

ORAL MEDICINE AND
RADIOLOGY- CRRI WORK
DONE

27.04.2020-02.05.2020

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

27.4.2020:

SUPERVISION AND DISCUSSION BY
Dr.SIVAN SATHISH, MDS, MFDS RCPS
PROFESSOR AND HOD, ORAL MEDICINE



<u>Work done</u>	<u>Participants</u>	<u>Timing</u>
Discussion on Basic science NEET Discussion	CRRI, IV Year	10.30-11.30 am
Discussion with PGs on <i>Shock</i>	PGs, CRRIs, Final year, Third year	11.30-01.00 pm
Combined theory class with final years- COMPUTED TOMOGRAPHY	CRRI, Final year, III Year	02 pm -03 pm
NEET Questions Discussion	CRRI, III Year	03 pm- 04 pm

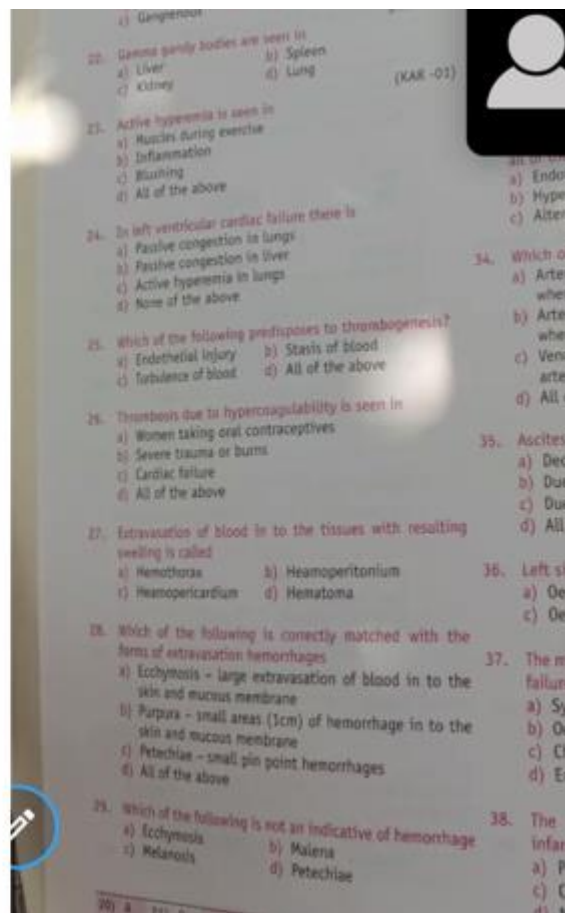
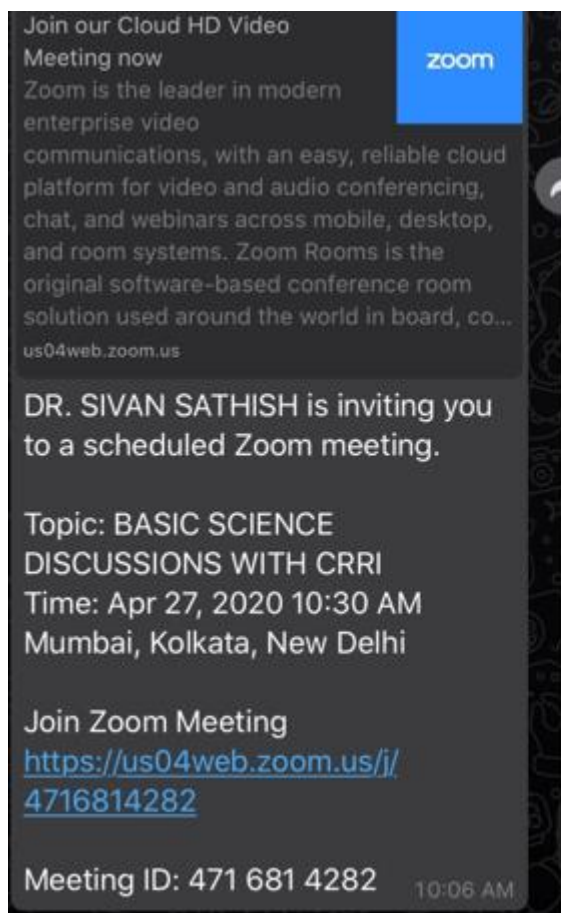
- **NAME OF STAFFS PARTICIPATED: 4**

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

PICTURES:















Close Participants (14)

-  Pavithra RathinaSw...
-  P Prabhavathi.s
-  P prasanth
-  PRITHIVIKA RAJ
-  Priya Darshini
-  SK Sharmista Karunaka.
-  SS sivan sathish
-  SR SOUNDHAR RAJAN
-  S Sumithra
-  Y Yamini.N

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Close Participants (14)

- Search
-  SA Sai Archana (me)
 -  PL Pavithra laksh... (hos
 -  MS Moomina Sattar
 -  PR Pavithra Rajkumar
 -  Pavithra RathinaSw...
 -  P Prabhavathi.s
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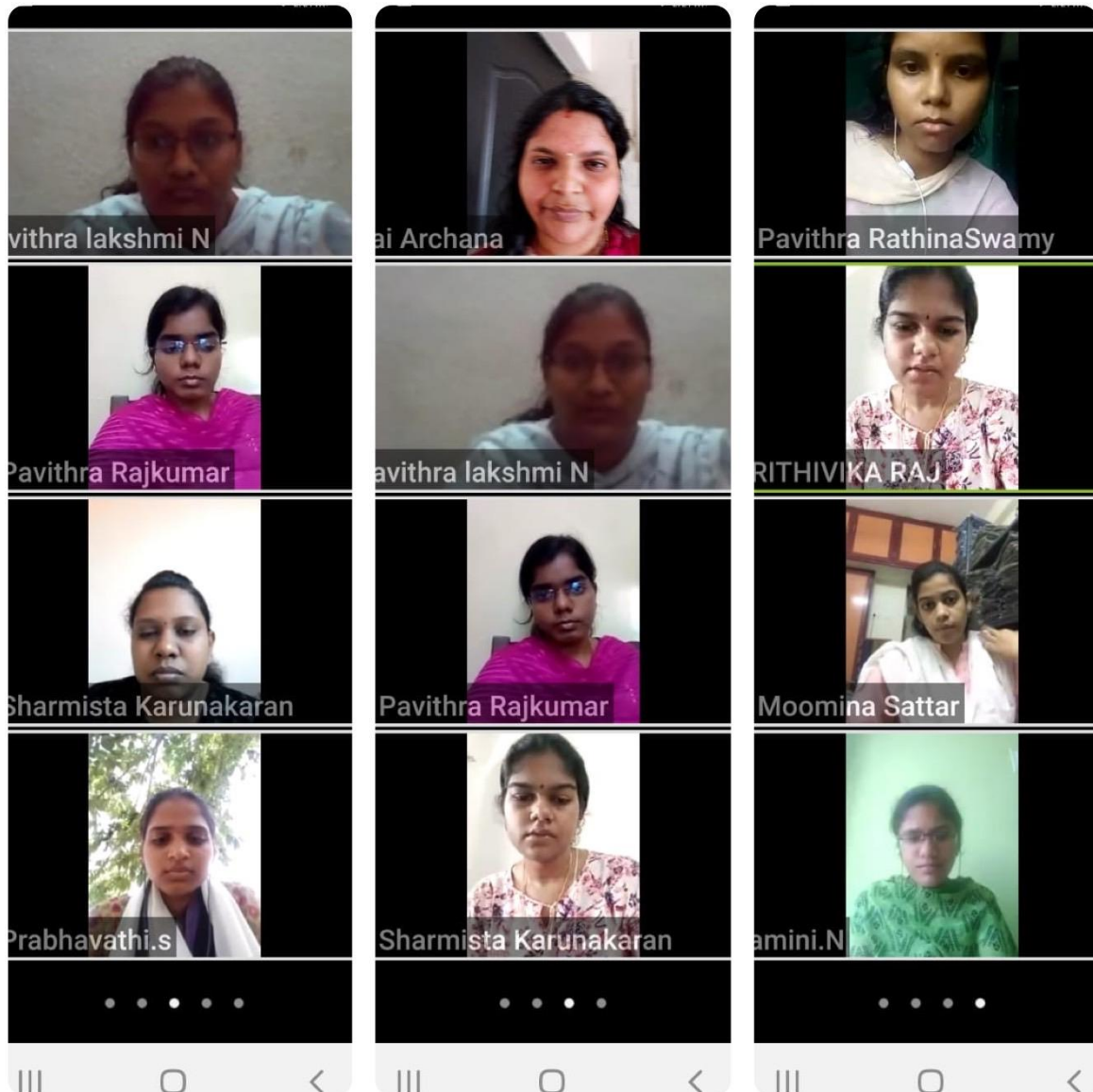


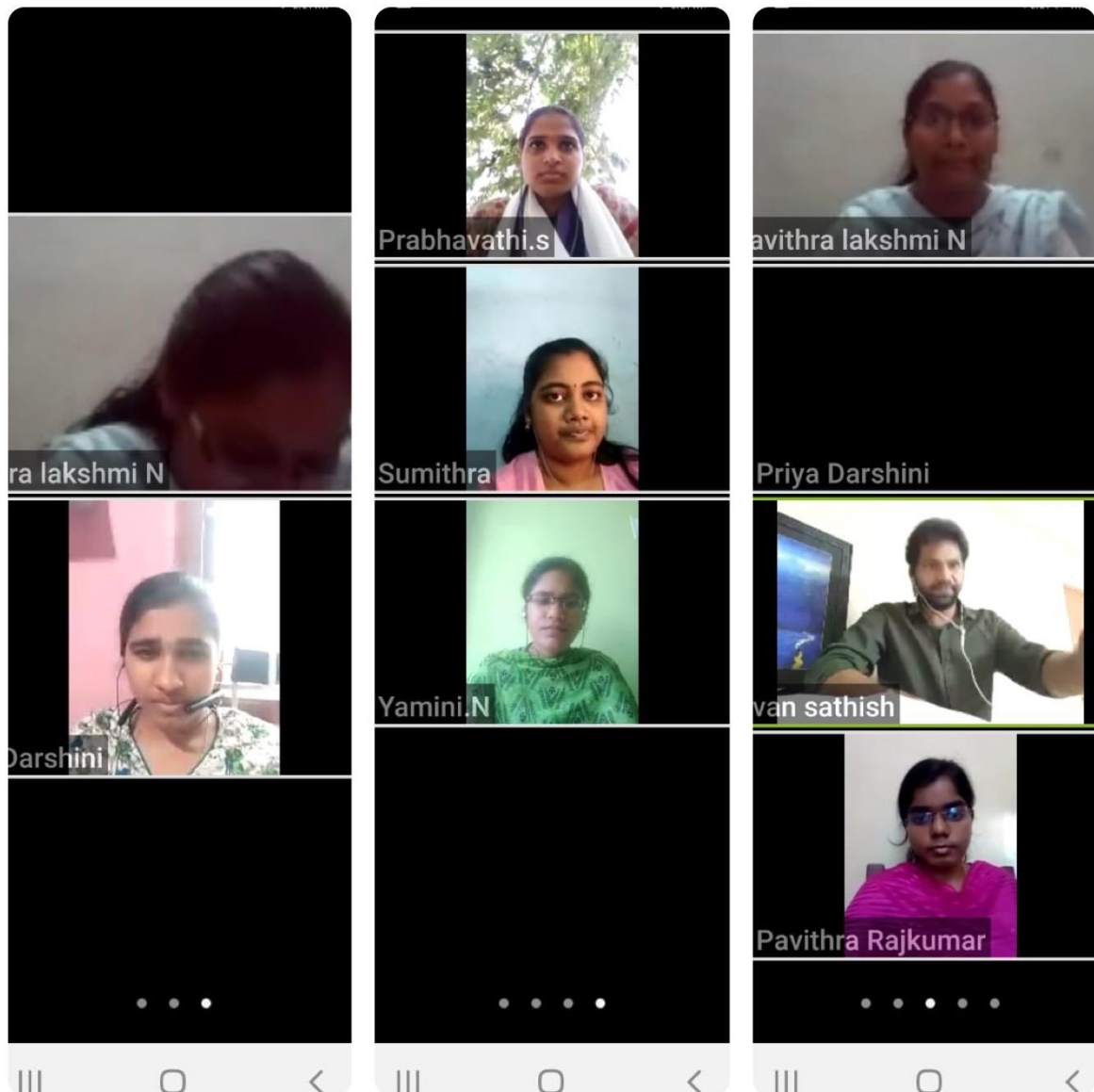
Close Participants (16)

- Search
-  Sai Archana (me)
 -  Pavithra laksh... (host)
 -  Priya Darshini
 -  Rizvi Chouhan
 -  sivan sathish
 -  SOUNDHAR RAJAN
 -  Vignesh ravi
 -  Moomina Sattar
 -  Pavithra Rajkumar
 -  Prabhavathi.s

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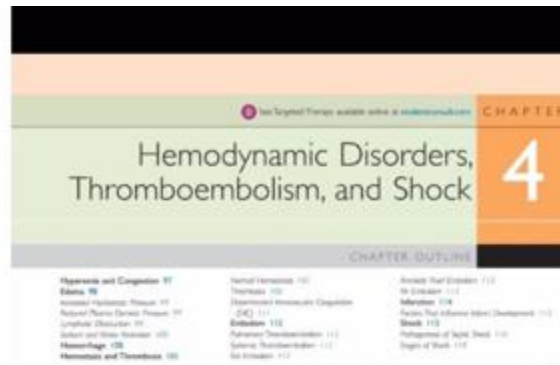
DR. SIVAN SATHISH is inviting you to a scheduled Zoom meeting.

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1:32 PM



The health of cells and tissues depends on the circulation of blood, which delivers oxygen and nutrients and removes wastes generated by cellular metabolism. Under normal conditions, as blood passes through capillary beds, proteins in the plasma are retained within the vasculature and there is little net movement of water and electrolytes into the tissues. This balance is often disturbed by pathologic conditions that alter endothelial function, increase vascular hydrostatic pressure, or decrease plasma protein content, all of which promote edema—the accumulation of fluid in tissues resulting from a net movement of water into extravascular spaces. Depending on the severity and location, edema may have minimal or profound effects. In the lower extremities, it may only make one's shoes feel snugger after a long sedentary day; in the lungs, however, where fluid can fill alveoli, causing life-threatening hypoxia.

The structural integrity of blood vessels is frequently compromised by trauma. Hemorrhage is the process of blood clotting that prevents excessive bleeding after blood vessel damage. Inadequate hemostasis may result in hemorrhage, which can compromise regional tissue perfusion and, if massive and rapid, may lead to hypotension, shock, and death. Conversely, inappropriate clotting (thrombosis) or migration of clots (embolism) can obstruct blood vessels, potentially causing ischemia, cell death (necrosis), infarction, thromboembolism (in the heart of three major causes of morbidity and death in developed countries: myocardial infarction, pulmonary embolism (PE), and cerebrovascular accident (stroke)).

With this in a preface, we begin our discussion of hemodynamic disorders with conditions that increase tissue blood volume.































HYPEREMIA AND CONGESTION

Hyperemia and congestion both refer to an increase in blood volume within a tissue, but have different underlying mechanisms. Hyperemia is an active process resulting from arteriolar dilation and increased blood inflow, as occurs at sites of inflammation or in exercising skeletal muscle. Hyperemic tissues are redder than normal because of engorgement with oxygenated blood. Congestion is a passive process resulting from impaired outflow of venous blood from a tissue. It can occur symmetrically, as in cardiac failure, or locally as a consequence of an isolated venous obstruction. Congested tissues have an abnormal blue-red color (cyanosis) that stems from the accumulation of deoxygenated hemoglobin in the affected area. In long-standing chronic congestion, inadequate tissue perfusion and persistent hypoxia may lead to perivascular cell death and secondary tissue fibrosis, and the elevated intravascular pressures may cause edema or sometimes rupture capillaries, producing local hemorrhages.

MORPHOLOGY































The surface of hyperemic or congested tissue has wet and typically more bluish. On microscopic examination, acute pulmonary congestion is marked by blood-engorged vascular capillaries and variable degrees of alveolar septal edema and interstitial hemorrhage. In chronic pulmonary congestion, the septa become thickened and fibrotic, and the alveolar spaces contain numerous macrophages laden with hemosiderin ("heart failure cells") derived from phagocytosed red cells in alveoli.

Participants (17)

-  **PRITHIVIKA RAJ**  
-  **Priyadarshini Ganes...**  
-  **Priyadharshini A**  
-  **Rizvi Chouhan**  
-  **Rohithchandru K**  
-  **SAHANASHREE M**  
-  **Sharmista Karunaka...**  
-  **SOUNDHAR RAJAN**  
-  **Sumithra**  
-  **Yamini.N**  

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-  **RAKSHA RAMK...** (host)  
-  **Moomina Sattar**  
-  **Sivan sathish**  
-  **Vignesh ravi**  
-  **christeffi**  
-  **NIKILESH RAJ**  
-  **PRITHIVIKA RAJ**  
-  **Priyadarshini Ganes...**  
-  **Privadharshini A**  

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28.4.2020:

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<u>Work done</u>	<u>Participants</u>	<u>Timing</u>
Discussion on NEET Questions	CRRi	10.30-11.00 am
Clinical case presentation	CRRi, Final year	11.00-11.30 am
Discussion with PGs on <i>Fibrous Dysplasia</i>	PGs, CRRIs, Final year, Third year	11.30-01.00 pm
Clinical case presentation	CRRi, Final year	02 pm -03 pm
Theory class on Ideal long case	III Year, CRRi, Final Year	03 pm- 04 pm

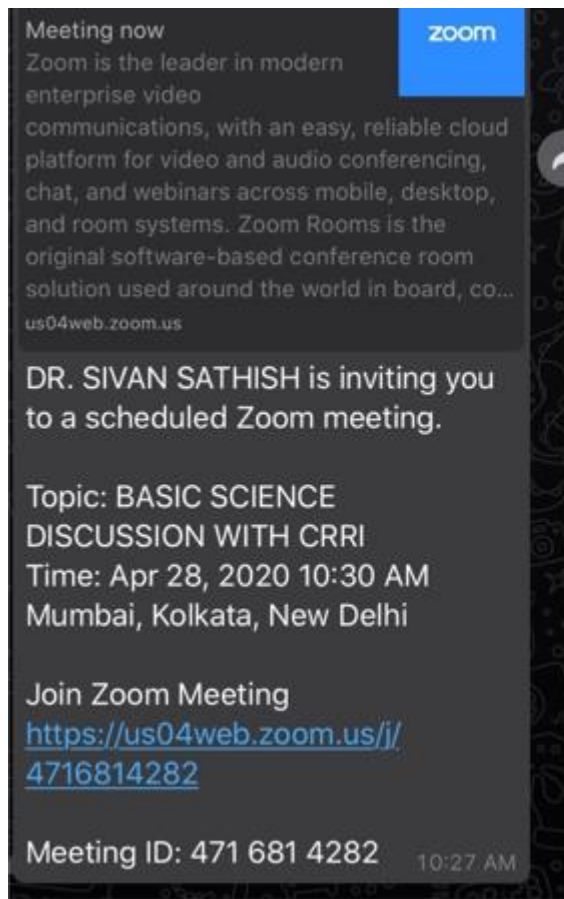
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CHAPTER 4 Hemodynamic Disorders, Thromboembolism, and Shock

Edema ("elephantiasis") by producing inguinal lymphatic and lymph node fibrosis. Infiltration and obstruction of superficial lymphatics by breast cancer may cause edema of the overlying skin; the characteristic bumpy pitted appearance of the skin of the affected breast is called *pitting edema* (orange pitted). Lymphedema also may occur as a complication of surgery. One relatively common setting for this form of edema is in women with breast cancer who undergo axillary lymph node dissection and/or irradiation, both of which can damage and obstruct lymphatic drainage, resulting in severe lymphedema of the arm.

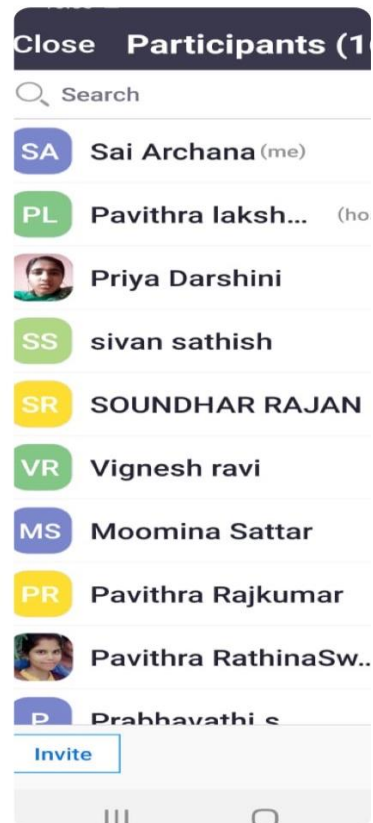
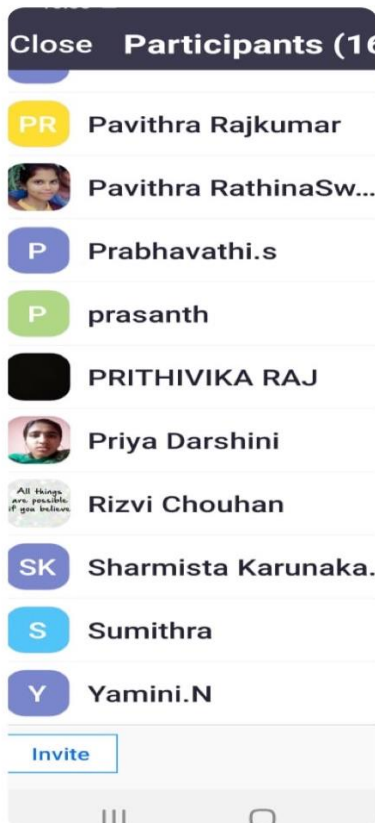
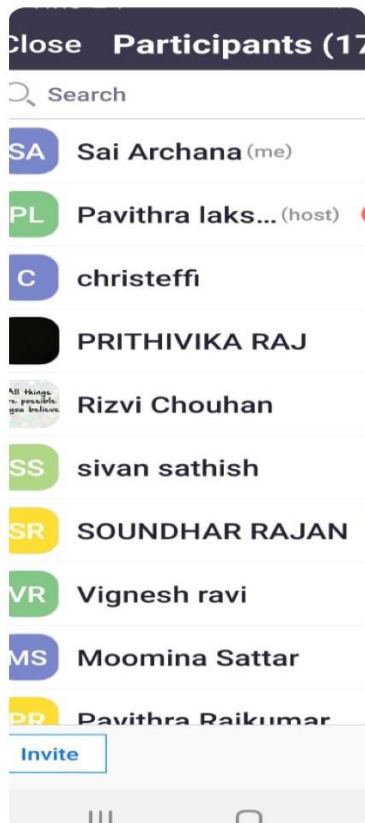
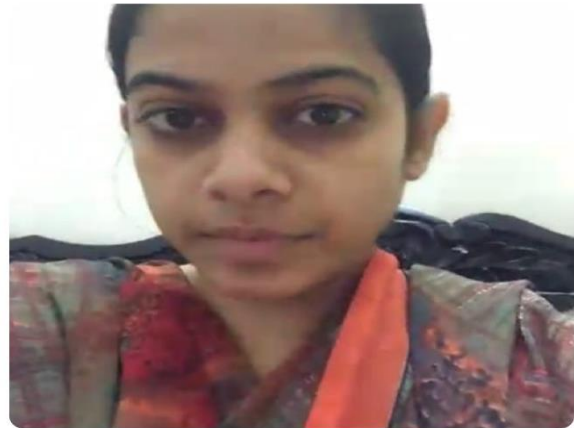
Sodium and Water Retention
 Excessive retention of salt (and its obligate associated water) can lead to edema by increasing hydrostatic pressure (because of expansion of the intravascular volume) and reducing plasma oncotic pressure. Excessive salt and water retention are seen in a wide variety of diseases that compromise renal function, including prerenal, renal, and postrenal causes, including prerenal, renal, and postrenal causes (Chapter 38).

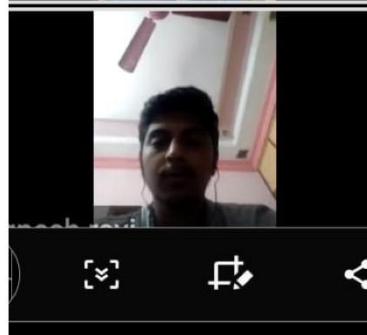
MORPHOLOGY
 Edema is easily recognized on gross inspection: intercapillary spaces become distended and numerous of the extravascular spaces (ECV) increase. Although any tissue can be affected, edema most commonly is encountered in subcutaneous tissue, lungs, and brain. Subcutaneous edema can be difficult but easily recognized preferentially in parts of the body positioned the greatest distance from the heart, where hydrostatic pressures are highest. Thus, edema typically is most pronounced in the legs with standing and the person with increasing a relatively normal dependent edema. Finger pressure over edematous subcutaneous tissue imparts the increased fluid, leaving a large, deep depression; this appearance is called *pitting edema*. Edema resulting from renal dysfunction or congestive syndrome also results in a large, compressible mass (e.g., the spleen, causing pericardial edema). With pulmonary edema, the lungs often are wet to three times their normal weight, and cutaneous edema (Chapter 22) can be localized (e.g., because of edema in a limb) or generalized depending on the nature and extent of the pathologic process or injury. With generalized edema, the skin is removed as the gross and becomes flaccid against the skin.

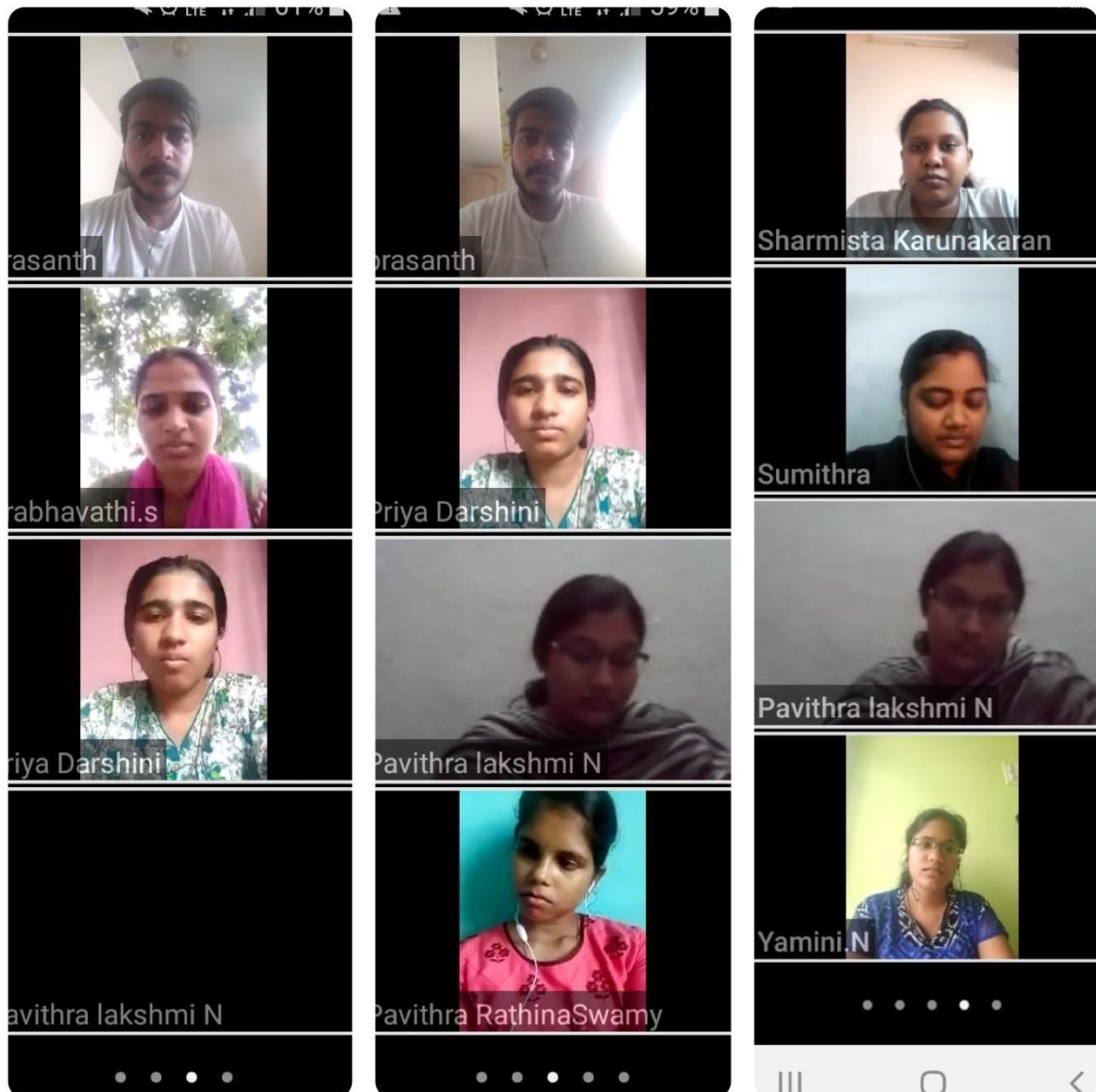
Clinical Features
 The effects of edema vary, ranging from locally annoying to life-threatening. Subcutaneous edema is important to recognize primarily because it signals potential underlying cardiac or renal disease; however, when significant, it also can impair wound healing and the clearance of infectious agents. Pulmonary edema is a common clinical problem; it is most frequent in the setting of left ventricular failure, but also may occur in renal failure, acute respiratory distress syndrome (Chapter 11), and inflammatory and infectious disorders of the lung. Severe cases result in pulmonary edema.

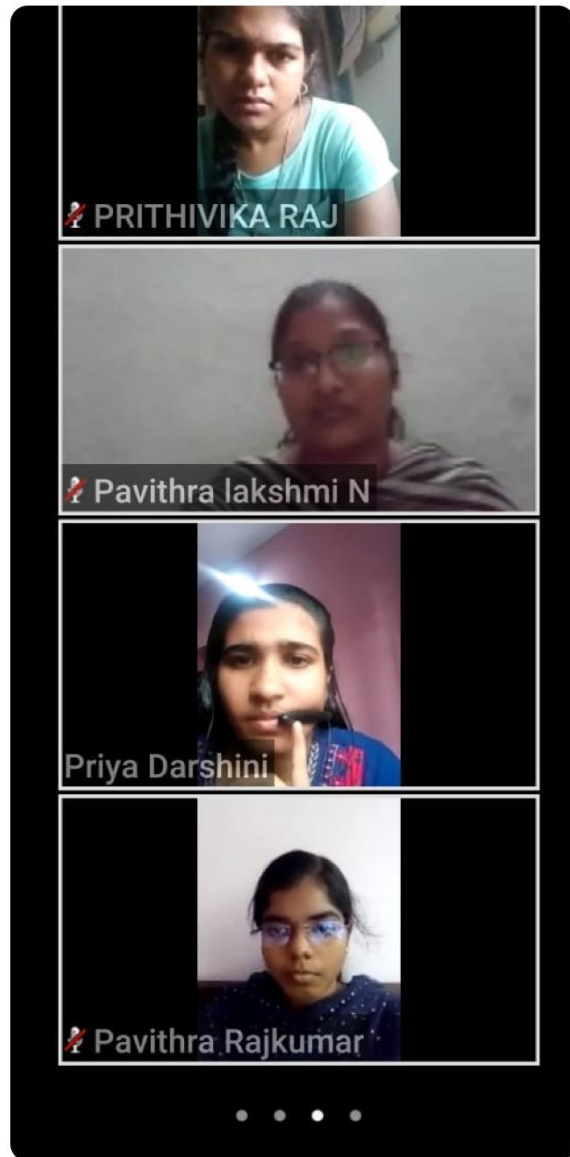
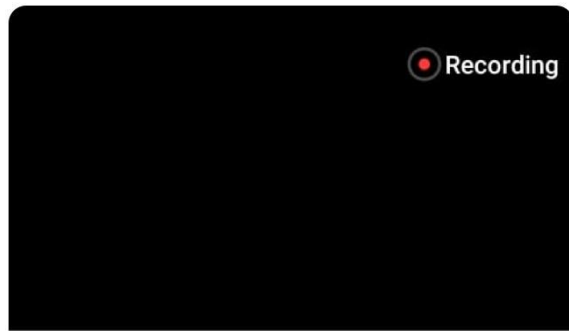
Edema results from the movement of fluid from the vasculature into the interstitial space; the fluid may be protein-poor (transudate) or protein-rich (exudate).
 Edema may be caused by:
 - Increased hydrostatic pressure (e.g., heart failure)
 - Increased vascular permeability (e.g., inflammation)
 - Decreased colloid osmotic pressure resulting from reduced plasma albumin
 - Decreased lymphatic flow (e.g., lymph node removal)
 - Increased flow (e.g., mitral regurgitation)
 - Lymphatic obstruction (e.g., inflammation or neoplasia)
 - Sodium retention (e.g., renal failure)

HEMORRHAGE
 Hemorrhage, defined as the extravasation of blood from vessels, is most often the result of damage to blood vessels or defective clot formation. As described earlier, capillary bleeding can occur in chronically congested tissues. Venous, arteriovenous, or inflammatory or neoplastic erosion of a vessel wall also may lead to hemorrhage, which may be extensive if the affected vessel is a large vein or artery.
 The risk of hemorrhage (often after a seemingly insignificant injury) is increased in a wide variety of clinical disorders collectively called *hemorrhagic diatheses*. These have diverse causes, including inherited or acquired defects in vessel walls, platelets, or coagulation factors, all of which impair function properly to ensure hemostasis. These are discussed in the next section. Other two forms of clinical features of hemorrhage, regardless of the cause.
 Hemorrhage may be classified by different appearance and clinical consequences.
 - Hemorrhage may be external or subcutaneous within a tissue as a hematoma, which ranges in significance from trivial (e.g., a bruise) to fatal (e.g., a massive intracranial hemorrhage resulting from rupture of a dissecting aortic aneurysm) (Chapter 10). Large bleeds into body cavities are described variously according to location—hemoptysis, hemopericardium, hemoperitoneum, or hemorheum (peritoneum). Extensive hemorrhage can also result in jaundice from the massive breakdown of red cells and hemoglobin.









29.4.2020:

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<u>Work done</u>	<u>Participants</u>	<u>Timing</u>
Discussion on NEET Questions	CRRRI	10.30-11.00 am
Clinical case presentation	CRRRI, Final year	11.00-11.30 am
Discussion with PGs on <i>Bleeding disorders</i>	PGs, CRRIs, Final year, Third year	11.30-01.00 pm
Clinical case presentation	CRRRI, Final year	02 pm -03 pm
NEET Questions discussion	CRRRI, Final Year	03 pm- 04 pm

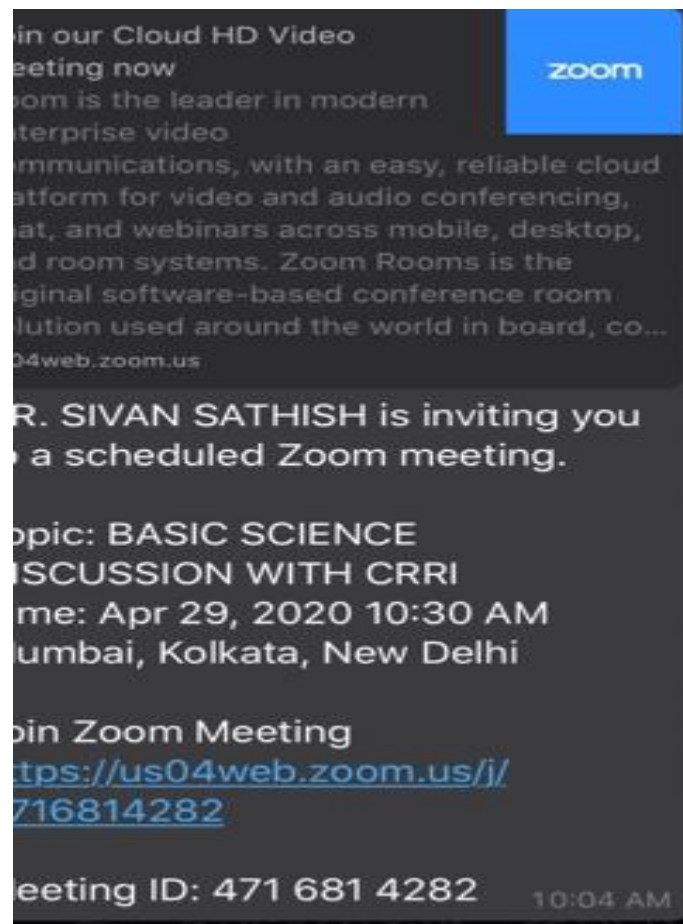
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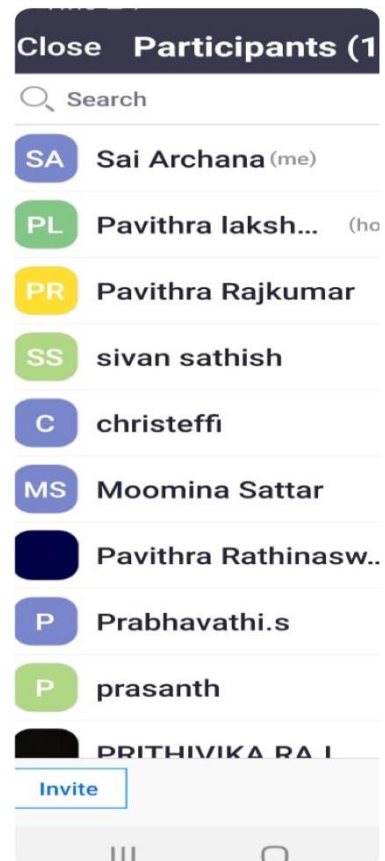
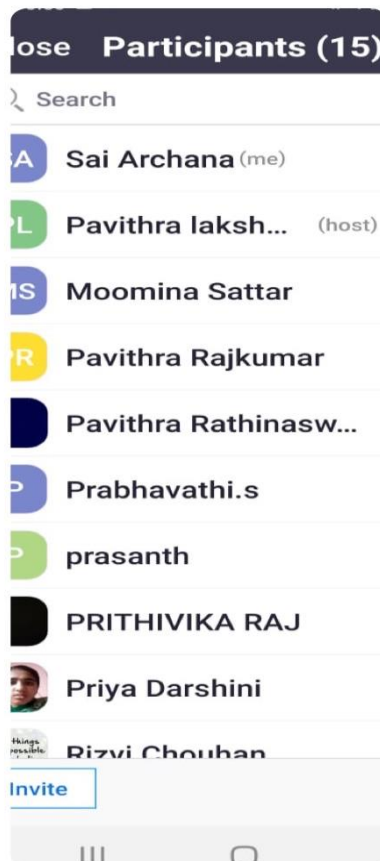
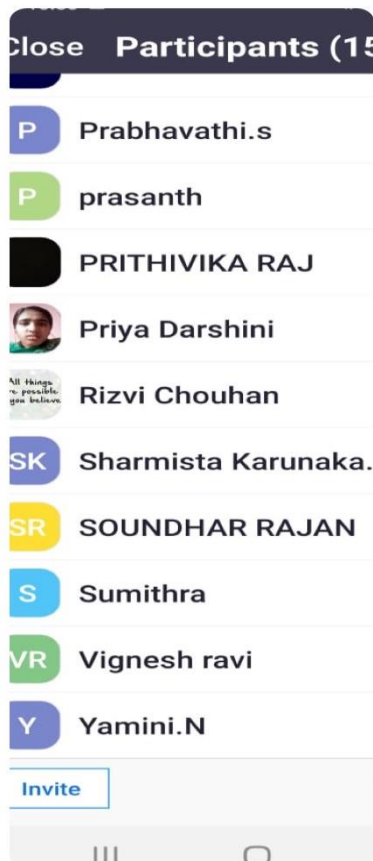
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by shifting the pattern of gene expression in endothelium to one that is "prothrombotic." This change is sometimes referred to as endothelial activation or dysfunction and can be produced by diverse exposures, including physical injury, infectious agents, abnormal blood flow, inflammatory mediators, metabolic abnormalities, such as hypercholesterolemia or homocysteinemia, and toxins absorbed from cigarette smoke. Endothelial activation is believed to have an important role in triggering arterial thrombotic events.

The role of endothelial cell activation and dysfunction in arterial thrombosis is also discussed in Chapters 10 and 11. Here it suffices to mention several of the major prothrombotic alterations:

- **Procoagulant changes:** Endothelial cells activated by cytokines downregulate the expression of thrombomodulin, already described as a key modulator of thrombin activity. This may result in sustained activation of thrombin, which can in turn stimulate platelets and augment inflammation through PAFs expressed on platelets and inflammatory cells. In addition, inflamed endothelium also downregulates the expression of other anticoagulants, such as protein C and tissue factor protein inhibitor, changes that further promote a procoagulant state.
- **Anti-fibrinolytic effects:** Activated endothelial cells secrete Plasminogen activator inhibitors (PAI), which limit fibrinolysis and downregulate the expression of t-PA, alterations that also favor the development of thrombosis.

Abnormal Blood Flow

Turbulent and static blood flow contributes to arterial and cardiac thrombosis by causing endothelial injury or dysfunction, as well as by forming stases or currents and local pockets of stasis. Stasis is a major factor in the development of venous thrombosis. Under conditions of normal laminar blood flow, platelets (and other blood cells) are found mainly in the center of the vessel lumen, separated from the endothelium by a shear-protecting layer of plasma. By contrast, stasis and turbulence have the following deleterious effects:

- Both promote endothelial cell activation and enhanced procoagulant activity, in part through flow-induced changes in endothelial gene expression.
- Stasis allows platelets and leukocytes to come into contact with the endothelium when the flow is sluggish.
- Stasis also slows the washout of activated clotting factors and impedes the inflow of clotting factor inhibitors.

Turbulent and static blood flow contributes to thrombosis in a number of clinical settings. Unreversed atherosclerotic plaques not only expose subendothelial ECM but also cause turbulence. Abnormal aortic and arterial dilations called aneurysms create local stasis and consequently are fertile sites for thrombosis (Chapter 1). Acute myocardial infarction results in locally noncontractile myocardium. Ventricular remodeling after more remote infarction can lead to aneurysm formation. In both cases, cardiac mural thrombi are more easily formed because of the local blood stasis (Chapter 11). Mitral valve stenosis (Chapter 12, after rheumatic heart disease) results in left atrial

distention. In conjunction with atrial fibrillation, this produces stasis and is a major factor in the development of thrombi. Hypertension, such as polycythemia vera, contributes to flow and causes small vessel thromboses, and the resultant risk of thrombosis.

Hypercoagulability

Hypercoagulability refers to an abnormally high tendency of the blood to clot, and is typically caused by alterations in coagulation factors. It contributes independently to arterial or intracardiac thrombosis but is an important underlying risk factor for venous thrombosis. The alterations of the coagulation pathways that predispose affected persons to thrombosis can be divided into primary (genetic) and secondary (acquired) disorders (Table 4.2).

Primary (inherited) hypercoagulability is most often caused by mutations in the factor V and prothrombin genes:











- Approximately 25 to 35% of whites carry a specific factor V mutation (called the Leiden mutation), after the Dutch city where it was first described. Among those with recurrent deep venous thrombosis (DVT), the frequency of this mutation approaches 80%. The mutation alters an active acid residue in factor V and renders it

Table 4.2 Hypercoagulable States

Primary (Genetic)
Common (>1% of the Population)
Factor V mutation (F508A mutation factor V Leiden)
Prothrombin mutation (G20210A variant)
Increased levels of factor VIII, IX, or XI or fibrinogen
Rare
Anti-thrombin III deficiency
Protein C deficiency
Protein S deficiency
Very Rare
Hyperhomocysteinemia (increased homocysteine)
Hyperhomocysteinemia (increased homocysteine)
Secondary (Acquired)
High Risk for Thrombosis
Dehydration and oral or intravenous
Myocardial infarction
Acute leukemia
Tissue injury (surgery, trauma, burn)
Cancer
Thrombotic thrombocytopenic syndrome
Disseminated intravascular coagulation
Heparin-induced thrombocytopenia
Anti-phospholipid antibody syndrome
Lower Risk for Thrombosis
Cardiomegaly
Hypertension
Hyperhomocysteinemia (pregnancy and postpartum)
Oral contraceptive use
Sickle cell anemia
Smoking

<http://medscape.com/viewarticle/944444>











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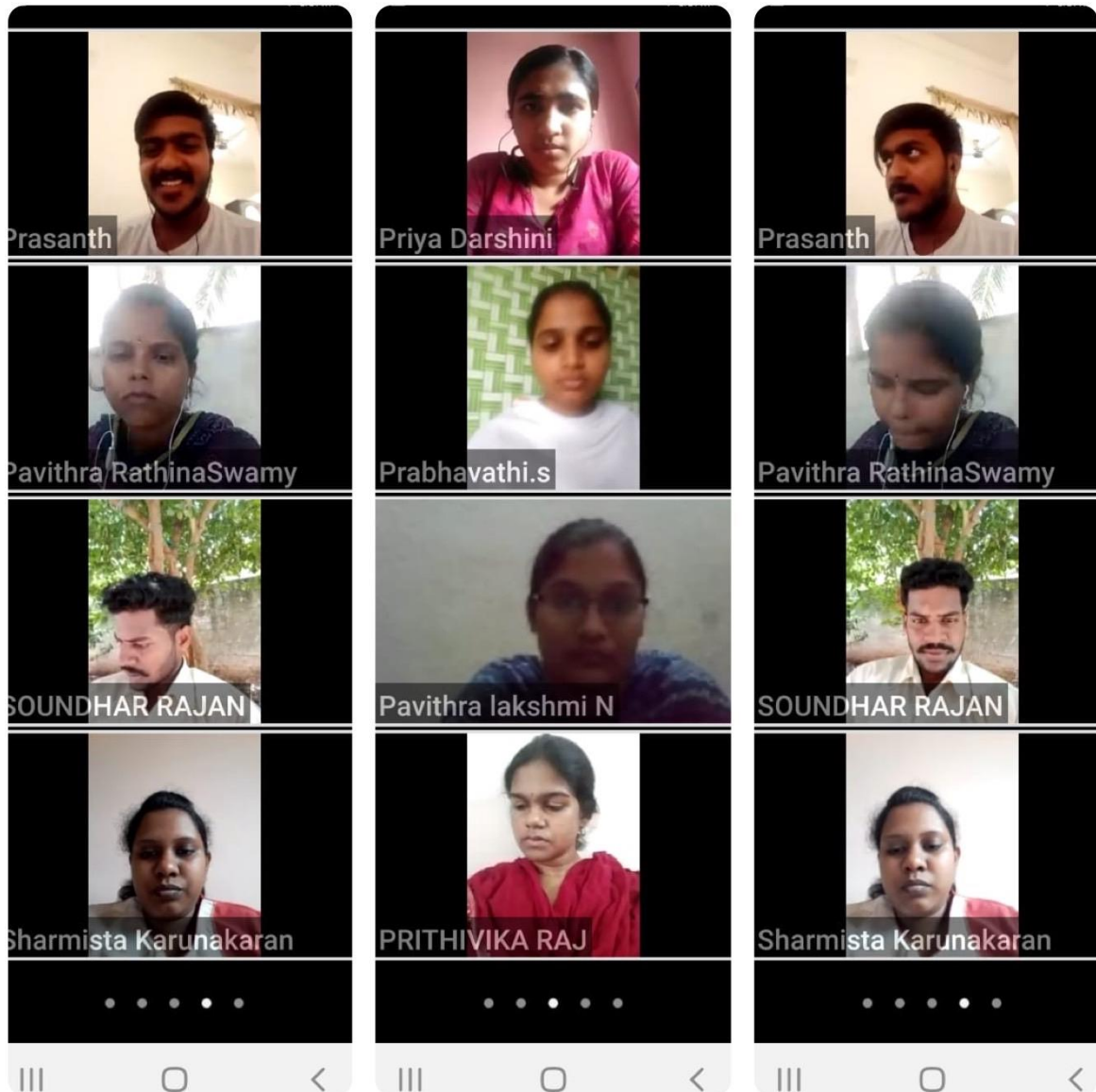
Sumithra

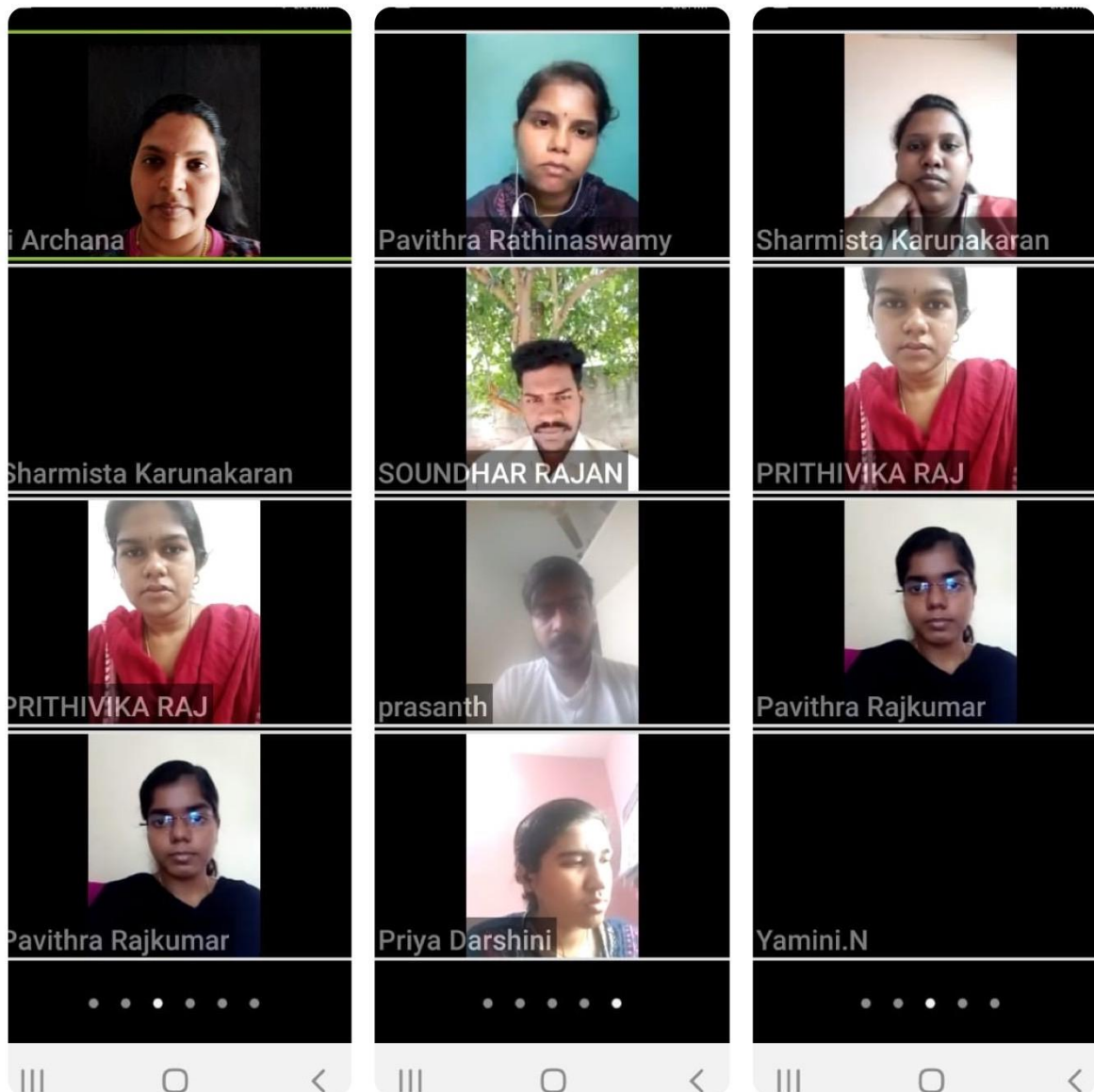


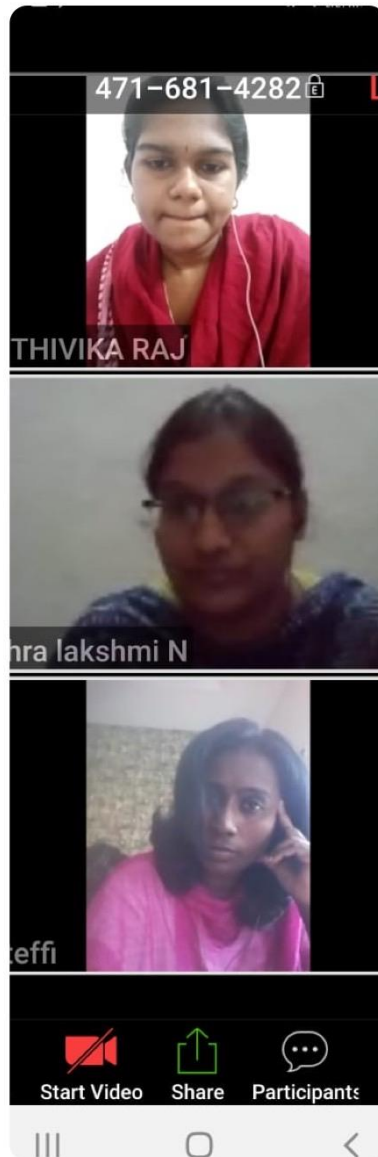
Yamini.N



Pavithra Rajkumar







30.4.2020:

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Discussion with PGs on <i>Corticosteroids in dentistry</i>	PGs, CRRIs, Final year, Third year	11.30-01.00 pm
Clinical case presentation	CRRI, Final year	02 pm -03 pm
NEET Questions discussion	CRRI, Final Year	03 pm- 04 pm

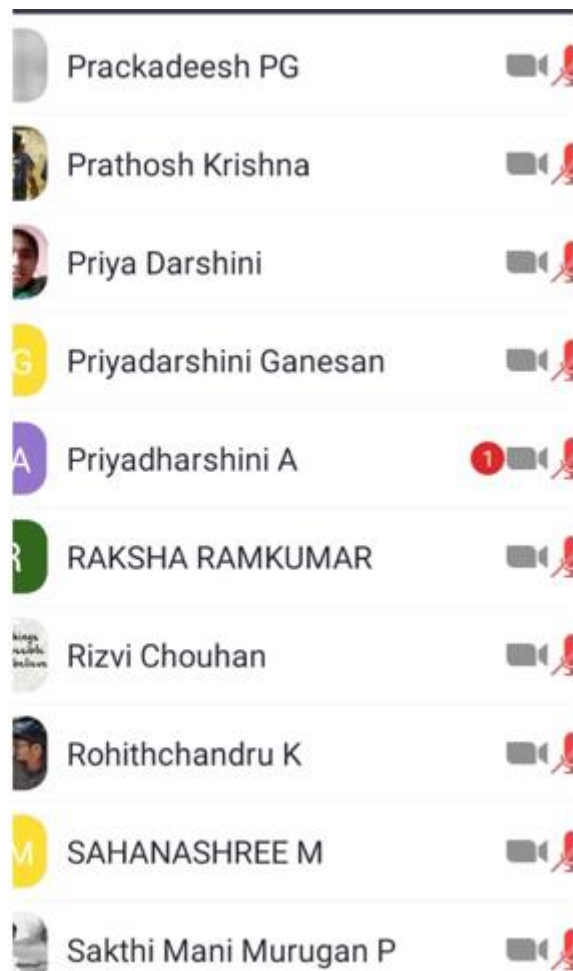
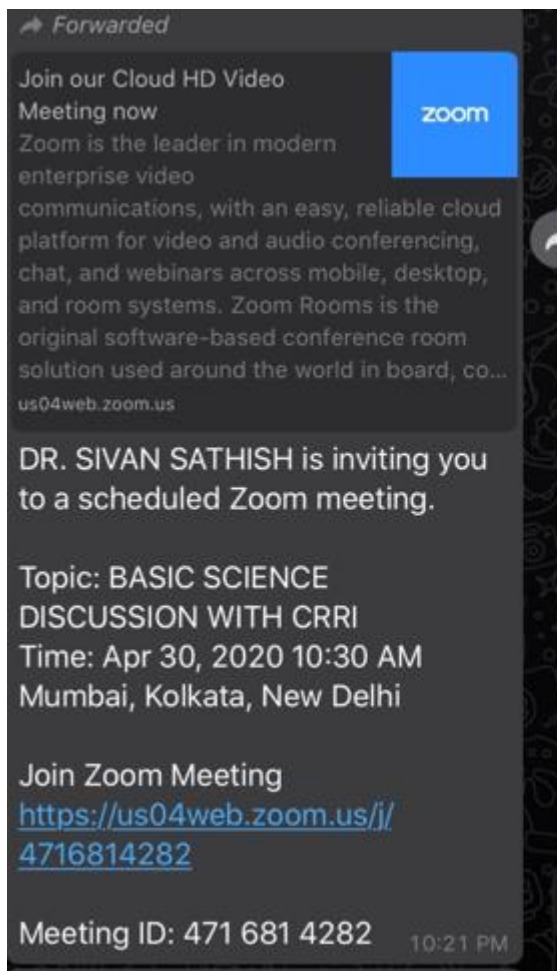
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PICTURES:



Close **Participants**

Search

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- PL Pavithra lakshmi N (f)
- PRITHIVIKA RAJ
- SR SOUNDHAR RAJAN
- NR NIKILESH RAJ
- PR Pavithra Rajkumar
- Pavithra RathinaSw
- P Prabhavathi.s
- Prackadeesh PG

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- PG Priyadarshini Ganes
- PA Priyadarshini A
- R RAKSHA RAMKUMA
- All things are possible if you believe Rizvi Chouhan
- Rohithchandru K
- SM SAHANASHREE M
- Sakthi Mani Muruga
- SK Sharmista Karunaka
- S Sumithra
- Y Yamini.N

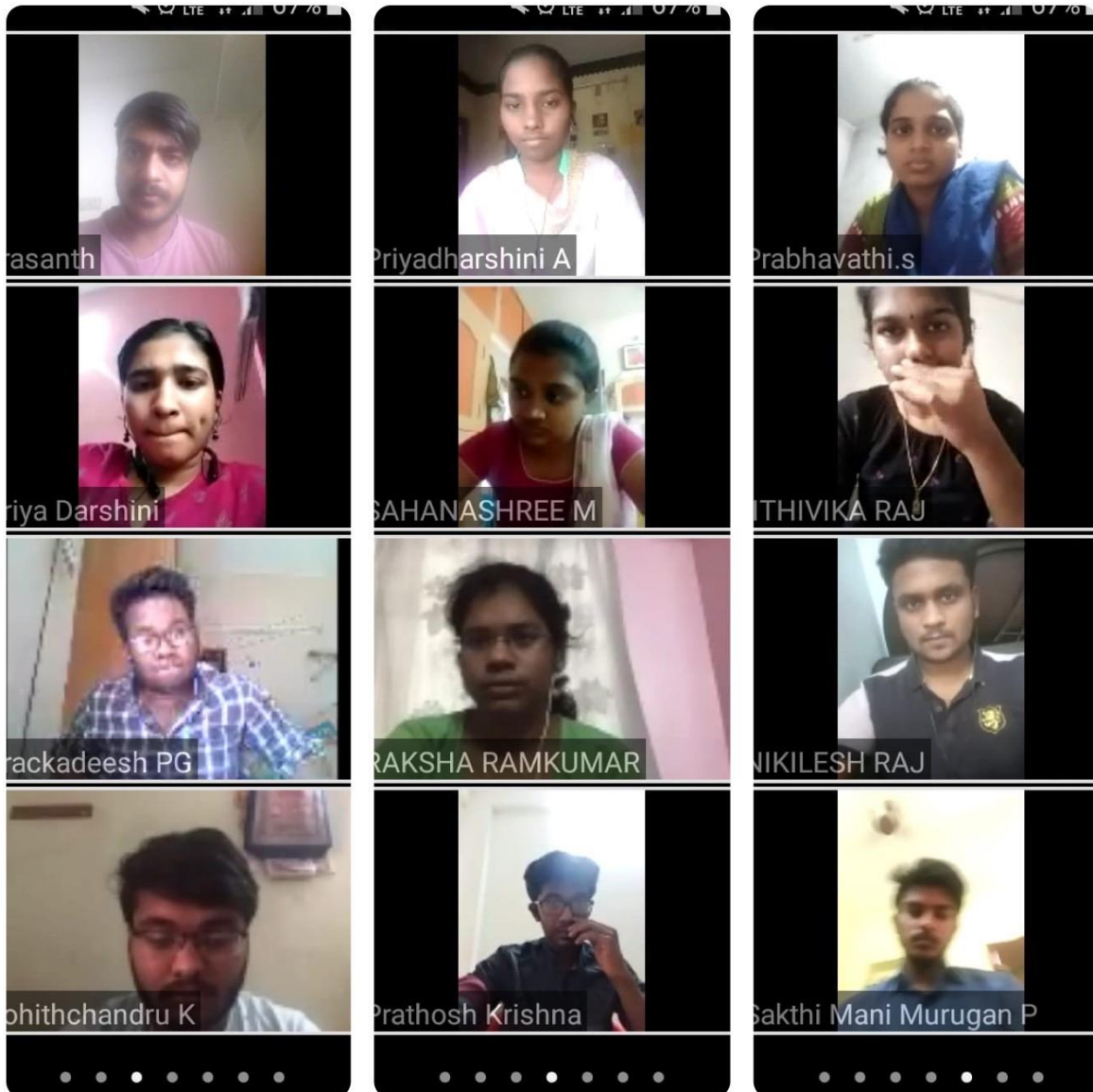
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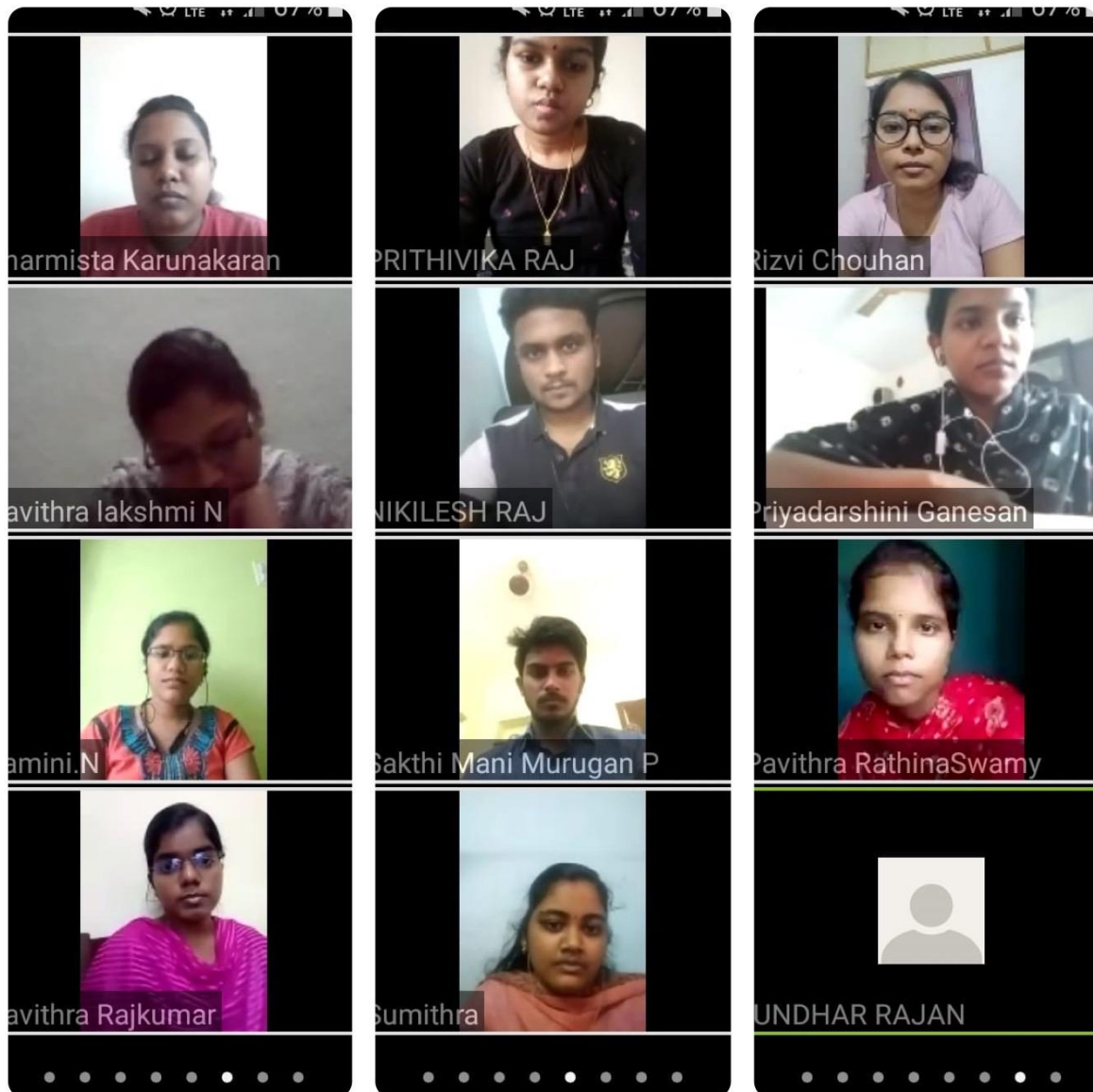
Close **Participant**

Search

- P prasanth (me)
- PL Pavithra lakshmi N (f)
- SS sivan sathish
- SR SOUNDHAR RAJAN
- NR NIKILESH RAJ
- PR Pavithra Rajkumar
- Pavithra RathinaSw
- P Prabhavathi.s
- Prackadeesh PG

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02.05.2020:

**SUPERVISION AND DISCUSSION BY
Dr.SIVAN SATHISH, MDS, MFDS RCPS
PROFESSOR AND HOD, ORAL MEDICINE**



<u>Work done</u>	<u>Participants</u>	<u>Timing</u>
Discussion on NEET Questions	CRRRI	10.30-11.00 am
Clinical case presentation	CRRRI, Final year	11.00-11.30 am
Discussion with PGs on <i>Radiotherapy in oral cancer</i>	PGs, CRRRIs, Final year, Third year	11.30-01.00 pm
Clinical case presentation	CRRRI, Final year	02 pm -03 pm
NEET Questions discussion	CRRRI, Final Year	03 pm- 04 pm

- **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

PICTURES:

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Topic: BASIC SCIENCE
DISCUSSION WITH CRRI
Time: May 2, 2020 10:30 AM
Mumbai, Kolkata, New Delhi











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Meeting ID: 471 681 4282 10:39 AM

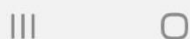
Table 8.1 Distinguishing features of exotoxins and endotoxins

Exotoxins	Endotoxins
1. Protein	Lipopolysaccharides
2. Heat labile	Heat stable
3. Actively secreted by cells; diffuse into surrounding medium	Form part of cell wall; do not diffuse into surrounding medium
4. Readily separable from cultures by physical means such as filtration	Obtained only by cell lysis
5. Action often synergistic	No synergistic action
6. Specific pharmacological effect for each exotoxin	Effect nonspecific; action common to all endotoxins
7. Specific tissue affinity	No specific tissue affinity
8. Active in very minute doses	Active only in very large doses
9. Highly antigenic	Weakly antigenic
10. Action specifically neutralised by antibody	Neutralisation by antibody ineffective

Close Participants (18)











-  Kauvya Karunanidhi
-  Kishan Kumar
-  Kudiarasi Rajendran
-  M.Lakshmipriya
-  Moomina Sattar
-  PRITHIVIKA RAJ
-  Ragavi vijayan
-  Sharmista Karunaka...
-  Sumithra
-  Yamini.N

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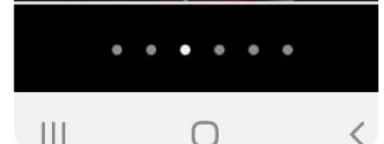
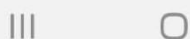


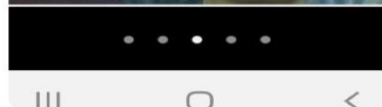
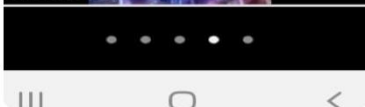
Close Participants (18)

Search

-  Sai Archana (me)
-  Lakshika Sree (host)
-  Rizvi Chouhan
-  sivan sathish
-  SOUNDHAR RAJAN
-  Kadambari Ramasw..
-  Kanchana Mala
-  Kauvya Karunanidhi
-  Kishan Kumar
-  Kudiarasi Rajendran

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Polymorphic nodules	Hepatitis B virus antigens in some cases
Reactive arthritis	Bacterial antigens (e.g., <i>Yersinia</i>)
Serum sickness	Various proteins (e.g., foreign serum proteins and erythrocyte globulin)
Arthus reaction (experimental)	Various foreign proteins

requently, the complexes may be formed at sites where antigen has been "planted" previously (called *in situ* immune complexes). The antigens that form immune complexes may be exogenous, such as a foreign protein that is injected or produced by an infectious microbe, or endogenous, if the individual produces antibody against self antigens (autoimmunity). Examples of immune complex disorders and the antigens involved are listed in Table 5.5. Immune complex-mediated diseases tend to be systemic, but often preferentially involve the kidney (glomerulonephritis), joints (arthritis), and small blood vessels (vasculitis), all of which are common sites of immune complex deposition.

Systemic Immune Complex Disease. Acute serum sickness is a classic example of immune complex disease; administration of foreign serum to a human, e.g., serum from immunized horses used for protection against diphtheria. In modern times, the disease is infrequent and usually seen in individuals who receive antibodies from other individuals or species, e.g., horse or rabbit antihymocytoglobulin administered to deplete T cells in recipients of organ grafts. Nevertheless, it is an informative model that has taught us a great deal about immune complex disorders.

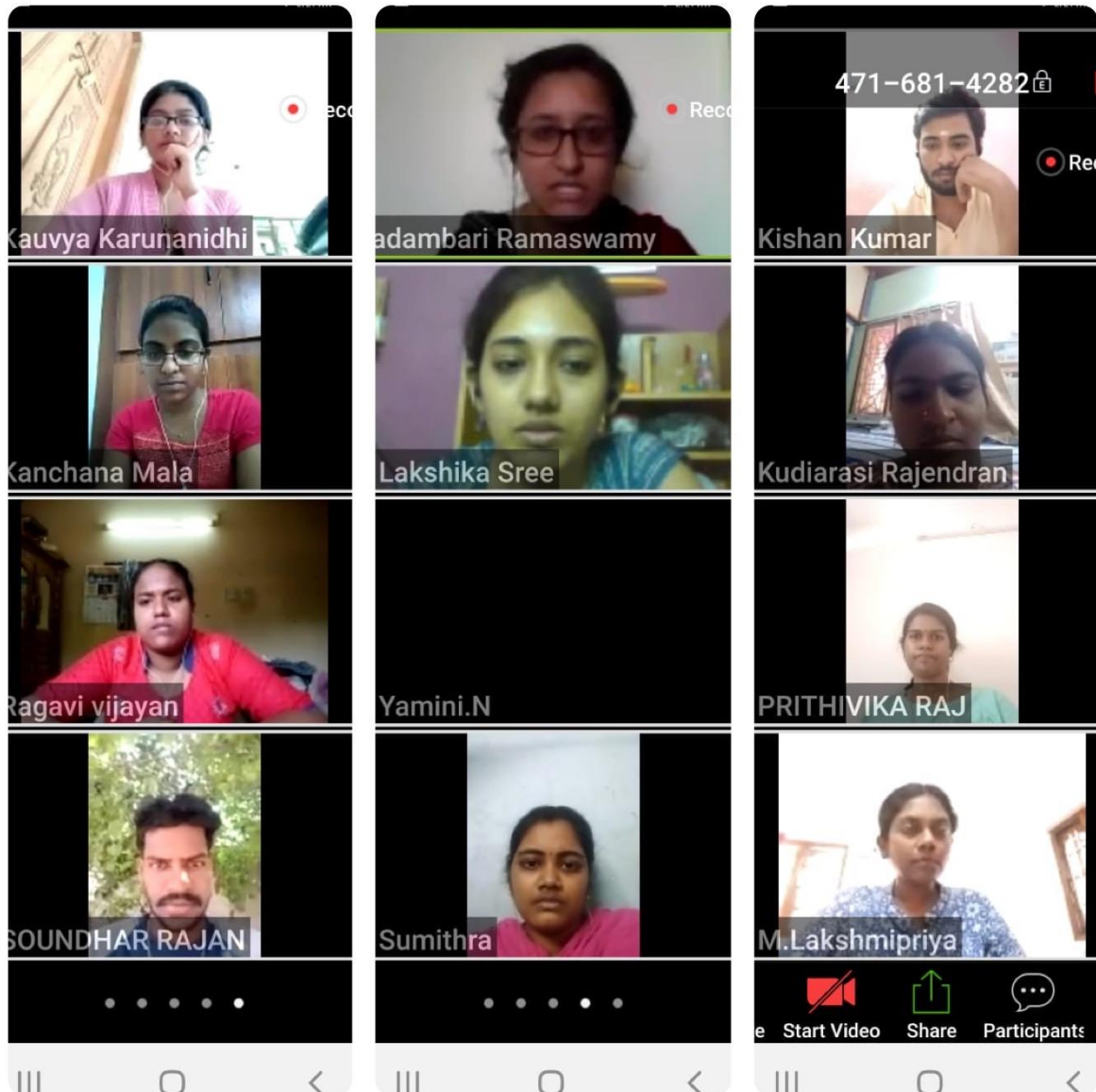
The pathogenesis of systemic immune complex disease can be divided into three phases (Fig. 5.15).

Formation of Immune Complexes. The introduction of a protein antigen triggers an immune response that results in the formation of antibodies, typically about 1 week after the injection of the protein. These antibodies are secreted into the blood, where they react with the antigen still present in the circulation and form antigen-antibody complexes.

Deposition of Immune Complexes. In the next phase, the circulating antigen-antibody complexes are deposited in various tissues. The factors that determine whether immune complex formation will lead to tissue deposition and disease are not fully understood, but the major influences seem to be the characteristics of the complexes and local vascular alterations. In general, complexes of medium size but are formed when antigen is in slight excess are the most pathogenic. Chronic immune blood is filtered at high



Fig. 5.15 Immune complex disease of systemic immune complex-mediated



Close

Participants (18)

Search

SA Sai Archana (me)

Lakshika Sree (host)

C christeffi

KR Kadambari Ramasw..

SS sivan sathish

KM Kanchana Mala

Kanmani Raju

Kauvya Karunanidhi

Kishan Kumar

KR Kudiarasi Rajendran

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Close

Participants (18)

Kishan Kumar

KR Kudiarasi Rajendran

M M.Lakshmipriya

MS Moomina Sattar

PRITHIVIKA RAJ

Ragavi vijayan

Rizvi Chouhan

SR SOUNDHAR RAJAN

S Sumithra

Y Yamini.N

Invite

Close

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