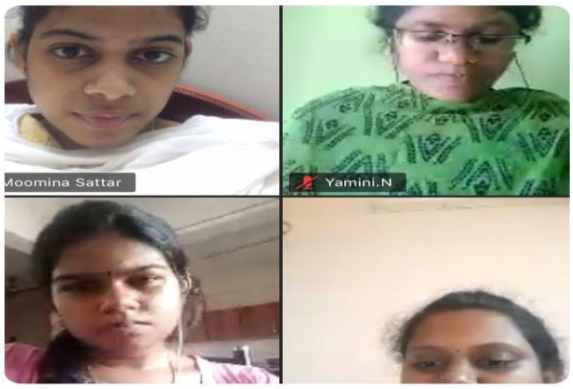

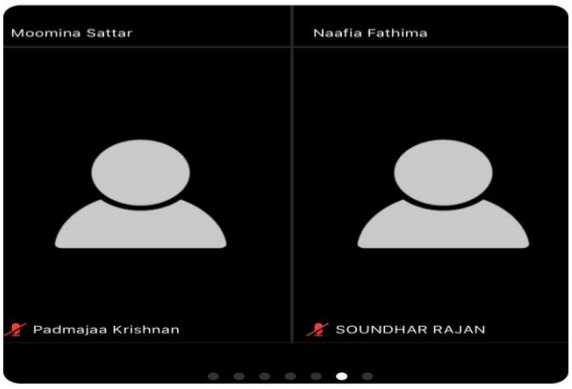

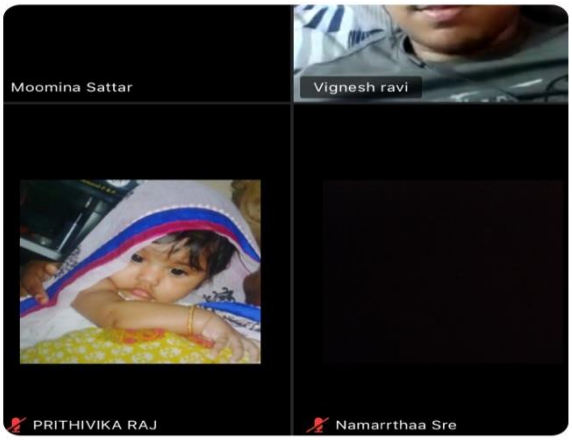

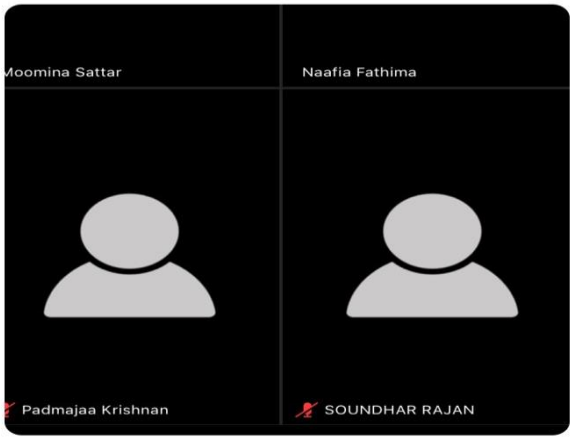

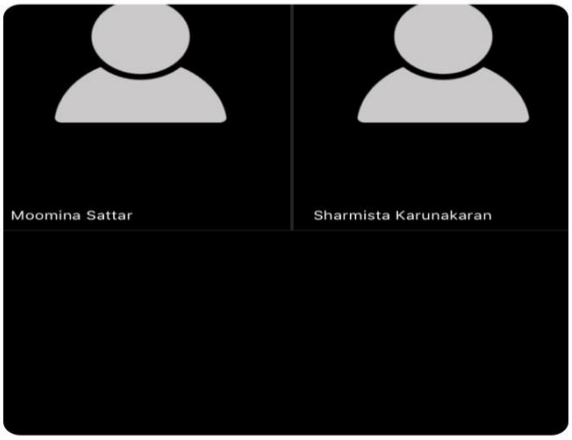

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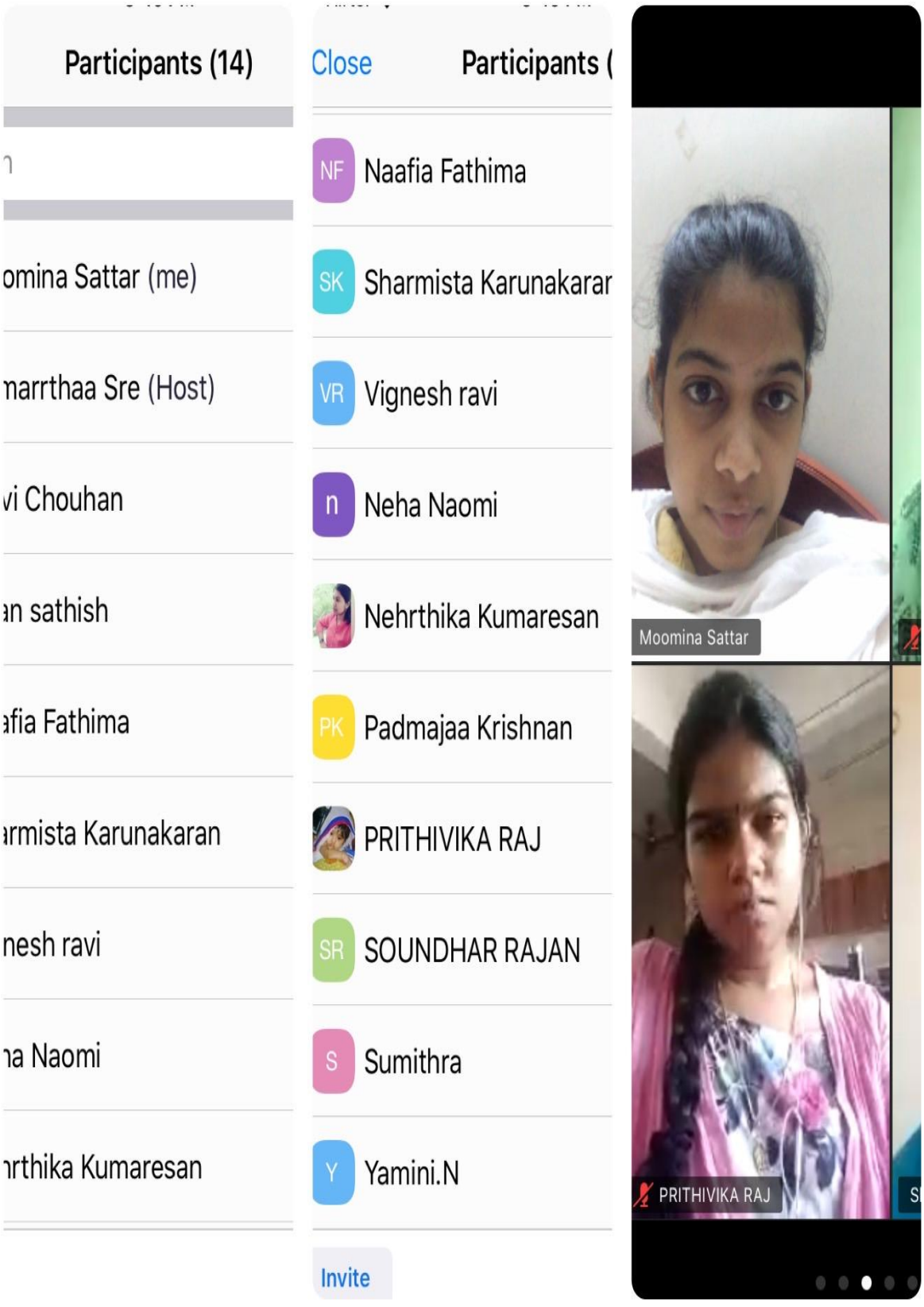

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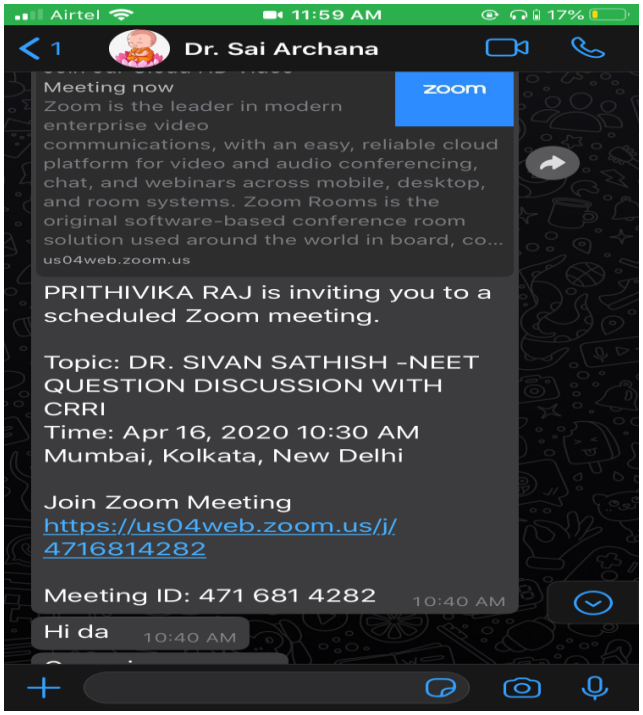

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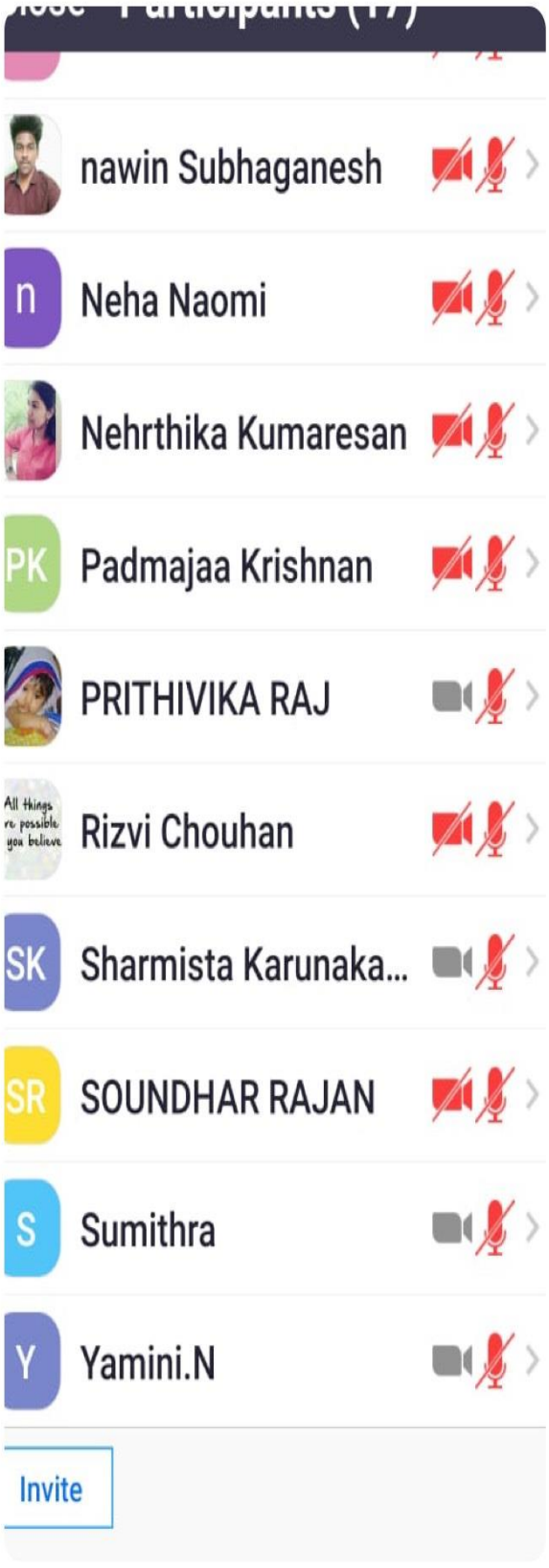

88 SECTION I Cellular and Molecular Basis for Medical Physiology

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 4–1B). 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-μm 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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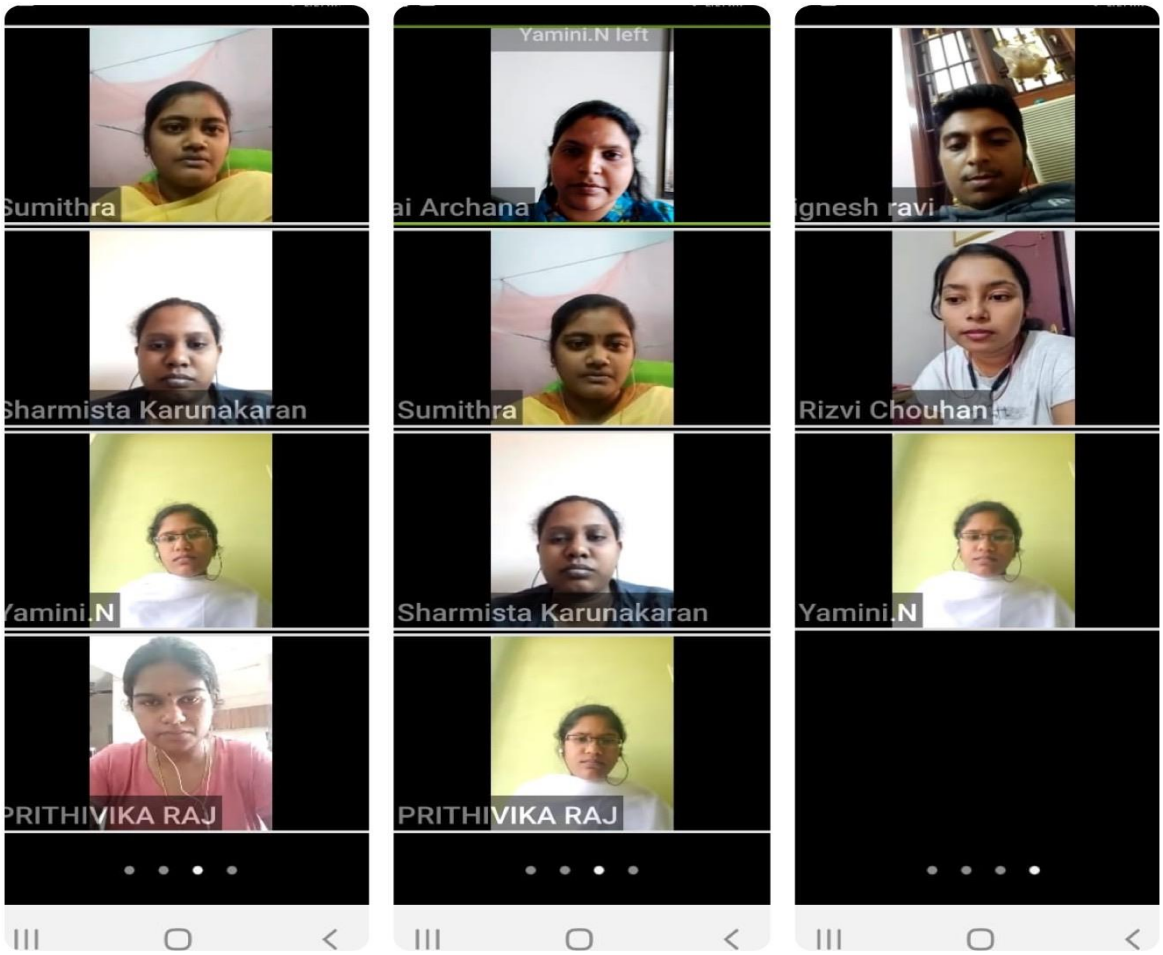

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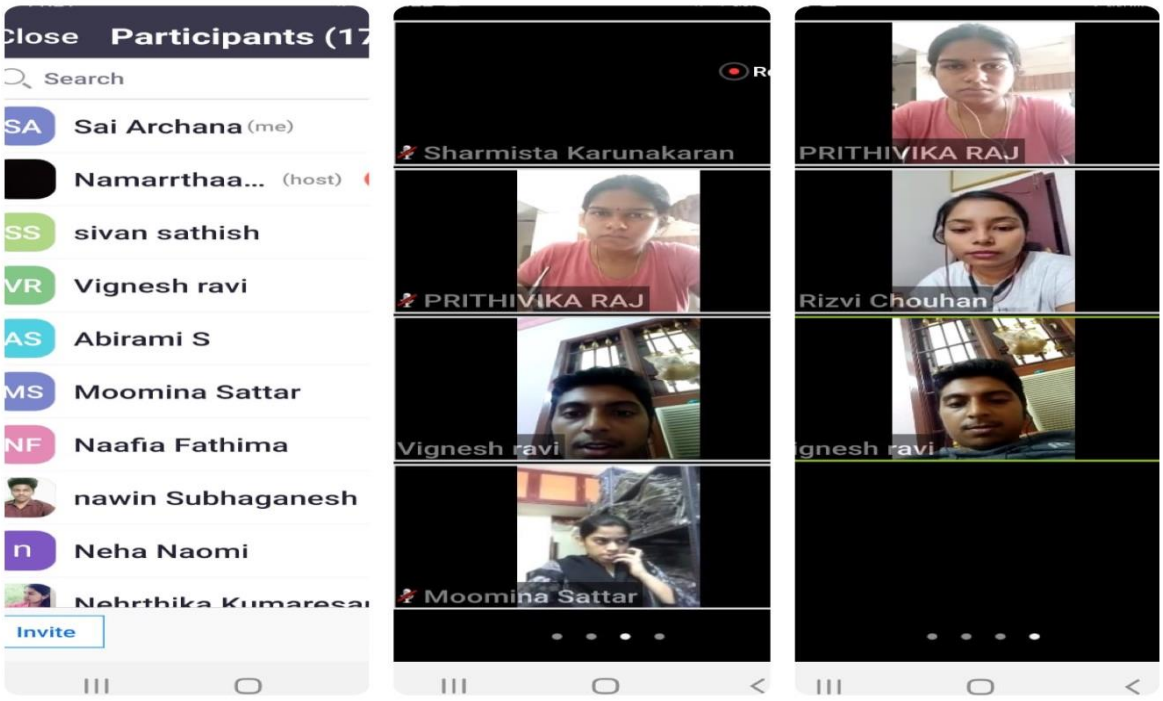

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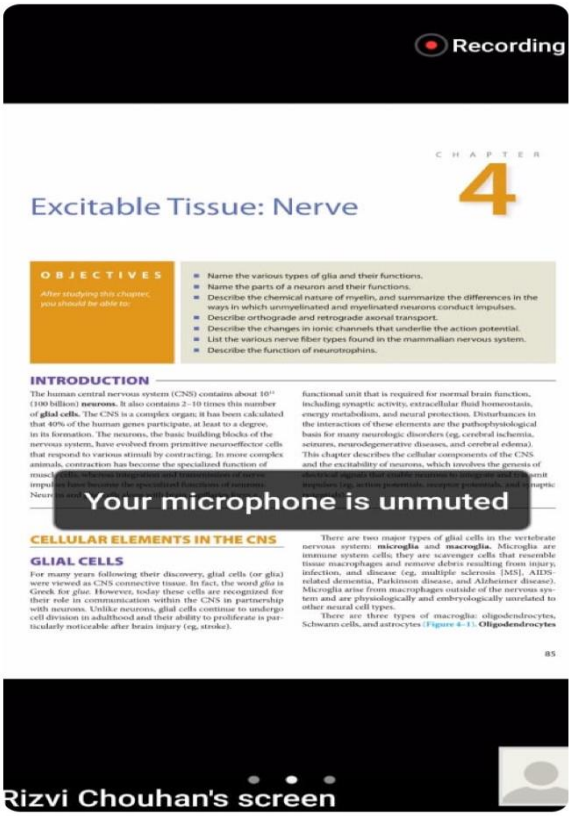

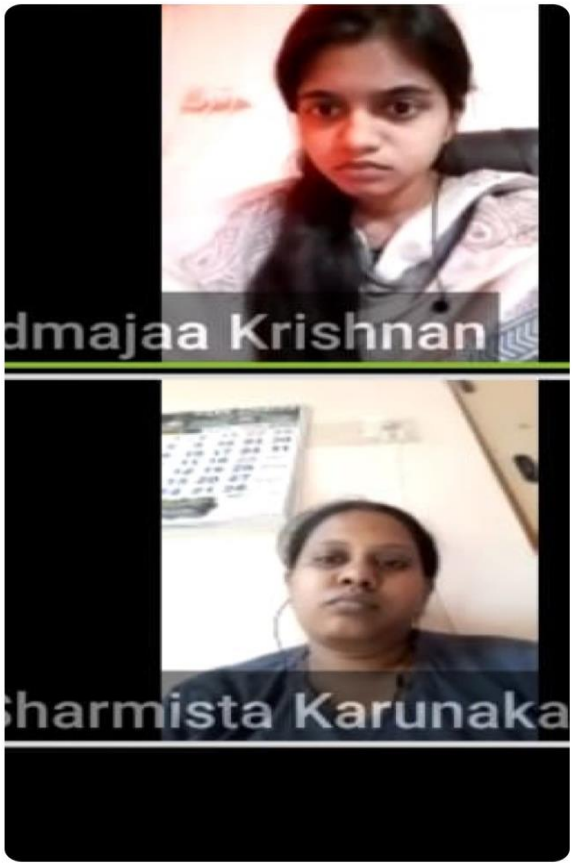

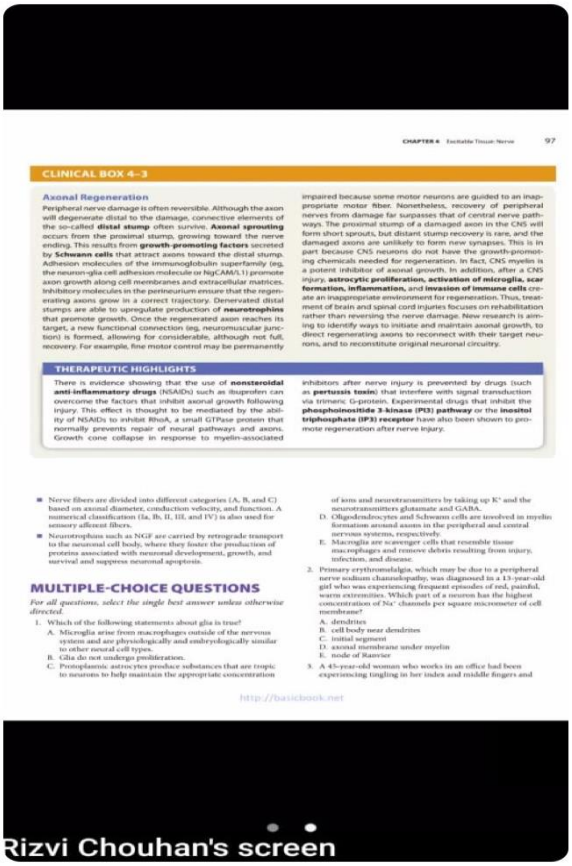

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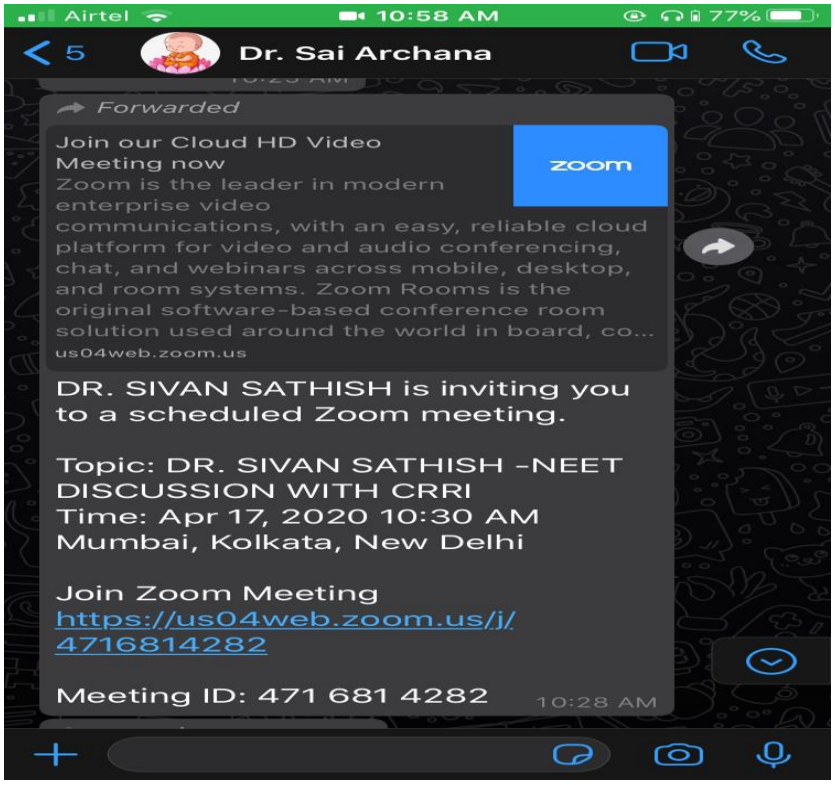


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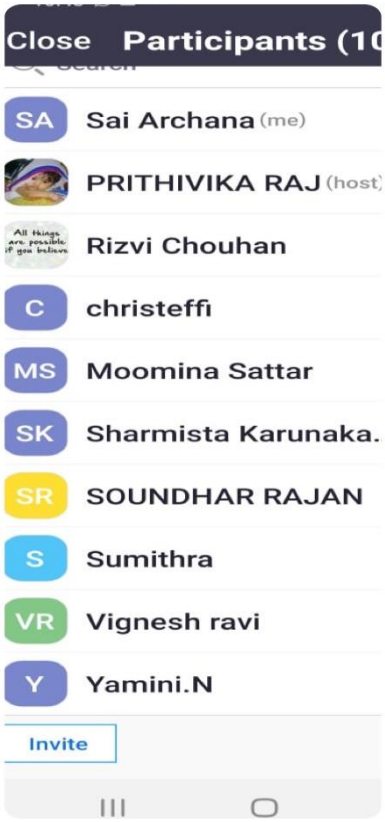
Rizvi Chouhan
sivan sathish
Sai Archana
Sharmista Karunakaran
SOUNDHAR RAJAN
Sumithra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Participants (10
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PRITHIVIKA RAJ (host)
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christeffi
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Moomina Sattar
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Vignesh ravi
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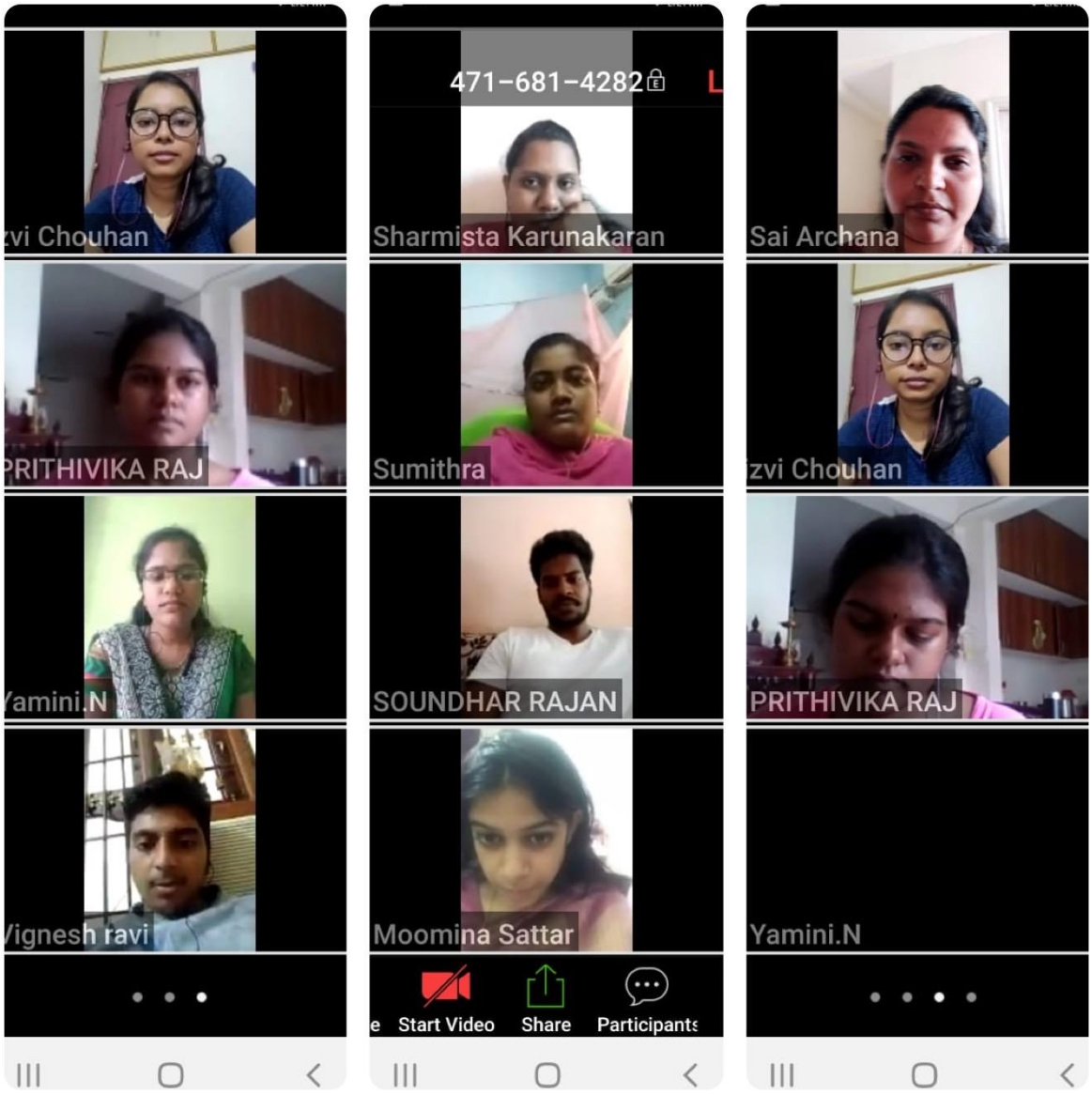


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DR. SIVAN SATHISH is inviting you to a scheduled Zoom meeting.
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CHAPTER 5 Excitable Tissue: Muscle 105
channel with Ca2+ as its natural ligand. In skeletal muscle, Ca2+ entry from the extracellular 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
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Rizvi Chouhan's screen

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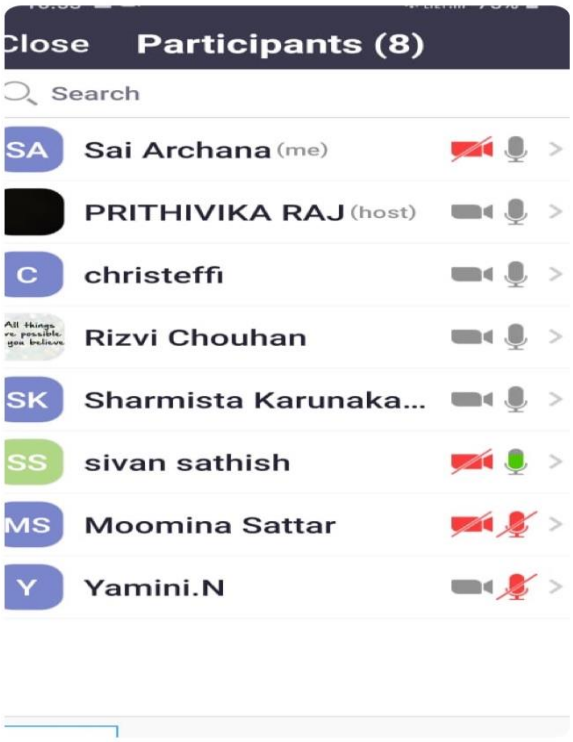
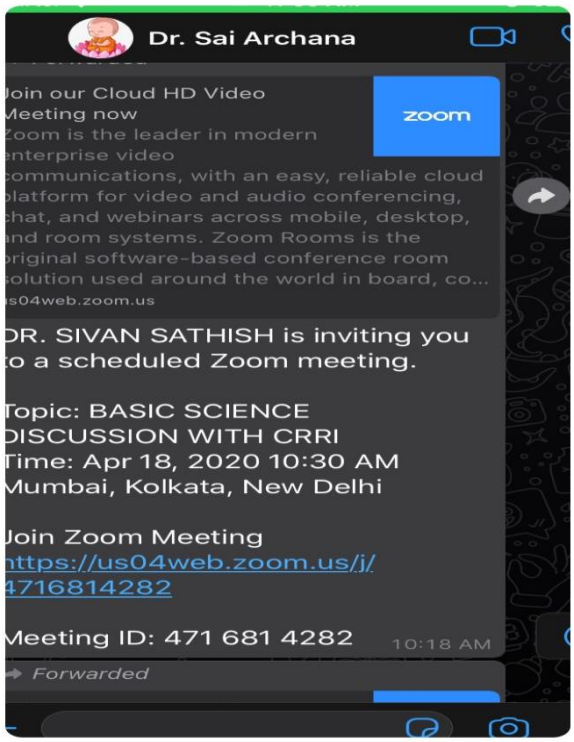
PICTURES:





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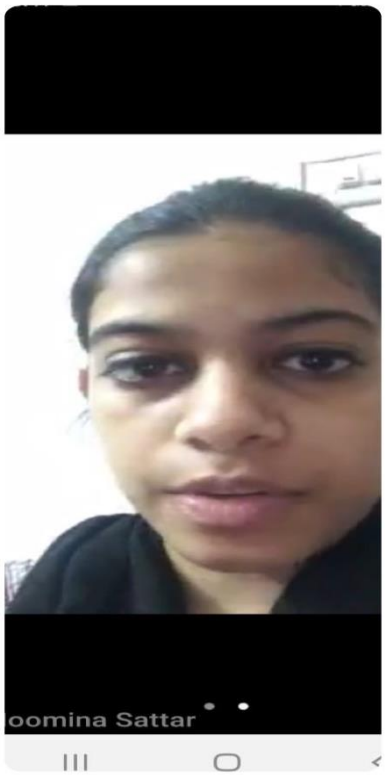

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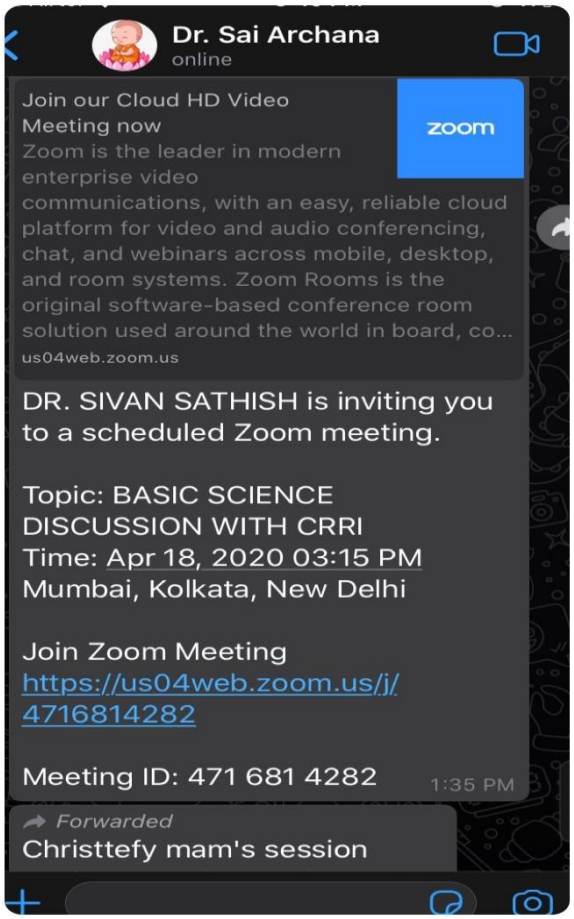

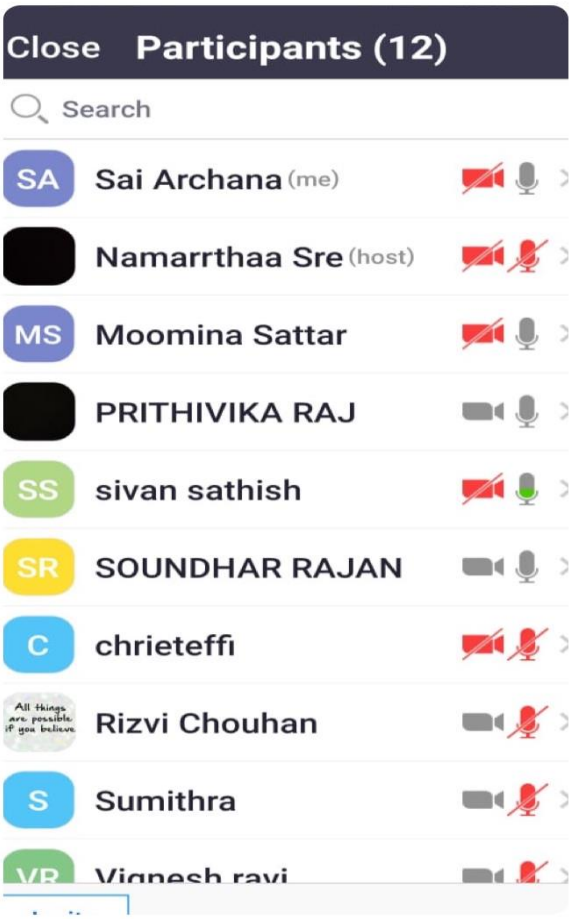

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