9nflammationis a Reprotective Response involving host cells, blood vesseles, Protein other mediators. to eliminate The and initial cause of cellinjury as well as necrotic cells and tissues and initiate The process of Repair. Characterstics: O- Inflammation accomplish its mission by first diluting destorying or neutring or neutring harmful agents 2)-Healing and Repair of all at site of injury 3helps clear infection but also Inflammation capable of normal tissue 4)-Inflammation is normal and beneficial (5)but Rx very strong. Prolonge or inappropriate lead to chronic anflammation 6)onflammation act as innate immunity - Protectic Response Function:

Main Goal is to bring

Cell - and mole rule of host

defenre - leukocytes + plasma protein

to site of infection

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O

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	4		Da	te:	
Cardinal	Sign	of infla	mmatron:-	[]	s If
Heat -	calor	ri in N	4 4		
Redness -	Rubor	Al John 17	Le stora	S 3	
Swelling -		i di consti	rd Sendo	*	
Pain	dolor		. A.		
Loss of fun-	function	laesa.	Scotts.		
fine at the second	PROTECTION OF THE PROTECTION O		nmatron:		1-
O- Vascu	lar Event	7 ac	tivated by m	eduation	derived
2 - Cellular	Respons	re I from	1 Plasma Pro	tein and	cells
					2(3)
9nf lamm	ation 9ndu	ced by	Mediators :-	645 °	Daniel m
O- Mediator	y Pr	oduced by	Plasma Protein	251	Inde co
and	uco an Eli	ammatron	- Response	to injury	stimul
Me	chanism:-		and the 21 P	1855	the new me
@- Microba			Protorer		
enter	the cell,	does	Infection and	damage	(1)-
		acsophoges			
valench.	3- M	ast ceus	L		-(3)
- W. D.	J. Tarpenii	Lytos	ecrete O- (ylok	ines latori	rsteade.
					9nflammatic
m)	Service T		O_ Vascular	change	Lamas Ju
wid refresh	Terror	rion .	Q- cellular	r change	
		healing $\rightarrow R$	pair		-61
	Mr. 2 - 9	sometime lead	d -> chron	uic Inflam	mation
1	(L)-	CONTRACT CONTRACTOR			

D -4		
11/11/01	Date:	

Type	of	inflammation!	ar i
		02 type	of inflammation.
	O -	- Acute Inflamm	ation
	@-	Chronic Inflamm	ation

	Acute	Chronic
	Acute	Information.
O -	who have full and are to	In a charge in
Onset	Rapid onset	Slow onset
2 -	·	
Duration	Short duration	Long duration.
	few minutes	Long duration. days - Years
3 -	Maria James A - matter	molling mulita
Component	contain fluid+ Plasma	Contain lymphocytes,
	Protein	Macrophages
%	does soft householder in	The state of the s
"	Bredominantly neutro-	Bre dominantly
	phills deukocyte	Predominantly macrophages
B -		1211 18
Systemic	Sever often high	low grade fever
Menifistation	Louisidad	
out come.		Fibrosis
	Absces formation	Tisrue destriction
0 -	8 mg/) = [16	West States
Cardinal Sign	Prerent	slightly /Abrent
- unha		niceday

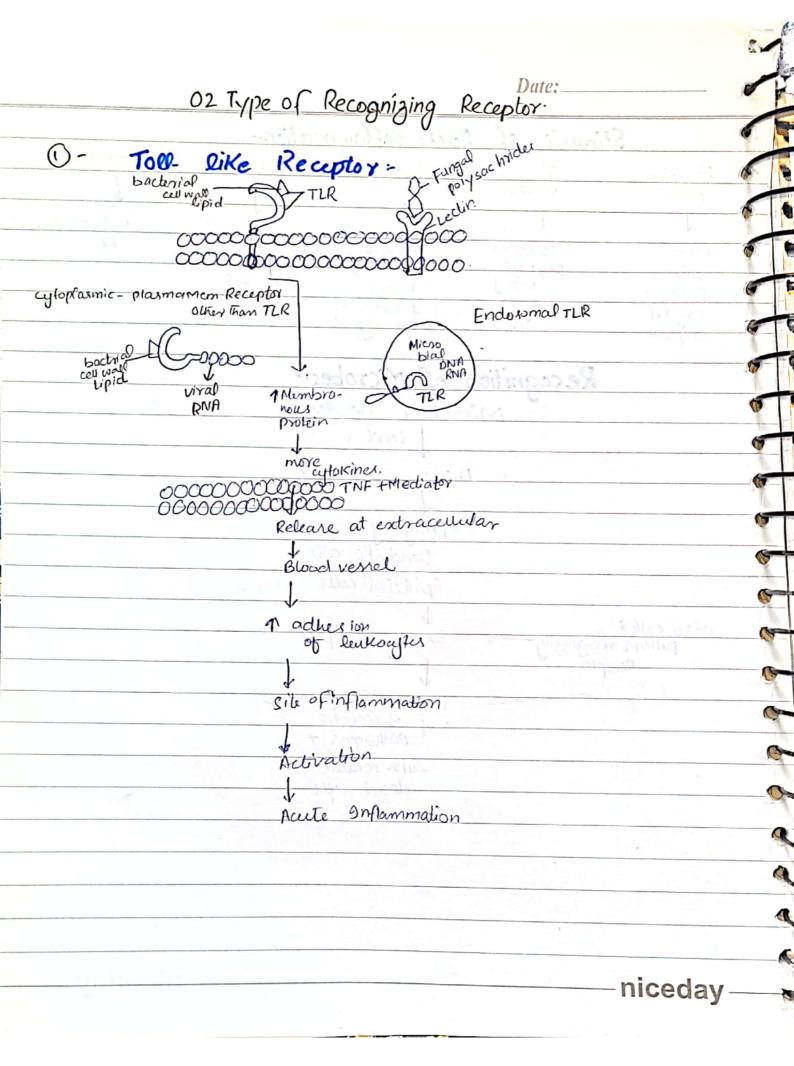
6		Date:
Blood vesseles	Vasodilation	Proliferation
	V COSO CO (CI IIOTT	
Mecliators	Histomine	T-lymphogtes.
	Brady Kinin	GFs.
6 -		
*	Tirrue + fibrosis.	Tirrue - fibrosis
	Mild and self	Severe+
	limited	progressive
,		a lama a cons
	Acute Inflam	matorialist
	Two component.	
O- Vas		milesping -U
Q- Celli	ular changes	
Vas	cular changes:	ration in vessel calibar
5	O- Alter	ration in vessel calibar
,		-1/
	1	Blood flow
	100° Car As M. Carlo	Vasodilation -
, sådn	made serre day	SOL SOLULIA CONTRACTOR
113011	Q-↑ vas	cular Permeability.
	/Creps profice	1
	on Canada Halial	Mow plana Protein
3)- Hetivator	of endothelial	to leave includation
1 adh	esion of leukocytes	W TOO GHOT
Miaro	ation of leukocytes	
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		Illocuay

with the set of the section
(8)
Venous dilation 18.F
0
Leakage of
20200
protein > edema
Acute 3rd In
Jan Bayerra . Cult
f leukocytes from Circulation
Deports values s
nulation at site of injury
44
vation of leukoujes
ainly Meutophills
(polymorphonudear)
Quikoujtes)
iminate
affecting agents.
ARTHUR DO TO BE TO THE
enthorns to universities to
The sale of the sa
Substance de lessant by the
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Date:		

	muli of A	cute Inflammat	ion:-	Amouse D.
orenign	Tissue	Trauma	Infection	9mmone Rx
Bodies	Necrosii	y 1	7. 1	1
1	1		1	a. Linamina
splinten		7	Bacterial	Autoimmune Rx
	Ischemia	Thermal	Viral	~ X
Dirts Sidures	——Physical—— 9njury	Borns	Fungal	
		Irracliation	parasitic	
crystal deposites	Chemical	Toxicity	^	
Cleposito		JIMA V) II
	De con Line	-C Microbaci-		
	Recognition	of Microbes:	1157	
	Mic	sobe + Deadcell I send	J	
		send		
		Danger Signals to		KATE CO.
	1	1	100000	
	4 10	Phagocy ter	Kil	Are true
		Dendritic cells		
		Epilhelial cells		
		1	4	
also called	recognizing ->	Express Receptor		
also called Paltern - Rec	contor	U		
, cec	7	4	A Property of the Control of the Con	
		sense the pre	une	
		sense The pres of Infections pathogens +	E L	
	the state of the state of	radiograms /	2	
		sub-related +		
	-57	dead cells	1	
		Dish bashertellaria		
1				
		- Carrier Carrier Ca		
	46-12-			

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	Date:
2- Inflammasom	Valendar character -: 9
311/10/10/10/10/10/10/10/10/10/10/10/10/1	Multi protein cutoplasmic complex.
	Multi protein cytoplasmic complex.
	Products of dead cells
	Ouric Acid
	2 ATP extracellular
0000000000	00000000000000000000000000000000000000
bacaças	Janay Abacasa
	gnflammasome.
	Acton K + efflux ROS
Conve	Endogenous Crystals. Enogenous Crystals
(caspare 1) (caspare 1)	aspares active)
Nucleus	The bridge of the second of th
(0000)	+convert (IL-1B)
Pro IL-1B. Pro IL-1B	\rightarrow
gene bond youldo	secrete sonor
	wold boot IL-IB.
15.5	
	gnc adhesion of leukoayte
e evaluation	
Autoimmunity/ Autoinflam	motory Acute and Tammatron
syndrom: Goat	urate cystal), amyloid in brain (Alzahmir diseare) ofoshof crystal (cutierosaclaresis) Type 2 diabetes
s y rio io i	deshot crystal (cutrerosactoresis) Type 2 diabeter obesity
L diales of	IL-1B antagonists for the treatment of them
Autoactivation of inflammosome.	AVERAMIETE ST. III
La I	No. of the contract of the con
Release IL-1B	D. C.

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Date:

Vascular changes:	
Normally:- 1 hydro Static Pres.	
Secreted IL1B.,TNF.	ļ
Blood Verrel	
Vasocli Patron	
1 B-F 1 Vascular Permeability.	(6
(Carpana) To (Carpana)	6
1 Blood cells 1 Membrenous proteins.	(6
1) Change in vasular caliberand Blood flow:	6
Anflom atory Stimulus	0
G-Stasis:- Small dilated verid e- RBC. Vasoconstriction.	0
D-Margination: Leukouje acemulate vasulor endotwial surface 1 B-Flow engorgment of down stream	9
Cours - Redness erthyma. Stasts, Margin ation	
Hed niceday—	

Date:	Date:		
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@- Inc Vascular Permeability:-

1 vasular Permeability

movement of Protein + fluid

Two Type of action

A COUNTY OF THE PARTY OF THE PA		VE COCKET
high to see	Transudate	Exudate.
Defination:	One Vasualar Permeability	1 V- Permeability
is of made	Deads to & osmotic Presuse	1 O-P-
	1 Protein loss.	Damage serous membrane
~: 55	1 hydroslatic Presure	out flow of water + Botein
Ü		a 18 Station Report has a
Edema	No inflomatory edema	Inflammatory Edema.
1 - Calendar	Jaco With	20 contract (out 1
Protein	Low protein Content	1419th protein Content.
- Period E		CHAPTER TREET BETTER TO THE
Coagulant	No tendency to Coagulate	Coagulate.
122		(10.2 (10.1)
fibrinogen	No libringen	Fibringen 1 content.
J		1.01
LDH	Low LDH	high LDI+
		(2) = It Ironsaybann
spaces:	Abrent	Interendotrelal space Present
3/40		
SeaK	Fluid leakage	Protein + Fluid leakage
mol.	, det	
		niceday-
- Landson		Hickary

	Nephrotic synchrom	Malignany	
	Pulmonary embolism	Tb.	
	1 hydroslatic presune	Fluidt Bolin l	ealtage
	The state of the s	C do menoner	o
	Low Protein	المناف	vasodilation
		1	colloid Press
	Fluid & colloid		Protein-
4 1 - 1	leakage ornotric Prem	e saluter ma -no	Delings
	1 Protein loss	•	
	7 MON 34	and to deliber to chees	
water s	Course of 1 Vascul	ar Permeability:	
1100 814 1	Coure of Vascul	Ma utilization	[dama]
)- Endolha	Coure of Vascul vial Cell Contraction: histornine		in
1100 814 1	Coure of Vascul	TNF 2nterluk	etaloud.
)- Endolha	Coure of Vascul elial Cell Contraction: histamine bractycinin meet atox	Ma utilization	etaloud.
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)- Endolha	Coure of Vascul elial Cell Contraction: histornine brackycomin meet also endottetial cell contraction	TNF Interluk Lett contraction Jong liver	aisloy9 n
)- Endolha	Coure of Vascul elial Cell Contraction: histamine bractycinin meet atox endotretial cell contraction	TNF Interluk Lett contraction Jong liver	aisloy9 n
)- Endolha	Coure of Vascul elial Cell Contraction: histornine brackylcinin meet also encloteetial cell contraction Short lived (15-30min)	TNF Interluk cell contractio long liver (4-6 hon)	mluged)
)- Endolha	Coure of Vascul elial Cell Contraction: histamine brackylcinin meet also endottetial cell contraction Short Ired (15-30min) Gap-form	TNF Interluk Lett contraction Jong liver	mluged)
)- Endolha	Coure of Vascul elial Cell Contraction: histornine brackylcinin meet also encloteetial cell contraction Short lived (15-30min)	TNF Interluk Cell contractio Jong liver (4-6 hon) 24 hour.	Reagular mlugaa)

O-leukocytes Recruitment: Four steps: O- Margination and Rolling along resser wall. O- adhesion to endothelium. O- Transmigration b/w andwhelial cell. O- Migration towards chemotaxis. (a) Migration and Rolling:- O2 sub steps: O Migration O- Rolling.	Date:	
direct ondirect Beginafler 2-12 hours Caure 1 vasualar Promodility Cellular Event: O2 Steps: O- Leukocytes Recruitment > in ECF O- Leukocytes Activation. Caure acute Inflammation but it may prolong to damage other normal cells O- Leukocytes Recruitment: Four steps: O- Margination and Rolling along ressel wall. O- adhesion to endothelium O- Transmigration blue endothelial cell. O- Migration and Rolling: O2 sub steps: Migration O3 sub steps: Migration O4 solling:	2) - endottelial cell grijury:	
Beginafter 2-12 hours Caure 1 vasualer Permerbility Cellular Event: 02 Steps: 0- Leukocytes Recruitment > in ECF G- Leukocytes Activation. Caure acide Inflammation but it may prolong to damage other normal cells O- Leukocytes Recruitment: Four steps: 0- Margination and Rolling along ressel wall. G- adhesion to endothelium G- Fransmigration b/w andothelial cell. O- Migration towards chemotaxis. (a) Migration and Rolling:- O2 sub steps: © Migration © Rolling:		
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Deleukocytes Recruitment: Four steps: O- Margination and Rolling along ressel wall. O- adhesion to endothelium O- Transmigration blw andothelial all. O- Migration towards Chemotaxis. O2 substeps: O3 Migration O4 Rolling:		
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O- Leukocytes Recruitment: Four steps: O- Margination and Rolling along ressel wall. O- adhesion to endothelium. O- Transmigration b/w endothelial cell. O- Migration towards Chemotaxis. (a) Migration and Rolling:- O2 sub steps: O Migration O- Rolling.	but it may prolong to damage other normal	cells
Four steps: O- Margination and Rolling along ressel wall. O- adhesion to endothelium. O- Transmigration b/w and ondethelial cell. O- Migration towards Chemotaxis. (a) Migration and Rolling:- O2 substeps: O Migration O- Rolling.		
 ②- adhesion to endothelium. ③- Transmigration b/ω endothelial cell. ④- Migration towards Chemotaxis. (a) Migration and Rolling:- O2 sub steps ⑤ Migration ⑥- Rolling. 	Four steps.	
(a) Migration and Rolling:- O2 sub steps (b) Migration O2 Polling.		
(a) Migration and Rolling:- Oz sub steps Migration P- Rolling:	2- adhesion to endothelium.	
(a) Migration and Rolling:- Oz sub steps Migration P- Rolling.	3- Transmigration b/w endethelial cell.	
Oz sub steps Mig ration B- Rolling.	G- Migration towards chemotaxis.	
Oz sub steps Mig ration B- Rolling.	The fide and the fide of the f	
Mig ration B- Rolling.	(a) Migration and Rolling:	
Migration Rolling.	02 sub steps	
(B- Rolling.		
		-
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Date:	
Duic.	

	Blood from capillanes
	entering
	post capillary Venule
	Cause swept process of circulating cells
	Celluday Event
	(a) RBCs are small.
	They move faster
	They are pushed away from endotelion
h	b) Jeukogter large in size
	More less faster
	pushed away towards endothelim
	so that interaction is easily. > Margination
(b) R	offing:- and partial the analytic was a market was a constitution of the constitution
	Cytokines and other mediator
	activates the endothelial cells which
	express adhesion molecules or Receptor
	or ligand to which leukoute is loosly
	attach and detects again & again
	Rolling of leukocyte slasts.
	Les de la competit de
	Professional Contract

Recept	ors on endothelium and leukoayte
Conta	in Ecdomain for binding sugar.
Types	
E sele	
P selec	tin Receptor on platelet endothelium
CD6	2ρ
L-sele	ctin. Receptor on xurface of mast
CD62L	
	A PARTY EVENT RESERVE
They are	in low amount - unactivated form
but	when stimuli come -> activate in large amou
n sa a sa a sa	
Histomin	CytoKine TNF
ProThrombin	IL-1
12:	paliting the party of the party
pselectin	E-Lselectin
1	
	Cast Clata Coul out (ex.
Focilitate leukoughe binding due to Rolling Phenomena	Binding due to rolling
Colling Phonomena	Phonomena.
Kening Provision	

	Date:
@- Adhesion:	
Rolling leutoayt	e.
griticale	
Aethesive	
medialedby	-9ntegrins-
hinds to	6- Transmembrone alycoprotein
leukoujle function associated Antigun (1)	6 - Transmembrane dry wpisteri
Macrophage 1 antigen.	O-Types ICAM
	VCFIII
Binds to VLA-4.	3- mediate adhesion.
very Late ontigen 4	@ Show less affanity when inactivated.
	8- high affanity when activation of lymphogyles
	of lymphouses
chemokines TNF-IL1	4 mg 3
Leuko gtes endoltrelia	el integrin eigands
activated	Altafones e e e e e
Conformational 1 expression change in affainity integ	not ligards for
change in affairity integ	grins.
of integrins on leu 160 cyles	O Con altal
- Signed	I for allach ment
Recieve signal for attachment	
	A SAME TO MAKE A PROPERTY OF THE PARTY OF TH
Calledia	•
firm adhesion.	
	-1
The Mark of the Control of the Contr	
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Date:
3. Transmigration:
Leukoyte migrate mough Verrele wall
By sequeezing blowceus at I/C Junction
called diapep di pedes
after dipedes leukoytes Rebare
collagenases.
help them to pars through. Vasculor membrane
@- Migration towards chemotaxis:- extra vasation of leukocytes
move loward site of infection along. e- chemical gracient
called chemotonis
N-formyl meth
Proclamber of medicine and an armine
Cytolkines. (chumotkines family) Produck AA: Chemotactic Molecules
(LTB4 Bind - Receptor
Release eylos Keleton contract le eliment
Leukoaytes move by extending pseudopods.
puer leukoug ter
direction depends on chumo knes Receptor

	Date:
In acute Inflammation	" twigger in heard"
6-24 hrs.	Newhophill Short lived
Replace by Monoyler	longer
	- U
a least the annual to the	
Leukocyte Activatio	n:-
Duko cu te	e at reached
at	e at reached site of microbe
	ttog en
The state of the s	
HCC I.	Migrafica bisards
Pen	form following function.
Phagacutais of Particals	
grim cellular Destruction	of phagouplosed microbes
Liberation of substance that	it destory extracellular
microbes and dead cells.	Court Transfer
Production of mediators.	
	The same of the sa
Phagocytosis of Partic	147 5 5 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1
00 1	- 0

B- Leukoujle bind to Micro-organism throug Receptor
Recognize

component of Microbes-I dead cells
opponines

	Date:
	@- Microbe also have coating by phenomena of opsonization
	by phenomena of opsonization.
8	3- Recognize by Receptor and engulf by loukoxyles
	G- Most imp opsonins.
S	Antibodies of immunoglobins
<u>~</u>	9 19 9.
	Bind to mitrobial surface antigen
9	↓
0	Biseak down The product of complement protein C3
6	·
	plasma Carbohydrate - binding Lectin.
0	B- Fc receptors for Igg (FCBRI)
0	Complement Rocepter 1 3 for compliment " 3 Fragments
0	" 3 J Fragments
0	(197) collectins.
a	
0	Diedice of anconized . Decentar -> enoultment
0	Binding of opsonized -> Receptor -> engulfment
0	{ 1 courtage
2	Phagalybrome attach actiation
9	to degradation of Missoles
	Remation of Phagocytic vacule
8	Vacile
	niceday —
a la	Inocady

Date:	
Morphological Changes:	
There are Four morphological changes.	
O- Serous Inflammation	
@- Fibrinous onflammation	
3- Suppurative Inflammation.	
g- wer.	
O- serous Inflammation:	
The second secon	and
O- outpouring of water fluid dep	
with less protein of in	رمورا
@- Secretion from.	
Fluid in serious 6- Plasma	
and cavity 7 effusion Q- Mesothelia Cul Jining Prest	
Pericar	
periton	NOT .
3- Accumulation of serous effusion either	
within epidermis. beneath the epidenmisof skin.	
Example:-	
O- Vival Infection	
@- Skin blister.	
The state of the second section of the section of the section of the second section of the sectio	
	-
nicoda	

R

	Date:
Fibrinous Inflammation:	Marchelofal
> Severe anjury	V
Toy total land to the land of the	7- 82 97 9TV
1 Vascular Pormeat	510Hu
L L	Minister of the parties
allow large mo	leale
to pass through	h The
endothelial Bo	arrier (Fibrinogen)
	Ų
> 9nflammation of Boo	
such as meninges, pl	Leura and
Perican diam	
both to my odd stay with	
→ Exuadate com Remove E	by Macrophages
100 0 10 10 10 10 10 10 10 10 10 10 10 1	Fibrinotytic.
Lead to Res	V
0.014.014.001	
of more fibrin o	cure.
No Resolution	13.0
with the wind the second of th	white willing
organization	ocure
new	ly B-Formation Fibroblast
Scar form	alton
Example:-	HE TO THE REAL PROPERTY.
Fibrinous p	pericardium onflammation.
	i magori,
	niceday –

Date:
Suppurative 9nflammation: punlent.
Pus collection (Neutrophills, necrotic cells and edemal fluid)
Caure by pyogenic (pus forming) Such as staphylococci.
Abscesses formation. Local collection of pus. have central large necrotic Region.
neutrophills Rimmed by preserved peutrophills Limined by preserved neutrophills surrounding zone of Blood Verrele
surrounding by fibroblast prolification scar formation.
Ulcer:- Local defect or extravation of the surface of organ. occur duo to necrosis of tissue " Inflammation near a surface
Commly occur in G17 Uninony Tract Subcutoneous tissues af law extremities of old people Scar-Formation + accomulation of Lymphocyte

Peptic gostri won.

Define Inflammation?

Inflammation is protective Response involving host cells blood vessele, Protein. and madiator to eliminate the cause of injury as well as necrotic cell and process of repair staril.

- 150 A Develop States of Post 9- In this process dilution of harmful agent destorying
- D- Repair and healing occure
- (3- 9t is beneficial Rx and normal (4)- act as innate immunity.
- of prolong cause chronic inflamm ation (S)-

Briefy mention of morphological Pattern of acute inflammation?

There are four pattern of acute inflammation

- Serous Inflammation (I)-
- fibrinoid anflammation (2)-
- Suppurative Inflammation
- (4)-Uleer.



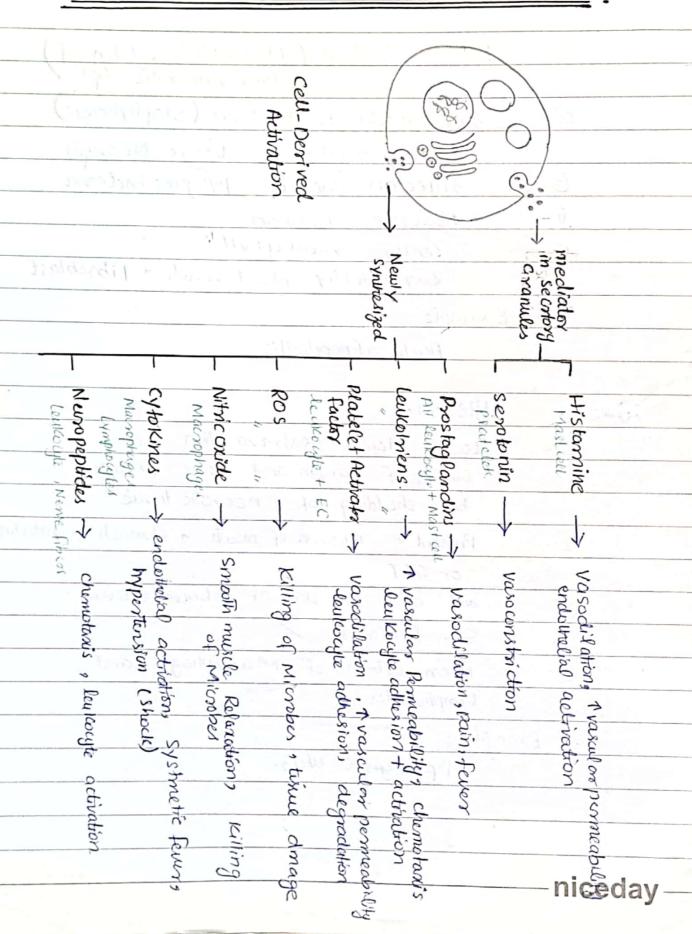
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<u>(1)-</u>	Serous Inflammation:
0-	serous grip igniniquon
<i></i>	Outnousing of water Fluid
U -	Outpouring of watery fluid e-less protein
<u>(2)</u>	Cacaretion Form .) Plasma
	-) Mesothelial cells linning
FU.	uid- effusion O- pleusa
	Q- · pericardiam
	3- · peritonium
3-	Accumulation of fluid within epidermis
	Example.
, U	Viral Infection
	Skin blister
	Lamiron type in Dendented in the way
@-	Fibrinous Inflammation:
0-	Severe grijvry Large protein leakage
2-	1 Vascular Permeability
3)-	Large molecule librinogen pass the membrane
4-	Fibrin is form Lyinto tirue.
G-	Deposite in EC space
<u></u>	of fibrin is not removed then
	Scar formation ocure
Exc	ample:
	Ocuse in body cavity of Mervinges
	Penicardium

D 4	
Date:	
111100	

<u> 31 00000</u>	suppurative Inflammation:
_	
()-	Pus collection (Neutrophils, debrisof) edema, necrotic Cell
	edema, necrotic cell
1	Infection due to bacteria (Staphylococci)
	raure liqufective tirrue Necrosis.
3-	Infection due to progenic bacteria
O -	Abscesses formation
5 -	Contex: -> neutrophy//
2.6	surrounded by blood vessele + fibroblast
	Example
	Acute appendicitis.
	Ficulty at period of the
Q-	ulcer :-
0-	Local defect + extravasation of
<u> </u>	surface of organ and tissue produced
	by shedding of necrotic tirsue
O -	Present in mucousa of mouth a stomach. Intestine
5	or GIT
3-	and also in skin or sabaceous tissue-
<u> </u>	Scar formation
6)-	Acemulation of Macrophager and
	lymphocytes
r~	ample:
E X	Peptic gastric Weer
	1970 0

Q :- Chemical Mediators of gnflammation:



Date: Protein Plasma Activation Csb-9 complex allock Factor XII -Ca, Csa, Csb) (amaphylation) plasma coagulation nooth musula contraction. Vasodilation, Pain Endothelial activation leukouste recouriment niceday

	Date:
G:- Describe <u>Vascular</u> change	% :-
Vascular Changes:	
Two changes	
O- Vasoclilation	(
@- 9nc Vascular Permeability	
O- Vasadilation:	
O- Vasodilation	v' ®
	Y
D- 1 Blood flow	
	stream
capillary bed	
@ - coure Redness	
stasis	
Heat	
swallowing	
Loss of function	
3- Inc Vascular Permeability	J:•
and the second s	
	Protein
0000000	, see the
O- gocreare Hydrostatic preriuse	
Q- Decreare Golloid osmotic Pressu	
3- Retaction of endothelial cells by	hi staning
@- and injury cause by bur	
G- 0.10 1 1	
	niceday

D .	•	100
Date:	41	-

(i) Exudate:	41290011	Colleger Cha
0 0		Fluid - Protein V
		leakage
)) @-	Vasodilation
	3-	Stasis
space	Ø -	Increase endothelial Space
Example	Ø-	high Protein Content
	6 -	1 Fibronogen content
② Tb	. D	high LDH
	3	Damage serous
		membranc
(ii) Transudate:		coagulating
		947(4
	0-	Fluid leakage
- Land	D-	+ osmotic colloid Pre-
)) ③-	dec Protein SynThesis
	<u></u>	anc Protein loss.
Fluid Ceakag	B	low Protein content
Example:-	<u>6</u> -	1 fibrinogen
O- Nephrotic Syndrome	<i>Ø-</i>	1 LDH low.
@- Pulmonary embolism	8-	1 hydrostatic Presure
	11	, : 1
in the makes that we		1 11.7
	Tight of	
	Example O Malignany To Transudate:- Example:- O- Nephrotic Syndrome Pulmonary embolism	Example & O- O- Example & O- O- O- O- O- O- O- O- O- O-

niceday -

G :-	
Cellular Changer:	
Cellular Changes: Steps of cellular changes	
O- Margination	
Q- Rolling	
3- Adhesion	
O- Trans migration_	
Margination:	
0- Blood enter in vanules.	
that's why of pushed	
Towards endother win.	
Towards endothelium. RBC small size	
3)- leu Koaytes align in a away from endonelium	
30000 grit powerug	
(TO) (B) Leu Kocyte Marginale	
endothelfalum.	
Paul in a second	
Rolling:- O- Rolling is mediated by Slectin	
here integrin in low affanity state-	
2- Selection Receptor on endothelial	
Ligand on leukocytes	
@ From type of colordin	ت
3 Three type of selection	
E-selectin L-selectin	

Pselectin.

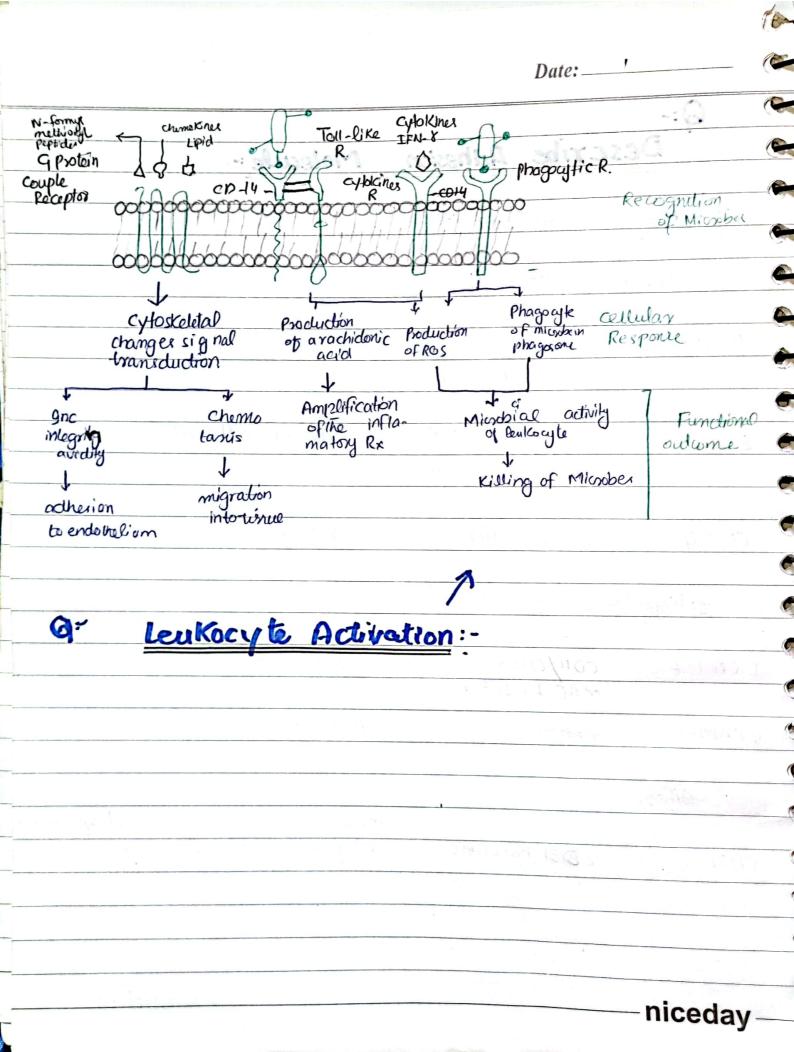
- Alaska	
Lselection	
(Garantegnio	
Selection Selection (Selection ()	
P & C034	
- selectin- Receptor on endothelial all	CD 62 E
selectin Receptor on platelets endothelium	CD63P
	CD62L
Adhesion:	
O- Adhesion is mediated by ontegrin	
here antegrins in high offanity state.	
3- Integrin Receptor on leukoujte	
but ligand on endothelium.	
(ICAMI) (ICAMI)	
integria selection antegria	
- Two yee of ligand of ontegrins	
, •	A
0- (ICAM-1) - bind to leukeagtes ass	ociated e- Unligen 1
0- (VCAM) bind to VLA-4-gntiger	1 4

	Date:
Q	Transmiarations
9	Transmigration:- O- Leukoute migrate through Vesselevall
	the sunction
	by squezing blw cell-and I/C Junction
	called dipediesis-
	@- This is occurredue to the CD31 dimmer
	dipedenis.
	(20)
	c031 & CO31
	3- CD31 hom type Interaction

Date:	
l lato	
Duic.	

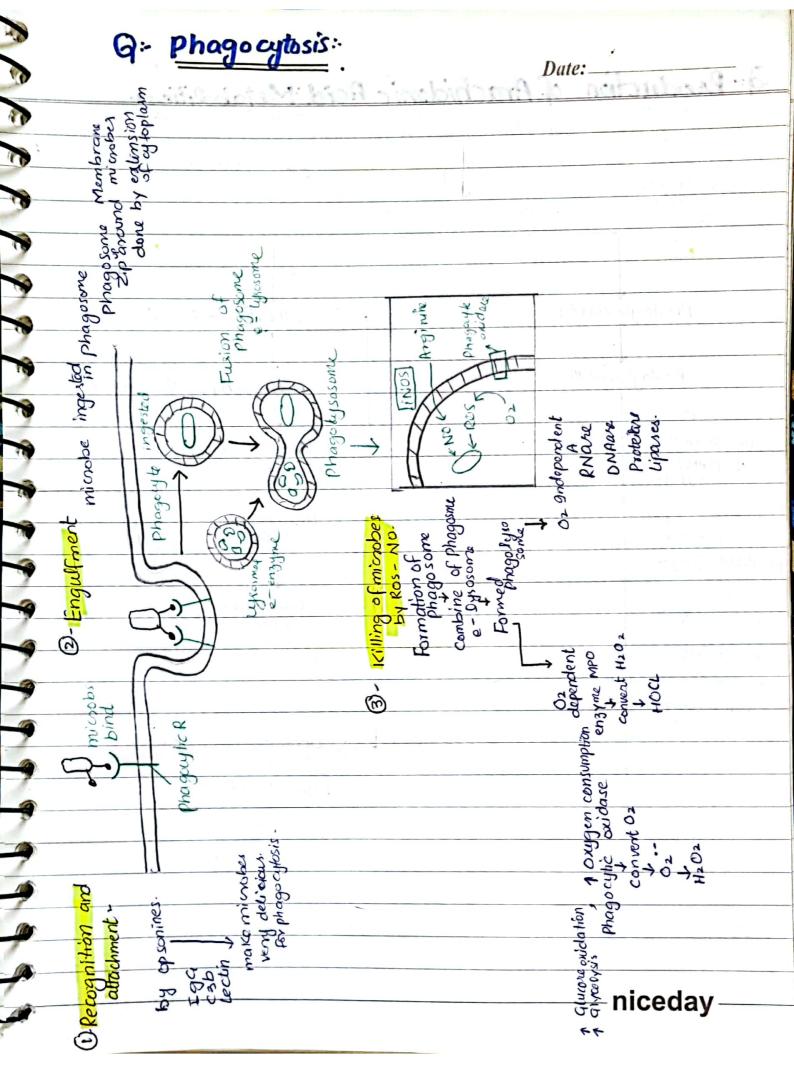
Desi	cribe Adhesion	Molecule
Endothelial	Leu Koc y les Molecule	Role.
Selectins	Selectin Ligands.	
		- letie L Lo
P- selectin	Sialy1-Lewisx	Rolling
	modified proteins	<u> </u>
	100	to the species and the second
E selectin	Sialy1-lewis X	Rolling + adhesion
	modified proteins	
		3 1 May 7 1 May 1 1 May
CD 34	L- selectin	Rolling
gntegrins	- 9 ntegnin Ligands	
<u> </u>	- Consta	de Legions & Adie
ICAM-1	0011/0018	Adhesion
<u> </u>	MAC 1, LFA-1	
VCAM-1	VLA-4	Adhesion
V C/		
OThers		
		Tran diaped
CD31	(B131 homotypic	Tran diaper Migration of burkoustes
	·	

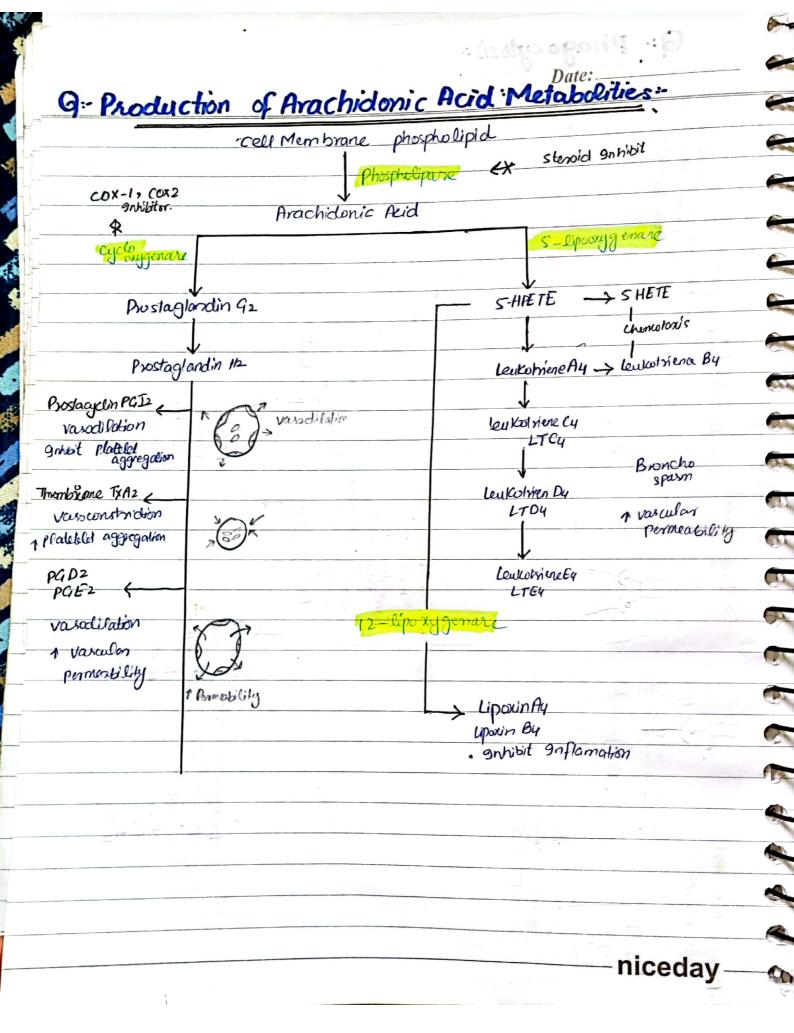
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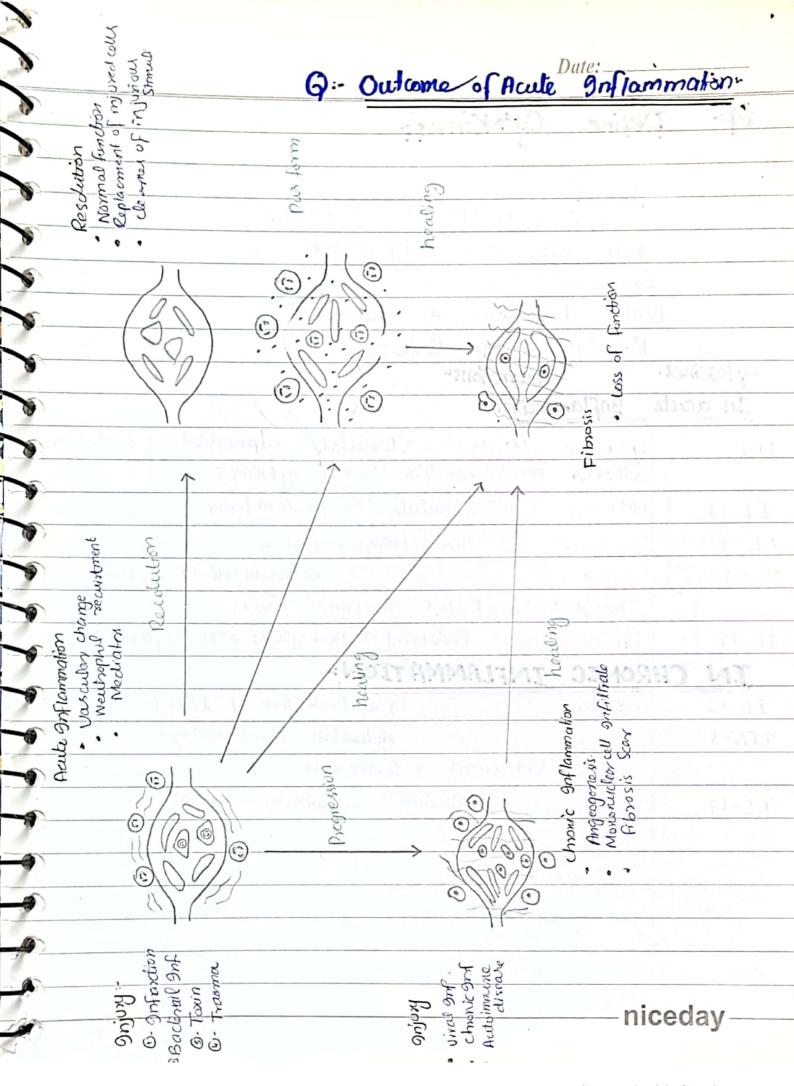


0	Date:	_
1	G: Chemotaxis:	
15	Desine:-	
	"Movement of leukoutes from	
P	verrele lumen into a damage	
-	area is called chemoloxis-1,	
4	Prouss:-	
	and the state of t	<u> </u>
1	Extravasation of Leukocytes	
1	1	
1	move towards site	
1	of infection along e-	
1	Chimical gradient called	
	chenotaxis.	
-		
9	can be exogenous endogenous.	
1	O-Bacterial product (N-formyl methionine Termini)	•.
7	Termini)	
9	@- (ytokines	
9	(chemokines)	
6	3- Product of AA	
1	(LTB4)	_
1		
1		
*	niceday –	
10	Iniocuay	

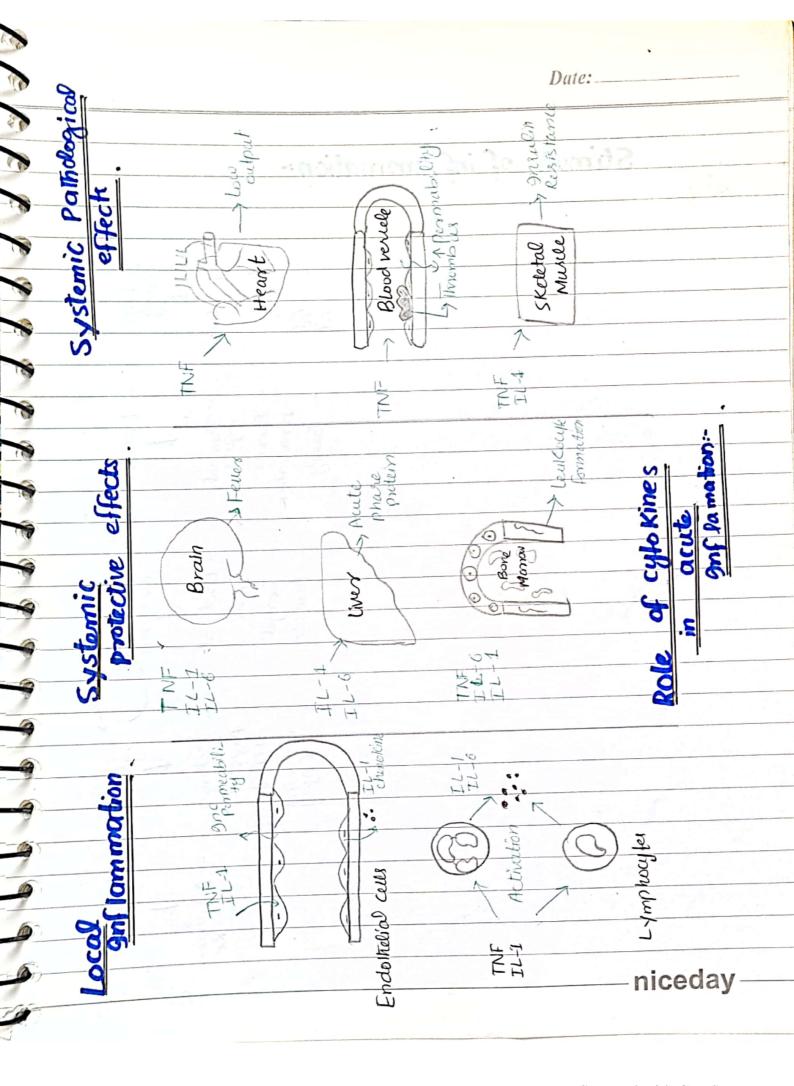
Date:	
Chemotaxic molecule.	
ψ	
Bind Receptor	
Releare cytoskeletoh contractile elements	<u></u>
contractile elements	
- Compation	
Leukocyte move by The formation of pseudopods	
Pseudopaus	5
anchor ECM	â
anchor EC 1	
Pull The leukocytes	
direction depend on chemotassis	-
Receptor	9
Leathers it is seen in	
by preudopality	
towards stimul.	
	6
Carried States and Carried State	-
	4
	-
nio-d	
niceday —	20

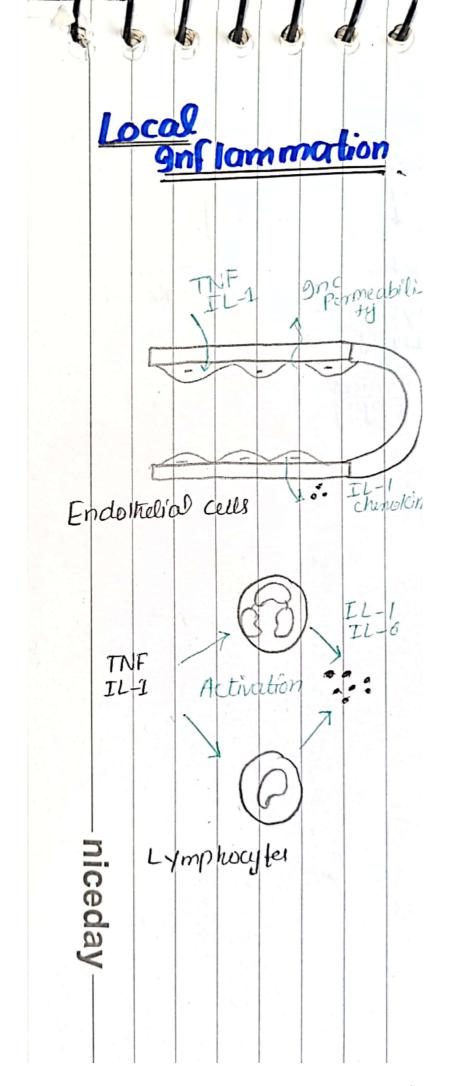


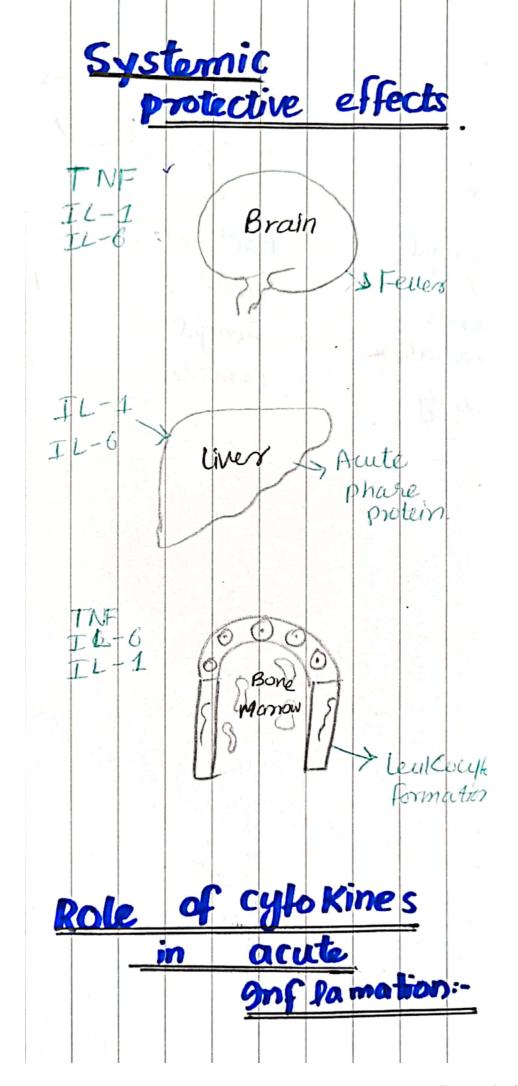


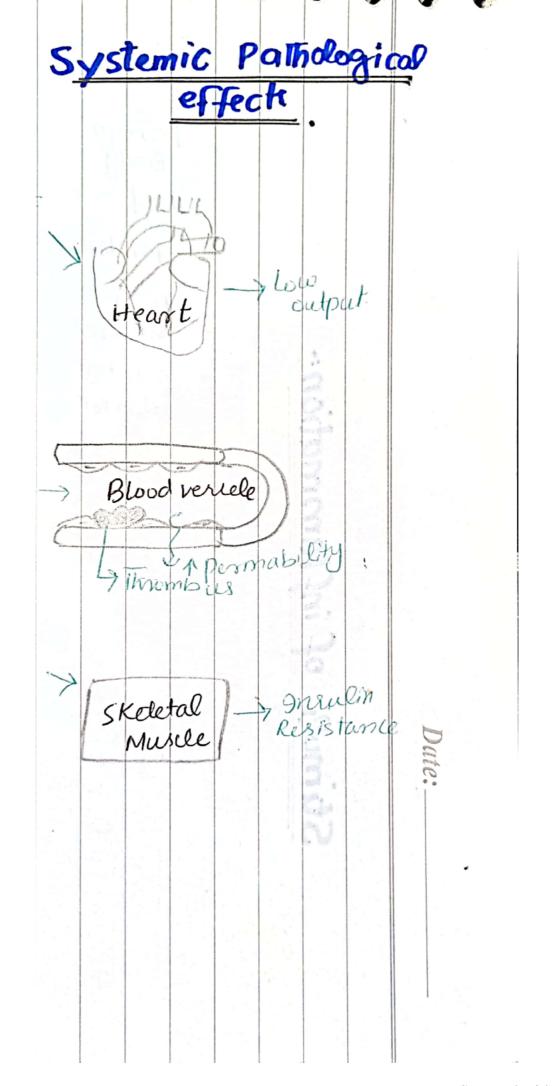


9:- 1	Define CytoKines:
	Define:
	Group of Low MW- polypeptide /protein
	which are secreted by activated
4	immuno cytes or some matrix cell and
	possess high activity and function:"
\rightarrow	Regulate Smune Response
Cytokines	Functions.
<u>In acut</u>	e 9nflammation:
TNF	(Macrophage/Mastell) Stimulate expression of endolheld
	adhesion and rectetion of other aytokines.
IL-1	(Macrophage) Stimulate TNF (Role in fever)
IL-6	(Macrophages) acute phare Response
Chemokines	(Macrophages, Trymphocylo, Masteer) - Recuritment of leukocytes.
	. migration of celle innormal tirule
IL-17	(T-lymphocytes) Remitment of neutrophills e- Monocytes
IN C	HRONIC INFLAMMATION:
IL-12	(Dendritic cell illacrophage) and Production of IFN-8
IFN-8	(Telymphouples NKCell) Activation of macrophages
	Kill microber - tumor cells
IL-17	(T-Cymphouter) Recitiment of neutrophill - monocytes
	niceday —









	Date:	
5		
Stimuli of infla	mmation:	- 1
		2
	())	- A
	peintess Dists Sutures Crystal deposit	2000 S
	7.6 6 3	Ň
	- Thermal injury Burns Thradiation	Tran
3	tiotion of the state of the sta	- dma
i i i i i i i i i i i i i i i i i i i	3	#
a a	())	4
	Backerial Virus Rumgas Panasite	20.
	Site Site	25.50
		70
	- 2 - 2 - 2 - 2 - 2 - 2 - 2 - 2 - 2 - 2	7
	Professions of the series	fection
	Awair.	Eq
	R. X	× Ž
	6 P	iooda
		iceday-

	Ques 1:-	Date:
3	Gues 1:-	
	1)- Pain in s	Right lower quadrant 12 hr.
		ny. Performed
		in wall of appendix
8	A	the contract to be a second
		olog: make rolls
8	(Acute Appendicitis)	acute suppurative Inflammation
		Symptoms:
		> Pus formation in Center
6 0-	loss of appetite	Then neutrophill
2-		p and Then surounded by
3-		+ diffated Blood verrele and
(ý)-	vom ting	fibro blast
<u>(G)</u> -		
(i)		Seesage Life Lyr 240
D -	- TWBC	And in the first termination
8-	Anovenia	Epident U. Gerthijke de Lepaine.
9-	Tendoness at MucBumy	points.
		Control of the Contro
(1)	Ques 2:-	The second of th
	12 year	old boy injury a tender, Red. hot
	swelling develop	ned at local site.
)——	Trace se	quence of event of this Picture.
	Ans:-	Mylphiza 4 min
	Redness	. tumos I symptom of
	loss of function	pain acute Inflammation
2)	heat	
9	We the second second	
7		niceday——

Date:
> Sequence of event of acute inflamation
Notes
Ques 3:-
cellular exudate form in acute inflammation
Ans: (a) What factor altroct WBC out of Blood Vesseler
Chemokines.
act on WBC and stimulate cell to migrate
Maugh spaces towards chemical conc-gradient
Chemokines IL-8
CD31.
(b) What are the object of their exudation out
of verrele? how do they achieve this objectives
Phagogytosi's process Notes
GN04:
CBC Test of 22 year old student
abnormal Leukocyte count
Weeks complain of sneezing - and watering eyes
in spring- summer of paralogation
Which is cell type mereare
Ans:
anc Release of histornine
in Response of allergic substance from
Mast cell- and basophills which
caure 1 existrophills

Date:
QN05-
25 old man Red hot fluctuant swelling in arm
after recieving IM-injection.
Local fluid exudate formation
Describe mechanism of Fluid exudate formation
Ans:-
Major Three Component
O- Vasodilation
@- 1 Vasular Permeablity
3- Emigration
CANCELLEY DESCRIPTION OF THE PROPERTY OF THE P
Explain Vasuelor Permeability Notes
Bisheltal sajen- water of fliction
QNO6:
30 old have wife , skin blisher e-serous fluid
Resulting from burn.
Name the morphological Pattern.
Ans:- Williams
Serous Inflammation
30/003 20/10/19/11/10
6N07-
3 Major component of acute Inflammation
f
Cellular.
Humsal Plasma Proleare

Platelets activate factor

Arachidonic Acid

Que. 8:-	Date:
	20 year old patient
To the first	Appendicitis.
	Poun in R iliae fossa.
	Appendent Removed (Red. Swell)
- 11 1/10	show edema.
	Enlist macanism caure edema
Ans-	Alagora Mario Companial
	Change in Vasuelar flow-
	vasodilation by histamine
	" NO. Whaping
	Vascular Permeability
	Space formation
	Endothelial anjusy-Leakage of fluid
	100/16
1.402.1	u a casa de la compania de la casa de la cas
	(5a,(3a 9nfla
	C3b Phagoulus
	the state of the s
	0
	Fevus IL-1-TNF
	Pair pg. Brady Kinin

Difference b/w

Acute Inflammation.	Chronic Inflammation.
D- Defi	nation
Rapidly reponding	gnflammation of prolong duration
Protective mechanism	in which tissue injured-
derivering of neutrophills	and attempts to repair.
to site of infection	
for cleaning The invaders	kishodstin i die in i i
and intiating The process	arena to the
of necrotic trisue	Prochy Krimin
Q- Major co	u involve
	E No Com Strickly and
Neutrophills	Mononuclear Cells
Macrophages	Monogte
	/ Fibroblast
3- 0	nset
9 mediate Onset	Delayed
JITOE ON GIO	
G- Dura	tion
Few days	up to monits.
1 200 01-1	
©- System	ic Manifestation
High grade fever	Low grade fever
.,,	/
6- Care	dinal Sign
Present	Absent
- 12 s, and 1 2 s	niceday —

		,	}
	She	Date:	•
O- outce	omes	32171314112	
Healing water simul		Fibrosis	Acute
Pus formation	inch-	Tissue destruction	
compact to a special k		1733000 00317700	1002
A DAVINGS AND A COLUMN AND A STATE OF THE ABOVE AND A STATE OF THE ABOV	Vesseles		
Vasodilation	VC33003	Parl'Contin	·h
30. (01/1017		Proliferation	· · ·
@- Med	diators	Marci set pains	ola di
Histamine	244.01	T- lymphocyte	W. Long
Braoly Kinin		qFs .	· \-
O- Vasu	wood cha	nae	
Vasodilaton	acor and	New Vessel	
1 vascular Permeability	1	formation	turk -
		and a secondary	
The property of the			
	The same		
bayalad		The board day	baine!
	TO LICE	4(1 4)	
est our man		The state of the s	Trans.
Treatest.	SCAT DIN	31.12	
Low grode from			WH.
The state of the s		Fred Gues	15111
	L Cost, T		
Rosent			Postory
		4	

Differentiate b/w

Date:_

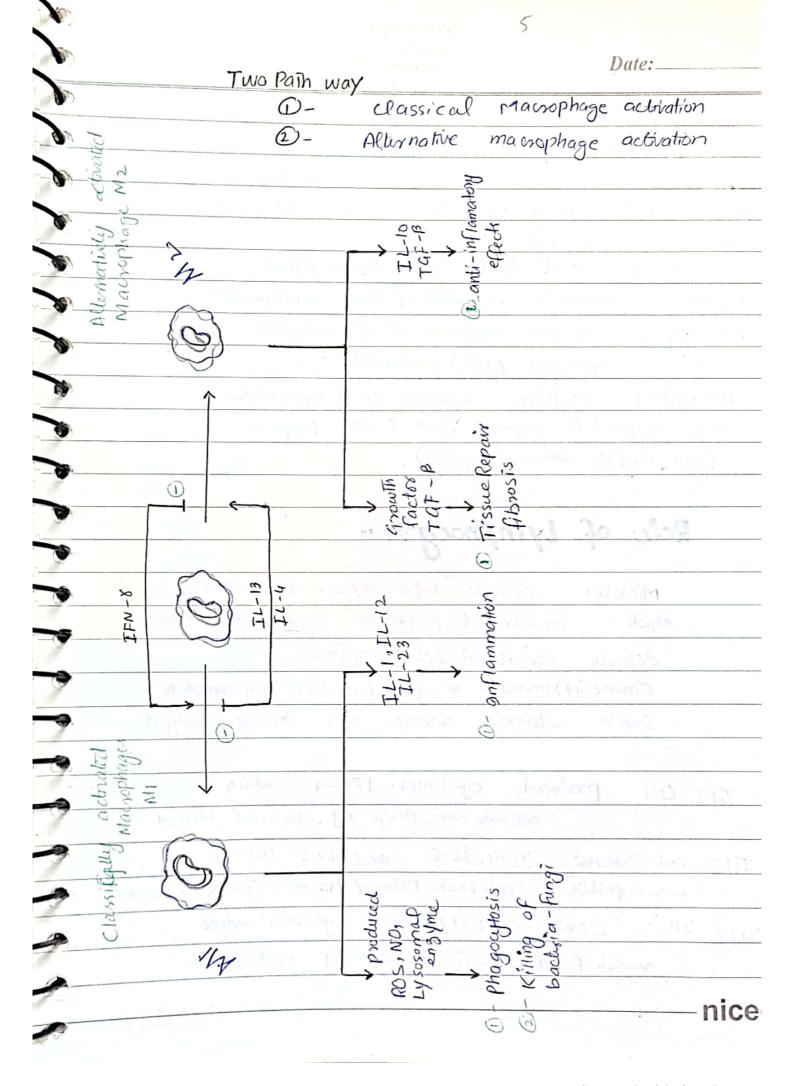
Fransudate	Exudate
O- 0.P	
1 osmotic Pressure	1 osmotic Pressure
17	Perfection of the state of
@- Feaburer	
1 Protein Joss	Damage serous membrane
1 hydrosatic Pressure	
3- Edema-	
No inflammatory edema	9nflammatory edema
Q- protein.	
1 Protein Content	1 protein content
@ coagulation	
No tendency to coagulate	coagulation
O- Fibrinogen	
No fibrinogen	Fibrinogen content
@- LOH	
LOW LDH	high LDH
®- Spaces	
Abrent	Ontenendo Thelial _space present
	niceday -

@- Leakage	Date:
Fluid leakage	Protein + fluid leakage
@- caure-	
Nephralie Syndrone	Malignancy
Pulmonary embolism	Tb
1 Diagram	7
Diameter Francisco	000 000
0 0 0	A hydre by Common or a second
va sedife	alian o o o o o))
teinloss	Spaces
fluid leakage	Protein 0
	The state of the s
Andrew wishing in	
	1 Proton Control - Section 1
A PERCUIPON TO THE OPEN	
	The Total occurs of the trade of
Contract Contract	
	Va lie proger and all all
FO 7 Will	Har-
the problem of the period	to the second

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110170	
LI III C.	The second secon

Define: 9t is a Response of prolonge cluration (months) Inwhich inflamation. tirrue injury - and Then Repairing owne prolonge of prolonge Ourse Prolonged exposure to potentially toxic agents O- either exegenous- endogenous O- persistant 9nfections: O- persistant 9nfections: O- either exegenous- endogenous O- persistant 9nfections: They may evoke such as mycobacteria They may evoke fungi > Virus, parasites They may evoke type hypersens	Chronic 9nflammation:	
cluration (months) Inwhich inflormations birrue injury - and then Repairing property to the polarity of the p	Desine:	
cause injury - and then Repairing positions occurse proposed from Repairing profit of the form of the	It is a Response of prolonge	
Course: O- Persistant Infections. O- Hypersenstivity disease O- Prolonged exposure to potentially toxic agent, O- either exegenous- endogenous O- ommune- Mediate Inflammatory diseases O- by microorganism that are difficult to eractic cate such as mycobacteria fungi, Visus, parasites imane Rx delayed type hypersens O- sometime acute bacterial Info of lung I progressive to chronic lung disease O- Chronic Inflammation Play imp role I disease which is cause by	duration (months) Inwhich inflamation.	tivi
Course: O- Persistant Infections. O- Hypersenstivity disease O- Prolonged exposure to potentially toxic agent, O- either exegenous- endogenous O- ommune- Mediate Inflammatory diseases O- by microorganism that are difficult to eractic cate such as mycobacteria fungi, Visus, parasites imane Rx delayed type hypersens O- sometime acute bacterial Info of lung I progressive to chronic lung disease O- Chronic Inflammation Play imp role I disease which is cause by	terrue injury - and Then Repairing portion	
O- either exegenous— endogenous O- either exegenous— endogenous O- persistant Infection's: O- by microorganism that are difficult to eradic cate such as mycobacteria fungi, Vixus, parasites imune Rx = delayed type hypersens O- sometime acute bacterial onf of lung I progressive to chronic lung disease O- Chronic Inflammation Play imp vole	prolonding from house demand and head	rδ
O- either exegenous— endogenous O- either exegenous— endogenous O- persistant Infection's: O- by microorganism that are difficult to eradic cate such as mycobacteria fungi, Vixus, parosites imune Rx = delayed type hypersens O- sometime acute bacterial onf of lung I progressive to chronic lung disease O- Chronic Inflammation Play imp vole	Cause:	
D- either exegenous—endogenous B- 9mmune- Mediate Inflammatory diseases 1 O- Persistant Infection's: O- by microorganism that are difficult to eradic cate such as mycobacteria fungi, Vixus, parasites imune Rx = delayed type hypersens D- Sometime acute bacterial Info of lung progressive to chronic lung disease O- Chronic Inflammation Play imp vole	10- Persistant Infections.	
O- either exegenous— endogenous B- 9mmune- Mediate Inflammatory diseases ** O- Persistant Infection's:- O- by microorganism that are difficult to eradic cate such as mycobacteria fungi, Virus, parosites imune Re in delayed type hypersens O- sometime acute bacterial Infection progressive to chronic lung disease O- Chronic Inflammation Play imp vole	D- Hypersenstivity diseare	
D- either enegenous- endogenous B- 9mmune- Mediate Inflammatory diseases 1 D- Persistant Infections: 1 D- Persistant Infections: 1 D- Persistant Infections: 1 D- By microorganism that are difficult 1 to eradic cate 1 Such as mycobacteria They may evolve 1 fungi , Visus, parasites mure Rx delayed 1 type hypersens 2 Sometime acute bacterial Infections 1 progressive 1 progressive 1 to chronic lung disease 1 D- Hyper sensitivity Disease 1 D- Chronic Inflammation Play imp vole 1 disease which is cause by	13- Prolonged exposure to potentially toxic agent,	
B- 9 mmune- Mediate Inflammatory diseases 1 O- Persistant Infections: O- by microorganism that are difficult to eradic cate such as mycobacteria They may evolce fungi, Visus, parasites imune Rx = delayed type hypersens D- sometime acute bacterial onf of lung progressive to chronic lung disease Hyper senstivity Disease O- Chronic Inflammation Play imp vole in disease which is cause by		
* O- Persistant Infection's:- O- by microorganism that are difficult to eractic cate such as mycobacteria They may evoke fungi; Visus, parasites mune Rx -> delayed type hypersens O- sometime acute bacterial onf of lung I progressive to chronic lung disease Typer senstivity Disease O- Chronic Inflammation Play imp vole	B- 2mmune- Mediate Inflammatory diseases	
O- by microorganism that are difficult to eraclic cate such as mycobacteria They may evolce fungi, Visus, parasites imune Rx = delayed type hypersons O- sometime acute bacterial onf of lung progressive to chronic lung disease Typer sensitivity Disease O- Chronic Inflammation Play imp vole in disease which is cause by	was a first than the same of t	
such as mycobacteria They may evolve fungi, Virus, parasites imune Rx delayed fungi, Virus, parasites imune Rx delayed type hypersons D- sometime acute bacterial onf of lung progressive to chronic lung disease The chronic lung disease C- Chronic Inflammation play imp vole in disease which is cause by	* O- Persistant Infection's:	
such as mycobacteria They may evolve fungi, Virus, parasites imume Rx > delayed type hypersons D- Sometime acute bacterial onf of lung progressive to chronic lung disease The chronic lung disease Co- Chronic Inflammation Play imp vole in disease which is cause by	O- by microorganism that are difficult	
fungi, Visus, parasites The type hypersons (2)— Sometime acute bacterial onf of lung I progressive to chronic lung disease Hyper Senstivity Disease (3)— Chronic Inflammation Play improbe in disease which is cause by	to erradic cate	
fungi, Visus, parasites The type hypersons 2- Sometime acute bacterial and of lung 1 progressive to chronic lung disease 1 D- Chronic Inflammation play improbe in disease which is cause by	such as mycobacteria 7 They may evolve	-
D- Sometime acute bacterial ont of early to progressive to chronic lung disease Hyper senstivity Disease D- Chronic Inflammation play imp vole in disease which is cause by	fungi, Visus, parasites Type hyperse	ทร
to chronic lung disease Hyper senstivity Disease B- Chronic Inflammation play imp vole in disease which is cause by	3. cometime acute bacterial ont of sung	
# D- Hyper sensitivity Disease (D- Chronic Inflammation play imp vole in disease which is cause by	to choic lung disease	
(b)- Chronic Inflammation play improve	JARDER DESCRIPTION OF DESCRIPTION OF THE PROPERTY OF THE PROPE	
(b)- Chronic Inflammation play improve	+ 2)- Hyper senstivity Disease	
in direare which is cause by excessive abnormal activation of immune system	B- Chronic Sinflammation ray improve	
of immune system	in direase which is cause by	
	of immune system	

Date:
3- Auto immune diseare Ocure.
On this autoantigen evoke
a self-perpetuating immune.
Rx That result chronic
tirrue damage
Example:- Rheumatoid arthritis
Multiple Sclesosis.
· Allergens - bronchical asthma
A 3- Prolonged exporuse to potentially toxic agents
either enogenous- endogenous:
De garagnie Wertelle De Caronited discorde
O. Exugenous agent → sillica nondegradaable
anhale for long time coure lung Diceaue Silicosis
@- Deposition of cholestral +1pid in the wall of arteries
An at a few times and the second seco
Commence of the same of the sa
* G- some other disease:
Neurode genrative direare
L> Al zheimes Diseare
Metabolic Syndron
Type 2 diabetes
For Married of Wally and Carlotter
mally 2 growing the
niceday—



secrete medialio

scar form

Function of Macro phages -

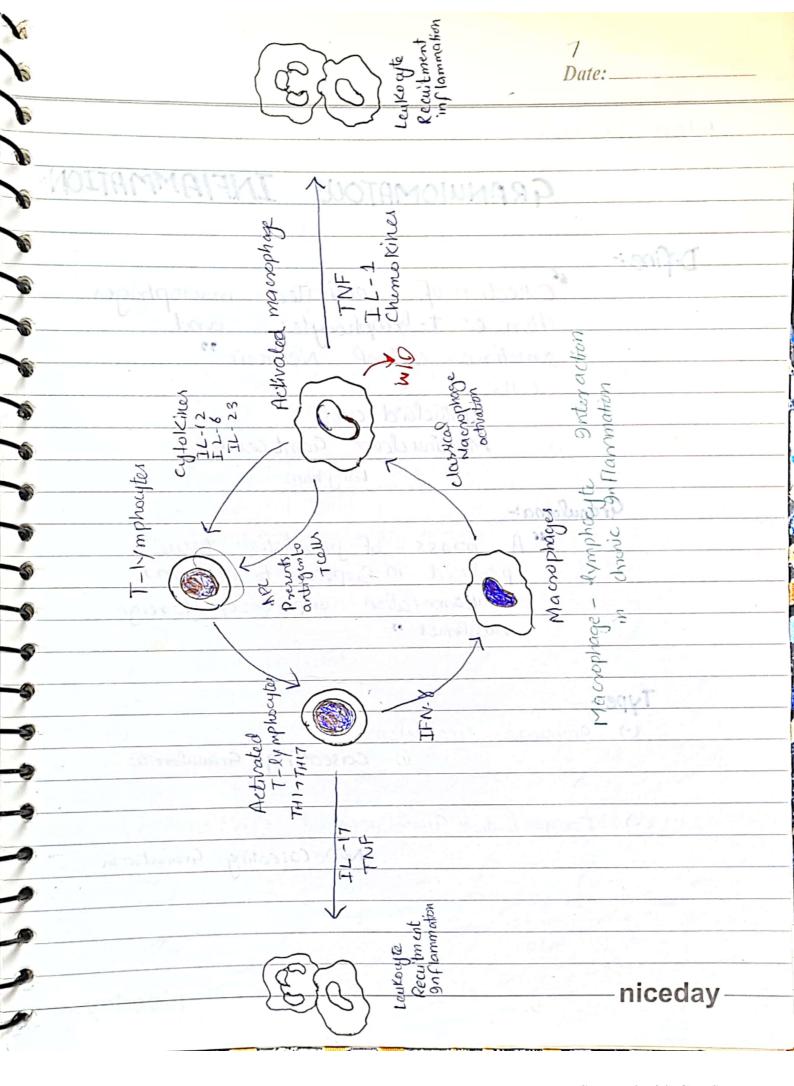
- Mossophage is a phagosytes to eliminate and ingest <u>()</u> and dead cells. Microbes
- Macrophages Initiate The Process of tisrue Repair 2)-
- Macrophage involve in formation of scar and fibrosis. (3) -
- Macrophages secrete mediator of inflammation (4)-Such as cytokines (TNF) ecosanoid
- Mocrophage display antigen to T-lymphaytes **6**and respond to signals from T cells help in (cell mediate immune Response).

Role of Lymphocytes -

Microbes activate T-Lymphocytes - B-Lymphocytes. which amplify and propagate chronic Inflammation défense against enfections pathogens. Chronic inflammation > granulomatous inflammation. Seen in autoimmune direare and hypersensitivity diseare

produced cytokines IFN-Y which THI COLL activate mausophage by classical Method

TH2 all secrete IL-4, IL-5 and IL13 activate eiosenophills alternate Pathway For Manophage activation THIT CELL secrete IL-17 and cytokines induce secretion of chemokines for recuiting neutrophills



B-lymphocyles, Mastall, Neutrophills

GRANULOMATOUS INFIAMMATION:

Define -

"Collection of activated macrophages often e- +-lymphocytes and sometime central Necrosis Cells

- Epitheloid cells
- Multinu clear Gaint cells.

Lang-hans

Granuloma:

mass of granulation tissue produced in Response to infection, Inflammation or presence of foreign substances "

Type:

(9 gmmune Granuloma

O- Caseating Granuloma

(.) Forign Bodies Granulomas

Non Caseating Granuloma

- niceday -

9		
Date:		
Duic.		

Types of Repairing.

Two-Lypes

I Healing of skin wound

Fibrosis in the injured parachymal cus

Healing of skin Wound:

step.

O- Epithelial regenration was morning dusti

O - Formation of C.T scan

Type of healing

Primary healing

2nd intention, 2ndary healing

First intention

- clean unifected (I)-
- surgically incised
- (3) –
- With less cell and tirrue loss
 Edger of wound are approximated by surgical sutures Scenty granulation, Neat linear scor

gnitial hemorhage + Hemostatic Function:

Incison

destruption

epithelial membrane peals of epithelial cell of connective tissue Death

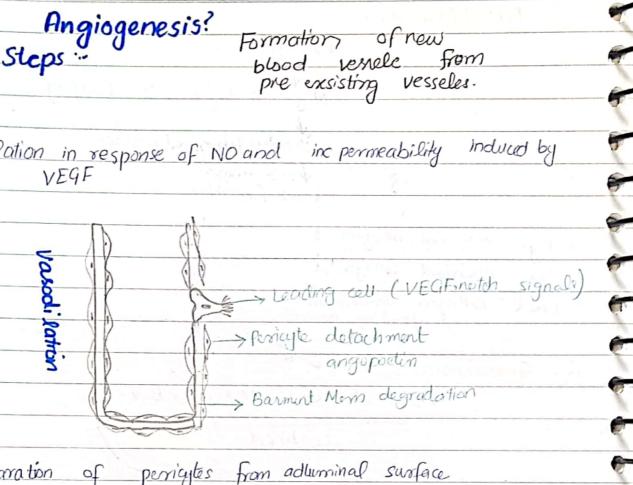
3- Months.

Recontmont of preendotholial colls
Suppresses of entothelial prolification - migralion

Angiogenesis? Steps:

Formation of new blood versele from pre exsisting vesseles.

①induced by Vasodifation in response of NO and VEGF inc permeability



- of pericites from adhuminal surface 2)-Separation
- of endothelial cells toward area of Migration 3-Orpanisation injury.
- Prolification of endotholial cells 4 -
- into capillary -lubes. Remodelling **O**-
- prolification + Migration Suppression of endothelial **6**-
- of B. Membrone Deposition 0 -

niceday-

Rentridaling -

	Date:
	Secondana heading
A Commission of the Commission	A Periogte Reconsistment
	ECM
	elongation of vascular stalk
and the same of th	The sharment Mem.
Comment of the second	; endotholium
	Acitios basemarshage:
	Bar 6 John A. Lange Bridge
3	
	9nflammatary Phase:

Secondary healing:

Open wound.

Large tissue defect

1 loss of tissue and cells

Wound not approximated by surgical suture.

Slow process

Ugly scar formation

O- gritial haemorrhage.

Wound space is filled e blood and fibrinclot



large defect

10- 9nflammatory Phase:

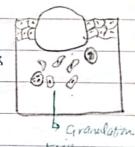
acute Inflammation

Appearence of macrophages

3- epithelial changes:-

Proliferation of epidermal cells

and migration of epidermal cells
Angeginess

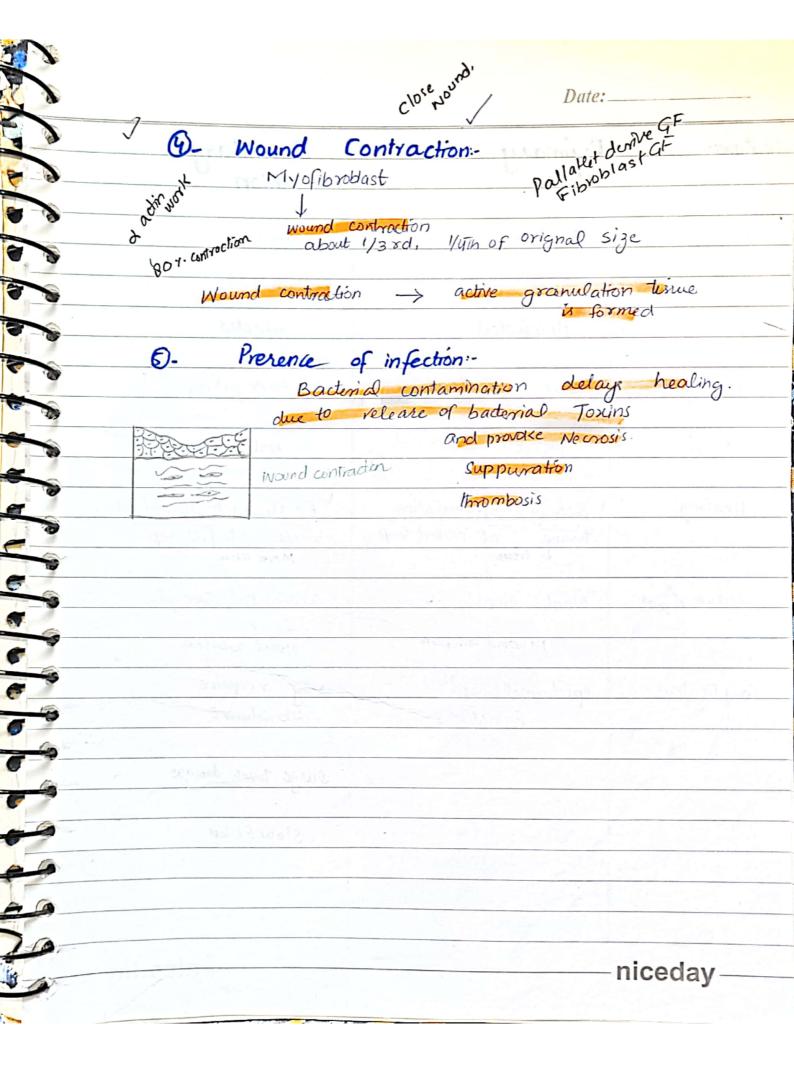


(9- Granulation Tissue:

Proliferation of fibroblast and neovas cularization

> deep red

very fragile



BBBB Date:

cleanliness of	Union	
Ue antiness of		union
Cleanliness of		
	Clean	unclean
wound.		
		Project Laborator
9nfection	Uninfected	9nfected
	of infection-	E. Presence
Margins	Surgical clean	grregular
Sutures	wred	Not wred
Healing	Scanty granulation	Exuberant granulation
	tirrue at incised Gap	Wire to fill gap
	V tisue	Move tissue
Out come score	Neat linear Scar	Contracted Omegwar
		Scar
	No wound contract in	Wound contraction
Complication	epidermal cyst	-May require
	Formation	debyidment
	Small dange	Darge Tireno damago
V	Ropid pi	Slow Process
		niceday

Complication of Wound healing:- 8- Injection - Of wound due to bacteria delay healing 9- Injection - Of wound due to bacteria delay healing 13- Pigmentation - Prist like enfour due to staining e haemosidin 13- Deficient scar formation - Inadequate formation of granulation tirue 15- Injectional huminian Weak scar formation 15- Lyportrophied scars - 1 scar , agly and painful 16- Hyportrophied scars - 1 scar , agly and painful 16- Keloid formation 1 contagen formation (Claw like) + Klevid. 1 scar twice graw beyond the boundaries seen in blacks 1 seen in blacks 1 formation of Cicatrisation		
8- 9njection - Of wound due to bacteria. delay healing Pigmentation - (epidermal cyst formation) Pigmentation - Rust like colour due to staining e haemosidin P- Deficient scar formation - Onadequate formation of granulation tirrue B. graisional humician Weak scar formation Lymay be site of bursting open of a wound Weak scar formation Congressional humician open of a wound Congressional formation A collagen formation (Clawlike) 7Klepid. Scartuine grow beyond the boundaries delivered on regression seen in blacks O- Excessive Contraction expection 1 wound contraction may result in formation of Gicatrication		Complication of Wound healing
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B- Deficient scar formation: Bradequate formation of granulation where Bray be site of bursting open of a wound A collagen formation (Clawlike) The boundaries bursting open of Collagen formation (Clawlike) The boundaries bursting open of Collagen formation (Clawlike) The boundaries scartuice grow beyond the boundaries bursting open of Collagen formation (Clawlike) The boundaries seen in blacks Contraction: Contraction Contracti	2) -	9 mpl antation: (epidermal cyst formation.)
B- Deficient scar formation: Bruce Bruce Weak scar formation Lymay be site of bursting open of a wound B- Keloid formation A collagen formation (Clawlike) The boundaries bursting open of a wound Collagen formation (Clawlike) The boundaries scartuice grow beyond the boundaries seen in blacks B- Excessive Contraction: expected in formation of Cicatribation	3)-	Pigmentation: Rust like lolour due to staining e haemosidin
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Scartifice grow beyond the boundaries seen in blacks On Excessive Contraction: extension 1 wound contraction may result in formation of Cicatrisation.	<i>©</i> -	10- Age - (1)
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@- Excessive Contraction- externation 1 wound contraction may result in formation of Cicatrisation	se	continue army heyord the boundaries coop in blooks
expression 1 wound contraction may result in formation of Cicatrisation	ï-	
in formation of cicatrisation	0	
		in Constitute of Containation
, la sound Malgarial to .> Peyronie's disease	-	, love moderned Mulgadust to .> Peyronie's disease
(a) Desmoids. Dupuytren's contractures.	(A)	

Date:
(9)- Neoplasia:
Cardnoma formation
Factor Influencing Healing:
Local Factors:
8- Infection
&- Poor blood supply yundone
&- Forgnbodies
@- Movement delays wound healing
@ gonising Radiation > delays granulation time form
&- Exposure to UV light.
I will broken to see in the second
Systemic Factors :-
O- Age Anymid grant direction Deficercy hormond obstitute
VitC and zinc delay healing
&- Infection
&- uncontrolled DM
Bl Administered of Glucocorticoids

defect of newhophill forction and

Haemologic Abnormality.

23 mil 22 (1960) & marsy use Newtropenia.

	Date:
0- Keloids: and Hypertrophic scan	Factor from Blood-
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- Acumulation	ů ,
2 d = 24 complian	Scor.
Scar tirrue growbeyond t	oundaries of original
Wound, det Re	gress
(8) - Exuberant granulation (provid f	
1 granulation	
Protude abou	e level of surounding skin
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deformity our	Corne
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Scar Formation:

laked		1	
<u>(1)</u> -	Formation of new	blood verrele	(Angeogenesis)
②	Migration and prolifica		
3-	Deposition of C.NT		
4-	Maturation and Rec	ognization of fibra	sous tissue
	to produced isto	able scan	47
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3-		> Formation of Gran	watron Tosue
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0	0,000	> Scar formation	(4)(1) 20
<u>Q</u> -	00	C.T deposition	***
	000	a should	s attacked the

Growth	Factors : robanis : roban
	Functions
Epidermal Growth Factor (EGF) Nacrophages - calivary Gorkerathages	Milogenic for Karetinocytes and fibroblast Slimulates formation of granular tissue EGF
Transforming (GF) Macrophage-Keratinoaytes	Proliferation of hepatocytes TGF
Hepatocyte (GF) (Scaller) Fibroblast, stromal all	1 Prolification of hepatogles., 1 cell motility F
Vascular endothelial (GF) Merinchymal cell	Stimulate Prolification of endothelialcoll A vascular primeablety Anger 5+ VEGF
Macrophoger, Smorth, Kenton	activates Prolifertion of Fibriblast sendothelial cells ECM protein Synthesis PDQF W.C. Angw.
Fibroblast GF. (1.2) Macrophages, Mastell	ECM protein Synthesis, Stimulate Angeogenesia Nound con FGF
Tranforming (GFB): T-lymphoused, Massophazus smooth Munde	TGF-B. suppress cicute inflammation
Keralinouyte (GF) Fibroblat	Stimulate Keratinocyte migration,
	niceday—

	Granuloma	tous Inflammation
Direare	Caure	
	ating	
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	tuberclosis	activated macrophages simmed by
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		Lang hans Gaint Cell
	here in a profit	
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	Pallidom	V central cell necrosis
		6 1 H
Cat-Scratch	gran we	Vstallale granuloma
direare.	bacillus	V Central Granular debris
	1	v Gaints cells
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Leprosy	Mycobactnium Deprae	Non caseating Granuloma
		v Acid fat bacill in macrophage
Sacrcoidosis	Unknown	V Non case ating Granwloma. V activated manophages
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