RAMA UNIVERSITY

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FACULTY OF NURSING

Chapter-01

INFLAMMATION

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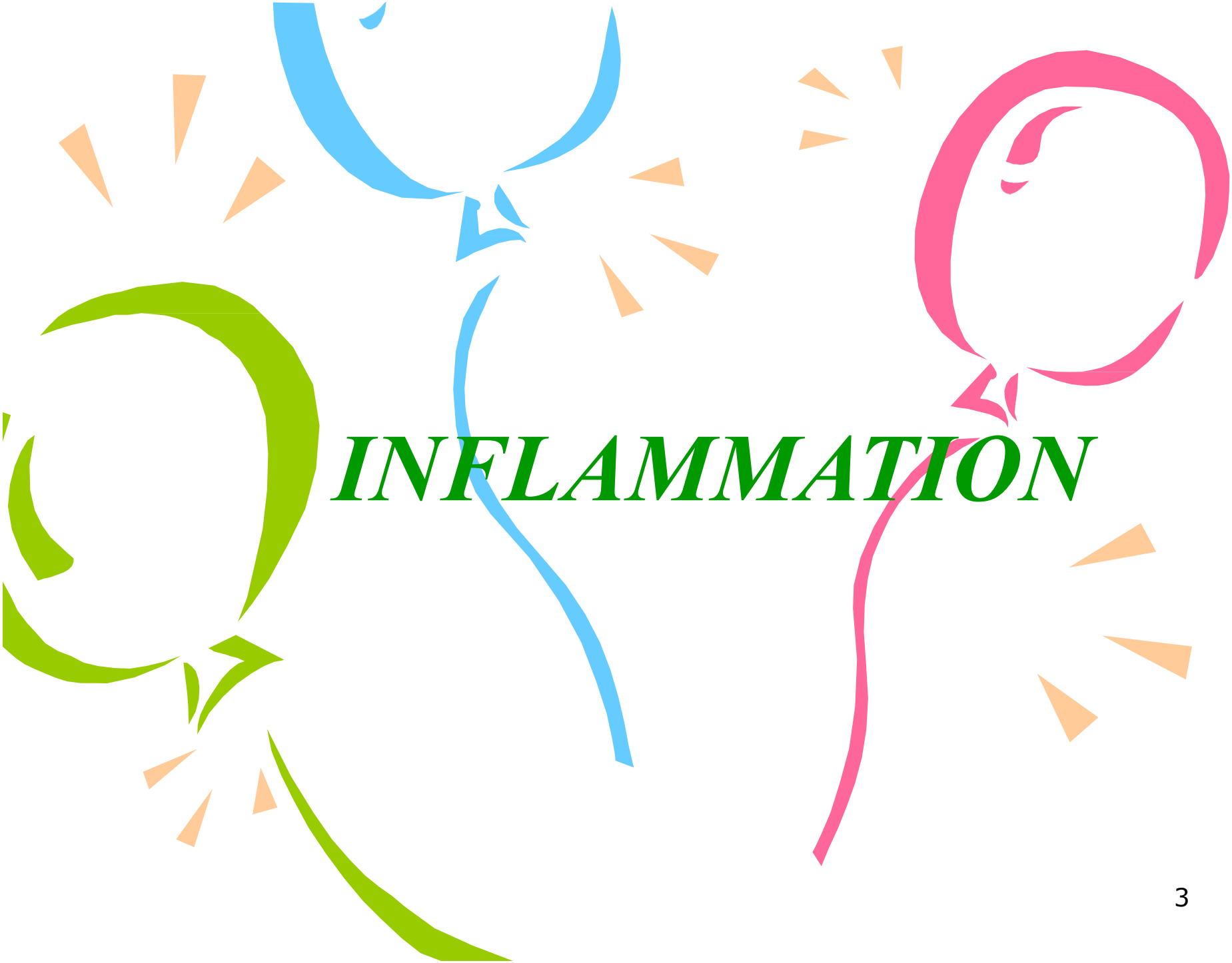
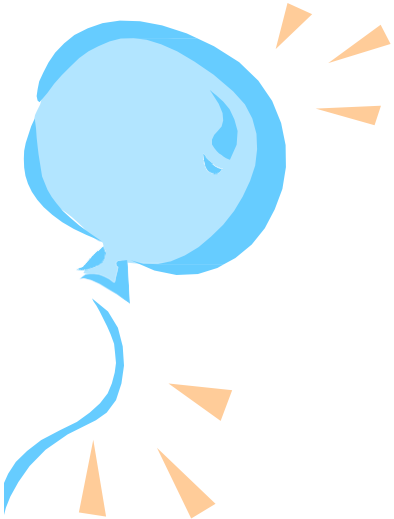
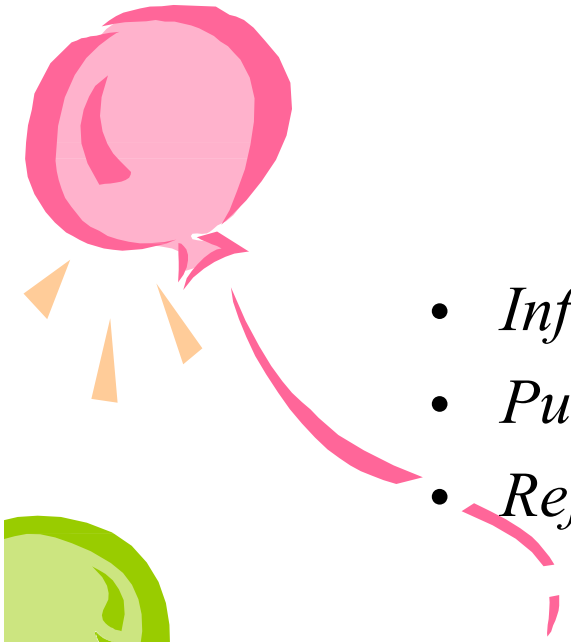
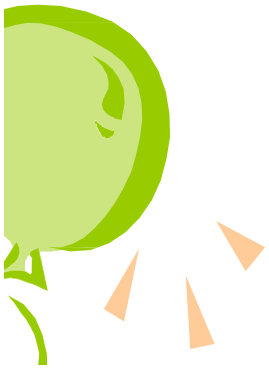
LECTURER (MSN)


INFLAMMATION







Contents

- *Introduction*
- *Defination & causes*
- *Signs of inflammation*
- *Types of inflammation*
  - *Acute*
  - *Chronic*
  - *Granulomatous*
- *Inflammatory cells*
- *Pulpal, periodontal, gingival inflammation*
- *References*

Introduction

Inflammation:

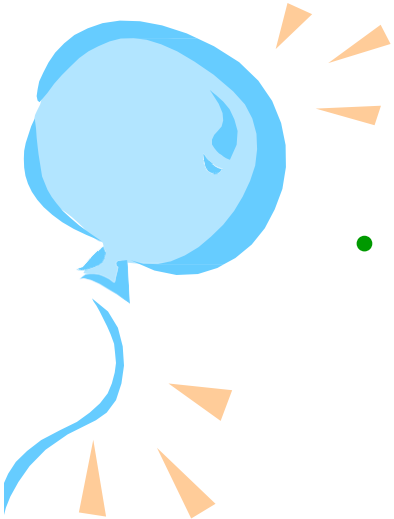
1. *To eliminate the cause of the tissue damage,*
2. *To repair injured and damaged tissue.*

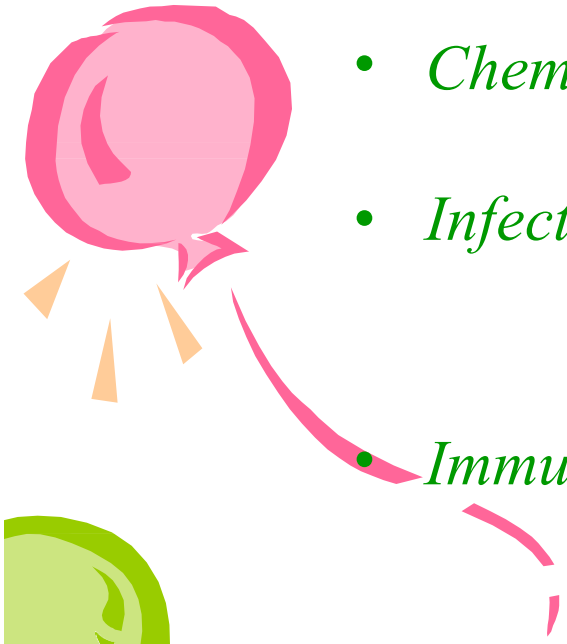
- *Inflammation is necessary for the survival of the host.*
- *In the absence of inflammation the body would be unable to kill and eliminate infectious agents.*
- *One of the innate defense mechanisms of the body.*

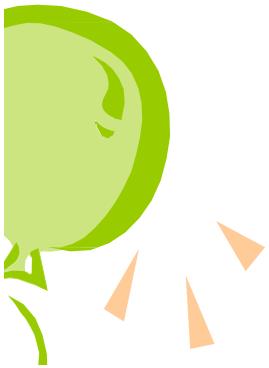
Defination

- *Inflammation is defined as complex series of events that occurs in vascularized living tissues in response to local injury or tissue damage.*

- *Inflammation is a programmed local tissue response peculiar to vascularized living tissues.* →

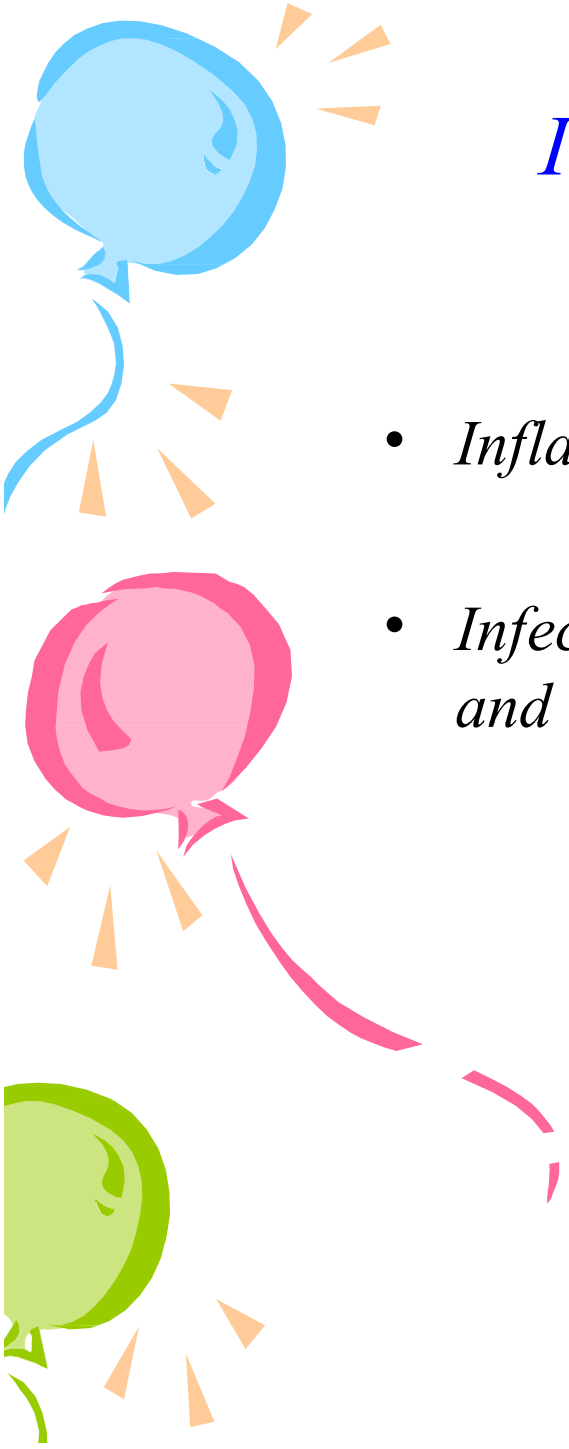






Causes of inflammation

- *Physical agents –*
  - *heat*
  - *cold*
  - *radiation*
  - *mechanical trauma*
- *Chemical agents –*
  - *organic and inorganic poisons*
- *Infective agents –*
  - *bacteria and virus*
- *Immunological agents –*
  - *cell mediated*
  - *antigen-antibody reactions*

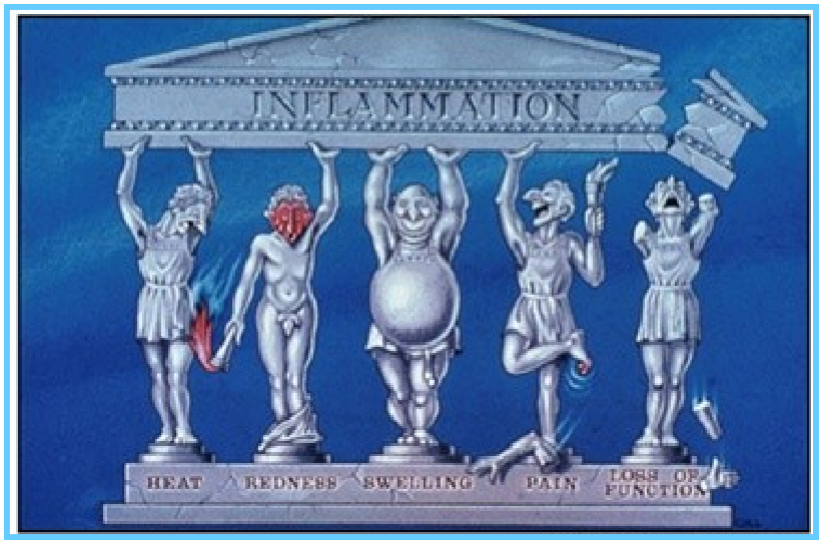


Inflammation and infection..

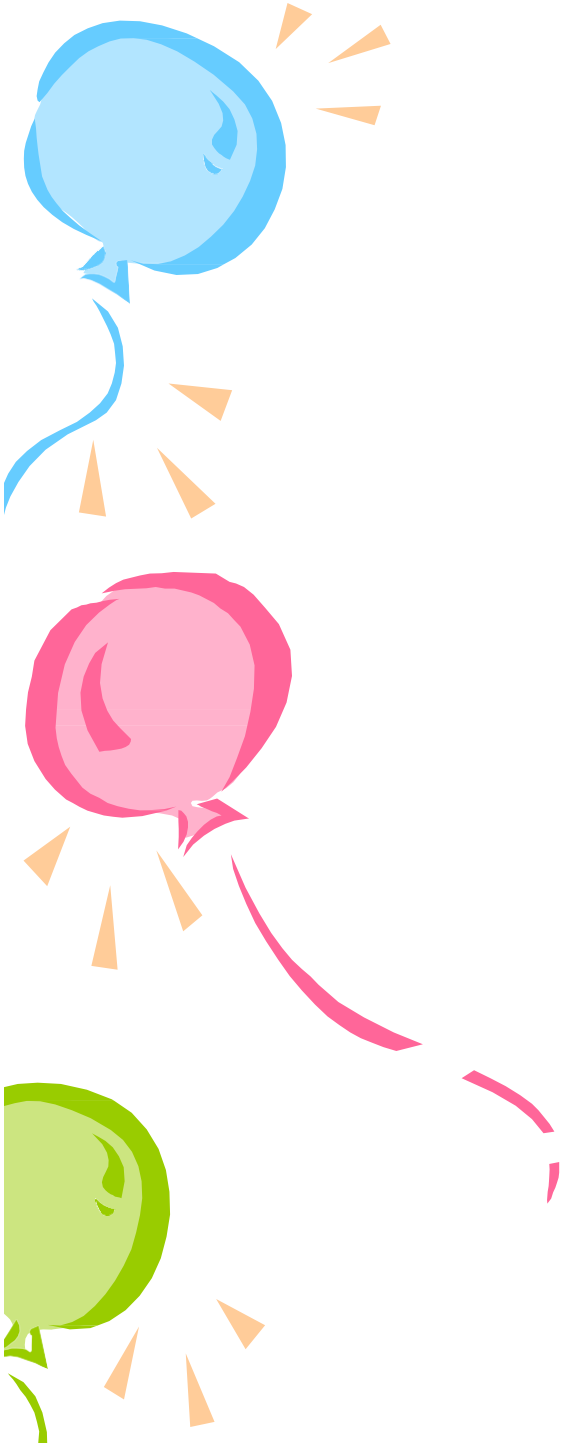
- *Inflammation ◊ protective response by the body*
- *Infection ◊ invasion into body by harmful microbes and their resultant ill-effects by toxins.*

Signs of inflammation

- *Rubor (redness)*
- *Tumor (swelling)*
- *Calor (heat)*
- *Dolor (pain)*
- *Functio lasea (loss of function)*



English	*Greek/Latin*	*Caused By*
Redness	*Rubor*	*Hyperemia*
Warmth	*Calor*	*Hyperemia*
Swelling	*Tumor*	*Increased vascular permeability*
Pain	*Dolor*	*Low pH*
Loss of function	*Functio laesa*	*Pain, swelling*

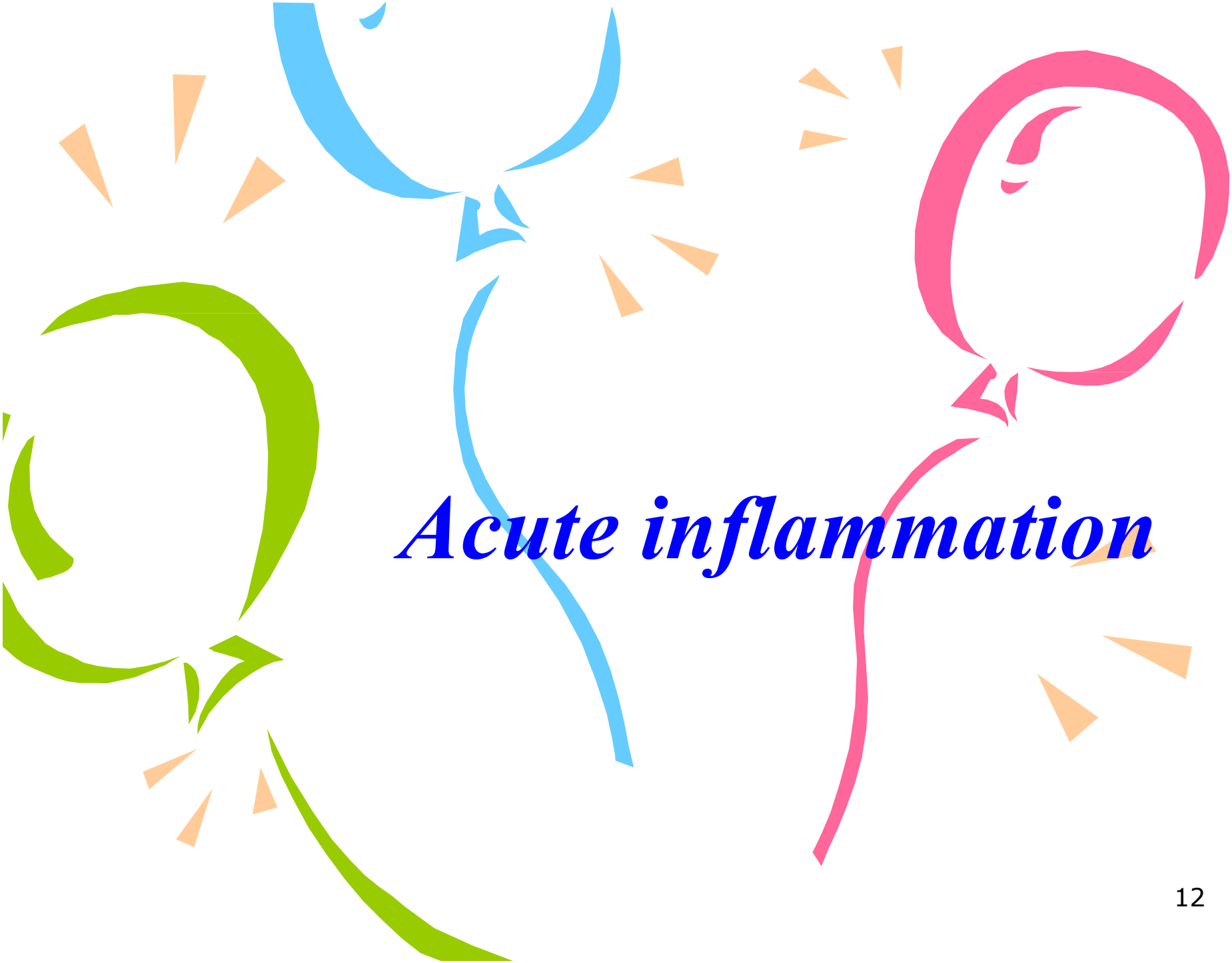


Classification

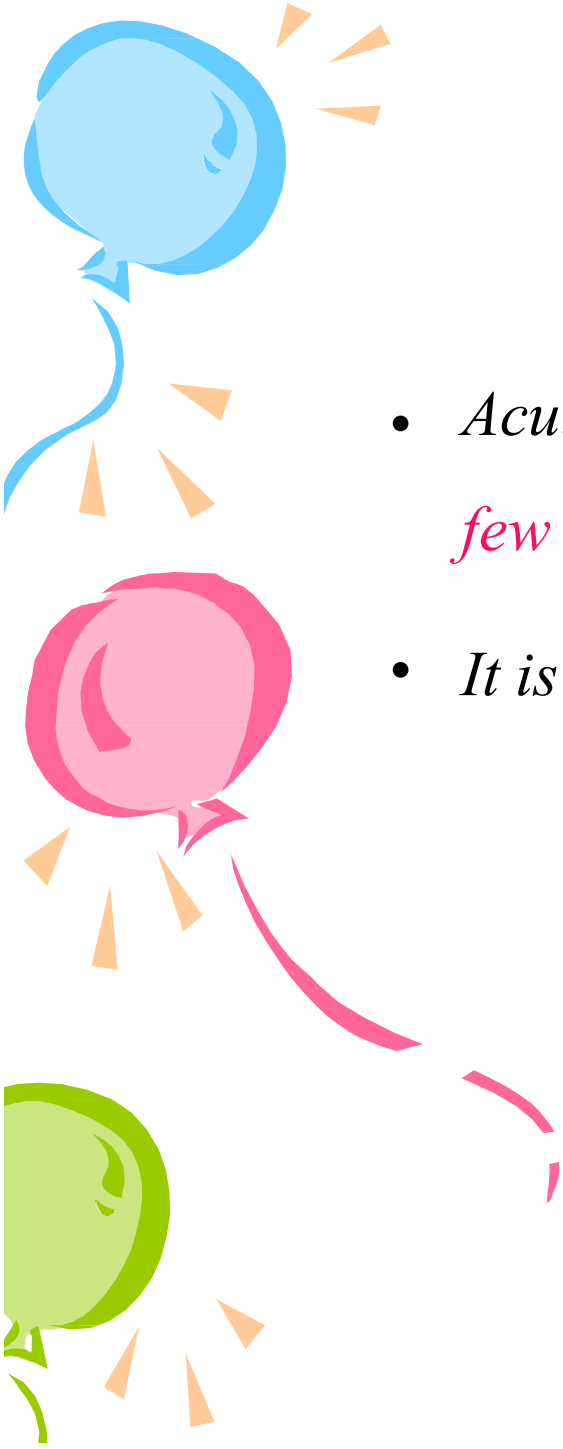
Inflammation (Duration & capacity)

Acute

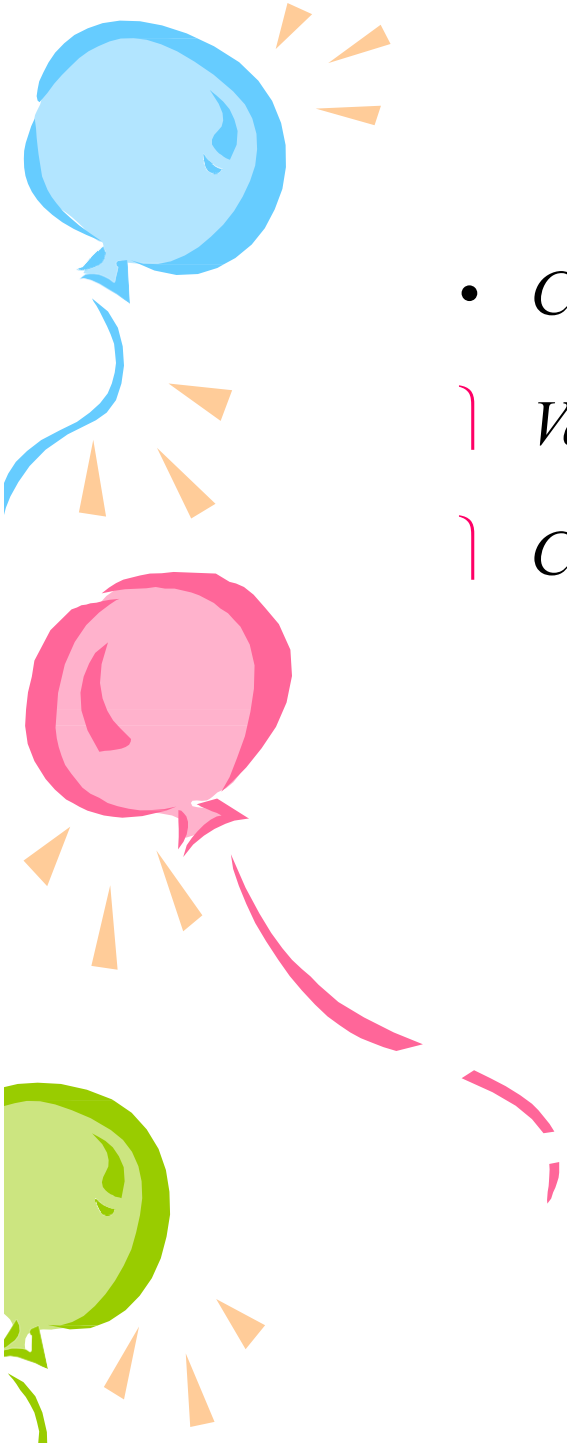
Chronic



Acute inflammation



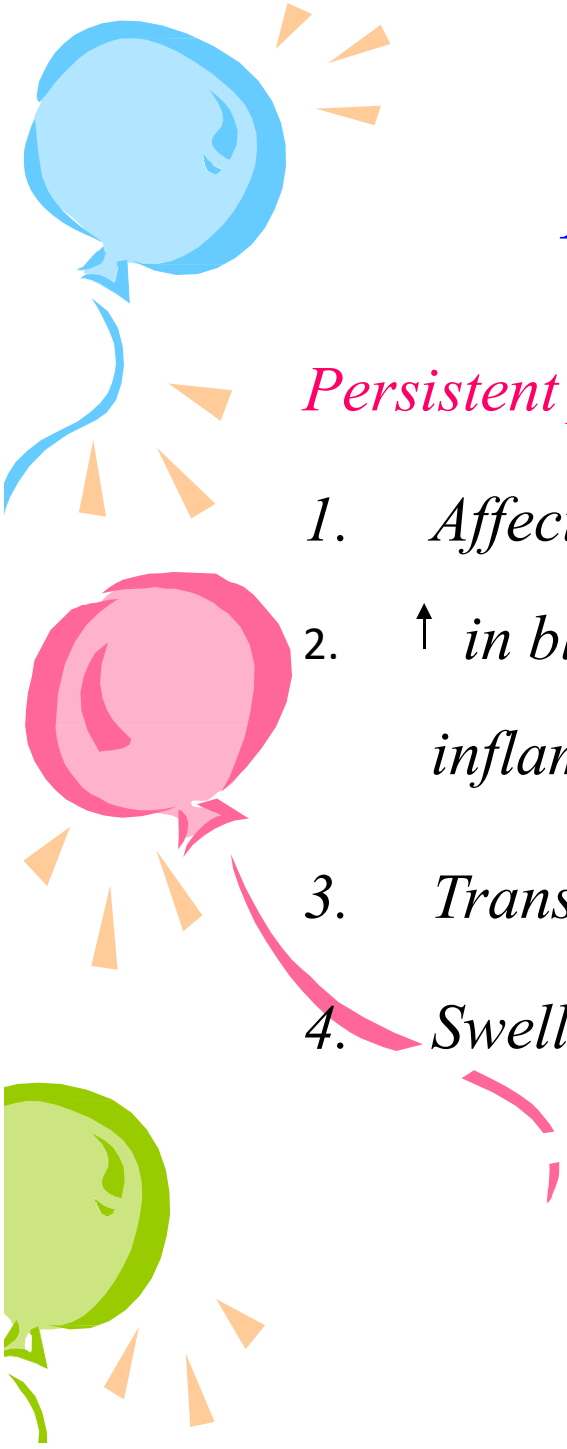
- *Acute inflammation persists for few minutes to few days and resolves on its own thereafter.*
- *It is a healthy response most often.*



- *Changes in acute inflammation…..*
  - *Vascular events*
  - *Cellular events*

Vascular events in acute inflammation

- *Haemodynamic changes*
- *Changes in vascular permeability*



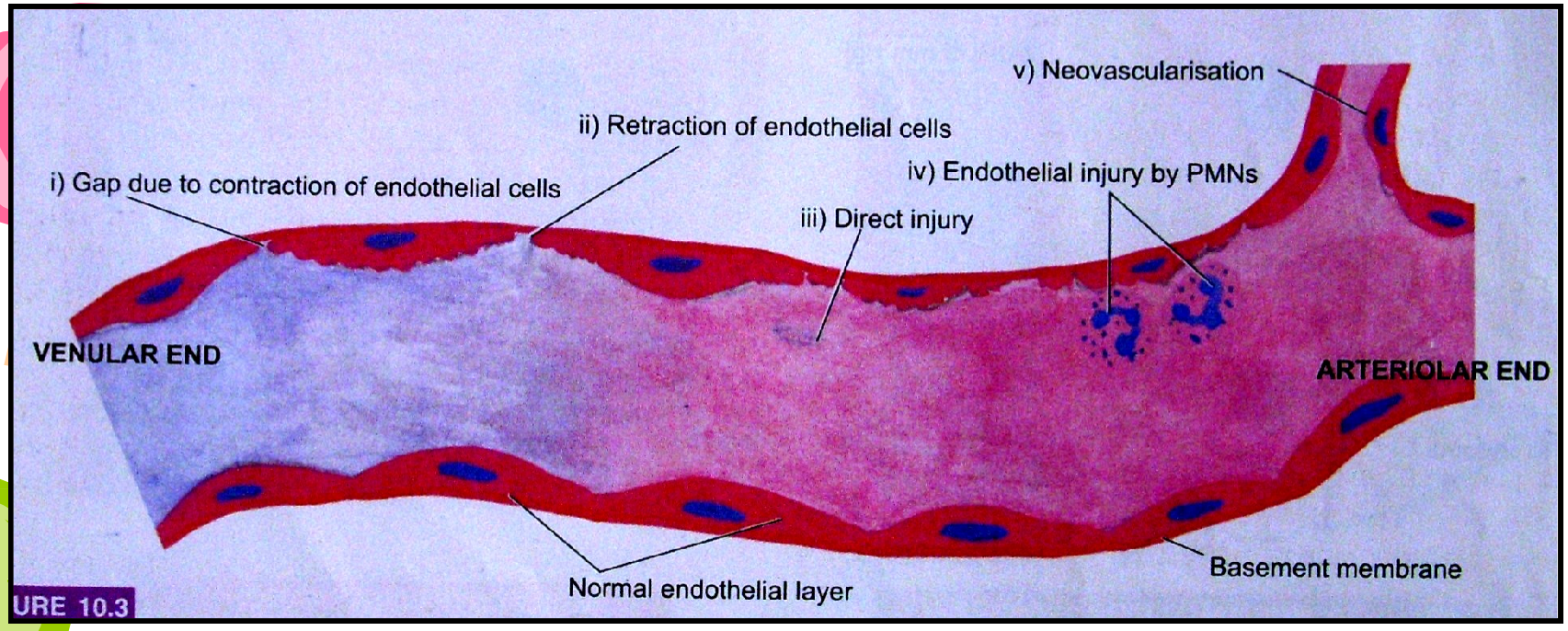
1) Haemodynamic changes

Persistent progressive vasodilation –

1. *Affects venules & capillaries*
2. *↑ in blood vol ◊ redness and warmth at site of inflammation.*
3. *Transudation of fluid in extracellular space*
4. *Swelling at local site of acute inflammation*

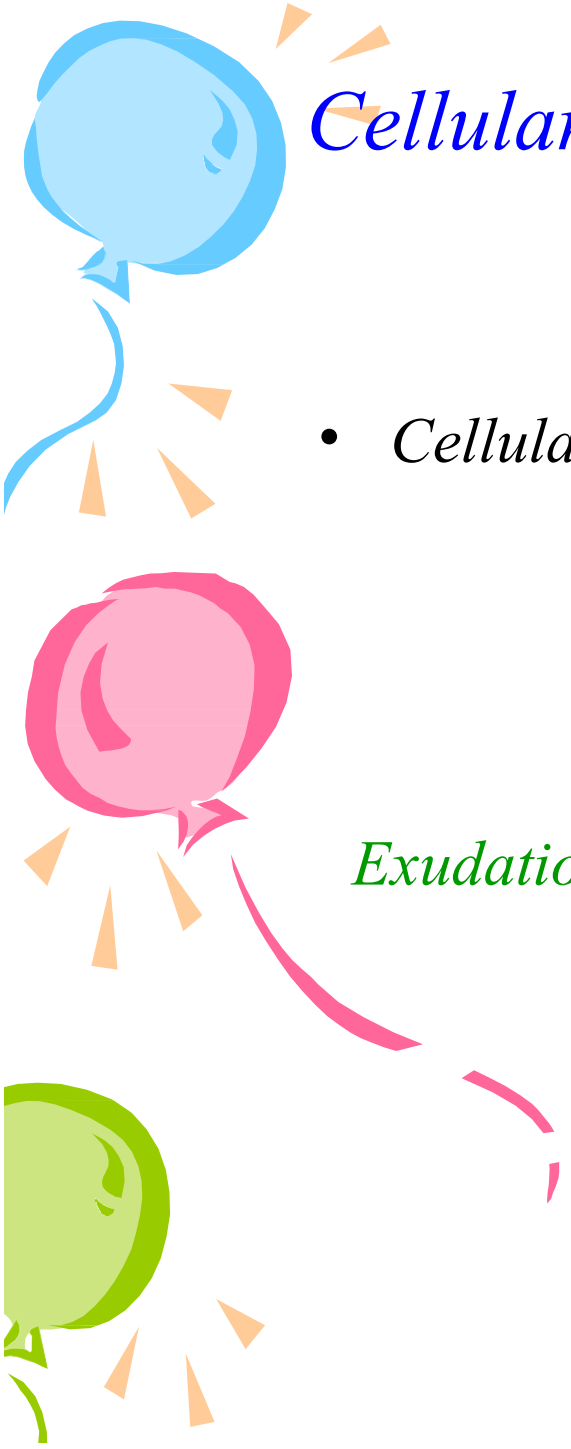
2) Increased vascular permeability

- *In acute inf normally nonpermeable endothelial layer of microvasculature becomes leaky.*



Cellular events in acute inflammation

- *Cellular phase of inflammation consists of 2 process*



Exudation of leukocytes

phagocytosis

1) Exudation of leukocytes

- *The changes leading to migration of leucocytes are:*

  Changes in the formed elements of blood

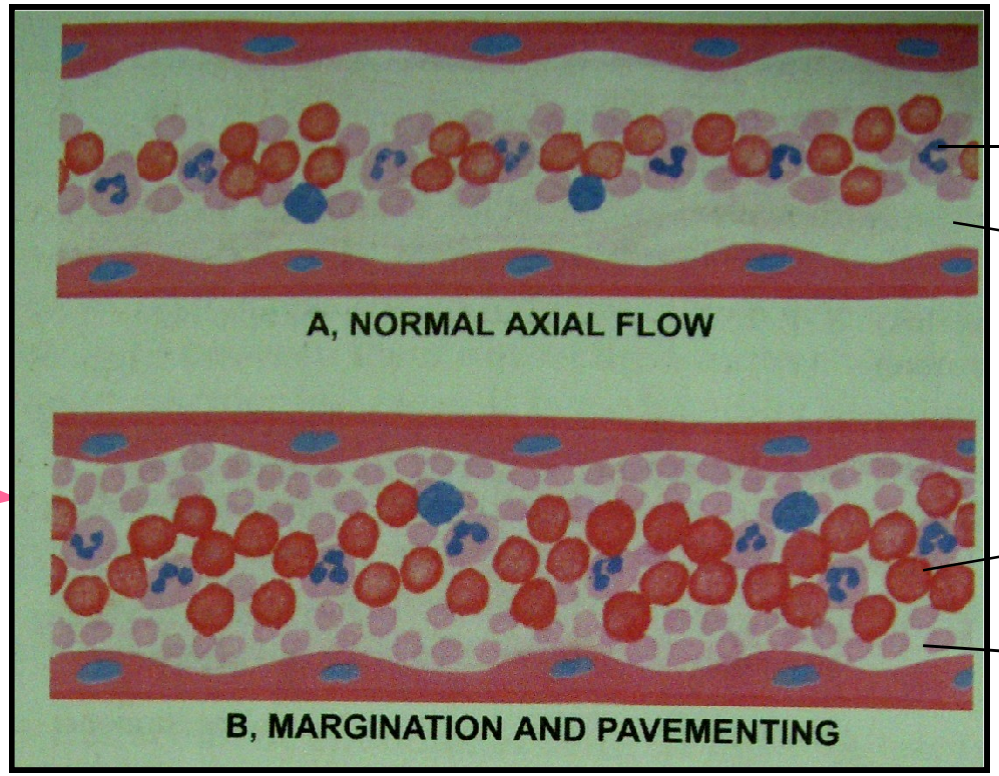
  Rolling and adhesion

  Emigration

  Chemotaxis

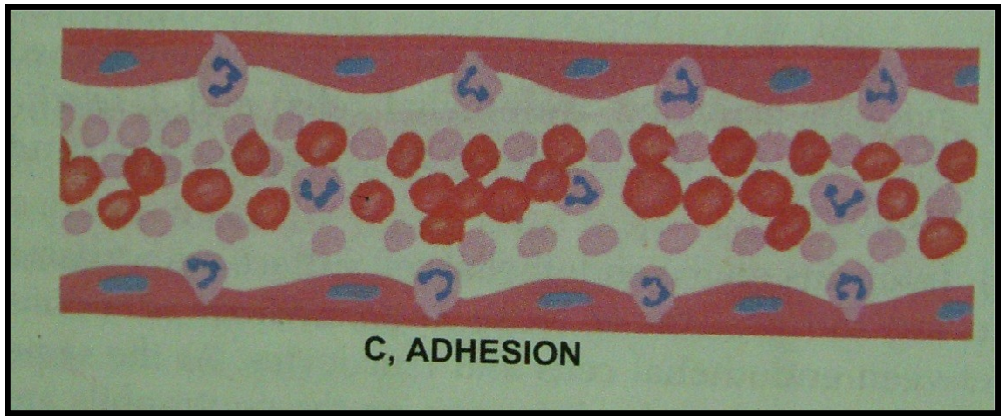
1)changes in the formed elements:

- *Rate of blood flow is increased*
- *Stasis(change in normal axial flow of blood)*



2)Rolling and adhesion

- *PMS's roll over the endothelial cells.*
- *Bond btn leukocytes and endothelial cells.*

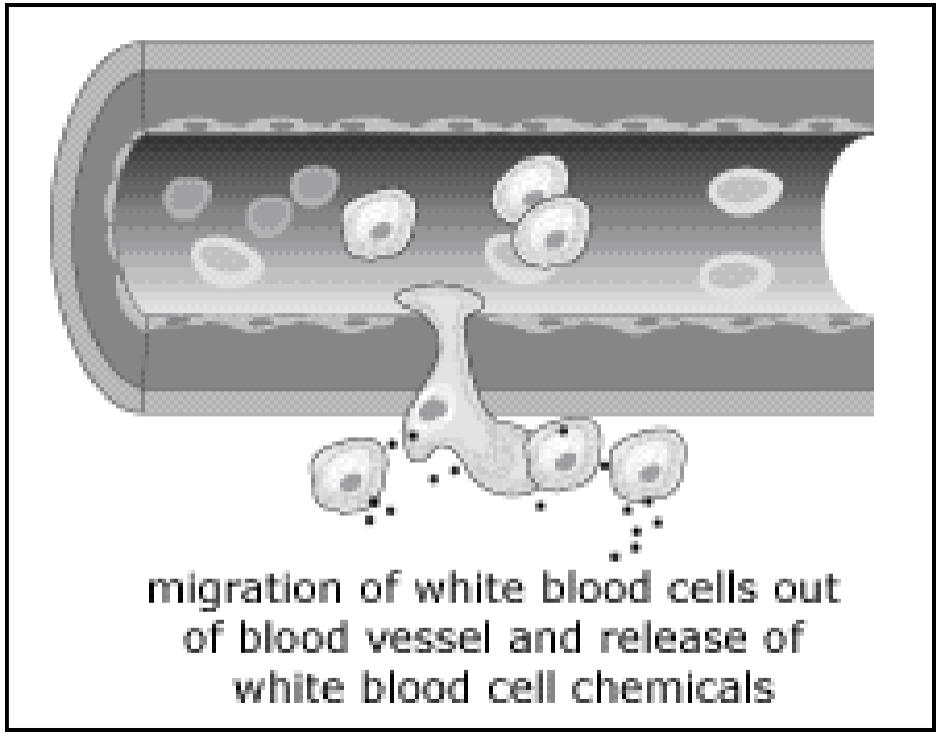


3) Emigration and diapedesis –

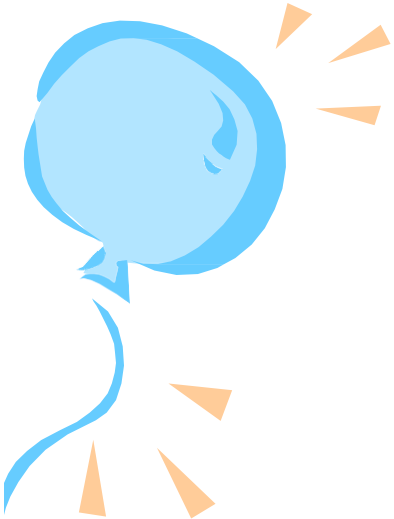
neutrophils throw cytoplasmic pseudopods

↓

cross the basement membrane



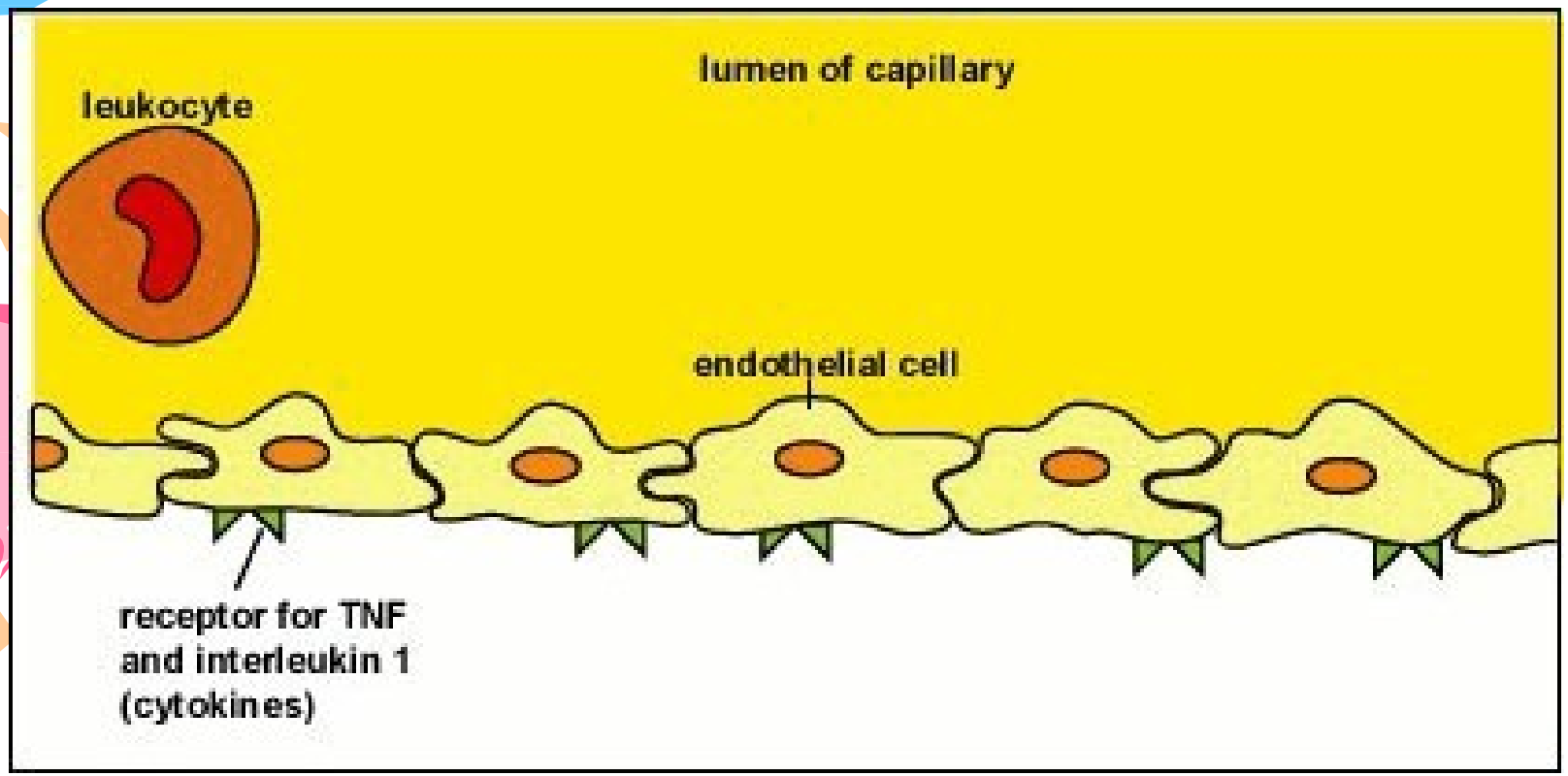
migration of white blood cells out of blood vessel and release of white blood cell chemicals

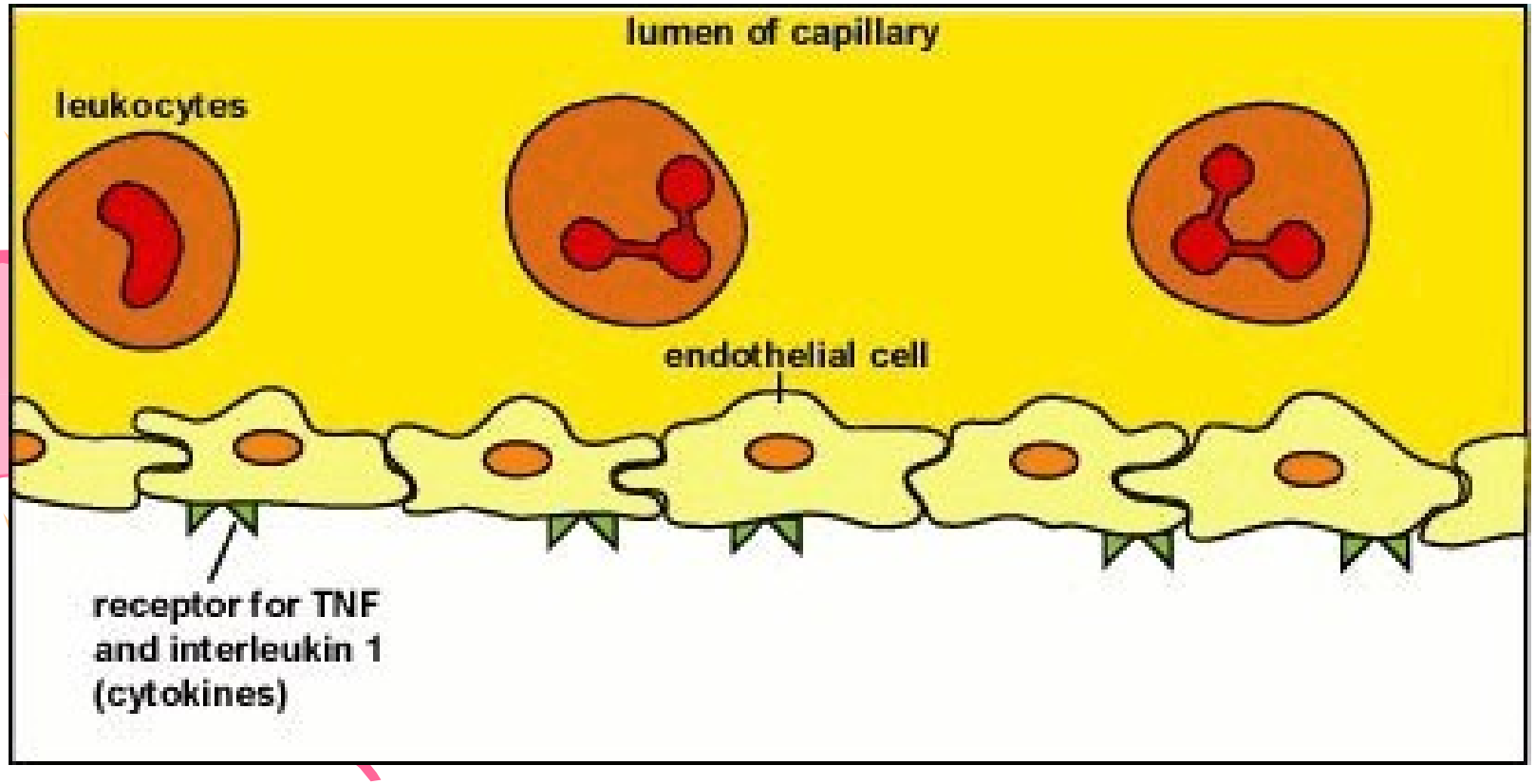


4)Chemotaxis –

- *The chemotactic factor-mediated transmigration of leukocytes after crossing several barriers to reach the interstitial tissues is called chemotaxis*

- *Chemotactic substances – chemokines eg – leukotrines, platelet factor,cytokines, etc*

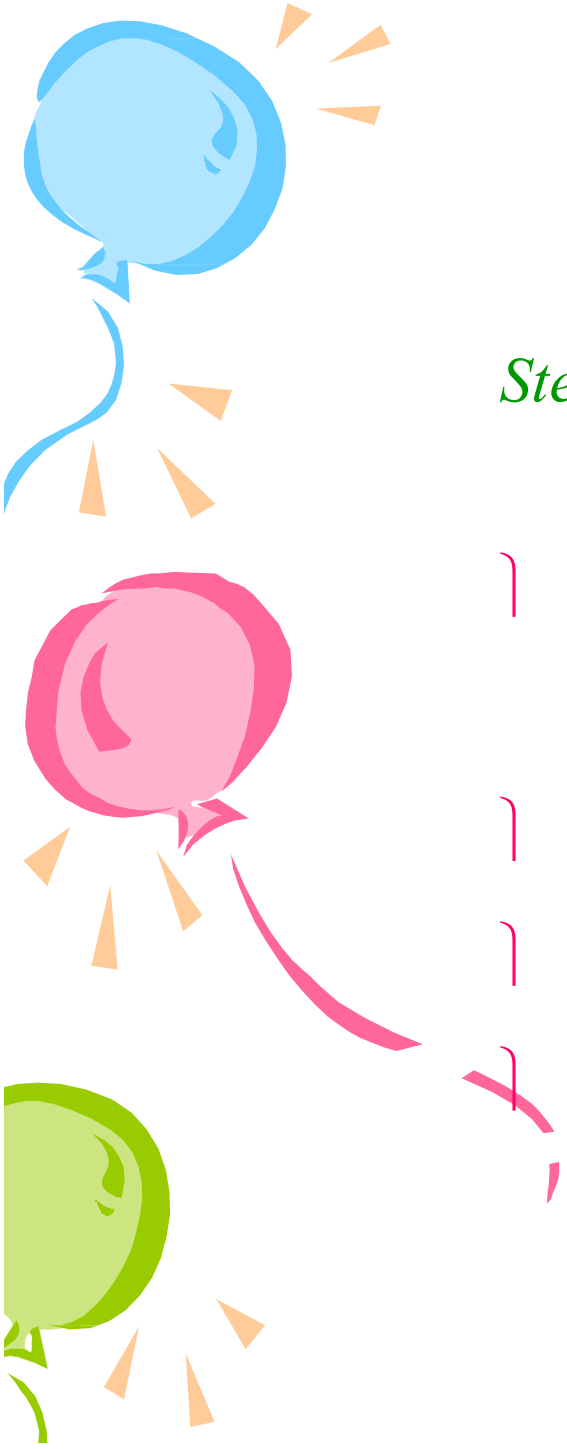

lumen of capillary
leukocyte
endothelial cell
receptor for TNF
and interleukin 1
(cytokines)


lumen of capillary
leukocytes
endothelial cell
receptor for TNF
and interleukin 1
(cytokines)

2) Phagocytosis

- *Phagocytosis is defined as the process of engulfment of solid particulate material by the cells*
- *Phagocytes (cell eating cells)*
- *cells*

microphages *macrophages*

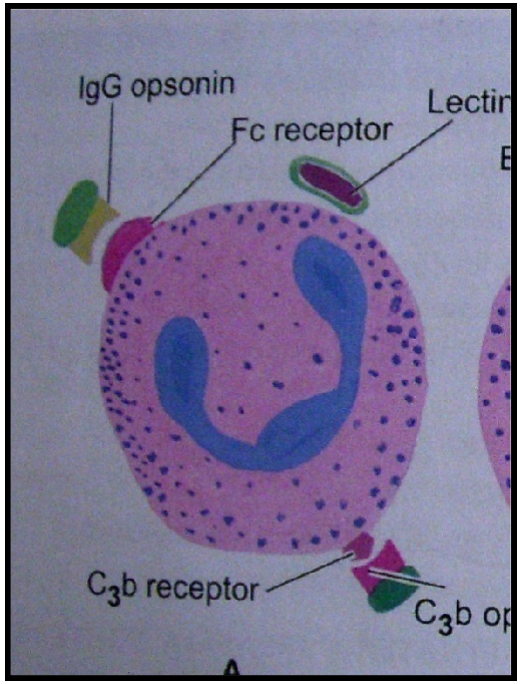


Steps in phagocytosis

- *Recognition and attachment stage (opsonisation)*
- *Engulfment stage*
- *Secretion (degranulation) stage*
- *Digestion or degradation stage*

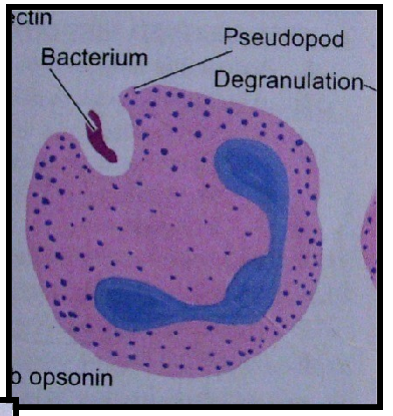
Recognition and attachment stage:

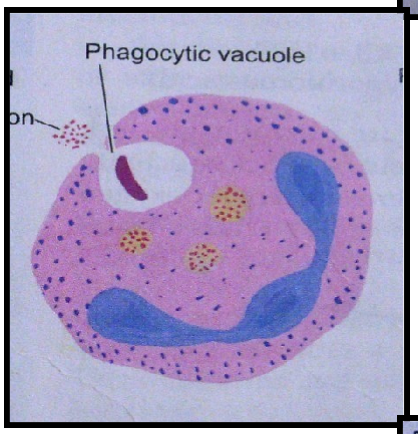
- *To bond the bacteria & the cell membrane of the phagocytic cell, the microorganisms get coated with opsonins which are naturally occurring factors in the serum.*
- *The main opsonins present in the serum are I_gG opsonin, C_{3b}opsonin & lectins*

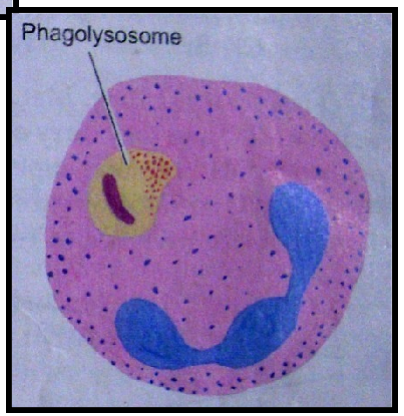


Engulfment stage

- *Cytoplasmic pseudopods are formed around the particle, enveloping it in a phagocytic vacuole.*
- *Eventually the plasma membrane enclosing the phagocytic vacuole breaks the cell surface*
- *The lysosomes of the cell fuse with the vacuole and form phagolysosome or phagosome.*

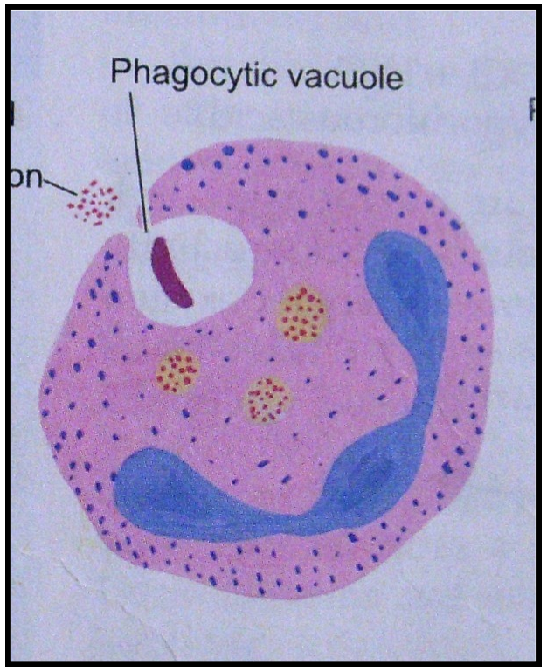






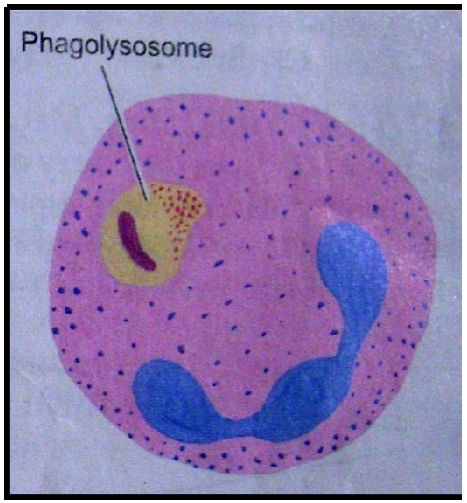
Degranulation stage :

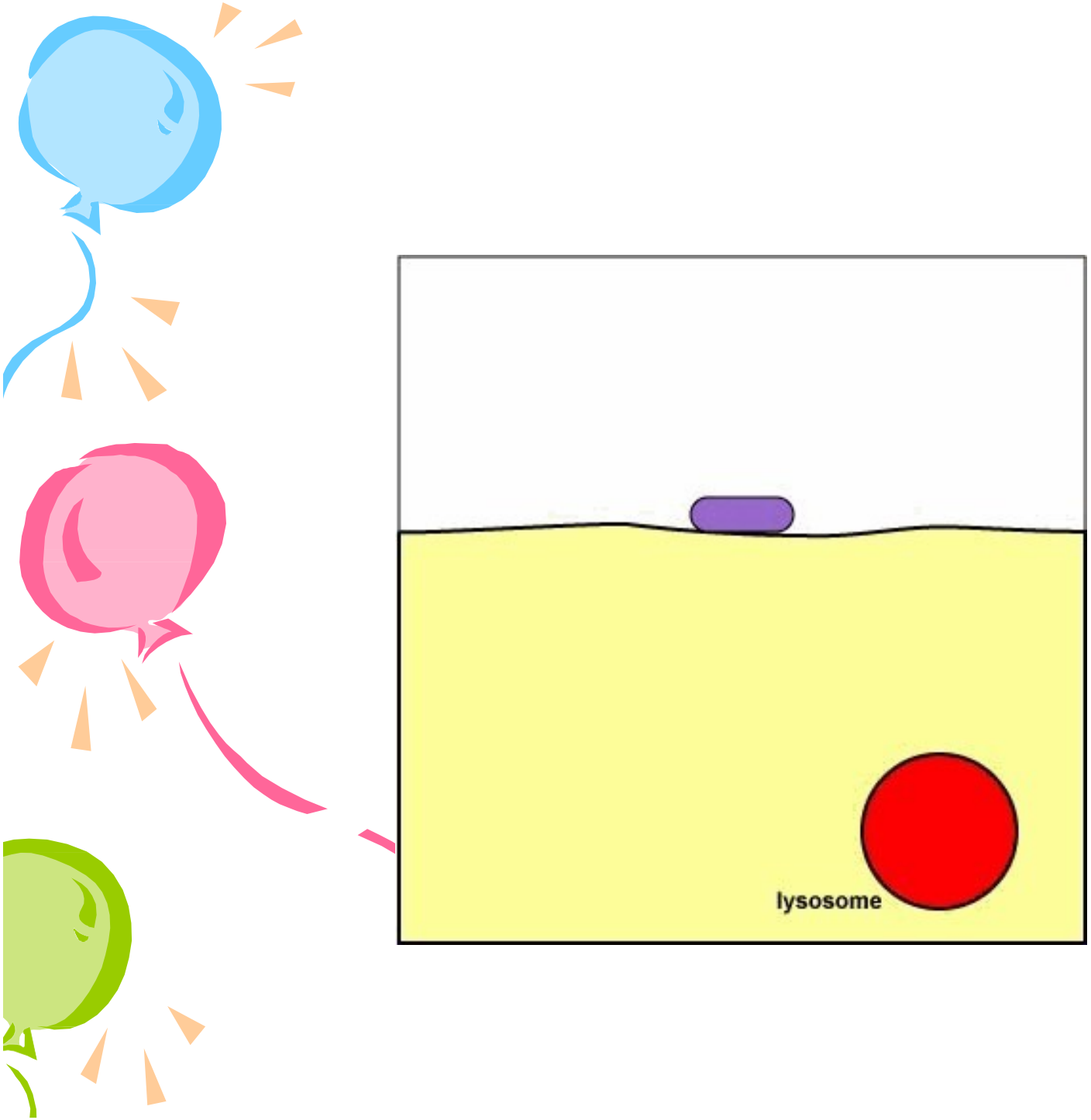
- *preformed granule products of PMN's are discharged.*
- *Specific or secondary granules of PMN's are released with interlukin2,TNF,superox ide oxygen.*

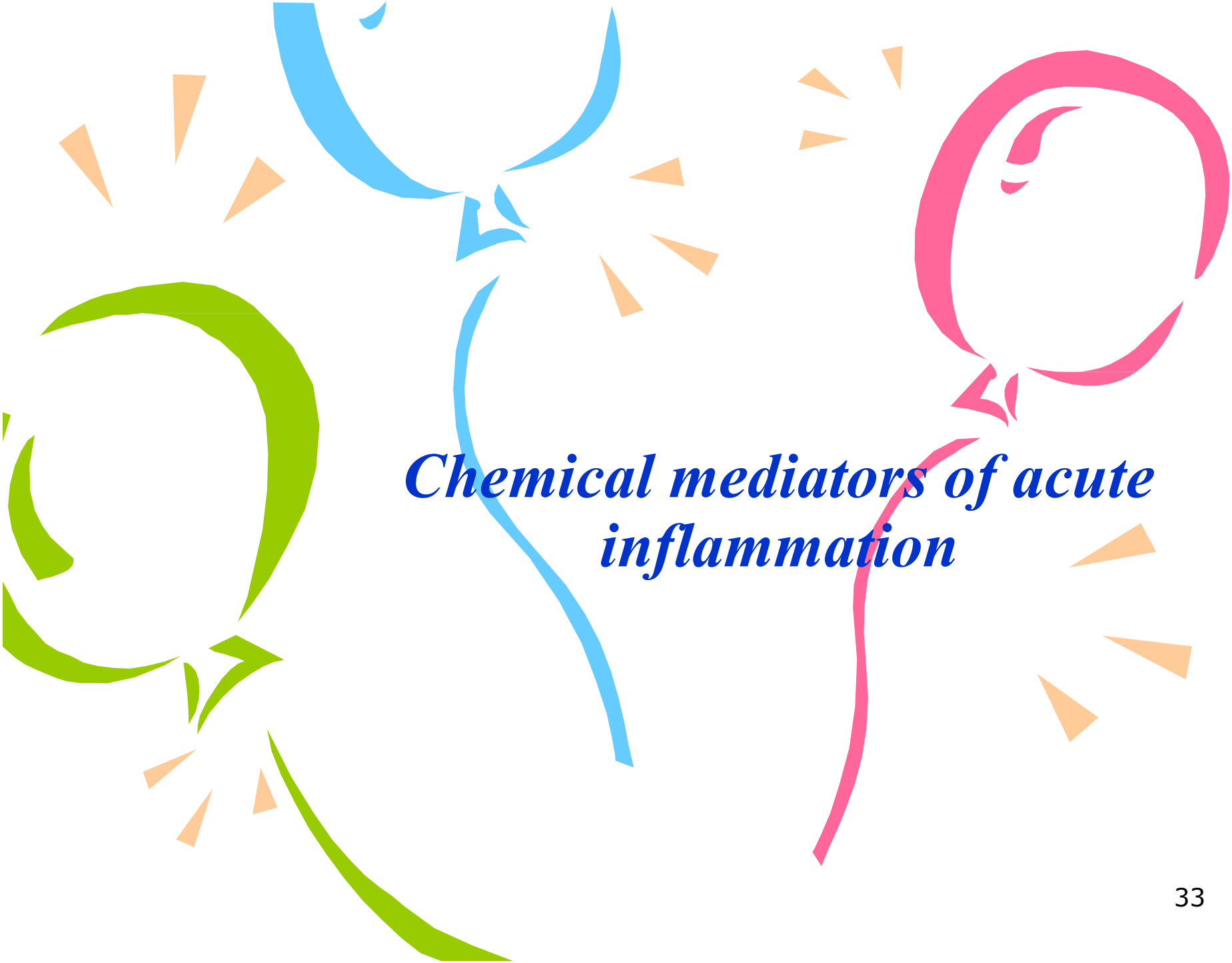


Killing/degradation stage:

- *Killing & digestion of the microorganisms by the phagocytes as scavenger cells is done.*
- *The microorganisms are degraded by the hydrolytic enzymes.*

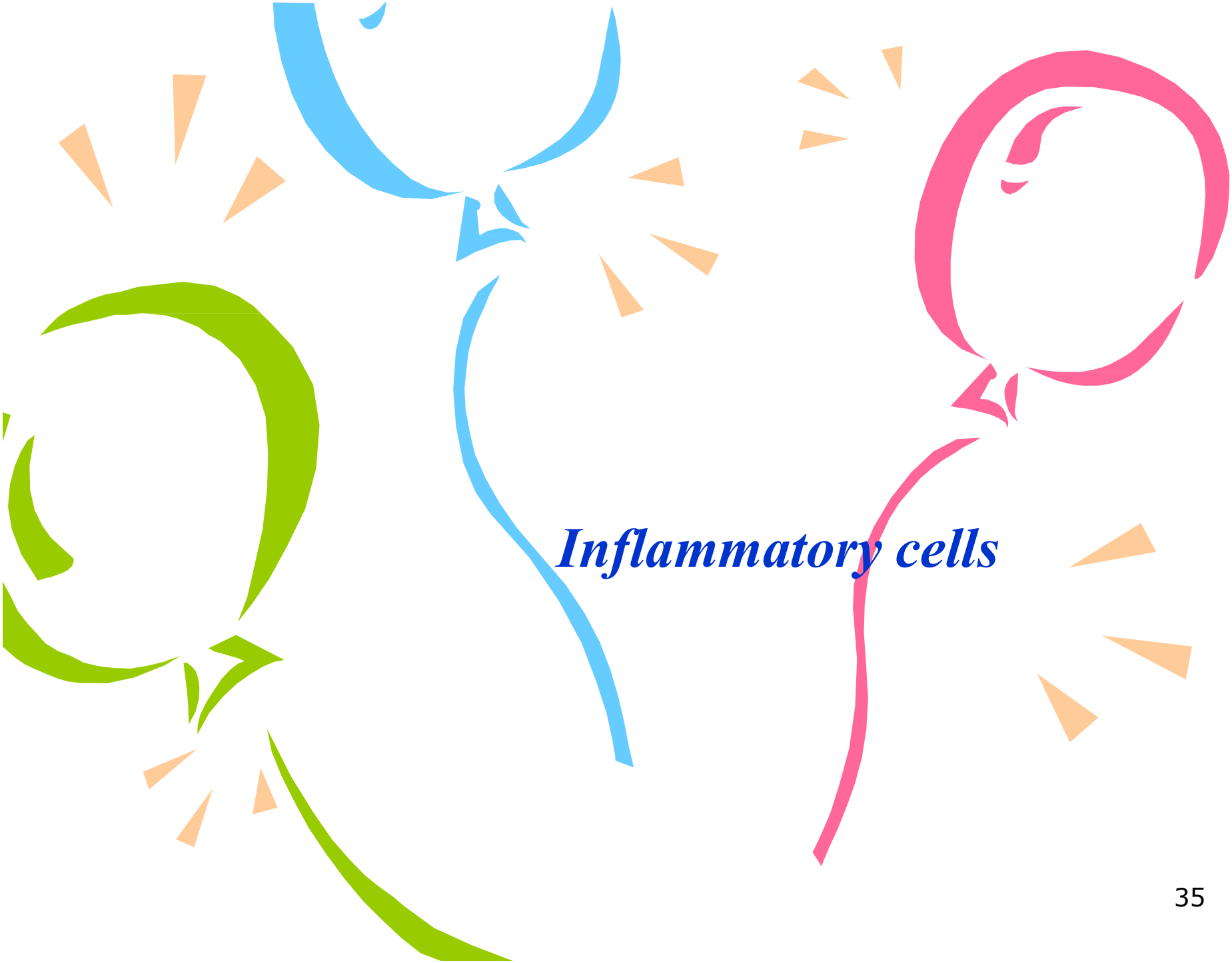


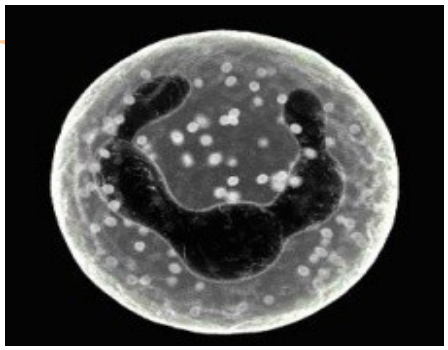

lysosome



Chemical mediators of acute inflammation

	Source	Mediator	Main action
Cell derived	Mast cells, basophils, platelets	Histamine	Increased permeability
	Platelets	Serotonin	Increased permeability
	Inflammatory cells	Lysosomal enz, NO,O metabolites leukotrines, prostaglandins, cytokines,	Tissue damage ↑ permeability Vasodilation Fever
	Clotting & fibrinolytic sys.	Fibrin split products	Increased permeability
Plasma derived	Kinin sys.	Kinin/bradykinin	Increased permeability
	Complement sys.	Anaphylatoxins, C_{3a}, C_{4a}, C_{5a}	Increased permeability

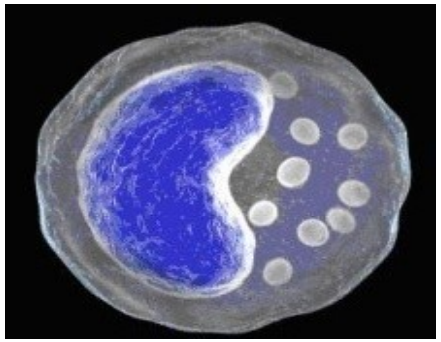

Inflammatory cells



Polymorpho nuclear neutrophils

Initial phagocytosis,

acute inflammatory cell

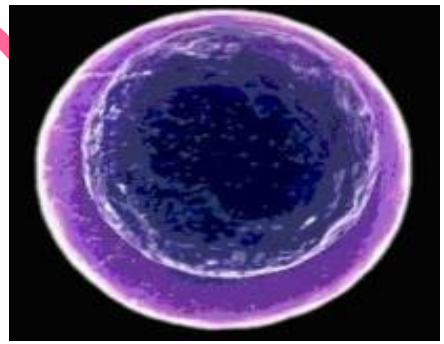


Monocyte/macrophage

Bacterial phagocytosis

Chronic inflammatory cell

Regulates Lymphocyte response

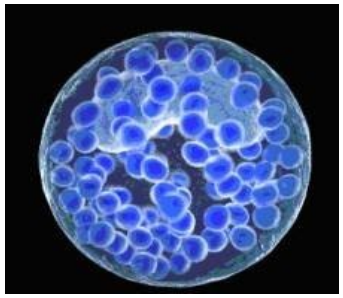


Lymphocyte

Humoral & cell mediated response

Chronic inflammatory cell

Regulates Macrophage response

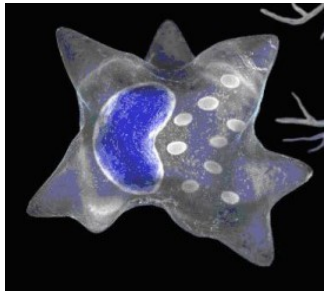


Eosinophil

Allergic states

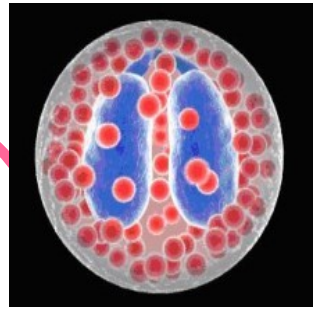
Parasitic infestations

Chronic inflammatory cell



Mast cell

Receptor for IgE anti-bodies



Plasma cell

Chronic inflammatory cell

Derived from B cells

Morphology of acute inflammation

1.Pseudomembranous inflammation-

- *it's a inflammatory response of the mucous surface to toxins of diphtheria or irritant gases.*
- *denudation of the epithelium*
- *plasma exudes on the surface where it coagulates & together with the necrosed epithelium forms a false membrane.*

2.Ulcer-

- *Ulcers are local defects on the surface of an organ produced by inflammation.*

3.Suppuration(Abscess formation)-

- *neutrophilic infiltrate in the inflamed tissue results in tissue necrosis.*
- *A cavity is formed which is called an abscess & contains a purulent exudate*
- *Boil or Furuncle is an acute inflammation of the hair follicles in the dermal tissues.*

4. *Cellulitits-*

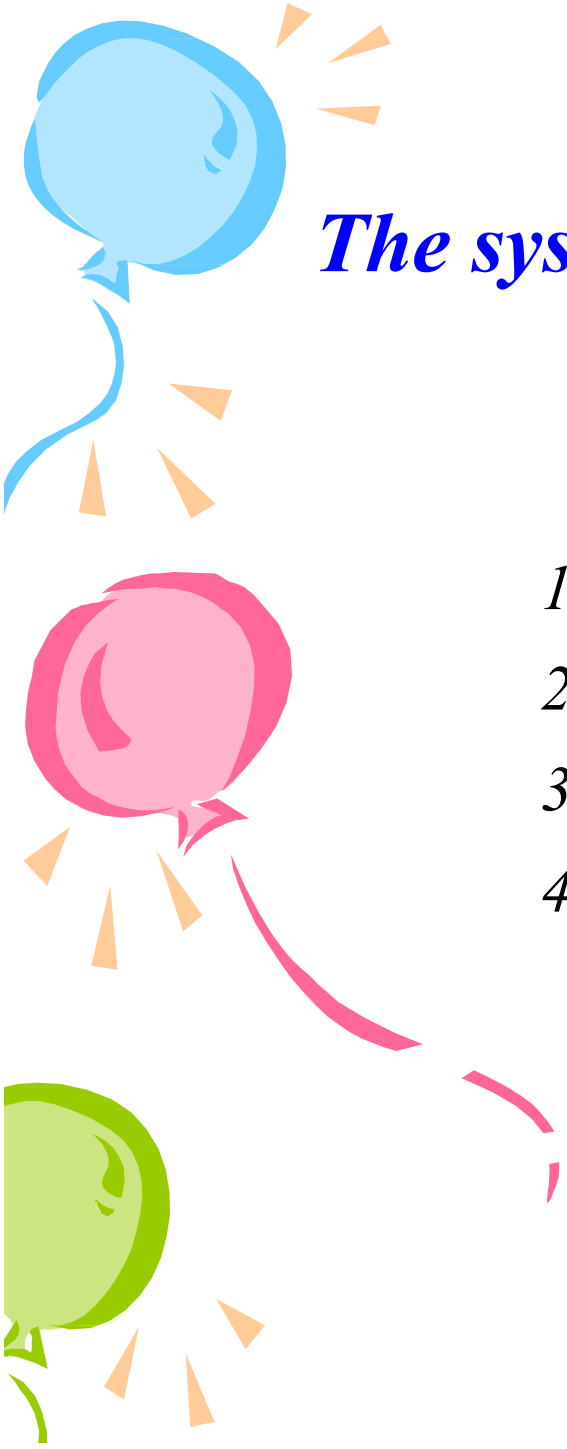
it's a diffuse inflammation of soft tissues resulting from spreading effects of substances like hyaluronidase released by some bacteria.

5. *Bacterial infection of the blood-*

a) Bacteraemia

b) Septicemia

c) Pyaemia



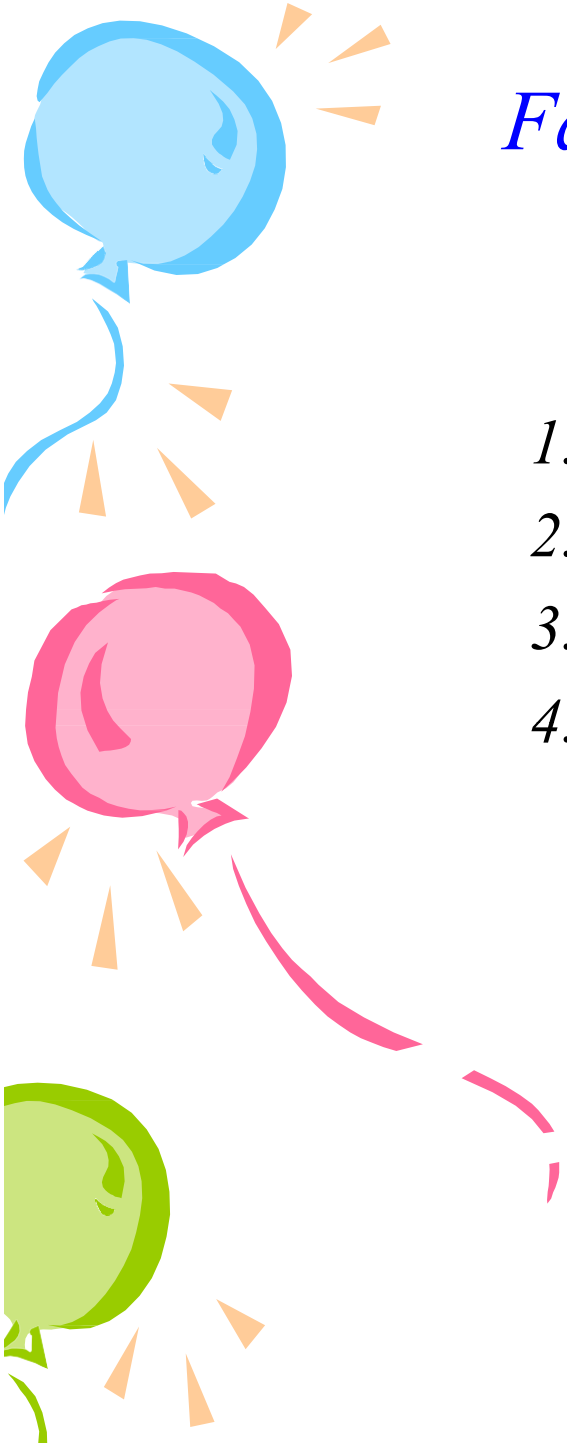
The systemic effects of inflammation

1.fever

2.Leucocytosis

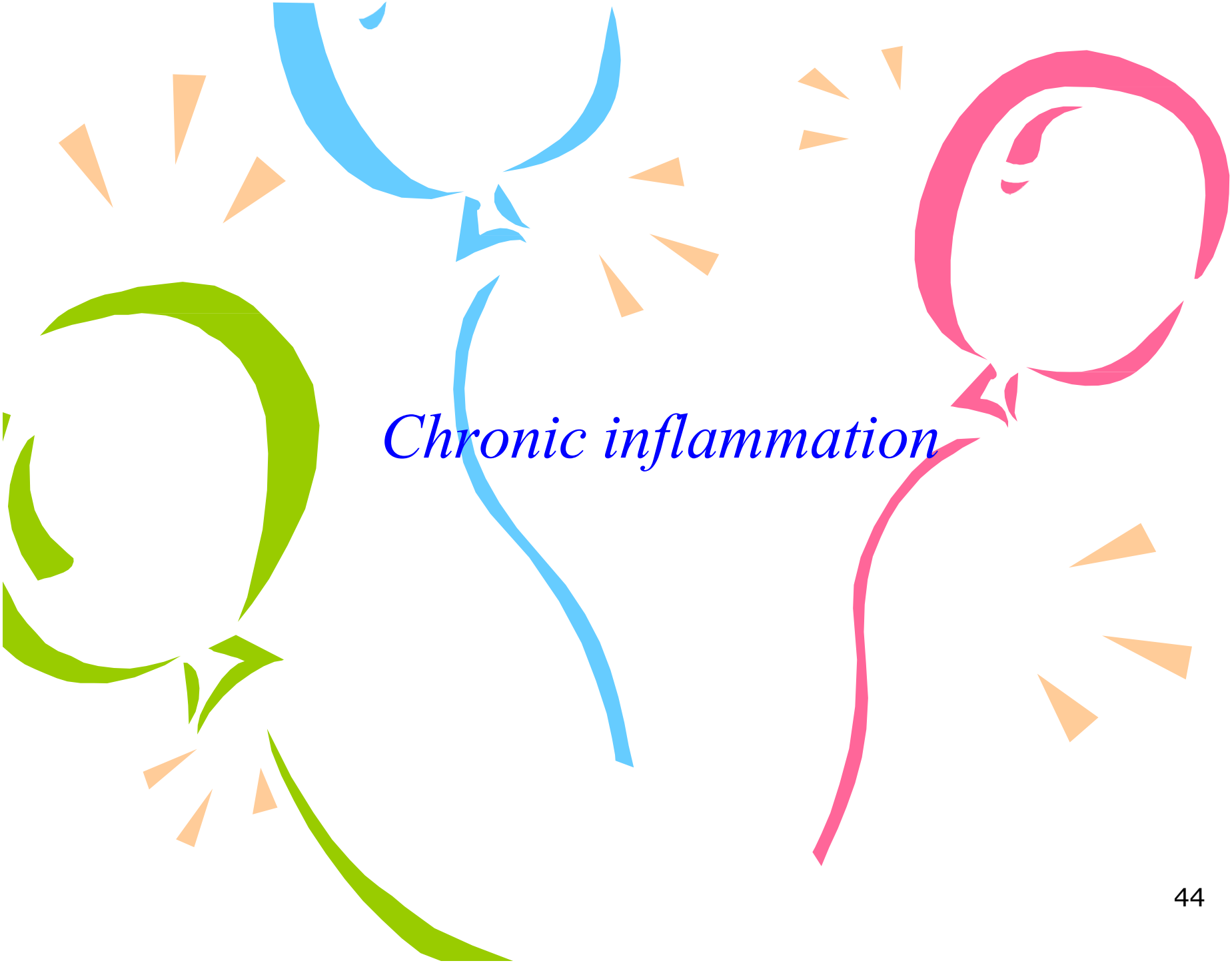
3.Lymphadenitis

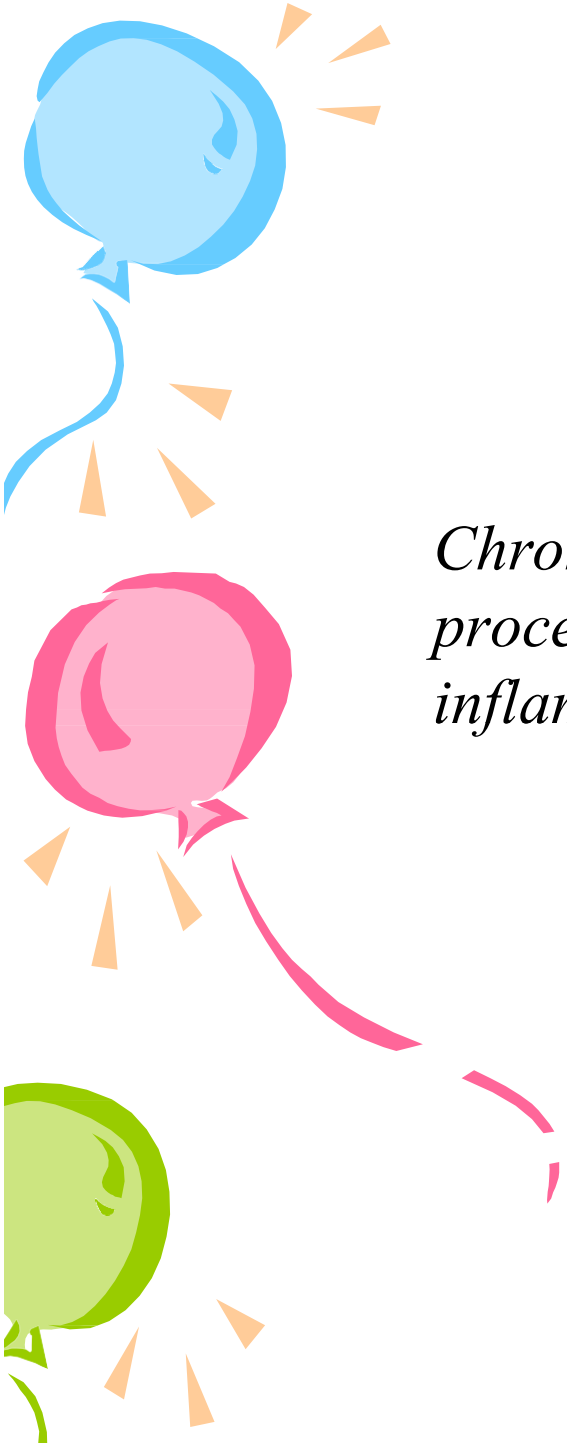
4.Shock



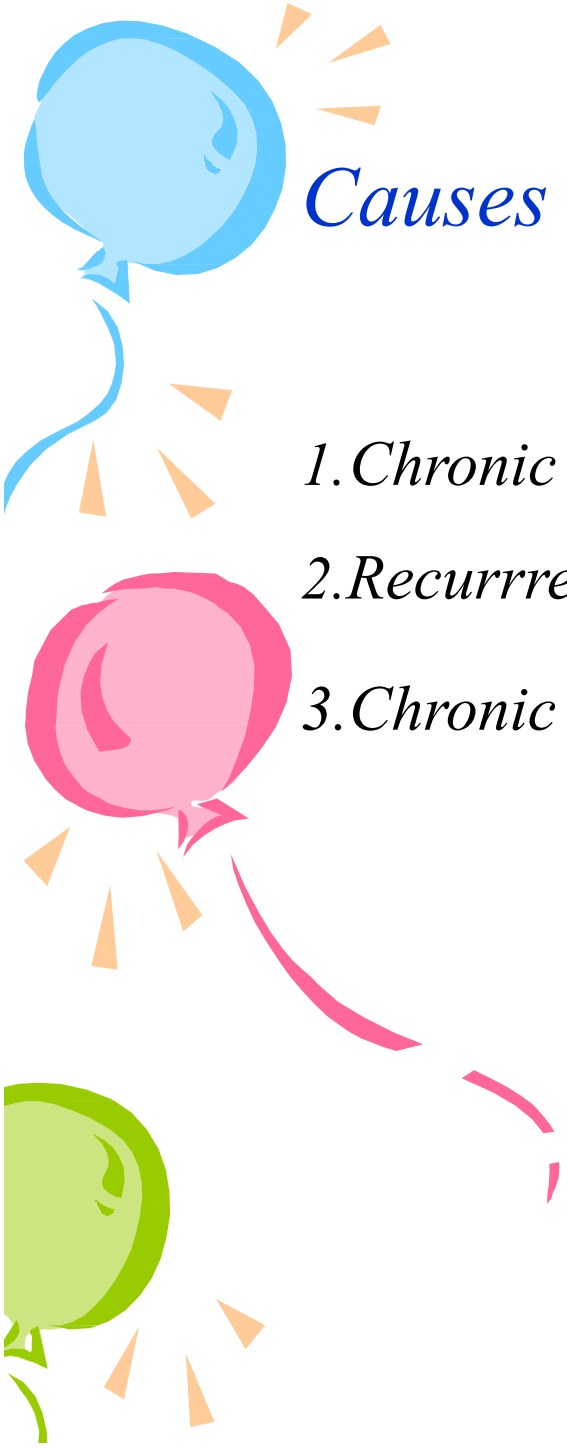
Fate of acute inflammation

1. *Resolution*
2. *Healing by scarring*
3. *Progression to suppuration*
4. *Progression to Chronic inflammation.*


Chronic inflammation



Chronic inflammation can be defined as a prolonged process in which tissue destruction and inflammation occur at the same time.



Causes of Chronic inflammation

1.Chronic inflammation following Acute inflammation

2.Recurrrent attacks of acute inflammation

3.Chronic inflammation starting de novo

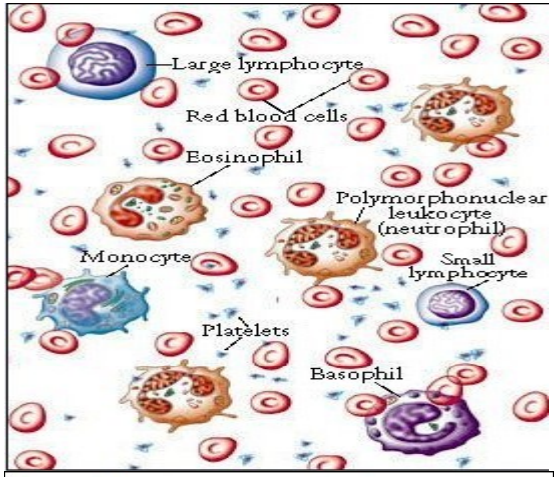
General features of chronic inflammation

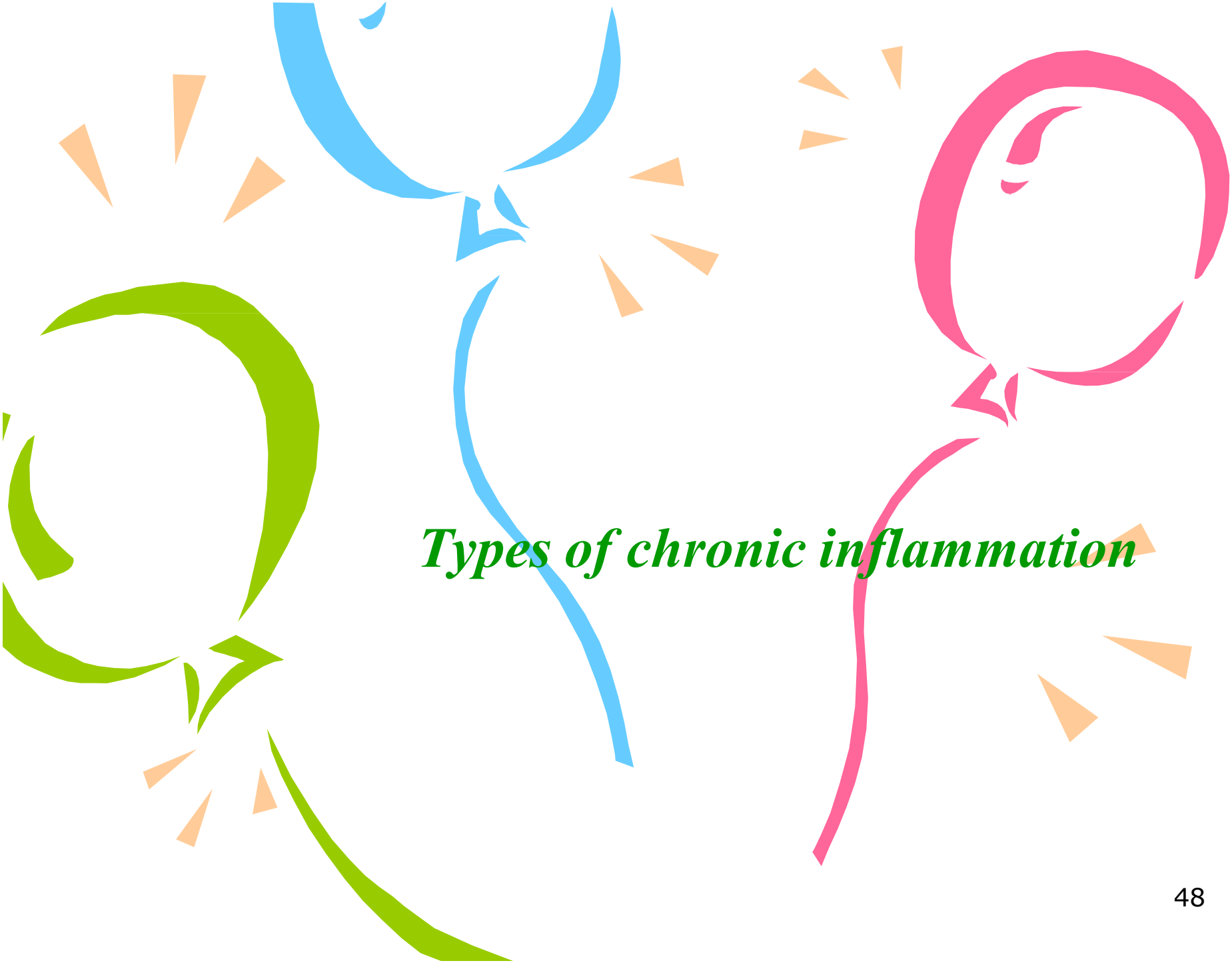
1.Mononuclear cell infiltration-

phagocytes, circulating monocytes, macrophages & giant cells.

2.Tissue destruction or necrosis.

3.Proliferative changes- small blood vessels & fibroblasts

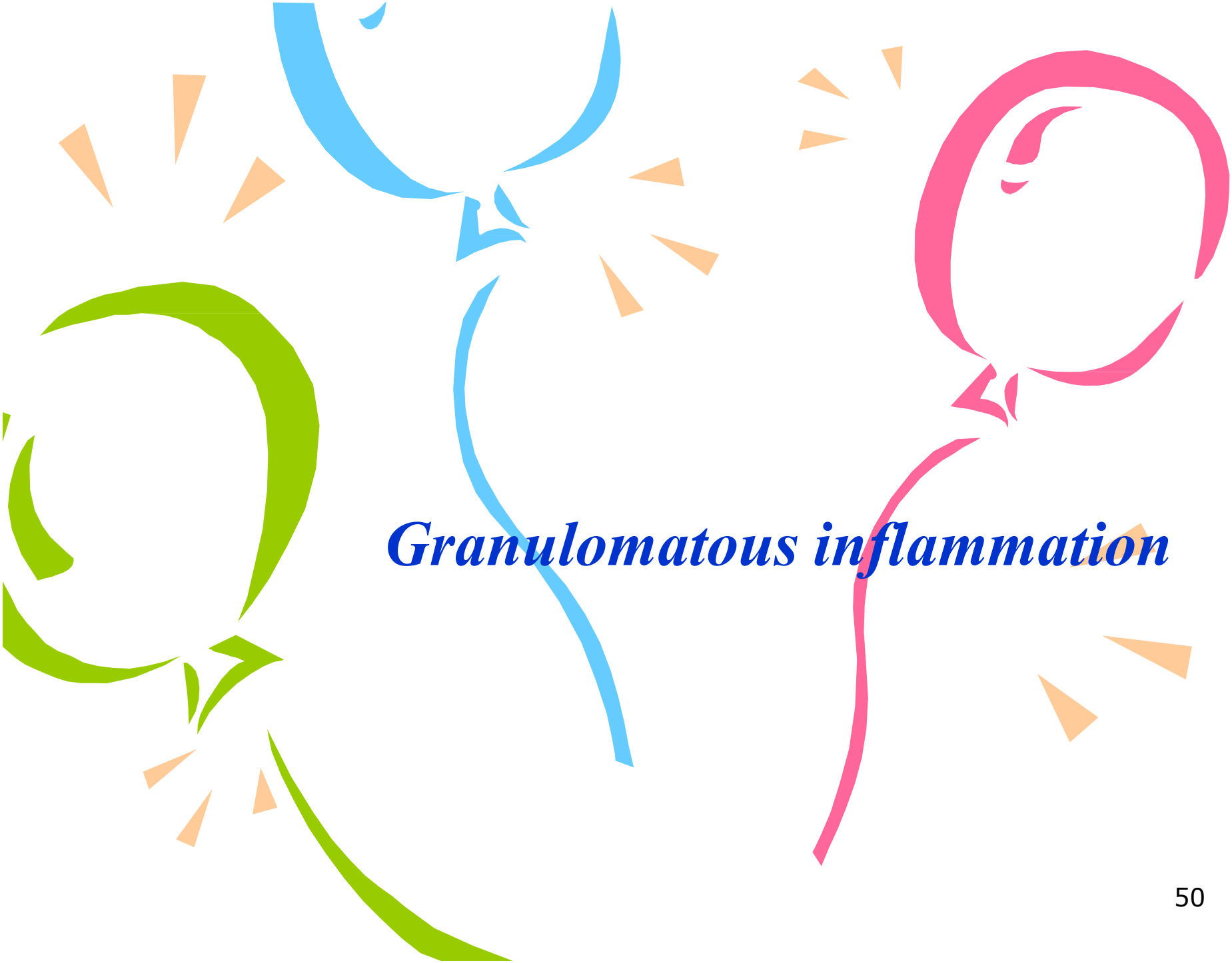




Types of chronic inflammation

1.Nonspecific inflammation
2.Specific

According to histological findings
1.Chronic nonspecific inflammation
2.Chronic Granulomatous inflammation



Granulomatous inflammation

- *It is a circumscribed , tiny lesion, about 1 mm in diametre, composed predominantly of collection of modified macrophages called epithelioid cells*

disease	*Oral manifestation*
TB	*TB ulcers & gingiva*
Actinomycosis	*lumpy jaw,abscess, sinuses*
Syphilis	*Mucocutaneous lesions, painless lymphadenopathy*

Inflammation of pulp

Classification of pulpitis

1. *Inflammatory diseses of pulp*

- *Reversible pulpitis*

  acute and chronic

- *Irreversible pulpitis*

  acute(heat or cold)

  chronic(asymptomatic, hyperplastic pulpitis,int respn)

2. *Pulp degeneration*

   calcific (radiograpically)

   others (histologycally)

- *Necrosis*

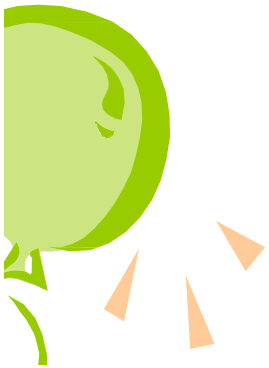
Causes

1)Physical

- *Mechanical – cavity or crown preparations*
- *Thermal – cavity preparation,polishing, conduction fillings.*
- *Electrical – dissimilar fillings.*

2) Chemical – phosphoric acid, monomer

3) Bacterial - caries



Inflammation of PDL

- *Acute*
  - *acute alv abscess*
  - *acute apical periodontitis*
  - *vital*
  - *nonvital*
- *Chronic*
  - *chronic alv abscess*
  - *granuloma*

Condensing osteitis

Inflammation of gingiva

- *Acute, chronic, recurrent gingivitis(course and duration)*
- *Localised ,generlised gingivitis (distribution)*
- *Marginal,papillary, diffuse gingivitis (combination)*

References

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- *GROSSMMAN'S endodontic practice 12th edn*
- *Ingles endodontics 6th edition.*


Thank you

THANK YOU