1.1.1: The Institutional ensures effective curriculum planning and delivery through a well-planned and documented process including Academic calendar and conduct of continuous internal Assessment

INDEX

AVANTHI INSTITUTE OF PHARMACEUTICAL SCIENCES
(Approved by PCI, AICTE & Affiliated to JNTUH)
Gunthapally (V), Abdullapurmet (M), R.R. Dist., Near Ramoji Filmcity, Hyderabad - 501 512.

Date: 14.06.2019

CIRCULAR

This is to inform that the below mentioned staff members are appointed as Institutional Academic Committee members for the Academic year 2019-20 to discuss Institutional academic matters.

S NO	NAME OF THE FACULTY	DESIGNATION	SIGNATURE
1	Dr.K. BALAJI ,PRINCIPAL, AIPS	CHAIRPERSON	
2	Dr. Y JAYAPRADHA, DIRECTOR-HR	MEMBER	
3	Dr. NIHAR RANJAN DAS,VICE PRINCIPAL,AIPS IQAC COORDINATOR	MEMBER	
4	Dr. M. RAMAKRISHNA PROFESSOR	MEMBER	
5	B. MANJULA ASSOCIATE PROFESSOR	MEMBER	
6	Dr. BISWAT BISWAL	MEMBER	
7	Dr.ARIFA BEGUM	MEMBER	
8	Dr. M. PADIGYA PATEL		
9	M.RAJASHEKAR,PHYSICAL DIRECTOR	MEMBER	
10	S.SRIDEVI,LIBRARIAN	MEMBER	

PRINCIPAL

Copy to:

1.ALL HODs

2.IQAC Coordinator

PRINCIPAL
Avanthi's Institute of Pharmaceutical Sciences
Gunthapally (V), Hayath Nagar (M),
Ranga Reddy Dist.

INSTITUTIONAL ACADEMIC PLANNING & ADVISORY COMMITTEE

Institutional Academic Planning & Advisory Committee Members For the Academic Year 2019-20

S NO	NAME OF THE FACULTY	DESIGNATION
1	Dr.K. BALAJI ,PRINCIPAL, AIPS	CHAIRPERSON
2	Dr. Y JAYAPRADHA, DIRECTOR-HR	MEMBER
3	Dr. NIHAR RANJAN DAS,VICE PRINCIPAL,AIPS IQAC COORDINATOR	MEMBER
4	Dr. M. RAMAKRISHNA ASSOCIATE PROFESSOR	MEMBER
5	B. MANJULA ASSOCIATE PROFESSOR	MEMBER
6	Dr. BISWAT BISWAL	MEMBER
7	Dr.ARIFA BEGUM	MEMBER
8	Dr. M. PADIGYA PATEL	MEMBER
9	M.RAJASHEKAR,PHYSICAL DIRECTOR	MEMBER
10	S.SRIDEVI, LIBRARIAN	MEMBER

Functions of the Academic Committee:

1. The academic committee is responsible for imbibing the best practices to provide an improved academic system for the present and future students.

2. The committee is also accountable for practices, such as conducting academic award functions to honor students for academic excellence.

3. Propose the academic requirements (Theory, Laboratory and Examination related) of each Department.

4. Scheduling of various academic activities.

5. Review of the academic activities.

6.Perform such other functions as may be assigned by the governing body

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AIPS/AC/2021-2022/01

Date: 18.06.2019

CIRCULAR

This is to inform all the staff members that Institutional Academic Committee will be meeting on 02.06.2019 at 10.00 AM in the Principal's chamber to discuss the following agenda. All members are requested to attend the meeting without fail.

Agenda:

1. Preparation of institute academic calendar of 2019-20
2. Value added courses
3. Hospital training sessions and visits
4. Pharmacological and Analytical Project works
5. Research works and collaboration
6. Workshops/FDPs
7. Industrial visits
8. Training and Placements
9. Sports/NSS activities
10. Any other issues

AVANTHI INSTITUTE OF PHARMACEUTICAL SCIENCES * ABDULLAPURMET *

PRINCIPAL

- PRINCIPAL
Avanthi's Institute of Pharmaceutical Science
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Copy to:

1. All HODS
2. IQAC coordinator
3. All the Committee Members

MINUTES OF THE INSTITUTIONAL ACADEMIC PLANNING & ADVISORY COMMITTEE

The Institutional Academic Committee meeting was held on 02.06.2019 at 10AM in Principal's chamber. The principal welcomed the staff and briefed on the above objective of the Institutional Academic Committee meeting. The principal started the deliberations by discussing the Academic issues and emphasized the need to concentrate on new University regulations.

Agenda Item 1:

Preparation of Institute academic calendar of 2019-20

Resolution:

- Dr. Nihar Ranjan Das IQAC Coordinator, prepared the college Academic Calendar based on the Academic Calendar issues by the University and is handed over to the Head of the Department of Pharmacy.
- Department wise Academic Calendar was prepared by the Head of the Department basing on the Calendar issued by the Coordinator and was sent to the IQAC coordinator for his approval.
- Timetables were prepared and workloads were allotted to the faculty based

Academic

Calendar of the institute as per the curriculum of the current semester.

Agenda Item 2

Value added Courses

Resolution:

Dr. Biswath Biswal, Professor, The member of the committee have been proposed that value added courses should be included in each department though it's not included in the curriculum as it finds important for the development and employability of the students.

Agenda Item 3:

Hospital training sessions and visits.

Resolution:

The members suggested that every student should complete atleast one internship per year.

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Agenda Item 4:

Pharmacological and Analytical Project works

Resolution:

The members of the committee assigned the faculty to guide the students in project

Agenda Item 5:

Research Works

Resolutions

- Dr. Y. Jayapradha, advised the faculty members to Publish at least one research paper per semester in High Indexed Journal. The entire remaining faculty were suggested to publish one paper in Scopus journal.
- B. Manjula advised all the faculty members to attend the FDP every year.
- Dr. M. Padiya patel, advised all the faculty members to undergo Internship Academic Interaction programmes.

Agenda Item 6:

Training and placements

Resolution:

- The Principal, AIPS staff members discussed and took a resolution and informed and the faculty members to implement the following from the academic year.
- Students who cleared all the subjects and secured CGPA above 7 should enroll for GPAT Programme.
- Students who cleared all the subjects and obtained CGPA between 6-7 should enroll for PGECET Programme.
- All the remaining students should attend CRT classes conducted by the college.
- M. Rama Krishna, informed the faculty members to organize various activities in the form of Competitions, Guest lectures, Career guidance, Entrepreneurship programmes etc for the students to improve their knowledge, skills and keep them abreast with the changing demands of the industries.

Agenda Item 7:

Workshops/FDPS

Resolution: Dr. Y. Jayapradha , suggested the faculty to attend the FDP every year.

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- She suggested the importance of proving training programmes to non-teaching staff in Ms Office, Ms Word and Excel which are very useful in drafting and for preparing documents.
- She also advised the English faculty to train the junior faculty and nonteaching staff to compose emails, notices, official letters, circulars which are necessary for the needs of their job and also for the professional development of the institution

Agenda Item 8:

Industrial Visits

Resolution:

- Dr. Arifa begum proposed an idea of organizing regular industrial visits for the students *Inreputed industries like Pfizer, Aurabindo*
- To acquire knowledge on the working of men and machinery in different pharmacy

Industries

- Dr. Nihar Ranjan Das suggested for arranging at least two guest lecturers to students in a semester.

Agenda Item 9:

Sports/NSS Activities

Resolution:

- M. Rajashekar proposed organizing Sports activities for the students and encourages the students to participate in competitions at the university, state or national level Tournaments
- He also informed the faculty members to conduct various technical events and NSS activities like Blood donation camps, Plantation drive, Swacch Bharat Campaign, Health check-up programs etc.

Agenda Item 10

Any other Issues Resolution:

Resolution

- Dr. Nihar Ranjan Das, the IQAC coordinator instructed all the staff members to maintain updated stock registers, Maintenance registers, Complaint registers etc of all the laboratories duly verified by the committee.
- It was also resolved after the discussion and should follow IQAC Audit Action Taken Report.

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Avanthi's Institute of Pharmaceutical Sciences
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Ranga Reddy Dist.

List of Academic Planning & Adivisory Committee Members attended

S.no	Name of the faculty	Designation	Signature
1	Dr.K. BALAJI ,PRINCIPAL, AIPS	CHAIRPERSON	
2	Dr. Y JAYAPRADHA, DIRECTOR-HR	MEMBER	
3	Dr. NIHAR RANJAN DAS,VICE PRINCIPAL,AIPS IQAC COORDINATOR	MEMBER	
4	Dr. M. RAMAKRISHNA PROFESSOR	MEMBER	
5	B. MANJULA ASSOCIATE PROFESSOR	MEMBER	
6	Dr. BISWAT BISWAL	MEMBER	
7	Dr.ARIFA BEGUM	MEMBER	
8	Dr. M. PADIGYA PATEL	MEMBER	
9	M.RAJASHEKAR,PHYSICAL DIRECTOR	MEMBER	
10	S.SRIDEVI, LIBRARIAN	MEMBER	

PRINCIPAL

AVANTHI INSTITUTE OF PHARMACEUTICAL SCIENCES * ABDULLAPURMET *

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Date: 05.07.2019

DEPARTMENT OF PHARMACY

CIRCULAR

This is to inform that the Department Academic Committee (DAC) will be held on 10.07.2019 10:30AM at Principal Sir's chamber

Agenda:

1. Preparation of Department progress for the academic year 2019-20
2. Value added courses related to medical coding, Clinical SAS
3. Certificate courses/ Internship programs on Instrumentation handling
4. Project works on Pharmacological activities and Analytical designs
5. Research works on Plant extracts and their Pharmacological action
6. Training and Placements with respect to Multinational Pharmaceutical Industry needs
7. Industrial visits to formulation Pharmaceutical Industries
8. Extracurricular/ Co-curricular activities
9. Sports/NSS activities
10. Any other issues

PRINCIPAL

Copy to:

1. All HODS
2. IQAC coordinator
3. All the Committee Members

AVANTHI INSTITUTE OF PHARMACEUTICAL SCIENCES * ABDULLAPURMET *

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MINUTES OF THE INSTITUTIONAL ACADEMIC PLANNING & ADVISORY COMMITTEE

The Institutional Academic Committee meeting was held on 02.07.2019 at 10AM in Principal's chamber. The principal welcomed the staff and briefed on the above objective of the institutional Academic Committee meeting. The principal started the deliberations by discussing the Academic Issues and emphasized the need to concentrate on new University regulations.

Agenda Item 1:

Preparation of Department progress for the academic year 2019-20

Resolution:

Dr. M. Rama Krishna, HOD Pharmacy Analysed the results of B.Pharmacy 2019-20 academic year and expressed satisfaction for getting more than 86% of pass percentage Committee congratulated the faculty who met the target of 91% or more.

Agenda Item 2:

Value added Courses related to medical coding, Clinical SAS

Resolution:

The members of the committee have been proposed that value added courses related to medical coding, medical scribing and clinical SAS related to be included in each department though it's not included in the curriculum as it finds important for the development and employability of the B. Pharmacy

The members of the committee have been proposed that value added courses related to Quality Assurance and Quality control, Pharmaceutical technology and Pharmacological Assays should be included in each department though its not included in the curriculum as it finds important for the development and employability of the M.Pharmacy students.

Agenda Item 3:

Certificate courses/Internship programs on Instrumentation handling

Resolution:

- The members suggested that every B. Pharmacy students should complete certification courses/Internship courses related to latest instrumentation handling, thesis writing courses.

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Agenda Item 4:

Project works on Pharmacological activities and Analytical designs

Resolution:

- The members of the committee assigned the faculty to guide the B.Pharmacy students in project works related to plant extracts and pharmacological activities, pharmaceutics related projects and analytical projects.
- The members of the committee assigned the faculty to guide the students to perform real time projects related to drug design and drug development

Agenda Item 5:

Research works on Plant Extracts and their Pharmacological

action

Resolution:

- Dr. K. Balaji, Principal adviced the faculty members to publish atleast one research Paper per semester in High Indexed Journal. The entire remaining faculty were

Suggested

to publish one paper in Scopus journal.

- Dr. K. Nagaraju Professor advised all the faculty members to attend the FDP programs every year.

Agenda Item 6:

Training and placements with respect to Multinational Pharmaceutical Industry needs

Resolution:

- The Principal, AIPS staff members discussed and took a resolution and informedand the faculty members to implement the following from the academic year:
- Students who cleared all the subjects and secured CGPA above 7 should enroll for GPAT Programme Students who cleared all the subjects and obtained CGPA between 6-7 should
- All the remaining students should attend CRT classes conducted by the college.
- Dr. M. Rama Krishna, informed the faculty members to organize various activities in the form of Competitions, Guest lectures, Career guidance, Entrepreneurship programmes etc

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for the students to improve their knowledge, skills and keep them abreast with the changing demands of the industries.

Agenda Item 7:

Industrial Visits to formulation Pharmaceutical Industries

Resolution:

- Dr. G. SAIKIRAN proposed an idea of organizing regular industrial visits for the students in reputed multinational Pharmacy industries like Pfizer, Aurabindo, Dr. Reddys Laboratories, DIVIS Laboratories.
- To acquire knowledge on the working of men and machinery in different pharma industries.
- P. Lavanya suggested for arranging at least two guest lecturers to students in a Semester.

Agenda Item 8:

Sports/NSS Activities

Resolution:

- M. Rajashekar proposed organizing Sports activities for the students and encourages the students to participate in competitions at the university, state or national level tournaments.
- He also informed the faculty members to conduct various technical events and NSS He activities like Blood donation camps. Plantation drive, Swacch Bharat Campaign, Health check-up programs etc.,

Agenda Item 9:

Any other Issues

Resolution:

- The IQAC coordinator instructed all the staff members to maintain updated stock registers, Maintenance registers, Complaint registers etc of all the laboratories duly verified by the committee.
- It was also resolved after the discussion and should follow IQAC Audit Action Taken Report

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Avanthi's Institute of Pharmaceutical Sciences
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AVANTHI INSTITUTE OF PHARMACEUTICAL SCIENCES ABDULLAPURMET

List of DAC Members attended

s.no	Name of the faculty	Designation	Signature
1	Dr.K. BALAJI ,PRINCIPAL, AIPS	CHAIR PERSON	
2	Dr. Y JAYAPRADHA, DIRECTOR-HR	MEMBER	
3	Dr. NIHAR RANJAN DAS,VICE PRINCIPAL,AIPS IQAC COORDINATOR	MEMBER	
4	Dr. M. RAMAKRISHNA PROFESSOR	MEMBER	
5	B. MANJULA ASSOCIATE PROFESSOR	MEMBER	
6	Dr. BISWAT BISWAL	MEMBER	
7	Dr.ARIFA BEGUM	MEMBER	
8	Dr. M. PADIGYA PATEL	MEMBER	
9	M.RAJASHEKAR,PHYSICAL DIRECTOR	MEMBER	

HOD

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AVANTHI INSTITUTE OF PHARMACEUTICAL SCIENCES * ABDULLAPURMET *

DEPARTMENT OF PHARMACY PRACTICE

CIRCULAR

Date: 01.06.2019

This is to inform that the Department Academic Committee (DAC) will be held on 06.06.2019 10:30AM at Principal Sir's chamber.

Agenda:

1. Preparation of department progress for the academic year 2019-20
2. Hospital training and Hospital visits
3. Clinical Project works
4. Community centers correlated training
5. Placement in Pharma - IT Sector Companies.
6. Value added courses
7. Research works

8 Sports/NSS activities

9. Any other issues

PRINCIPAL

- PRINCIPAL
Avanthi's Institute of Pharmaceutical Sciences
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AVANTHI INSTITUTE OF PHARMACEUTICAL SCIENCES * ABDULLAPURMET *

Copy to:

1.All HODS

2.IQAC coordinator

3. All the Committee Members

MINUTES OF THE INSTITUTIONAL ACADEMIC PLANNING & ADVISORY COMMITTEE

The Institutional Academic Committee meeting was held on 06.06.2019 at 10AM in Principal's chamber. The principal welcomed the staff and briefed on the above objective of the Institutional Academic Committee meeting. The principal started the deliberations by discussing the Academic issues and emphasized the need to concentrate on new University regulations.

Agenda Item 1:

Preparation of Department progress for the academic year 2019-20

Resolution:

- Dr. B.Manjula, HOD Pharmacy Practice analysed the results of Pharm.D 2019-2020 academic year and expressed satisfaction for getting more than 85% of pass percentage. Committee congratulated the faculty who met the target of 90% or more.

Agenda Item 2:

Hospital training and Hospital visits

Resolution:

- Dr. P. Swathi suggested faculty to train the students to participate in bed side learning.
- Dr. Ravi prakash proposed an idea of organizing regular hospital visits for the students in reputed hospitals like Global hospital, Gandhi hospital

Agenda Item 3:

Clinical Project works:

Resolution:

The members suggested that every student should complete atleast one clinical project which includes both cases and controls:

Agenda Item 4

Community centers centers correlated training

Resolution:

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The members of the committee assigned the Pharmacy practice faculty to The members of the committee assigned the Pharmacy practice faculty to guide the students to participate in community center correlated training such as B.P monitoring, Glucose monitoring

Agenda Item 5:

Placement in Pharma - IT Sector Companies:

Resolution:

- The Principal, AIPS staff members discussed and took a resolution and informed and the faculty members to implement the following from the academic year.
- Students should attend CRT classes conducted by the college.
- Dr. Md Abdul Azeem informed the faculty members to organize various activities in the form of Competitions, Guest lectures, Career guidance, Entrepreneurship programmes etc for the students to improve their knowledge, skills and keep them abreast with the changing demands of the industries.

Agenda Item 6:

Value added courses

Resolution:

The members of the committee have been proposed that value added courses related to clini-SAP, clinical research, Pharmacovigilance should be included in each department though its not included in the curriculum as it finds important for the development and employability of the students.

Agenda Item 7:

Rearch works

Resolution:

- Dr. B.Manjula advised the faculty members to publish atleast one research paper per semester in High Indexed Journal. The entire remaining faculty were suggested to publish one paper in
  Scopus journal

Agenda Item 8:

Sports/NSS activities

Resolution:

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- M.Rajashekar proposed organizing Sports activities for the students and encourages the students to participate in competitions at the university, state or national level tournaments.
- MD Abdul Azeem also informed the faculty members to conduct various technical events and NSS activities like Blood donation camps, Plantation drive, Swacch Bharat Campaign, Health check-up programs etc.

Agenda Item 9:

Any other Issues

Resolution:

- Dr. Nihar Ranjan Das The IQAC coordinator instructed all the staff members to maintain *updated stock registers, Maintainance registers, and Complaint registers of all the* laboratories duly verified by the committee.
- It was also resolved after the discussion and should follow IQAC Audit Action Taken Report

PRINCIPAL
Avanthi's Institute of Pharmaceutical Sciences
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Ranga Reddy Dist.

AVANTHI INSTITUTE OF PHARMACEUTICAL SCIENCES * ABDULLAPURMET *

List Of DAC Members attended

S.no	Name of the faculty	Designation	Signature
1	Dr.K. BALAJI ,PRINCIPAL,AIPS	CHAIR PERSON	
2	Dr.Y.JAYAPRADHA,DIRECTOR-H.R	MEMBER	
3	Dr.NIHAR RANJAN DAS,VICE PRINCIPAL,AIPS IQAC COORDINATOR	MEMBER	
4	B.MANJULA ASSOCIATE PROFESSOR	MEMBER	Manjula.B
5	MD. ABDUL AZEEM, ASSOCIATE PROFESSOR	MEMBER	
6	P. SWATHI PATEL, ASSISTANT PROFESSOR	MEMBER	P. Swathi
7	Dr. RAVIPRAKASH, ASSISTANT PROFESSOR	MEMBER	
8	M.RAJASHEKAR,PHYSICAL DIRECTOR	MEMBER	Rajashekar
9	S.SRIDEVI,LIBRARIAN	MEMBER	

HOD

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PRINCIPAL

- PRINCIPAL
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JAWAHARLAL NEHRU TECHNOLOGICAL UNIVERSITY HYDERABAD
REVISED ACADEMIC CALENDAR (2019-20)
M.Tech. / M.Pharm. I Year - I & II Semesters

M.Tech. / M.Pharm. I Year - I Semester

S. No	EVENT	DATE	Duration
1	Commencement of Instruction	26th Aug. 2019	--
2	Dussehra recess	7th to 19th Oct. 2019	2 weeks
3	First Mid Term Examinations	31st Oct. to 2nd Nov. 2019	--
4	Submission of First Mid Term Exam Marks to University on or before	8th Nov. 2019	--
5	Parent-Teacher Meeting	9th Nov. 2019	--
6	Last date of Instruction	24th Dec. 2019	--
7	Second Mid Term Examinations	27th to 30th Dec. 2019	16 weeks
8	Preparation Holidays and Practical Examinations	31st Dec. 2019 to 7th Jan 2020	1 week
9	Submission of Second Mid Term Exam Marks to University on or before	7th Jan. 2020	--
10	End Semester / Supplementary Examinations	8th to 25th Jan. 2020	2 weeks

M.Tech. / M.Pharm. I Year - II Semester

S. No	EVENT	DATE	Duration
1	Commencement of Instruction	27th Jan. 2020	--
2	First Mid Term Examinations	19th to 21st March 2020	--
3	Submission of First Mid Term Exam Marks to University on or before	28th March 2020	--
4	Parent-Teacher Meeting	11th April 2020	--
5	Last date of Instruction	13th May 2020	--
6	Second Mid Term Examinations	14th to 16th May 2020	16 weeks
7	Practical Examinations	18th to 20th May 2020	--
8	Submission of Second Mid Term Exam Marks to University on or before	20th May 2020	--
9	Summer Vacation	21st May to 30th June 2020	6 weeks
10	End Semester / Supplementary Examinations	1st to 15th July 2020	2 weeks

AVANTHI INSTITUTE OF PHARMACEUTICAL SCIENCES * ABDULLAPURMET *

23.10.19

DIRECTOR
ACADEMIC & PLANNING, JNTUH

PRINCIPAL
Avanthi's Institute of Pharmaceutical Sciences
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JAWAHARLAL NEHRU TECHNOLOGICAL UNIVERSITY HYDERABAD

ACADEMIC CALENDAR (2019-20)

FOR NON-AUTONOMOUS CONSTITUENT & AFFILIATED COLLEGES

M.TECH./M.PHARMACY II YEAR - I & II SEMESTER

M.Tech./M. Pharmacy II Year - I Semester

S. No	EVENT	DATE	Duration
1.	Commencement of III Semester	15th July 2019	--
2.	Preparation of Project Work Proposals	10th Aug. 2019	4 weeks
3.	Project Work Review–I, Project approval (Part-I commencement)	13th to 19th Aug. 2019	--
4.	Last date for submission of list of approved students	20th Aug. 2019	--
5.	Comprehensive Viva-Voce	21st Aug. to 25th Oct. 2019	--
6.	Dussehra recess	7th to 12th Oct. 2019	1 week
7.	Last date for submission of Comprehensive Viva-Voce Marks	28th Oct. 2019	--
8.	Project Work Review -II (Phase-I)	11th to 14th Dec. 2019	--
9.	# Project Work Review -II(Phase-II)	27th to 30th Dec. 2019	--
10.	Last date for submission of PRC-II marks	2nd Jan. 2020	--
11.	**Part-I Duration**	13th Aug. to 14th Dec. 2019	**18 weeks**

M.Tech./M.Pharmacy II Year - II Semester

S. No	EVENT	DATE	Duration
1.	Commencement of IV Semester (Project Work Continuation)	16th Dec. 2019	--
2.	Project Work Review -III (Phase –I)	12th to 16th May 2020	--
3.	Last date for submission of Project Work Review-III (Phase-I) Marks	20th May 2020	--
4.	* Date of eligibility of thesis submission	20th May 2020	--
5.	Submission of Thesis and Project Viva –Voce Examination (Phase-I) follows	---	--
6.	**Part-II Duration**	16th Dec. 2019 to 16th May 2020	**22 weeks**
7.	# Project Work Review – III (Phase –II)	19th to 23rd Aug. 2020	--
8.	Last date for submission of Project Work Review –III (Phase-II) Marks	26th Aug. 2020	--
9.	Submission of Thesis and Project Viva –Voce Examination (Phase-II) follows	---	--

* After completion of 40 weeks from the date of approval of project work proposal and subject to approval of Project Work Review-III.

\# Phase-II will be conducted only for unsuccessful students in Phase –I

Note: 1 The unsuccessful students in Project Work Review-II (Phase-II) shall appear for Project Work Review-II at the time of Project Work Review-III. These students shall reappear for Project Work Review-III in the next academic year at the time of Project Work Review -II only after completion of Project Work Review -II, and then Project Work Review -III follows.

2 The unsuccessful students in Project Work Review -III (Phase-II) shall reappear for Project Work Review -III in the next academic year at the time of Project Work Review –II only.

3 The Project Viva-Voce External examination Marks must be submitted on the day of examination to the University.

DIRECTOR
ACADEMIC & PLANNING, JNTUH

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JAWAHARLAL NEHRU TECHNOLOGICAL UNIVERSITY HYDERABAD
REVISED ACADEMIC CALENDAR (2019-20)
B. Pharmacy I Year I & II Semesters

I Sem

S. No	EVENT	DATE	Duration
1	Commencement of Instruction	26th Aug. 2019	--
2	Dussehra recess	7th to 19th Oct. 2019	2 weeks
3	First Mid Term Examinations	31st Oct. to 2nd Nov. 2019	--
4	Submission of First Mid Term Exam Marks to University on or before	8th Nov. 2019	--
5	Parent-Teacher Meeting	9th Nov. 2019	--
6	Last date of Instruction	24th Dec. 2019	--
7	Second Mid Term Examinations	27th to 30th Dec. 2019	16 weeks
8	Preparation Holidays and Practical Examinations	31st Dec. 2019 to 7thJan 2020	1 week
9	Submission of Second Mid Term Exam Marks to University on or before	7th Jan. 2020	--
10	End Semester / Supplementary Examinations	8th to 25th Jan. 2020	2 weeks

II Sem

S. No	EVENT	DATE	Duration
1	Commencement of Instruction	27th Jan. 2020	--
2	First Mid Term Examinations	19th to 21st March 2020	--
3	Submission of First Mid Term Exam Marks to University on or before	28th March 2020	--
4	Parent-Teacher Meeting	11th April 2020	--
5	Last date of Instruction	13th May 2020	--
6	Second Mid Term Examinations	14th to 16th May 2020	16 weeks
7	Preparation Holidays and Practical Examinations	18th to 23rd May 2020	--
8	Submission of Second Mid Term Exam Marks to University on or before	23rd May 2020	--
9	End Semester / Supplementary Examinations	25th May to 6th June 2020	2 weeks
10	Summer Vacation	8th June to 4th July 2020	4 weeks

23.10.2019

DIRECTOR
ACADEMIC & PLANNING, JNTUH

AVANTHI INSTITUTE OF PHARMACEUTICAL SCIENCES * ABDULLAPURMET *

PRINCIPAL
Avanthi's Institute of Pharmaceutical Sciences
Gunthapally (V), Hayath Nagar (M),
Ranga Reddy Dist.

JAWAHARLAL NEHRU TECHNOLOGICAL UNIVERSITY HYDERABAD

REVISED ACADEMIC CALENDAR (2019-20)

FOR NON-AUTONOMOUS CONSTITUENT& AFFILIATED COLLEGES

B. TECH./B.PHARM. II, III & IV YEARS I & II SEMESTERS

I SEM

S. No	EVENT	DATE	Duration
1	Commencement of Instruction	15th July 2019	--
2	First Mid Term Examinations	12th to 14th Sept. 2019	--
3	Submission of First Mid Term Exam Marks to University on or before	20th Sept. 2019	--
4	Parent-Teacher Meeting	21st Sept. 2019	--
5	Dussehra recess	7th to 19th Oct. 2019	2 weeks
6	Last date of Instruction	20th Nov. 2019	17 weeks
7	Second Mid Term Examinations	21st to 23rd Nov. 2019	--
8	Preparation Holidays and Practical Examinations	25th to 30th Nov. 2019	1 week
9	Submission of Second Mid Term Exam Marks to University on or before	30th Nov. 2019	--
10	End Semester Examinations	2nd to 14th Dec. 2019	2 weeks

II SEM

S. No	EVENT	DATE	Duration
1	Commencement of Instruction	16th Dec. 2019	--
2	First Mid Term Examinations	10th to 12th Feb. 2020	--
3	Submission of First Mid Term Exam Marks to University on or before	19th Feb. 2020	--
4	Parent-Teacher Meeting	14th March 2020	--
5	Last date of Instruction	7th April 2020	16 weeks
6	Second Mid Term Examinations	8th to 11th April 2020	--
7	Preparation Holidays and Practical Examinations	13th to 18th April 2020	1 week
8	Submission of Second Mid Term Exam Marks to University on or before	18th April 2020	--
9	End Semester Examinations	20th April to 2nd May 2020	2 weeks
10	Summer Vacation	4th May to 4th July 2020	9 weeks

21.10.19

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AVANTHI INSTITUTE OF PHARMACEUTICAL SCIENCES * ABDULLAPURMET *

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JAWAHARLAL NEHRU TECHNOLOGICAL UNIVERSITY HYDERABAD

REVISED ACADEMIC CALENDAR (2019-20)

PHARM.D (Regular) and (PB) I YEAR

PHARM. D (Regular) and (Post Baccalaureate) I YEAR

Description	Period	Duration
Commencement of instruction	26th Aug. 2019	--
Dussehra Recess	7th to 19th oct. 2019	(2 weeks)
First mid examinations	18th to 23rd Nov. 2019	(1 week)
Submission of First Mid Term Exam Marks to University on or before	30th Nov. 2019	--
Parent-Teacher Meeting	14th Dec. 2019	--
Second mid examinations	10th to 15th Feb. 2020	(1 week)
Submission of Second Mid Term Exam Marks to University on or before	22nd Feb. 2020	--
Parent-Teacher Meeting	14th March 2020	--
Last date of Instruction	2nd May 2020	(32 weeks)
Third mid examinations	3rd to 9th May 2020	(1 week)
Preparation and Practical Examinations	11th to 23rd May 2020	(2 weeks)
Submission of Third Mid Term Exam Marks to University on or before	16th May 2020	--
End / Supplementary Examinations	25th to 6th June 2020	(2 w)
Summer vacation	8th June to 4th July 2020	(4 w)

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JAWAHARLAL NEHRU TECHNOLOGICAL UNIVERSITY HYDERABAD

REVISED ACADEMIC CALENDAR (2019-20)

PHARM. D (Regular) II, III, IV, V, VI YEARS and PHARM.D (PB) II YEAR

PHARM. D (Regular) II, III, IV, V YEAR and PHARM.D (PB) II YEAR

Description	Period	Duration
Commencement of instruction	1st July 2019	--
First mid examinations	16th to 21st Sept. 2019	(1 week)
Submission of First Mid Term Exam Marks to University on or before	30th Sept. 2019	--
Dussehra Recess	7th to 19th Oct.2019	(2 weeks)
Parent-Teacher Meeting	9th Nov. 2019	--
Supplementary Examinations	2nd to 8th Nov. 2019	(1 week)
Second mid examinations	6th to 11th Jan. 2020	(1 week)
Submission of Second Mid Term Exam Marks to University on or before	18th Jan. 2020	--
Parent-Teacher Meeting	8th Feb. 2020	--
Last date of Instruction	28th Mar. 2020	(34 weeks)
Third mid examinations	30th Mar. to 4th April 2020	(1 week)
Submission of Third Mid Term Exam Marks to University on or before	13th April 2020	--
Preparation and Practical Examinations	6th to 18th April 2020	(2 weeks)
End / Supplementary Examinations	20th April to 2nd May 2020	(2 weeks)
Summer vacation	4th April to 4th July 2020	(9 weeks)

PHARM. D (Regular) VI YEAR and PHARM.D (PB) III YEAR

Description	Period	Duration
Commencement of internship in general ward	1st July to 28th Dec. 2019	(6 months)
Report submission of internship in general ward	30th Dec. 2019	--
Commencement of internship in Specialty ward -1	31st Dec. 2019 to 29th Feb. 2020	(2 months)
Report submission of internship in Specialty ward -1	2nd Mar. 2020	--
Commencement of internship in Specialty ward – 2	3rd Mar. to 2nd May 2020	(2 months)
Report submission of internship in Specialty ward–2	4th May 2020	--
Commencement of internship in Specialty ward – 3	5th May to 4th July 2020	(2 months)
Report submission of internship in Specialty ward - 3	6th July 2020	--
Final viva of internship	8th July 2020	--

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INSTITUIONAL ACADEMIC CALENDER

ACADEMIC YEAR 2019-2020

S.NO	DATE	NAME OF THE EVENT
JUNE	12.06.2019	ACADEMIC COMMITTEE MEETING
	22.06.2019	INSTITUIONAL ACADEMIC COMMITEE MEETING
	26.06.2019	DEPARTMENT OF PHARMACY ACADEMIC COMMITEE MEETING
JULY	01.07.2019	COMMENCEMENT OF INSTRUCTIONS OF pharm D 2^{ND} ,3^{RD} ,4^{TH} & 5^{TH} YEARS
	01.07.2019-28.12.2019	COMMENCEMENT OF INTERNSHIP IN GENERAL WARD OF PHARM D 3^{RD} & 6^{TH} YEARS
	10.07.2019	DEPARTMENT OF PHARMACY ACADEMIC COMMITEE MEETING
	13.07.2019	SAY NO TO PLASTIC
	15.07.2019	COMMENCEMENT OF INSTRUCTIONS OF I SEMESTER OF 2^{ND} ,3^{RD} & 4^{TH} YEARS
	15.07.2019	COMMENCEMENT OF INSTRUCTIONS OF III SEMESTER FOR M PHARM 2^{ND} YEAR
	24.07.2019	GOVERNING BODY MEETING
	29.07.2019	BONALU
AUG	12.08.2019	BAKRID
	10.08.2019	Preparation of Project Work Proposals FOR M PHARM 2^{ND} YEAR 1^{ST} SEM
	13.08.2019	Project Work Review-I, Project approval (Part-1 commencement) FOR M PHARM II YEAR 1^{ST} SEM
	13.08.2019	Part-1 Duration FOR MPHARM
	15.08.2019	SPORTS AND CULTURAL ACTIVITIES ON THE OCCASION OF INDEPENDENCE DAY
	18.08.2019	WORLD HUMANITARIAN DAY

	20.08.2019	Last date for submission of list of approved students FOR M PHARM II YEAR 1ST SEM
	21.08.2019	Comprehensive Viva-Voce FOR M PHARM FOR 2ND YEAR
	24.08.2019	SRI KRISHNA ASTAMI
	26.08.2019	GENDER EQUALITY
	26.08.2019	COMMENCEMENT OF INSTRUCTION FOR M PHARM I YEAR 1ST SEMESTER
	26.08.2019	COMMENCEMENT OF INSTUCTIONS OF I SEMESTER FOR B PHARM 1ST YEAR
	26.08.2019	COMMENCEMENT OF INSTRUCTIONS OF PHARM D 1ST YEAR
SEPT	02.09.2019	VINAYAKA CHAVITHI
	02.09.2019	AWARENESS PROGRAMME ON HIGHER EDUCATION
	03.09.2019	NATIONAL NUTRITION DAY
	05.09.2019	SPORTS AND CULTURAL ACTIVITIES ON THE OCCASION OF TEACHER'S DAY
	10.09.2019	MOHARAM
	12.09.2019-14.09.2019	FIRST MID TERM EXAMINATIONS OF I SEMESTER FOR B PHARM 2ND ,3RD & 4TH YEARS
	16.09.2019-21.09.2019	FIRST MID TERM EXAMINATIONS OF PHARM D 2ND ,3RD ,4TH & 5TH YEARS
	25.09.2019	CULTURAL ACTIVITIES ON THE OCCASION OF WORLD PHARMACIST DAY
	26.09.2019	GUEST LECTURE FOR B PHARM
	28.09.2019	BATHUKAMMA
	30.09.2019	CULTURAL ACTIVITIES ON THE OCCASION OF FRESHEERS DAY CELEBRATIONS
OCT	02.10.2019	MATHATMA GANDHI JAYANTHI
	01.10.2019	CULTURAL ACTIVITIES ON THE OCCASION OF BATHUKAMMA CELEBRATIONS
	07.10.2019-12.10.2019	PARENT TEACHER MEETING FOR PHARM D 2ND ,3RD ,4TH & 5TH YEARS

	14.10.2019-02.11.2019	SUPPLEMENTARY EXXAMINATIONS OF PHARM D 2ND ,3RD ,4TH & 5TH YEARS
	06.10.2019	DURGASTAMI
	07.10.2019-19.10.2019	DUSSEHRA RECESS FOR M PHARM B PHARM & PHARM D
	08.10.2019	VIJAYA DASAMI
	18.10.2019	GUEST LECTURE FOR PHARM D
	24.10.2019-26.10.2019	FIRST MID TERM EXAMINATIONS OF I SEMESTER FOR B PHARM 1ST YEAR
	28.10.2019	Last date for submission of Comprehensive Viva-Voce FOR M PHARM 2ND YEAR 1ST SEM
	30.10.2019	EID MILADUN NABI
	31.10.2019	FIRST MID TERM EXAMINATIONS FOR M PHARM I YEAR 1ST SEM
NOV	08.11.2019	SUBMISSION OF FIRST MID TERM EXAM MARKS TO UNIVERSITY ON OR BEFORE FOR M PHARM 1ST YEAR 1ST SEM
	09.11.2019	PARENT TEACHER MEETING OF I SEMESTER FOR B PHARM & M PHARM 1ST YEAR
	12.11.2019	KARTIIKA PURNIMA
	14.11.2019	CHILDREN'S DAY
	17.11.2019-23.11.2019	MEDICAL CAMP
	18.11.2019	FIRST MID TERM EXAMINATIONS FOR PHARM D 1ST YEAR
	18.11.2019-25.11.2019	SPORTS AND CULTURAL ACTIVITIES ON THE OCCASION NATIONAL PHARMACY WEEK
	20.11.2019	LAST DATE OF INSTRUCTIONS OF I SEMESTER FOR B PHARM 2ND ,3RD & 4TH YEARS
	21.11.2019-23.11.2019	SECOND MID TERM EXAMINATIONS OF I SEMESTER FOR B PHARM 2ND ,3RD & 4TH YEARS
	22.11.2019	TREE PLLANTATION SWATCH BHARAT
	25.11.2019-30.11.2019	PREPARATION HOLIDAYS AND PRACTICAL EXAMINATIONS FOR B PHARM I SEMESTER OF 2ND ,3RD & 4TH YEARS

	23.11.2019	GENDER SENSITIZATION PROGRAMME
DEC	01.12.2019	WORLD AIDS DAY
	02.12.2019-14.12.2019	END SEMESTER EXAMINATIONS OF I SEMESTER FOR B PHARM 2ND ,3RD & 4TH YEARS
	08.12.2019	TELENGANA HARITHAHARAM
	10.12.2019	AWARENESS ON WEB COUNSELLING
	11.12.2019	Project Work Review-II (Phase-1) FOR M PHARM 2ND YEAR 1ST SEM
	14.12.2019	PARENT TEACHER MEETING OF PHARM D 1ST YEAR
	15.12.2019	AWARENESS OF COVID SAFETY MEASURES
	16.12.2019	COMMENCEMENT OF INSTRUCTIIONS OF II SEMESTER FOR B PHARM 2ND ,3RD & 4TH YEARS
	16.12.2019	Commencement of IV Semester(Project Work Continuation) FOR M PHARM 2ND YEAR 2ND SEM Part-II Duration FOR M PHARM 2ND YEAR 2ND SEM
	17.12.2019	LAST DATE OF INSTRUCTIONS OF I SEMESTER FOR B PHARM 1ST YEAR
	18.12.2019-20.12.2019	SECOND MID TERM EXAMINATIONS OF I SEMESTER FOR B PHARM 1ST YEAR
	18.12.2019	WOMEN'S RIGHTS
	21.12.2019-28.12.2019	PREPARATION HOLIDAYS AND PRACTICAL EXAMINATIONS OF I SEMESTER FOR 1ST YEAR FOR B PHARM
	24.12.2019-30.12.2019	SPORTS AND CULTURAL ACTIVITIES ON THE OCCASION ANNUAL DAY CELEBRATIONS
	24.12.2019	LAST DATE OF INSTRUCTIONS FOR M PHARM I ST YEAR 1ST SEM
	25.12.2019	CHRISTMAS
	26.12.2019	BOXING DAY
	27.12.2019	Project Work Review-II(Phase-11) FOR M PHARM II YEAR 1ST SEM
	27.12.2019	SECOND MID TERM EXAMINATIONS FOR M PHARM 1ST YEAR 1ST SEM

	30.12.2019-04.01.2020	SECOND MID TERM EXAMINATIONS OF PHARM D 2ND ,3RD ,4TH & 5TH YEARS
	30.12.2019	END I SEMESTER/SUPPLEMENTARY EXAMINATIONS OF 1ST YEAR
	31.12.2019	PREPARATION HOLIDAYS AND PRACTICAL EXAMINATIONS
	31.12.2019-29.02.2020	COMMENCEMENT OF INTERNSHIP IN SPECIALTY OF PHARM D 3RD & 6TH YEARS
JAN	01.01.2020	NEW YEARS DAY
	02.01.2020	Last date for submission of PRC-II marks FOR M PHARM 2ND YEAR 1ST SEM
	06.01.2020	AWARENESS PROGRAMME ON DRUG MENACE
	07.01.2020	Submission of Second Mid Term Exam Marks to University on or before FOR M PHARM I YEAR 1ST SEM
	08.01.2020	END SEMESTER/SUPPLEMENTARY EXAMINATIONS FOR M PHARM I YEAR 1ST SEM
	12.01.2020	NATIONAL YOUTH DAY
	13.01.2020	COMMENCEMENT OF INSTRUCTIONS OF II SEMESTER FOR B PHARM 1ST YEAR
	14.01.2020	BHOGI
	15.01.2020	SANKRANTI
	22.01.2020	BETI BACHAO BETI PADHAO
	24.01.2020	SAVE GIRL CHILD
	26.01.2020	SPORTS AND CULTURAL ACTIVITIES ON THE OCCASION OF REPUBLIC DAY
	27.01.2020	COMMENCEMENT OF INSTRUCTIONS OF II SEMESTER FOR M PHARM 1ST YEAR
FEB	05.02.2020	GOVERNING BODY MEETING
	07.02.2020	SPORTS AND CULTURAL ACTIVITIES ON THE OCCASION OF BIO-ADHYAYAN 2020 FACULTY DEPARTMENT PROGRAMME
	08.02.2020	PARENT TEACHER MEETING OF PHARM D 2ND ,3RD ,4TH & 5TH YEARS

	02.02.2020	DISTRICT LEVEL INTER PHARMA COLLEGE CRICKET TOURANAMENT
	10.02.2020-12.02.2020	FIRST MID TERM EXAMINATIONS OF II SEMESTER FOR B PHARM 2ND ,3RD & 4TH YEARS
	10.02.2020-15.02.2020	SECOND MID TERM EXAMINATION OF PHARM D 1ST YEAR
	21.02.2020	MAHA SHIVARATRI
	24.02.2020	SPORTS AND CULTURAL ACTIVITIES ON THE OCCASION OF NATIONAL WOMEN'S DAY
MARCH	02.03.2020	REPORT SUBMISSION OF INTERNSHIP IN SPECIALTY WARD-1 FOR PHARM D 3RD & 6TH YEARS
	03.03.2020-02.05.2020	COMMENCEMENT OF INTERNSHIP IN SPECCAALITY WARD-2 FOR PHARM D 3RD & 6TH YEARS
	05.03.2020-07.03.2020	FIRST MID TERM EXAMINATIONS OF II SEMESTER FOR B PHARM 1ST YEAR
	08.03.2020	INTERNATIONAL WOMENS DAY
	09.03.2020	HOLI
	14.03.2020	SPORTS AND CULTURAL ACTIVITIES ON THE OCCASION OF TRADITIONAL DAY
	14.03.2020	PARENT-TEACHER MEETING OF II SEMESTER FOR B PHSARM 2ND ,3RD & 4TH YEARS
	19.03.2020	FIRST MID TERM EXAMINATIONS OF II SEMESTER FOR M PHARM 1ST YEAR
	25.03.2020	UGADI
	23.03.2020-28.03.2020	THIRD MID TERM EXAMINATIONS OF PHARM D 2ND ,3RD ,4TH & 5TH YEARS
	28.03.2020	Submission of FIRST Mid Term Exam Marks to University on or before FOR M PHARM 2ND SEM
	30.03.2020-11.04.2020	PREPARATION AND PRACTICAL EXAAMINATIONS FOR PHARM D 2ND ,3RD ,4TH & 5TH YEARS
APRIL	02.04.2020	SRI RAMA NAVAMI
	05.04.2020	BABU JAGJIVAN RAM'S BIRTHDAY

	07.04.2020	WORLD HEALTH DAY
	08.04.2020-11.04.2020	SECOND MID TERM EXAMINATIONS OF II SEMESTER FOR B PHARM 2ND ,3RD & 4TH YEARS
	10.04.2020	GOOD FRIDAY
	11.04.2020	PARENT-TEACHER MEETING OF II SEMESTER FOR B PHARM &M PHARM 1ST YEAR
	13.04.2020-18.04.2020	PREPARATION HOLIDAYS AND PRACTICAL EXAMINATIONS OF II SEMESTER OF 2ND ,3RD & 4TH YEARS
	20.04.2020-02.05.2020	END II SEMESTER EXAMINATIONS OF 2ND ,3RD & 4TH YEARS
	20.04.2020-02.05.2020	END II SEMESTER EXAMINATIONS FOR B PHARM 2ND ,3RD & 4TH YEARS
	13.04.2020-25.04.2020	END/SUPPLEMENTARY EXAMINATIONS FOR PHARM D 2ND ,3RD ,4TH & 5TH YEARS
	14.04.2020	Dr. AMBEDKAR'S BIRTHDAY
	20.04.2020	WORLD EARTH DAY
	26.04.2020-04.07.2020	SUMMER VACATIONS FOR PHARM D 2ND ,3RD ,4TH & 5TH YEARS
MAY	03.05.2020-09.05.2020	TIIIRD MID TERM EXAMINATIONS OF 1ST YEAR
	01.05.2020	LAST DATE OF INSTRUCTIONS OF II SEMESTER FOR B PHARM 1ST YEAR
	02.05.2020-05.05.2020	SECOND MID TERM EXAMINATIONS OF II SEMESTER FOR B PHARM 1ST YEAR
	04.05.2020-04.07.2020	SUMMER VACATION OF II SEMESTER FOR B PHARM 2ND ,3RD & 4TH YEARS
	06.05.2020-12.05.2020	PREPARATION HOLIDAYS AND PRACTICAL EXAMINATIONS OF II SEMESTER FOR B PHARM 1ST YEAR Project Work Review –III (Phase-1) FOR M PHARM 2ND YEAR 2ND SEM
	04.05.2020	REPORT SUBMISSION OF INTERNSHIP IN SPECIALTY WARD-2 FOR PHARM D 3RD & 6TH YEARS

	05.05.2020-04.07.2020	COMMENCEMENT OF INTERNSHIP IN SPECIALTY WARD-3 FOR PHARM D 3RD & 6TH YEARS
	07.05.2020	ACADEMIC COMMITTEE MEETING
	11.05.2020-23.05.2020	PREPARATION AND PRACTICAL EXAMINATIONS OF 1ST YEAR
	13.05.2020	LAST DATE OF INSTRUCTIONS FOR MPHARM I YEAR 2ND SEM
	14.05.2020	SECOND MID TERM EXAMINATIONS FOR M PHARM I ST YEAR 2ND SEM
	18.05.2020	PRACTICAL EXAMINATIONS FOR MPHARM I YEAR 2ND SEM
	20.05.2020	Submission of Second Mid Term Exam Marks to University on or before FOR M PHARM I 2ND SEM Last date for submission of Project Work Review-III(Phase-1) Marks FOR M PHARM 2ND YEAR 2ND SEM
	20.05.2020	BLOOD DONATION CAMP
	21.05.2020	SUMMER VACATION FOR M PHARM I YEAR 2ND SEM
	25.05.2020	RAMZAN
	28.05.2020-04.07.2020	SUMMER VACATIONS OF II SEMESTER FOR B PHARM 1ST YEAR
	28.05.2020	WORLD NUTRITION DAY
JUNE	08.06.2020-04.06.2020	SUMMER VACATION FOR PHARM D 1ST YEAR
	25.06.2020-06.06.2020	END SEMESTER/ SUPPLEMENTARY EXAMINATIONS FOR PHARM D 1ST YEAR
JULY	01.07.2020	END SEMESTER/ SUPPLEMENTARY EXAMINATIONS FOR M PHARM 1ST YEAR 2ND SEM
AUGUST	19.08.2020	Project Work Review – III (Phase-II) FOR M PHARM 2ND YEAR 2ND SEM
		Last date for submission of Project Work Review-III (Phase-II) Marks
OCT	02.10.2020	AWARENESS PROGRAMME ON NATIONAL. PEACE ON THE OCCASSION OF BIRTH ANNIVERSARY MAHATHMA GANDHI

DEPARTMENT OF PHARMACY PRACTICE

ACADEMIC CALENDER 2019-2020

PHARM.D I YEAR – V YEAR

DESCRIPTION	I YEAR	II YEAR	III YEAR	IV YEAR	V YEAR
COMMENCEMENT OF CLASSWORK	26.08.2019	01.07.2019	01.07.2019	01.07.2019	01.07.2019
I MID OF EXAMINATION	18.11.2019	16.09.2019	16.09.2019	16.09.2019	16.09.2019
SUBMISSION OF FIRST MID TERM EXAM MARKS TO UNIVERSITY ON OR BEFORE	30.11.2019	30.09.2019	30.09.2019	30.09.2019	30.09.2019
II MID OF EXAMINATION	10.02.2020	06.01.2020	06.01.2020	06.01.2020	06.01.2020
SUBMISSION OF SECOND MID TERM EXAM MARKS TO UNIVERSITY ON OR BEFORE	22.02.2020	18.01.2020	18.01.2020	18.01.2020	18.01.2020
III MID OF EXAMINATION	11.05.2020	30.03.2020	30.03.2020	30.03.2020	30.03.2020
SUBMISSION OF THIRD MID TERM EXAM MARKS TO UNIVERSITY ON OR BEFORE	16.05.2020	13.04.2020	13.04.2020	13.04.2020	13.04.2020
PREPARATION AND PRACTICALS	11.05.2020	06.04.2020	06.04.2020	06.04.2020	06.04.2020
END EXAMINATIONS	25.06.2020	20.04.2020	20.04.2020	20.04.2020	20.04.2020

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PHARM D VI YEAR

S.NO	DESCRIPTION	VI YEAR
1	COMMENCEMENT OF INTERNSHIP IN GENERAL WARD	01.07.2019
2	REPORT SUBMISSION OF INTERNSHIP IN GENERAL WARD	30.12.2019
3	COMMMENCEMENT OF INTERNSHIP IN SPECIALITY WARD-1	31.12.2019
4	REPORT SUBMISSION OF INTERNSHIP IN SPECIALITY WARD -1	02.03.2020
5	COMMENCEMENT OF INTERNSHIP IN SPECIALITY WARD-2	03.03.2020
6	REPORT SUBMISSION OF INTERNSHIP IN SPECIALITY WARD -2	04.05.2020
7	COMMENCEMENT OF INTERNSHIP IN SPECIALITY WARD-3	05.05.2020
8	REPORT SUBMISSION OF INTERNSHIP IN SPECIALITY WARD -3	06.07.2020
9	FINAL VIVA OF INTERNSHIP	08.07.2020

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DEPARTMENT OF PHARMACY

ACADEMIC CALENDER 2019-2020

M PHARM I & II YEAR

EVENT	I YEAR	
EVENT	**SEM-I**	**SEM-II**
COMMENCEMENT OF CLASSWORK	26.08.2019	27.01.2020
I MID OF EXAMINATION	31.10.2019	19.03.2020
SUBMISSION OF FIRST MID TERM EXAM MARKS TO UNIVERSITY ON OR BEFORE	*08.11.2019*	*28.03.2020*
II MID OF EXAMINATION	27.12.2019	14.05.2020
SUBMISSION OF SECOND MID TERM EXAM MARKS TO UNIVERSITY ON OR BEFORE	07.01.2020	20.05.2020
PREPARATION AND PRACTICALS	31.12.2019	08.05.2020
END EXAMINATIONS	08.01.2020	01.07.2020

DESCRIPTION	II YEAR
I SEM	
COMMENCEMENT OF I SEM CLASSWORK	15.07.2019
PREPARATION OF PROJECT WORK PROPSALS	10.08.2019
PROJECT WORK REVIEW- 1	13.08.2019
LAST DATE FFOR SUBMISSION OF PRC-1	20.08.2019
LAST DATE FOR SUBMISSION OF COMPHERNSIVE VIVA VOCE MARKS	28.10.2019
PROJECT WORK REVIEW- 2(PHASE-1)	11.12.2019
PROJECT WORK REVIEW- 2(PHASE-2)	27.12.2019
LAST DATE FOR SUBMISSION OF PRC-II MARKS	02.01.2020

AVANTHI INSTITUTE OF PHARMACEUTICAL SCIENCES * ABDULLAPURMET

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DESCRIPTION	II YEAR
II SEM	
COMMMENCEMENT OF II SEMESTER	16.12.2019
PROJECT WORK REVIEW-III (PHASE-I)	12.05.2020
LAST DATE FOR SUBMISSION OF PROJECT WORK REVIEW-III	20.05.2020
DATE OPF ELIGIBITY OF THESIS SUBMISSION	20.05.2020
SUBMISSION OF THESIS AND PROJECT VIVA VOCE EXAMINATION	-
PROJECT WORK REVIEW-III (PHASE-II)	19.08.2020
LAST DATE FOR SUBMISSION OF PROJECT WORK REVIEW-III (PHASE-II)	26.08.2020
SUBMISSION OF THESISAND PROJECT VIVA VOCE EXAMINATION (PHASE-II)	-

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DEPARTMENT OF PHARMACY

ACADEMIC CALENDER 2019-2020

B.PHARMACY

EVENT	I YEAR		II YEAR		III YEAR		IV YEAR	
COMMENCEMENT OF CLASSWORK	26.08.2019	27.01.2020	15.07.2019	16.12.2019	15.07.2019	16.12.2019	15.07.2019	16.12.2019
I MID OF EXAMINATION	31.10.2019	19.03.2020	12.09.2019	10.02.2020	12.09.2019	10.02.2020	12.09.2019	10.02.2020
II MID OF EXAMINATION	27.12.2019	14.05.2020	21.11.2019	08.04.2020	21.11.2019	08.04.2020	21.11.2019	08.04.2020
PREPARATION AND PRACTICALS	31.12.2019	08.05.2020	25.11.2019	13.04.2020	25.11.2019	13.04.2020	25.11.2019	13.04.2020
END EXAMINATIONS	08.01.2020	25.05.2020	02.12.2019	20.04.2020	02.12.2019	20.04.2020	02.12.2019	20.04.2020

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(Approved by PCI, AICTE & Affiliated to JNTUH)
Gunthapally (V), Abdullapurmet (M), R.R. Dist., Near Ramoji Filmcity, Hyderabad - 501 512.

DEPARTMENT OF PHARMACY PRACTICE

A.Y 2019-20 TIME TABLE

PHARM.D VI YEAR W.E.F: 01.07.2019 COLLEGE TIMINGS:9:30AM-3:50PM

DAY	9.30AM-10.20AM	10.20AM-11.10AM	11.10AM-12.00PM	12.00AM-12.50PM	12.50PM-1.20PM	1.20P.M-2.10PM	2.10PM-3.00PM	3.00PM-3.50PM
MON	CARDIOLOGY	NEPHROLOGY	NEUROLOGY	UROLOGY	L	CRITICAL CARE	PULMONORY	CASE PRESENTATION
TUE	PULMONORY	CRITICAL CARE	UROLOGY	NEPHROLOGY	U	NEUROLOGY	CARDIOLOGY	CASE PRESENTATION
WED	CRITICAL CARE	PULMONORY	NEPHROLOGY	CARDIOLOGY	N	UROLOGY	NEUROLOGY	CASE PRESENTATION
THU	UROLOGY	NEUROLOGY	PULMONORY	CRITICAL CARE	C	NEPHROLOGY	CARDIOLOGY	CASE PRESENTATION
FRI	NEUROLOGY	CRITICAL CARE	UROLOGY	PULMONORY	H	CARDIOLOGY	NEPHROLOGY	CASE PRESENTATION
SAT	CARDIOLOGY	NEPHROLOGY	CRITICAL CARE	UROLOGY		PULMONORY	NEUROLOGY	CASE PRESENTATION

Monday	Dr.P. Swathi/ Dr. K. Anusha	Assistant Professor/ Assistant Professor
Tuesday	Dr.. Ravinayak/ Dr. MD. Abdul Azeem	Assistant Professor/ Assistant Professor
Wednesday	Dr. Raviprakash/ Dr. Ravinayak	Assistant Professor/ Assistant Professor
Thursday	Dr.P. Swathi/ Dr. K. Anusha	Associate Professor
Friday	Dr.. Ravinayak/ Dr. MD. Abdul Azeem	Assistant Professor/ Assistant Professor
Saturday	Dr. Raviprakash/ Dr. Ravinayak	Assistant Professor/ Assistant Professor

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DEPARTMENT OF PHARMACY PRACTICE

A.Y 2019-20 TIME TABLE

PHARM.D V YEAR W.E.F: 01.07.2019 COLLEGE TIMINGS:9:30AM-3:50PM

DAY	9.30AM-10.20AM	10.20AM-11.10AM	11.10AM-12.00PM	12.00AM-12.50PM	12.50PM-1.20PM	1.20P.M-2.10PM	2.10PM-3.00PM	3.00PM-3.50PM
MON	CR	P&PE	CPK&PDM	TEST	L	P&PE	SEMINAR	CR
TUE	CR	SEMINAR			U	CPK&PDM	P&PE	TEST
WED	HOSPITALVISIT				N	HOSPITALVISIT		
THU	CPK&PDM	CR	P&PE	CLERKSHIP	C	HOSPITALVISIT		
FRI	HOSPITALVISIT				H	HOSPITALVISIT		
SAT	HOSPITALVISIT					HOSPITALVISIT		

SUBJECTNAME	FACULTYNAME	DESIGNATION
Clinical Research	Dr.P. Swathi	Assistant Professor
Pharmaco Epidemiology and Pharmaco Economics	Dr. K. Anusha	Assistant Professor
Clinical pharmacokinetics & Pharmacotherapeutic drug Monitoring	Dr.. Ravinayak	Assistant Professor
Clerkship*	Dr.. Ravinayak	Assistant Professor
Project work(six months)	Dr. K. Anusha/P.Swathi	Assistant Professor

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DEPARTMENT OF PHARMACY PRACTICE

A.Y 2019-20 TIME TABLE

PHARM.D IV YEAR W.E.F: 01.07.2019 COLLEGE TIMINGS:9:30AM-3:50PM

DAYS	9.30AM-10.20AM	10.20AM-11.10AM	11.10AM-12.00PM	12.00AM-12.50PM	12.50PM-1.20PM	1.20P.M-2.10PM	2.10PM-3.00PM	3.00PM-3.50PM
MON	CT	B&RM	P.THER-III	HP	L	BPK (T)	TEST	CP
TUE	BPK	P.THER-III (T)	CP	TEST	U	B&RM	HP(T)	CT
WED	P.THER-III	SEMINAR			N	HOSPITALVISIT(P.THER-III)		
THU	P.THER-III	HP	HP	CP	C	LIBRARY/SPORTS		
FRI	CP(T)	HOSPITALVISIT			H	HOSPITALVISIT		
SAT	B&RM	BPK				BPK	TEST	CT

SUBJECTNAME	FACULTYNAME	DESIGNATION
Pharmacotherapeutics-III	Dr. Raviprakash	Assistant Professor
Hospital pharmacy	Dr. Ravinayak	Assistant Professor
Clinical pharmacy	Dr. MD. Abdul Azeem	Associate Professor
Biostatistics and Research methodology	I. Swathi	Assistant Professor
Biopharmaceutics and pharmacokinetics	Dr. Arifabegum	Assistant Professor
Clinical toxicology	Dr. P. Swathi	Assistant Professor
Pharmacotherapeutics-III Lab	Dr. Raviprakash	Assistant Professor
Hospital pharmacy Lab	Dr. Ravinayak	Assistant Professor
Clinical pharmacy Lab	Dr. MD. Abdul Azeem	Associate Professor
Biopharmaceutics and pharmacokinetics Lab	I. Swathi	Assistant Professor

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DEPARTMENT OF PHARMACY PRACTICE

A.Y 2019-20 TIME TABLE

PHARM.D III YEAR W.E.F: 01.07.2019 COLLEGE TIMINGS:9:30AM-3:50PM

DAYS	9.30AM-10.20AM	10.20AM-11.10AM	11.10AM-12.00PM	12.00AM-12.50PM	12.50PM-1.20PM	1.20P.M-2.10PM	2.10PM-3.00PM	3.00PM-3.50PM
MON	P.A LAB			P.A	L	M.C	P.J	SEMINARS
TUE	M.C LAB			P.THER.–II	U	P.F (T)	P.THER –II	P.COL-II
WED	P.F LAB			P.THER.–II	N	M.C (T)	P.COL-II	P.A
THU	P.A	P.F	P. THER.-II LAB (HOSPITALVIST)		C	P.THER.-II LAB(HOSPITALVISIT)		
FRI	P.COL-II LAB			M.C	H	P.J	P.THER.II (T)	P.COL-II (T)
SAT	P.F	P.J	LIBRARYSPORTS			M.C	P.COL-II	P.A(T)

SUBJECTNAME	FACULTYNAME	DESIGNATION
Pharmacology-II	Santhoshi kumari	Assistant Professor
Pharmaceutical Analysis	Dr. Raviprakash	Assistant Professor
Pharmacotherapeutics-II	Dr. K. Anusha	Assistant Professor
Pharmaceutical Jurisprudence	Dr. K. Ravinayak	Assistant Professor
Medicinal Chemistry	Dr. Arifa begum	Assistant Professor
Pharmaceutical Formulations	P. Srilatha /S. Sandhya rani	Assistant Professor/ Assistant Professor
Pharmacology-II	Santhoshi kumari	Assistant Professor
Pharmaceutical Analysis-Lab	Dr. Raviprakash	Assistant Professor
Pharmacotherapeutics-II-Lab	Dr. K. Anusha	Assistant Professor
Medicinal Chemistry-Lab	Dr. Arifa begum	Assistant Professor
Pharmaceutical Formulations-Lab	P. Srilatha/ S. Sandhya rani	Assistant Professor/ Assistant Professor

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DEPARTMENT OF PHARMACY PRACTICE

A.Y 2019-20 TIME TABLE

PHARM.D II YEAR W.E.F: 01.07.2019 COLLEGE TIMINGS:9:30AM3:50PM

DAYS	9.30AM-10.20AM	10.20AM-11.10AM	11.10AM-12.00PM	12.00AM-12.50PM	12.5PM-1.20PM	1.20P.M-2.10PM	2.10P M-3.00P M	3.00PM - 3.50PM
MON	P.COL-I	CP	P.PHY.	P.THER.-I	L	LIBRARY/SPORTS		
TUE	P.THER.-I	MICRO	P.PHY	LIBRARY	U	SEMINARS		CP
WED	P.PHY.	MICRO	P.COL-I	MICRO	N	MICRO		
THU	P.COL-I	LIBRARY	P.COG&PHYTO	P.COG& PHYTO	C	P.COG&PHYTO.		
FRI	CP	P.PHY	P.THER.-I(T)	P.COL-I(T)	H	SEMINARS		
SAT	MICRO (BS)	P.THER.-I	P.THER.-ILAB(HOSPITALVISIT)			P.THER.-ILAB (HOSPITAL VISIT)		

Subject Name	Faculty Name	Designation
Pathophysiology	Dr. Ravi prakash	Assistant Professor
Pharmaceutical Microbiology	Dr. Ravinayak	Assistant Professor
Pharmacognosy & Phytopharmaceuticals	S. Sandhya rani	Assistant Professor
Pharmacology-I	Santhoshi kumari	Assistant Professor
Community Pharmacy	P. Swathi	Assistant Professor
Pharmacotherapeutics-I	Dr. K.Anusha	Assistant Professor
PharmaceuticalMicrobiology –Lab	Dr. Ravinayak	Assistant Professor
Pharmacognosy & Phytopharmaceuticals-Lab	S. Sandhya rani	Assistant Professor
Pharmacotherapeutics-I-Lab	Dr. K.Anusha	Assistant Professor

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PHARM.D I YEAR W.E.F: COLLEGE TIMINGS:9:30AM 3:50PM

DAYS	9.30AM-10.20AM	10.20AM-11.10AM	11.10AM-12.00PM	12.00AM-12.50PM	12.50PM-1.20PM	1.20P.M-2.10PM	2.10PM-3.00PM	3.00PM-3.50PM
MON	B.CHEM.	HAP	POC	P.CEU (T)	L	PIC		
TUE	PIC	RM	POC (T)	TEST	U	LIBRARY/SPORTS		
WED	HAP	POC	B.CHEM. (T)	PIC (T)	N	POC		
THU	POC	RM/RB	P. CEU	PIC	C	HAP		
FRI	B.CHEM.	RM/RB	P. CEU	B. CHEM.	H	B.CHEM.		
SAT	HAP	RM/RB	TEST	HAP (T)		P.CEU.		

Subject name	Faculty name	Designation
Human Anatomy And Physiology	Dr. P. Swathi	Assistant Professor
Pharmaceutics	I. Swathi /S. Sandhya rani	Assistant Professor
Medicinal Biochemistry	Dr. MD. Abdul Azeem	Associate Professor
Pharmaceutical Organic Chemistry	Dr. Arifa Begum	Assistant Professor
Pharmaceutical Inorganic Chemistry	Santhoshi Kumari	Assistant Professor
Remedial Mathematics/ Biology	T. Bhargavi	Assistant Professor
Human Anatomy And Physiology Lab	Dr. P. Swathi	Assistant Professor
Pharmaceutics Lab	I. Swathi /S. Sandhya rani	
Medicinal Biochemistry Lab	Dr. MD. Abdul Azeem	Associate Professor
Pharmaceutical Organic Chemistry Lab	Dr. Arifa Begum	Assistant Professor
Pharmaceutical Inorganic Chemistry-Lab	Santhoshi Kumari	Assistant Professor

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PHARM D WORK LOAD 2019-2020

S.No	Name of the faculty	Subjects	Class	No of periods	Total Workload	signature
1	MD.ABDUL AZEEM	M.BIO	I YR	7	14	
		CP	IV YR	7		
2	Dr. P. SWATHI PATEL	HAP	I YR	7	17	
		CP	II YR	3		
		CT	IV YR	3		
		CR	V YR	4		
3	Dr. RAVI NAYAK	HP	IV YR	6	16	
		PJ	III YR	2		
		P.MICRO	II YR	7		
		CLERKSHIP	V YR	1		
4	Dr. K. ANUSHA	P.THER-II	III YR	7	18	
		P.THER-I	II YR	7		
		EPIDEMOLOGY	V YR	4		
5	Dr. D. RAVIPRAKASH	PATHO	II YR	4	21	
		P.THRER-III	IV YR	7		
		CP&PTDM	V YR	3		
		PA	III YR	7		
6	Dr. ARIFA BEGUM	POC	I YR	7	21	
		MC	III YR	7		
		BPPK	IV YR	7		
7	SANTHOSHI KUMARI	P.COL-I	II YR	7	16	
		PIC	I YR	6		
		BSRM	IV YR	3		
8	I. SWATHI	P.CEUT	I YR	6	20	
		PF	III YR	6		
		P.CEU	II-II(A,B) B.PHARM	8		
9	S. SANDHYA RANI	PF	III YR	6	19	
		P.COG&PHYTO	II YR	7		
		P.CEUT	I YR	6		

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S.No	Name of the faculty	Subjects	Class	No of periods	Total Workload	signature
10	Dr. P.SRILATHA	PF	III YR	6	19	
		P.COG&PHYTO	II YR	7		
		P.CEU	I YR	6		

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JAWAHARLAL NEHRU TECHNOLOGICAL UNIVERSITY HYDERABAD

(Established by Act No. 30 of 2008)

Kukatpally, Hyderabad, Telangana (India).

ACADEMIC REGULATIONS OF B.PHARM. (REGULAR/FULL TIME) STUDENTS WITH EFFECT FROM THE ACADEMIC YEAR 2017-18 (R-17)

1.0 Under-Graduate Degree Programme in Pharmacy

1.1 JNTUH offers a 4-year (8 semesters) **Bachelor of Pharmacy** (B.Pharm.) degree programme, under Choice Based Credit System (CBCS) at its affiliated colleges with effect from the academic year 2017-18.

2.0 Eligibility for admission

2.1 Admission to the under graduate programme shall be made either on the basis of the merit rank obtained by the qualified candidate in entrance test conducted by the Telangana State Government (EAMCET) or the University or on the basis of any other order of merit approved by the University, subject to reservations as prescribed by the government from time to time.

2.2 The medium of instructions for the entire under graduate programme in Pharmacy will be **English** only.

3.0 B.Pharm. Programme structure

3.1 A student after securing admission shall pursue the under graduate programme in B.Pharm. in a minimum period of **four** academic years (8 semesters), and a maximum period of **eight** academic years (16 semesters) starting from the date of commencement of first year first semester, failing which student shall forfeit seat in B.Pharm course.

A student shall register for all subjects for covering 196 credits and each student shall secure 196 credits (with CGPA $\geq$ 5) required for the completion of the under graduate programme and award of the B.Pharm. degree.

3.2 **UGC/ AICTE** specified definitions/ descriptions are adopted appropriately for various terms and abbreviations used in these academic regulations/ norms, which are listed below.

3.2.1 Semester scheme

Each under graduate programme is of 4 academic years (8 semesters) with the academic year being divided into two semesters of 22 weeks ($\geq$ 90 instructional days) each, each semester shall have - 'Continuous Internal Evaluation (CIE)' and 'Semester End Examination (SEE)'. Choice Based Credit System (CBCS) and Credit Based Semester

System (CBSS) as indicated by UGC and curriculum / course structure as suggested by AICTE are followed.

3.2.2 Credit courses

All subjects/ courses are to be registered by the student in a semester to earn credits which shall be assigned to each subject/ course in an L: T: P: C (lecture periods: tutorial periods: practical periods: credits) structure based on the following general pattern.

- One credit for one hour/ week/ semester for theory/ lecture (L) courses.
- One credit for two hours/ week/ semester for laboratory/ practical (P) courses or tutorials (T).

Courses like environmental science, human values and professional ethics, gender sensitization lab and other student activities like NCC/NSO and NSS are identified as mandatory courses. These courses will not carry any credits.

3.2.3 Subject Course Classification

All subjects/ courses offered for the under graduate programme in Pharmacy (B.Pharm. degree programmes) are broadly classified as follows. The university has followed almost all the guidelines issued by AICTE/UGC.

S. No.	Broad Course Classification	Course Group/ Category	Course Description
1	Foundation Courses (FnC)	BS – Basic Sciences	Includes mathematics, physics and chemistry subjects.
2		PS - Pharmaceutical Sciences	Includes fundamental Pharmacy Subjects.
3		HS – Humanities and Social sciences	Includes subjects related to humanities, social sciences and management.
4	Core Courses (CoC)	PC – Professional Core	Includes core subjects related to the parent discipline.
5	Elective Courses (EℓC)	OE – Open Electives	Includes elective subjects related to inter-disciplinary areas of Pharmacy or other than Pharmacy
6	Core Courses	Project Work	B.Pharm. project or UG project or UG major project
7		Seminar	Seminar/ Colloquium based on core contents related to parent discipline.
10	Minor courses	-	1 or 2 Credit courses (subset of HS)
11	Mandatory Courses (MC)	-	Mandatory courses (non-credit)

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4.0 Course registration

4.1 A 'faculty advisor or counselor' shall be assigned to a group of 15 students, who will advise student about the under graduate programme, its course structure and curriculum, choice/option for subjects/ courses, based on their competence, progress, pre-requisites and interest.

4.2 The academic section of the college invites 'registration forms' from students before the beginning of the semester through 'on-line registration', ensuring 'date and time stamping'. The on-line registration requests for any 'current semester' shall be **completed before the commencement of semester end examinations of the 'preceding semester'**.

4.3 A student can apply for **on-line** registration, **only after** obtaining the '**written approval**' from faculty advisor/counselor, which should be submitted to the college academic section through the Head of the Department. A copy of it shall be retained with Head of the Department, faculty advisor/ counselor and the student.

4.4 If the student submits ambiguous choices or multiple options or erroneous entries during **on-line** registration for the subject(s) / course(s) under a given/ specified course group/ category as listed in the course structure, only the first mentioned subject/ course in that category will be taken into consideration.

4.5 Subject/ course options exercised through **on-line** registration are final and **cannot** be changed or inter-changed; further, alternate choices also will not be considered. However, if the subject/ course that has already been listed for registration by the Head of the Department in a semester could not be offered due to any unforeseen or unexpected reasons, then the student shall be allowed to have alternate choice either for a new subject (subject to offering of such a subject), or for another existing subject (subject to availability of seats). Such alternate arrangements will be made by the Head of the Department, with due notification and time-framed schedule, within the **first week** after the commencement of class-work for that semester.

4.6 **Open Electives**: Students have to choose one open elective (OE-I) in II year II semester, one (OE-II) in III year I semester, and one (OE-III) in III year II semester and one (OE-IV) in IV year II semester from the list of Open Electives.

5.0 Subjects/ courses to be offered

5.1 A typical section (or class) strength for each semester shall be 60.

5.2 A subject/ course may be offered to the students, **only if** a minimum of 20 students (1/3 of the section strength) opt for it. The maximum strength of a section is limited to 80 (60 + 1/3 of the section strength).

5.3 If more entries for registration of a subject come into picture, then the Head of Department concerned shall decide, whether or not to offer such a subject/ course for **two (or multiple) sections**.

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6.0 Attendance requirements:

6.1 Attendance in all classes (Lectures/Laboratories/Project Work) is compulsory. The minimum required attendance in aggregate of all the subjects/ courses including the attendance of mid-term examination / Laboratory etc. is 75%. Two periods of attendance for each theory subject shall be considered, if the student appears for the mid-term examination of that subject. A student shall not be permitted to appear for the Semester End Examinations (SEE), if his attendance is less than 75% (excluding attendance in mandatory courses environmental science, human values and professional ethics, gender sensitization Lab, NCC/NSO, NSS and Industrial Training) for that semester.

6.2 Condoning of shortage of attendance (between 65% and 75%) up to a maximum of 10% (considering the days of attendance in sports, games, NCC, NSS activities and Medical grounds) in each semester shall be granted by the College Academic Committee on genuine and valid grounds, based on the student's representation with supporting evidence.

6.3 A stipulated fee shall be payable towards condoning of shortage of attendance.

6.4 Shortage of attendance below 65% in aggregate shall in **no case be condoned**.

6.5 Students whose shortage of attendance is not condoned in any semester are not eligible to take their end examinations of that semester. They get detained and their registration for that semester shall stand cancelled. They will not be promoted to the next semester. They may seek re-registration for all those subjects registered in that semester in which student was detained, by seeking re-admission into that semester as and when offered; in case if there are any open electives, the same may also be re-registered if offered. However, if those electives are not offered in later semesters, then alternate electives may be chosen from the **same** set of elective subjects offered under that category.

6.6 A student fulfilling the attendance requirement in the present semester shall not be eligible for readmission into the same class.

7.0 Academic requirements

The following academic requirements have to be satisfied, in addition to the attendance requirements mentioned in item no.**6.**

7.1 A student shall be deemed to have satisfied the academic requirements and earned the credits allotted to each subject/ course, if student secures not less than 35% marks (26 out of 75 marks) in the semester end examination, and a minimum of 40% of marks in the sum total of the CIE (Continuous Internal Evaluation) and SEE (Semester End Examination) taken together; in terms of letter grades, this implies securing **'C'** grade or above in that subject/ course.

7.2 Promotion Rules

S. No.	Promotion	Conditions to be fulfilled
1	**First year first semester to first year second semester**	**Regular course of study of first year first semester.**

2	**First year second semester to second year first semester**	**(i) Regular course of study of first year second semester.** **(ii) Must have secured at least 24 credits out of 48 credits i.e., 50% of credits up to first year second semester from all the relevant regular and supplementary examinations, whether the student takes those examinations or not.**
3.	**Second year first semester to second year second semester**	**Regular course of study of second year first semester.**
4	**Second year second semester to third year first semester**	**(i) Regular course of study of second year second semester.** **(ii) Must have secured at least 58 credits out of 96 credits i.e., 60% of credits up to second year second semester from all the relevant regular and supplementary examinations, whether the student takes those examinations or not.**
5	**Third year first semester to third year second semester**	**Regular course of study of third year first semester.**
6	**Third year second semester to fourth year first semester**	**(i) Regular course of study of third year second semester.** **(ii) Must have secured at least 86 credits out of 144 credits i.e., 60% of credits up to third year second semester from all the relevant regular and supplementary examinations, whether the student takes those examinations or not.**
7	**Fourth year first semester to fourth year second semester**	**Regular course of study of fourth year first semester.**

7.3 A student shall register for all subjects covering 196 credits as specified and listed in the course structure, fulfills all the attendance and academic requirements for 196 credits, 'earn all 196 credits' by securing SGPA ≥ 5.0 (in each semester) and CGPA (at the end of each successive semester) ≥ 5.0 to successfully complete the under graduate programme.

7.4 After securing the necessary 196 credits as specified for the successful completion of the entire under graduate programme, the student can avail exemption of two subjects up to 6 credits, that is, two open elective subjects for optional drop out from these 196 credits earned; resulting in 190 credits for under graduate programme performance evaluation, i.e., the performance of the student in these 190 credits shall alone be taken into account for the calculation of 'the final CGPA (at the end of under graduate programme, which takes the SGPA of the IV year II semester into account), and shall be indicated in the

grade card of IV year II semester. However, the performance of student in the earlier individual semesters, with the corresponding SGPA and CGPA for which grade cards have already been given will not be altered.

7.5 If a student registers for some more **'extra subjects'** other than those listed subjects totaling to 196 credits as specified in the course structure, the performances in those **'extra subjects'** (although evaluated and graded using the same procedure as that of the required 196 credits) will not be taken into account while calculating the SGPA and CGPA. For such **'extra subjects'** registered, % of marks and letter grade alone will be indicated in the grade card as a performance measure, subject to completion of the attendance and academic requirements as stated in regulations 6 and 7.1 – 7.4 above.

7.6 A student eligible to appear in the end semester examination for any subject/ course, but absent from it or failed (thereby failing to secure **'C'** grade or above) may reappear for that subject/ course in the supplementary examination as and when conducted. In such cases, CIE assessed earlier for that subject/ course will be carried over, and added to the marks to be obtained in the SEE supplementary examination for evaluating performance in that subject.

7.7 A student **detained in a semester due to shortage of attendance, may be re-admitted when the same semester is offered in the next academic year for fulfillment of academic requirements**. The academic regulations under which student has been readmitted shall be applicable. However, no grade allotments or SGPA/ CGPA calculations will be done for the entire semester in which student has been detained.

7.8 A student detained **due to lack of credits, shall be promoted to the next academic year only after acquiring the required academic credits.** The academic regulations under which student has been readmitted shall be applicable to him.

Note: (1) The SGPA will be computed and printed on the marks memo only if the candidate passes in all the subjects offered and gets minimum B grade in all the subjects.

(2) CGPA is calculated only when the candidate passes in all the subjects offered in all the semesters.

8.0 Evaluation - Distribution and Weightage of marks

8.1 The performance of a student in every subject/course (including practicals and UG major project) will be evaluated for 100 marks each, with 25 marks allotted for CIE (Continuous Internal Evaluation) and 75 marks for SEE (Semester End-Examination).

8.2 For theory subjects, during a semester, there shall be two mid-term examinations. Each mid-term examination consists of one objective paper, one descriptive paper and one assignment. The objective paper and the essay paper shall be for 10 marks each with a total duration of 1 hour 20 minutes (20 minutes for objective and 60 minutes for essay paper). The objective paper is set with 20 bits of multiple choice, fill-in the blanks and matching type of questions for a total of 10 marks. The essay paper shall contain 4 full

questions out of which, the student has to answer 2 questions, each carrying 5 marks. While the first mid-term examination shall be conducted on 50% of the syllabus, the second mid-term examination shall be conducted on the remaining 50% of the syllabus. Five marks are allocated for assignments (as specified by the subject teacher concerned). The first assignment should be submitted before the conduct of the first mid-examination, and the second assignment should be submitted before the conduct of the second mid-examination. The total marks secured by the student in each mid-term examination are evaluated for 25 marks, and the average of the two mid-term examinations shall be taken as the final marks secured by each student in internals/sessionals. If any student is absent from any subject of a mid-term examination, an on-line test will be conducted for him by the university. The details of the question paper pattern are as follows,

- The end semester examinations will be conducted for 75 marks consisting of two parts viz. i) **Part- A** for 25 marks, ii) **Part - B** for 50 marks.
- Part-A is compulsory question which consists of ten sub-questions. The first five sub-questions are from each unit and carry 2 marks each. The next five sub-questions are one from each unit and carry 3 marks each.
- Part-B consists of five questions (numbered from 2 to 6) carrying 10 marks each. Each of these questions is from one unit and may contain sub-questions. For each question there will be an "either" "or" choice, which means that there will be two questions from each unit and the student should answer either of the two questions.

8.3 For practical subjects there shall be a continuous internal evaluation during the semester for 25 sessional marks and 75 semester end examination marks. Out of the 25 marks for internal evaluation, day-to-day work in the laboratory shall be evaluated for 15 marks and internal practical examination shall be evaluated for 10 marks conducted by the laboratory teacher concerned. The semester end examination shall be conducted with an external examiner and the laboratory teacher. The external examiner shall be appointed from the clusters of colleges which are decided by the examination branch of the university.

8.4 There shall be an Industrial Training in IV year I semester. For the Industrial Training, the student shall be required to work for at least 150 hours spread over four weeks in a Pharmaceutical Industry/Hospital. It includes Production unit, Quality Control department, Quality Assurance department, Analytical laboratory, Chemical manufacturing unit, Pharmaceutical R&D, Hospital (Clinical Pharmacy), Clinical Research Organization, Community Pharmacy, etc. After the IV year I semester and before the commencement of IV year II semester, the student shall submit satisfactory report of the work and certificate duly signed by the authority of training organization to the head of the institute.

8.5 **Practice School:** In the IV year I semester, every candidate shall undergo a practice school for a period of 150 hours evenly distributed throughout the semester. The student

shall opt any one of the domains for practice school declared by the departmental committee from time to time. At the end of the practice school, every student shall submit a printed report (in triplicate) on the practice school he/she attended (not more than 25 pages). The report shall be submitted to the departmental committee consisting of Head of the Institution, Head of the Department and a senior faculty member. The practice school report shall be evaluated for 100 marks and grade point shall be awarded.

8.6 Out of a total of 100 marks for the UG major project, 25 marks shall be allotted for internal evaluation and 75 marks for the end semester examination (viva voce). The end semester examination of the project work shall be conducted by a committee consisting of external examiner, Head of the Department, supervisor of the project and a senior faculty member. The evaluation of UG major project shall be made at the end of IV year II semester. The internal evaluation shall be on the basis of two seminars given by each student on the topic of UG major project.

8.7 The laboratory marks and the sessional marks awarded by the college are subject to scrutiny and scaling by the university wherever necessary. In such cases, the sessional and laboratory marks awarded by the college will be referred to a committee. The committee will arrive at a scaling factor and the marks will be scaled accordingly. The recommendations of the committee are final and binding. The laboratory records and internal test papers shall be preserved in the respective institutions as per the university rules and produced before the committees of the university as and when asked for.

8.8 For mandatory courses environmental science, human values and professional ethics, gender sensitization lab and Industrial Training a student has to secure 40 marks out of 100 marks (i.e. 40% of the marks allotted) in the continuous internal evaluation for passing the subject/course.

8.9 For mandatory courses NCC/ NSO and NSS, a 'satisfactory participation certificate' shall be issued to the student from the authorities concerned, only after securing ≥ 65% attendance in such a course.

8.10 No marks or letter grade shall be allotted for all mandatory/non-credit courses.

9.0 Grading procedure

9.1 Marks will be awarded to indicate the performance of student in each theory subject, laboratory / practicals and UG major project. Based on the percentage of marks obtained (Continuous Internal Evaluation plus Semester End Examination, both taken together) as specified in item 8 above, a corresponding letter grade shall be given.

9.2 As a measure of the performance of student, a 10-point absolute grading system using the following letter grades (as per UGC/AICTE guidelines) and corresponding percentage of marks shall be followed:

% of Marks Secured in a Subject/Course (Class Intervals)	Letter Grade (UGC Guidelines)	Grade Points
Greater than or equal to 90%	O (Outstanding)	10

80 and less than 90%	A^+ (Excellent)	9
70 and less than 80%	A (Very Good)	8
60 and less than 70%	B^+ (Good)	7
50 and less than 60%	B (Average)	6
40 and less than 50%	C (Pass)	5
Below 40%	F (FAIL)	0
Absent	Ab	0

9.3 A student obtaining '**F**' grade in any subject shall be deemed to have '**failed**' and is required to reappear as a 'supplementary student' in the semester end examination, as and when offered. In such cases, internal marks in those subjects will remain the same as those obtained earlier.

9.4 A student who has not appeared for examination in any subject, '**Ab**' grade will be allocated in that subject, and student shall be considered '**failed**'. Student will be required to reappear as a 'supplementary student' in the semester end examination, as and when offered.

9.5 A letter grade does not indicate any specific percentage of marks secured by the student, but it indicates only the range of percentage of marks.

9.6 A student earns grade point (GP) in each subject/ course, on the basis of the letter grade secured in that subject/ course. The corresponding 'credit points' (CP) are computed by multiplying the grade point with credits for that particular subject/ course.

Credit points (CP) = grade point (GP) x credits .... For a course

9.7 The student passes the subject/ course only when **GP ≥ 5 ('C' grade or above)**

9.8 The semester grade point average (SGPA) is calculated by dividing the sum of credit points (ΣCP) secured from all subjects/ courses registered in a semester, by the total number of credits registered during that semester. SGPA is rounded off to **two** decimal places. SGPA is thus computed as

$$\text{SGPA} = \{ \sum_{i=1}^{N} C_i \, G_i \} / \{ \sum_{i=1}^{N} C_i \} \text{ .... For each semester,}$$

where 'i' is the subject indicator index (takes into account all subjects in a semester), 'N' is the no. of subjects '**registered**' for the semester (as specifically required and listed under the course structure of the parent department), C_i is the no. of credits allotted to the i^{th} subject, and G_i represents the grade points (GP) corresponding to the letter grade awarded for that i^{th} subject.

9.9 The cumulative grade point average (CGPA) is a measure of the overall cumulative performance of a student in all semesters considered for registration. The CGPA is the ratio of the total credit points secured by a student in **all** registered courses in **all** semesters, and the total number of credits registered in **all** the semesters. CGPA is rounded off to **two** decimal places. CGPA is thus computed from the I year II semester onwards at the end of each semester as per the formula

$$\text{CGPA} = \{ \sum_{j=1}^{M} C_j \, G_j \} / \{ \sum_{j=1}^{M} C_j \} \text{ ... for all S semesters registered}$$

(i.e., up to and inclusive of S semesters, S ≥ 2),

where '**M**' is the **total** no. of subjects the student has '**registered**' i.e., from the 1st semester onwards up to and inclusive of the 8th semester, 'j' is the subject indicator index (takes into account all subjects from 1 to 8 semesters), C_j is the no. of credits allotted to the j^{th} subject, and G_j represents the grade points (GP) corresponding to the letter grade awarded for that j^{th} subject. After registration and completion of first year first semester, the SGPA of that semester itself may be taken as the CGPA, as there are no cumulative effects.

Illustration of calculation of SGPA

Course/Subject	Credits	Letter Grade	Grade Points	Credit Points
Course 1	4	A	8	4 x 8 = 32
Course 2	4	O	10	4 x 10 = 40
Course 3	4	C	5	4 x 5 = 20
Course 4	3	B	6	3 x 6 = 18
Course 5	3	A+	9	3 x 9 = 27
Course 6	3	C	5	3 x 5 = 15
	Total Credits = 21			Total Credit Points = 152

SGPA = 152/21 = 7.24

Illustration of calculation of CGPA

Course/Subject	Credits	Letter Grade	Grade Points	Credit Points
I Year I Semester				
Course 1	4	A	8	4 x 8 = 32
Course 2	4	A+	9	4 x 9 = 36
Course 3	4	B	6	4 x 6 = 24
Course 4	3	O	10	3 x 10 = 30
Course 5	3	B+	7	3 x 7 = 21
Course 6	3	A	8	3 x 8 = 24
I Year II Semester				
Course 7	4	B+	7	4 x 7 = 28
Course 8	4	O	10	4 x 10 = 40
Course 9	4	A	8	4 x 8 = 32
Course 10	3	B	6	3 x 6 = 18
Course 11	3	C	5	3 x 5 = 15
Course 12	3	A+	9	3 x 9 = 27
	Total Credits = 42			Total Credit Points = 327

CGPA = 327/42 = 7.79

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9.10 For merit ranking or comparison purposes or any other listing, **only** the '**rounded off**' values of the CGPAs will be used.

9.11 For calculations listed in regulations 9.6 to 9.9, performance in failed subjects/ courses (securing **F** grade) will also be taken into account, and the credits of such subjects/ courses will also be included in the multiplications and summations. After passing the failed subject(s) newly secured letter grades will be taken into account for calculation of SGPA and CGPA. However, mandatory courses will not be taken into consideration.

10.0 Passing standards

10.1 A student shall be declared successful or 'passed' in a semester, if student secures a GP≥5 ('C' grade or above) in every subject/course in that semester (i.e. when student gets an SGPA ≥ 5.00 at the end of that particular semester); and a student shall be declared successful or 'passed' in the entire under graduate programme, only when gets a CGPA≥5.00 for the award of the degree as required.

10.2 After the completion of each semester, a grade card or grade sheet (or transcript) shall be issued to all the registered students of that semester, indicating the letter grades and credits earned. It will show the details of the courses registered (course code, title, no. of credits, and grade earned etc.), credits earned, SGPA, and CGPA.

11.0 Declaration of results

11.1 Computation of SGPA and CGPA are done using the procedure listed in 9.6 to 9.9.

11.2 For final percentage of marks equivalent to the computed final CGPA, the following formula may be used.

% of Marks = (final CGPA – 0.5) x 10

12.0 Award of degree

12.1 A student who registers for all the specified subjects/ courses as listed in the course structure and secures the required number of 196 credits (with CGPA ≥ 5.0), within 8 academic years from the date of commencement of the first academic year, shall be declared to have '**qualified**' for the award of the B.Pharm. degree.

12.2 A student who qualifies for the award of the degree as listed in item 12.1 shall be placed in the following classes.

12.3 Students with final CGPA (at the end of the under graduate programme) ≥ 8.00, and fulfilling the following conditions -

(i) Should have passed all the subjects/courses in '**first appearance**' within the first 4 academic years (or 8 sequential semesters) from the date of commencement of first year first semester.

(ii) Should have secured a CGPA ≥ 8.00, at the end of each of the 8 sequential semesters, starting from first year first semester onwards.

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(iii) Should not have been detained or prevented from writing the end semester examinations in any semester due to shortage of attendance or any other reason, shall be placed in **'first class with distinction'**.

12.4 Students with final CGPA (at the end of the under graduate programme) ≥ 6.50 but < 8.00, shall be placed in **'first class'**.

12.5 Students with final CGPA (at the end of the under graduate programme) ≥ 5.50 but < 6.50, shall be placed in **'second class'**.

12.6 All other students who qualify for the award of the degree (as per item 12.1), with final CGPA (at the end of the under graduate programme) ≥ 5.00 but < 5.50, shall be placed in **'pass class'**.

12.7 A student with final CGPA (at the end of the under graduate programme) < 5.00 will not be eligible for the award of the degree.

12.8 Students fulfilling the conditions listed under item 12.3 alone will be eligible for award of **'university rank'** and **'gold medal'**.

13.0 Withholding of results

13.1 If the student has not paid the fees to the university/ college at any stage, or has dues pending due to any reason whatsoever, or if any case of indiscipline is pending, the result of the student may be withheld, and student will not be allowed to go into the next higher semester. The award or issue of the degree may also be withheld in such cases.

14.0 Transitory regulations

A. **For students detained due to shortage of attendance**:

1. A Student who has been detained in I year of R09/R13/R15/R16 Regulations due to lack of attendance, shall be permitted to join I year I Semester of R17 Regulations and he is required to complete the study of B. Pharmacy programme within the stipulated period of eight academic years from the date of first admission in I Year.
2. A student who has been detained in any semester of II, III and IV years of R09/R13/R15/ R16 regulations for want of attendance, shall be permitted to join the corresponding semester of R17 regulations and is required to complete the study of B. Pharmacy within the stipulated period of eight academic years from the date of first admission in I Year. The R17 Academic Regulations under which a student has been readmitted shall be applicable to that student from that semester.

   See rule (C) for further Transitory Regulations.

B. **For students detained due to shortage of credits**:

3. A student of R09/R13/R15/R16 Regulations who has been detained due to lack of credits, shall be promoted to the next semester of R17 Regulations only after acquiring the required credits as per the corresponding regulations of his/her first admission. The student is required to complete the study of B. Pharmacy within the stipulated period of

eight academic years from the year of first admission. The R17 Academic Regulations are applicable to a student from the year of readmission onwards.

See rule (C) for further Transitory Regulations.

C. **For readmitted students in R17 Regulations**:

4. A student who has failed in any subject under any regulation has to pass those subjects in the same regulations.

5. The maximum credits that a student acquires for the award of degree, shall be the sum of the total number of credits secured in all the regulations of his/her study including R17 Regulations. The performance evaluation of the student will be done after the exemption of two subjects if total credits acquired are ≤ 206, three subjects if total credits acquired are > 206 (see R17 Regulations for exemption details).

6. If a student readmitted to R17 Regulations, has any subject with 80% of syllabus common with his/her previous regulations, that particular subject in R17 Regulations will be substituted by another subject to be suggested by the University.

Note: If a student readmitted to R17 Regulations, has not studied any subjects/topics in his/her earlier regulations of study which is prerequisite for further subjects in R17 Regulations, the College Principals concerned shall conduct remedial classes to cover those subjects/topics for the benefit of the students.

15.0 Student transfers

15.1 There shall be no branch transfers after the completion of admission process.

15.2 There shall be no transfers from one college/stream to another within the constituent colleges and units of Jawaharlal Nehru Technological University Hyderabad.

15.3 The students seeking transfer to colleges affiliated to JNTUH from various other Universities/institutions have to pass the failed subjects which are equivalent to the subjects of JNTUH, and also pass the subjects of JNTUH which the students have not studied at the earlier institution. Further, though the students have passed some of the subjects at the earlier institutions, if the same subjects are prescribed in different semesters of JNTUH, the students have to study those subjects in JNTUH in spite of the fact that those subjects are repeated.

15.4 The transferred students from other Universities/institutions to JNTUH affiliated colleges who are on rolls to be provide one chance to write the CBT (internal marks) in the **failed subjects and/or subjects not studied** as per the clearance letter issued by the university.

15.5 The autonomous affiliated colleges have to provide one chance to write the internal examinations in the **failed subjects and/or subjects not studied,** to the students transferred from other universities/institutions to JNTUH autonomous affiliated colleges who are on rolls, as per the clearance (equivalence) letter issued by the University.

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16.0 Scope

16.1 The academic regulations should be read as a whole, for the purpose of any interpretation.

16.2 In case of any doubt or ambiguity in the interpretation of the above rules, the decision of the Vice-Chancellor is final.

16.3 The university may change or amend the academic regulations, course structure or syllabi at any time, and the changes or amendments made shall be applicable to all students with effect from the date notified by the university authorities.

JAWAHARLAL NEHRU TECHNOLOGICAL UNIVERSITY HYDERABAD

(Established by Act No. 30 of 2008)

Kukatpally, Hyderabad, Telangana (India).

Academic Regulations for B.Pharm. (Lateral Entry Scheme) w.e.f the AY 2018-19

1. Eligibility for award of B. Pharm. Degree (LES)

The LES students after securing admission shall pursue a course of study for not less than three academic years and not more than six academic years.

2. The student shall register for 147 credits and secure 147 credits with CGPA ≥ 5 from II year to IV year B.Pharm. programme (LES) for the award of B.Pharm. degree. **Out of the 147 credits secured, the student can avail exemption up to 6 credits**, that is, two open elective subjects resulting in 141 credits for B.Pharm programme performance evaluation.

3. The students, who fail to fulfil the requirement for the award of the degree in six academic years from the year of admission, shall forfeit their seat in B.Pharm.

4. The attendance requirements of B. Pharm. (Regular) shall be applicable to B.Pharm. (LES).

5. Promotion rule

S. No	Promotion	Conditions to be fulfilled
1	Second year first semester to second year second semester	Regular course of study of second year first semester.
2	Second year second semester to third year first semester	(i) Regular course of study of second year second semester. (ii) Must have secured at least 29 credits out of 48 credits i.e., 60% of credits up

		to second year second semester from all the relevant regular and supplementary examinations, whether the student takes those examinations or not.
3	**Third year first semester to third year second semester**	**Regular course of study of third year first semester.**
4	**Third year second semester to fourth year first semester**	**(i) Regular course of study of third year second semester.** **(ii) Must have secured at least 58 credits out of 96 credits i.e., 60% of credits up to third year second semester from all the relevant regular and supplementary examinations, whether the student takes those examinations or not.**
5	**Fourth year first semester to fourth year second semester**	**Regular course of study of fourth year first semester.**

6. All the other regulations as applicable to B. Pharm. 4-year degree course (Regular) will hold good for B. Pharm. (Lateral Entry Scheme).

MALPRACTICES RULES

DISCIPLINARY ACTION FOR / IMPROPER CONDUCT IN EXAMINATIONS

	Nature of Malpractice/Improper conduct	Punishment
	If the student:	
1. (a)	Possesses or keeps accessible in examination hall, any paper, note book, programmable calculators, cell phones, pager, palm computers or any other form of material concerned with or related to the subject of the examination (theory or practical) in which student is appearing but has not made use of (material shall include any marks on the body of the student which can be used as an aid in the subject of the examination)	Expulsion from the examination hall and cancellation of the performance in that subject only.
(b)	Gives assistance or guidance or receives it from any other student orally or by any other body language methods or	Expulsion from the examination hall and cancellation of the performance in that subject only of all the students involved. In case of an

	communicates through cell phones with any student or persons in or outside the exam hall in respect of any matter.	outsider, he will be handed over to the police and a case is registered against him.
2.	Has copied in the examination hall from any paper, book, programmable calculators, palm computers or any other form of material relevant to the subject of the examination (theory or practical) in which the student is appearing.	Expulsion from the examination hall and cancellation of the performance in that subject and all other subjects the student has already appeared including practical examinations and UG major project and shall not be permitted to appear for the remaining examinations of the subjects of that semester/year. The hall ticket of the student is to be cancelled and sent to the university.
3.	Impersonates any other student in connection with the examination.	The student who has impersonated shall be expelled from examination hall. The student is also debarred and forfeits the seat. The performance of the original student who has been impersonated, shall be cancelled in all the subjects of the examination (including practicals and UG major project) already appeared and shall not be allowed to appear for examinations of the remaining subjects of that semester/year. The student is also debarred for two consecutive semesters from class work and all university examinations. The continuation of the course by the student is subject to the academic regulations in connection with forfeiture of seat. If the imposter is an outsider, he will be handed over to the police and a case is registered against him.
4.	Smuggles in the answer book or additional sheet or takes out or arranges to send out the question paper during the examination or answer book or additional sheet, during or after the examination.	Expulsion from the examination hall and cancellation of performance in that subject and all the other subjects the student has already appeared including practical examinations and UG major project and shall not be permitted for the remaining examinations of the subjects of that semester/year. The student is also debarred for two consecutive semesters from class work and all university examinations. The continuation of the course by the student is subject to the academic regulations in connection with forfeiture of seat.
5.	Uses objectionable, abusive or offensive language in the answer paper or in letters to the examiners or writes to the examiner requesting him to award pass marks.	Cancellation of the performance in that subject.

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6.	Refuses to obey the orders of the chief superintendent/assistant – superintendent / any officer on duty or misbehaves or creates disturbance of any kind in and around the examination hall or organizes a walk out or instigates others to walk out, or threatens the officer-in charge or any person on duty in or outside the examination hall of any injury to his person or to any of his relations whether by words, either spoken or written or by signs or by visible representation, assaults the officer-in-charge, or any person on duty in or outside the examination hall or any of his relations, or indulges in any other act of misconduct or mischief which result in damage to or destruction of property in the examination hall or any part of the college campus or engages in any other act which in the opinion of the officer on duty amounts to use of unfair means or misconduct or has the tendency to disrupt the orderly conduct of the examination.	In case of students of the college, they shall be expelled from examination halls and cancellation of their performance in that subject and all other subjects the student(s) has (have) already appeared and shall not be permitted to appear for the remaining examinations of the subjects of that semester/year. The students also are debarred and forfeit their seats. In case of outsiders, they will be handed over to the police and a police case is registered against them.
7.	Leaves the exam hall taking away answer script or intentionally tears of the script or any part thereof inside or outside the examination hall.	Expulsion from the examination hall and cancellation of performance in that subject and all the other subjects the student has already appeared including practical examinations and UG major project and shall not be permitted for the remaining examinations of the subjects of that semester/year. The student is also debarred for two consecutive semesters from class work and all university examinations. The continuation of the course by the student is subject to the academic regulations in connection with forfeiture of seat.
8.	Possess any lethal weapon or firearm in the examination hall.	Expulsion from the examination hall and cancellation of the performance in that subject and all other subjects the student has already appeared including practical examinations and UG major project and shall not be permitted for the remaining examinations of the subjects of that semester/year. The student is also debarred and forfeits the seat.
9.	If student of the college, who is not a student for the particular examination or	Student of the colleges expulsion from the examination hall and cancellation of the

	any person not connected with the college indulges in any malpractice or improper conduct mentioned in clause 6 to 8.	performance in that subject and all other subjects the student has already appeared including practical examinations and UG major project and shall not be permitted for the remaining examinations of the subjects of that semester/year. The student is also debarred and forfeits the seat. Person(s) who do not belong to the college will be handed over to police and, a police case will be registered against them.
10.	Comes in a drunken condition to the examination hall.	Expulsion from the examination hall and cancellation of the performance in that subject and all other subjects the student has already appeared including practical examinations and UG major project and shall not be permitted for the remaining examinations of the subjects of that semester/year.
11.	Copying detected on the basis of internal evidence, such as, during valuation or during special scrutiny.	Cancellation of the performance in that subject and all other subjects the student has appeared including practical examinations and UG major project of that semester/year examinations.
12.	If any malpractice is detected which is not covered in the above clauses 1 to 11 shall be reported to the university for further action to award suitable punishment.	

Malpractices identified by squad or special invigilators

1. Punishments to the students as per the above guidelines.
2. Punishment for institutions : (if the squad reports that the college is also involved in encouraging malpractices)
   a. A show cause notice shall be issued to the college.
   b. Impose a suitable fine on the college.
   c. Shifting the examination centre from the college to another college for a specific period of not less than one year.

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JAWAHARLAL NEHRU TECHNOLOGICAL UNIVERSITY HYDERABAD
(Established by Act No.30 of 2008)
Kukatpally, Hyderabad–500085, Telangana State (India)

Academic Regulations of M.Pharm. (Regular/Full Time) Programmes, 2019-20 (R19)
(CBCS)
(Effective for the students admitted into I year from the Academic Year 2019-20 and onwards)

1.0 **Post-Graduate Degree Programmes in Pharmacy (PGP in Pharmacy)** Jawaharlal Nehru Technological University Hyderabad (JNTUH) offers **Two** Years (**Four** Semesters) full-time Master of Pharmacy (M.Pharm.) Degree programmes, under Choice Based Credit System (CBCS) at its constituent (non-autonomous) and affiliated colleges in different specializations.

2.0 **Eligibility for Admissions**

2.1 Admission to the PGPs shall be made subject to eligibility, qualification and specializations prescribed by the University from time to time, for each specialization under each M.Pharm. programme.

2.2 Admission to the post graduate programme shall be made on the basis of either the merit rank or Percentile obtained by the qualified student in the relevant qualifying GPAT Examination/ the merit rank obtained by the qualified student in an entrance test conducted by Telangana State Government (PGECET) for M.Pharm. programmes / an entrance test conducted by JNTUH/ on the basis of any other exams approved by the University, subject to reservations as laid down by the Govt. from time to time.

2.3 The medium of instructions for all PG Programmes will be **ENGLISH** only.

3.0 **M.Pharm. Programme (PGP in Pharmacy) Structure**

3.1 The M.Pharm. Programmes in Pharmacy of JNTUH are of Semester pattern, with **Four** Semesters consisting of **Two** academic years, each academic year having **Two** Semesters (First/Odd and Second/Even Semesters). Each Semester shall be of 22 weeks duration (inclusive of Examinations), with a minimum of 90 instructional days per Semester.

3.2 The student shall not take more than four academic years to fulfill all the academic requirements for the award of M.Pharm. degree from the date of commencement of first year first semester, failing which the student shall forfeit the seat in M.Pharm. programme.

3.3 **UGC/AICTE** specified definitions/descriptions are adopted appropriately for various terms and abbreviations used in these PG academic regulations, as listed below:

3.3.1 **Semester Scheme**

Each Semester shall have 'Continuous Internal Evaluation (CIE)' and 'Semester End Examination (SEE)'. Choice Based Credit System (CBCS) and Credit Based Semester System (CBSS) are taken as 'references' for the present set of Regulations. The terms 'SUBJECT' and 'COURSE' imply the same meaning here and refer to 'Theory Subject', or 'Lab Course', or 'Design/Drawing Subject', or 'Mini Project with Seminar', or 'Dissertation', as the case may be.

3.3.2 **Credit Courses**

All subjects/courses are to be registered by the student in a semester to earn credits which shall be assigned to each subject/course in an L: T: P: C (Lecture Periods: Tutorial Periods: Practical Periods: Credits) structure based on the following general pattern:

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- One credit for one hour/week/semester for theory/lecture (L) courses
- One credit for two hours/ week/semester for laboratory/ practical (P) courses or tutorials (T)

Other student activities like study tour, guest lecture, conference/workshop participations, technical paper presentations and mandatory courses (***Audit Courses***) will not carry any credits.

3.3.3 Subject Course Classification

All subjects/courses offered for the Post-Graduate Programme in Pharmacy (M.Pharm. Degree Programme) are broadly classified as follows. The University has followed in general the guidelines issued by AICTE/UGC.

S.No.	Broad Course Classification	Course Group/ Category	Course Description
1	Core Courses (CoC)	PC- Professional Core	Includes subjects related to the Specialization in Pharmacy
		Dissertation	M.Pharm. Project or PG Project or Major Project
		Mini Project with Seminar	Seminar based on core contents related to the Specialization in Pharmacy
2	Elective Courses (EIE)	PE - Professional Electives	Includes elective subjects related to the Specialization in Pharmacy
		OE - Open Electives	Elective subjects which include inter-disciplinary subjects or subjects in an area outside the Specialization in Pharmacy
3	***Mandatory Courses***	--	***Non-Credit Audit Courses***

4.0 Course Registration

4.1 A 'Faculty Advisor or Counselor' shall be assigned to each specialization, who will advise on the Post Graduate Programme (PGP), its Course Structure and Curriculum, Choice/Option for Subjects/ Courses, based on his competence, progress, pre-requisites and interest.

4.2 The Academic Section of the College invites 'Registration Forms' from students within 15 days from the commencement of class work through 'ON-LINE SUBMISSIONS', ensuring 'DATE and TIME Stamping'. The ON-LINE Registration Requests for any 'CURRENT SEMESTER' shall be completed BEFORE the commencement of SEEs (Semester End Examinations) of the 'PRECEDING SEMESTER'.

4.3 A Student can apply for ON-LINE Registration, ONLY AFTER obtaining the 'WRITTEN APPROVAL' from his Faculty Advisor, which should be submitted to the College Academic Section through the Head of Department (a copy of it being retained with Head of Department, Faculty Advisor and the Student).

4.4 If the Student submits ambiguous choices or multiple options or erroneous entries during ON-LINE Registration for the Subject(s) / Course(s) under a given/ specified Course Group/ Category as listed in the Course Structure, only the first mentioned Subject/ Course in that Category will be taken into consideration.

4.5 Subject/ Course Options exercised through ON-LINE Registration are final and CANNOT be changed, nor can they be inter-changed; further, alternate choices also will not be considered. However, if the Subject/ Course that has already been listed for Registration by the University in a Semester could not be offered due to unforeseen or unexpected reasons, then the Student will be allowed to have alternate

choice either for a new Subject, if it is offered, or for another existing Subject (subject to availability of seats). Such alternate arrangements will be made by the Head of Department, with due notification and time-framed schedule, within the FIRST WEEK from the commencement of Class-work for that Semester.

5.0 Attendance Requirements

The programmes are offered based on a unit system with each subject being considered a unit. Attendance is calculated separately for each subject.

5.1 Attendance in all classes (Lectures/Laboratories) is compulsory. The minimum required attendance in each theory subject (***also mandatory(audit) courses***) including the attendance of mid-term examination / Laboratory etc. is 75%. Two periods of attendance for each theory subject shall be considered, if the student appears for the mid-term examination of that subject. ***This attendance should also be included in the fortnightly upload of attendance to the University. The attendance of mandatory(audit) courses should be uploaded separately to the University.*** A student shall not be permitted to appear for the Semester End Examinations (SEE), if his attendance is less than 75%.

5.2 A student's Seminar report and presentation on Mini Project shall be eligible for evaluation, only if he ensures a minimum of 75% of his attendance in Seminar presentation classes on Mini Project during that Semester.

5.3 **Condoning of shortage of attendance** (between 65% and 75%) up to a maximum of 10% (considering the days of attendance in sports, games, NCC, NSS activities and Medical grounds) in each subject (Theory/Lab/Mini Project with Seminar) of a semester shall be granted by the College Academic Committee on genuine reasons.

5.4 A prescribed fee per subject shall be payable for condoning shortage of attendance after getting the approval of College Academic Committee for the same. The College Academic Committee shall maintain relevant documents along with the request from the student.

5.5 Shortage of Attendance below 65% in any subject shall in **no case be condoned.**

5.6 A Student, whose shortage of attendance is not condoned in any Subject(s) (Theory/Lab/Mini Project with Seminar) in any Semester, is considered as 'Detained in that Subject(s), and is not eligible to write Semester End Examination(s) of such Subject(s), (in case of Mini Project with Seminar, his/her Mini Project with Seminar Report or Presentation are not eligible for evaluation) in that Semester; and he/she has to seek re-registration for those Subject(s) in subsequent Semesters, and attend the same as and when offered.

5.7 A student fulfills the attendance requirement in the present semester, shall not be eligible for readmission into the same class.

5.8 **a)** A student shall put in a minimum required attendance in at least **three theory subjects (excluding *mandatory(audit)* course)** in first Year I semester for promotion to first Year II Semester.

b) A student shall put in a minimum required attendance in at least **three theory subjects (excluding *mandatory(audit)* course)** in first Year II semester for promotion to second Year I Semester.

6.0 Academic Requirements

The following academic requirements must be satisfied, in addition to the attendance requirements mentioned in item no. 5. The performance of the candidate in each semester shall be evaluated subject-

wise, with a maximum of 100 marks per subject / course (theory / practical), based on Internal Evaluation and Semester End Examination.

6.1 A student shall be deemed to have satisfied the academic requirements and earned the credits allotted to each subject/course, if he secures not less than 40% of marks (30 out of 75 marks) in the End Semester Examination, and a minimum of 50% of marks in the sum total of CIE (Continuous Internal Evaluation) and SEE (Semester End Examination) taken together; in terms of Letter Grades and this implies securing 'B' Grade or above in a subject.

6.2 A student shall be deemed to have satisfied the academic requirements and earned the credits allotted to Mini Project with seminar, if student secures not less than 50% marks (i.e. 50 out of 100 allotted marks). The student would be treated as failed, if student (i) does not submit a seminar report on Mini Project or does not make a presentation of the same before the evaluation committee as per schedule or (ii) secures less than 50% marks in Mini Project with seminar evaluation. The failed student shall reappear for the above evaluation when the notification for supplementary examination is issued.

6.3 A student shall register for all subjects for total of **68** credits as specified and listed in the course structure for the chosen specialization, put in required the attendance and fulfill the academic requirements for securing **68** credits obtaining a minimum of 'B' Grade or above in each subject, and all **68** credits securing Semester Grade Point Average **(SGPA)** ≥**6.0** (in each semester) and final Cumulative Grade Point Average **(CGPA)** (i.e., CGPA at the end of PGP) ≥ **6.0**, and shall ***pass all the mandatory(audit) courses*** to complete the PGP successfully.

Note: (1) The SGPA will be computed and printed on the marks memo only if the candidate passes in all the subjects offered and gets minimum B grade in all the subjects.

(2) CGPA is calculated only when the candidate passes in all the subjects offered in all the semesters

6.4 Marks and Letter Grades obtained in all those subjects covering the above specified **68** credits alone shall be considered for the calculation of final CGPA, which will be indicated in the Grade Card /Marks Memo of second year second semester.

6.5 If a student registers for extra subject(s) (in the parent specialization or other specializations of Pharmacy) other than those listed subjects totaling to **68** credits as specified in the course structure, the performance in extra subject(s) (although evaluated and graded using the same procedure as that of the required **68** credits) will not be considered while calculating the SGPA and CGPA. For such extra subject(s) registered, percentage of marks and Letter Grade alone will be indicated in the Grade Card/Marks Memo, as a performance measure, subject to completion of the attendance and academic requirements as stated in items 5 and 6.1 - 6.3.

6.6 When a student is detained due to shortage of attendance in any subject(s) in any semester, no Grade allotment will be made for such subject(s). However, he is eligible for re-registration of such subject(s) in the subsequent semester(s), as and when next offered, with the academic regulations of the batch into which he is re-registered, by paying the prescribed fees per subject. In all these re-registration cases, the student shall have to secure a fresh set of internal marks and Semester End Examination marks for performance evaluation in such subject(s), and SGPA/CGPA calculations.

6.7 A student eligible to appear for the Semester End Examination in any subject, but absent from it or failed (failing to secure 'B' Grade or above), may reappear for that subject at the supplementary examination as and when conducted. In such cases, his Internal Marks assessed earlier for that subject will be carried over, and added to the marks secured in the supplementary examination, for

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the purpose of evaluating his performance in that subject.

6. 8 A Student who fails to earn **68** credits as per the specified course structure, and as indicated above, within **four** academic years from the date of commencement of his first year first semester, shall forfeit his seat in M.Pharm. programme and his admission **shall stand cancelled.**

7.0 Evaluation - Distribution and Weightage of Marks

The performance of a student in each semester shall be evaluated subject- wise (irrespective of credits assigned) for a maximum of 100 marks.

7.1 For the theory subjects 75 marks shall be awarded for the performance in the Semester End Examination and 25 marks shall be awarded for Continuous Internal Evaluation (CIE). The Continuous Internal Evaluation shall be made based on the average of the marks secured in the two Mid-Term Examinations conducted, first Mid-Term examinations in the middle of the Semester and second Mid-Term examinations during the last week of instruction. Each Mid-Term Examination shall be conducted for a total duration of 120 minutes with Part 'A' as compulsory consisting of 5 questions carrying 2 marks each (10 marks), and Part 'B' with 3 questions to be answered out of 5 questions, each question carrying 5 marks (15 marks). The details of the Question Paper pattern for Semester End Examination (Theory) are given below:

- The Semester End Examination will be conducted for 75 marks. It consists of two parts.

  i) Part A for 25 marks, ii) Part B for 50 marks.
- Part A is compulsory and consists of 5 questions, one from each unit and carrying 5 marks each.
- Part B consists of 5 questions carrying 10 marks each. There will be two questions from each unit and only one should be answered.

7.2 For practical subjects, 75 marks shall be awarded for performance in the Semester End Examinations and 25 marks shall be awarded for day-to-day performance as Internal Marks.

7.3 For conducting laboratory end examinations of all PG Programmes, one internal examiner and one external examiner are to be appointed by the Principal of the College and this is to be informed to the Director of Evaluation within two weeks, before commencement of the lab end examinations. The external examiner should be selected from outside the College concerned but within the cluster. No external examiner should be appointed from any other College in the same cluster/any other cluster which is run by the same Management.

7.4 There shall be Mini Project with Seminar during I year II semester for internal evaluation of 100 marks. The Departmental Academic Committee (DAC) will review the progress of the mini project during the seminar presentations and evaluate the same for 50 marks. Mini Project Viva Voce will be evaluated by the DAC for another 50 marks before the semester end examinations. Student shall carryout the mini project in consultation with the mini project supervisor which may include critically reviewing the literature, project implementation and submit it to the department in the form of a report and shall make an oral presentation before the DAC consisting of Head of the Department, Mini Project supervisor and two other senior faculty members of the department. The student has to secure a minimum of 50% of marks in i) seminar presentation and ii) mini project viva voce, to be declared successful. If he fails to obtain the minimum marks, he has to reappear for the same as and when scheduled.

7.5 Every candidate shall be required to submit a dissertation on a topic approved by the Dissertation Review Committee.

7.6 A Dissertation Review Committee (DRC) shall be constituted with the Head of the Department as

Chairperson, Dissertation Supervisor and one senior faculty member of the Department offering the M.Pharm. programme.

7.7 Registration of Dissertation Work: A candidate is permitted to register for the Dissertation Work after satisfying the attendance requirement in all the subjects, both theory and laboratory.

7.8 After satisfying 7.7, a candidate must present in Dissertation Work Review - I, in consultation with his Dissertation Supervisor, the title, objective and plan of action of his Dissertation work to the Dissertation Review Committee (DRC) for approval within four weeks from the commencement of Second year First Semester. Only after obtaining the approval of the DRC can the student initiate the Dissertation work.

7.9 If a candidate wishes to change his supervisor or topic of the Dissertation, he can do so with the approval of the DRC. However, the DRC shall examine whether or not the change of topic/supervisor leads to a major change of his initial plans of Dissertation proposal. If yes, his date of registration for the project work starts from the date of change of Supervisor or topic as the case may be.

7.10 A candidate shall submit his Dissertation progress report in two stages at least with a gap of **three** months between them.

7.11 The work on the Dissertation shall be initiated at the beginning of the II year and the duration of the Dissertation is two semesters. A candidate is permitted to submit Dissertation Thesis only after successful completion of all theory and practical courses with the approval of DRC not earlier than 40 weeks from the date of approval of the Dissertation work. For the approval of DRC the candidate shall submit the draft copy of thesis to the Head of the Department and make an oral presentation before the DRC.

7.12 The Dissertation Work Review - II in II Year I Sem. carries internal marks of 100. Evaluation should be done by the DRC for 50 marks and the Supervisor will evaluate the work for the other 50 marks. The Supervisor and DRC will examine the Problem Definition, Objectives, Scope of Work, Literature Survey in the same domain and progress of the Dissertation Work. A candidate has to secure a minimum of 50% of marks to be declared successful in Dissertation Work Review - II. If he fails to obtain the minimum required marks, he has to reappear for Dissertation Work Review - II as and when conducted.

7.13 The Dissertation Work Review - III in II Year II Sem. carries 100 internal marks. Evaluation should be done by the DRC for 50 marks and the Supervisor will evaluate it for the other 50 marks. The DRC will examine the overall progress of the Dissertation Work and decide whether or not the Dissertation is eligible for final submission. A candidate has to secure a minimum of 50% of marks to be declared successful in Dissertation Work Review - III. If he fails to obtain the required minimum marks, he has to reappear for Dissertation Work Review - III as and when conducted. For Dissertation Evaluation (Viva Voce) in II Year II Sem. there are external marks of 100 and it is evaluated by the external examiner. The candidate has to secure a minimum of 50% marks in Dissertation Evaluation (Viva-Voce) examination.

7.14 Dissertation Work Reviews - II and III shall be conducted in phase I (Regular) and Phase II (Supplementary). Phase II will be conducted only for unsuccessful students in Phase I. The unsuccessful students in Dissertation Work Review - II (Phase II) shall reappear for it at the time of Dissertation Work Review - III (Phase I). These students shall reappear for Dissertation Work Review - III in the next academic year at the time of Dissertation Work Review - II only after completion of Dissertation Work Review - II, and then Dissertation Work Review - III follows. The unsuccessful students in Dissertation Work Review - III (Phase II) shall reappear for Dissertation Work Review - III in the next academic year only at the time of Dissertation Work Review - II (Phase I).

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7.15 After approval from the DRC, a soft copy of the thesis should be submitted for ANTI-PLAGIARISM check and the plagiarism report should be submitted to the University and be included in the final thesis. The Thesis will be accepted for submission, if the similarity index is less than **30%**. If the similarity index has more than the required_percentage, the student is advised to modify accordingly and re-submit the soft copy of the thesis after one month. The maximum number of re-submissions of thesis after plagiarism check is limited to TWO. The candidate has to register for the Dissertation work and work for two semesters. After three attempts, the admission is liable to be cancelled. The college authorities are advised to make plagiarism check of every soft copy of theses before submissions.

7.16 Three copies of the Dissertation Thesis certified by the supervisor shall be submitted to the College/School/Institute, after submission of a research paper related to the Dissertation work in a UGC approved journal. A copy of the submitted research paper shall be attached to thesis.

7.17 The thesis shall be adjudicated by an external examiner selected by the University. For this, the Principal of the College/School/Institute shall submit a panel of **three** examiners from among the list of experts in the relevant specialization as submitted by the supervisor concerned and Head of the Department.

7.18 If the report of the external examiner is unsatisfactory, the candidate shall revise and resubmit the Thesis. If the report of the examiner is unsatisfactory again, the thesis shall be summarily rejected. Subsequent actions for such dissertations may be considered, only on the specific recommendations of the external examiner and /or Dissertation Review Committee. No further correspondence in this matter will be entertained, if there is no specific recommendation for resubmission.

7.19 If the report of the examiner is satisfactory, the Head of the Department shall coordinate and make arrangements for the conduct of Dissertation Viva-Voce examination. The Dissertation Viva-Voce examination shall be conducted by a board consisting of the Supervisor, Head of the Department and the external examiner who adjudicated the Thesis. The candidate has to secure a minimum of 50% of marks in Dissertation Evaluation (Viva-Voce) examination.

7.20 If he fails to fulfill the requirements as specified in 7.19, he will reappear for the Dissertation Viva-Voce examination only after three months. In the reappeared examination also, if he fails to fulfill the requirements, he will not be eligible for the award of the degree, unless he is asked to revise and resubmit his Dissertation Work by the board within a specified time period (within **four** years from the date of commencement of his first year first semester).

7.21 The Dissertation Viva-Voce External examination marks must be submitted to the University on the day of the examination.

7.22 ***For mandatory(audit) courses, a student has to secure 40 marks out of 100 marks (i.e. 40% of the marks allotted) in the continuous internal evaluation for passing the subject/course. These marks should also be uploaded along with the internal marks of other subjects.***

7.23 ***No marks or letter grades shall be allotted for mandatory(audit) courses. Only Pass/Fail shall be indicated in Grade Card.***

8.0 Re-Admission/Re-Registration

8.1 Re-Admission for Discontinued Student

A student, who has discontinued the M.Pharm. degree programme due to any reason whatsoever, may be considered for **'readmission'** into the same degree programme (with the same specialization) with the academic regulations of the batch into which he gets readmitted, with prior permission from the authorities concerned, subject to item 6.6.

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8.2 If a student is detained in a subject (s) due to shortage of attendance in any semester, he may be permitted to **re-register** for the same subject(s) in the same category (core or elective group) or equivalent subject, if the same subject is not available, as suggested by the Board of Studies of that department, as and when offered in the subsequent semester(s), with the academic regulations of the batch into which he seeks re-registration, with prior permission from the authorities concerned, subject to item 3.2

8.3 A candidate shall be given one chance to re-register and attend the classes for a maximum of two subjects, if the internal marks secured by a candidate are less than 50% and failed in those subjects but fulfilled the attendance requirement. A candidate must re-register for failed subjects within four weeks of commencement of the class work and secure the required minimum attendance. In the event of the student taking this chance, his Continuous Internal Evaluation (internal) marks and Semester End Examination marks obtained in the previous attempt stand cancelled.

9.0 Examinations and Assessment - The Grading System

9.1 Grades will be awarded to indicate the performance of each student in each Theory Subject, or Lab/Practicals, or Mini Project with Seminar, Dissertation, etc., based on the percentage of marks obtained in CIE + SEE (Continuous Internal Evaluation + Semester End Examination, both taken together) as specified in Item 7 above, and a corresponding Letter Grade shall be given.

9.2 As a measure of the student's performance, a 10-point Absolute Grading System using the following Letter Grades (UGC Guidelines) and corresponding percentage of marks shall be followed:

% of Marks Secured in a subject/Course (Class Intervals)	**Letter Grade (UGC Guidelines)**	**Grade Points**
90% and above (≥ 90%, ≤ 100%)	O (Outstanding)	10
Below 90% but not less than 80% (≥80%, <90%)	A^+ (Excellent)	9
Below 80% but not less than 70% (≥70%, <80%)	A (Very Good)	8
Below 70% but not less than 60% (≥60%, <70%)	B^+ (Good)	7
Below 60% but not less than 50% (≥ 50%, <60%)	B (above Average)	6
Below 50% (< 50%)	F (FAIL)	0
Absent	**Ab**	**0**

9.3 A student obtaining F Grade in any Subject is deemed to have 'failed' and is required to reappear as 'Supplementary Candidate' for the Semester End Examination (SEE), as and when conducted. In such cases, his Internal Marks (CIE Marks) in those subjects will remain as obtained earlier.

9.4 If a student has not appeared for the examinations, 'Ab' Grade will be allocated to him for any subject and shall be considered 'failed' and will be required to reappear as 'Supplementary Candidate' for the Semester End Examination (SEE), as and when conducted.

9.5 A Letter Grade does not imply any specific marks percentage; it is only the range of percentage of marks.

9.6 In general, a student shall not be permitted to repeat any Subject/ Course (s) only for the sake of 'Grade Improvement' or 'SGPA/ CGPA Improvement'.

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9.7 A student earns Grade Point (GP) in each Subject/ Course, on the basis of the Letter Grade obtained by him in that Subject/ Course. The corresponding 'Credit Points' (CP) are computed by multiplying the Grade Point with Credits for that particular Subject/ Course.

Credit Points (CP) = Grade Point (GP) x Credits .... For a Course

9.8 The student passes the Subject/ Course only when he **gets GP ≥6 (B Grade or above).**

9.9 The Semester Grade Point Average (SGPA) is calculated by dividing the Sum of Credit Points (ΣCP) secured from ALL Subjects/ Courses registered in a Semester, by the Total Number of Credits registered during that Semester. SGPA is rounded off to TWO Decimal Places. SGPA is thus computed as

$$\text{SGPA} = \{ \sum_{i=1}^{N} C_i G_i \} / \{ \sum_{i=1}^{N} C_i \} \text{ .... For each Semester,}$$

where 'i' is the Subject indicator index (taking into account all Subjects in a Semester), 'N' is the no. of Subjects 'REGISTERED' for the Semester (as specifically required and listed under the Course Structure of the parent Department), C_i is the no. of Credits allotted to the i^{th} Subject, and G_i represents the Grade Points (GP) corresponding to the Letter Grade awarded for that i^{th} Subject.

9.10 The Cumulative Grade Point Average (CGPA) is a measure of the overall cumulative performance of a student over all Semesters considered for registration. The CGPA is the ratio of the Total Credit Points secured by a student in ALL registered Courses in ALL Semesters, and the Total Number of Credits registered in ALL the Semesters. CGPA is rounded off to TWO Decimal Places. CGPA is thus computed from the I Year Second Semester onwards, at the end of each Semester, as per the formula

$$\text{CGPA} = \{ \sum_{i=1}^{M} C_j G_j \} / \{ \sum_{i=1}^{M} C_j \} \text{ ... for all S Semesters registered}$$

(ie., upto and inclusive of S Semesters, S ≥2),

where 'M' is the TOTAL no. of Subjects (as specifically required and listed under the Course Structure of the parent Department) the Student has 'REGISTERED' for from the 1st Semester onwards upto and inclusive of the Semester S (obviously M > N), 'j' is the Subject indicator index (taking into account all Subjects from 1 to S Semesters), C_j is the no. of Credits allotted to the j^{th} Subject, and G_j represents the Grade Points (GP) corresponding to the Letter Grade awarded for that j^{th} Subject. After registration and completion of I Year I Semester however, the SGPA of that Semester itself may be taken as the CGPA, as there are no cumulative effects.

Illustration of calculation of SGPA

Course/Subject	Credits	Letter Grade	Grade points	Credit Points
Course 1	4	A	8	4*8 = 32
Course 2	4	O	10	4*10 = 40
Course 3	4	B	6	4*6 = 24
Course 4	3	B	6	3*6 = 18
Course 5	3	A+	9	3*9 = 27
Course 6	3	B	6	3*6 = 18
	21			159

SGPA = 159/21 = 7.57

Illustration of calculation of CGPA

Semester	Credits	SGPA	Credits * SGPA
Semester I	24	7	24*7 = 168
Semester II	24	6	24*6 = 144
Semester III	24	6.5	24*6.5 = 156
Semester IV	24	6	24*6 = 144
	96		612

CGPA = 612/96 = 6.37

10.0 Award of Degree and Class

10.1 If a student who registers for all the specified Subjects/ Courses as listed in the Course Structure, satisfies all the Course Requirements, and passes the examinations prescribed in the entire PG Programme (PGP), and secures the required number of **68** Credits (with CGPA ≥6.0), shall be declared to have 'QUALIFIED' for the award of the M.Pharm. Degree in the chosen specialization of Pharmacy that he was admitted into.

10.2 Award of Class

After a student has earned the requirements prescribed for the completion of the programme and is eligible for the award of M.Pharm. Degree, he shall be placed in one of the following three classes based on the CGPA:

Class Awarded	CGPA
First Class with Distinction	≥ 7.75
First Class	6.75≤ CGPA < 7.75
Second Class	6.00≤ CGPA < 6.75

A student with final CGPA (at the end of the **PGP) < 6.00** shall not be eligible for the Award of Degree.

11.0 Withholding of Results

If the student has not paid the dues, if any, to the University or if any case of indiscipline is pending against him, the result and degree of the student will be withheld and he will not be allowed into the next semester.

12.0 General

12.1 **Credit**: A unit by which the course work is measured. It determines the number of hours of instructions required per week. One credit is equivalent to one hour of teaching (lecture or tutorial) or two hours of practical work/field work per week.

12.2 **Credit Point:** It is the product of grade point and number of credits for a course.

12.3 Wherever the words "he", "him", "his", occur in the regulations, they shall include "she", "her".

12.4 The academic regulation should be read as a whole for the purpose of any interpretation.

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12.5 In case of any doubt or ambiguity in the interpretation of the above rules, the decision of the University is final.

12.6 The University may change or amend the academic regulations or syllabi at any time and the changes or amendments made shall be applicable to all the students with effect from the dates notified by the University.

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MALPRACTICES RULES

DISCIPLINARY ACTION FOR IMPROPER CONDUCT IN EXAMINATIONS

S.No	Nature of Malpractices/Improper conduct	Punishment
	If the candidate:	
1.(a)	Possesses or keeps accessible in examination hall, any paper, note book, programmable calculators, Cell phones, pager, palm computers or any other form of material concerned with or related to the subject to the examination (theory or practical) in which he is appearing but has not made use of (material shall include any marks on the body of the candidate which can be used as an aid in the subject of the examination).	Expulsion from the examination hall and cancellation of the performance in that subject only.
(b)	Gives assistance or guidance or receives it from any other candidate orally or by any other body language methods or communicates through cell phones with any candidate or persons in or outside the exam hall in respect of any matter.	Expulsion from the examination hall and cancellation of the performance in that subject only of all the candidates involved. Incase of an outsider, he will be handed over to the police and a case is registered against him.
2.	Has copied in the examination hall from any paper, book, programmable calculators, palm computers or any other form of material relevant to the subject to the examination (theory or practical) in which the candidate is appearing.	Expulsion from the examination hall and cancellation of the performance in that subject and all other subjects the candidate has already appeared including practical examinations and project work and shall not be permitted to appear for the remaining examinations of the subjects of that Semester/year. The Hall Ticket of the candidate is to be cancelled and sent to the University.
3.	Impersonates any other candidate in connection with the examination.	The candidate who has impersonated shall be expelled from examination hall. The candidate is also debarred and forfeits the seat. The performance of the original candidate, who has been impersonated, shall be cancelled in all the subjects of the examination (including practicals and project work) already appeared and shall not be allowed to appear for examinations of the remaining subjects of that semester/year. The candidate is also debarred for two consecutive semesters from class work and all University examinations. The continuation of the course by the candidate is subject to the academic regulations in connection with forfeiture of seat. If the imposter is an outsider, he will be handed over to the police and a case is registered against him.
4.	Smuggles in the Answer book or additional sheet or takes out or arranges to send out the question paper during the examination or	Expulsion from the examination hall and cancellation of performance in that subject and all the other subjects the candidate has already

	answer book or additional sheet, during or after the examination.	appeared including practical examinations and project work and shall not be permitted for the remaining examinations of the subjects of that semester/year. The candidate is also debarred for two consecutive semesters from class work and all University examinations. The continuation of the course by the candidate is subject to the academic regulations in connection with forfeiture of seat.
5.	Uses objectionable, abusive or offensive language in the answer paper or in letters to the examiners or writes to the examiner requesting him to award pass marks.	Cancellation of the performance in that subject.
6.	Refuses to obey the orders of the Chief Superintendent/Assistant – Superintendent/ any officer on duty or misbehaves or creates disturbance of any kind in and around the examination hall or organizes a walk out or instigates others to walk out, or threatens the officer-in charge or any person on duty in or outside the examination hall of any injury to his person or to any of his relations whether by words, either spoken or written or by signs or by visible representation, assaults the officer-in- charge, or any person on duty in or outside the examination hall or any of his relations, or indulges in any other act of misconduct or mischief which result in damage to or destruction of property in the examination hall or any part of the College campus or engages in any other act which in the opinion of the officer on duty amounts to use of unfair means or misconduct or has the tendency to disrupt the orderly conduct of the examination.	Incase of students of the college, they shall be expelled from examination halls and cancellation of their performance in that subject and all other subjects the candidate(s) has (have) already appeared and shall not be permitted to appear for the remaining examinations of the subjects of that semester/year. The candidates also are debarred and forfeit their seats. In case of outsiders, they will be handed over to the police and a police case is registered against them.
7.	Leaves the exam hall taking away answer script or intentionally tears of the script or any par there of inside or outside the examination hall.	Expulsion from the examination hall and cancellation of performance in that subject and all the other subjects the candidate has already appeared including practical examinations and project work and shall not be permitted for the remaining examinations of the subjects of that semester/year. The candidate is also debarred for two consecutive semesters from class work and all University examinations. The continuation of the course by the candidate is subject to the academic regulations in connection with forfeiture of seat.
8.	Possess any lethal weapon or firearm in the examination hall.	Expulsion from the examination hall and cancellation of the performance in that subject and all other subjects the candidate has already appeared including practical examinations and project work and shall not be permitted for the

		remaining examinations of the subjects of that semester/year. The candidate is also debarred and forfeits the seat.
9.	If student of the college, who is not a candidate for the particular examination or any person not connected with the college indulges in any malpractice or improper conduct mentioned in clause 6 to 8.	Student of the colleges expulsion from the examination hall and cancellation of the performance in that subject and all other subjects the candidate has already appeared including practical examinations and project work and shall not be permitted for the remaining examinations of the subjects of that semester/year. The candidate is also debarred and forfeits the seat. Person(s) who do not belong to the College will be handed over to police and, a police case will be registered against them.
10.	Comes in a drunken condition to the examination hall.	Expulsion from the examination hall and cancellation of the performance in that subject and all other subjects the candidate has already appeared including practical examinations and project work and shall not be permitted for the remaining examinations of the subjects of that semester/year.
11.	Copying detected on the basis of internal evidence, such as, during valuation or during special scrutiny.	Cancellation of the performance in that subject and all other subjects the candidate has appeared including practical examinations and project work of that semester/year examinations.
12.	If any malpractice is detected which is not covered in the above clauses 1 to 11 shall be reported to the University for further action to award suitable punishment.	

Malpractices identified by squad or special invigilators

1. Punishments to the candidates as per the above guidelines.
2. Punishment for institutions: (if the squad reports that the college is also involved in encouraging malpractices)
   - (i) A show cause notice shall be issued to the college.
   - (ii) Impose a suitable fine on the college.
   - (iii) Shifting the examination centre from the college to another college for a specific period of not less than one year

AVANTHI INSTITUTE OF PHARMACEUTICAL SCIENCES * ABDULLAPURMET *

- PRINCIPAL
Avanthi's Institute of Pharmaceutical Sciences
Gunthapally (V), Hayath Nagar (M),
Ranga Reddy Dist.

रजिस्ट्री सं. डीएल (एन)-04/0007/2003--05 REGISTERED No. DL(N)—04/0007/2003—05

भारत का राजपत्र
The Gazette of India

साप्ताहिक/WEEKLY

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सं० 19] नई दिल्ली, शनिवार, मई 10—मई 16, 2008 (वैशाख 20, 1930)
No. 19] NEW DELHI, SATURDAY, MAY 10—MAY 16, 2008 (VAISAKHA 20, 1930)

इस भाग में भिन्न पृष्ठ संख्या दी जाती है जिससे कि यह अलग संकलन के रूप में रखा जा सके।
(Separate paging is given to this Part in order that it may be filed as a separate compilation)

भाग III—खण्ड 4
[PART III—SECTION 4]
[सांविधिक निकायों द्वारा जारी की गई विविध अधिसूचनाएं जिसमें कि आदेश, विज्ञापन और सूचनाएं सम्मिलित हैं]
[Miscellaneous Notifications including Notifications, Orders, Advertisements and Notices issued by Statutory Bodies]

भारतीय रिज़र्व बैंक

मुंबई-400001, दिनांक 9 अप्रैल 2008

सदर्भ : बैंपविवि. सं. आईबीडी.-14241/23.13.048/2007-08--भारतीय रिज़र्व बैंक अधिनियम, 934 (1934 का 2) की धारा 42 की उप-धारा (6) के खण्ड (ग) के अनुसरण में भारतीय रिज़र्व बैंक इसके द्वारा निदेश देता है कि उक्त अधिनियम की दूसरी अनुसूची में निम्नलिखित परिवर्तन किये जाएं :--

''अरब बांगलादेश बैंक लिमिटेड'' शब्दों के स्थान पर ''एबी बैंक लिमिटेड'' शब्द होंगे।

आनन्द सिन्हा
कार्यपालक निदेशक

[PUBLISHED IN THE GAZETTE OF INDIA, No.19, PART III, SECTION 4]

Ministry of Health and Family Welfare
(Pharmacy Council of India)

New Delhi, 10th May, 2008.

Pharm.D. Regulations 2008

Regulations framed under section 10 of the Pharmacy Act, 1948 (8 of 1948).

(As approved by the Government of India, Ministry of Health vide, letter No.V.13013/1/2007-PMS, dated the 13th March, 2008 and notified by the Pharmacy Council of India).

No.14-126/2007-PCI.— In exercise of the powers conferred by section 10 of the Pharmacy Act, 1948 (8 of 1948), the Pharmacy Council of India, with the approval of the Central Government, hereby makes the following regulations, namely:-

CHAPTER-I

1. Short title and commencement. – (1) These regulations may be called the Pharm.D. Regulations 2008.

   (2) They shall come into force from the date of their publication in the official Gazette.

2. Pharm.D. shall consist of a certificate, having passed the course of study and examination as prescribed in these regulations, for the purpose of registration as a pharmacist to practice the profession under the Pharmacy Act, 1948.

CHAPTER-II

3. Duration of the course. –

a) Pharm.D: The duration of the course shall be six academic years (five years of study and one year of internship or residency) full time with each academic year spread over a period of not less than two hundred working days. The period of six years duration is divided into two phases –

Phase I – consisting of First, Second, Third, Fourth and Fifth academic year.

Phase II – consisting of internship or residency training during sixth year involving posting in speciality units. It is a phase of training wherein a student is exposed to actual pharmacy practice or clinical pharmacy services and acquires skill under supervision so that he or she may become capable of functioning independently.

b) Pharm.D. (Post Baccalaureate): The duration of the course shall be for three academic years (two years of study and one year internship or residency) full time with each academic year spread over a period of not less than two hundred working days. The period of three years duration is divided into two phases –

Phase I – consisting of First and Second academic year.

Phase II – consisting of Internship or residency training during third year involving posting in speciality units. It is a phase of training wherein a student is exposed to actual pharmacy practice or clinical pharmacy services, and acquires skill under supervision so that he or she may become capable of functioning independently.

4. Minimum qualification for admission to. –

a) Pharm.D. Part-I Course – A pass in any of the following examinations -

(1) 10+2 examination with Physics and Chemistry as compulsory subjects along with one of the following subjects:

Mathematics or Biology.

(2) A pass in D.Pharm course from an institution approved by the Pharmacy Council of India under section 12 of the Pharmacy Act.

(3) Any other qualification approved by the Pharmacy Council of India as equivalent to any of the above examinations.

Provided that a student should complete the age of 17 years on or before 31st December of the year of admission to the course.

Provided that there shall be reservation of seats for the students belonging to the Scheduled Castes, Scheduled Tribes and other Backward Classes in accordance with the instructions issued by the Central Government/State Government/Union Territory Administration as the case may be from time to time.

b) Pharm.D. (Post Baccalaureate) Course -

A pass in B.Pharm from an institution approved by the Pharmacy Council of India under section 12 of the Pharmacy Act:

Provided that there shall be reservation of seats for the students belonging to the Scheduled Castes, Scheduled Tribes and other Backward Classes in accordance with the instructions issued by the Central Government/State Government/Union Territory Administration as the case may be from time to time.

5. Number of admissions in the above said programmes shall be as prescribed by the Pharmacy Council of India from time to time and presently be restricted as below –
   i) Pharm.D. Programme – 30 students.
   ii) Pharm.D. (Post Baccalaureate) Programme – 10 students.

6. Institutions running B.Pharm programme approved under section 12 of the Pharmacy Act, will only be permitted to run Pharm.D. programme. Pharm.D. (Post Baccalaureate) programme will be permitted only in those institutions which are permitted to run Pharm.D. programme.

7. Course of study. – The course of study for Pharm.D. shall include the subjects as given in the Tables below. The number of hours in a week, devoted to each subject for its teaching in theory, practical and tutorial shall not be less than that noted against it in columns (3), (4) and (5) below.

TABLES

First Year :

S.No.	Name of Subject	No. of hours of Theory	No. of hours of Practical	No. of hours of Tutorial
(1)	(2)	(3)	(4)	(5)
1.1	Human Anatomy and Physiology	3	3	1
1.2	Pharmaceutics	2	3	1
1.3	Medicinal Biochemistry	3	3	1
1.4	Pharmaceutical Organic Chemistry	3	3	1
1.5	Pharmaceutical Inorganic Chemistry	2	3	1
1.6	Remedial Mathematics/ Biology	3	3*	1
	Total hours	**16**	**18**	**6 = (40)**

* For Biology

Second Year:

S.No	Name of Subject	No. of hours of Theory	No. of hours of Practical	No. of hours of Tutorial
(1)	(2)	(3)	(4)	(5)
2.1	Pathophysiology	3	-	1
2.2	Pharmaceutical Microbiology	3	3	1
2.3	Pharmacognosy & Phytopharmaceuticals	3	3	1
2.4	Pharmacology-I	3	-	1
2.5	Community Pharmacy	2	-	1
2.6	Pharmacotherapeutics-I	3	3	1
	Total Hours	**17**	**9**	**6 = 32**

Third Year:

S.No.	Name of Subject	No. of hours of Theory	No. of hours of Practical	No. of hours of Tutorial
(1)	(2)	(3)	(4)	(5)
3.1	Pharmacology-II	3	3	1
3.2	Pharmaceutical Analysis	3	3	1
3.3	Pharmacotherapeutics-II	3	3	1
3.4	Pharmaceutical Jurisprudence	2	-	-
3.5	Medicinal Chemistry	3	3	1
3.6	Pharmaceutical Formulations	2	3	1
	Total hours	**16**	**15**	**5 = 36**

Fourth Year:

S.No.	Name of Subject	No. of hours of Theory	No. of hours of Practical/ Hospital Posting	No. of hours of Tutorial
(1)	(2)	(3)	(4)	(5)
4.1	Pharmacotherapeutics-III	3	3	1
4.2	Hospital Pharmacy	2	3	1
4.3	Clinical Pharmacy	3	3	1
4.4	Biostatistics & Research Methodology	2	-	1
4.5	Biopharmaceutics & Pharmacokinetics	3	3	1
4.6	Clinical Toxicology	2	-	1
	Total hours	**15**	**12**	**6 = 33**

Fifth Year:

S.No.	Name of Subject	No. of hours of Theory	No. of hours of Hospital posting*	No. of hours of Seminar
(1)	(2)	(3)	(4)	(5)
5.1	Clinical Research	3	-	1
5.2	Pharmacoepidemiology and Pharmacoeconomics	3	-	1
5.3	Clinical Pharmacokinetics & Pharmacotherapeutic Drug Monitoring	2	-	1
5.4	Clerkship *	-	-	1
5.5	Project work (Six Months)	-	20	-
	Total hours	**8**	**20**	**4 = 32**

* Attending ward rounds on daily basis.

Sixth Year:

Internship or residency training including postings in speciality units. Student should independently provide the clinical pharmacy services to the allotted wards.

(i) Six months in General Medicine department, and

(ii) Two months each in three other speciality departments

8. Syllabus. – The syllabus for each subject of study in the said Tables shall be as specified in Appendix -A to these regulations.

9. Approval of the authority conducting the course of study. – (1) No person, institution, society or university shall start and conduct Pharm.D or Pharm.D. (Post Baccalaureate) programme without the prior approval of the Pharmacy Council of India.

(2) Any person or pharmacy college for the purpose of obtaining permission under sub-section (1) of section 12 of the Pharmacy Act, shall submit a scheme as prescribed by the Pharmacy Council of India.

(3) The scheme referred to in sub-regulation (2) above, shall be in such form and contain such particulars and be preferred in such manner and be accompanied with such fee as may be prescribed:

Provided that the Pharmacy Council of India shall not approve any institution under these regulations unless it provides adequate arrangements for teaching in regard to building, accommodation, labs., equipments, teaching staff, non-teaching staff, etc., as specified in Appendix-B to these regulations.

10. Examination. – (1) Every year there shall be an examination to examine the students.

(2) Each examination may be held twice every year. The first examination in a year shall be the annual examination and the second examination shall be supplementary examination.

(3) The examinations shall be of written and practical (including oral nature) carrying maximum marks for each part of a subject as indicated in Tables below :

TABLES

First Year examination :

S.No.	Name of Subject	Maximum marks for Theory			Maximum marks for Practicals		
		Examination	Sessional	Total	Examination	Sessional	Total
1.1	Human Anatomy and Physiology	70	30	100	70	30	100
1.2	Pharmaceutics	70	30	100	70	30	100
1.3	Medicinal Biochemistry	70	30	100	70	30	100
1.4	Pharmaceutical Organic Chemistry	70	30	100	70	30	100
1.5	Pharmaceutical Inorganic Chemistry	70	30	100	70	30	100
1.6	Remedial Mathematics/ Biology	70	30	100	70*	30*	100*
				600			600 = 1200

* for Biology.

Second Year examination :

S.No.	Name of Subject	Maximum marks for Theory			Maximum marks for Practicals		
		Examination	Sessional	Total	Examination	Sessional	Total
2.1	Pathophysiology	70	30	100	-	-	-
2.2	Pharmaceutical Microbiology	70	30	100	70	30	100
2.3	Pharmacognosy & Phytopharmaceuticals	70	30	100	70	30	100
2.4	Pharmacology-I	70	30	100	-	-	-
2.5	Community Pharmacy	70	30	100	-	-	-
2.6	Pharmacotherapeutics-I	70	30	100	70	30	100
				600			300 = 900

Third Year examination :

S.No.	Name of Subject	Maximum marks for Theory			Maximum marks for Practicals		
		Examination	Sessional	Total	Examination	Sessional	Total
3.1	Pharmacology-II	70	30	100	70	30	100
3.2	Pharmaceutical Analysis	70	30	100	70	30	100
3.3	Pharmacotherapeutics-II	70	30	100	70	30	100
3.4	Pharmaceutical Jurisprudence	70	30	100	-	-	-
3.5	Medicinal Chemistry	70	30	100	70	30	100
3.6	Pharmaceutical Formulations	70	30	100	70	30	100
				600			500 = 1100

Fourth Year examination :

S.No.	Name of Subject	Maximum marks for Theory			Maximum marks for Practicals		
		Examination	Sessional	Total	Examination	Sessional	Total
4.1	Pharmacotherapeutics-III	70	30	100	70	30	100
4.2	Hospital Pharmacy	70	30	100	70	30	100
4.3	Clinical Pharmacy	70	30	100	70	30	100
4.4	Biostatistics & Research Methodology	70	30	100	-	-	-
4.5	Biopharmaceutics & Pharmacokinetics	70	30	100	70	30	100
4.6	Clinical Toxicology	70	30	100	-	-	-
				600			400 = 1000

Fifth Year examination :

S.No.	Name of Subject	Maximum marks for Theory			Maximum marks for Practicals		
		Examination	Sessional	Total	Examination	Sessional	Total
5.1	Clinical Research	70	30	100	-	-	-
5.2	Pharmacoepidemiology and Pharmacoeconomics	70	30	100	-	-	-
5.3	Clinical Pharmacokinetics & Pharmacotherapeutic Drug Monitoring	70	30	100	-	-	-
5.4	Clerkship *	-	-	-	70	30	100
5.5	Project work (Six Months)	-	-	-	100**	-	100
				300			200 = 500

* Attending ward rounds on daily basis.

** 30 marks – viva-voce (oral)
70 marks – Thesis work

11. Eligibility for appearing Examination.— Only such students who produce certificate from the Head of the Institution in which he or she has undergone the Pharm.D. or as the case may be, the Pharm.D. (Post Baccalaureate) course, in proof of his or her having regularly and satisfactorily undergone the course of study by attending not less than 80% of the classes held both in theory and in practical separately in each subject shall be eligible for appearing at examination.

12. Mode of examinations.— (1) Theory examination shall be of three hours and practical examination shall be of four hours duration.

    (2) A Student who fails in theory or practical examination of a subject shall re-appear both in theory and practical of the same subject.

    (3) Practical examination shall also consist of a viva –voce (Oral) examination.

    (4) Clerkship examination – Oral examination shall be conducted after the completion of clerkship of students. An external and an internal examiner will evaluate the student. Students may be asked to present the allotted medical cases followed by discussion. Students' capabilities in delivering clinical pharmacy services, pharmaceutical care planning and knowledge of therapeutics shall be assessed.

13. Award of sessional marks and maintenance of records.— (1) A regular record of both theory and practical class work and examinations conducted in an institution imparting training for Pharm.D. or as the case may be, Pharm.D. (Post Baccalaureate) course, shall be maintained for each student in the institution and 30 marks for each theory and 30 marks for each practical subject shall be allotted as sessional.

    (2) There shall be at least two periodic sessional examinations during each academic year and the highest aggregate of any two performances shall form the basis of calculating sessional marks.

    (3) The sessional marks in practicals shall be allotted on the following basis:-

        (i) Actual performance in the sessional examination (20 marks);

        (ii) Day to day assessment in the practical class work, promptness, viva-voce record maintenance, etc. (10 marks).

14. Minimum marks for passing examination.— A student shall not be declared to have passed examination unless he or she secures at least 50% marks in each of the subjects separately in the theory examinations, including sessional marks and at least 50% marks in each of the practical examinations including sessional marks. The students securing 60% marks or above in aggregate in all subjects in a single attempt at the Pharm.D. or as the case may be, Pharm. D. (Post Baccalaureate) course examination shall be declared to have passed in first class. Students securing 75% marks or above in any subject or subjects shall be declared to have passed with distinction in the subject or those subjects provided he or she passes in all the subjects in a single attempt.

15. Eligibility for promotion to next year.— All students who have appeared for all the subjects and passed the first year annual examination are eligible for promotion to the second year and, so on. However, failure in more than two subjects shall debar him or her from promotion to the next year classes.

16. Internship.— (1) Internship is a phase of training wherein a student is expected to conduct actual practice of pharmacy and health care and acquires skills under the supervision so that he or she may become capable of functioning independently.

    (2) Every student has to undergo one year internship as per Appendix-C to these regulations.

17. Approval of examinations.— Examinations mentioned in regulations 10 to12 and 14 shall be held by the examining authority hereinafter referred to as the university, which shall be approved by the Pharmacy Council of India under sub-section (2) of section 12 of the Pharmacy Act, 1948. Such approval shall be granted only if the examining authority concerned fulfills the conditions as specified in Appendix–D to these regulations.

18. Certificate of passing examination.— Every student who has passed the examinations for the Pharm.D. (Doctor of Pharmacy) or Pharm.D. (Post Baccalaureate) (Doctor of Pharmacy) as the case may be, shall be granted a certificate by the examining authority.

CHAPTER-III
Practical training

19. Hospital posting.— Every student shall be posted in constituent hospital for a period of not less than fifty hours to be covered in not less than 200 working days in each of second, third & fourth year course. Each student shall submit report duly certified by the preceptor and duly attested by the Head of the Department or Institution as prescribed. In the fifth year, every student shall spend half a day in the morning hours attending ward rounds on daily basis as a part of clerkship. Theory teaching may be scheduled in the afternoon.

20. Project work.— (1) To allow the student to develop data collection and reporting skills in the area of community, hospital and clinical pharmacy, a project work shall be carried out under the supervision of a teacher. The project topic must be approved by the Head of the Department or Head of the Institution. The same shall be announced to students within one month of commencement of the fifth year classes. Project work shall be presented in a written report and as a seminar at the end of the year. External and the internal examiners shall do the assessment of the project work.

    (2) Project work shall comprise of objectives of the work, methodology, results, discussions and conclusions.

21. Objectives of project work.— The main objectives of the project work is to—

    (i) show the evidence of having made accurate description of published work of others and of having recorded the findings in an impartial manner; and

    (ii) develop the students in data collection, analysis and reporting and interpretation skills.

22. Methodology.— To complete the project work following methodology shall be adopted, namely:—

    (i) students shall work in groups of not less than *two* and not more than *four* under an authorised teacher;

    (ii) project topic shall be approved by the Head of the Department or Head of the Institution;

    (iii) project work chosen shall be related to the pharmacy practice in community, hospital and clinical setup. It shall be patient and treatment (Medicine) oriented, like drug utilisation reviews, pharmacoepidemiology, pharmacovigilance or pharmacoeconomics;

    (iv) project work shall be approved by the institutional ethics committee;

    (v) student shall present at least three seminars, one in the beginning, one at middle and one at the end of the project work; and

    (vi) two-page write-up of the project indicating title, objectives, methodology anticipated benefits and references shall be submitted to the Head of the Department or Head of the Institution.

23. Reporting .— (1) Student working on the project shall submit jointly to the Head of the Department or Head of the Institution a project report of about 40-50 pages. Project report should include a certificate issued by the authorised teacher, Head of the Department as well as by the Head of the Institution

(2) Project report shall be computer typed in double space using Times Roman font on A4 paper. The title shall be in bold with font size 18, sub-tiles in bold with font size 14 and the text with font size 12. The cover page of the project report shall contain details about the name of the student and the name of the authorised teacher with font size 14.

(3) Submission of the project report shall be done at least one month prior to the commencement of annual or supplementary examination.

24. Evaluation.— The following methodology shall be adopted for evaluating the project work—

(i) Project work shall be evaluated by internal and external examiners.

(ii) Students shall be evaluated in groups for four hours (i.e., about half an hour for a group of four students).

(iii) Three seminars presented by students shall be evaluated for twenty marks each and the average of best two shall be forwarded to the university with marks of other subjects.

(iv) Evaluation shall be done on the following items:

	Marks
a) Write up of the seminar	(7.5)
b) Presentation of work	(7.5)
c) Communication skills	(7.5)
d) Question and answer skills	(7.5)
Total	(30 marks)

(v) Final evaluation of project work shall be done on the following items:

	Marks
a) Write up of the seminar	(17.5)
b) Presentation of work	(17.5)
c) Communication skills	(17.5)
d) Question and answer skills	(17.5)
Total	(70 marks)

Explanation.— For the purposes of differentiation in the evaluation in case of topic being the same for the group of students, the same shall be done based on item numbers b, c and d mentioned above.

COURSE FILE

SUBJECT:	PATHOPHYSIOLOGY
ACADEMIC YEAR:	2019-2020

NAME OF THE FACULTY:	**Dr. RAVIPRAKASH**
DESIGNATION:	**ASSISTANT PROFESSOR**
DEPARTMENT:	**PHARMACY PRACTICE**
BRANCH	**PHARM.D**
YEAR	**II YEAR**

PRINCIPAL
AVANTHI INSTITUTE OF PHARMACEUTICAL SCIENCES
Gunthapally (V), Abdullapurmet (M),
R.R. Dist., Telangana.

Course File Index

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1	VISION AND MISSION
2	COURSE OUTCOMES
3	COURSE SYLLABUS
4	LESSON PLAN
5	ACADEMIC CALENDER
6	TIME TABLE
7	LECTURE NOTES
8	UNIVERSITY QUESTION PAPER
9	INTERNAL QUESTION PAPER
10	INTERNAL QUESTION PAPER WITH ANSWER KEY
11	ASSIGNMENT QUESTION PAPER
12	STUDENT ASSIGNMENT
13	RESULT
14	ATTAINMENT

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Gunthapally (V), Abdullapurmet (M)
R.R. Dist. Telangana

COURSE FILE

COURSE DESRIPTION/COURSE INFORMATION SHEET

NAME OF THE DEPARTMENT: PHARM.D

COURSE TITLE	PATHOPHYSIOLOGY			
COURSE CODE	PH201			
REGULATION	R8		YEAR	II
COURSE STRUCTURE	LECTURES	TUTORIALS	PRACTICALS	CREDITS
	3	1	-	-
COURSE TEACHER	Dr. RAVIPRAKASH			
NO.OF HOURS ALLOTED PER WEEK	LECTURES	TUTORIALS	PRACTICALS	
	3	1	-	

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1.VISION & MISSION OF THE INSTITUTION

VISION	TO DEVELOP HIGHLY SKILLED PROFESSIONALS WITH ETHICS AND HUMAN VALUES
MISSION	WE ARE COMMITTED TO PROVIDE A POSITIVE AND PROFESSIONAL LEARNING ENVIRONMENT WHERE ALL STUDENTS ARE INSPIRED TO STRIVE FOR EXCELLENCE IN ORDER TO ACHIEVE THEIR POTENTIAL AS DIGNIFIED AND COMPETENT PHARMACISTS,TECHNOLOGY INNOVATORS,MANAGERS AND LEADERS IN GLOBAL SOCIETY THROUGH A COHESIVE NETWORK THE PARENTS,STUDENTS,COLLEGE STAFF AND INDUSTRY.

COURSE HANDOUT

- **PROGRAM OUTCOMES & PROGRAM SPECIFIC OUTCOMES (POs) & (PSOs)**
- **COURSE OUTCOMES(COs)**
- **DETAILED SYLABUS**

Program Outcomes (POs) and (PSOs)

PO 1 Pharmacy Knowledge: Provide high quality, evidence-based, patient-centeredcare in cooperation with patients, prescribers and members of the inter professional health care team

PO 2 Practical Skill: Demonstrate mastery and application of core knowledge and skills in relation to the evolving biomedical, clinical, epidemiological and social-behavioral sciences.

PO 3 Professional Identity: Evaluate practice and care, and promote continuous improvement in one's own patient care and pharmacy services

PO 4 Problem Solving: Demonstrate self-calibration skills and a commitment to the lifelong learning needed to provide high quality care

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PO 5 Communication: Effectively utilize information, informatics and technology to optimize learning and patient care

PO 6 Planning Ability: Demonstrate effective interpersonal written and verbal skills, adapt to socioeconomic and cultural factors as well as situational applications

PO 7 Leadership Skills & Team Work : Demonstrate exemplary professional, ethical and legal behaviors, complying with all federal, state and local laws and regulations related to pharmacy practice

PO 8 Life Long Learning: Demonstrate awareness and responsiveness to the system of health care, effectively utilizing systems of care to provide cost-effective, optimal care

PO9 Pharmaceutical Ethics: Honour personal values and apply ethical principles in professional and social context. Demonstrate behavior that recognizes cultural and personal variability in values, communication and life styles.

PO10 Pharmacist and Society: Apply reasoning informed by the contextual knowledge to asses societal, health, safety and legal issues and the consequent responsibilities relevant to the profession.

PO11 Environment and Society: Understand the impact of professional pharmacy solutions in societal and environmental context and demonstrate the knowledge of, and need for sustainable development.

PSO1: Able to apply the knowledge gained during the course of the program in drug discovery and development, their safety and efficacy and current technologies in Pharmaceutical industry

PSO 2: Able to apply the knowledge of ethical and management principles required to work in a team as well as to lead a team.

PSO3: Able to do multidisciplinary jobs in the pharmaceutical industries and would be able to write effective project reports in multidisciplinary environment in the context of changing technologies.

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CO1: Initiate drug therapy and the anticipated therapeutic goals by therapeutic intervention
CO2: Know the effective use of Non pharmacological therapeutic interventions in the treatment of specific diseases, conditions and symptoms.
CO3: Demonstrate the ability to effectively communicate and work collaboratively together with others in the small group setting
CO4: Have moral reasoning, ethical judgement and professionalism

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DETAILED SYLLABUS

S. No.	Topic
01	**Basic principles of cell injury and Adaptation**
	a) Causes, Pathogenesis and morphology of cell injury b) Abnormalities in lipoproteinaemia, glycogen infiltration and glycogen infiltration and glycogen infiltration and glycogen storage diseases
02	**Inflammation**
	a) Pathogenesis of acute inflammation, Chemical mediators in inflammation, Types of chronic inflammation b) Repairs of wounds in the skin, factors influencing healing of wounds
03	**Diseases of Immunity**
	a) Introduction to T and B cells b) MHC proteins or transplantation antigens c) Immune tolerance
04	**Cancer**
	Differences between benign and malignant tumors Histological diagnosis of malignancy invasions and metastasis patterns of spread disturbances of growth of cells classification of tumors general biology of tumors spread of malignant tumors, etiology and pathogenesis of cancer
05	**Types of shock, mechanisms, stages and management**
06	**Biological effects of radiation**
07	**Environmental and nutritional diseases**
	i) Air pollution and smoking- SO2, NO, NO2, and CO ii) Protein calorie malnutrition, vitamins, obesity, pathogenesis of starvation

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08	**Pathophysiology of common diseases**
	a. Parkinsonism b. Schizophrenia c. Depression and mania d. Hypertension, e. Stroke (ischaemic and hemorrhage) f. Angina, CCF, Atherosclerosis, Myocardial infarction g. Diabetes Mellitus h. Peptic ulcer and inflammatory bowel diseases i. Cirrhosis and Alcoholic liver diseases j. Acute and chronic renal failure k. Asthma and chronic obstructive airway diseases
09	**Infectious diseases**
	Sexually transmitted diseases (HIV, Syphilis, Gonorrhea) Urinary tract infections Pneumonia Typhoid, Tuberculosis, Leprosy Malaria Dysentery (bacterial and amoebic) Hepatitis- infective hepatitis
TEXT BOOKS	a. Pathologic basis of disease by- Cotran, Kumar, Robbins b. Text book of Pathology- Harsh Mohan c. Text book of Pathology- Y.M. Bhinde
REFERENCES	a. Clinical Pharmacy and Therapeutics; Second edition; Roger Walker; Churchill Livingstone publication

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LECTURE PLAN

S. No.	Topic	No of Lecture Hours	Teaching Learning Process
	Topic-1		
01	**Basic principles of cell injury and Adaptation**	**10**	
	a) Causes, Pathogenesis and morphology of cell injury	04	Chalk & Board
	b) Abnormalities in lipoproteinaemia, glycogen infiltration and glycogen infiltration and glycogen infiltration and glycogen storage diseases	06	Power Point Presentation
	Topic-2		
02	**Inflammation**	**12**	
	a) Pathogenesis of acute inflammation, Chemical mediators in inflammation, Types of chronic inflammation	06	Power Point Presentation
	b) Repairs of wounds in the skin, factors influencing healing of wounds	06	Chalk & Board
	Topic-3		
03	**Diseases of Immunity**	**20**	
	a) Introduction to Tand B cells	02	Power Point Presentation
	b) MHC proteins or transplantation antigens	02	Power Point Presentation
	c) Immune tolerance	16	Chalk & Board
	Topic-3		

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04	**Cancer**	**20**	
	Differences between benign and malignant tumors	02	Power Point Presentation
	Histological diagnosis of malignancy	02	Power Point Presentation
	invasions and metastasis	03	Chalk & Board
	patterns of spread	02	Power Point Presentation
	disturbances of growth of cells	02	Chalk & Board
	classification of tumors	03	Power Point Presentation
	general biology of tumors	02	Power Point Presentation
	spread of malignant tumors,.	02	Power Point Presentation
	etiology and pathogenesis of cancer	02	Power Point Presentation
	TOPIC-5		
05	**Types of shock, mechanisms, stages and management**	**05**	Power Point Presentation
	TOPIC-6		
06	**Biological effects of radiation**	**04**	Power Point Presentation
	TOPIC-7		
07	**Environmental and nutritional diseases**	**05**	
	i) Air pollution and smoking- SO2,NO, NO2, and CO	02	Power Point Presentation
	ii) Protein calorie malnutrition, vitamins, obesity, pathogenesis of starvation	03	Power Point Presentation
	TOPIC-8		
08	**Pathophysiology of common diseases**	**22**	
	a. Parkinsonism	02	Power Point Presentation
	b. Schizophrenia	02	Power Point Presentation
	c. Depression and mania	02	Chalk & Board
	d. Hypertension,	02	Power Point Presentation

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	e. Stroke (ischaemic and hemorrhage)	02	Power Point Presentation
	f. Angina, CCF, Atherosclerosis, Myocardial infarction	02	Power Point Presentation
	g. Diabetes Mellitus	02	Chalk & Board
	h. Peptic ulcer and inflammatory bowel diseases	02	Power Point Presentation
	i. Cirrhosis and Alcoholic liver diseases	02	Power Point Presentation
	j. Acute and chronic renal failure	02	Chalk & Board
	k. Asthma and chronic obstructive airway diseases	02	Power Point Presentation
	TOPIC-9		
09	**Infectious diseases**	**10**	
	Sexually transmitted diseases (HIV, Syphilis, Gonorrhea)	02	Power Point Presentation
	Urinary tract infections	01	Power Point Presentation
	Pneumonia	01	Power Point Presentation
	Typhoid, Tuberculosis, Leprosy	03	Power Point Presentation
	Malaria Dysentery (bacterial and amoebic)	02	Power Point Presentation
	Hepatitis- infective hepatitis	01	Power Point Presentation

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JAWAHARLAL NEHRU TECHNOLOGICAL UNIVERSITY HYDERABAD

ACADEMIC CALENDAR (2019-20)

PHARM.D (Regular) and (PB) I YEAR

PHARM. D (Regular) and (Post Baccalaureate) I YEAR

Description	Period	Duration
Commencement of instruction	26th Aug. 2019	--
Dussehra Recess	7th to 12th oct. 2019	(1 w)
First mid examinations	11th to 16th Nov. 2019	(1 w)
Submission of First Mid Term Exam Marks to University on or before	23rd Nov. 2019	--
Parent-Teacher Meeting	14th Dec. 2019	--
Second mid examinations	3rd to 8th Feb. 2020	(1 w)
Submission of Second Mid Term Exam Marks to University on or before	16th Feb. 2020	--
Parent-Teacher Meeting	14th April 2020	--
Last date of Instruction	25th April 2020	(32 w)
Third mid examinations	27th April to 2nd May 2020	(1 w)
Preparation and Practical Examinations	4th to 16th May 2020	(2 w)
Submission of Third Mid Term Exam Marks to University on or before	9th May 2020	--
End / Supplementary Examinations	18th to 30th May 2020	(2 w)
Summer vacation	1st June to 4th July 2020	(5 w)

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JAWAHARLAL NEHRU TECHNOLOGICAL UNIVERSITY HYDERABAD
ACADEMIC CALENDAR (2019-20)
PHARM. D (Regular) II, III, IV, V, VI YEARS and PHARM.D (PB) II & III YEARS

PHARM. D (Regular) II, III, IV, V YEAR and PHARM.D (PB) II YEAR

Description	Period	Duration
Commencement of instruction	1st July 2019	--
First mid examinations	16th to 21st Sept. 2019	(1 week)
Submission of First Mid Term Exam Marks to University on or before	30th Sept. 2019	--
Dussehra Recess	7th to 12th Oct.2019	(1 week)
Parent-Teacher Meeting	9th Nov. 2019	--
Supplementary Examinations	14th Oct. to 2nd Nov. 2019	(3 weeks)
Second mid examinations	30th Dec. 2019 to 4th Jan. 2020	(1 week)
Submission of Second Mid Term Exam Marks to University on or before	11th Jan. 2020	--
Parent-Teacher Meeting	8th Feb. 2020	--
Last date of Instruction	21st Mar. 2020	(32 weeks)
Third mid examinations	23rd to 28th Mar. 2020	(1 week)
Submission of Third Mid Term Exam Marks to University on or before	6th April 2020	--
Preparation and Practical Examinations	30th Mar. to 11th April 2020	(2 weeks)
End / Supplementary Examinations	13th to 25th April 2020	(2 weeks)
Summer vacation	26th April to 4th July 2020	(10 weeks)

PHARM. D (Regular) VI YEAR and PHARM.D (PB) III YEAR

Description	Period	Duration
Commencement of internship in general ward	1st July to 28th Dec. 2019	(6 months)
Report submission of internship in general ward	30th Dec. 2019	--
Commencement of internship in Specialty ward -1	31st Dec. 2019 to 29th Feb. 2020	(2 months)
Report submission of internship in Specialty ward -1	2nd Mar. 2020	--
Commencement of internship in Specialty ward – 2	3rd Mar. to 2nd May 2020	(2 months)
Report submission of internship in Specialty ward–2	4th May 2020	--
Commencement of internship in Specialty ward – 3	5th May to 4th July 2020	(2 months)
Report submission of internship in Specialty ward - 3	6th July 2020	--
Final viva of internship	8th July 2020	--

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DEPARTMENT OF PHARMACY PRACTICE

A.Y 2019-20 TIME TABLE

PHARM.D II YEAR **W.E.F: 01/07/2019** **COLLEGE TIMINGS:9:30AM-3:50PM**

DAYS	9.30AM-10.20AM	10.20AM-11.10AM	11.10AM-12.00PM	12.00AM-12.50PM	12.5PM-1.20PM	1.20P.M-2.10PM	2.10PM-3.00P M	3.00PM - 3.50PM
MON	P.COL-I	CP	P.PHY.	P.THER.-I	L	LIBRARY/SPORTS		
TUE	P.THER.-I	MICRO	P.PHY	LIBRARY	U	SEMINARS		CP
WED	P.PHY.	MICRO	P.COL-I	MICRO	N	MICRO		
THU	P.COL-I	LIBRARY	P.COG&PHYTO	P.COG& PHYTO	C	P.COG&PHYTO.		
FRI	CP	P.PHY	P.THER.-I(T)	P.COL-I(T)	H	SEMINARS		
SAT	MICRO	P.THER.-I	P.THER.-I LAB(HOSPITALVISIT)			P.THER.-I LAB (HOSPITAL VISIT)		

Subject Name	Faculty Name	Designation
Pathophysiology	Dr. Raviprakash	Assistant Professor
Pharmaceutical Microbiology	Dr. Ravinayak	Assistant Professor
Pharmacognosy & Phytopharmaceuticals	S. Sandhya rani	Assistant Professor
Pharmacology-I	Santhoshikumari	Assistant Professor
Community Pharmacy	Dr. P. Swathi	Assistant Professor
Pharmacotherapeutics-I	Dr. K. Anusha	Assistant Professor
Pharmaceutical Microbiology -Lab	Dr. Ravinayak	Assistant Professor
Pharmacognosy & Phytopharmaceuticals-Lab	S. Sandhya rani	Assistant Professor
Pharmacotherapeutics-I-Lab	Dr. K. Anusha	Assistant Professor

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Cancer: Etiology, Pathophysiology, Types

Cancer is defined as the uncontrolled proliferation of cell populations that defy the normal rules of cell division. Such cells are known as cancer cells. Normal cells are directly monitored, and their growth, proliferation and cell division are monitored by signal transduction. However, cancer cells have developed autonomous mechanisms for their growth and reproduction.

Cancer is a disease in which normal cells transform into cancerous cells through a process called carcinogenesis

Clinically, there are many types of cancer, but biologically, the origins of cancers are similar due to defective gene expression.

There are several factors that cause normal cells to turn into cancer cells. These factors or substances are known as carcinogens

All cells are thought to carry specific oncogenes that cause cancer

Oncogenes are genes that induce tumors. Under certain conditions, these genes are induced to rapidly proliferate into malignant neoplasms

Etiological agents that induce cancer:

1. Environmental factors:

tobacco, smokes, diets, environmental pollutants etc . Heavy smoking cause lung, oral cavity and oesophagus cancer. Excessive intake of alcohol cause liver cancer.

2. Chemical carcinogen: Nickel compounds, cadmium, arsenic, nitrosamines, trichloroethylene, arylamines, benzopyrene, aflatoxins, reactive oxygen radicals etc

3. Physical carcinogen: UV rays (ultraviolet), ionizing radiation (x-rays and gamma rays)

4. Biological carcinogen:

Virus:Virus has also been associated with various types of cancers. These viruses are called oncoviruses .

Rous sarcoma virus (RSV) is the first discovered retro-virus causing cancer.

(Oncovirus); Human papilloma virus (HPV), Epstein-BarrVirus, (EBV), Hepatitis B virus, Herpes virus

Hepatitis B and C virus is casually related with hepato-cellular carcinoma

Cytomegalovirus (CMV) is associated with kaposi's sarcoma

Human papilloma virus (HPV) is a chief suspect of cervix cancer

Bacteria; Helicobacter pylori,

5. Endogenous factors:

Mutations, change in DNA replication, metabolic reactions generating, reactive oxygen radicals, Immune system defects, Ageing

Cancer pathophysiology :-

.Regardless of difference in types of cancer histologically and physiologically, there is existence of a common pathophysiological process of malignant tumors or cancer development in the organism. The commonly accepted basis of the pathogenesis of cancer is the damage to the genetic apparatus of cells (such as mutation, disturbance of gene expression, activation of tumor promoter gene, inactivation of tumor suppressor genes,etc.)

Damage to the genetic apparatus of the cell along with inactivation of anti-tumor genes takes place and is essential for the development of malignant tumors. The inactivation of tumor suppressor gene is one of the natural physiological reactions of the body. When this reaction becomes pathological, it results in cancer.

At the cellular level, the development of cancer is viewed as a multi-step process involving mutation and selection for cells with progressively increasing capacity for proliferation, survival, invasion, and metastasis.

First step: Mutation and tumor initiation.

Genetic alteration leads to mutation in a single cell which results into abnormal proliferation of that cell known as tumor cell.

Second step: Cell proliferation and Tumor progression.

Tumor progression continues as additional mutations occur within cells of the tumor population.

The mutated cells have some selective advantage over normal cell as such cells shows rapid growth and division. The descendants of a cell bearing such additional mutation will consequently become dominant within the tumor population

Third step: Clonal selection and malignancy

Cell proliferation of tumor then leads to new clone of tumor cells with increased growth rate or other properties (such as survival, invasion, or metastasis) that confer a selective advantage. The process is called clonal selection

Clonal selection continues throughout tumor development, so tumors continuously become more rapid-growing and increasingly malignant

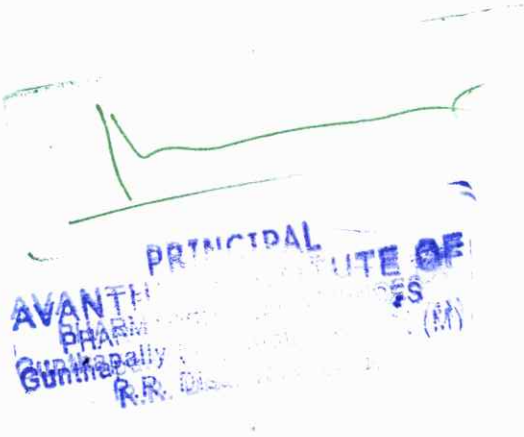
For example: In colon cancer, the earliest stage in tumor development is increased proliferation of colon epithelial cells. A clonal selection occurs in which, a single cell within these proliferative cell population give rise to a small benign neoplasm. Further rounds of clonal selection lead to the growth of benign neoplasm with increase in size and proliferative potential resulting in malignant carcinoma. The cancer cells then continue to proliferate and spread through the connective tissues of the colon wall. Eventually the cancer cells penetrate the wall of the colon and invade other abdominal organs, such as the bladder or small intestine. In addition, the cancer cells invade blood and lymphatic vessels, allowing them to metastasize throughout the body

Fourth step: Metastasis

Metastasis is a complex process in which cancer cells break away from the primary tumor and circulate through the bloodstream or lymphatic system to other sites in the body. At new sites, the cells continue to multiply and eventually form additional tumors comprised of cells that reflect the tissue of origin.

The ability of tumors, such as pancreatic cancer and uveal (iris, ciliary body, or choroid of eye) cancers, to metastasize contributes greatly to their lethality.

Many fundamental questions remain about the clonal structures of metastatic tumors, phylogenetic relationships among metastases, the scale of ongoing parallel evolution in metastatic and primary sites, how the tumor disseminates, and the role that the tumor micro-environment plays in the determination of the metastatic site.



Parkinsonism is an umbrella term that describes Parkinson's disease and conditions with similar symptoms like multiple system atrophy or corticobasal degeneration.

SYMPTOMS AND CAUSES

What are the symptoms?

The best-known symptoms of Parkinson's disease involve loss of muscle control. However, experts now know that muscle control-related issues aren't the only possible symptoms of Parkinson's disease.

Motor-related symptoms

Motor symptoms — which means movement-related symptoms — of Parkinson's disease include the following:

Slowed movements (bradykinesia). A Parkinson's disease diagnosis requires that you have this symptom. People who have this describe it as muscle weakness, but it happens because of muscle control problems, and there's no actual loss of strength.

Tremor while muscles are at rest. This is a rhythmic shaking of muscles even when you're not using them and happens in about 80% of Parkinson's disease cases. Resting tremors are different from essential tremors, which don't usually happen when muscles are at rest.

Rigidity or stiffness. Lead-pipe rigidity and cogwheel stiffness are common symptoms of Parkinson's disease. Lead-pipe rigidity is a constant, unchanging stiffness when moving a body part. Cogwheel stiffness happens when you combine tremor and lead-pipe rigidity. It gets its name because of the jerky, stop-and-go appearance of the movements (think of it as the second hand on a mechanical clock).

Unstable posture or walking gait. The slowed movements and stiffness of Parkinson's disease cause a hunched over or stooped stance. This usually appears as the disease gets worse. It's visible when a person walks because they'll use shorter, shuffling strides and move their arms less. Turning while walking may take several steps.

Blinking less often than usual. This is also a symptom of reduced control of facial muscles.

Cramped or small handwriting. Known as micrographia, this happens because of muscle control problems.

Drooling. Another symptom that happens because of loss of facial muscle control.

Mask-like facial expression. Known as hypomimia, this means facial expressions change very little or not at all.

Trouble swallowing (dysphagia). This happens with reduced throat muscle control. It increases the risk of problems like pneumonia or choking.

Unusually soft speaking voice (hypophonia). This happens because of reduced muscle control in the throat and chest.

Non-motor symptoms

Several symptoms are possible that aren't connected to movement and muscle control. In years past, experts believed non-motor symptoms were risk factors for this disease when seen before motor symptoms. However, there's a growing amount of evidence that these symptoms can appear in the earliest stages of the disease. That means these symptoms might be warning signs that start years or even decades before motor symptoms.

Non-motor symptoms (with the potential early warning symptoms in bold) include

Autonomic nervous system symptoms. These include orthostatic hypotension (low blood pressure when standing up), constipation and gastrointestinal problems, urinary incontinence and sexual dysfunctions.

Depression.

Loss of sense of smell (anosmia).

Sleep problems such as periodic limb movement disorder (PLMD), rapid eye movement (REM) behavior disorder and restless legs syndrome.

Trouble thinking and focusing (Parkinson's-related dementia).

sSTAGES OF PARKINSONS DISEASE

the Movement Disorder Society-Unified Parkinson's Disease Rating Scale (MDS-UPDRS) classifies four different areas of how Parkinson's disease affects you:

Part 1: Non-motor aspects of experiences of daily living. This section deals with non-motor (non -movement) symptoms like dementia, depression, anxiety and other mental ability- and mental health-related issues. It also asks questions about pain, constipation, incontinence, fatigue, etc.

Part 2: Motor aspects of experiences of daily living. This section covers the effects on movement-related tasks and abilities. It includes your ability to speak, eat, chew and swallow, dress and bathe yourself if you have tremors and more.

Part 3: Motor examination. A healthcare provider uses this section to determine the movement-related effects of Parkinson's disease. The criteria measure effects based on how you speak, facial expressions, stiffness and rigidity, walking gait and speed, balance, movement speed, tremors, etc.

Part 4: Motor complications. This section involves a provider determining how much of an impact the symptoms of Parkinson's disease are affecting your life.

Familial Parkinson's disease

Parkinson's disease can have a familial cause, which means you can inherit it from one or both of your parents. However, this only makes about 10% of all cases.

Idiopathic Parkinson's disease

idiopathic Parkinson's disease happens because of problems with a protein called α-synuclein. Proteins are chemical molecules that have a very specific shape. When some proteins don't have the correct shape — a problem known as protein misfolding — body can't use them and can't break them down.

The proteins build up in various places or in certain cells (tangles or clumps of these proteins are called Lewy bodies). The buildup of these Lewy bodies (which doesn't happen with some of the genetic problems that cause Parkinson's disease) causes toxic effects and cell damage.

Protein misfolding is common in many other disorders, such as Alzheimer's disease, Huntington's disease, multiple forms of amyloidosis and more.

Induced Parkinsonism

There are conditions or circumstances experts have linked to parkinsonism.

The possible causes are:

Medications. Several medications can cause a parkinsonism-like effect. The Parkinson's-like effects are often temporary if you stop taking the medication that caused them before the effects become permanent. However, the effects can linger for weeks or even months after you stop taking the medication.

Encephalitis. Inflammation of your brain, known as encephalitis, can sometimes cause parkinsonism.

Toxins and poisons. Exposure to several substances, such as manganese dust, carbon monoxide, fumes from welding, or certain pesticides, can lead to parkinsonism.

Damage from injuries. Repeated head injuries, such as those from high-impact or contact sports like boxing, football, hockey, etc., can cause brain damage. The term for this is "post-

traumatic parkinsonism.".

DIAGNOSIS AND TESTS

Blood tests (these can help rule out other forms of parkinsonism).

Computerized tomography (CT) scan.

Genetic testing.

Magnetic resonance imaging (MRI).

Positron emission tomography (PET) scan.

New lab tests -

Researchers have found possible ways to test for possible indicators or Parkinson's disease. Both of these new tests involve the alpha-synuclein protein but test for it in new, unusual ways. While these tests can't tell you what conditions you have because of misfolded alpha-synuclein proteins, that information can still help your provider make a diagnosis.

The two tests use the following methods.

Spinal tap. One of these tests looks for misfolded alpha-synuclein proteins in cerebrospinal fluid, which is the fluid that surrounds your brain and spinal cord. This test involves a spinal tap (lumbar puncture), where a healthcare provider inserts a needle into your spinal canal to collect some cerebrospinal fluid for testing.

Skin biopsy. biopsy of surface nerve tissue includes collecting a small sample of your skin, including the nerves in the skin. The samples come from a spot on your back and two spots on your leg. Analyzing the samples can help determine if you're alpha-synuclein has a certain kind of malfunction that could increase the risk of developing Parkinson's disease.

MANAGEMENT AND TREATMENT

How is it treated, and is there a cure?

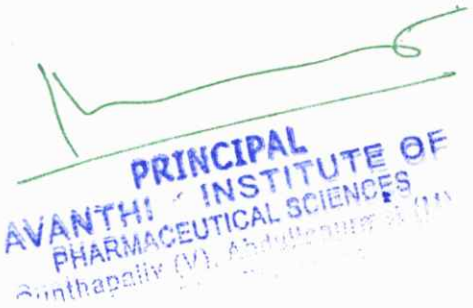
For now, Parkinson's disease is not curable, but there are multiple ways to manage its symptoms. The treatments can also vary from person to person, depending on their specific symptoms and how well certain treatments work. Medications are the primary way to treat this condition.

A secondary treatment option is a surgery to implant a device that will deliver a mild electrical

current to part of your brain (this is known as deep brain stimulation). There are also some experimental options, such as stem cell-based treatments, but their availability often varies, and many aren't an option for people with Parkinson's disease.

medications and treatments

Medication treatments for Parkinson's disease fall into two categories: Direct treatments and symptom treatments. Direct treatments target Parkinson's itself. Symptom treatments only treat certain effects of the disease.



Water soluble vitamins

1. B1 (thiamine) deficiency

Poor diet and ETOH

Wet BeriBeri: cardiac failure

Dry BeriBeri: polyneuropathy

Wernicke Encephalopathy – ophthalmoplegia and nystagmus, ataxia

Korsakoff syndrome: confabulation

2. B2 (riboflavin) deficiency

Cheilosis (cracks/fissures at angles of mouth)

Glossitis (inflam/atrophy of tongue)

Facial dermatitis

Normocytic anemia

3. B3 (niacin) deficiency

Pellagra: that is dermatitis, diarrhea, dementia

4. B6 (pyridoxine) deficiency

Peripheral neuropathy

Cheilosis, stomatitis, glossitis, irritability, confusion, depression

Treated by Isoniazid

5. B9 (folic acid)

Macrocytic, Normochromic, No Neuro sxs

Leafy greens

Purine and thymidine Synth for DNA

Preggers (NTDs), Sickle Cell

Treated by Phenytoin, Methotrexate, Trimethoprim, pyrimethamine

6. B12 (cobalamin) Deficiency

Macrocytic, megaloblasticanemia; Pernicious anemia

Neurologic Symptoms : paresthesias, subacute combined degeneration due to abnormal myelin.

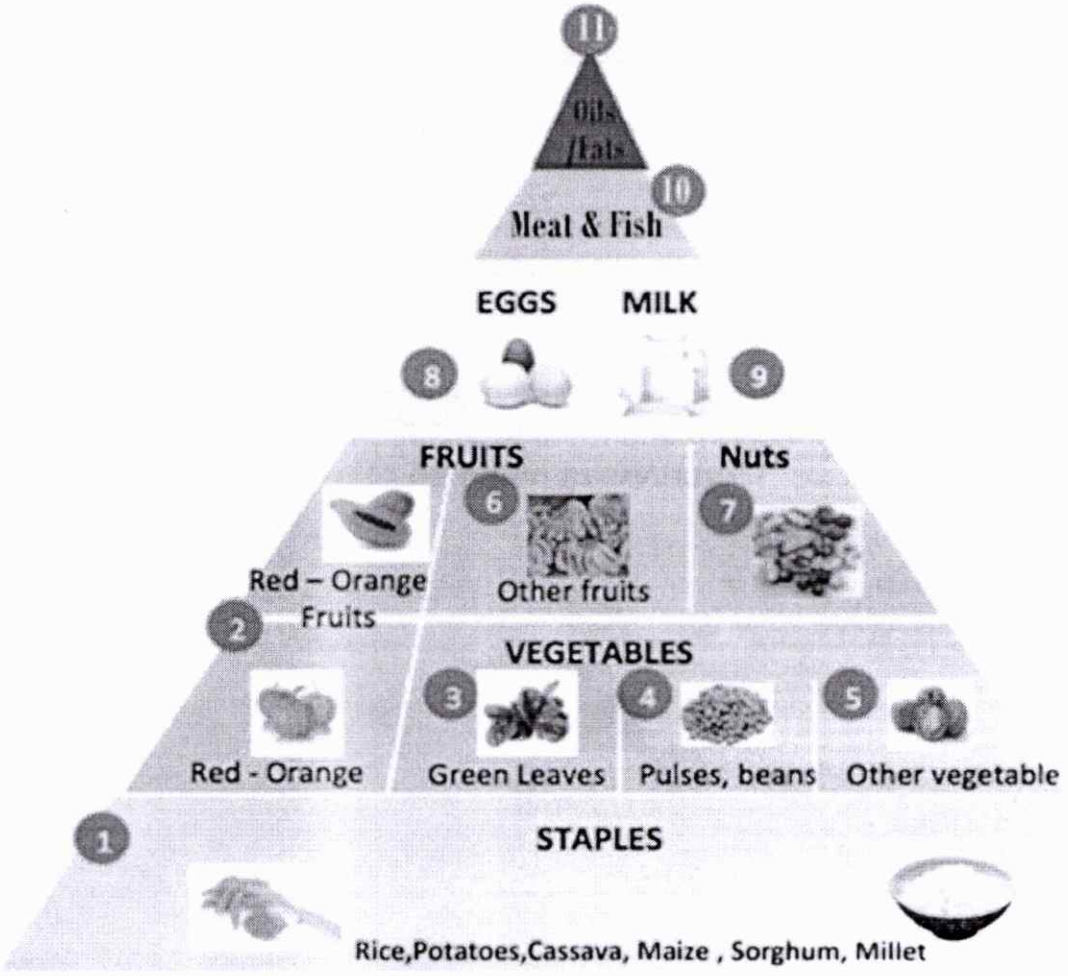
Prolonged deficiency leads to irreversible nervous system damage.

7. Vitamin C deficiency

Scurvy, hemorrhagic diathesis, poor wound healing

Nutrient	Function	Food sources
Vitamin A	Strengthens our immunity which helps us fight off infections Improves vision in dim light Keeps the skin and the linings of some parts of the body, such as the nose, healthy	Dark green leafy vegetable spinach, broccoli and carro pumpkin, liver, fish, kidney produce such as yoghurt, e margarine
Vitamin D	Helps the body absorb calcium Keeps bones and teeth healthy	Sun light, fish liver oils, mill margarine, eggs, liver
Vitamin E	Helps maintain cell structure by protecting cell membranes	Soya, groundnuts, fortified oil, wholegrain cereals, egg butter, tomatoes
Vitamin K	Helps with blood clotting	Vegetables such as spinac cauliflower, and cabbage, b liver, meat, eggs
B-group Vitamins	Help the body release energy from food Keep the skin, eyes and the nervous system healthy	Millet, sorghum, beans, pe meat, milk, fresh fruit, gree vegetables, wholegrain cer
Vitamin C	Helps with wound healing Strengthens our immunity which helps us fight off infections	Citrus fruits such as orange tangerines, red and green p tomatoes, broccoli, potatoe
Folic acid	Helps form healthy red blood cells Helps reduce the risk of central nervous system defects such as spina bifida in unborn babies	Leafy green vegetables su broccoli, and lettuce, liver, fruits such as oranges, ban avocados and melons
Iron	Helps make red blood cells, which carry oxygen around the body	Liver, meat, offal, beans, n ground nuts, eggs, most da vegetables such as amara parsley
Calcium	Helps build strong bones and teeth Helps muscles and nerves function normally Helps to ensure blood clots normally	Milk, cheese and other dai leafy vegetables, such as c okra

The food pyramid: All of these types of food should be eaten but the foods at the b should be eaten most and those at the top more sparingly. ***For a healthy diet, a mir of 5 food groups need to be eaten every day.***



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Vitamin	Chemical Name	Deficiency Diseases
Fat soluble Vitamins		
A	Retinol, Retinal, Retinoic acid	Night-blindness and keratomal
D	Ergocalciferol (D_2), Cholecalciferol (D_3)	Rickets and Osteomalacia
E	Tocopherol	Mild hemolytic anemia in newbo
K	Phylloquinone (K_1), Menaquinones (K_2)	Bleeding diathesis
Water soluble vitamins		
B_1	Thiamine	Beriberi
B_2	Riboflavin	Ariboflavinosis
B_3	Niacin, Niacinamide	Pellagra
B_5	Pantothenic acid	Paresthesia
B_6	Pyridoxine, Pyridoxamine, Pyridoxal	Anemia peripheral neuropathy
B_7	Biotin	Dermatitis
B_9	Folic acid, Folinic acid	Neural tube defects
B_{12}	Cyanacobalamine	Megaloblastic anemia
C	Ascorbic acid	Scurvy

FAT SOLUBLE VITAMINS:-

1. Vitamin A deficiency

Night blindness, dry skin, growth failure

Bitot spots, keratomalacia, xerophthalmia

2. Vitamin D deficiency

Rickets in children, osteomalacia in adults, hypocalcemic tetany

3. Vitamin E deficiency

Hemolyticanemia, posterior and spinocerebellar tract demyelination
Nystagmus

Looks like B12 def but without labs or hypersegmented neutrophils

4. Vitamin K deficiency

Hemorrhage –Inc INR, PT (2, 7, 9, 10)

bruising

Mineral Deficiencies

- Mg deficiency symptoms

Nervousness, muscle tremors, ataxia

Heart damage

*Death-uncommon

- Zn deficiency

Alopecia

Pustular skin rash – Perioral and extremities

Hypogonadism

Impaired wound healing

Impaired Taste

Looks like B12 def but without labs or hypersegmented neutrophils

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Pustular skin rash – Perioral and extremities

Hypogonadism

Impaired wound healing

Impaired Taste

Immune dysfunction

- Cu deficiency

Skin and hair depigmentation

Brittle hair

Neuro Sx – Ataxia and peripheral neuropathy

Osteoporosis

Sideroblasticanemia

- Selenium deficiency

Thyroid dysfunction

Cardiomyopathy

Immune dysfunction

- Chromium deficiency

Impaired glucose tolerance

Features of malabsorption in celiac disease

General symptoms- bulky, foul smelling, floating stools

Fat & Protein: loss of muscle mass, loss of SC fat, fatigue

Iron: pallor, fatigue

Ca + Vit D: osteomalacia, osteoporosis

Vit K: bruising

Vit A: hyperkeratosis

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HISTOLOGICAL DIAGNOSIS OF MALIGNANCY

When the diagnosis of Cancer is suspected based on clinical examination & other investigations then the diagnosis must be confirmed.

The most reliable method to confirm the diagnosis of Cancer is "Histological examination of biopsy"

Histological method is based on Microscopic examination of properly ~~fixed~~ fixed tissue

Histological method is most valuable in arriving at the accurate diagnosis.

The tissue must be fixed in 10% Formalin for

⇒ light microscopic examination.

⇒ The tissue must be fixed in Glutaraldehyde for Electronmicroscopic examination

⇒ "The histological diagnosis is Made on the base of cytological features"

Benign tumours resemble Normal tissue & are unable to invade & metastasize.

Malignant tumours are identified by

clippercells, invasion, metastasis.

+

Microscopic features.

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Meanings

-ness = study of structures without the use of a microscope

pattern = design

3 Basal polarity =

Basal polarity is an cardinal molecular feature of adult eukaryotic epithelial cells.

⇓

Basal polarity is involved in key cellular processes like cell migration & Maintenance of tissue architecture"

4 pleomorphism = Multiple shapes & Sizes

6 Nucleo-cytoplasmic ratio = "Is a measurement used in cell biology"

N:C ratio = $\frac{\text{Size of nucleus of the cell}}{\text{Size of cytoplasm of cell}}$

N:C ratio = Indicates the maturity of cells

NOTE: As the cells matures the size of the nucleus decreases.

⑥ Anisonucleosis : Morphological manifestation of Nuclear injury

characterised by Variation in the size of the nucleus.

⑦ hyperchromatism :- A condition in which all nucleus stains more intensely than normal.

⑧ "Nuclear atypia = abnormal appearance of cell nuclei atypical nuclei are generally pleomorphic.

FEATURES	BENIGN	MALIGNANT
I Clinical & gross features:		
1. Boundaries	Encapsulated (or) well circumscribed	Poorly circumscribed & Irregular
2. Surrounding tissue	Frequently compressed.	Generally invaded
3. Size	Generally small	Generally large.
4. Secondary changes	Occur Very Rarely.	Occur more frequently.
5. Shape.	Oval or spherical	Irregular.

III Microscopic features

1. Pattern.	closely resembles the tissue of origin	poorly resembles the tissue of origin
2. Basal polarity	Retained	lost
3. Pleomorphism.	Absent	present
4 Nucleocytoplasmic ratio	Normal.	Increased.
5. Anisonucleosis.	Absent	Present
6. Hyperchromatism	Absent	present
7. Mitosis	Typical mitosis	Atypical & abnormal.
8. Tumour giant cells.	May be present Without atypical nucleus.	present with Atypical nucleus

9.	Chromosomal abnormalities	less often	Frequently present
10	Function.	Well maintained	May be maintained or lost as abnormal
III	Growth rate	Relatively slow.	Generally Rapid.
IV	Local Invasion	No.	frequently present.
V	Metastasis	Absent	present
VI	Prognosis	local complications	Death by local & metastatic complication.

IMMUNE TOLERANCE

Defination: Ability of the Immune system To recognise "Self tissue and Antigens"

Immune tolerance is present since foetal life

Immune tolerance is achived by following Mechanisms:

(i) clonal elimination (ii) clonal anergy

(iii) Suppressor T cells.

(i) Clonal elimination: "During Embryonic development

According to This theory: T-cells Maturing in the thymus acquire immune tolerance.

"These T cells are then eliminated by apoptosis"

(ii). Clonal Anergy: According to this theory:

T-cells which acquired immune tolerance are Not eliminated

BUT

Become Non responsive / Inactive

(iii) Suppressor T cells: According to this theory Immune tolerance is achived by Suppressor T cells which stops the immune system from becoming Over active.

Important steps of the ischaemic cascade

Without adequate blood supply and thus lack of oxygen, brain cells lose their ability to produce energy – particularly adenosine triphosphate (ATP).

Cells in the affected area switch to anaerobic metabolism, which leads to a lesser production of ATP but releases a by-product called lactic acid.

Lactic acid is an irritant, which has the potential to destroy cells by disruption of the normal acid-base balance in the brain.

ATP-reliant ion transport pumps fail, causing the cell membrane to become depolarized; leading to a large influx of ions, including calcium (Ca++), and an efflux of potassium.

Intracellular calcium levels become too high and trigger the release of the excitatory amino acid neurotransmitter glutamate.

Glutamate stimulates AMPA receptors and Ca++-permeable NMDA receptors, which leads to even more calcium influx into cells.

Excess calcium entry overexcites cells and activates proteases (enzymes which digest cell proteins), lipases (enzymes which digest cell membranes) and free radicals formed as a result of the ischaemic cascade in a process called excitotoxicity.

As the cell's membrane is broken down by phospholipases, it becomes more permeable, and more ions and harmful chemicals enter the cell.

Mitochondria break down, releasing toxins and apoptotic factors into the cell.

Cells experience apoptosis.

If the cell dies through necrosis, it releases glutamate and toxic chemicals into the environment around it. Toxins poison nearby neurons, and glutamate can overexcite them.

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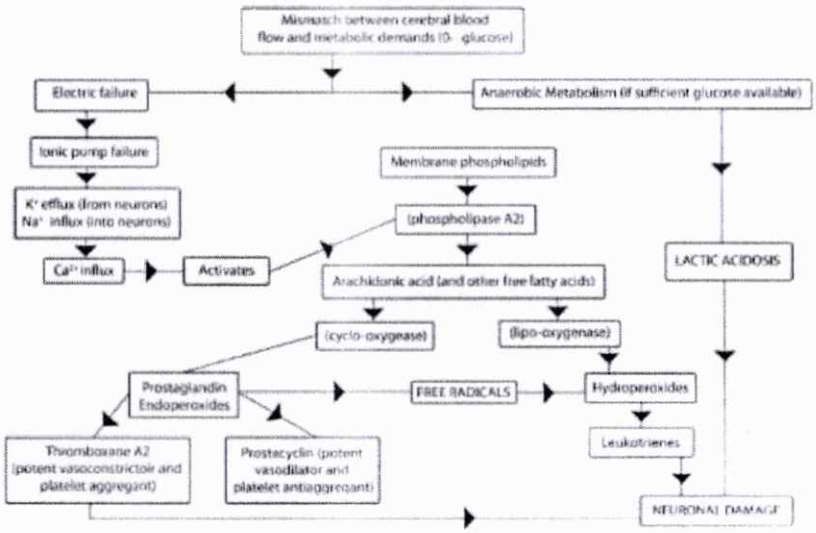
The Ischaemic Cascade



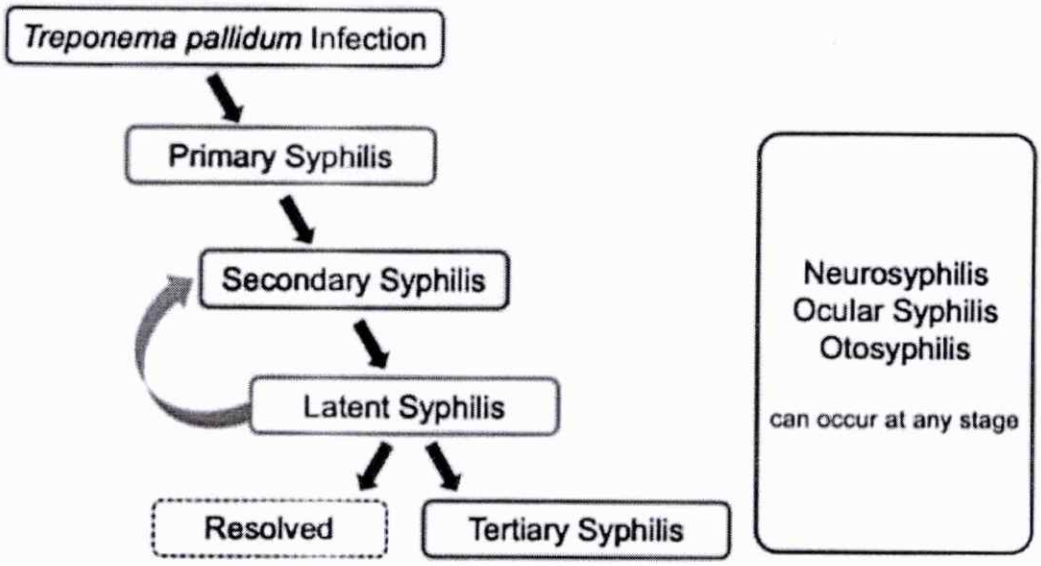
The loss of vascular structural integrity results in a breakdown of the protective blood brain barrier and contributes to cerebral oedema, which can cause secondary progression of the brain injury.

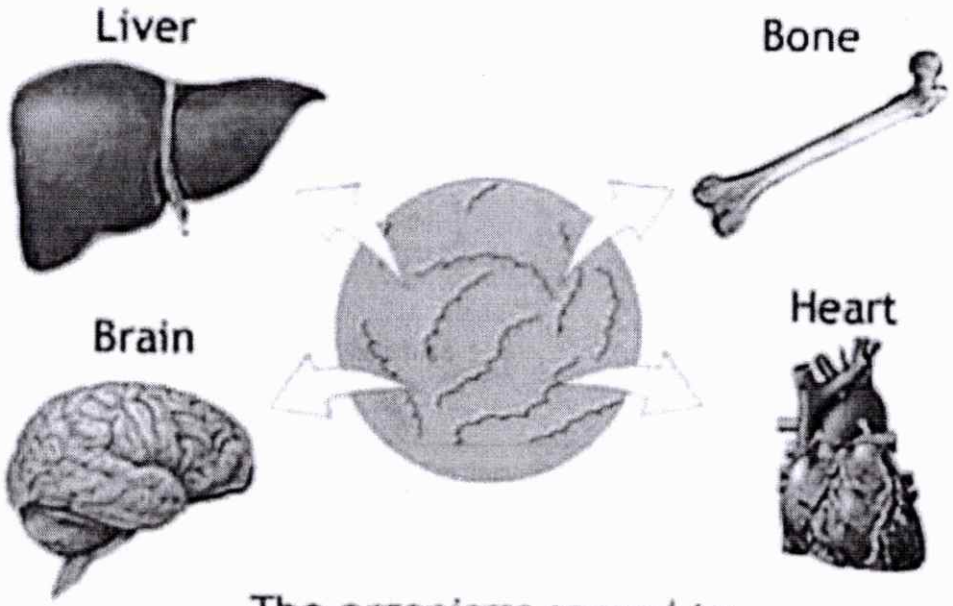
Pathophysiology of haemorrhagic stroke

Haemorrhagic strokes are due to the rupture of a blood vessels leading to compression of brain tissue from an expanding haematoma. This can distort and injure tissue. In addition, the pressure may lead to a loss of blood supply to affected tissue with resulting infarction, and the blood released by brain haemorrhage appears to have direct toxic effects on brain tissue and vasculature.

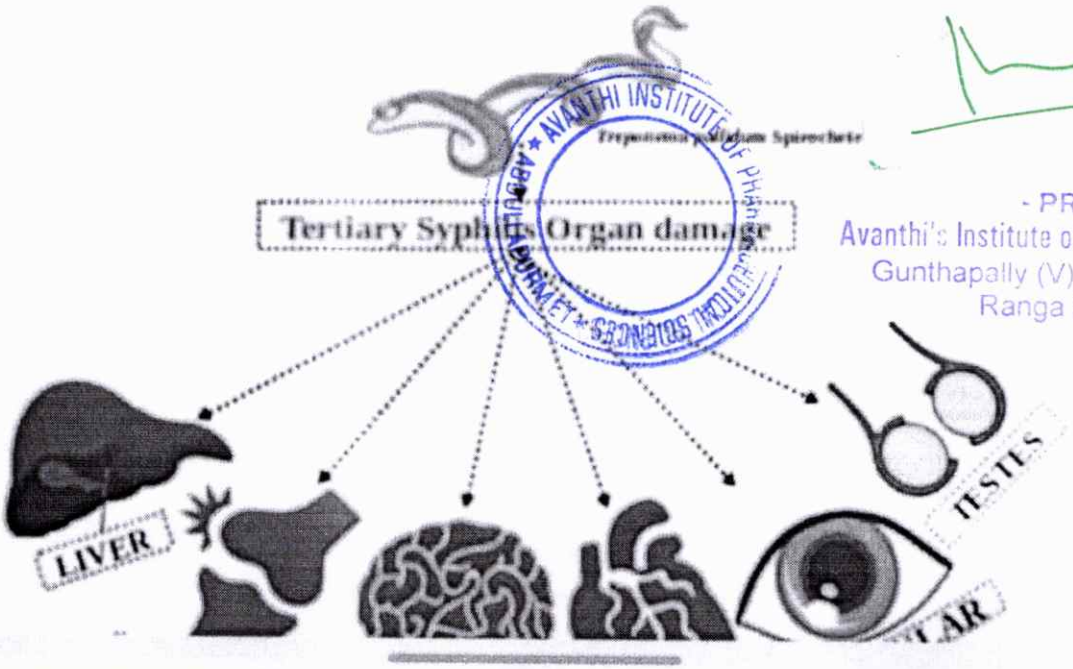
Intracerebral haemorrhage is caused by rupture of a blood vessel and accumulation of blood within the brain. This is commonly the result of blood vessel damage from chronic hypertension, vascular malformations, or the use of medications associated with increased bleeding rates, such as anticoagulants, thrombolytics, and antiplatelet agents.

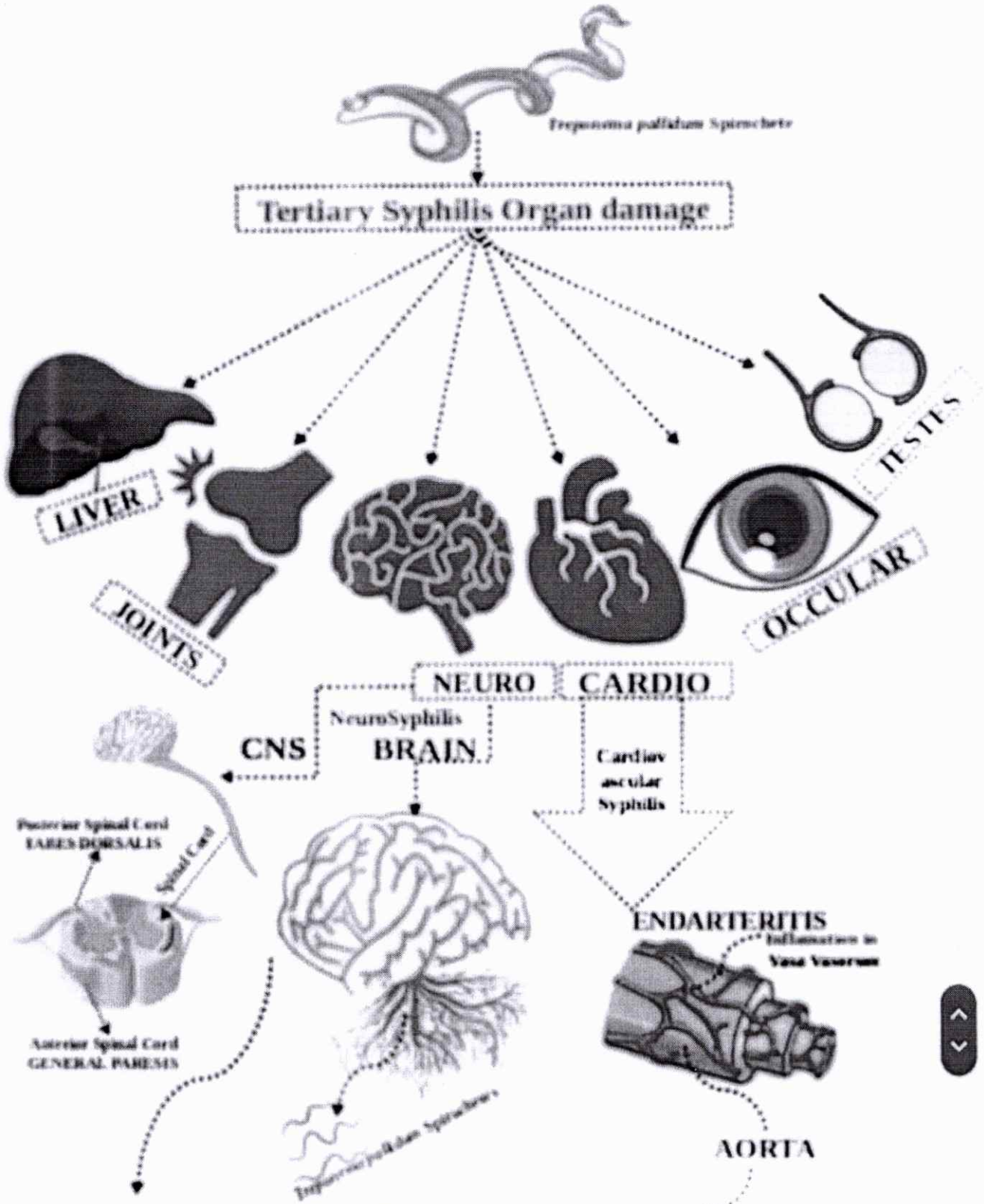
Subarachnoid haemorrhage is the gradual collection of blood in the subarachnoid space of the brain dura, typically caused by trauma to the head or rupture of a cerebral aneurysm.





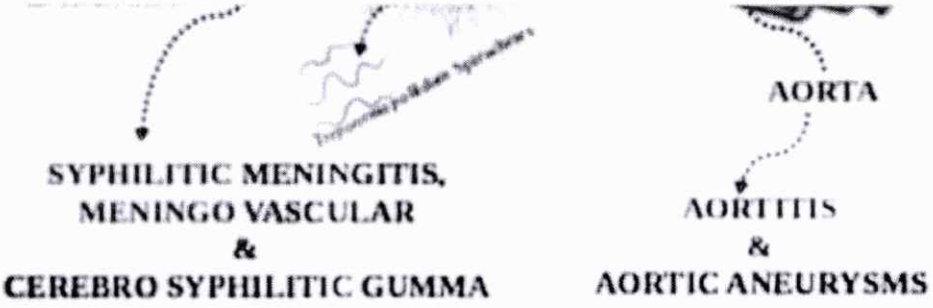
The organisms spread to various organs causing lesions or gummas





Syphilis is a bacterial infection typically spread through sexual contact. Congenital syphilis occurs when infection is passed from parent to fetus during pregnancy and can result in stillbirth, infant death, or other health issues.

Syphilis can present at different stages, so early detection and treatment are important to prevent disease progression and



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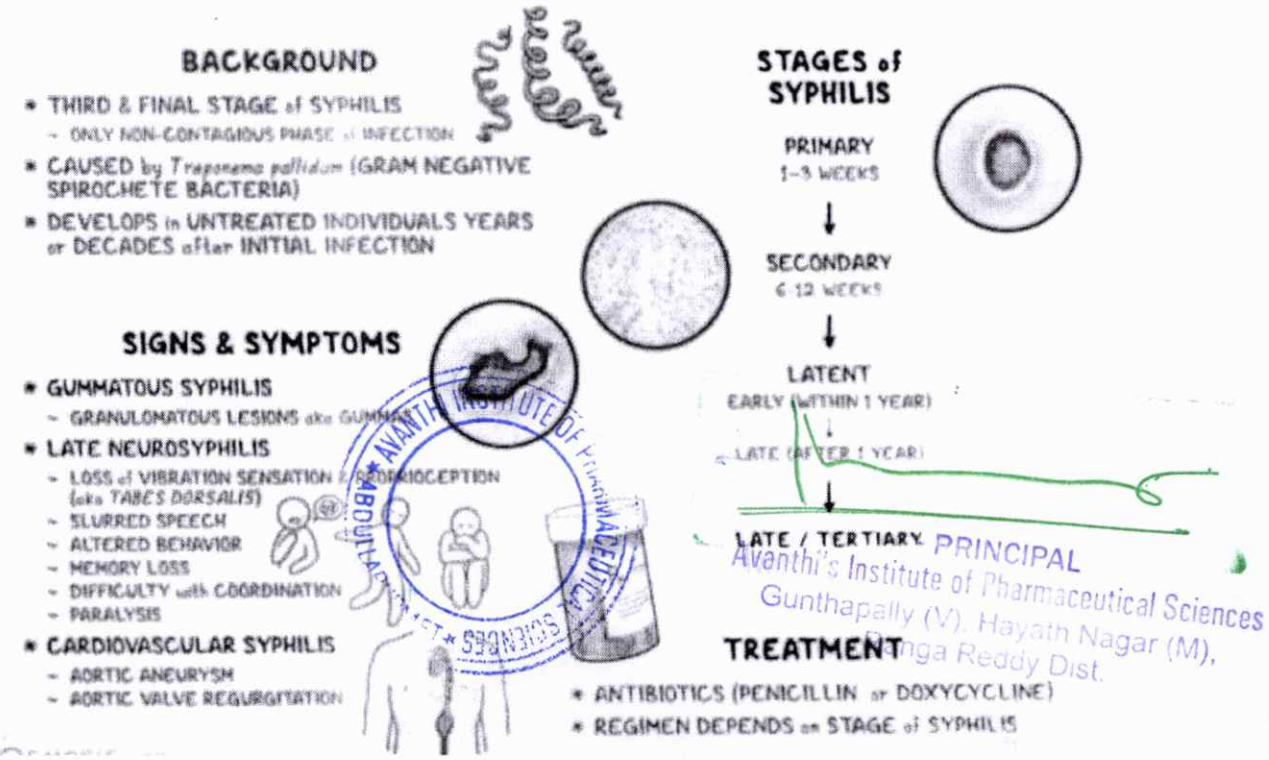
Syphilis can present at different stages, so early diagnosis and treatment are important to prevent disease progression and transmission to others.

Stage	Time period	Symptoms
Primary	10-90 days after infection	Painless ulcer (chancre) on the genitals or mouth Typically heals on its own within 3-6 weeks
Secondary	Varies, typically 4-10 weeks after primary stage	Full-body rash (can involve palms of hands and soles of feet) and flu-like symptoms (eg, fever, headache, sore throat)
Latent	After untreated secondary syphilis	No symptoms but the infection is still present Can still be transmitted congenitally
Tertiary	Years or decades after initial infection	May cause damage to the brain, nerves, eyes, heart, blood vessels, liver, bones, and joints May be life-threatening

Neurosyphilis (infection of the brain and spinal cord) can occur at any stage and cause meningitis, stroke, hearing loss, blindness, paralysis, and dementia.



IMMUNOPATHOLOGY

⇓

"Moldy hay = అచ్చు పట్టిన వరి గడ్డి"

Autoimmune disease = Disease caused by Antibodies (or) T-cells generate against substances Naturally present in the body

Autoimmune disease involves formation of Antibodies against own tissue antigens"

Clonal anergy = Absence of Normal immune response To a particular antigen"

Suppressor T cells = Type of T cell which stops the Immune system from becoming Overactive"

Sequestered = "వేరుపరచబడిన"

graft = a piece of living tissue that is transplanted surgically

"Compatibility = అనుకూలత = a state in which two things are able to exist together without problems.

"Human Leucocyte Antigen System"

(HLA System)

(or)

Human Leucocyte Antigen Complex

(HLA Complex)

(or)

Major Histocompatibility Complex

(MHC)

"Human leucocyte antigens are the antigens located on the

(i) plasma membrane of all the nucleated cells of body and the platelets

"Human leucocyte antigens were first discovered on the

(ii) plasma membrane of leucocytes

(or)

"HLA were first discovered on Leucocytes"

(iii) HLA's are Not a Component of Immune system

BUT

"It plays an important role in the regulation of Immune System"

(iv) Human Leucocyte antigens are highly pleomorphic"

The genes which code for HLA's are located on short arm of chromosome 6"

(V) These genes occupy four regions (A,B,C,D) on the short arm of chromosome 6

Depending upon the characteristics of HLA's. They are divided into 3 classes.

1. Class I HLA's : CD8 T cells carry receptors for "class I HLA's"

⇓ So

Class I HLA's are identified by CD8 T cells.

2. Class II HLA's : B cells & CD4 T cells carry receptors for class II HLA's

⇓ So

"Class II HLA's are identified by B cells, CD4 cells."

3 Class III HLA's : Components of the Complement system

IMPORTANCE OF HLA SYSTEM

(or)

SIGNIFICANCE OF HLA SYSTEM

1. Organ transplantation 2 Regulation of immune system
3. Diseases associated with HLA's.

1. ORGAN TRANSPLANTATION:

"Immunsystem of the organ recipient will recognise

The HLA's on the donar organ.

Then

The immunesystem of the organ recipient can accept it or reject it.

"Organ rejection is seen in genetically Non-identical Transplants"

"In organ rejection BCA CMI & HI are involved"

2. REGULATION OF IMMUNE SYSTEM:

Class I HLA's regulate the functioning of CMI.

Class II HLA's regulate the functioning of HI.

3. DISEASES ASSOCIATED WITH HLAs:

Number of diseases has been found to have a close association with specific human leucocyte antigens

Eg: Ankylosing Spondylitis
Rheumatoid Arthritis
DM-I.

The exact mechanism of this close association between the disease and type of HLA is Not known.

CBT - time 12 min

(1) Anxiolytic BZDs - chlordiazepoxide, Diazepam
↓
hypnotic relief ... orally
...

(2) SSRIs - alter the level production of 5-HT in brain
make ... difficult.

Cont'd Etiology of HBV

Structure of HBV - consists of 4 overlapping genes which encode 4 different proteins

1) S gene - codes 4 HBsAg viz present on outer surface of ... small spherical particles & tubular struc. of virus.

2) P gene - largest; codes 4 DNA polymerase

3) C gene - codes 4 two nucleocapsid proteins - HBeAg & HBcAg.

4) X " " - HBxAg viz a small non-particulate protein

Pathophy. of V. Hepatitis :-

1) Carrier state < asymptomatic healthy carrier ... ; " carrier c disease - may have chronic hepatitis or cirrhosis

2) Asympt. Infec^n - only ↑ liver enz. or presence of Antibodies otherwise have no symptoms.

3) Acute Hepatitis :- 4 phases.

1) Incub.° period - HAV - 4 wks
HBV - 10 "
HCV - 7 "
D - 6 "
E - 2-8 wks

2) Pre-icteric phase — prodromal constitutional Sympt i.e. anorexia, nausea, vomit, fatigue, arthralgia, headache, low grade fever f/b jaundice.
→ ↑ liver enz.

3) Icteric phase - onset of clinical jaundice & above sympt resol
→ dark urine due 2 bilirubinuria
→ clay colored stools due (cholestasis)
→ pruritus due 2 ↑ serum bile acids
→ ↓ loss & abd. discomfort.
* Lasts 4 1-4 wks

4) Post-icteric - lasts 4 2-12 wks & is a recovery phase
- longer in HBV & HCV Infec"
- 1% of acute cases progress 2 fulminant Hepatitis

Changes in Morphology :- 1) Hepatocellular Injury — ballooning degen"
— acidophilic "
& presence of councilman's
— diagnostic necrosis

2) Inflam. infiltration by mononuclear cells — bridging "
3) Kupffer cells hyperplasia
4) Cholestasis - intracytoplasmic bile pigment granules.
5) Regeneration - tubular diarrangt is noticed due 2 necrosis

4) CHRONIC HEPATITIS :- continuing / relapsing hepatitis for > 6 months along 2 serologic & pathologic evidences of inflam & necrosis.

→ Pathologic features common in HBV & HCV Infec" are —
a) Piecemeal necrosis — necrosis destruction of hepatocytes one after the other at limiting plate of portal tract
Expanded portal tract is characteristic.

b) portal tract lesions – expanded portal tract due 2 proliferation of bile ductules.

c) Intra-lobular lesions – same changes occur as in morphologic changes in Acute Hepatitis.

d) Bridging fibrosis – Also, in Hep. C, fatty changes can be seen & in Hep. B, ground glass hepatocytes indicating presence of HBsAg in the cytoplasm.

} irreversible damage.

Grading of Hepatitis :- Hepatitis Activity Index (HAI) is calculated by considering 2 things -

a) Necroinflammatory Activity – from 0 to 4 ['0' = no necrosis/inflam
4 = highest "]

b) Stage of fibrosis – scored from 0 to 6 [0 - no fibrosis
6 = Cirrhosis]

Clinical features of Chronic :-

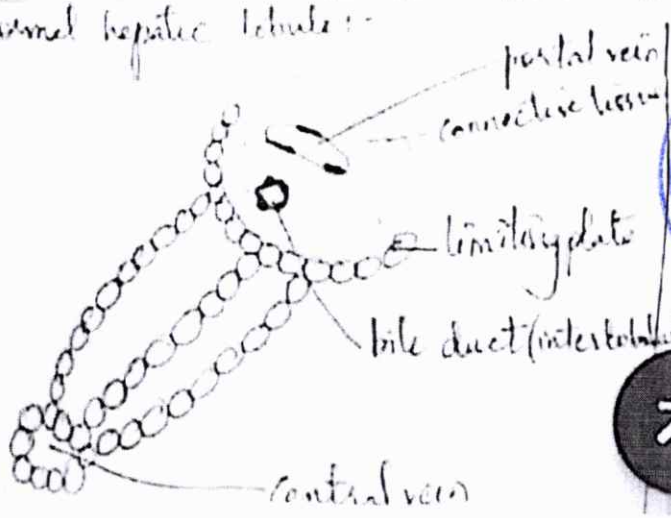
- ↑ liver enz. (transaminases)
- mild hepatomegaly & tenderness ; splenomegaly
- ↑ Prothrombin time, hyperbilirubinemia, hyperglobulinemia, ↑↑ ALP
- circulating immune complexes to HBV & HCV.

5) FULMINANT HEPATITIS :- occurs in 2 patterns -

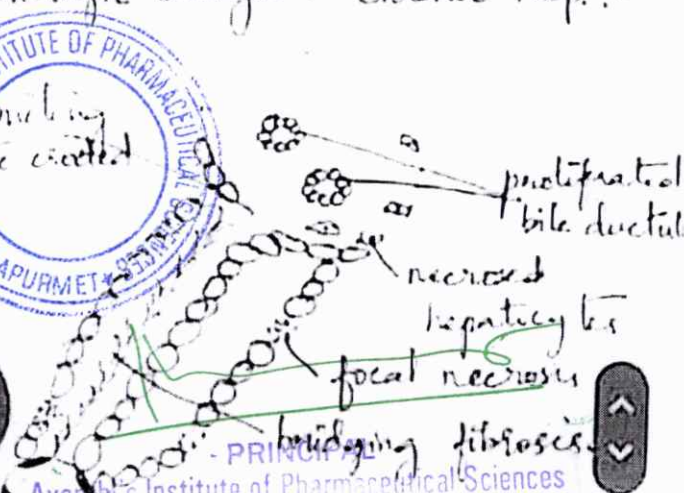
a) Sub-massive – not very fast progressing & extends upto 3 months

b) Massive – Rapidly progressing liver failure & occurs within 2-3 weeks

normal hepatic lobule :-

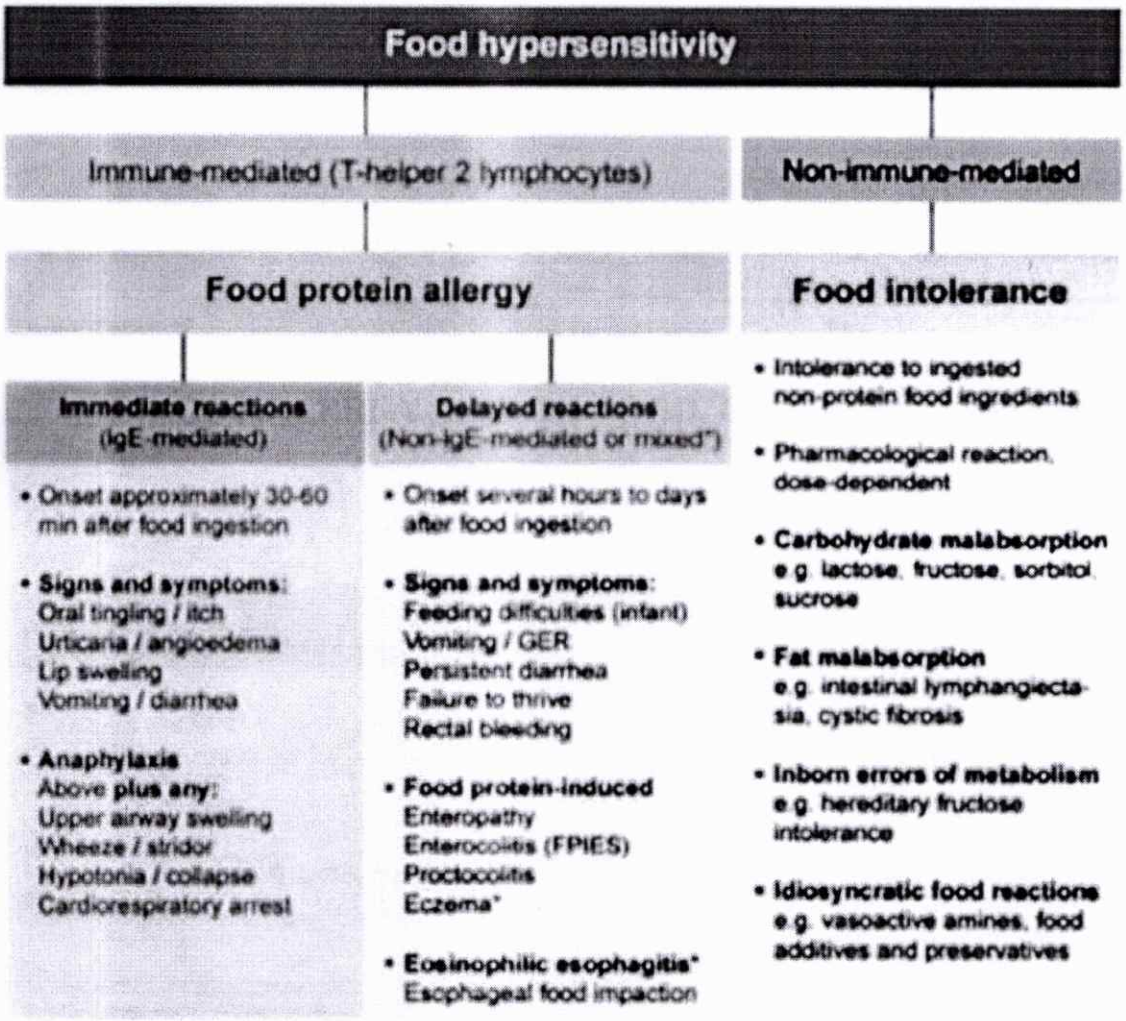


pathologic changes in Chronic Hep. :-



ALLERGY DUE TO FOOD, CHEMICALS AND DRUGS

Allergy due to Food-



Allergy Due to Chemicals-

- Immune system overreacts to chemicals that are normally harmless. These chemicals can be in products that are-

Allergy due to chemicals:

Chemical allergens are small molecules able to form a sensitizing complex once they bound to proteins.

One of the most frequent manifestations of chemical allergy is contact hypersensitivity, which can have serious impact on quality of life.

Allergic contact dermatitis is a predominantly CD8+T cell-mediated immune disease, resulting in erythema and eczema.

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Toxic chemicals found in shampoo	• Sodium Lauryl Sulfate (SLS) • SLS is an inexpensive detergent and surfactant that is widely used in shampoo, body wash, shaving cream, toothpaste, and other products. • The American College of Toxicology found that SLS easily penetrates the skin and can circulate in the body for up to five days, leaving residues in the heart, liver, lungs, and brain. • SLS can strip moisture and oils from the hair and skin, causing rashes, hair loss, and a condition similar to dandruff. 2. Sodium Laureth Sulfate (SLES) ➢ It is frequently contaminated by 1,4 dioxane, a byproduct of the ethylation oxide used to make harsh petrolum-based ingredients more gentle. ➢ It is a known carcinogen and suspected of causing kidney damage. ➢ 1,4 Dioxane will not be found in the list of ingredients on your shampoo bottle because it is a byproduct and not part of the formulation. ➢ Dioxane has a long life in the body, primarily because the liver cannot metabolize it effectively.
Fragrance	• These are highly toxic and can result in liver toxicity, dam... to the central nervous system, allergies, brain fog, obesity, asthma, headache, contact dermatitis, organ toxicity, and cancer. • They are made from petrochemicals and phthalates, and have been linked to learning disabilities and other developmental abnormalities in children whose mothers had high levels in their bodies during gestation. • Byproducts from the production of fragrances include dioxin and formaldehyde.
Cosmetic Allergy	• The chemicals present in cosmetics causes allergy in some peoples. • The groups of allergens that appear to most frequently cause cosmetic allergy are;- • fragrances, • preservatives, and • paraphenylenediamine (PPD) found in hair dyes.

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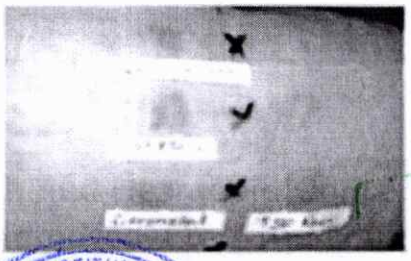
Other allergens used in cosmetics that can cause cosmetics allergy include:

- ❑ Lanolin (wool alcohol)
- ❑ Coconut diethanolamide
- ❑ Glyceryl monothioglycolate
- ❑ Methyldibromo glutaronitrile
- ❑ Rosin (colophony)
- ❑ Propolis
- ❑ Thiomersal
- ❑ Sunscreen allergens
- ❑ Nail cosmetic allergens

What is the treatment for cosmetics allergy?

Contact dermatitis should clear rapidly once the cosmetic allergen is removed. Over-the-counter creams and ointments containing mild topical steroids, such as hydrocortisone 0.5-2.5%, may be used to help control itching, swelling, and redness. In more severe cases, a prescription steroid cream may be required, as well as antibiotic medication if the skin becomes blistered and infected. Bland emollients such as cetomacrogol cream can be used to soothe and relieve dryness.



Treatments for Chemical allergy

•Avoid exposure to allergen. Medications may be used to alleviate some symptoms e.g. antihistamines and topical steroids.

• Oral steroids may be needed in severe cases but this is rare.

Treatments for Chemical allergy

- Avoid exposure to allergen. Medications may be used to alleviate some symptoms e.g. antihistamines and topical steroids.
- Oral steroids may be needed in severe cases but this is rare.
- Moisturizers can alleviate skin symptoms and antibiotics may be needed if secondary skin infections develop from initial skin symptoms such as rashes.

Signs and Symptoms-

- Red skin
- Scaly patches
- Blisters that ooze
- Burning or itching, which may be intense
- Swelling of the eyes, face, and genital area
- Hives
- Sun sensitivity
- Darkened, "leathery," and cracked skin
-

Allergy Due to Drugs-

Mechanism-

Immunological reactions	Often mediated by a chemically reactive metabolite---------Non detoxification of metabolite that causes direct cytotoxicity and that leads to direct tissue damage + necrosis.
Pseudoallergic reactions	Direct mast cell activation and degranulation by drugs (opiates, vancomycin and radiocontrast media) that causes clinically indistinguishable form Type I hypersensitivity but not involve IgE (non immunologic reactions)
Pharmacogenetic variation	It may involve: Red cell enzyme defects Porphyria Malignant hyperthermia.

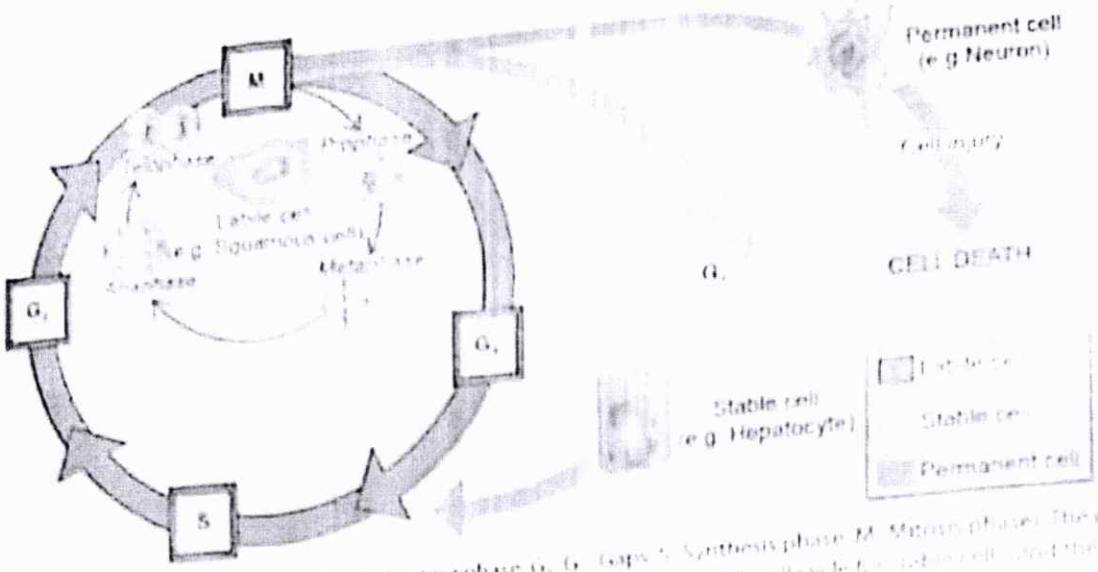


Figure 4.44 Parenchymal cells in relation to cell cycle (G0 - Resting phase, G1 - Gap 1, S - Synthesis phase, M - Mitosis phase). The inner circle shown with green line represents cell cycle for labile cells and the outer circle shown with red line represents cell cycle for permanent cells. Compare them with traffic signals - green stands for ... red stands for ... and yellow signal for ... which applies here to stable cells which can be stimulated to enter cell cycle and not to ... permanent cells.

1. *Labile cells* which are continuously dividing cells remain in the cell cycle from one mitosis to the next.
2. *Stable cells* are in the resting phase (G0) but can be stimulated to enter the cell cycle.
3. *Permanent cells* are non-dividing cells which have left the cell cycle and are destined to die after cell injury.

Regeneration of any type of parenchymal cells involves the following 2 processes:

i) Proliferation of original cells from the margin of injury with migration so as to cover the gap.
ii) Proliferation of migrated cells with subsequent differentiation and maturation so as to reconstitute the original tissue.

REPAIR

Repair is the replacement of injured tissue by fibrous tissue. Two processes are involved in repair:

- Granulation tissue formation.
- Wound contraction and strength.

Repair response takes place by participation of mesenchymal cells (consisting of connective tissue stem cells, fibrocytes and histiocytes), endothelial cells, macrophages, platelets, and the parenchymal cells of the injured organ.

Granulation Tissue

The term granulation tissue ... pinkish granular ... and pink appearance of the ... corresponds histologically to proliferation of new small blood vessels which are slightly lifted on the surface and has a thin covering of fibroblasts and young collagen.

The process of granulation tissue formation can be discussed under following 4 phases which are not mutually exclusive and have enough overlapping (Fig. 4.45):

1. BLEEDING PHASE: Following trauma, there is bleeding which may stop after a few hours (on an average within 4-6 hours) but may vary, this is followed by clotting of blood at the site of injury.

2. INFLAMMATORY PHASE: Inflammation is an essential component of healing process. After blood clotting, fibrin and fibronectin remain in the tissues which form the substrate for

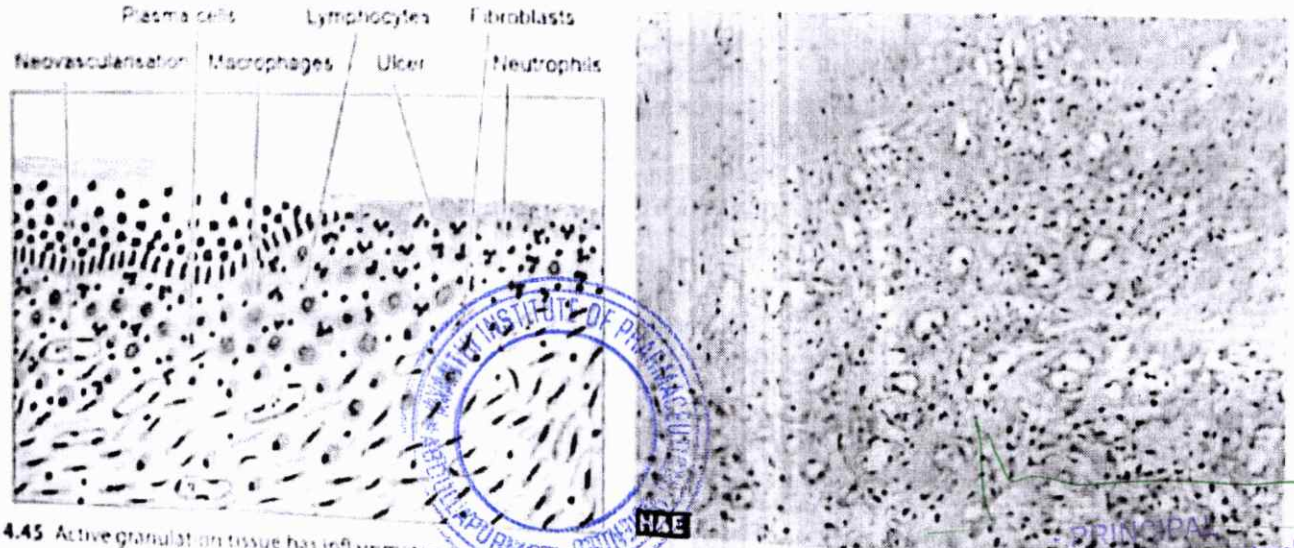


Figure 4.45 Active granulation tissue has inflammatory cells, ... newly formed blood vessels and young fibrous tissue in background.

adhesion of various inflammatory cells. Inflammatory response involves vascular [illegible] mediators (page [illegible]) [illegible] exudates within [illegible] followed by influx of phagocytic cells (macrophages, [illegible]) within 24 hours. Dead and dying cells, necrotic debris, fibrin mesh and clot are cleared off by combination of proteolytic [illegible] liberated from neutrophils, [illegible] enzymes from dead tissue [illegible] activity of macrophages. The latter events [illegible] debridement phase.

3. PROLIFERATION PHASE [illegible] of repair material [illegible] within first [illegible] However [illegible] (4-6 months) [illegible] or new vascular [illegible]

i) Angiogenesis (neovascularisation): [illegible] vessels at the [illegible] cells from [illegible] proliferated [illegible] develop [illegible] vessels are [illegible] of new granulation [illegible] these blood vessels differentiate into muscular [illegible] thin-walled venules and true capillaries.

The process of angiogenesis is stimulated with proteolytic destruction of basement membrane. Angiogenesis takes place under the influence of following factors:

a) Vascular endothelial growth factor (VEGF) elaborated by mesenchymal cells while its receptor (VEGF-R) is present in endothelial cells [illegible]

b) Platelet derived growth factor (PDGF), transforming growth factor-β (TGF-β), basic fibroblast growth factor (b-FGF) and surface integrins are associated with cellular proliferation.

ii) Fibrogenesis: The newly formed blood vessels are present in an amorphous ground substance or matrix. The new fibroblasts have features intermediate between those of fibroblasts and smooth muscle cells (myofibroblasts). By about 4th day, predominantly type III collagen fibrils appear. The myofibroblasts have surface receptors for fibronectin molecules which form bridges between collagen fibrils. As repair matures, weak type III collagen fibrils are reabsorbed by collagenase and replaced with type I collagen, while the number of active fibroblasts and new blood vessels decreases.

4. REMODELLING PHASE: This phase begins around the time when proliferation phase is at peak (i.e. 2-3 weeks following injury). The main events in remodelling are refinement of collagen and its associated extracellular matrix. Type I collagen fibrils that have replaced now have more cross-links and greater tensile strength. This results in formation of inactive looking scar; this process is known as *cicatrisation*.

Wound Contraction and Strength

The wound starts contracting after 2-3 days and the process is completed by the 14th day. During this period, the wound is reduced by approximately 80% of its original size. Contracted wound results in rapid healing since lesser surface area of the injured tissue has to be replaced.

The wound is strengthened by proliferation of fibroblasts and myofibroblasts which get structural support from the extracellular matrix (ECM) (page 18). In addition to providing structural support, ECM can direct cell migration, attachment, differentiation and organisation. ECM is not a static structure but the matrix proteins comprising it undergo marked remodelling during foetal life which slows down in adult tissues. These matrix proteins are degraded by a family of metalloproteinases which are under regulatory control of inhibitors of metalloproteinases. ECM has five main components: collagen, adhesive glycoproteins, basement membrane, elastic fibres, and proteoglycans (page 18). In wound healing, the deposition of proteoglycans precedes collagen laying.

The strength of wound also depends upon certain factors such as the site of injury, depth of incision and area of wound. After removal of stitches on around 7th day, the wound strength is approximately 10% which reaches 80% in about 3 months.

FACTORS INFLUENCING TISSUE REPAIR

While several local and general factors delay wound healing, certain therapeutic influences stimulate healing.

A. LOCAL FACTORS

[illegible] factors acting locally delay wound healing.
[illegible] important factor [illegible] delays [illegible] healing.
2. [illegible] blood supply to wound [illegible] slow healing, e.g. [illegible] the face heal quickly due to rich blood supply while injury [illegible] with varicose ulcers having poor blood supply heals slowly.
3. *Foreign bodies* including sutures interfere with healing and cause intense inflammatory reaction and infection.
4. *Movement* delays wound healing.
5. Exposure to *ionising radiation* delays granulation tissue formation.
6. Exposure to *ultraviolet light* facilitates healing.
7. *Type, size and location of injury* determine whether healing takes place by resolution or organisation.

B. GENERAL FACTORS

A few general and systemic factors influence healing:
1. *Age:* Wound healing is rapid in young and somewhat slow in aged and debilitated people due to poor blood supply to the injured area in the latter.
2. *Nutrition:* Deficiency of dietary constituents like protein, vitamin C (scurvy), vitamin A and zinc delays the wound healing.
3. *Systemic infection* delays wound healing.
4. *Administration of glucocorticoids and NSAIDs* delay wound healing.
5. *Uncontrolled diabetics* are more prone to develop infections and hence delayed wound healing.
6. *Haematologic abnormalities* like defect of neutrophil functions (chemotaxis and phagocytosis), neutropenia and bleeding disorders slow the process of wound healing.
7. *Colder temperature* delays healing.

C. THERAPEUTIC INFLUENCES

If there is delayed healing, appropriate therapy may be employed which stimulates healing by more efficient response but does not change the natural process of healing. Therapy works by influencing the chemical environment of the repair tissue to bring healing [illegible] delayed healing. Four basic principles of wound care are:
[illegible] removal of necrotic debris
[illegible] moist wound healing environment by proper dressing
[illegible] protect the wound from further injury, and
[illegible] provide nutritional substances essential for wound healing (extra [illegible], calories and nutrients).

Besides, all local and general factors listed above which may delay healing should be attended and corrected, wherever possible.

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KEY POINTS Tissue Repair: Regeneration and Repair

- [illegible]
- [illegible] cells: stable cells continue to divide [illegible] (e.g. epidermis, mucosa); stable cells decrease [illegible] their ability to proliferate (e.g. liver, kidney), while permanent cells cease to regenerate around the time of birth (e.g. neurons, myocardium).
- Repair is healing by formation of granulation tissue [illegible] inflammatory reaction by the body [illegible] clearance by proteolytic enzymes and [illegible] and proliferation of fibroblasts.
- The [illegible] strengthened by proliferation of fibroblasts and [illegible] which get structural support from the extracellular matrix (ECM).
- ECM [illegible] by collagen, adhesive glycoproteins, [illegible] membrane, elastic tissue and proteoglycans.
- Various local and general factors may delay wound healing, some of which need to be attended for better wound healing.

SELECTED EXAMPLES OF TISSUE REPAIR

[illegible] of general aspects of regeneration and repair [illegible] specific examples of tissue repair [illegible] under two headings:

Healing of skin wounds which represents the classic example of combination of regeneration and repair.

Healing in specialised tissues. Here, examples of healing in bone and a few [illegible] are described.

HEALING OF SKIN WOUNDS

Wound healing can be accomplished in one of the following two ways:

- Healing by first intention *(primary union)*
- Healing by second intention *(secondary union)*

HEALING BY FIRST INTENTION (PRIMARY UNION)

[illegible] Fig.

1. [illegible]

2. **Acute inflammatory response.** This occurs within 24 hours [illegible] from the margins of incision. By [illegible] macrophages.

3. **Epithelial changes.** [illegible] from both [illegible] differentiated into superficial and deeper layers.

4. **Organisation.** [illegible] epithelialised surface is formed.

5. **Suture tracks.** Each suture track is a separate wound and incites the same phenomena as in healing of the primary wound, i.e. filling the space with haemorrhage, some inflammatory cell reaction, epithelial cell proliferation along the suture track from both margins, fibroblastic proliferation and formation of young collagen. When sutures are removed around 7th day, much of epithelialised suture track is avulsed and the remaining epithelial tissue in the track is absorbed. However, sometimes the suture track gets infected *(stitch abscess)*, or the epithelial cells may persist in the track *(implantation or epidermal cysts)*.

Thus, the scar formed in a sutured wound is neat due to close apposition of the margins of wound; the use of adhesive tapes or metal clips avoids removal of stitches and its complications.

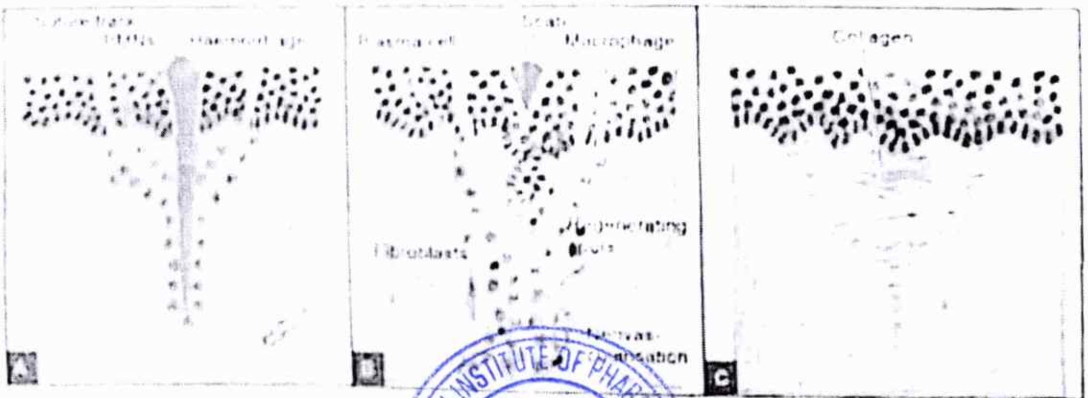

Figure 4.46 Primary union of skin wounds. A, [illegible] as well as suture track on either side are filled with blood clot and there is inflammatory response from the margins. B, [illegible] cells migrate along the incised margin on either side as well as around the suture track. Formation of granulation tissue also begins [illegible]. Removal of suture at around 7th day results in scar tissue at the sites of incision and suture track. (PMNs: polymorphonuclear neutrophils)

TRANSPLANTATION

Transplantation is a process of Taking an organ (OR) living tissue

AND

Implanting this organ (or) living tissue in another part of the Body

(OR).

Implanting this organ (or) living tissue in Another body.

Classification:

Transplantation is classified into:

1. Autografts = Grafts in which the Donor and recipient is the same individual
2. Isografts = Grafts between the Donor & recipent of the same genotype.
3. Allografts = Grafts between the Donor & recipent of same species But a different genotype
4. Xenografts = Grafts between the Donor & recipent of different species

For a successful transplantation without rejection

"Donor HLA should match recipient HLA"

Greater the genetic variability between

Donor and recipient HLA

The

stronger will be the rejection reaction

&

The

rapid will be the rejection reaction.

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MECHANISM OF GRAFT REJECTION

Rejection of allografts involves

(i) Cell mediated Immunity.

(ii) Humoral Immunity

(i) CELL MEDIATED IMMUNE RESPONSE:

Cell mediated immune response is Mainly responsible for graft rejection

Cell mediated immune response is mediated by T cells.

MECHANISM:

The T cells of the recipient upon comming in Contact with the HLA's of the Donor organ

⇓

The T cells are sensitised due to Mismatch between Donor & recipient HLA

⇓ NOW

The sensitised T cells undergoes clonal proliferation to give Cytotoxic T cells, T-Helper cells, etc

⇓

The graft is attacked & distroyed.

2. HUMORAL IMMUNG RESPONSE:

In some cases of rejection reactions humoral immuno response will also play a role along with CMI.

"Preformed Circulating Antiantibodies"

Due to

Prior sensitization of the recipient

Before

Transplantation.

Eg: Blood transfusions.

CLASSIFICATION OF REJECTION REACTIONS

1. Hyperacute rejection 2-Acute rejection 3 Chronic rejection

① Hyperacute rejection:

Rejection starts within "Minutes to hours"
after
placing the transplant.

Destruction of the graft occurs.

Hyperacute rejection reactions are mediated by
Preformed Antibodies against the donor antigen

② Acute rejection:

Rejection starts within few days to few months
after
Implantation / transplantation.

Acute rejection reactions are mediated
by CMI (Main) HI (Additional).

Destruction of regions of graft occurs

③ Chronic rejection

Chronic rejection occurs as a result of
repeated attacks of acute rejection.
(or)
Chronic rejection is seen after a prolonged

Months to years

The underlying cause of chronic rejection is Immunological (or) Ischaemia.

Progressive deterioration in the functioning of transplanted Organ is seen in chronic patients with rejection.

HYPERSENSITIVITY (OR) ALLERGY

⇓

"Exaggerated immune response to an antigen."

Depending on the speed of hypersensitivity · Hypersensitivity reactions are broadly of two types:

(duration)

① IMMEDIATE TYPE:

"After the administration of Antigen

With in

Seconds to minutes

Hypersensitivity reaction will occur."

Immediate type of hypersensitivity reactions are (mainly) Mediated by "Humoral Immunity".

Immediate type of Hypersensitivity are further classified into:

reactions.

TYPE I

TYPE II

TYPE III.

Hypersensitivity reactions.

② DELAYED TYPE:

After the administration of Antigen within

24 – 48 hours ⇒ (So the name delayed.)

Hypersensitivity reactions will occur.

- Delayed hypersensitivity reactions are mainly mediated by CMI.

Ex: TYPE – IV hypersensitivity reactions.

TYPE I HYPERSENSITIVITY REACTION.

(OR)

ANAPHYLATIC REACTION

"Rapidly developing exaggerated immune response to an antigen to which the person is previously sensitised."

"Anaphylatic reaction is Mediated by IgE antibodies"

Mechanism:

"IgE antibodies sensitise Mastcells of tissues & Basophils of Blood"

↓ leading to

release of Anaphylatic mediators [Histamines, 5-HT, VIP, LTB_4, LTD_4, TXA_2, PGD_2, E_2, PAF]; Chemotactic factors of anaphylaxis for Neutrophils, Eosinophils].

Effects of anaphylatic mediators:

Increased Vascular permeability.

Smooth muscle contraction

Initially Vasoconstriction f/b Vasodilation.

Shock

Increased gastric secretion, Nasal secretion

Lacrimation.

Anaphylaxis — Systemic anaphylaxis
Local anaphylaxis.

Systemic anaphylaxis:

Clinical manifestations: Itching
Rashes
Contraction of respiratory bronchioles.
Diarrhoea.
pulmonary edema.
Shock
Death.

Ex:
Stings by honeybee
Sting by Wasp
Administration of drugs mainly penicillin.

Local anaphylaxis (atopic reaction).

⇒ Commonly seen in 10% population.

⇒ 50% of local anaphylaxis reactions are due to genetic predisposition.

Ex: "Allergic rhinitis occurs due to pollen sensitisation of Conjunctiva and Nasal passages"

(ii) ~~Allergen~~ Bronchial asthma. Occurs due to sensitisation of Bronchi by inhaled allergens.

(iii) ~~Food~~ Food allergy occurs due to ingested Allergens like ~~Fish~~, Milk, Tamato.

TYPE-II HYPERSENSITIVITY REACTIONS

(OR)

CYTOTOXIC REACTION

An immunological reaction in which:

a Noncytotropic antibody

Combines with a

Specific antigen on the cell surface.

To

Form a ~~Antigen~~ Complex.

This complex will activate the complement system leading to cell lysis (or) cell damage.

3 types of Mechanisms are involved in Cytotoxic reactions:

1. cytotoxic antibodies to blood ~~cells~~ cells.
2. Cytotoxic antibodies to tissue Components.
3. Antibody dependent cell mediated Cytotoxicity

cytotoxic antibodies to blood cells:

This mechanism involves

Antibody (IgG or IgM) combines with antigens present on the surface of blood cells [RBC, WBC, platelets]

⇓ So

An immune complex is formed

⇓ This leads to

activation of the complement system.

⇓

Direct lysis of blood cells will occur

Examples:

1. Autoimmune haemolytic Anaemia:

Autoantibodies combines with antigens present on the surface of RBC.

⇓ So

Ultimately lysis of RBC's occurs.

2. Transfusion reactions due to Mismatched blood transfusion.

3. Haemolytic disease of Newborn (OR) Erythroblastosis foetalis:

Fetal RBC are destroyed by the Isoantibodies of the Mother by crossing the placenta.

4 Idiopathic thrombocytopenic Purpura (ITP):

Autoantibodies Combine with the surface antigens of the platelets

⇓

Ultimately the platelets are lysed or destroyed.

5. Drug induced Cytotoxic antibodies:

Certain:

Drugs or their metabolites act as ~~Hapten~~ Haptens

These:

Drugs or their metabolites binds to protein present on the (Blood cell) surface to form into an antigen.

⇓

Antibodies Combine with these antigens

⇓

Ultimately leading to lysis of these Blood cells:

2. Cytotoxic antibodies to tissue Components:

In this mechanism

Autoantibodies Combines with certain

Certain Components of tissue cells.

Causing

Certain diseases / tissue injury

Examples:

(i) Myasthenia gravis:

Autoantibodies to Acetylcholine receptors of skeletal muscle are formed.

⇓ Causing

Damage to skeletal muscle cell

⇓ leading to

Muscle weakness.

(ii) Male sterility:

Antisperm antibodies are formed.

This antisperm antibodies Combines with the sperm cell.

⇓ Causing: Injury to sperm cell + Impaired motility,

3. Antibody dependent cell mediated Cytotoxicity: (ADCC)

Cytotoxicity by this Mechanism is Mediated by

Monocytes, Neutrophils, Eosinophils, NK cells.

Antibodies involved are IgG.

"Antibody coated target cell" is

lysed by the above WBC "through Fc receptors present on leucocytes.

Examples: Tumour cells.

Pathogens.

TYPE III HYPERSENSITIVITY REACTION

(OR)

IMMUNE COMPLEX REACTION

Type III hypersensitivity reaction involves a

"Direct Combination of Antigen with Antibody"

⇓

This Immune Complex (Ag-Ab complex) leads to activation of Complement system

⇓

Causing Cell injury (or) Tissue damage.

Immune Complex mediated tissue injury is caused by two types of Antigens:

① Exogenous Antigens = Pathogens [Bacteria, Fungi, Virus, Protozoa etc]

Certain drugs & its metabolites

Certain chemicals.

② Endogenous antigens =

Blood components [Ab's, Tumour antigens]

Antigens on Cells & tissues: [ex: Nuclear antigens in SLE]

Depending upon the location of Antigens:

Type III hypersensitivity reactions are of 2 types:

1. Local Type III hypersensitivity reaction.
2. Systemic Type III hypersensitivity reaction.

① Local Type III Hypersensitivity reaction:

Ex: Injection of antitetanus serum.

Farmer's lung = allergic alveolitis = In response to Bacterial antigen from Mouldy hay.

2. Systemic Type III hypersensitivity reaction (OR) Circulating immune Complex ~~Reaction~~ Disease (OR) Serum sickness.

After the antigen is introduced into circulation. It initiates formation of Antibodies

⇓

An (Ag–Ab Complex) (or) Immune Complex is formed

⇓

The circulating immune Complex is diposited at different tissue sites.

~~Mainly~~ The circulating immune Complexes are mainly diposited at Basement membrane exposed to circulating ~~blood~~.

Ex: Basement membrane of glomeruli

Synovial membrane of joints.

⇓

Following deposition of Immune complexes in the tissues

Activation of complement system occurs.

Causing

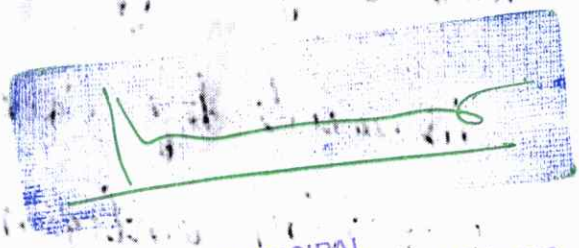
Tissue damage

Ex: Various forms of glomerulonephritis

Rheumatoid arthritis.

Scleroderma.

Arthritis occuring transiently during Infections



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TYPE IV HYPERSENSITIVITY REACTION

(OR)

CELL MEDIATED HYPERSENSITIVITY REACTION

Type IV Hypersensitivity reaction is Mediated by

"T cells which are specifically sensitised to a particular Antigen"

Type IV Hypersensitivity reaction
- Classical delayed hypersensitivity reaction.
- T cells mediated Cytotoxicity.

Classical delayed hypersensitivity reaction:

Mediated by CD4 T cells specifically sensitised to particular antigen

⇓

When the sensitised CD4 T cells comes in Contact with the particular antigen

⇓

CD4 T cells possess receptors on cell membrane to bind with the antigen

⇓

Slowly developing inflammation

⇓

Cell damage (or) Tissue damage

Ex: Tuberculin skin test (OR) Mantoux test

Intradermal injection of "Tuberculoprotein"

(i) UNsensitised person = No response -Ve

(ii) Sensitised person — Previous Infection
                        — BCG Vaccination

Develops

Delayed hypersensitivity reaction
(or)
Delayed inflammatory reaction
(Skin rash, Edema).

In 48 hours

This skin rash, edema will disappear slowly

T cell-mediated Cytotoxicity:

Mediated by CD8-T cells specifically sensitised to a particular antigen.

CD8-T cells (or) Cytotoxic T cells are formed in response to Antigens like:

- Virus infected cells.
- Tumour cells
- Transplantable transplanted cells
- Isocompatible transplanted tissues.

Toxicity in gonococcal infections is largely attributable to the endotoxic effects of LOS.

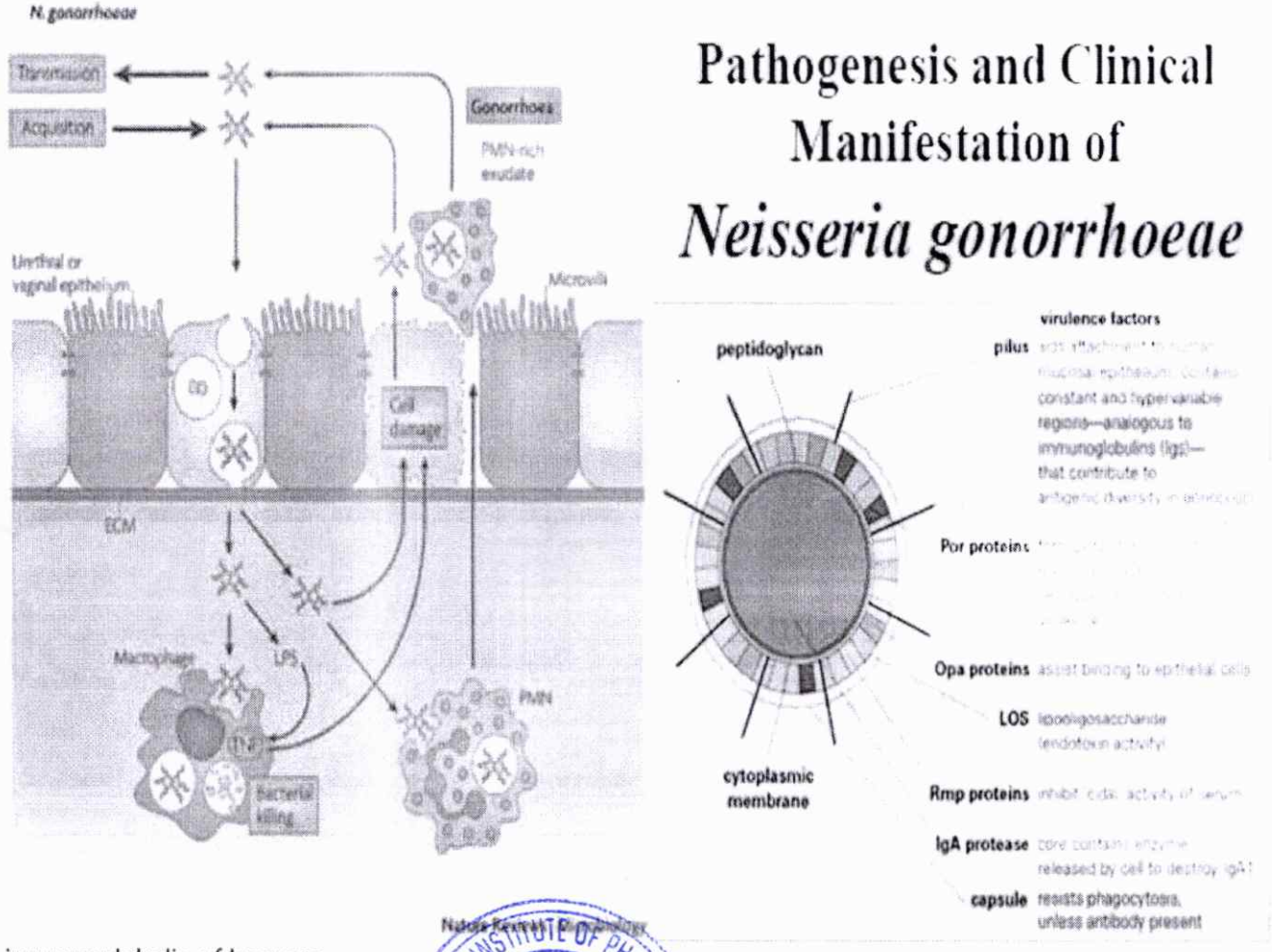
Capsule, lipooligosaccharide (endotoxin), and outer cell membrane proteins I-III are important in antigenic variation and for eliciting an inflammatory response.

E. Other proteins

Lip (H8) is a surface exposed protein that is heat modifiable like Opa.

The Fbp (ferric-binding protein), similar in molecular weight to Por, is expressed when the available iron supply is limited, such as in human infection.

Gonococci elaborate an IgA1 protease that splits and inactivates IgA1, a major mucosal



immunoglobulin of humans.

Pathogenesis and Clinical Manifestation of Neisseria gonorrhoeae

Pathogenesis of Neisseria gonorrhoeae

Gonorrhoeal infection is generally limited to superficial mucosal surfaces lined with columnar epithelium.

Pili and Opa proteins facilitate adhesion of the gonococcus to epithelial cells of the urethra, rectum, cervix, pharynx, and conjunctiva, thereby making colonization possible.

Pili, PorB, and Opa proteins mediate gonococci to attach to mucosal cells, penetrate into the cells and multiply, and then pass through the cells into the subepithelial space where infection is established.

The gonococcal LOS stimulates release of the proinflammatory cytokine tumor necrosis factor-α (TNF-α), which causes most of the symptoms associated with gonococcal disease.

Antibodies to LOS can activate complement, releasing complement component C5a, which has a chemotactic effect on neutrophils; however, IgG and secretory IgA1 antibodies directed against Rmp protein can block this bactericidal antibody response.

The gonococcus requires Iron for growth and survival in vivo.

The pathogen acquires this necessary nutrient by expression of specific transport systems that remove and internalize the iron from human iron binding proteins including transferrin, lactoferrin and hemoglobin.

Clinical manifestations of Neisseria gonorrhoeae

Gonococci most often colonize the mucous membrane of the genito -urinary tract or rectum. There, the organisms may cause a localized infection with the production of pus or may lead to tissue invasion, chronic inflammation, and fibrosis. A higher proportion of females than males are generally asymptomatic, and these individuals act as the reservoir for maintaining and transmitting gonococcal infections.

A. Genitourinary tract Infections

Acute infections in males

In males, yellow, purulent urethral discharge and painful urination

In females, infection occurs in the endocervix and extends to the urethra and vagina. A greenish-yellow cervical discharge is most common, often accompanied by intermenstrual bleeding.

Genital infections include acute purulent urethritis, prostatitis, and epididymitis in males and acute cervicitis in females.

The disease may progress to the uterus, causing salpingitis (inflammation of the fallopian tubes), pelvic inflammatory disease (PID), and fibrosis.

Pelvic inflammatory disease (PID) may cause sterility, ectopic pregnancy or perihepatitis also referred to as Fitz-Hugh–Curtis syndrome.

B. Rectal Infections

Prevalent in men who have sex with men, rectal infections are characterized by constipation, painful defecation, and purulent discharge.

C. Pharyngitis

Pharyngitis is contracted by oral-genital contact.

Infected individuals may show a purulent pharyngeal exudates.

D. Ophthalmia neonatrum

This infection of the conjunctival sac is acquired by newborns during passage through the birth canals of infected mothers.

If untreated, acute conjunctivitis may lead to blindness.

F. Disseminated infection

Most strains of gonococci have a limited ability to multiply in the bloodstream.

Therefore, bacteremia with gonococci is rare.

However, some strains of gonorrhoeae do invade the bloodstream and may result in a disseminated infection in which the organism can cause fever; a painful, purulent arthritis; and small, single, scattered pustules on the skin, whose base becomes erythematous due to dilation or congestion of capillaries.

Pathogenesis of Starvation:

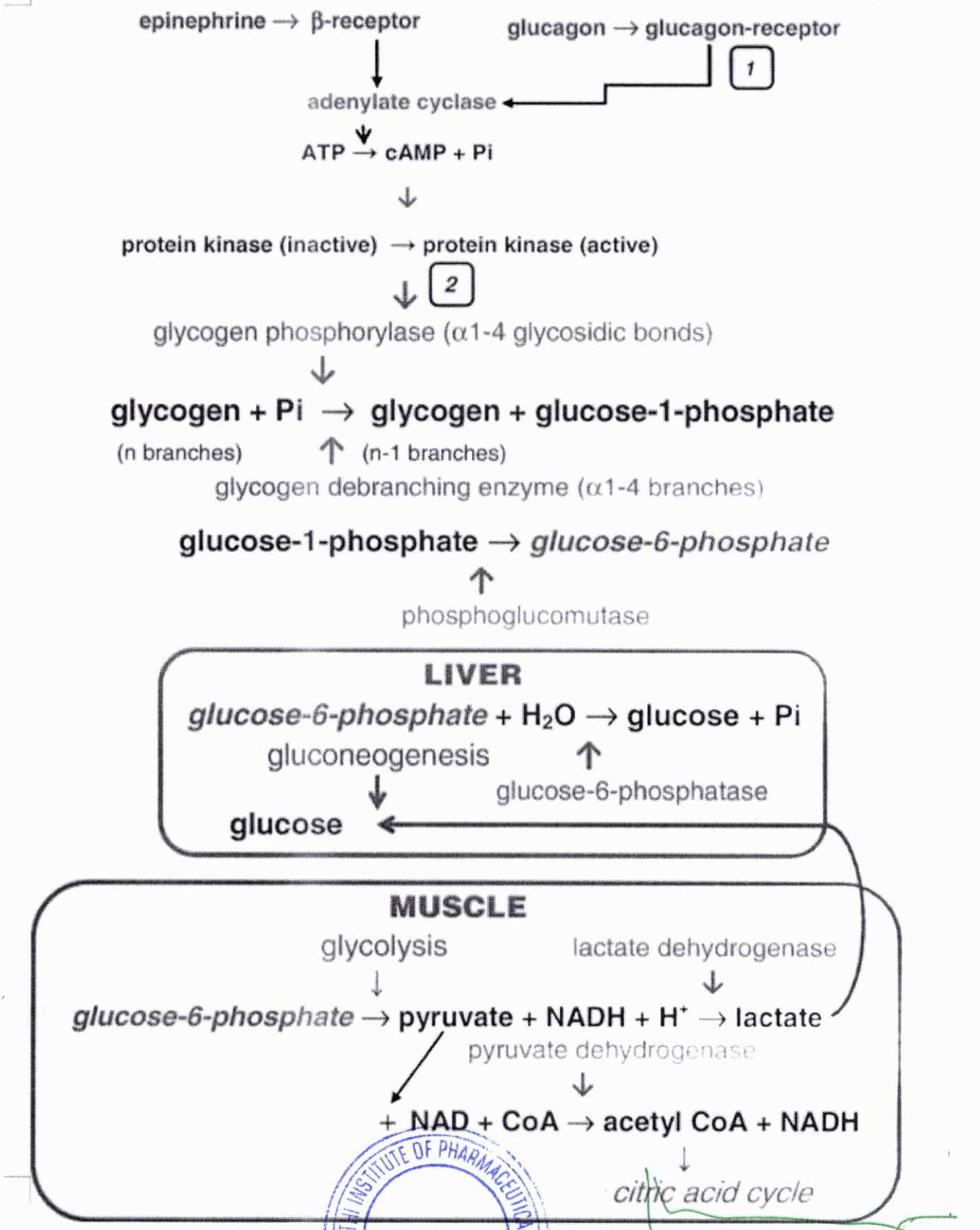
Starvation is a condition of stoppage of food intake leading to overall deprivation of nutrients.

When food intake ceases, the body enters the starvation response. Initially, the body's glycogen stores are used up in about 24 hours. The level of insulin in circulation is low and the level of glucagon is very high. The main means of energy production
is lipolysis. Gluconeogenesis converts glycerol into glucose and the Cori cycle converts lactate into usable glucose. Two systems of energy enter the gluconeogenesis: proteolysis provides alanine and lactate produced from pyruvate, while acetyl CoA produces dissolved nutrients (Ketone bodies), which can be detected in urine and are used by the brain as a source of energy. In terms of insulin resistance, starvation conditions make more glucose available to the brain.

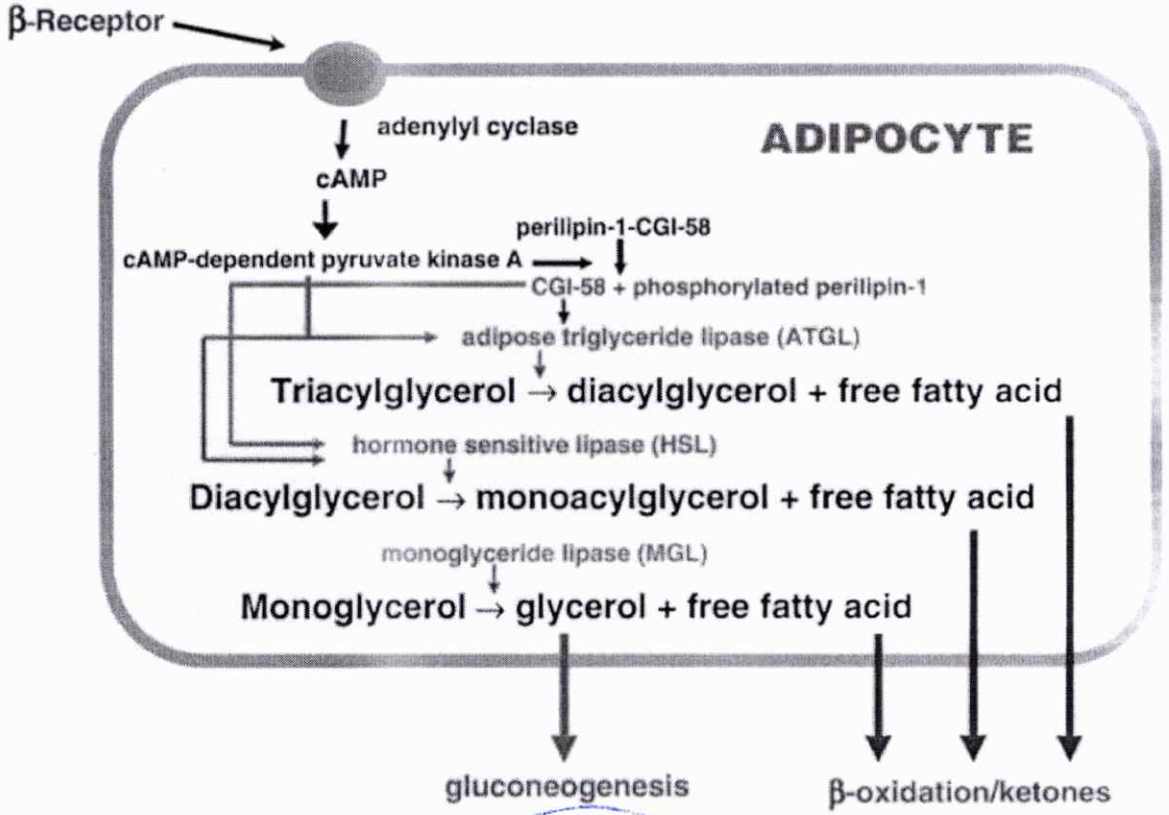
Common Causes

- Anorexia nervosa
- Bulimia nervosa
- Clinical Depression
- Coma
- Crash dieting
- Digestive disease
- Famine
- Fasting
- Malnutrition
- Overpopulation
- Poverty

- PRINCIPAL
Avanthi's Institute of Pharmaceutical Sciences
Gunthapally (V), Hayath Nagar (M),
Ranga Reddy Dist.



Effect of starvation on Adipocytes:



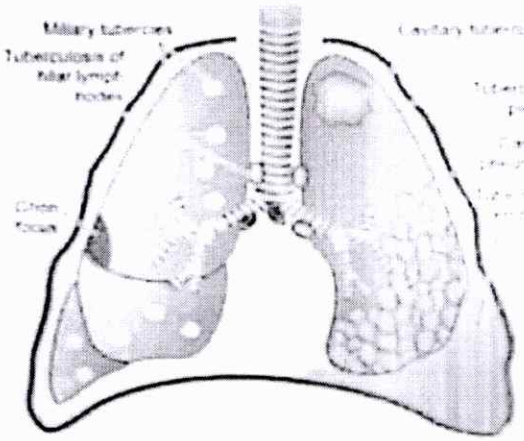


Figure 4.33 Spectrum of lesions in the lungs and pleura in various types of pulmonary tuberculosis

LEPROSY

Leprosy or Hansen disease (after discovery of the causative organism by Hansen in 1874) was first described in ancient Indian text going back to 6th Century BC as a chronic non-fatal infectious disease. It affects mainly the cooler parts of the body such as the skin, mouth, respiratory tract, eyes, peripheral nerves superficial lymph nodes and testis. In leprosy, the earliest and most severe involvement is of the skin and nerves. Histologic lesions are caused by granulomas from endothelial reformation or by bacilli ingested from blood by reticuloendothelial systems; other organs such as the liver, spleen, bone marrow and regional lymph nodes are also involved. Advanced cases may develop secondary amyloidosis and renal disease, both of which are of immunologic origin.

CAUSATIVE ORGANISM

The disease is caused by *Mycobacterium leprae* which closely resembles *Mycobacterium tuberculosis* but the organism is less acid-fast and has characteristic *neurotropism*. The organisms in tissues appear as compact rounded masses (*globi*) or are arranged in parallel fashion like *cigarettes in a pack*.

M. leprae can be demonstrated in tissue sections, in split skin smears by splitting the skin scrapings from cut edges of dermis and in nasal smears by the following techniques:

1. *Acid-fast (Ziehl Neelsen or ZN) staining*. The staining procedure is similar as for demonstration of *M. tuberculosis* (page 90) but can be decolourised by lower concentration (5%) of sulphuric acid (less acid fast).
2. *Fite-Faraco staining*. This procedure is a modification of ZN procedure and is considered better for more adequate staining of tissue sections (**Fig. 4.34**).
3. *Gomori methenamine silver (GMS) staining* can also be employed.
4. *Molecular methods* e.g. PCR.
5. *IgM antibodies to PGL-1 antigen* is seen in 95% cases of lepromatous leprosy but only in 50% cases of tuberculoid leprosy.

The slit smear technique gives a reasonably quantitative measure of *M. leprae* when stained with ZN method and examined using 100× oil objective for determining the density of bacteria in the lesion (*bacterial index, BI*). BI is scored from 1+ to 6+ (range from 1 to 10 bacilli per 100 fields to > 1000 per field) as *multibacillary leprosy*, while BI of 0+ is termed *paucibacillary leprosy*. This forms the basis of WHO classification of leprosy practiced by field workers.

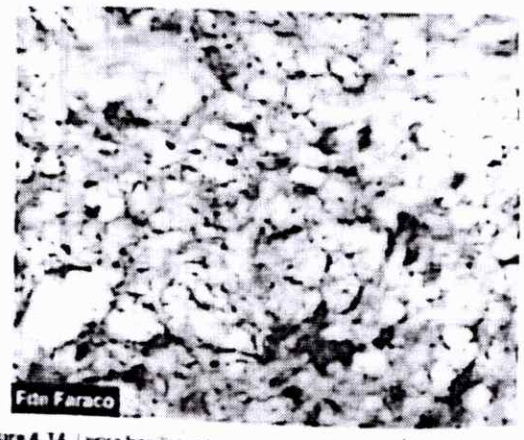


Figure 4.34 Lepra bacilli in LL are seen as globi and cigarettes in a pack appearance inside the foam macrophages (Fite-Faraco stain)

the immune reaction of host lymphocytes and macrophages. Another unique feature of leprosy is [illegible] in peripheral nerves which is due to binding of trisaccharide of *M. leprae* to basal lamina of Schwann cells [illegible].

2. Genotype of the host [illegible] known by MHC [illegible] leprosy [illegible] the host [illegible] in different individuals is variable.

3. T cell response There is variation in T cell response in two main forms of leprosy.

i) Unlike tubercle bacilli, there is not only activation of CD4+ T cells but also of CD8+ T cells.

ii) CD4+ [illegible] not only helper [illegible]

iii) The [illegible] CD4+ T cells [illegible] (helper cells) T_H1 [illegible] in response [illegible] lepra bacilli and macrophages.

iv) In [illegible] response is [illegible] by CD4+ T cells, while [illegible] although there is excess of CD8+ T cells (suppressor T cells), the macrophages and suppressor T cells fail to de[illegible] bacilli [illegible].

4. Humoral response Though the patients of lepromatous leprosy [illegible] immune response such as high levels of immunoglobulins (IgG, IgA, IgM) and antibodies to mycobacterial antigens, these antibodies do not have any protective role against lepra bacilli.

Based on the above unique immunologic features in leprosy, lesions in leprosy are classified into 5 distinct clinicopathologic types and three forms of reactional leprosy (described below). An intradermal immunologic test, lepromin test, has been described which is not a diagnostic test but is used for classifying leprosy on the basis of immune response.

LEPROMIN TEST Intradermal injection of **lepromin**, an antigenic extract of *M. leprae*, reveals delayed hypersensitivity reaction in patients of tuberculoid leprosy.

1) An early positive reaction appearing as an indurated area in 24-48 hours is called *Fernandez reaction*.

2) A delayed granulomatous lesion appearing after 3-4 weeks is called *Mitsuda reaction*.

This test indicates that cell-mediated immunity patients of tuberculoid leprosy show good immune response.

On the other hand, patients of lepromatous leprosy are negative by the lepromin test. It indicates that cell-mediated immunity is greatly suppressed in lepromatous leprosy.

[illegible] of type of hypersensitivity [illegible] The [illegible] of tuberculoid type [illegible] cells [illegible] T suppressor cells at the periphery [illegible] of lepromatous leprosy [illegible]

CLASSIFICATION

RIDLEY AND JOPLING'S CLASSIFICATION [illegible] two main forms of leprosy are distinguished as:

1. Lepromatous type representing *low resistance*; and
2. Tuberculoid type representing *high resistance*.

Salient differences between these two forms of leprosy are summarised in **Table 4.8**.

Since both these types of leprosy represent two opposite poles of host immune response, these are also called polar forms of leprosy. Cases not falling into either of the two poles are classified as borderline and indeterminate types.

Based on clinical, histologic and immunologic features, modified Ridley and Jopling classification has been described which divides leprosy into 5 groups as under:

TT: Tuberculoid Polar (*High resistance*)
BT: Borderline Tuberculoid
BB: Mid-Borderline (Dimorphic)
BL: Borderline Lepromatous
LL: Lepromatous Polar (*Low resistance*)

VARIANTS In addition, a few variant forms of leprosy which are not included in Ridley-Jopling classification have been described:

- *Indeterminate leprosy*: This is an initial non-specific stage of any type of leprosy.
- *Pure neural leprosy*: In these cases, skin lesions which are the cardinal feature of leprosy are absent but instead neurologic involvement is the main feature.
- *Histoid leprosy*: Described by Wade in 1963, this is a variant of LL in which the skin lesions resemble nodules of dermatofibroma and the lesions are highly positive for lepra bacilli.

REACTIONAL LEPROSY Based on shift in immune status of [illegible] in patients of leprosy on treatment, two types of reactional leprosy are distinguished: type I (reversal reaction) and type II (erythema nodosum leprosum).

Type I: Reversal reactions The polar forms of leprosy [illegible] undergo no change in clinical and histopathological picture. The borderline groups are unstable and may move across the spectrum in either direction with upgrading or downgrading of patient's immune state. Accordingly, there may be two types of borderline reaction:

TABLE 4.8 Differences between lepromatous and tuberculoid leprosy

	FEATURE	LEPROMATOUS LEPROSY	TUBERCULOID LEPROSY
1.	*Skin lesions*	Symmetrical, multiple, hypopigmented or erythematous or maculopapular or nodular lesions (leonine facies)	Asymmetrical, single or a few lesions, well-defined, hypopigmented and erythematous macular lesions
2.	*Nerve involvement*	Present but late, sensory disturbance is less severe	Present with distinct sensory disturbance
3.	*Histopathology*	Collection of foamy macrophages or lepra cells in the dermis separated from epidermis by a clear zone, lymphocytes absent [illegible] only	Hard tubercle similar to granulomatous lesion, eroding the basal layer of epidermis; no clear zone; lymphocytes plenty
4.	*Bacteriology*	Lepra cells highly positive for lepra bacilli seen as globi or 'cigarettes-in-pack' appearance (multibacillary type)	Lepra bacilli few, seen in destroyed nerves as granular or beaded forms (paucibacillary type)
5.	*Complications*	Type 2 reactional leprosy (ENL) may occur	Neurologic damage causing sensory loss and paralysis may occur
6.	*Immunity*	Suppressed (low resistance)	Good immune response (high resistance)
7.	*Lepromin test*	Negative	Positive
8.	*Prognosis*	Progressive disease, bad prognosis	Milder disease, better prognosis

❖ *Upgrading reaction* is characterised by increased cell-mediated immunity and occurs in patients of borderline lepromatous (BL) type on treatment who upgrade or shift towards tuberculoid type.

❖ *Downgrading reaction* is characterised by lowering of cellular immunity and is seen in borderline tuberculoid (BT) type who downgrade or shift towards lepromatous type.

Type II: Erythema nodosum leprosum (ENL) ENL occurs in lepromatous patients after treatment. It is characterised by tender cutaneous nodules, fever, iridocyclitis, synovitis and lymph node involvement.

HISTOPATHOLOGY OF LEPROSY

Usually skin biopsy from the margin of lesions is submitted for diagnosis and for classification of leprosy. The histopathologic diagnosis of multibacillary leprosy [BL, LL and BB] poses no problem, while the indeterminate leprosy and tuberculoid lesions are paucibacillary and their pathologic diagnosis is made together with clinical evidence.

In general, for histopathologic evaluation of skin biopsy in all suspected cases of leprosy, the following general *features* should be looked for:

i) Cell type of granuloma
ii) Nerve involvement
iii) Bacterial load
iv) Presence and absence of lymphocytes
v) Relation of granuloma with epidermis and adnexa.

The salient features in major types of leprosy are as follows:

1. Lepromatous leprosy. The following features characterise lepromatous polar leprosy (Fig. 4.35):

i) In the dermis, there is proliferation of macrophages with foamy change, particularly around the blood vessels, nerves and dermal appendages. The foamy macrophages are called *lepra cells* or *Virchow cells*.

ii) The lepra cells are heavily laden with acid-fast bacilli demonstrated with AFB staining. The AFB may be seen as compact globular masses *(globi)* or arranged in parallel fashion like *'cigarettes-in-pack'* (see Fig. 4.34).

iii) The dermal infiltrate of lepra cells characteristically does not encroach upon the basal layer of epidermis, which is separated from epidermis by a subepidermal uninvolved [illegible] zone.

iv) The epidermis overlying the lesion [illegible] may even be atrophic.

2. Tuberculoid leprosy. The [illegible] the following histologic features:

i) The dermal lesions show [illegible] tubercles composed of epithelioid cells [illegible] and peripheral mantle of lymphocytes.

ii) Lesions of tuberculoid leprosy [illegible] except when [illegible] cells and lymphocytes.

iii) The granulomas [illegible] epidermis, [illegible] there is [illegible].

iv) The *lepra cells* are [illegible].

3. Borderline leprosy. The [illegible] of the three forms of borderline leprosy are as [illegible]:

i) *Borderline tuberculoid (BT)* [illegible] epithelioid cells and plentiful lymphocytes. There is a narrow [illegible] subepidermal zone. Lepra bacilli are scanty and found [illegible].

ii) *Borderline lepromatous (BL)* [illegible] predominance of histiocytes, a few epithelioid cells and [illegible] lymphocytes. Numerous lepra bacilli [illegible].

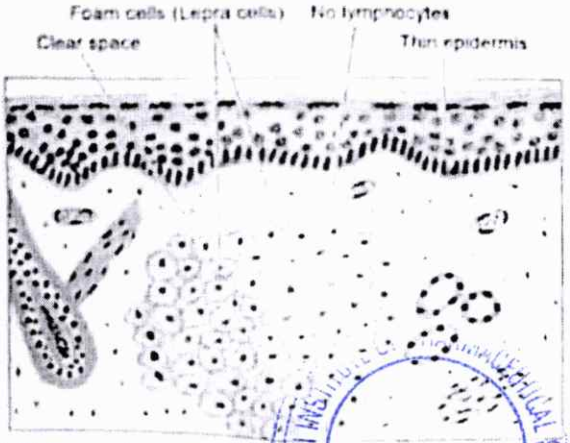
iii) *Mid-borderline (BB)* or dimorphic [illegible] sheets of epithelioid cells with no giant cells. Some lymphocytes are seen in the perineurium. Lepra bacilli are present, mostly in nerves.

4. Indeterminate leprosy. The histopathologic features are non-specific, so that the diagnosis of non-specific chronic dermatitis may be made. However, a few features help in suspecting leprosy as under:

i) Lymphocytic or mononuclear cell infiltrate [illegible], particularly around skin adnexal structures like hair follicles and sweat glands or around blood vessels.

ii) Nerve involvement, if present, is strongly supportive of diagnosis.

iii) Confirmation of diagnosis is made by finding of lepra bacilli.



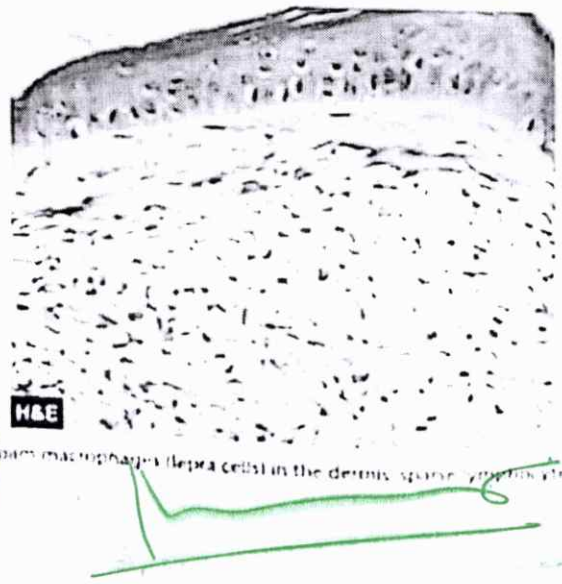


Figure 4.35 Lepromatous leprosy. (a) [illegible] collection of [illegible] foam macrophages (lepra cells) in the dermis, sparing [illegible] clear subepidermal zone.

Figure 4.36 Tuberculoid leprosy: Granuloma in the dermis with the basal layer of the epithelium. The granuloma is composed of epithelioid cells with sparse Langhans giant cells and lymphocytes

5. Pure neural leprosy. Histopathologic features described in skin lesion of various forms of leprosy may be seen in the nerve biopsy specimens. Pure neural leprosy may be AFB positive or AFB negative.

6. Histoid leprosy. Following features of histoid leprosy are seen in these lesions:

i) Whorls and fascicles of spindle cells in the upper dermis after a clear subepidermal space.

ii) On close scrutiny, these cells have foamy cytoplasm.

iii) The cytoplasm of these cells is laden with lepra bacilli.

7. Reactional leprosy. Two types of reactional leprosy show following features:

i) Type I reaction: Reversal reactions. These may be upgrading or downgrading type of reaction.

- *Upgrading reaction* shows an increase of lymphocytes, oedema of the lesions, necrosis in the centre and reduced BI.
- *Downgrading reaction* shows dispersal and spread of the granulomas and increased presence of lepra bacilli.

ii) Type II reaction: ENL. The lesions in ENL show infiltration by neutrophils and eosinophils and prominence of vasculitis. Inflammation often extends deep into the subcutaneous fat causing panniculitis. Bacillary load is increased. Secondary amyloidosis may follow repeated attacks of ENL in leprosy.

CLINICAL FEATURES

The two main forms of leprosy show distinctive clinical features.

1. Lepromatous Leprosy

i) The skin lesions in LL are generally symmetrical, multiple, slightly hypopigmented and erythematous macules, papules, nodules or diffuse infiltrates. The nodular lesions may coalesce to give *leonine facies* appearance.

ii) The lesions are hypoaesthetic or anaesthetic initially. Sensory disturbance is not as distinct as in TT.

2. Tuberculoid Leprosy

i) The skin lesions in TT occur as either single or as a few asymmetrical lesions which are hypopigmented and erythematous macules.

ii) There is a distinct sensory impairment.

Long-term cases of either type may develop secondary amyloidosis. Anti-leprosy vaccines have been developed but are undergoing human trials yet. Since the incubation period for leprosy is quite long, the efficacy of such vaccines will be known after a number of years.

KEY POINTS Leprosy

- Leprosy or Hansen disease is a chronic infectious disease that affects mainly the cooler parts of the body such as the skin, mouth, respiratory tract, eyes, peripheral nerves, superficial lymph nodes and testis.
- The disease is caused by *Mycobacterium leprae* which closely resemble *Mycobacterium tuberculosis* but is less acid fast and has characteristic neurotropism.
- The disease spreads by close contact for a long duration, often lasting for several years.
- Based on clinical, pathologic and immunologic features, leprosy is classified into polar tuberculoid (high resistance), polar lepromatous (low resistance), and borderline towards either type. A few variants are reactional (type I upgrading and downgrading, type II or ENL), histoid, and pure neural leprosy.
- Lepromatous type has foam cell granulomas (multibacillary on lepra stain) while tuberculoid type has epithelioid cell granulomas (paucibacillary on lepra stain).

SYPHILIS

Syphilis is a venereal (sexually-transmitted) disease caused by spirochaete *Treponema pallidum*, characterised by episodes of active disease interrupted by periods of latency. Other treponemal diseases are yaws, pinta and bejel. The word 'syphilis' is derived from the name of the mythological handsome boy, Syphilus, who was cursed by Greek god Apollo with the disease.

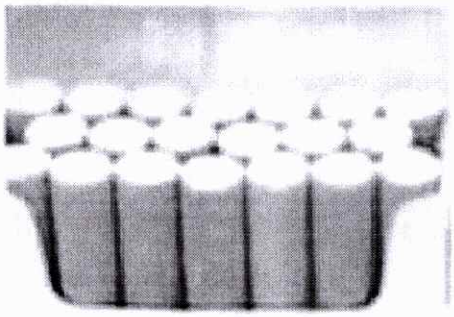
CAUSATIVE ORGANISM

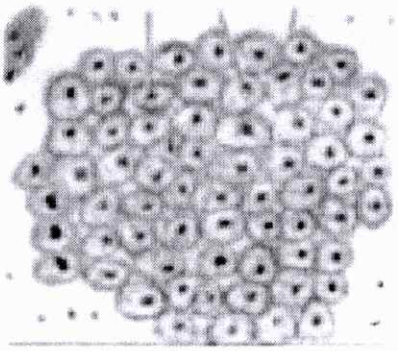
Treponema pallidum is a coiled spiral filament 10 µm long that moves actively in fresh preparations. The organism cannot be stained by the usual methods and can be demonstrated in the exudates and tissues by:

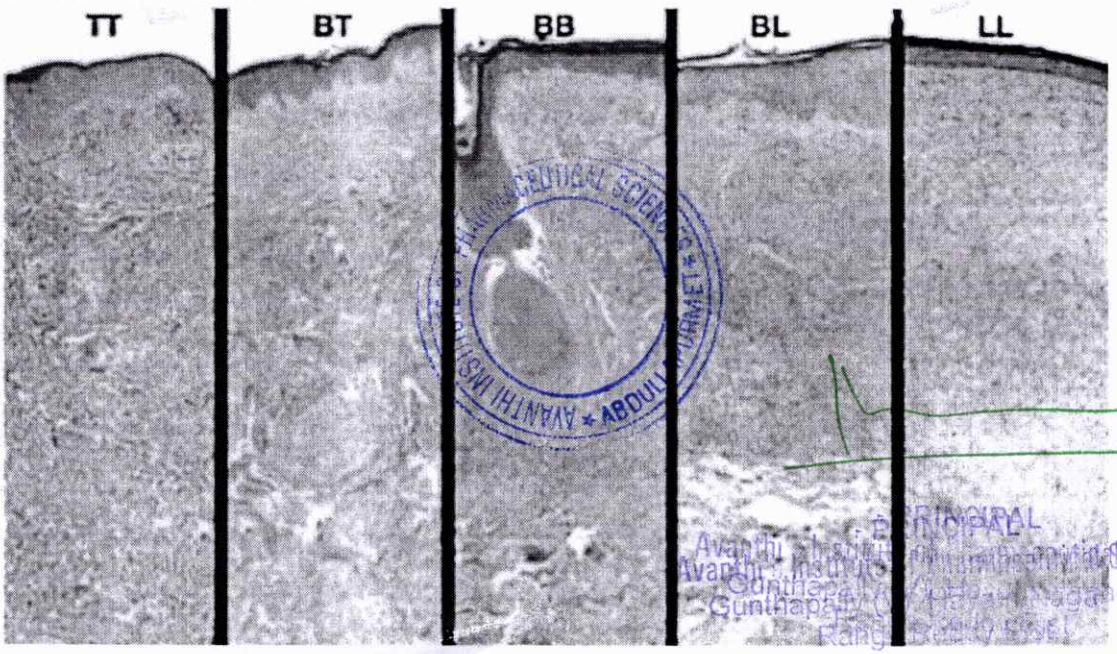
1. *Dark ground illumination* (DGI) in fresh preparation,
2. *Fluorescent antibody technique*,
3. *Silver impregnation techniques*, and
4. *Nucleic acid amplification* technique by PCR.

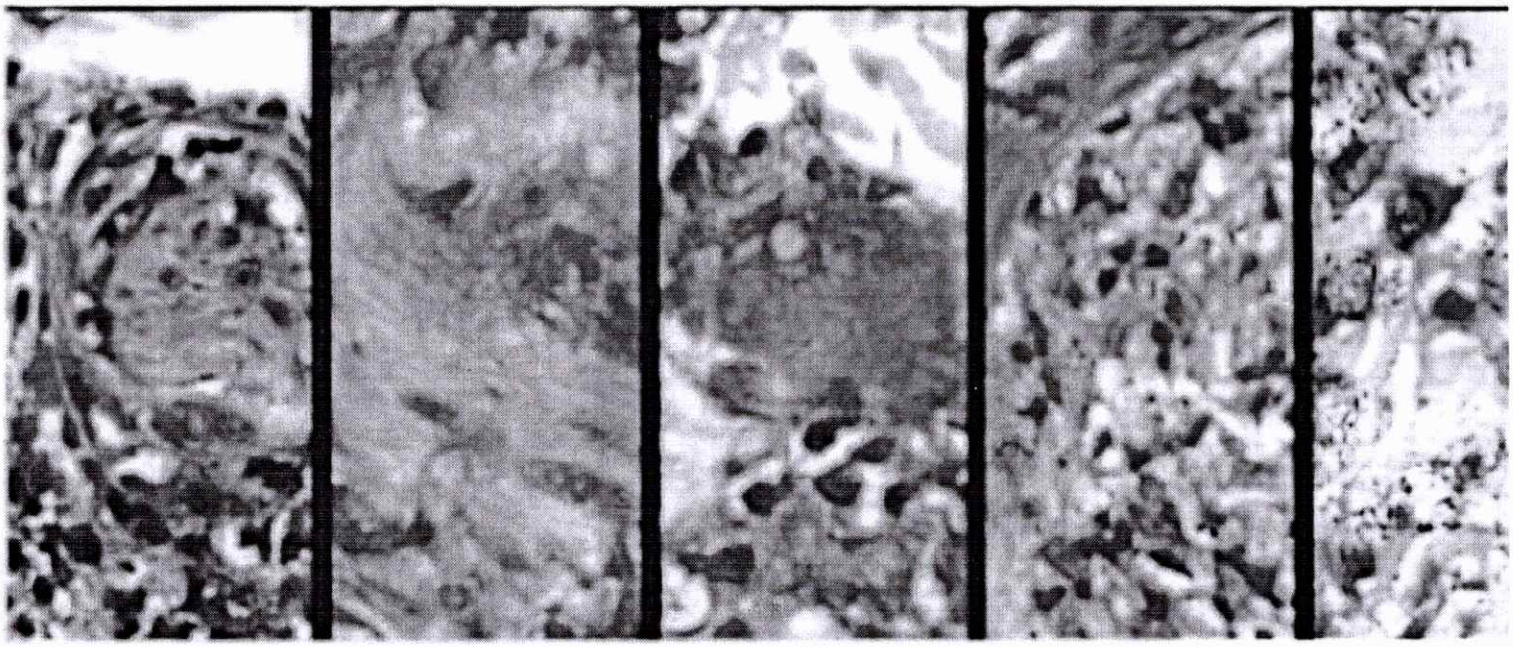
Hansen's	Tuberculoid Leprosy	Lepromatous Leprosy
	Paucibacillary Hansen Disease	Multibacillary Hansen Disease
	few bacteria → less infectious	Many bacteria → More infectious
	Strong T. cell immunity	weak T. cell immunity
	Activation + Granuloma formation	NO Activation + NO Granuloma formation
	Few Erythematous/Hypopigmented plaques	Many Erythematous/Hypopigmented plaques
	Peripheral nerve damage. Complete sensory loss	Peripheral nerve damage. Patchy sensory loss
	Gross NERVE ENLARGEMENT	NO Gross NERVE ENLARGEMENT
	Predominantly "Lymphocytes" & epithelioid cells	Predominantly foamy macrophages
Lepromin	Reactive to Lepromin skin test	NOT Reactive to Lepromin skin test
IG's levels	NORMAL	↑↑↑
Erythema Nodosum	Absent	Present
D_x	Skin Test (Granuloma formation) for	MICROSCOPY (Many bacteria)
T_x	Rifampin + Dapsone	Rifampin + Dapsone + Clofazimine

- **The lepra cells are heavily laden with acid-fast bacilli demonstrated with AFB staining. The AFB may be seen as compact globular *masses (globi) or arranged in parallel fashion like 'cigarettes-in-pack'*.**









Lepromin test

- **Integral lepromin**
- **Bacillary lepromin**
- **0.5 ml ID injection**
- ***Early reaction of Fernandez*: erythema & induration within 24 - 48 hrs, remains 3 – 5 days**
- ***Late reaction of Mitsuda*: 1 -2 weeks after inj., peak 4 weeks, nodule – ulecration – healing**

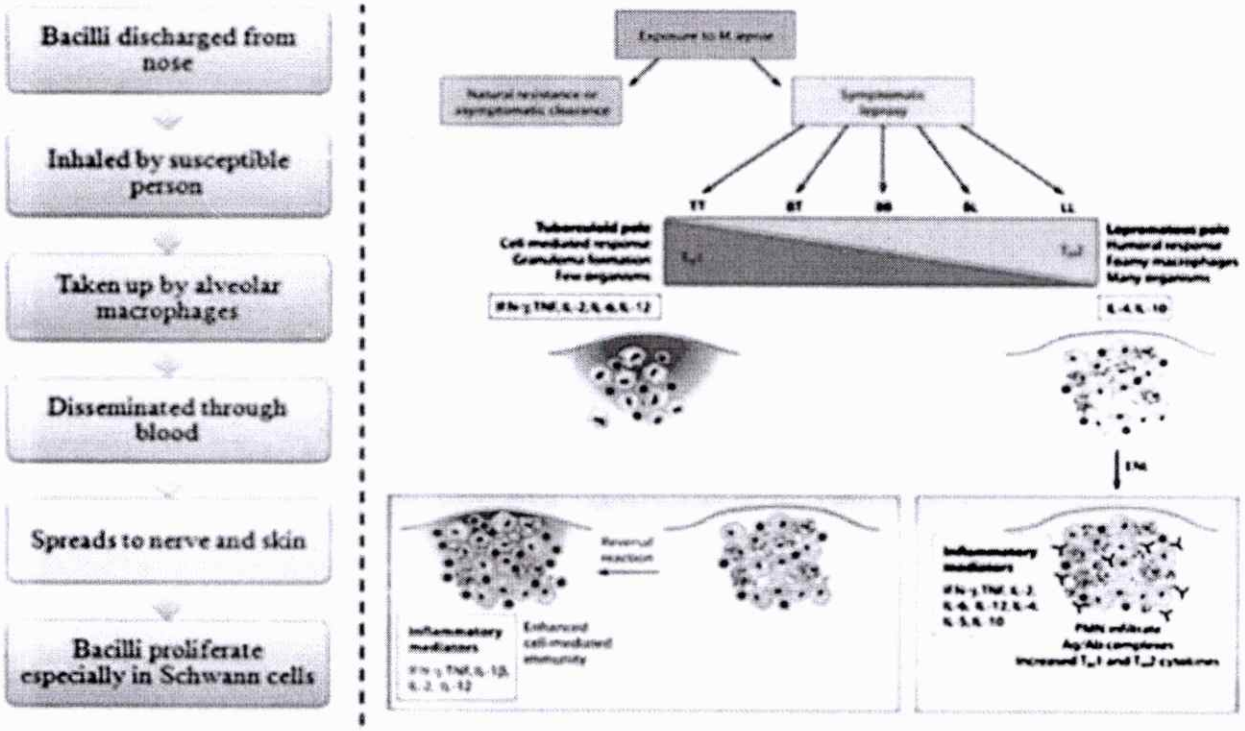


Table 18.7 Characteristics of the five points on the spectrum of leprosy

Characteristic	TT	BT	BB	BL	LL
Bacilli seen in skin	–	±	+	++	+++
Bacilli in nasal secretions	–	–		+	+++
Granuloma formation	+++	++	+	–	–
Reaction to lepromin	+++	+	±	–	–
Antibodies to *M. leprae*	±	±	+	++	+++
Main phagocytic cell	Mature epithelioid	Immature epithelioid	Immature epithelioid	Macrophage	Macrophage
In-vitro correlates of CMI	+++/++	+	+	–	–
Type 1 reactions	–	+	+	+	–
Type 2 reactions	–	–	±	++	

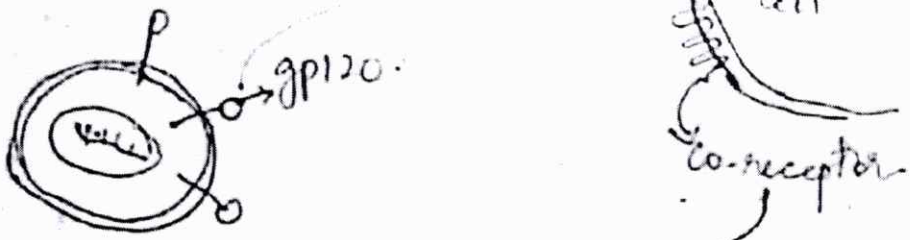
CMI, cell-mediated immunity. See text for other abbreviations.

HIV / AIDS — 1 - US & world wide * common
2 - Asia & Southern Africa

immunocompromised.

HIV — invades CD4+ cells — macrophages, T helper, dendritic

by binding @ 2 sites on host cell.



- CXCR-4 & CCR-5 are chemokine coreceptors for HIV entry into CD4+ cells

* used during later stages of HIV infection
* T_H cells

* used during early stages
* T cells, macroph, monocytes, dendritic cells.

* Mutations in co-receptors of HIV infecting becomes difficult.
a) Homozygous mutation — resistant to HIV
b) Heterozygous mutation — slower disease
= one normal allele & one mutated allele @ a gene locus

⇒ structure of HIV - diagram

its a ss-RNA retrovirus

gains entry into cell to inject its ssRNA into T_H cell

→ Enz. reverse transcriptase converts ssRNA into a complementary strand. Proviral just means it is going to join the T_H cell DNA.

⇒ Thus proviral DNA enters nucleus of T_H cell & joins into its DNA.

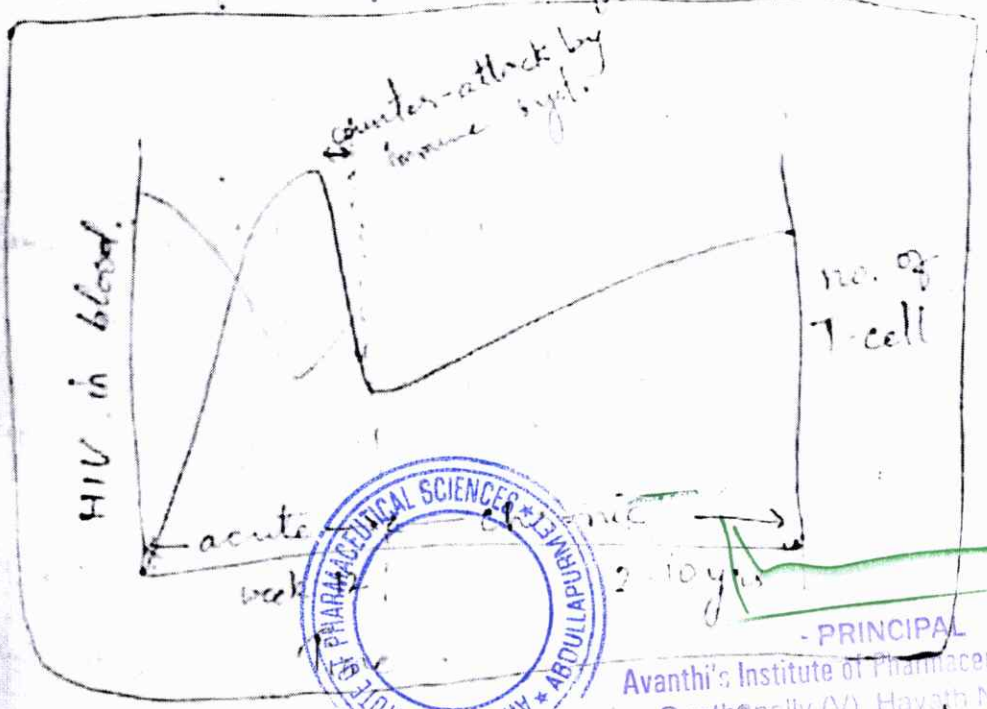
[Transcription by RT: RT (RNA dependent DNA polymerase enz. is a DNA polymerase enz. that transcribes ssRNA to DNA.

⇒ When T_H cells is activated due to any infection, it starts producing proteins & immune response & accordingly also translates & transcribes new HIV viruses which come out of cell enter to infect more body cells.

⇒ HIV characteristic feature is to make slight errors in replication. These are still called HIV but behave slightly different & invade diff. types of cells.

Acute infn :-

- R5 strain of HIV binds to CCR5 coreceptor of macroph, dendritic cells & TH cells of any epith./mucosal tissue then enters lymphnodes where it affects all these cells.
- Symptom start. - fever
- In response to infn immune syst. counterattacks to control replication thus amt. of virus in blood ↓ but detectable thru test within 12 weeks. After which pt enters chronic/clinically latent phase which lasts for 2-10 yrs. Chronic phase :-



- At the start of chronic phase, amt of virus in blood ↑ slowly & no of T cells ↓ at the rate

of 1-2 billion Tcells/day.
Through out chronic phase, Tcell count = >500cell/mm³
even then body fights infections but infections
can get severe.

- In Chronic phase, X4 strain of HIV develops which targets CXCR4 coreceptor. which is essentially on T-cells & stay in lymphoid tissues & destroy CD4-Tcells.

- when Tcell count drops <200-500/mm³, pt symptoms like • Swollen lymphnodes (lymphadenopathy)
  • hairy leukoplakia (white patch on tongue caused by EBV)
  • Oral Candidiasis (yeast infec.n)

- when Tcells become <200 cells/mm³, pt. becomes severely immunocompromised, which is called HIV infec.n to AIDS progression [HIV in blood ↑]

Symptoms - persistent fever,
fatigue,
weight loss.

AIDS defining conditions - Recurrent Bact. Pneumonia
Pneumocystis Pneumonia
Esoph. Candidiasis,
Cancers - Kaposis Sarcoma,
lymphoma.

Absent

30/≤23 → 2, 3, 5, [illegible] 19, 21, 24, 26, 27, 30.

≥16/23 - 2, 3, 4, 5, 9, 10, 26, 27, 29

≤15 - 2, 3, 9, 19, 26, 29,

6/6 - 6, 7, 10, 22, 25, 26

13/6 - 4, 11, 20, 23, 26

14/6 - 32

15/6 - 27, 30

Patho - disease. 6/11/23

physiology - Functions

study of changes in functions or functionality of different body process under disease state.

Cell Injury

Defination:

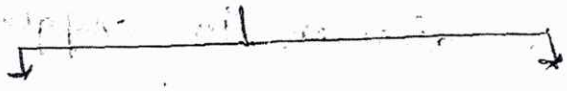
Cell injury is define as functional and morphology changes and variety of stress a cell experiences due to changes in it's internal and external environment.

Morphology - At tissue level (structural changes)

Etiology - study of cause (or) different factors causing disease.

Pathogenesis / pathophysiology (Sequencial changes progression of disease).

Etiology



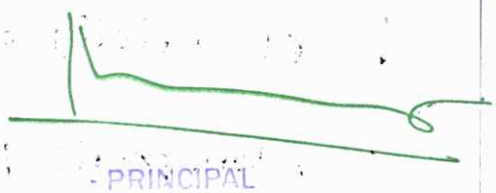
Genetics Acquired

cell injury - Reversible - cell funct^{n} can be restored

Irreversible - cannot be restored

- Etiology:

1. Genetics
2. Acquired



Acquired causes :-

Hypoxia and Ischemia
↓ O_2 ↓ blood supply

- Pathogenesis due to Hypoxia cell goes into stress. Continued stress will result in reversible cell injury further cell goes into irreversible cell injury as a result disease occurs.

- Hypoxia ($\downarrow O_2$)
↓
stress
↓
reversible cell injury
↓
irreversible cell injury
↓
Disease

- Hypoxia can result into interreptution of blood supply (or) disorders in oxygen carrying RBCs heart & lung diseases.

1. Physical agents :
- Mechanical trauma (road accident)
- Thermal trauma (excessive heat (or) cold)
- Electricity, radiation (ionsing (or) UV)
- Atomspheric pressure (rapid (or) sudden changes)

2. Chemical Agent & drugs :- 7/11/2023
- chemical poisons like arsenic, cyanide, mercury
- strng acid and bases
- environmental pollutants
- insecticides and pesticides.
- oxygens and high concentrat^n
- ~~Drugs~~:
  Hypertonic, glucose & salts
  social agents – alcohol, narcotic drugs
- therapeutic adm^n of drug

3. Microbial agents :- infect^n through fungia, bacteria, virus. (Path-gens)

4. Immunologic Agent / drugs.
- Auto immune
- hypersensitivity -
- Anaphylactic rxn

5. Nutritional derangements

6. Ageing

7. psychogenic disease → Schizophrenia. → Drug Addiction. → causing injury to brain cells.

8. Idiopathic causes / iatrogenic (means of unknown origin)

9. I
Iatrogenic causes – lack of intrest, carelessness of medical staff

Genetics Causes:

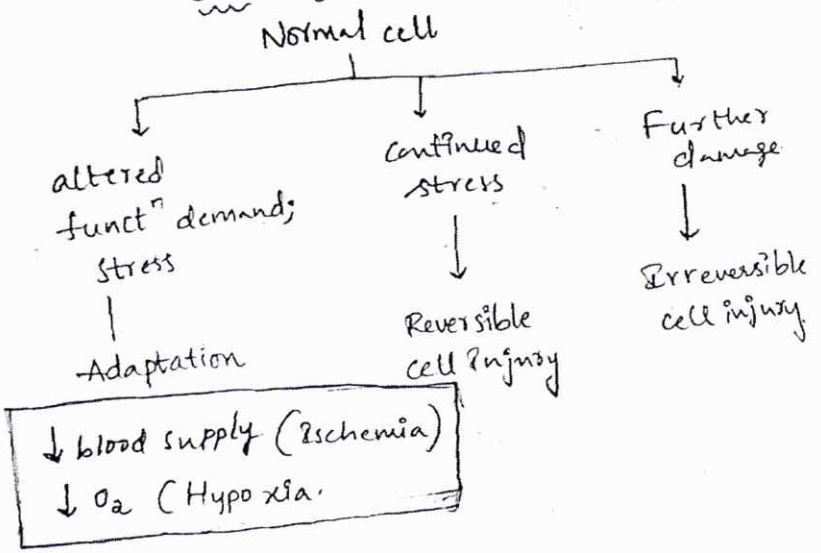
- Single gene defect, mutations, sickle cell anemia.
- developmental defect

Ex: folic acid of mother causes improper development of nervous system in fetal stage.

- In fetal stage heart contains a hole.

Cell Adaptations

9/11/2023



defination:

Cell adaptations are adjustments the cell makes in response to stress due to physiology (or) pathologic needs.

- Adjustments are done by
  - decrease (or) increase in cell size (atrophy) (hypertrophy)
  - and hyperplasia (Inc in no. of cell)
  - changing the pathway of cell differentiation.

metaplasia (change in cell type) and dysplasia (disordered cell development from foetal growth.)

- Atrophy:

Atrophy can be physiologic (or) pathologic

→ In physiologic eg:- Thymus gland in adults (shrinkage of Thymus gland).

- Brain atrophy due to increase in age.
- Osteoporosis due to increase in age.
- Atrophy of lymphatic tissue due to age.

Pathologic atrophy eg:

occurs in cond^n like-

- starvation (degrad^n of stored fat & sugar)
- Ischemia (Renal artery atherosclero-sis in atrophy kidney's).
- Disused atrophy (wasting of muscles of immobilized limb).

- Neuropathic atrophy
  eg: polio
- endocrine atrophy (Hypopituitarism
  Bleading to thyroid, adrenal gland
- Pressure atrophy
  (erossion of skull by Maningoma from the pia-arachnoid, erossion of sternum by Aneurism of arch of aorta).

14/11/2023

Idiopathic – Myopathy

Hypertrophy -

Increase in the size of Parenchymal cells resulting in increase in size of effected organal tissue

Causes :-

physiology :-

eg: - Uterine, Smooth muscle cells in pregnansy.

- Due to enlarge size of a uterin
- estrogenic stimulation.

pathologic :-

eg: hypertrophy of cardiac muscle in systemic hypertension.

- Hypertrophy of Smooth muscle in pyloric stenosis becoz lumen became narrow.
  (reduct^n in size / Shrinkage)
- Hypertrophy of Skeletal muscle in athelets and manual labourers.
- Compensatory Hypertrophy.
  eg: following nephrectomy (to remove the kidney through disection).

Hyperplasia :-

- Inc in no. of parenchymal cells.

Causes :

- cells are divided to 3 types

(1) Labile :- (divide).

- labile cell continue to proliferate throughout life.
- They never enter into Go phase.
  eg epithelial cells of skin, Mucous membrane

(2) Stable cells :

They retain proliferating capacity but donot divide unless stimulated

- When stimulated they move from G_0 to G_1 phase. ∴ $(G_0 - G_1)$ eg: parenchyme cells of liver.

(3) Permanent cells:

- They cannot proliferate after birth.
- They are in G_0 phase / resting phase.

eg: neurons & cardiac

Causes:

physiology:-

eg:-
- Hormonal Hyperplasia in pregnancy.
- compensatory Regenaration of liver after partial Hepatectomy.

pathologic:

eg:-
- endometerial hyperplasia following estrogen excess.
- Benign prostatic hyperplasia (BPH) in elder males.

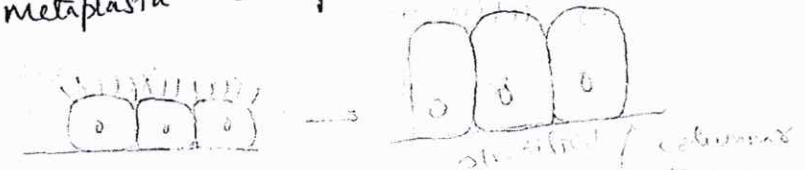
15/11/23

- Viral Warts - (type of skin infection)

Metaplasia :- 15/11/2023

Meta - transformation
plasia - growth.

- Metaplasia is a reversible change of one type of cell (epithelial (or) Mesenchymal) to another type in response to abnormal stimulus. It often reverse back to nrml when stimulus is removed.
- If stimulus persists for long epithelial metaplasia changes into cancer.



- 2 types of metaplasia occurs-

1. epithelial metaplasia:-

- It is more common
- The meta plastic change can be patchy (small portion is patch cells) / diffuse
- It results in replace by strnger but less specialised epithelium and deprivation of blood supply protective mucous secretion. Hence becoming more

Prone to infection.

eg:- Depending on the type of epithelium transformed.

It is of two types:

1. Squamous
2. columnar

1. Squamous:

eg:- In bronchees of heavy smokers.

- In the uterine endo cervix in prolapse of uterus. (portion of uterus inner)
- In vit A deficiency, i.e., Xerophthalmia. (cells of retina changes)

2. Columnar metaplasia:

(thickness of cell type) (precancerous condition)

eg: Barrett's Esophagus there is change of nrml squamous epithelium in lower esophagus to columnar epithelium.

- change of Pseudostratified columnar epithelium in chronic bronchitis and bronchiectasis to columnar typesl.

~~less~~

Mesenchymal Metaplasias:-

- It is less common.
- There is one transformation of one adult type of mesenchymal tissue to another.

(1) Oseous metaplasia - i.e., formation of bone in fibrous tissue & cartilage.

eg: Arterial wall of elderly people.

(2) cartilagenous metaplasia.

(more mobility)

occurs when there is undue mobility

eg: In healing of fractures.

Dysplasia:-

disordered cellular development often preceeded / accompany with metaplasia and hypoplu hyperplasia.

- Also called Atypical → hyperplasia mostly occurs in epithelial cells
- Epithelial dysplasia is characterised by cell proliferation and cytologic changes like-
  - increase no. of layers of cells.
  - Disordered arrangements of cells in basal layer.
  - loss of basal polarity.

(nuclei moving away from membrane).

- Nuclear hyper chromatism

Eg:- Uterine cervix, respiratory tract, oral cavity & Oesophagus

- In cervix [CIN] cervical Intra epithelial Neoplasia of grade 1, 2, 3 can (Cancer) occured.

Pathogenesis of Ischemic/Hypoxic Injury :: 16/11/2023

Injury / Reversible
\ Irreversible

Reversible injury:-

Cell changes occur in cell -

1. ↓ ATP generation

- ATP is required for - membrane transport, phospholipid metabolism, protein and lipid Synthesis.

- ATP is required for aerobic respiration
- To maintain constant supply, ATP is generated from Glycogen (or) glucose by anaerobic glycolysis under Ischemic/Hypoxic state.

2) Lactic acidosis :

low oxygen supply/
failure of Mitochondria
↓
Cell switch to anerobic glycotic pathway (glycolytic)
↓
To maintain continues ATP generation
↓
Rapid depletion of glycogen (led to glycogen).
↓
Accumulation of lactic acid and low intra cellular pH.
↓
clumping of chromatin in the nucleus.

3) plasma membrane pump damage

decreased ATP
↓
Increased cellular fattyacid
↓
due to decreased phospholipid generation which helps in membrane repair, thus causes damage to the membrane and membrane pumps.
(Na^+, K^+ pump & calcium ATPase. Channel

(a) Na^+, K^+ ATPase

Na^+ - major extra cellular ion.

K^+ - major intra " "

Nrmly, Na^+ moves out & K^+ moves inside.

- These failure causes more sodium in cell.

& ↑ H_2O ~~—~~ results in,

- swelling of cell — hydropic swelling.

(b) Calcium pump

Calcium inflex

↓

fed Ca^{2+} into the cell (mitochondria)

↓

Mitochondrial swelling

amodifes phospholipid density

(4) Decreased protein Synthesis and dispersed ribosomes.

continued hypoxia

↓

swelling of endoplasmic reticulum, golgi apparatus and detachments of ribosomes from RER

↓

decreased protein Synthesis.

Pathogenesis of Irreversible cell injury

Two features differentiate irreversible cell injury from reversible injury.

→ Inability to reverse mitochondria dysfunction

→ Inability to reverse ~~mitochondrial~~ membrane disfunct^n

1. Mitochondria damage - ↑ Ca^{++} influx due to reperfusion
2. Membrane damage - Activat^n of phospholipases & ATPase
3. cyto skeletal - Activat^n of proteases
4. Nuclear - " " endonucleases
5. lysosomal cell death & phagocytosis - " " hydrolytic enzymes

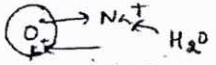
- Mitochondrial damage caused due to ↑ Ca^{++} influx
- Nuclear damage occuring in 3 form.
- Pyknosis - Condensation & dumping of nuclear mating it dark basophilic.
- Karyorrhexis - nuclear fragmentat^n into small bits dispense into cytoplasm
- Karyolysis - dissolut^n of the nucleus damaged DNA activates pro apoptitic proteins leading to cell death.

Morphology of Reversible 4/12/23

- Hydropic change / cloudy swelling / vascular degeneration

- lot of H_2O inside the cell [H_2O accumulation]

- It occurs due to the changes in Na^+ regulation [impairment]



(2) Hyaline change

glassy [translucent]

→ glass hyalinization is glassy, homogenous, eosinphilic appearance of protinaceous material

Hyalinisation

Intracellular [epithelium]

ex: (1) An proteinuria
(2) Thyphoid fever [enteric fever intestine damage]
(3) Alcoholic liver disease [malloy]
(4) viral infection [aggregates of intermediate filament]
(5) Russels bodies [excessive immuno globulin in RER]

extra cellular [connective Tiss]

(1) Hyaline arteriole sclerosis in renal vescles in DM & HTN
(2) Hyalinised glomeruli in chronic glomerulo nepheitis.
(3) Hyaline degenerat^n in leiomyoma of uterus.

(3) Mucoid

- Mucus is a secretary product of mucus gland. It is a combinat^n of protein [mucin] complexed with mucopoly saccharides.
- Mucin is a chief constituent of mucus
- Myxoid - connective T mucin
- PAS (Periodic Acid schiff) is a dye/stain used to differentiate epithilium (+ve) & connective T mucin (-ve)

Morphology of Irreversible cell injury

cell death → Necrosis - murder
→ Apoptosis [plan death] - suicide
→ Autolysis / cell digestion ← Rapid
— Intermediate
— slow

Rapid digestion occurs in - gastric mucosa, pancreas

- Intermediate - kidney, liver, heart
Slow - cartilage & bone [fibrous tissue]

Autolysis

It is disintegration of the cell by its own hydrolytic enzyme prsnt in lysosomes as a post-mortem change in tissues. Therefore not accompained by inflammatory response in surrounding tissue.

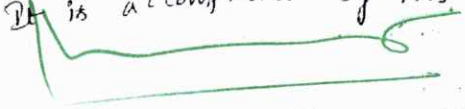
→ Morphologically identified by homogenous eosinphilic cytoplasm with loss of cellular details.

Apoptosis

programmed cell death. coordinated & generally programmed cell suicide in which unwanted host cells are eliminated by activation of intrinsic enzymes.

Necrosis

/focal (localised area of death of tissue) death along with degradation of tissue by hydrolytic enzymes. It is accompinied by inflammatory gxns.



Necrosis :

Two essential changes bring abt irreversible injury in necrosis

- cell digestion by lytic enzymes
- Denaturation of proteins

→ Necrosis morphologically identified by cytoplasmic changes like homogenous & eosinophilic cytoplasm & nuclear changes like pyknosis, karyolysis, karyorrhexis

5 types of Necrosis :

1. Coagulative necrosis
   - it is most common type
   - Caused by sudden cessation of blood flow
   - less often caused by bacterial, & chemical agent
   - organs effected are heart, kidney, spleen
   - Morphologically, early-stage coaggulative necrosis is called "infact" i.e, pale initially. later stage it becomes more yellowish, dense and shrunken

Cessation of blood flow
↓
death of tissue - called infract (early stage)
↓
late stage - More yellow & shrunken
↓
Coagulative necrosis

2. Liquefactive Necrosis

Caused due to ischemic injury, bacterial & fungal infection.

- occurs due to degradation of tissue by dominant effect of powerful hydrolytic enzymes

Thus forming semi fluid material at the centre

- infract of brain & abcess cavity
- Morphologic appearence - cyst like wall with fluid filled centre containing cell debris is seen.

3. Caseous Necrosis (cheesy)
   - it is found in centre of the foci of tubercular granulomas
   - it combines the features of 1st & 2nd types of necrosis.

4. Fat Necrosis
   - specially occur at fat rich anatomic locations in the body.

   ex:- Pancreatic necrosis

   - This results in hydrolysis of neutral fat present in adipose cell into glycerol & free fatty acid
   - FFA combines with calcium to form calcium soap which is called as "Saponification".
   - Damaged adipose tissue gives cloudy appearance with only free fatty acids remaining behind after glycerol leaks out.

5. Fibrinoid necrosis
   - It is characterised by deposition of fibrin like material which has staining properties like fibrin.
   - Identified by brightly eosinophilic, hyalin like deposition in vessel wall. Necrotic

tissue is surrounded by nuclear debris.

Apoptosis

Mechanism

Initiation of apoptosis

↓

occurs in cell membrane

step 1 :

- it occurs in cell membrane
- It has 2 signals for initation.

1st signal - withdrawl of normal cell survival signals i.e., absence of cytokines hormones growth factor

2nd signal - Actn of agents of cell injury i.e, heat, radiatn, hypoxia, toxins etc

Step 2 :-

- Initiatn of death signalling pathways
- Intrinsic mitochondrial pathway
- cytochrome c is the life line of intact mitochondria
- Its release into cytoplasm initiates mitochondrial degeneration & initiates apoptosis.
- it is regulated by BCL family of oncogenes (β-cells lymphoma) [oncogenes are cancer causing genes]

[Antioncogenes are tumor suppressor genes.

Antiapoptotic BCL family - BCL-2 BCL-XL & MCL-1 [mycloid cell leukemia] they prevent the leakage of cytochrom-c

pro-apoptotic - BCL family → BAX BAK (BAX-BCL2)

associated X protein; BAK - BCL-2 associated killer proteins) pro apoptic promote leakage of cytochrom-c].

Step 3: activat^n of initiator of caspase (enzymes)

(a) Mitochondrial Pathway

Cytochrome C (in cytosol) + Apoptosis-activating factor (ApAF-1) → Apoptosome complex.

Apoptasome complex + precursor Caspases (caspase-9) —cleavage of Caspase 9→ Activated form of caspases

(b) Death receptor initiated pathway

FADD + caspase 10 (inactive) —inhibited by FLIP protein→ caspase 8

FADD - Fas associated death domain & adaptor protein recruited to death inducing signalling

FLIP - FLICE inhibitory protein

FLICE - FADD like interleukin -1β converting enzyme

FAS

Step 4 - activat^n of apoptosis executing caspase activat^n of caspase -3 & 6 leads to their action on various cell components like DNA ase, Nuclear matrix proteins etc & leads to proteolytic enzyme actions on them like chromatin clumping, disruption of ER, damage to mitochandria & cyto skeliton.

Step 5: phagocytosis.

Gangrene — 3 types

1. wet
2. Dry
3. Gas

Gangrene - necrosis of tissue associated with super added putrefaction often following coagulative necrosis due to ischemia.

Putrefaction - is decomposition of protein, breakdown of tissue & liquefaction of organs. It is the fifth stage of death.

(1) Dry gangrene: [occur in arteries]

it is usually due to blockage (or) decreased arterial blood supply to any tissue. Mostly occurs in atherosclerosis.

→ organs - limbs, extremities of the body

→ A clear line of seperation lies b/w the healthy, life (live), viable tissue and gangrenous tissue consists of inflammatory granulation tissue. mech of ischemic - hypoxic injury

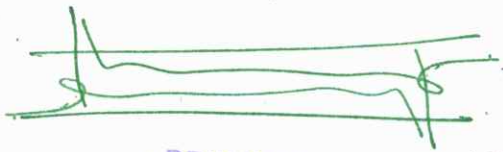
formatn of dry gangrene ↓

coagulative necrosis ↓

decreased blood supply ↓

few bacteria prsnt ↓

Amputation [cutting].



Morphology:

1. Dry, shriveled, shrunken, black

→ Black colour is due to rxn b/w hemoglobin & hydrogen sulphide (HS_2) forming black iron sulfide. Hb is released from hemolysed RBC & HS_2 is released from bacteria

HS_2 + Hb ⟶ black iron sulfide.
(bacteria) (hemolysed RBC)

6/12/23

Wet Gangrene: [venous blockage] blood is pooled.

- it occurs in moist tissues like bowel, lungs, mouth, cervix
- TWO most common ex ① diabetic foot - due to more glucose in tissue favouring bacterial growth.
② Bed sores - due to pressure on the site of affected in bed ridden patients [sacrum, buttucks, heal].

Etiology / Causes

Mostly due to venous supply blockage. Sometimes due to both arterial & venous blockage

Mechanism

Affected part is stuffed with blood
↓
favours overgrowth of ~~putrifaf~~ putrifactive bacteria
↓
Toxic products are more concentrated in affected area & may spread to other parts through circulation.
↓
Thus causing systemic symptoms of septicemia (infection of whole body bcoz blood).
↓
Death

Morphology:

Affected part is soft & swollen, putrid dark & rotten.

eg: gangrene of bowel, same as in dry gangrene their is coagulative necrosis but with stuffing of blood in damaged tissue giving black/dark colour.

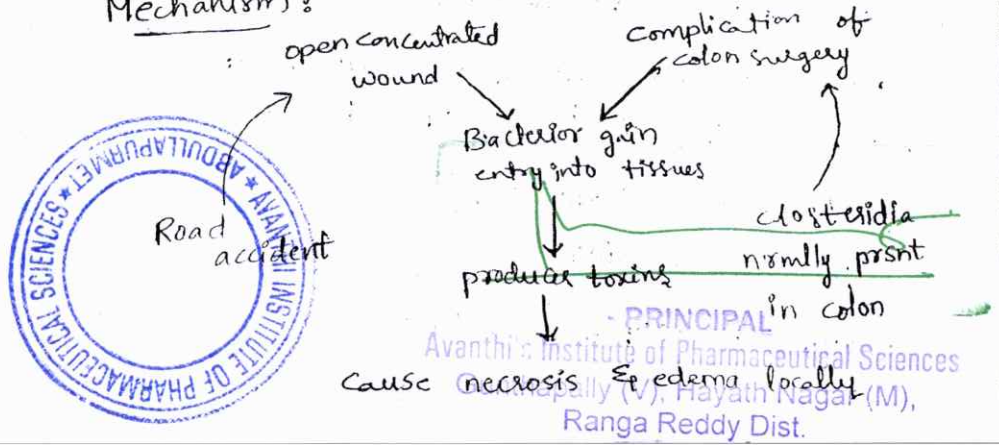
Spread:

Spreads to peritonium causing peritonitis
→ prognosis is severe due to severe septicimia when compared to dry gangrene no line of seperation exists.

3) Gas gangrene:

A special type of wet gangrene caused by gas forming Gram +ve clostridia.

Mechanism:



↓

Absorbed into circulation

↓

produce systemic manifestations (whole body) . (symptoms).

Morphology :

Muscle fibres undergoes coagulative necrosis with liquefaction

Hyperlipidemia (blood) / Hyperlipo proteinemia.

[↑lipids / lipoprotein in blood]

Lipoproteins :-

— These are lipid + protein complex bound to another protein. It allows fat to move through water in an out of cell.

Types of Lipoproteins

1. HDL (Good cholesterol)

It absorbs & transport cholestrol from blood & body parts to liver. Liver flushes it out from the body.

2. LDL [bad Cholesterol].

It constitutes most of the cholestrol ~~from~~ in the ~~blood~~ body and is a plaque builder.

3. VLDL [bad cholesterol]

produced in liver.

→ causes buildup of plaque in the arteries

→ Difficult to measures its levels in the blood directly.

→ It contains highest amt of triglycerides

→ 1/5th of triglycerides levels is equal to VLDL levels.

Lipo proteinemia

Hypolipidemia

→ low levels of lipids / Cholestcrol in the blood

Hyperlipidemia

→ High levels of lipids / cholesterol in blood.

fredricksons classification of hyperlipidemia

Types	chilomicrons	LDL β-LP	VLDL preβ-LP	floating β-LP
1. familial Hyper-chilomicronemia	+	−	slightly ↑es	
2a. familial hyper-cholesterolemia Chylomicronemia		+		
b. F. combained hyper cholesterol-emia		+	+	
3. " dys-β-lipo-proteinemia				
4. " Hyperlipidemia			+	
5. endogenous hyper triglyceridemia	+		+	

Chylomicrons -

largest form of LP responsible for transport of lipids throughout the body. synthesized in the intestinal enterocytes during active fat absorption & then transported to lymph.

Floating β-LP :-

These have the presence of VLDL having abnormally high cholesterol content & abnormal electrophoretic mobility.

Inflammation :

22/12/23

"response of living tissue to an injury.

→ 2 aims - . to eliminate the injurier agent which is causing injury.

. to remove damage tissue.

- Inflamat^n is the protective response to the body.
- Agents - physical, chemical, Biological, immunological
- Pathology : Black fungi leads to death, skin allergy, damages the organ.

- Inflamation involves 2 process:

1. inflammatory Response - early process.
2. Repair / healing - removal of damage tissue.
   → formation of tissue

2 types of inflammation.

Acute inflammat^n

- the inflammat^n response in short durat^n
- caused by all neutrophiles
- swelling at the site of damage.

chronic inflammat^n

- Agent causing injury for long duration.

Eg: Hepatitis

- caused by plasma cell, macrophages, lymphocytes.

- Signs of inflammat^{n} - swelling, Rubur (redness), Tumour (swelling), calor (heat), Dalar (pain).

- Cells of inflammation:

1. Neutrophils - involved in all acute inflam^{n}
2. Esnophils
3. Basophils
4. Lymphocytes
5. Macrophages
6. plasma cells

Neutrophils: should cross the wall of blood vessel. (Emigrat^{n} of N)

↓

movement of neutrophils to site of injury

↓

Engulfing the agent which is causing the injury

↓

Degranulation

↓

Complete degradation of injuries agent

↓

Healing / Repair will occur

Acute inflammatory: 23/12/23

Two changes occurs at the site of injury during acute inflammatory.

- Vascular changes
- cellular changes

Acute inflammatory 23/12/23

- Vascular changes
  - Hemodynamic changes
  - changes/increases vascular permiability
- cellular changes

- occurs in microvascular = Arterioles, capillaries, venules

Aorta → Artery → Arteriole → capillary → venule → Vein → Venacava

Hemodynamic changes:

The immediate changes occur at site of injury in tissue / cell

immediately changes occurs after tissue injury.

i) constriction of the arterioles.
- minor injury - 10-15 sec
- major injury - 5 mins

ii) Progressive dilation of microvasculation (Arteriole, Capillaries, Venules)

iii) increased intravascular volume in microvasculature.

iv) Redness & warmth.

v) increased in hydrostatic pressure in microvasculature

vi) Fluid escapes to extra vascular site of injury

vii) Swelling

viii) Blood becomes thick in the capillaries.

ix) migration of WBC from capillaries to site of injury.

Increased Vascular Permeability.

27/12/23

Fluid balance maintained by two pressures -

- Hydrostatic Pressure.
- Osmotic Pressure.

- factors responsed for movement of fluid and out of side. fluid in blood vessel.

(i) increased hydrostatic pressure in Micro - vascular.

(ii) increased osmotic pressure in Extra Vascular Tissue.

- Conditions of movements of fluid into the Micro Vasculature.

(i) increased hydrostatic pressure in extra Vascular Tissue

(ii) increased Osmotic pressure in micro Vasculature. maintains fluid.

- Albumin is respond for blood vessels. osmotic pressure.

- adquate of blood vessels more in albumins, the flow of blood vessels increases.

- less levels of albumin leads to hypoalbuminemia leads to swelling, fluid flows out side of blood vessels.

- hydrostatic pressure increases fluid flows outside of extra vascular Tissue.

- treatment of hypo albumins is albumin infusion. should be given.

Increased Vascular permeability

- Micro vasculature becomes leakly. It means; leaks fluid from the arterioles, capillaries venules.

- 5 mechanism responsible for ↑sed permeability of Vasculature are -

(i) contraction of endothelium of MV

(ii) Retraction of endothelium of MV

(iii) Endothelial damage Mediated by leukocytes

Direct injury to endothelium

(v) Neo Vascularisation

① Contraction of endothelium blood vessels of MV

- Contract^{n} of endothelium leads to increased of movement of fluid outside the MV.
- leads to Tis of blood capillaries.
- contract^{n} of endothelium is in increased Vascular permeability.
- thickness is ↓ed
- it is mainly seen in venules.
- it occurs at site of injury immediately
- it occurs 15-30 mins duration.

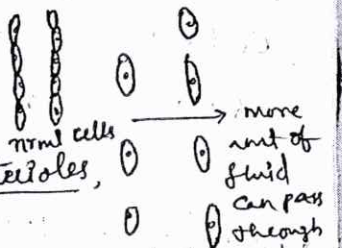
② Retraction of endothelium (going back) blood vessels of MV

The gap junction is retracted the blood vessels at gaps

- it leads to movements of blood vessels
- it occurs for 4-6 hrs after injury.
- it occurs at venules
- They promote communicat^{n} b/w cells.
- Duration - 2 to 4 hrs, mechanism stops.

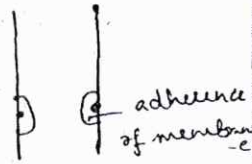
③ Direct injury to endothelium of Microvasculature:- 28/12/23

- Direct injury to endothelium leads to dead/ necrosis of endothelium cells.
- ↓
- Detachment of DE cells.
- This mechanism seen in arterioles, capillaries & venules.
- occurs after 2-12 hrs after tissue injury.
- sometimes it occurs immediately after tissue injury.
- Bec^{o}z of dead cells more amount of fluid can pass through gaps.



④ Endothelium damage mediated by leukocytes

- During the process of migration of blood vessels, the leukocytes gets attached to membrane.
- Leukocytes gets attached sometimes to the injury sites
- leukocytes releases upon activation to free radicles and damage the adj. cells at the site of injury



(5) Neovascularisation

Newly formed blood vessels. Capillaries are more leakely than normal MV.

Cellular changes / Events:

- cellular changes (i) Migration of leukocytes
  (ii) phagocytosis.

- Migration of leukocytes

migration of WBC. at site of injury.
↓
changes in formed elements of blood.

- Blood become thick, more amt of fluid is occured.
central stream of cells moves to peripheral
central stream of cells.

- Adhesion of WBC of endothelium
↓
Migration of WBC to site of injury.

- WBC will damage the basement membrane of capillaries. Endothelium at the site of injury.

basement membrane of capillaries

Phagocytosis

cell eating the foreign body
(or)
engulfing the pathogens / foreign body by the cell and destruction of foreign body.

- 2 types of phagocytosis
  - Neutrophils
  - Macrophages.

- Macrophages prsnt in liver known as Kupffer cells
Macrophages prsnt in skin - dendritic cells
Macrophages prsnt in brain - Microglia.

29/12/2023

steps:

1. Recognision / Identification of pathogen of attachment of pathogen.
2. Engulfment of pathogen
3. killing & degradation of pathogens

Recognision of attachment of Attachment of pathogen:

- identify and function the receptors, pathogens
- specialized cells are prsnt on cell membrane of phagocytes.
- They are two types of receptors.
  1. Scavange receptors
  2. Mannosa receptor
- process of phagocytosis is increased by specilized proteins called opsonins.

they will coat the surface of pathogens by them they enhance the process of the phagocytosis.

- The main function is easily recognised the pathogens. if attached by the cell surface receptors.

② Engulfment of pathogens

- cell membrane

1. formation of pseudopodium by phagocytes
   ↓ temporary structure
   formed pseudopodium envelop the pathogens is known as phagosome
   ↓
   Internalisation of phagosome – enters into cytoplasm
   ↓
   fusion of phagosome with lysosomes.
   ..and the structure called phagolysosomes.

③ Killing & degradation of pathogens

mechanisms are involve -

(I) oxidative Bacterial mechanism oxygen free radicals ($O_2^{\cdot}$, H_2O_2, $OH^{\cdot}$). HOCl, HOBr, HOI.

(a) Myloperoxidase dependent Killing

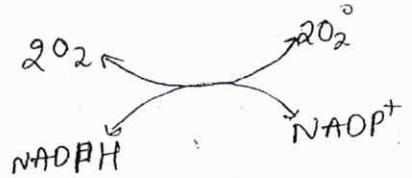
(b) Myloperoxidase independent Killing.

(II) oxidative Bacterial mechanism by lysosomes, granules

(III) Non oxidative Bactericidal Mechanism.

(I) oxidative Bacterial Mechanism free radicles :-

- Oxygen free radicals are response for killing pathogens (bacterial)
- enzymes phagocytic cells having phagosomes consume more amount of oxygen.
- These (phagosome) membrane of pathogen. contain the enzymes likes NADPH oxidase.
- These enzyme response for the generation of O_2 free radicles.
- it will oxidise NADPH to $NADP^+$



② Myloperoxidase dependent killing

- H_2O_2 - having more antibacterial form. action.

$$2O_2^{\cdot} + 2H \longrightarrow H_2O_2$$

(i) generation of oxidation oxygen free radicles.

(ii) conversion of free radicles.

$$H_2O_2 \xrightarrow[Cl^-,\ Br^-,\ I^-]{MPO} \begin{matrix} HOCl \\ HOBr \\ HOI \end{matrix}$$

MPO present only in neutrophils potent antimicrobial act^n

HOCl, HOI, HOBr kills the pathogens & Degrade the pathogens

- MPO occurs in neutrophils
- MPIO occurs in Macrophages.

⑥ Myloperoxide independent killing

It occurs in Macrophages.

H_2O_2 — $OH^{\cdot}$ – Fenton
H_2O_2 — $OH^{\cdot}$ – Haubel

becz it does not contain Myloperoxide NADPH enzyme.

II O.B.M by lysosomes, Granules:

fusion of phagosomes with lysosomes, after fuson all the enzymes are degraded & destruction of pathogens.

III Non-OBM:

- It is Antifungal, Anti bacterial paracytics action.
- Nitric oxide is released by endothelium at the site of injury.

~~Nitric oxide~~ - 03/1/2024

Chemical Mediators of Inflammation

The chemicals substance which are prsnt in body & release, at the site of injury.

- They will enhances the process of inflammatory.
- They play role in or increasing the vascular permeability of MV.
- Vasodilation of Microvasculature.
- Pain
- Fever.

- chemical Mediators are two types.
  - cell derived
  - plasma derived.

- cell derived -
  cells forms at site of injury.

plasma derived -
  Forms plasma at site of injury.

cell derived :-

Classified into 5 types.

1. Amines
2. prostaglandin
3. cytokines
4. platelet activating ~~fto~~ factors.
5. oxygen free radicles & NO

Neurokines & peptide

1. Amines

- Histamines – prsnt in mast cells
  Serotonin – in brain – neurotransmitter.

2. prostaglandins – PGE_2, PGE_1, $PGF_{2\alpha}$, prosta cycline.
   NSAIDs block prostaglandins.
   Thromboxanes, Leukotrines.

- components of lysosomes

plasma derived –

1. Kinin System
2. clotting System
3. fibrinolytic system
4. complement system

cell derived

04/1/2024

- Neuropeptidis
  components of lysosomes
- Vasoactive Amines are first chemical to release at the site of injury, 15-30mins after injury.
- Histamines – Mast cells, Basophils, platelets granules.
- Histamine is the main chemical that occurs in Asthma
- Histamine is main mediator of micro vascular permeability
- Histamine is a chemical responsible for increase vasculability & Itching.

Serotonin

- Regulate Apetite
- It stored in Enterochromafin cells of Intestine
- It also stored in high conc. of platelets
- It plays imp. role of platelet aggregation
- In case of inflammatoly Selotonin is same as Histamine.
- when compare to serotonin, histamine is very high potent.
- serotonin is imp for platelets, coagulate than i.e., formation of clot.

Ecosanoids :-

Arachidonic acid-

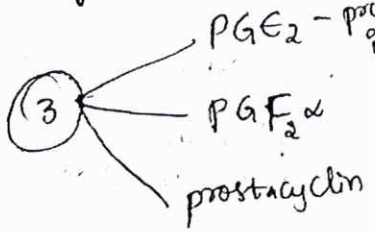
- It is fatty acid
- it is component of cell membrane.

- when there is injury, the activation of Arachidonic acid
- Activated Arachidonic acid is converted into prostaglandins, thromboxanes, leukotrines with the help of enzymes, cyclooxygenase & Lipooxygenase enzyme (LT).

PG, Tx - polent inflammatory mediator
They are responsible for pain, Increase Vascular permeability

- COX is blocked by NSAIDs - then there is no product^{n} of PG, Tx (which means stops swelling).

Prostaglands



- PGE_2 responsible for Vasodilation, Bronchodilation.
side effect of NSAIDs - reduce product^{n} of gastric bleeding - vaso dilation.

⊖ NSAIDs is a contraindicator in patient with peptic ulcers, BP, Asthma.

$PGF_2\alpha$

- responsible for Broncho contriction, Vaso dilation.

Prostacyclin: responsible for vaso constriction - Broncho-contriction.

- All these three are ↑s Vascular permeability of in ~~increase~~ chemicals.

05/1/2024

Thromboxanes:

Thrombo - clot
It is released by platelets.
It causes blood clotting and also caused Vasoconstriction.

Leucotrienes:

- Identified in WBC
- these ~~cells~~ chemical are released by all injured cells of body
slow chemical responses, & sustained response.
- play imp role in Asthma.
drug - leukotrines Antagonist - Montilleukast - tratio
- slow release & continuous action

Montek-lc — Histamine
↓
Leukotrines

- Pathway - lipo oxygnase pathway.

Broncho constriction & Vasoconstriction.
Montilleukast block the leukotrine receptors.

Cytokines

- Cyto - cell
- These are essential for cell to cell communication
- They play imp role in controlling inflammation
- These are proteins and peptides.
- There are 200 cytokines as been identified and also involved in cell growth.

Neurokines / peptides

1. Substance P:
2. Neuropeptide Y
3. Vasoactive Intestinal peptide

- These are responsible for causing Pain during inflammation.
- They also cause Vasoconstrictions.

Lysosomes

- Sucidal bag.
- Killing & degradation of foreign body

+ The contains present in the lysosomes are Granules.

- These Granules are further divided into
  - primary
  - secondary
  - Teritary

1° - Non-specific lysosomes

2° - Specific lysosomes

3° - Teritary lysosomes

enzymes for Killing & degradation of Pathogens:

- lysozymes
- proteinases
- Hydrolases
- peroxidase
- Lipases

Platelets Activating factors:-

Platelets Activates is a lipids synthesized by every damage cell / tissue.

- Responsible of platelets

↓

Aggression

↓

Adhesion

↓

clot formation

- enhancing the process of inflammation.

Oxygenase oxygen free radicals :-

- responsible for killing of degradations of pathogens

$H_2O_2 \longrightarrow 2O_2^{\cdot} + 2H^{\cdot}$

Plasma derived chemical Mediators :-

End products of (1) Kinin system → Brady Kinin.

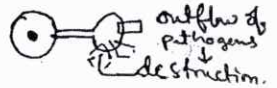
(2) ~~clot~~ complement system → Membrane attack complex in C_{3b} - C_{9}.

(3) Clotting system

(4) fribinolytic system

- Brady Kinin plays imp role in pulmonary hyper-tension [↑es BP of arterioles]
- these are responsible for vasodilation, increased Vascular Permeability and inflammation.
- responsible for dry cough.
- when ever ~~ag~~ ab pathogens cells of the body enter and recognised ↑ bind to the antigens. this is known as Antigen antibody complex.
- There will be destruction of pathogens by complement system leads to

- These complement system form holes on pathogens leads to death of pathogens.
- ~~Ag~~ & Ab complex is responsible for activation of complement system.



6/1/2024

complement system

↓

cascade of enzymatic rxn.

1 protein gets activated and breakdown of 2 parts bcoz part breaks another protein and thus final products C_{3b} - C_{5}+C_{9} gets holes on the membrane ~~and~~ &

- It occurs by antigen-antibody complex and kills pathogens.
- HIV :- It is identified by neutrophils, Macrophages. (Antigen printing cells)

↓

Antigen cells.

↓

lymph nodes

↓

Action of immune cells.

↓

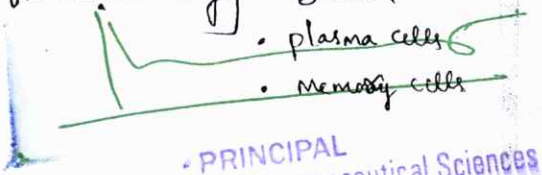
mediated

responsible for cell immunity

- Cytotoxic T cells
- T-Helper cells
- T-suppressor cells
- Memory T cells

B-cells

- plasma cells
- Memory cells



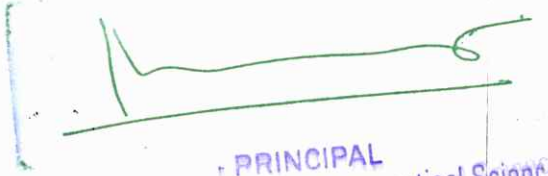
- Plasma cells - it will produce antibodies against pathogens.

Cytotoxic - Release the chemicals & kills the pathogens

T. Helper cells - Activation of immune cells.

T. Suppressor - cntrls the immune cells.

plasma cells
↓
antibody
↓
actvn of ab - ag complex
↓
complement system
↓
it destroy the pathogens by forming holes



SYPHILIS

- Syphilis is a sexually transmitted disease which will disrupt the mucus membrane, with the help of a causative agent Treponema pallidum (spirochete). (infect the reproductive part) Venereal (spiral shape bacteria)

Etiology:

- sexually transmitted & non-sexual, by blood transfusions, open abrasions & ulcers and exudates.
- It will be occured in more often to Homo-sexuals & Bisexuals.

Pathopysiology

→ when a bacteria enters into a healthy persons, it chooses a pooper mucus membrane and gets attach to the membrane with the help of Surface pili.

→ When it gets completely binded over the mucus membrane and it starts releasing some proteins and it shows its action in 3 different stages.

- primary stage - formation of chancres
- secondary stage - abrasions, exudates
- teritiary stage - cerebrell and neurological alteration

GONORRHOEA

+ Neisseria gonorrhoea (gram-ve bacteria).
gonococcal Species - Causative agent.

- Gonorrhoea is a sexual transmitted diseases which will affect the endothelium membrane & is manifested as urethritis, cervicitis & Conjunctivitis.

Etiology -

Neisseria gonorrhoea which is gram-ve bacteria

Pathophysiology

29/12/23

Neisseria gonorrhoea
↓
entering into the urogenital tract
↓
in order to survive, it compete with the residual bacteria in Urogenital tract
⇓
it gets nutrition from the Urogenital tract.
⇓ cell components
produces peptidoglycogone, ~~lipid phosph~~ Lipopolysaccharides (LPS), Membrane, secreting vesicles to get strengthen
⇓
Adhesion into the UGT [endothelial / epithelial cells]
⇓
influx of the Neutrophils.
↓
Relea starts inflammatory process
↓
release in the cell exudates
↓
it is helps in next transmission by damage of different organs.

Clinical Manifestations :-

- Itching
- Paining
- Redness
- Swelling
- Burning micturation.

Diagnostic Evalution:

- swap test - ruling out of the organism involved.
- drugs against
- Gram -ve bacteria

HIV :

- Sexually transmitted virus disease.
- It is a single stranded virus. (RNA virus)

HIV
↓
(it contain genetic Material) ← CD4 cells (Helper Cells) → selective tropism
↓
integration of viral genetic Material

with the host of genetic material.

↓

Replication & synthesis of viral DNA

↓

Attacking the Multiple CD_4 Helper cells

↓

Immune Supressor System activates

↓

immuno comprimizatn

↓

Disease possible infection

Disruption of membrane receptors that identify antigens (Ag)

↓

No antibody (ab) product^n

↓

cell death

↓

(AIDS) infections.

- HIV is a single stranded RNA virus which causes Aids.

Etiology

+ve agent - HIV.

Causes other infected prsn.

Patho :-

HIV · virus gets entry into the host immune cells by selective prog tropism and gets integrated with the host cell genetic material and forms / produces viral DNA.

→ Latent stn: the viral DNA gets replicated & starts a next attack to the rest of the CD_4 helper cells.

→ The process of immuno comprimization by the HIV is seen in two different mechanism.

(i) Synthesis of immune Suppressor System activates (A^+).

(ii) Disrupt the membrane receptors that recognises the surface antigens.

by these processess there is complete immuno compromised & no antibody product^n which leads to Aids & possible infections.

clinical

- frequent infection
- fever
- loss of weight
- Aepitits ~~Appitis~~ ~~Appetite~~
- Melige
- Fatigue.

Diagnostic evaluation

- ELISA
- Antivirus drugs like ~~Zobodine~~, ~~Entecavir~~, ~~Zudovidine~~.
(Anti-retro viral)

Entecavir, zidovudine (ZDV)

Urinary Tract Infections (UTI) 02/01/2024

- UTI is defined as a possible grp of syndromes which clinically manifested as bacteruria, pyuria, Pyelonephritis [Inflammation of nephrons].

Etiology

- E.coli
- Enterobacter
- ~~Streptococci~~ ecus
- ~~Staphylococcus aureus~~ Strephlyococcus aureus
- Pseudomonas ausiginosa
- Klebsella pneumoniae.
- St. Mirabilies.
- St. pyogenes.

→ A healthy prsns will have a count of bacteria which is very limited and also mostly it includes resident bacteria called colonial bacteria

- A prsn with UTI will have a thousand bacteria per/ml of urine.

* Types of UTI based on site of infection -

- Acute
- Chronic
- Urithcritis
- Cystitis
- Ureteritis
- Nephritis

- Types of UTI based on severity -
  - Acute - restricted to urethra, urinary bladder
  - Chronic - ureter & functional organ of kidney.

- this severity base infection can be manifested in three ways.
  - Ascending
  - descending
  - lymphatic type infection

- prostatis - mostly seen in mens.
Acystitis - mostly seen in womens.

Pathop:

Invasion/Entry of Bacteria into the urinary tract

↓

Adhering to the mucosal epithelial membrane.

↓

Colonization of the bacteria

↓

Activation / Release the of cytokines, prosta (fever)

- glanding of Glycopeptides [membrane damage]

↓

Fever | Inflammation ↓ Swelling of the urinary tract ↓ Obstruction to the flow of urine ↓ incomplete voiding ↓ Urgency to urine | Lining of the UT membrane gets damaged

- Dysuria - pain while urinating
- Burning Micturition

Virulence defence — pili — fimbrae

Host defence Mechanism — Glycosamine glycan

- finbrae - low pH, concn of urine; Urea ↑es.

Virulence mechanism	Host defence Mechanism
* adhering the surface pili or finbrae	* low pH
* production of cell wall components like peptidoglycan layer.	* conc^n urine → increased
* resisting the washing out action done by glycosamine -glycan	* urea contained presence of Glycosamine -glycan.
* production of hemolycins.	* Activated Immune system

Signs of Symptoms -

- Burning Miturition (Burning sensation during urinating).
- Dysuria (Pain while urinating)
- pyuria (pus in urine)
- inflammation
- Itching
- Irritation
- Hyper tension
- Hemoturia (blood in urine).

Pneumonia

03/01/2024

- Acute and chronic respiratory infection are common to all age grp ppl.
- This respiratory tract infection may some times leads to lyf threatening condn.

definations:

pneumonia is difined as inflammation to the lung parenchyma from the distal to the terminal bronchioles

- The mode of entry of the pathogens into the air way tract is seen four different types.
  - Inhalation
  - Aspiration
  - Hematogenous
  - Direct contact

1. Inhalation - The microbes can entered through the air i.e., inhaled

2. Aspiration: the contents from the oropharengial region can move towards the air way tract

3. Hematogenous: The microbes can entered to the systemic circulation.

4. Direct contact: Direct contact with the infected persons.

→ Respiratory tract is provided with a defence mechanism by different ways -

1. oro-pharengial defence mechanism and Nasopharengial defence mech.
2. Muco-ciliary (M. ciliary) defence mech.
3. phagocytosis by the Macrophages & Immuno-globuling (Ig) in the alveolar region.

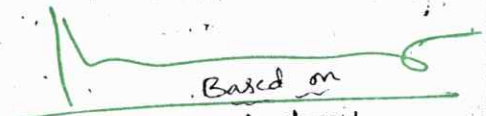
→ The disturbance in the defence mechanism is seen in different types -

1. Unconsciousness
2. Immobile ciliary mm
3. Inappropriate activation of immune system
4. Obstruction of endo-tracheal region.
5. Inappropriate glotis reflexus.

08/1/24

Types of Pneumonia:-

clinical classification:-



Based on Etiology

→ Bacterial Pneumonia
- Viral Pneumonia

Based on Anatomy

- Lobar pneumonia
- Lobular Pneumonia/Bronchopneumonia
- Interstitial Pneumonia

- Bacterial
  - lobar
  - lobular / Broncho Pneumonia
- Others.
  - → Pneumococcal pneumonia.
  - → Pseudomonas Pneumonia
  - → Klebsella Pneumonia
  - → Hemophyllus Pneumonia.

Bacterial Pneumonia :-

→ These type of Pneumonia will effect a single lobe or multiple lobes with a bacterial entry.

(i) Lobar Pneumonia :

→ In these a part of lobe a two (or) more lobes are involved.

1) Pneumocystis Pneumonia
2) Staphylococcal Pneumonia.
3) strepto coccal Pneumonia
4) Others

Pneumocystis Pneumonia :-

→ These type is involved with the entry of a bacteria which is Strepto Coccals species.

Staphylococcal Pneumonia :

→ The causative agent involve is staphylococcal aureus.

Strepto coccal Pneumonia :

The agent involved in strepto coccal Pneumonia which focuses on Hematogenus mode of infection.

Others :

* this type is involving agents like hemophilus influenzae.
* Klebsella pneumonia species which occurs after an episodes Measels and Mumps.

Morphological changes :-

M. Changes can be seen in 4 different stages.--

- stage 1 - stage of congestion / initial stage.
- 2 - Red Hepatisation / Early consolidation.
- 3 - Grey Hepatisation / late consolidation
- 4 - Resolution

I. stage of congestion / Initial stage :

This phase can be seen at the initial days like 1st (or) 2nd day.

→ They will be Histological changes

- Macrophages can be seen.
- Neutrophils can be seen.
- presence of minimal no of RBC

→ Secretion of the fibrin can be seen.

- a cut piece of the lung will show some fluid exudates and presence of neutrophils and macrophages with engulfed Pathogens.

9/1/24

II. Red Hepatisation: [span - 2-4 days]

- At these stage the appearance of the lung will be just as the ~~liver~~ liver
- It is red in colour and can seen massive alveolar patches and also fibrin, neutrophils and Red cells.
- The texture of the lung is slightly harden

III. Grey Hepatisation:

- At this stage the red hepatisation preceeds to grey in colour which again hardens it texture becz of the fibrin.
- The count of the neutrophils are slightly decreased.
- Massive alveolar hardening can be seen and this entire process takes places in the span of 5-8 days.

IV. Resolutions:

This stage can be seen if the chemotherapy was not initiated. once if the antibiotic therapy was started the resolution phase can be appear with in 3 days.

- The fibrin will be liquified with some enzymatic rxn. which allows a nrml air spaces.
- massive, opaqueness can be seen in the lungs
- Traces of neutrophils can also be seen
- 12-15 days [span].

Lobular Pneumonia: / Bronchi

- This type of Pneumonia will affect the terminal part of the bronchioles and extends upto alveoli. The pathogen will involve in the
- streptococcali, klebsiella, gram-ve bacilli like Pseudomonas.

Morphological changes:

- Opaqueness (consolidation)
- massive hardening of lungs
- fluid exudates (edema)

- A cut piece when touched can have exudation of the fluid

Clinical features:-

Lobar	Lobular / broncho
* Fever, chills, dyspnea, Tachycardia, Tachythmia, Tachypena	* Bed ridden illness, fatique, Tachy-cardia, SOB etc.
* physical finding - SOB, restlessness, fatique.	*. The secretion can be pulled towards the lobes lobules
* Radiological findings - X-ray - consolidatim of lungs can be seen	* consolidation can be seen a multiple lobules.

10/1/2024

Viral Pneumonia:

this is also known Mycoplasma Pneumonia (or) primary Atypical Pneumonia.

- This type involves more of the interstitium of lungs. These is no fluid exudates from the alveoli so the name Atypical Pneumonia was given.

- Ethiology:
  - adenovirus
  - Adenovirus
  - Rhinovirus
  - cytomegalovirus

Morphological changes:

→ Massive hardness of the interstitial of lung inflammation alveoli will be pinkish red colour.

11/1/2024

→ Histological changes

The histological changes that can be notice are-

- interstitial inflammation
- Nicrotising cells
- Reactive agents

* When a cut section of lung was touched it gives a froathy blood is oozing.

clinical manifestation:

SOB
Dysnia Dyspnea
cough with expectoration
Tachythmia Tachypenia
Tachycardia

• chills
• fever etc.

Others :

Pneumocystis Carinii Pneumonia

- PCP which is prsnt in air, when inhaled 50% of ppl will get effected with this type of infection
- It is manifested as hardening and the presence of (oozening) edematous fluid, fever, chills and cough extra symptoms can be seen.

Pseudmonas Pneumonia: / Legionella

- This is the aquatic type of gram-ve bacilli gets entry by drinking contaminated water. This is again clinically manifested with pneumonia symptoms.

Hypostatic Pneumonia

This type of Pneumonia can be seen in ppl who are unconscious, comatose and bed-ridden.

There is an entry of pathogen and leads to inflammation of the lung and other Pneumonia Symptoms.

Aeration Pneumonia :

Aspiration

- This type of Pneumonia is occured because of reflux of gastric contents into the lungs becoz of the presence of bacteria in the GIT is shifted to air way tract and causes Pneumonia

type of

Lipid Pneumonia :

This is of two types -

1. Exogenous lipid Pneumonia:

- Becoz of intake of lipid / oil contained food (or) drugs like liquid Paraffin will cause deposition and entry of lipid into the air way tract.

2. Endogenous lipid pneumonia :

- Becoz of the tissue breakdown the metabolised lyk lipids will cause a symptoms of Pneumonia.

Complications :

Pleural effusion cardiac problems lyk.

endocarditis, myocarditis & metastatic infections which somes called Hepatic dysfunction.

12/1/2024

Typhoid

→ Also known as Enteric fever.

→ A Typhoid fever causes acute illness which will effect the GIT and if left untreated leads to death.

Etiology

Salmonella typhi
Salmonella paratyphi

epidemiology:

- Across 1.6 million affected person, 6 lakhs persons are death

→ it is gram-ve bacteria.

Transmission

→ faecal-oral route.

→ The organism will be entering into other person if they have close direct contact.

→ Infected human will be carrier for other healthy person in getting the infection.

→ Through flies & cockroaches.

Pathophysiology:

Ingestion of contaminated b+ food & H_2O

↓

ingestion & invading of pathogen into the small intestine

↓

entry of pathogen into lymphatic systems

↓

In LS of organism gets matured & multiplied (incubator period)

↓

later enters into blood stream

↓

with the help of enterocytes & M-cells the pathogens & get multiplied.

↓

Enters into Gall bladder, intestine & tissues of lymphs.

↓

Got out in the form of feces.

Clinical Manifestation:

Early 1st week - Increase in temp, abdominal pain, constipation.

End of 1st week - progression of the fever, malise, [weakness], Abdominal tenderness

early 2nd week → Raised temperature, brady cardia, weight loss

End 2nd week → myalgia

early 3rd week → progression of 1st week

Symptoms

End 3rd → Prognosis of disease will be bad & leads to chronic phase & person will be a strng carrier.

Diagnosis:

1. widal test (Blood test) for ruling out the micro-organism involved.
2. Agglutinin test
3. Liver function test
4. Neurological manifestation

Treatment

Antipyretic

- Chloramphenicol

Floroquinonols.

18/1/24

Tuberculosis

Tuberculosis is a disease that not only affect lungs but also kidneys, spine and brain.

Epidemology:

The disease distribution is high in countries like USA, which have more no of reported cases.

- There is no Gender predominance.
- Age grp of 45-455 are affected with TB. with a minimal extend where as age of 55-65 have more reported cases.

Etiology:

Causative agent by Mycobacterium tuberculosis / tuberculi, Mycobacterium bovis; Mycobac. africanum, mycobac. Leprae (leprosy).

→* The bacteria will grow in the places where there is high oxygen tension like Apices of lungs and the parenchyma of kidneys.

→factors that contribute for transmission of TB.

- length of time exposed to the environment
- No. of organisms already prsnt.
- Depends on the host immune system
- Direct contact

* Mycobacterium Tuberculosis is an acid-fast bacilli which replicates for every 24 hrs.
* Each droplet contains around 2-3 micro organism

Pathophysiology: 22/1/24

- The pathophysiology of TB was studied in 3 diff stages -

- The immune phase
- 1° infection
- Reactivation phase.

Immune phase / Immune responses.

→ T-lymphocytes play a major role in activating immune systems for the cond? lyk. TB.

T-Helper-1, TH_2 also play a major role, of this TH_1 contribute very much.

→ Macrophages also play major role in invading the bacilli. Once the macrophages engulf the bacilli there is no infection but there are some immature macrophages though they engulf the bacteria they can't kill it but instead they prsnt the bact. bacili on the cell membranes.

→ Later on CD_4 marker cells are activated & produce cytokines like Interferon (IF)-γ, Interleukin-4, IL-10.

How the immune systems fails:

- There is an inhibition of binding of lysozomes & phagozomes which in term can produce enzyme to invade the bacilli.
- Lipoarabinomann is the bacteria cell wall component which will destroy the cell membrane of Host.
- production of cytokines which will form/convert macrophages to the immature macrophages.
- Scavenging of the O_2 where it leads to binding of O_2 & H_2 to form peroxides.

Primary Infection:

The bacilli will be entering into the host which is a air borne way where the bacilli gets engulfed by the macrophages at alveoli.

→ If the macrophages are killing the bacilli then there is no infection at all. failing to do so the bacilli will completely enters into the deeper parts of lungs lyk Hilum mediastinum. midastinal regial etc and also deep In vasculature.

→ later it chose a favourable cond^n where there is high O_2 tension lyk apexes of lungs & parenchyma of kidneys.

→ with in 2 weeks the T lymphocytes gets activated and can become bactericidal and surround the region with necrotizing material and forms a granuloma.

→ later some hypersensitivity rxn also takes places. after 2 months when was skin test was perform it will show a positive skin test with the prsnt of CD_4 cells & radiographyic evaluation points the granuloma which indicates the infection.

Reactivation phase:

This phase can be seen to 10% of ppl who are having very weak immune systems. At this stage the granulomass breakout and get liquified, spread the necrotizing material to the entire lungs where the infection is very high.

clinical Manifestation:

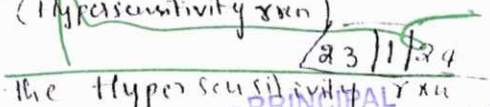
- cough
- night sweats
- SOB
- potts spine
- fatigue
- loss body weight
- Pyrexia
- Hematuria
- fever

→ the people who are co-infected with HIV there is a markable decreasing CD_4 cell count

Diagnostic evaluation:

Skin Mantoux test

- Mantoux test is the only diagnostic evalution which will be a positive skin test for TB Patient. It is done with a chemical lyk a Tuberculin purified protein derivative. This came in a injection of I, II where it gives out its rxn in 5-6 hrs.



- The induration of the hypersensitivity rxn of in patient infected with TB will be

5mm, 10mm & 15mm.

- If ≤ ≥ 5mm - Patient with T.B.
  ≥ 10mm, 15mm - T.B, Diabetes Milletus.
  ≤≥ 15mm - Serious illness & co-infection.

- Microscopy of Sputum, Sputum culture test PCR, chest radiography - other diagnostic Tat

Treatment:

- The drugs that are given for TB -

- ISONIAZIDE -H
- RIFAMPICIN -R
- PYRAZINAMIDE -Z
- ETAMBUTOL -E

included in cat-I, II, III, IV

Other drugs lyk -

- Streptomycin (500mg + 150)
- Kanamycin
- Neomycin
- Para Amminosalicylic Ac (PASA)

- dose streptomycin dose need to be adjusted acc. to weight (BMI)
- In case of pregnancy. there is inclusion of PASA,.
- In the case of renal failure patient the dose of Streptomycin dose is need to be adjusted.
- In case of pregnant women who effected with TB, the doctor will advice abortion abborsion at 1st trimester and a women with more 20 weeks the doctor will give advice with the drug PASA, kanamycin, Neomycin.

Malaria

24/1/24

- Malaria is a protozoal infection caused by the "female anopheles mosquito"

- Etiology
  plasmodium vivax.
  plasmodium Malariae.
  plasmodium ovale.
  plasmodium falciparum

- Of these four type P. falciparum will cause a harmful Malignant type of Malaria. where as, the other three will be a benign type.

25/1/24

Pathogenesis:

- When a female anopheles mosquito bites a person with already infected with plasmodiums where it undergoes a asexual phase in which a series of transformation like Hypnozoides, immature Sporozoites are form.

- When a female mosquito bites a person with already done asexual phase of protozoan, the immature sporozoides will convert into mature sporozoites and later on to Schizonts. This phase is sexual phase which is done in mosquito. when this female mosquito with already done sexual phase of protozoan, for its food and for reproduction bites a healthy human being then the infection is transmitted.

Clinical Manifestation:

Initial phase:

- Fever,
- Chills
- rigors
- Vomiting etc.

Second phase

Arethrocytic phase:

Hot phase

Cold phase

* Hot phase - High grade temp. 104°F
  electrolyte imbalance

* cold phase - cyanosis (discolouration)
  Sweating
  Nausea
  Vomiting
  heading

Complications:

Cardiac anurysm

Pulmonary edema

orthostatic Hypotension

Treatment:

- Artesunate
- chloroquine.
- MefLoquine.

♀ anopheles mosquito

↓

inno culated ē the Sporozite

↓

entry of Sporozoites into the blood stream

↓

Hepato cytes — Schizonts

dormant Hypnozoites (3-5).

↓

Merozoites (48-72 hrs - 24-36 with erythro infected)

Gametocyte ♂ Gametocytes } Sexual phase.

Ookinites

↓

Transmitted to a

Person and get infected

Code No: PH201

JAWAHARLAL NEHRU TECHNOLOGICAL UNIVERSITY HYDERABAD
Pharm.D II Year Regular/Supply Examinations, October - 2020
PATHOPHYSIOLOGY

Time: 2 hours **Max.Marks:70**

Answer any five questions
All questions carry equal marks

\- - -

1.a) Define Cell injury and write the pathogenesis of cell injury.
b) Write the morphology of cell injury. [10+4]

2.a) List any five different mediators of inflammation and their respective actions in the inflammatory process.
b) Write the pathogenesis of acute inflammation.
c) List the factors influencing healing of wounds. [5+5+4]

3.a) Write a note on major histocompatibility complex (MHC) proteins.
b) Explain different types of hypersensitivity reactions with examples.
c) Write the pathogenesis of amyloidosis. [4+5+5]

4.a) Write the etiology and pathogenesis of cancer.
b) Write the biological effects of radiation. [10+4]

5.a) Write the different types of shock, stages and management of shock.
b) List the various constituents of cigarette smoke and write the ill-effects of smoking. [10+4]

6.a) Write the etiology, pathophysiology and symptoms of hypertension.
b) Define angina pectoris and explain briefly the different types of angina. [10+4]

7.a) Write the risk factors and pathophysiology of type 2 diabetes mellitus.
b) Write the etiology and signs and symptoms of asthma. [8+6]

8.a) Write the pathogenesis of urinary tract infections.
b) Write the causative organism and signs and symptoms for the following:
i) Tuberculosis ii) AIDS iii) Typhoid iv) Pneumonia v) Bacterial dysentery. [4+10]

---ooOoo---

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II ND YEAR-I MID INTERNAL EXAMINATIONS

Subject: PHATHOPHYSIOLOGY

TIME: 2 hr marks:30

MID-1

1.PATHOGENSIS OF ACUTE INFLAMMATION

2.MORPHOLOGY OF IRREVERSIBLE CELL INJURY

3.BRIEF ABOUT MANAGEMENT OF BONE HEALING

4.BRIEF TYPES OF INFLAMMATION

5.EXPLAIN ABOUT ABNORMALITIES OF CELL INJURY

6.EXPLAIN ABOUT CELL MEDIATE IMMUNITY & HEMORAL IMMUNITY

7. WRITE A BRIEF NOTE ON AUTO IMMUNE DISEASE EXPLAIN ABOUT AIDS

8.DESCRIBE ABOUT DIFFERENT TYPES OF TRANSPLANTATION REJECTION

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AVANTHI INSTITUE OF PHARMACEUTICAL SCIENCES

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II-ND - YEAR II – Mid internal Examination

Subject:PHATHOPHYSIOLOGY

TIME: 2 hr marks: 30

MID-2

1.EXPLAIN IN DETAIL ABOUT PHATHOGENSIS OF CANCER

2.EXPALIN VARIOUS TYPES OF SHOCK & THEIR STAGES & MANAGEMENT

3.DESCRIBE IN DETAIL ABOUT VARIOUS ENIVERNOMENTAL DISEASES

4.WRITE A BRIEF NOTE ON (A)_PATHOGENISIS OF PARKINSONISM

(B)PATHOGENSIS OF COPD

5.EXPALIN IN DETAIL ABOUT PATHOGENSIS OF DIABETES MELLITEUS

6.WHAT IS INSFECTIONEOUS DISEASES,EXPLAIN ABOUT VARIOUS SEXUAL TRANSMITTEED DISEASES

7. WHAT IS ANGINA,EXPLAIN PATHOPHYSIOLOGY OF MYOCARDIAL INFRACTION

8.EXPLAIN ABOUT (A)BIOLOGICAL EFFECTS OF RADIATION

(B)PEPTICAL ULCER DISEASE.

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NAAC NATIONAL ASSESSMENT AND ACCREDITATION COUNCIL B++ GRADE

INTERNAL DISCRIPTIVE EXAM

PCI APPROVED

NAME : U. Raviteja

DATE: 03/02/20

ROLL No.: 19N01A0012

Subject: pathology

CLASS : II year mid-I SEM

SIGNATURE OF THE INVIGILATOR'S:

SIGNATURE OF THE STUDENT U Raviteja

TOTAL MARKS 28/30

1) Ans:

Acute Inflamation :-

Acute inflamation has a rapid onset of minutes or hours, usually resolves in a few days, has classic signs and symptoms, and has cellular infiltrate primarily composed of neutrophils. The erythema seen in acute inflamation results from increased blood flow to the affected area due to vasodilation.

Acute inflamation starts after a specific injury that will cause soluble mediators like cytokines, acute phase proteins and chemokines to promote the migration of neutrophils and macrophages to the area of inflamation. These cells are part of natural innate immunity that can take an active role in acute inflamation. If this inflamation does not resolve after six weeks, this will cause the acute inflamation to develop from subacute to the chronic form of inflamation.

* Causes:-

The causes or inducers of inflamation can classify into two main groups:- exogenous & endogenous inducers.

1. Exogenous inducers:-

This grouping can further subdivide into two classes main groups:- microbial & non-microbial exogenous inducers.

A. Microbial inducers:-

There are two classes of microbial inducers. The first class is pathogen associated molecular Patterns (PMPs) which are carried by all microorganisms

B. Non-microbial :-

Causes include allergens, toxic compounds, irritants, and foreign bodies that are too large to be digested or cause phagosomal damage in macrophages.

2. Endogenous

There are signs released by tissues that are either dead damaged, malfunctioned, or stressed.

2] Morphology of Irriversable cell injury:-

Necrosis is a type of irreversible cell injury characterized by cytoplasmic swelling, damage to the plasma membrane and organelle destruction. All of which Causes cell death.

Irreversible cell injury can be recognized by changes in the appearance of the nucleus and rupture of the cell membrane

The phenomena consistently characterize irreversible injury. The first is the inability to reverse mitochondrial dysfunction (lack of oxidative phosphorylation and ATP generation) even upon restoration of oxygen; the second is the development of profound disturbances in membrane function.

Hall mark of irreversible cell injury = membrane Damage.

There is a decrease in ATP because the cell is not receiving enough blood (oxygen and glucose). This decrease in ATP triggers the cascade that leads to cellular Swelling. The morphologic hallmarks of apoptosis include chromatic margination, nuclear condensation and fragmentation, and condensation of the cell with preservation of organelles.

Pathogenesis:-

This disorder in phospholipid metabolism is felt to be the critical lesion that produces irreversible cell injury in ischemia. It affects the endoplasmic and sarcoplasmic reticular membranes of liver and myocardial cells, respectively and probably the plasma membrane of both.

morphological, cell death can be classified into four different forms: Apoptosis, autophagy, necrosis and ~~ptosis~~. Apotosis, a type of cell death, is the fastest form of cell death and occurs depending on the caspase proteolytic cascade.

3) **Bone Healing:**

It is an intramembraneous bone healing that occurs through Haversian remodeling. The other type is secondary bone healing which occurs in non-rigid fixation modalities such as braces, external fixation, plates in bridging mode, intramedualry nailing...etc.. There are 2 main modes of bone healing; Primary bone healing is dictated by absolute stability Constructs that achieve a mechanical strain below 2%.. These fixation modalities achieve a mechanical strain b/w 2-10%. And it occurs via endochondral bone healing. Bone healing can involve a combination of primary & secondary Proces based on the stability throughout the constuct.

Bone fracture healing; is an intricate and fluent regenerative process that aims at restoring the damaged bone to its pre-injury state and cellular composition.

A fracture is a breach in the structural continuity of bone cortex, with a degree of injury to the surrounding soft tissues. following the fracture, secondary healing begins which consists of four steps.

1. Hematoma formation
2. Granulation tissue formation
3. Bony cells formation
4. Bone remodeling.

The type of fracture healing is governed by the achieved mechanical stability at the fracture site and, consequently, the strain. An appropriate mechanical stimulation such a strain, facilities tissues formation at the bony ends. The amount of the involved strain dictates the biological behavior of the cells involved in the healing process and, consequently, the type of bone healing.

4) Brief about inflamation :-

There are two types of inflamation: acute & chronic People are most familiar with acute inflamation. This is the redness, warmth, swelling, and pain around tissues and joints that occurs in response to an injury, like when you cut yourself. When the body is injured, your immune system releases white blood cells to surround & protect the area.

"Acute inflamation is how your body fights infections and helps speed up the healing process," says Dr. Shmerling, "In this way, inflammation is good because it protects the body." This process works the same if you have a virus like a cold or the flu. In contrast, when inflamation gets turned up too high and lingers for a long time, and the immune system continues to pump out white blood cells and chemical messengers that prolong the process, that's known as chronic inflamation.

"From the body's perspective, it's under consistent attack, so the immune system keeps fighting identinitely" says Dr. Shmerling.

When this happens, white blood cells may end up attacking nearby healthy tissues and organs!

5) Abnormalities of Cell injury:-

Cell damage (also known as cell injury) is a variety of changes of stress that a cell suffer due to external as well as internal environmental changes. Amongst other causes, this can be due to physical, chemical, infectious, biological, nutritional (or) immunological factors. Cell damage can be reversible or irreversible. Depending on the extent of injury, the cellular response may be adaptive and where possible, homeostasis is restored

Cell death occurs when the severity of the injury exceeds the cell's ability to repair itself. Cell death is relative to both the length of exposure to a harmful stimulus and the severity of the damage caused. Cell death may occur by necrosis (or) apoptosis.

* The 4 types of cell injury are
* Cell injury

* Apoptosis
* Necrosis
* Infarcts
* free radical injury.

Atrophy, Hypertrophy, Hyperplasia, metaplasia, and Dysplasia are all cellular adaptations to the demands of function or the effects from environmental stimuli (or) damaging diseases. All cells die – they are pre programmed genetically to do this (what is called "apoptosis")

8) Different types of transplantation rejections :-

Transplant rejection can be classified as hyperacute, acute, or chronic. Hyperacute rejection is usually caused by specific antibodies against the graft and occurs with in minutes or hours after grafting.

* There are 3 types of ~~of~~ transplant rejections :-

1) * Hyperacute rejection occurs a few minutes after the transplant when the antigens are completely unmatched.

2) * Acute rejection may occur any time from the first week after the transplant to 3 months afterward.

3) * chronic rejection can take place ~~over many years~~.

=> Acute rejection occurs days (or) weeks after transplantation and can be caused by specific lymphocytes in the recipient that recognize human leukocyte antigens in the

tissues (or) organ grafted. Finally, chronic rejections usually occurs months (or) years after organ (or) tissue transplantation. Various mechanisms involving chronic inflammation, humoral, and cellular immune reactions play essential roles in the immunopathogenesis of chronic rejections.

This activity reviews the evaluation and managment of chronic transplant rejections and highlights the role of interprofessional team members in collaborating to provide well-coordinated care and enhance outcome for affected patients.

Objectives:-

⇒ Identify the pathophysiology of chronic transplant rejection.

⇒ Describe the typical presentation of a patient with chronic transplant rejection.

⇒ Explain how to manage chronic transplant rejection

⇒ Explain the importance of improving coordination among the interprofessional team to enhance care for patients affected by chronic transplant rejection.

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NAAC NATIONAL ASSESSMENT AND ACCREDITATION COUNCIL B++ GRADE

INTERNAL DISCRIPTIVE EXAM

PCI APPROVED

NAME : V. priyanka

DATE: 23/11/2019

ROLL No.: 19N1T0022

Subject: pathophysiology

CLASS : IV year SEM. mid-II

SIGNATURE OF THE INVIGILATOR'S:

SIGNATURE OF THE STUDENT V. priyanka

TOTAL MARKS 24/30

(1) Pathogenesis of cancer is a pathophysiological understanding of how molecular and cellular events play a casual role in transforming tumors from benign to malignant state.

This often involves genetic, epigenetic, proteomic and metabolic alternations within solid tumors and blood cells.

Cancer can be viewed as the result of a succession of genetic changes during which a normal cell is transformed into a malignant one. One of the essential changes in a cell that cause this malignant transformation is evasion of cell death is

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(*) staging groups.

* Stage 0 means there's no cancer, only abnormal cells with the potential to become cancer....

* Stage I means the cancer is small & only in one area...

* Stage II & III mean the cancer is larger & has grown into nearby tissues or lymph nodes.

* Stage IV means the cancer has spread to other parts of your body.

(8) Exposure to high very levels of radiation, such as being close to an atomic blast, can cause acute health effects such as skin burns and acute radiation syndrome.

(*) Biological effects of Radiation.

. Radiation can cause biological damages on either by direct and indirect action.

. If radiation falls on human body, it produces moving electrons

(A) These electrons causes ionization, excitation resulting in chemical and molecular changes.

(A) Radiation can also produce free radicals, which are unpaired electrons that are chemically reactive.

B) A sore that develops on the lining of the oesophagous stomach or small intestine

Ulcers occur when stomach acid damages the lining of the digestive tract. common causes include the bacteria H. pylori and anti-inflammatory pain relievers including aspirin.

* The most common cause of ulcers is infection of the stomach by bacteria called Helicobacter pylori (H pylori) most people with peptic ulcers have these bacteria living in their digestive tract. Yet, many people who have these bacteria in their stomach do not develop an ulcer.

* Peptic ulcers can heal if the conditions that caused them go away. But it usually takes a medical diagnosis to identify the cause.

(5) * It is characterised by elevated blood glucose levels or hyperglycemia, which results from abnormalities in either insulin secretion or insulin action or both.

* Hyperglycemia manifests in various forms with a varied presentation and results in carbohydrate, fat and protein metabolic dysfunctions

* aureus is one of the most isolated pathogens in diabetic patients.

* Causing meningitis, sepsis, bacteremia, skin infections, and nasal carriage.

* Diabetes mellitus is a metabolic disease, involving inappropriately elevated blood glucs levels.

* DM has several categories, including type 1, type 2, maturity-onset diabetes of the young (MODY).

* gestational diabetes, neonatal diabetes and secondary causes due to endocrinopathies, steriod use.

* The diagnosis of DM can be made in the following situations.

a) occasional plasma glycemia > 200 mg/dl (11.1 mmol/L) (obtained at any time of day and without regard to when food was last ingested) and symptoms of DM (polyuria, polydypsia & inexplicable weight loss); b) fasting plasma glycemia.

(3) Environmental disease refers to any pathologic process having a characteristic set of signs & symptoms that are detrimental to the well-being of the individual and are the consequence of external factors, including exposure to physical or chemicals agents, poor nutrition and social or cultural behaviours.

* NCDs (notably heart disease, cancer & chronic respiratory diseases) are among the most prominent environmental diseases and are subsequently among the leading causes of death.

* Environmental hazards - like water & air pollution, extreme weather, or chemical exposures.

* - can effect human health in a number of ways, from contributing to chronic diseases like cancer or to acute illness like heat exhaustion.

(*) Environmental health examined the interaction between the environment and our health.

(6) Sexually transmitted diseases (STDs) are caused by sexually transmitted infections (STI). They are spread mainly by sexual contact. STIs are caused by bacteria, viruses or parasites. A sexually transmitted infection may pass from person to person in blood, semen, or vaginal and other body fluids

(*) Bacterial vaginosis, BV is a common, treatable vaginal condition which can increase your chance of getting an STD.

(*) Chlamydia.

(*) Gonorrhea

(*) Hepatitis

(*) Herpes

(*) HIV/AIDS & STD

(*) (HPV) Human papillomavirus

(*) Mycoplasma genitalium (mgen)

(1) * Sexually transmitted infections (STI)s are infections transmitted from person to person through sexual contact.

(2) * There are several types of shock : Septic shock caused by bacteria, anaphylactic shock caused by hypersensitivity or allergic reaction cardiogenic shock from heart damage, hypovolemic shock from blood & fluid loss, and neurogenic shock from spinal cord trauma.

Treatment for shock depends on the cause.

* The main types of shock are hypovolemic, Cardiogenic, & distributive shock.

* shock must be managed rapidly by identifying & treating acute, reversible causes, restoring intravascular Volume.

* infusing vasoactive drugs: using mechanical adjuncts when applicable, & treating vital functions until recovery.

(3) * It covers the four stages of shock. They include the initial stage, the compensatory stage, the progressive stage, & the refractory stage.

PATHOPHYSIOLOGY ASSIGNMENT - I

Name : P sai Teja

Roll NO : 17h51T0019

class : pharm - D II year

Topic : Diagnosis of pathology

Submitted to : Dr. Ravi prakash

5/5

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DIAGNOSIS OF PATHOLOGY

* Diagnostic Pathology :-

It identifies the causes of diseases based on morphological and/or clinical Pathology findings, as well as history, clinical signs, and ancillary test results.

It is important in all areas of Pathology, both in all areas of pathology, both in spontaneous and in experimentally induced disease.

Diagnostic Pathology test :-

Pathology means the study of diseases and its causes and Progression. Pathology tests cover blood tests, and tests on urine, stools (faeces) and bodily tissues. If you're sick, many of the decisions about your care will be based on the results of your blood and Pathology tests.

* Methods of diagnostic Pathology:-

Necroscopy, radiography, urinalysis, microscopic examination of tissues, haematological tests and anatomical Pathology are the different techniques used in Pathology, Diagnostic microbiology is another technique where microorganisms are isolated, cultured and results are interpreted.

Types of medical diagnosis

- Provisional: Based on physical exam and clinical findings.

* Histopathological:- Done by a Pathologist after examing sample tissue under a microscope.

* Final :- Done based on Provisional diagnosis and investigation.

* Post Pathology diagnosis :-

It is based on the combined result of the biopsy, gross examination, Processing, and microscopic examination.

There is a general format for diagnoses :- The organ or tissue biopsied. sepecific part of the organ or body where the sample came from. The biopsy Procedure.

* The four types of nursing diagnosis are :-

* problem- Focused Nursing Diagnosis
* Risk Nursing Diagnosis
* Health Promotion Diagnosis
* Syndrome Diagnosis
* Possible Nursing Diagnosis
* Etiology

Pathology diseases :-

Pathology (from the Greek word Pathologia, meaning the study of suffering) refers to the specialty of medical science concerned with the cause, development, structural /functional changes, and natural history associated with diseases.

Role of Pathologist in diagnosis:-

Pathologist are often called upon to grade tumors or to participate in their staging in order to estimate tumor prognosis. Tumor staging (eg. the well-known TNM system) has proved to be of great value in estimating prognosis

Pathology is the study of diseases. It is the bridge between science and medicine. It underpins every aspect of patient care, from diagnostic testing and treatment advice to using cutting-edge genetic technologies and preventing diseases.

* Five steps of Pathology:-

* History. Take a history....
* Examination. Do an examination..
* Investigation. These include blood and urine tests, and x-rays and scans...
* Diagnosis (initial or final).
* Diagnosis (final).

Pathology diagnosis and treatment :-

Pathologists are sent a sample of cells or tissues, which they record of their exam is called a Pathology report. Your care team uses this Pathology report to make a correct diagnosis. Working together, your care team and you will choose the best treatment plan for you.

* Pathology example :-

General pathology describes a complex and broad field that involves the study of mechanisms behind cell and tissue injury and understanding how the body responds to and repairs injury. Examples of areas that may be studied include necrosis, neoplasia, wound healing, inflamation and how cells adapt to injury.

* Dr. Rudolph how the father of Pathology.

* Importance of Diagnosis :-

Getting the right diagnosis is a key aspect of health Care, as it provides an explanation of a Patients health problem and informs subsequent health Care decisions (Holmboe and Durning). Diagnostic errors can lead to negative health outcomes, Psychological distress, and financial costs.

* How to write a Medical Diagnosis :-

1) 1Use standard Medical terminology throughout.
2) 2Take an inventory of the Patient's symptoms.
3) 3Read the Patient's medical history.
4) Examine the Patient and Perform diagnostic tests.
5) Create a working diagnosis
6) Rule out alternative Possibilites.

It is important in all areas of Pathology, both in spontaneous in experimentaly induced diseases.

* clinical diagnosis :-

clinical diagnosis. A diagnosis made on the basis of medical signs and reported symptoms, rather than diagnostic tests.

* Laboratory diagnosis :- A diagnosis based significantly on laboratory reports or test results, rather than the physical examination of the patient.

* Diagnostic tools detect Pathology

Diagnosis-aided tools include algorithms that assess one of the various histological features such as tumour grade, type, and extent. Accurate pathological diagnoses involve assessment and combination of multiple features by the trained human eye.

* Main Diagnosis :-

The primary diagnosis is the main condition treated or investigated during the relevant episode of healthcare. Where there is no definitive PATIENT DIAGNOSIS

* Advantages of Diagnosis:-

It makes things easier when communicating with professionals and once a person gets a diagnosis, it can help him/her to access some services, or it can also make finding information on specific problems easier.

* Diagnosis Report:-

A diagnostic report is the set of information that is typically provided by a diagnostic service when investigations are complete.

The information includes a mix of atomic results, text reports, images & codes.

PATHOPHYSIOLOGY * ASSIGNMENT - II

Name : V. priyanka

Roll No : 19HN1T0022

class : Pharm-D II year

Topic : Vitamin deficiency

Submitted to : Dr. Ravi prakash

5/5

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Vitamin Deficiency

Vitamin Deficiency is the condition of a long term lack of a vitamin. when caused by not enough vitamin intake it is classified as a primary deficiency, whereas when due to an underlying disorder such as malabsorption it is called a secondary deficiency. An underlying disorder can have 2 main causes :-

- Metabolic causes :- Genetic defects in enzyme (eg :- Kynurenirase) involved in the Kynurenine pathway of synthesis of niacin from tryptophan can lead to pellagra (niacin deficiency).
- Lifestyle Choices :- Life-style choices and habits that increase vitamin needs, such as smoking or drinking alcohol. Government guidelines on vitamin deficiencies advise certain intakes for healthy people, with specific value of women, men, bodies, children, the elderly and during pregnancy or breastfeeding. Many countries have mandated vitamin food fortification

Program to prevent Commonly occurring vitamin deficiencies.

→ Conversely, hypervitaminosis refers to Symptoms Caused by vitamin Intake in excess of needs, especially for fat-soluble vitamins that can accumulate in body tissue.

The history of the discovery of vitamin deficiencies Progressed over centuries from observation that certain condition - for example, Scurvy - could be prevented or treated with certain foods having high content of a necessary vitamin, to the identification and description of specific molecules essential for life and health. During the 20th century, several scientists were awarded the Nobel Prize in physiology or Medicine or the Nobel prize in chemistry for their roles in the discovery of vitamins.

* Food fortification is the process of adding micro-nutrients (essential trace elements and vitamins) to food as a public health policy which aims to reduce the number of people with dietary deficiencies within a population.

to which food. Vitamin fortification program Exist in one or more countries for folate, niacin, riboflavin thiamin, vitamin A, vitamin B_6, vitamin B_{12}, vitamin-D and vitamin E.

As of 21 December 2018, 81 Countries required food fortification with one or more vitamins. The most commonly fortified vitamin - as used 62 countries - is folate the most commonly fortified food is wheat flour.

A number of religion have published guidelines defining vitamin deficiencies and advising specific intake for healthy people, with different recommendations for women, men, infants the elderly, and during pregnancy and breast feeding including japan, the European union, the united States and Canada. These documents have been updated as research is Published in the US.

It's Important to maintain a balanced diet to get all the necessary nutrients, If you're Concerned It's best to Consult a healthcare professional for

Staple food of a region can lack particular nutrients due to the soil of the religion or from inherit inadequ-acy of a normal diet. Addition of micronutrients to staples and condiments can prevent large scale deficiency diseases in these cases.

As defind by the World Health Organization (WHO) and the food and agriculture organization of the united Nations (FAO), fortification refers to "the pratice of deliberately increasing the content of an essential micronutrient. i.e vitamins and minerals in a food irrespective of whether the nutrients were originally in the food before processing or not, so as to improve the nutrientional quality of the food supply and to Provide a public health benefit with minimal risk to health", whereas Enrichment is defined as 'synony mous with fortification and refers to the addition of micronutrients to a food which are lost conduct fortification programs and within lits all countries in the world that conduct fortification program, and within each country, what nutrients are added

There are different symptoms depending on the specific vitamin that's lacking. But some common symptoms include fatigue, weakness, hair loss, brittle nails, mouth ulcers, and poor wound healing. Remember it is always best to consult with a healthcare professional for a proper diagnosis.

Vitamin deficiency anemia is a lack of healthy red blood cells caused by lower than usual amounts of vitamins B-12 and folate. this can happen if you don't eat enough food containing vitamin B-12 and folate, or if your body has trouble absorbing & processing these vitamins.

Vitamin B_1 - deficiency may cause beriberi and dwarfism. vitamin B_2 - Deficiency can cause disorders in the digestive system, skin burning sensations and Cheilosis. vitamin B6 Deficiency of B6 causes convulsi-ons, Conjunctivitis and sometimes neurological disorder.

Vitamin D. vitamin D deficiency is especially common as we get older, since few food are naturally

rich in it...

Iron. Red blood cells, which carry oxygen throughout the body, rely on adequate iron stores.

A deficiency diseases can be defined as a diseases that is caused by the lack of essential nutrients or dietary elements such as vitamin and minerals in the human body.

vitamins and minerals are essential or bodily functions such as helping to fight injection, wound healing making our bones stong and regulating haemone. vitamins and minerals can cause toxicity if causmed in large amount.

To help prevent vitamin A deficiency, People should eat dark green leafy vegetables, yellow and orange fruit (such as papayas and oranges) carrots, and yellow vegetables (such as squash and pumpkin) Other food sources include milk and cereals that are fortified with vitamin A, liver, Egg, yolks and fish liver oils

Some vitamins causes acute or chronic toxicity, a condition called hypervitaminosis which occurs mainly for fat soluble vitamin if over-consumed by excessive supplementation. Hypervitaminosis A and hypervitaminosis D. are the most common example. Vitamin D toxicity does not result from sun exposure are consuming food rich in vitamin D, but rather from excessive intake of vitamin D supplement, possibly leading to hypercalcemia, nausea, weakness and kidney stones.

The united states, European union and japan among other countries, have established 'tolerable upper intake levels" for those vitamins which have documented toxicity.

Vitamins have diverse biochemical functions. vitamin A acts as a regulator of cell and tissue growth and differentiation, vitamin D provides a hormone-like function, regulating minerals metabolism for bones and other organs.

The B Complex vitamins function as Enzyme Cofactors or the precursors of them. Vitamins C and E function as antioxidants. Both deficient and Excess Intake of a vitamin can potentially cause clinically significand illness, although excess intake of water-Soluble vitamin is less likely to do so.

Department:		PHARM D							
Course Outcome Attainment - Internal Assessments									
Name of the Faculty:		Dr. RAVIPRAKASH		Academic Year:		2019-20			
Branch & Section:		PHARM D		Exam:		MID - II			
Course/Sub:		PATHO		Year/Semister:		II			
		CO2	C02	C02	C02	CO3	C03	C03	C03
Sl.No	Roll Number	Question No.							
		Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8
Maximum Marks		5	5	5	5	5	5	5	5
1	18GN1T0001	5		4	5	4		5	5
2	18GN1T0002		4	5	4	5		4	5
3	18GN1T0003		4	5	4	5	4	5	
4	18GN1T0004		4		4	5	5	5	4
5	18GN1T0005		5	5	5	5	4		3
6	18GN1T0006	5	3		5	5		4	5
7	18GN1T0007	4		4	5	5		4	5
8	18GN1T0008	4	5	5	4		5	5	
9	18GN1T0009	5	4	5	5	5	4		
10	18GN1T0010	4	5		5	3	5	5	
11	18GN1T0011			4	5	5	4	5	4
12	18GN1T0012			5	4	5	5	4	4
13	18GN1T0013	4	5	4	5		5		4
14	18GN1T0014	5	4	5	5	5	4		
15	18GN1T0015	5		4	5	5	4		4
16	18GN1T0017	4		4	5	5		4	5
17	18GN1T0018		5	4	5	4	4	5	
18	18GN1T0019	5		5	5	4	4		4
19	18GN1T0020		4	5		5	4	4	5
20	18GN1T0021		5	5		4	4	4	5
21	18GN1T0022		4	5	4	5	5	4	
22	18GN1T0023	5	5	4	5	4		4	
23	18GN1T0025	4	5	5	4	4	5		
24	18GN1T0026	5	5	4		5	4	5	4
25	17GN1T0020		4		5	4	5	4	
No. of students attempted		21	20	22	17	16	19	17	18
Max Marks Question wise		5	5	5	5	5	5	5	5
Target 50%		2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5
No. of Students above 50%		21	20	22	17	16	19	17	18
% of Students>50%		100.0	100	100	100.0	100.0	100	100	100.0
Attainment Level		3	3	3	3	3	3	3	3
Attainment table									
70-80%	1								
80-90%	2								
90-100%	3								

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Department:		PHARM D							
Course Outcome Attainment - Internal Assessments									
Name of the Faculty:		Dr. RAVIPRAKASH				2019-20			
Branch & Section:		PHARM D		Exam:		MID - I			
Course/Sub:		PATHO		Year/Semister:		II			
		CO1	C01	CO1	C01	CO1	C01	C02	C02
Sl.No	Roll Number	Question No.							
		Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8
Maximum Marks		5	5	5	5	5	5	5	5
1	18GN1T0001	5	4	4		5	5	4	
2	18GN1T0002	4	5	5	5	5		4	
3	18GN1T0003	5	4	5	5	5		4	
4	18GN1T0004		4	5	4		4	5	4
5	18GN1T0005	4		5	4	5	3	5	
6	18GN1T0006	5	5	4	4		4	5	
7	18GN1T0007	5	4	4		5	5		4
8	18GN1T0008	5	4	4		5	4		5
9	18GN1T0009	4	5	4		5	5		4
10	18GN1T0010	5	4		5	4	4		4
11	18GN1T0011	5	4	5	4	5		5	
12	18GN1T0012	4	5		5		4	5	5
13	18GN1T0013	5	5	5	4		5		3
14	18GN1T0014		4	5	5	4		5	5
15	18GN1T0015	5	4	5	5	4	4		
16	18GN1T0017	5	4	4	5		4		5
17	18GN1T0018		5	4	4	5		5	5
18	18GN1T0019	4		5	5		4	5	4
19	18GN1T0020	4	5	4		5	4		5
20	18GN1T0021		5	4		4	4	5	5
21	18GN1T0022	5	4	5			5	4	5
22	18GN1T0023	5	4	4		5		4	5
23	18GN1T0025	5		5	4		5	4	5
24	18GN1T0026	4		4	5		5	5	4
25	17GN1T0020	4			5	4	5	4	5
No. of students attempted		21	20	22	17	16	19	17	18
Max Marks Question wise		5	5	5	5	5	5	5	5
Target 50%		2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5
No.ofStudentsabove50%		21	20	22	17	16	19	17	18
% of Students>50%		100.0	100	100	100.0	100.0	100	100	100.0
Attainment Level		3	3	3	3	3	3	3	3

Attainment table									
70-80%	1								
80-90%	2								
90-100%	3								

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Course Outcome Attainment - Internal Assessments

Name of the Faculty:		Dr. RAVIPRAKASH		Academic Year:		2019-20			
Branch & Section:		PHARM D		Exam:		MID - III			
Course/Sub:		PATHO		Year/Semister:		II			
		CO1	C01	CO1	C01	CO1	C01	C02	C02
Sl.No	Roll Number	Question No.							
		Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8
Maximum Marks		5	5	5	5	5	5	5	5
1	18GN1T0001	5		4	5	4		5	5
2	18GN1T0002		4	5	4	5		4	5
3	18GN1T0003		4	5	4	5	4	5	
4	18GN1T0004		4		4	5	5	5	4
5	18GN1T0005		5	5	5	5	4		3
6	18GN1T0006	5	3		5	5		4	5
7	18GN1T0007	4		4	5	5		4	5
8	18GN1T0008	4	5	5	4		5	5	
9	18GN1T0009	5	4	5	5	5	4		
10	18GN1T0010	4	5		5	3	5	5	
11	18GN1T0011			4	5	5	4	5	4
12	18GN1T0012			5	4	5	5	4	4
13	18GN1T0013	4	5	4	5		5		4
14	18GN1T0014	5	4	5	5	5	4		
15	18GN1T0015	5		4	5	5	4		4
16	18GN1T0017	4		4	5	5		4	5
17	18GN1T0018		5	4	5	4	4	5	
18	18GN1T0019	5		5	5	4	4		4
19	18GN1T0020		4	5		5	4	4	5
20	18GN1T0021		5	5		4	4	4	5
21	18GN1T0022		4	5	4	5	5	4	
22	18GN1T0023	5	5	4	5	4		4	
23	18GN1T0025	4	5	5	4	4	5		
24	18GN1T0026		5	4		5	4	5	4
25	17GN1T0020	5	4		5	4	5	4	
No. of students attempted		21	20	22	17	16	19	17	18
Max Marks Question wise		5	5	5	5	5	5	5	5
Target 50%		2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5
No. of Students above 50%		21	20	22	17	16	19	17	18
% of Students>50%		100.0	100	100	100.0	100.0	100	100	100.0
Attainment Level		3	3	3	3	3	3	3	3
Attainment table									
70-80%	1								
80-90%	2								
90-100%	3								

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Department:	PHARM D		
Course Outcome Attainment External Examination			
Name of the Faculty:	Dr. RAVIPRAKASH	Academic Year:	2019-20
Branch & Section:	PHARM D	Exam:	EXTERNAL
Course:	PATHO	Year/Semister:	II

S.NO.	HALLTICKET NO	TOTAL(Max. Score Marks)
1	18GN1T0001	47
2	18GN1T0002	46
3	18GN1T0003	46
4	18GN1T0004	46
5	18GN1T0005	43
6	18GN1T0006	39
7	18GN1T0007	45
8	18GN1T0008	53
9	18GN1T0009	52
10	18GN1T0010	53
11	18GN1T0011	53
12	18GN1T0012	35
13	18GN1T0013	53
14	18GN1T0014	53
15	18GN1T0015	33
16	18GN1T0017	61
17	18GN1T0018	48
18	18GN1T0019	52
19	18GN1T0020	34
20	18GN1T0021	56
21	18GN1T0022	35
22	18GN1T0023	42
23	18GN1T0025	53
24	18GN1T0026	35
25	17GN1T0020	47

No. of students who attempted the subject	25
Max. Marks	70
No. of students secured > 26 marks	25
Percentage of students secured > 26 marks	100.0
Overall External Attainment level	3

Attainment table	
70-80%	**1**
80-90%	**2**
90-100%	**3**

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Department:	PHARM D		
Overall Course Outcome Attainment			
Name of the Faculty:	RAVIPRAKASH	Academic Year:	2019-20
Branch & Section:	PHARM D	Exam:	
Course:	PATHO	Semister:	II

Course Outcomes	1st Inter	2nd	3rd	Internal Exam(Avg.)	University Exam	Overall Attainment
Course outcome - 1	3	-	-	3	3	3
Course outcome - 2	3	3	-	3	3	3
Course outcome - 3	-	3	3	3	3	3
Course outcome - 4	-	-	3	3	3	3
Average				3	3	3

OVERALL ATTAINMENT OF THE SUBJECT = 0.25*INT + 0.75*EXT

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AVANTHI GROUP INSTITUTIONS

FACULTY MONTHLY PERFORMANCE REPORT

Name of the Faculty: B. Soujanya Month: September

Department: Pharmaceutical analysis

College: Avanthi Institute of Pharmaceutical Sciences

S.No	Subject Name	Year/ Semister	Classes Held in this month	Syllabus Details			No. of Classes required to complete the Syllabus	No. of Tests Conducted in this month	No. of Student Phone calls made	No. of parents visited in this month
				Pervious month Syllabus/ Units completed	Syllabus / Units completed in this month	Syllabus / Units completed from the beginning of the semister to till this month				
1	pharmaceutical Quality Assurance	III-II	15	03	01	04	30	-	-	-
2	~~Industrial~~ pharmacy	~~III-I~~								
3	Instrumental Methods of Analysis	III-I	13	2.5	01	3.5	35	-	-	-
4										
5										
6										

Class Incharge Details:

Class :

Strength :

No. of irregular Students:

Action Taken :

Last Semister Subjects taught and results

S No	Name of the subject	Pass %
1		
2		
3		
4		

Sudents Attendence Registers Checked by Principal Yes / No :

Faculty HOD Principal Director

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19-20

AVANTHI GROUP INSTITUTIONS

FACULTY MONTHLY PERFORMANCE REPORT

Name of the Faculty : Dr. Ravi Prakash Month : October - 2019

Department: PharmD

College: Avanthi Institute of Pharmaceutical Sciences

S.No	Subject Name	Year/ Semister	Classes Held in this month	Syllabus Details: Pervious month Syllabus/ Units completed	Syllabus Details: Syllabus / Units completed in this month	Syllabus Details: Syllabus / Units completed from the beginning of the semister to till this month	No. of Classes required to complete the Syllabus	No. of Tests Conducted in this month	No. of Student Phone calls made	No. of parents visited in this month
1	Pathophysiology	II.	13	1 1/2 unit	1/2 units	2 units	112	-	-	-
2										
3	Pharmaceutical Analysis	III.	16	1/2 unit	1/4 unit	1 unit	121	-	-	-
4										
5										
6										

Class Incharge Details:

Class :

Strength :

No. of irregular Students:

Action Taken :

Last Semister Subjects taught and results

S No	Name of the subject	Pass %
1		
2		
3		
4		

Sudents Attendence Registers Checked by Principal Yes / No :

Faculty HOD Principal Director

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19-20

AVANTHI GROUP INSTITUTIONS

FACULTY MONTHLY PERFORMANCE REPORT

Name of the Faculty : G. Swapna Month : March 2020

Department: pharmaceutical chemistry

College:

S.No	Subject Name	Year/ Semister	Classes Held in this month	Syllabus Details			No. of Classes required to complete the Syllabus	No. of Tests Conducted in this month	No. of Student Phone calls made	No. of parents visited in this month
				Pervious month Syllabus/ Units completed	Syllabus / Units completed in this month	Syllabus / Units completed from the beginning of the semister to till this month				
1	Pharmaceutical organic chemistry-I	I/I	12	1.5	1.5	3 units	25	—	—	—
2										
3	Medicinal chemistry II	III/II	18	3.0	1.5	4.5	5	—	—	—
4										
5										
6										

Class Incharge Details:

Class :

Strength :

No. of irregular Students:

Action Taken :

Last Semister Subjects taught and results

S No	Name of the subject	Pass %
1		
2		
3		
4		

Sudents Attendence Registers Checked by Principal Yes / No :

G Swapna

Faculty

HOD

Principal

Director

AVANTHI INSTITUTE OF PHARMACEUTICAL SCIENCES * ABDULLAPURMET *

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19-20

AVANTHI GROUP INSTITUTIONS

FACULTY MONTHLY PERFORMANCE REPORT

Name of the Faculty : MD. Abdul Azeem Month : April- 2020

Department:

College:

S.No	Subject Name	Year/ Semister	Classes Held in this month	Syllabus Details			No. of Classes required to complete the Syllabus	No. of Tests Conducted in this month	No. of Student Phone calls made	No. of parents visited in this month
				Pervious month Syllabus/ Units completed	Syllabus / Units completed in this month	Syllabus / Units completed from the beginning of the semister to till this month				
1	Medicinal Bio Chemistry.	I	16	12.5 units	1.5 units	14 units	—	—	—	—
2										
3	Clinical Pharmacy.	IV	End Examinations				—	—	—	—
4										
5										
6										

Class Incharge Details:

Class :

Strength :

No. of irregular Students:

Action Taken :

Last Semister Subjects taught and results

S No	Name of the subject	Pass %
1		
2		
3		
4		

Sudents Attendence Registers Checked by Principal Yes / No :

Faculty Azeem HOD Principal Director

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DATE: 23-03-2020

EXAMINATION BRANCH
NOTICE
TIME-TABLE
PHARM D V-YEAR III MID EXAMINATIONS

Time: 1.30PM to 3.30 PM

DATE & DAY	SUBJECT
30-03-2020/MON	CLINICAL RESEARCH
31-03-2020/TUE	PHARMACOEPIDEMIOLOGY& PHARMACOECONOMICS
01-04-2020/WED	CLINICALPHARMACOKINETICS& PHARMACOTHERAPEUTIC DRUG MONITORING

P. Nagaraju
EXAMINATION BRANCH

HOD

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COPY TO: 1) PRINCIPAL
2) ALL HODS
3)ALL STUDENTS
4)OFFICE
5) NOTICE BOARDS

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DATE: 02-01-2020

EXAMINATION BRANCH
NOTICE
TIME-TABLE
PHARM D V-YEAR II MID EXAMINATIONS

Time: 1.30PM to 3.30 PM

DATE & DAY	SUBJECT
06-01-2020/MON	CLINICAL RESEARCH
07-01-2020/TUE	PHARMACOEPIDEMIOLOGY& PHARMACOECONOMICS
08-01-2020/WED	CLINICALPHARMACOKINETICS& PHARMACOTHERAPEUTIC DRUG MONITORING

P. Nagaraju

EXAMINATION BRANCH **HOD** **PRINCIPAL**

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4)OFFICE
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DATE: 11-09-2019

EXAMINATION BRANCH
NOTICE
TIME-TABLE
PHARM D V-YEAR I MID EXAMINATIONS

Time: 1.30PM to 3.30 PM

DATE & DAY	SUBJECT
16-09-2019/MON	CLINICAL RESEARCH
17-09-2018/TUE	PHARMACOEPIDEMIOLOGY& PHARMACOECONOMICS
18.09.2022/WED	CLINICALPHARMACOKINETICS& PHARMACOTHERAPEUTIC DRUG MONITORING

P. Nagaraju
EXAMINATION BRANCH

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DATE: 23-03-2020

EXAMINATION BRANCH
NOTICE
TIME-TABLE
PHARM D IV-YEAR III MID EXAMINATIONS

Time: 1.30PM to 3.30 PM

DATE & DAY	SUBJECT
30-03-2020/MON	PHARMACOTHERAPEUTICS-III
31-03-2020/TUE	HOSPITAL PHARMACY
01-04-2020/WED	CLINICAL PHARMACY
03-04-2020/FRI	BIOSTATISTICS & RESEARCH METHODOLOGY
04-04-2020/SAT	BIOPHARMACEUTICS & PHARMACO KINETICS
06-04-2020/MON	CLINICAL TOXICOLOGY

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DATE: 02-01-2020

EXAMINATION BRANCH
NOTICE
TIME-TABLE
PHARM D IV-YEAR II MID EXAMINATIONS

Time: 1.30PM to 3.30 PM

DATE & DAY	**SUBJECT**
06-01-2020/MON	PHARMACOTHERAPEUTICS-III
07-01-2020/TUE	HOSPITAL PHARMACY
08-01-2020/WED	CLINICAL PHARMACY
09-01-2020/THU	BIOSTATISTICS & RESEARCH METHODOLOGY
10-01-2020/FRI	BIOPHARMACEUTICS & PHARMACO KINETICS
11-01-2020/SAT	CLINICAL TOXICOLOGY

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DATE: 11-09-2019

EXAMINATION BRANCH
NOTICE
TIME-TABLE
PHARM D IV-YEAR I MID EXAMINATIONS

Time: 1.30PM to 3.30 PM

DATE & DAY	SUBJECT
16-09-2019/MON	PHARMACOTHERAPEUTICS-III
17-09-2018/TUE	HOSPITAL PHARMACY
18.09.2022/WED	CLINICAL PHARMACY
19.09.2022/THU	BIOSTATISTICS & RESEARCH METHODOLOGY
20.09.2022/FRI	BIOPHARMACEUTICS & PHARMACO KINETICS
21.10.2022/SAT	CLINICAL TOXICOLOGY

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DATE: 23-03-2020

EXAMINATION BRANCH

NOTICE

TIME-TABLE

PHARM D III-YEAR III MID EXAMINATIONS

Time: 1.30PM to 3.30 PM

DATE & DAY	SUBJECT
30-03-2020/MON	PHARMACOLOGY-II
31-03-2020/TUE	PHARMACEUTICAL ANALYSIS
01-04-2020/WED	PHARMACOTHERAPEUTICS-II
03-04-2020/FRI	PHARMACEUTICAL JURISPRUDENCE
04-04-2020/SAT	MEDICINAL CHEMISTRY
06-04-2020/MON	PHARMACEUTICAL FORMULATIONS

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DATE: 02-01-2020

EXAMINATION BRANCH

NOTICE

TIME-TABLE

PHARM D III-YEAR II MID EXAMINATIONS

Time: 1.30PM to 3.30 PM

DATE & DAY	SUBJECT
06-01-2020/MON	PHARMACOLOGY-II
07-01-2020/TUE	PHARMACEUTICAL ANALYSIS
08-01-2020/WED	PHARMACOTHERAPEUTICS-II
09-01-2020/THU	PHARMACEUTICAL JURISPRUDENCE
10-01-2020/FRI	MEDICINAL CHEMISTRY
11-01-2020/SAT	PHARMACEUTICAL FORMULATIONS

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EXAMINATION BRANCH

NOTICE

TIME-TABLE

PHARM D III-YEAR I MID EXAMINATIONS

Time: 1.30PM to 3.30 PM

DATE & DAY	SUBJECT
16-09-2019/MON	PHARMACOLOGY-II
17-09-2018/TUE	PHARMACEUTICAL ANALYSIS
18.09.2022/WED	PHARMACOTHERAPEUTICS-II
19.09.2022/THU	PHARMACEUTICAL JURISPRUDENCE
20.09.2022/FRI	MEDICINAL CHEMISTRY
21.10.2022/SAT	PHARMACEUTICAL FORMULATIONS

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DATE: 23-03-2020

EXAMINATION BRANCH

NOTICE

TIME-TABLE

PHARM D II-YEAR III MID EXAMINATIONS

Time: 1.30PM to 3.30 PM

DATE & DAY	SUBJECT
30-03-2020/MON	PATHOPHYSIOLOGY
31-03-2020/TUE	PHARMACEUTICAL MICROBIOLOGY
01-04-2020/WED	PHARMACOGNOSY & PHYTOPHARMACEUTICALS
03-04-2020/FRI	PHARMACOLOGY-1
04-04-2020/SAT	COMMUNITY PHARMACY
06-04-2020/MON	PHARMACOTHERAPEUTICS-1

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DATE: 02-01-2020

EXAMINATION BRANCH

NOTICE

TIME-TABLE

PHARM D II-YEAR II MID EXAMINATIONS

Time: 1.30PM to 3.30 PM

DATE & DAY	SUBJECT
06-01-2020/MON	PATHOPHYSIOLOGY
07-01-2020/TUE	PHARMACEUTICAL MICROBIOLOGY
08-01-2020/WED	PHARMACOGNOSY & PHYTOPHARMACEUTICALS
09-01-2020/THU	PHARMACOLOGY-1
10-01-2020/FRI	COMMUNITY PHARMACY
11-01-2020/SAT	PHARMACOTHERAPEUTICS-1

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DATE: 11-09-2019

EXAMINATION BRANCH

NOTICE

TIME-TABLE

PHARM D II-YEAR I MID EXAMINATIONS

Time: 1.30PM to 3.30 PM

DATE & DAY	SUBJECT
16-09-2019/MON	PATHOPHYSIOLOGY
17-09-2018/TUE	PHARMACEUTICAL MICROBIOLOGY
18.09.2022/WED	PHARMACOGNOSY & PHYTOPHARMACEUTICALS
19.09.2022/THU	PHARMACOLOGY-1
20.09.2022/FRI	COMMUNITY PHARMACY
21.10.2022/SAT	PHARMACOTHERAPEUTICS-1

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DATE: 27-04-2020

EXAMINATION BRANCH

NOTICE

TIME-TABLE

PHARM D I-YEAR III MID EXAMINATIONS

Time: 1.30PM to 3.30 PM

DATE & DAY	***SUBJECT***
04-05-2020	HUMAN ANATOMY & PHYSIOLOGY
05-05-2020	PHARMACEUTICS
06-05-2020	MEDICINAL BIOCHEMISTRY
07-05-2020	PHARMACEUTICAL INORGANIC CHEMISTRY
08-05-2020	PHARMACEUTICAL ORGANIC CHEMISTRY
09-05-2019	REMEDIAL MATHEMATICS

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DATE: 06-02-2020

EXAMINATION BRANCH

NOTICE

TIME-TABLE

PHARM D I-YEAR II MID EXAMINATIONS

Time: 1.30 PM to 3.30 PM

DATE & DAY	SUBJECT
10-02-2020/MON	HUMAN ANATOMY & PHYSIOLOGY
11-02-2020/TUE	PHARMACEUTICS
12-02-2020/WED	MEDICINAL BIOCHEMISTRY
13-02-2020/THU	PHARMACEUTICAL INORGANIC CHEMISTRY
14-02-2020/FRI	PHARMACEUTICAL ORGANIC CHEMISTRY
15-02-2020/SAT	REMEDIAL MATHEMATICS

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DATE: 13-11-2019

EXAMINATION BRANCH

NOTICE

TIME-TABLE

PHARM D I-YEAR I MID EXAMINATIONS

Time: 1.30PM to 3.30 PM

DATE & DAY	SUBJECT
18-11-2019/MON	HUMAN ANATOMY & PHYSIOLOGY
19-11-2019/TUE	PHARMACEUTICS
20-11-2019/WED	MEDICINAL BIOCHEMISTRY
21-11-2019/THU	PHARMACEUTICAL INORGANIC CHEMISTRY
22-11-2019/FRI	PHARMACEUTICAL ORGANIC CHEMISTRY
23-11-2019/SAT	REMEDIAL MATHEMATICS

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EXAMINATION BRANCH

IV YEAR B.PHARM - I SEMESTER –R16, R15, R13,R09 REGULATIONS- SUPPLEMENTARY EXAMINATIONS OCTOBER-2020

TIMETABLE

TIME : AN: 2:30 PM TO 4:30 PM

DATE & DAY	R16	R15	R13	R09
12-10-2020 MONDAY	Biopharmaceutics and Pharmacokinetics	Biopharmaceutics &Pharmacokinetics	Biopharmaceutics &Pharmacokinetics	Biopharmaceutics and Pharmacokinetics
14-10-2020 WEDNESDAY	Pharmaceutical Analysis – II	Pharmacognosy III	Pharmacognosy III	Pharmaceutical Analysis - II
16-10-2020 FRIDAY	Pharmacology - III	Pharmacology-III	Pharmacology-III	Pharmacology III
19-10-2020 MONDAY	Medicinal Chemistry – II	Medicinal Chemistry – II	Medicinal Chemistry – II	Medicinal Chemistry II
21-10-2020 WEDNESDAY	Pharmacy Administration	Pharmacy Administration	Pharmacy Administration	Pharmacy Administration

DATE: 30-09-2020

CONTROLLER OF EXAMINATIONS

NOTE:

(i).ANY OMISSIONS OR CLASHES IN THIS TIME TABLE MAY PLEASE BE INFORMED TO THE CONTROLLER OF EXAMINATIONS IMMEDIATELY

(ii).EVEN IF GOVERNMENT DECLARES HOLIDAY ON ANY OF THE ABOVE DATES,THE EXAMINATIONS SHALL BE CONDUCTED AS USUAL

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E X A M I N A T I O N B R A N C H

III YEAR B.PHARMACY- I SEMESTER– R17 REGULATION COMPUTER BASED TEST (CBT) JANUARY-2020

TIMETABLES

BRANCH	23-1-2020 Thursday		24-1-2020 Friday		25-1-2020 Saturday
	10.00 AM TO 10.45 AM	2.00 PM TO 2.45 PM	10.00 AM TO 10.45 AM	2.00 PM TO 2.45 PM	10.00 AM TO 10.45 AM
B.PHARMACY III YEAR I SEM.	Medicinal Chemistry II	Industrial Pharmacy - I	Pharmacology II	Pharmacognosy and Phytochemistry - II	**(Open Elective-I)** Generic Product Development
					Green Chemistry
					Cell and Molecular Biology
					Cosmetic science

IV YEAR B.PHARMACY I SEMESTER– R16 REGULATION COMPUTER BASED TEST (CBT) JANUARY-2020

BRANCH	23-1-2020 Thursday		24-1-2020 Friday		25-1-2020 Saturday
	12.00 PM TO 12.45 PM	4.00 PM TO 4.45 PM	12.00 PM TO 12.45 PM	4.00 PM TO 4.45 PM	12.00 PM TO 12.45 PM
B.PHARMACY IV YEAR I SEM.	Pharmaceutical Analysis – II	Biopharmaceutics and Pharmacokinetics	Pharmacology - III	Medicinal Chemistry – II	Pharmacy Administration

II YEAR B.PHARMACY - I SEMESTER– R17 REGULATION COMPUTER BASED TEST (CBT) JANUARY-2020

BRANCH	27-1-2020 Monday		28-1-2020 Tuesday	
	10.00 AM TO 10.45 AM	2.00 PM TO 2.45 PM	10.00 AM TO 10.45 AM	2.00 PM TO 2.45 PM
B.PHARMACY II YEAR I SEM.	Pharmaceutical Organic Chemistry – II	Physical Pharmaceutics-I	Pharmaceutical Microbiology	Pharmaceutical Engineering

I YEAR B.PHARMACY - I SEMESTER– R17 REGULATION - COMPUTER BASED TEST (CBT) JANUARY-2020

BRANCH	27-1-2020 Monday		28-1-2020 Tuesday		29-01-2020 Wednesday	
	12.00 PM TO 12.45 PM	4.00 PM TO 4.45 PM	12.00 PM TO 12.45 PM	4.00 PM TO 4.45 PM	12.00 PM TO 12.45 PM	4.00 PM TO 4.45 PM
B.PHARMACY I YEAR I SEM.	REMEDIAL BIOLOGY	HUMAN ANATOMY AND PHYSIOLOGYI	PHARMACEUTICAL ANALYSIS I	PHARMACEUTICS I	PHARMACEUTICAL INORGANIC CHEMISTRY-I	COMMUNICATION SKILLS
	REMEDIAL MATHEMATICS					

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Sd/-

DATE: 17-01-2020

CONTROLLER OF EXAMINATIONS

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EXAMINATION BRANCH

II YEAR B.PHARM - I SEMESTER –R17, R16, R15 , R13, R09 REGULATIONS- REGULAR/SUPPLEMENTARY EXAMINATIONS DEC-2019

TIMETABLE

TIME: 10:00 AM TO 1:00 PM

DATE& DAY	R17	R16	R15	R13	R09
03-12-2019 TUESDAY	Pharmaceutical Organic Chemistry – II	Pharmaceutical Organic Chemistry – III	Pharmaceutical Organic Chemistry – II	Pharmaceutical Organic Chemistry-II	Pharmaceutical Organic Chemistry-II
05-12-2019 THURSDAY	Physical Pharmaceutics-I	Pharmacognosy I	Statistical Methods & Computer Applications	Statistical Methods & Computer Applications	Statistical Methods & Computer Applications
07-12-2019 SATURDAY	Pharmaceutical Microbiology	HospitaL and Community Pharmacy	Anatomy, Physiology &Pathophysiology	Anatomy Physiology &Pathophysiology	Physical Pharmacy – I Dispensing and Hospital Pharmacy
10-12-2019 TUESDAY	Pharmaceutical Engineering	Pharmaceutical Unit Operations – I	Pharmaceutical Unit Operations – I	Pharmaceutical Unit Operations – I	Pharmaceutical Unit Operations – I
12-12-2019 THURSDAY	---	Pharmaceutical Analysis-I	Physical Pharmacy – I	Physical Pharmacy – I	Anatomy Physiology & Pathophysiology Health Education and Pathophysiology

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DATE: 21-10-2019

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DATE: 15.12.2020

B. PHARMACY

PROJECT SHEDULE

For the academic year **2019-2020** all the **IV B. PHARM II SEMESTER** are here by informed that the students should undergo the course project as per the JNTUH R17 REGULATIONS.

S.NO:	REVIEW & ASSESSMENT	TOPIC	TENTATIVE SCHEDULE
1	PROJECT INITIALIZATION	FINALIZATION OF TITLE & PLAN OF WORK	23.12.2019 to 31.12.2019
2	REVIEW-1	REVIEW OF LITERATURE	06.012020 to 13.01.2020
3	REVIEW-2	METHODOLOGY & EXPECTED RESULTS	26.01.2020 to 4.02.2020
4	REVIEW-3	RESULTS & DISCUSSION, CONCLUSION	20.02.2020 to 05.03.2020

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Guidelines to students:

1. UG project work shall be carried out during IV Year II Semester.

2. Project will be evaluated for 100 marks. Student has to submit project work report at the end of semester.

3. Project shall be conducted in 3 Reviews

4. Project shall be completed before the commencement of SEE Theory examinations.

5. For Project the departmental committee consisting of Head of the Department, project supervisor and a senior faculty member shall evaluate the project work.

6. The student is deemed to have failed, if he/she

(i) Does not submit a report or does not make a presentation of the same before the evaluation committee as per schedule, or

(ii) Secures less than 40% marks in the sum total of the CIE and SEE taken together.

7. A student who has failed may reappear once for the above evaluation, when it is scheduled again; if he fails in such 'one reappearance' evaluation also, he has to reappear for the same in the next subsequent semester, as and when it is scheduled.

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8 For conducting viva-voce of project, University selects an external examiner from the list of experts in the relevant branch submitted by the Principal of the College.

9. A student who has failed may reappear once for the above evaluation, when it is scheduled again; if student fails in such 'one reappearance' evaluation also, he has to reappear for the same in the next subsequent semester, as and when it is scheduled.

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Department:		PHARM D							
Course Outcome Attainment - Internal Assessments									
Name of the Faculty:		Dr. RAVIPRAKASH		Academic Year:		2019-20			
Branch & Section:		PHARM D		Exam:		MID - II			
Course/Sub:		PATHO		Year/Semister:		II			
		CO2	C02	C02	C02	CO3	C03	C03	C03
Sl.No	Roll Number	Question No.							
		Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8
Maximum Marks		5	5	5	5	5	5	5	5
1	18GN1T0001	5		4	5	4		5	5
2	18GN1T0002		4	5	4	5		4	5
3	18GN1T0003		4	5	4	5	4	5	
4	18GN1T0004		4		4	5	5	5	4
5	18GN1T0005		5	5	5	5	4		3
6	18GN1T0006	5	3		5	5		4	5
7	18GN1T0007	4		4	5	5		4	5
8	18GN1T0008	4	5	5	4		5	5	
9	18GN1T0009	5	4	5	5	5	4		
10	18GN1T0010	4	5		5	3	5	5	
11	18GN1T0011			4	5	5	4	5	4
12	18GN1T0012			5	4	5	5	4	4
13	18GN1T0013	4	5	4	5		5		4
14	18GN1T0014	5	4	5	5	5	4		
15	18GN1T0015	5		4	5	5	4		4
16	18GN1T0017	4		4	5	5		4	5
17	18GN1T0018		5	4	5	4	4	5	
18	18GN1T0019	5		5	5	4	4		4
19	18GN1T0020		4	5		5	4	4	5
20	18GN1T0021		5	5		4	4	4	5
21	18GN1T0022		4	5	4	5	5	4	
22	18GN1T0023	5	5	4	5	4		4	
23	18GN1T0025	4	5	5	4	4	5		
24	18GN1T0026	5	5	4		5	4	5	4
25	17GN1T0020		4		5	4	5	4	
No. of students attempted		21	20	22	17	16	19	17	18
Max Marks Question wise		5	5	5	5	5	5	5	5
Target 50%		2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5
No. of Students above 50%		21	20	22	17	16	19	17	18
% of Students>50%		100.0	100	100	100.0	100.0	100	100	100.0
Attainment Level		3	3	3	3	3	3	3	3
Attainment table									
70-80%	1								
80-90%	2								
90-100%	3								

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Department:		PHARM D							
Course Outcome Attainment - Internal Assessments									
Name of the Faculty:		Dr. RAVIPRAKASH				2019-20			
Branch & Section:		PHARM D		Exam:		MID - I			
Course/Sub:		PATHO		Year/Semister:		II			
		CO1	C01	CO1	C01	CO1	C01	C02	C02
Sl.No	Roll Number	Question No.							
		Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8
Maximum Marks		5	5	5	5	5	5	5	5
1	18GN1T0001	5	4	4		5	5	4	
2	18GN1T0002	4	5	5	5	5		4	
3	18GN1T0003	5	4	5	5	5		4	
4	18GN1T0004		4	5	4		4	5	4
5	18GN1T0005	4		5	4	5	3	5	
6	18GN1T0006	5	5	4	4		4	5	
7	18GN1T0007	5	4	4		5	5		4
8	18GN1T0008	5	4	4		5	4		5
9	18GN1T0009	4	5	4		5	5		4
10	18GN1T0010	5	4		5	4	4		4
11	18GN1T0011	5	4	5	4	5		5	
12	18GN1T0012	4	5		5		4	5	5
13	18GN1T0013	5	5	5	4		5		3
14	18GN1T0014		4	5	5	4		5	5
15	18GN1T0015	5	4	5	5	4	4		
16	18GN1T0017	5	4	4	5		4		5
17	18GN1T0018		5	4	4	5		5	5
18	18GN1T0019	4		5	5		4	5	4
19	18GN1T0020	4	5	4		5	4		5
20	18GN1T0021		5	4		4	4	5	5
21	18GN1T0022	5	4	5			5	4	5
22	18GN1T0023	5	4	4		5		4	5
23	18GN1T0025	5		5	4		5	4	5
24	18GN1T0026	4		4	5		5	5	4
25	17GN1T0020	4			5	4	5	4	5
No. of students attempted		21	20	22	17	16	19	17	18
Max Marks Question wise		5	5	5	5	5	5	5	5
Target 50%		2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5
No.ofStudentsabove50%		21	20	22	17	16	19	17	18
% of Students>50%		100.0	100	100	100.0	100.0	100	100	100.0
Attainment Level		3	3	3	3	3	3	3	3

Attainment table									
70-80%	1								
80-90%	2								
90-100%	3								

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Course Outcome Attainment - Internal Assessments

Name of the Faculty:		Dr. RAVIPRAKASH		Academic Year:		2019-20			
Branch & Section:		PHARM D		Exam:		MID - III			
Course/Sub:		PATHO		Year/Semister:		II			
		CO1	C01	CO1	C01	CO1	C01	C02	C02
Sl.No	Roll Number	Question No.							
		Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8
Maximum Marks		5	5	5	5	5	5	5	5
1	18GN1T0001	5		4	5	4		5	5
2	18GN1T0002		4	5	4	5		4	5
3	18GN1T0003		4	5	4	5	4	5	
4	18GN1T0004		4		4	5	5	5	4
5	18GN1T0005		5	5	5	5	4		3
6	18GN1T0006	5	3		5	5		4	5
7	18GN1T0007	4		4	5	5		4	5
8	18GN1T0008	4	5	5	4		5	5	
9	18GN1T0009	5	4	5	5	5	4		
10	18GN1T0010	4	5		5	3	5	5	
11	18GN1T0011			4	5	5	4	5	4
12	18GN1T0012			5	4	5	5	4	4
13	18GN1T0013	4	5	4	5		5		4
14	18GN1T0014	5	4	5	5	5	4		
15	18GN1T0015	5		4	5	5	4		4
16	18GN1T0017	4		4	5	5		4	5
17	18GN1T0018		5	4	5	4	4	5	
18	18GN1T0019	5		5	5	4	4		4
19	18GN1T0020		4	5		5	4	4	5
20	18GN1T0021		5	5		4	4	4	5
21	18GN1T0022		4	5	4	5	5	4	
22	18GN1T0023	5	5	4	5	4		4	
23	18GN1T0025	4	5	5	4	4	5		
24	18GN1T0026		5	4		5	4	5	4
25	17GN1T0020	5	4		5	4	5	4	
No. of students attempted		21	20	22	17	16	19	17	18
Max Marks Question wise		5	5	5	5	5	5	5	5
Target 50%		2.5	2.5	2.5	2.5	2.5	2.5	2.5	2.5
No. of Students above 50%		21	20	22	17	16	19	17	18
% of Students>50%		100.0	100	100	100.0	100.0	100	100	100.0
Attainment Level		3	3	3	3	3	3	3	3
Attainment table									
70-80%	1								
80-90%	2								
90-100%	3								

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Department:	PHARM D		
Course Outcome Attainment External Examination			
Name of the Faculty:	Dr. RAVIPRAKASH	Academic Year:	2019-20
Branch & Section:	PHARM D	Exam:	EXTERNAL
Course:	PATHO	Year/Semister:	II

S.NO.	HALLTICKET NO	TOTAL(Max. Score Marks)
1	18GN1T0001	47
2	18GN1T0002	46
3	18GN1T0003	46
4	18GN1T0004	46
5	18GN1T0005	43
6	18GN1T0006	39
7	18GN1T0007	45
8	18GN1T0008	53
9	18GN1T0009	52
10	18GN1T0010	53
11	18GN1T0011	53
12	18GN1T0012	35
13	18GN1T0013	53
14	18GN1T0014	53
15	18GN1T0015	33
16	18GN1T0017	61
17	18GN1T0018	48
18	18GN1T0019	52
19	18GN1T0020	34
20	18GN1T0021	56
21	18GN1T0022	35
22	18GN1T0023	42
23	18GN1T0025	53
24	18GN1T0026	35
25	17GN1T0020	47

No. of students who attempted the subject	25
Max. Marks	70
No. of students secured > 26 marks	25
Percentage of students secured > 26 marks	100.0
Overall External Attainment level	3

Attainment table	
70-80%	**1**
80-90%	**2**
90-100%	**3**

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