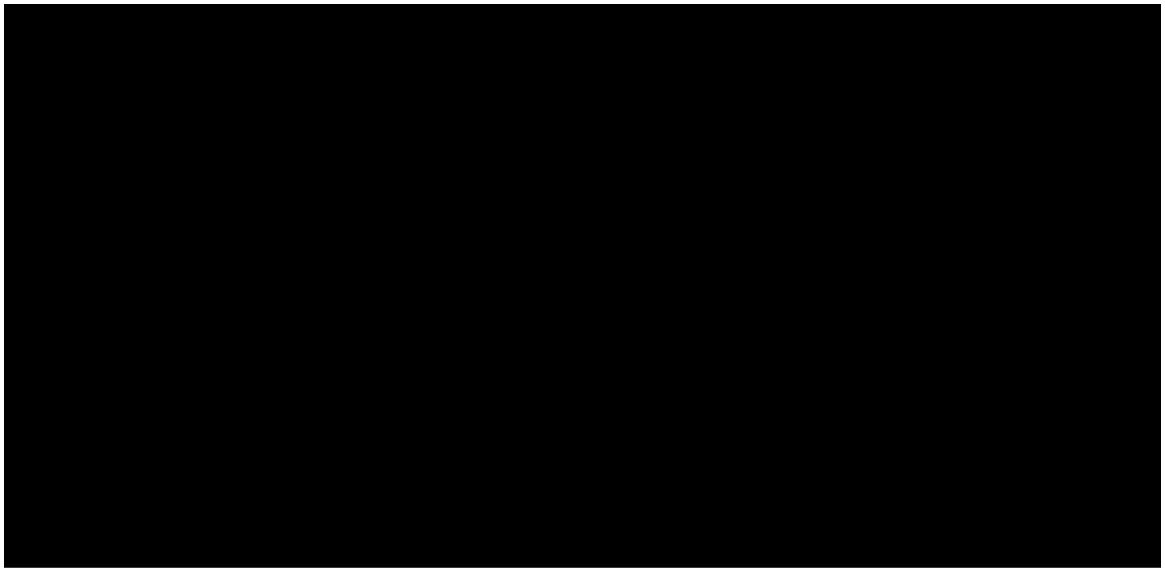
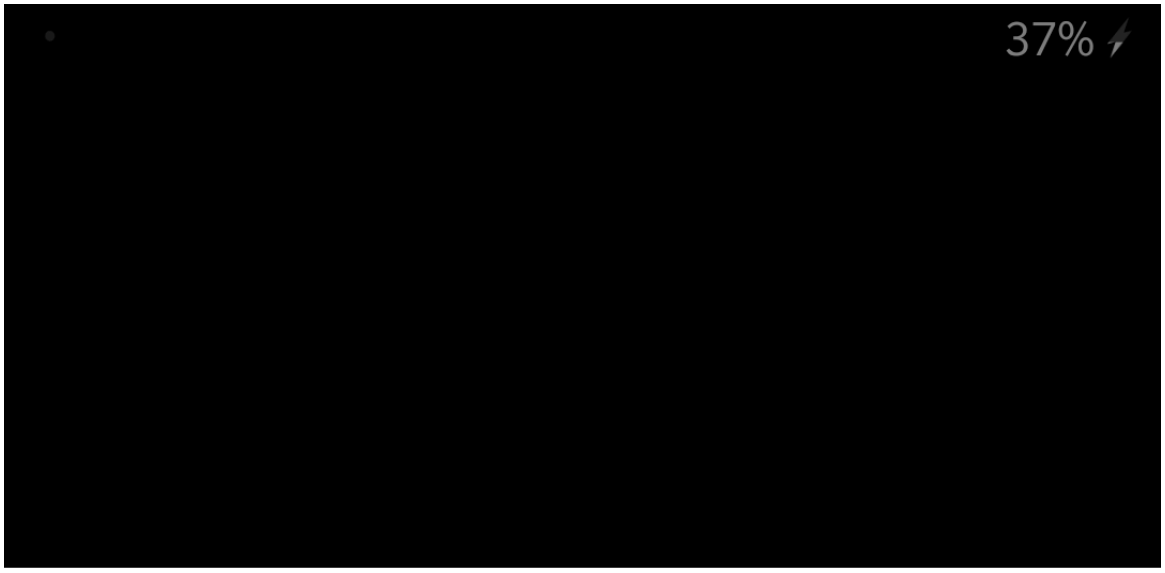


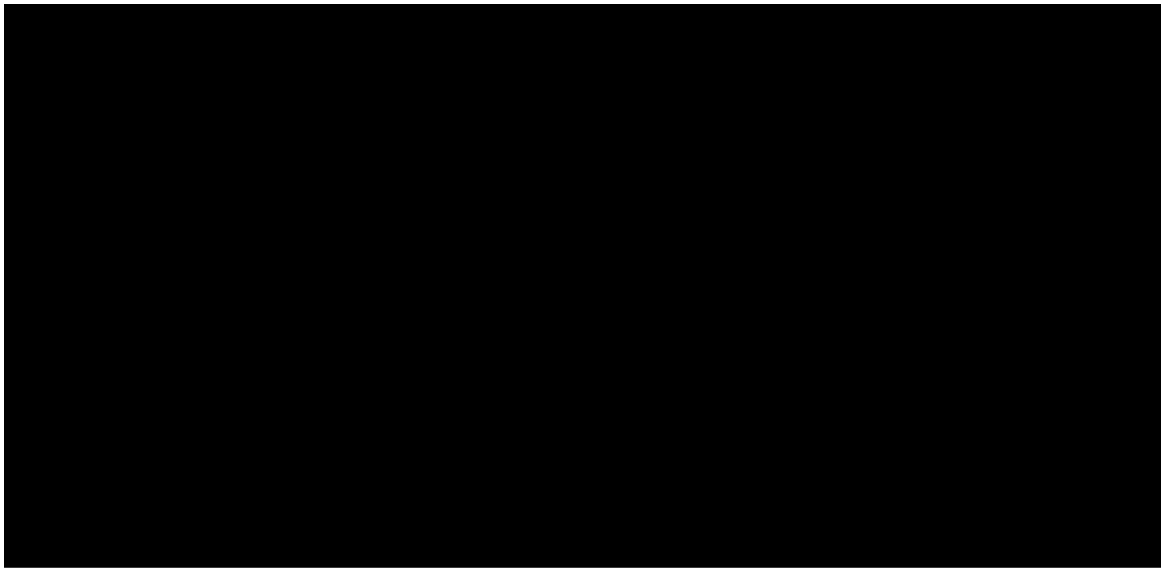
WHAT ARE HYPERSENSITIVITY REACTIONS?

- Immune responses that are exaggerated or inappropriate against an antigen or allergen.
- 4 types:-
- Type 1:-immediate hypersensitivity(anaphylactic reaction)
- Type 2:-Cytotoxic(antibody dependent)
- Type 3:-Immune complex reaction
- Type 4:-Cell mediated(delayed hypersensitivity)



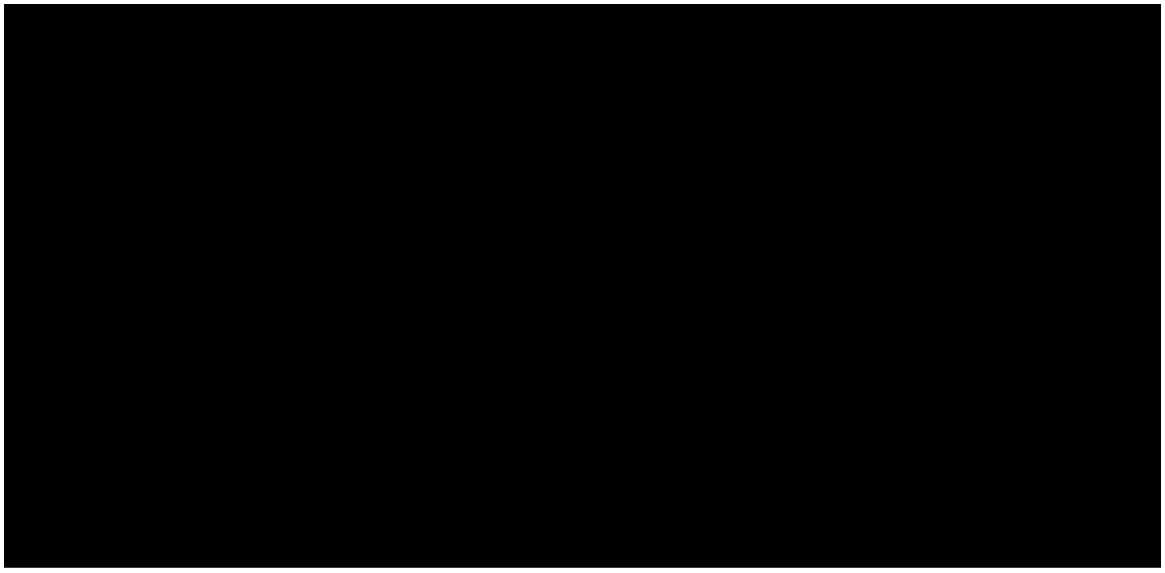
TYPE I HYPERSENSITIVITY

- Takes seconds to minutes
- Typically IgE mediated
- Involves 2 steps, sensitization & secondary exposure.
- Sensitization involves binding of allergen to APC>helperTcell in lymph node>conv.To TH2 cell>releases mediators>plasma cells release IgE and recruits eosinophils>IgE binds to mast cells.
- Secondary exposure after months cause binding of allergen to pre-sensitized mast cells>degranulation>release of mediators causing hypersensitivity.



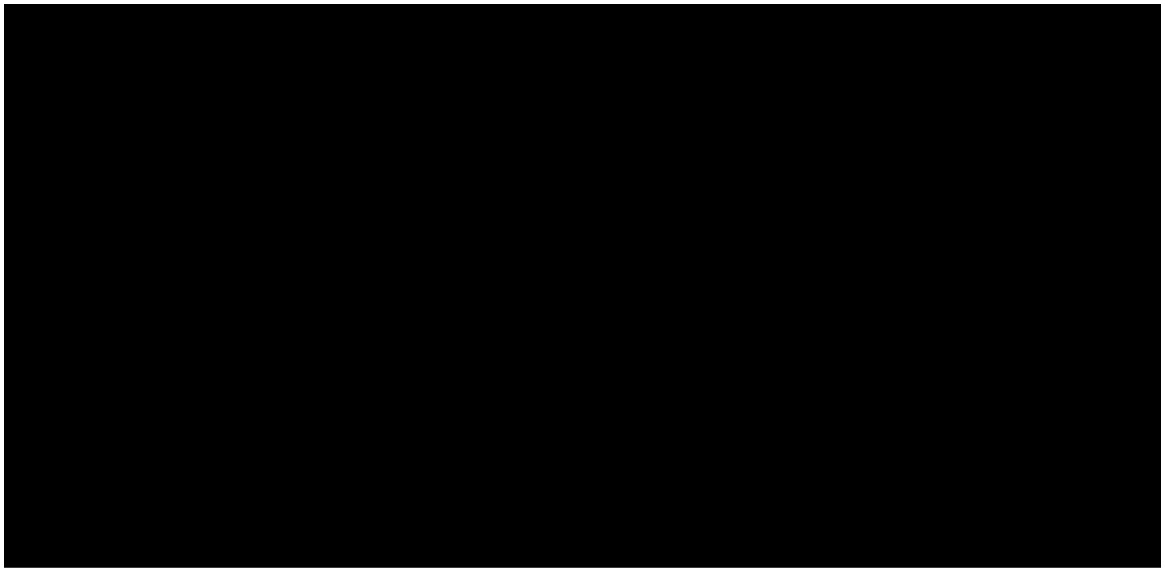
EARLY PHASE RESPONSE

- Occurs in seconds to minutes
- Release of histamine causes smooth muscle contraction >bronchoconstriction > difficulty breathing, blood vessel dilation &inc. permeability>edema>urticaria/hives.
- Responsive to antihistamines.



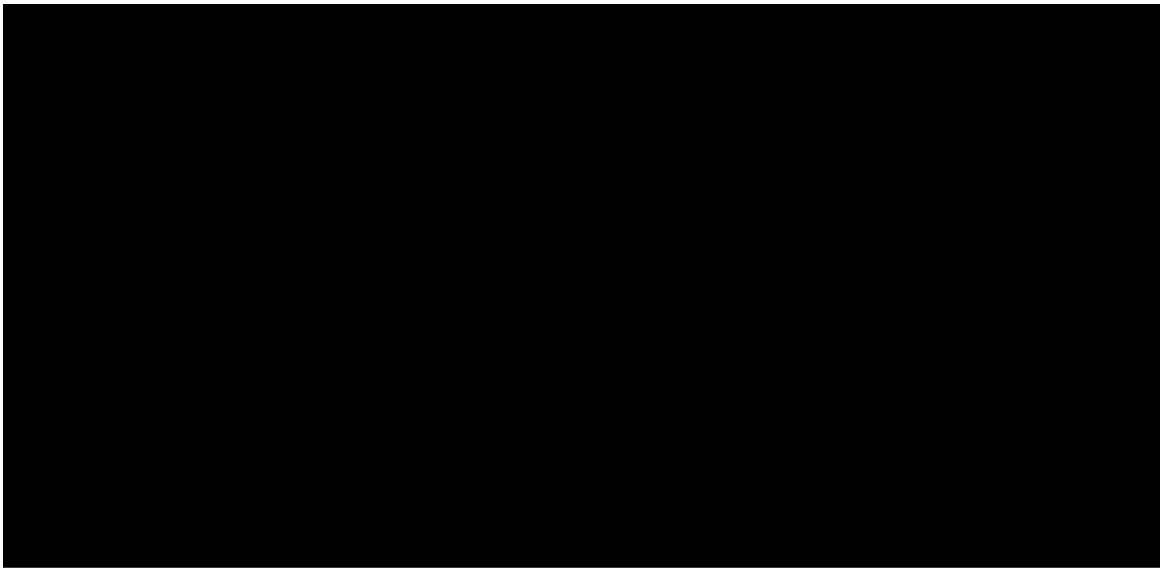
LATE PHASE RESPONSE

- Occurs 6-8 hrs. after exposure
- More severe reaction
- Involves mediators such as leukotrienes and prostaglandins
- Further recruitment of eosinophils ,basophils &TH2 cells>cascade
- Causes sustained bronchoconstriction and edema.
- Not responsive to antihistamines,requires steroids.



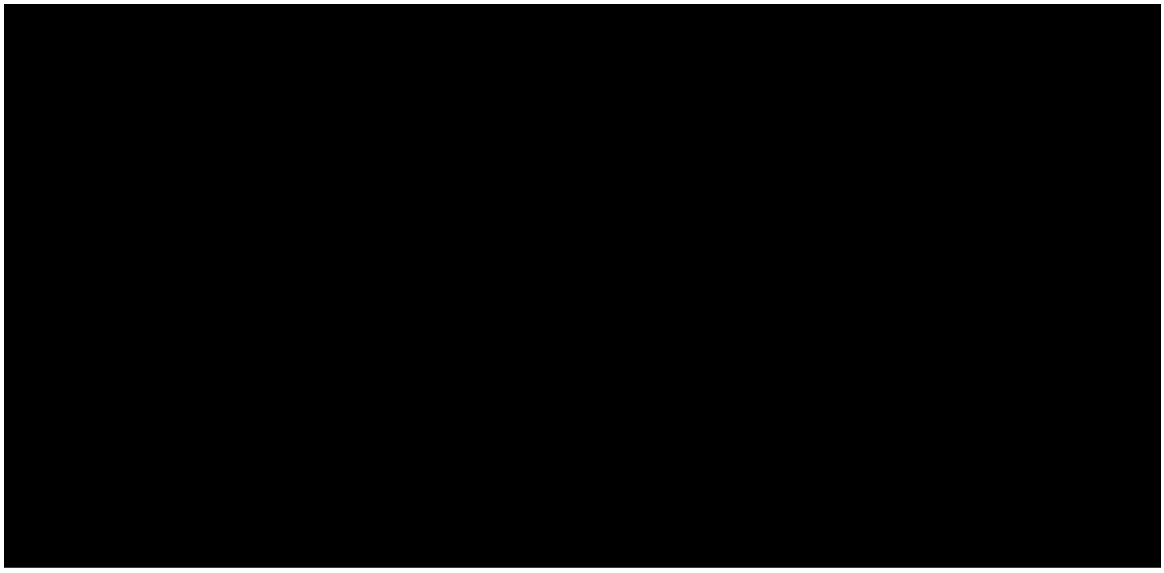
DIAGNOSIS

- History of exposure
- Family history of atopy
- Skin prick testing
- Serum allergen specific IgE
- Mast cell serum tryptase levels



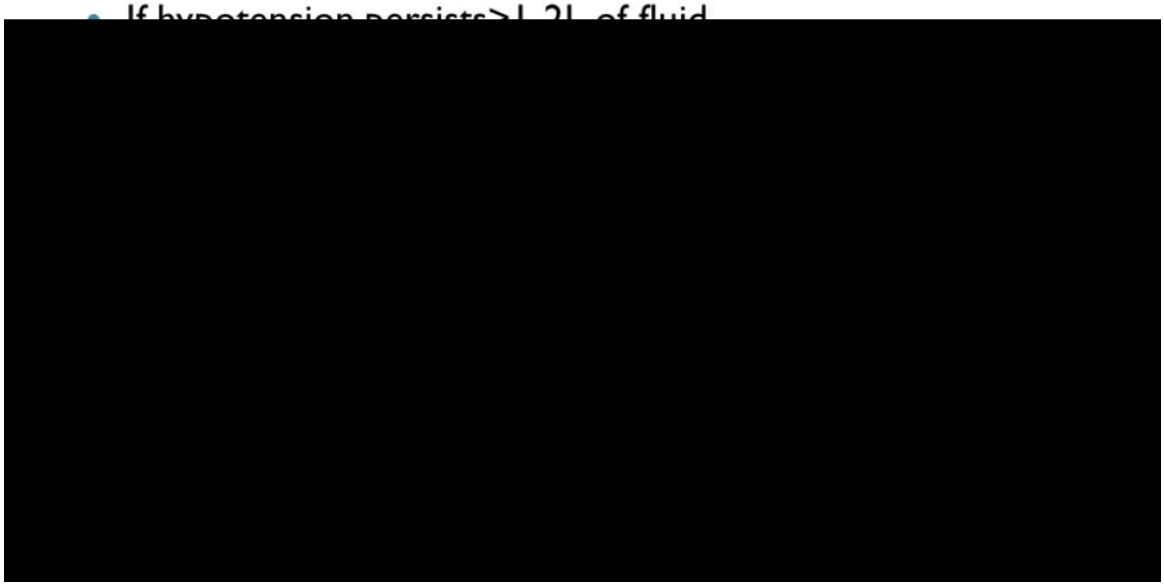
MANAGEMENT

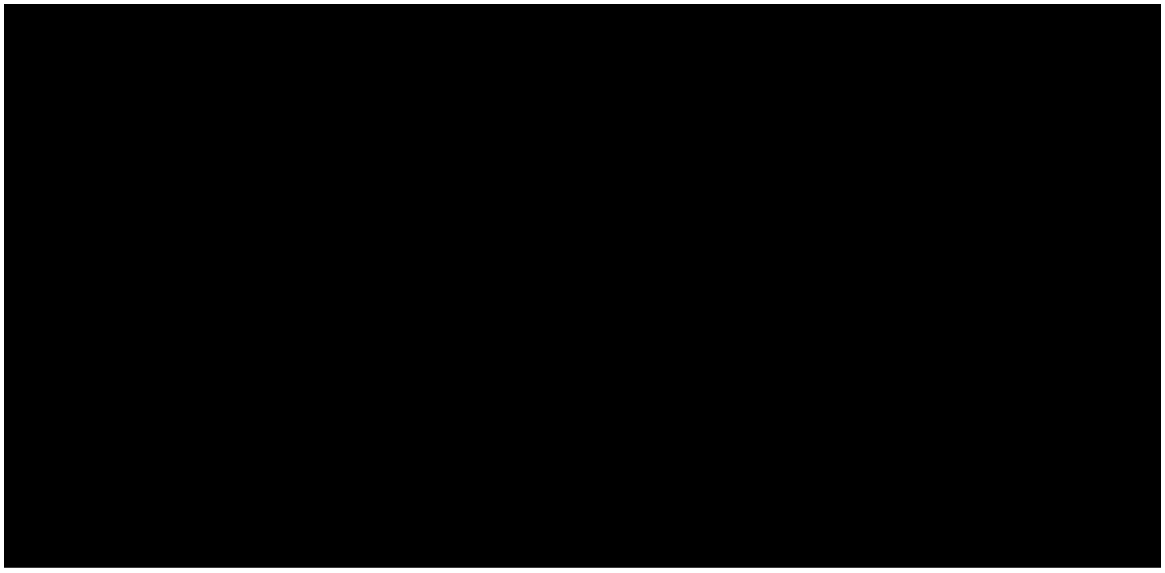
- Antihistamines
- Corticosteroids>PG &LT inhibition,recruitment of mast cells inhibition, vasoconstriction>red. Cell leakage &edema
- Leukotriene receptor antagonists
- Omalizumab(monoclonal IgE antibody).



ANAPHYLACTIC SHOCK

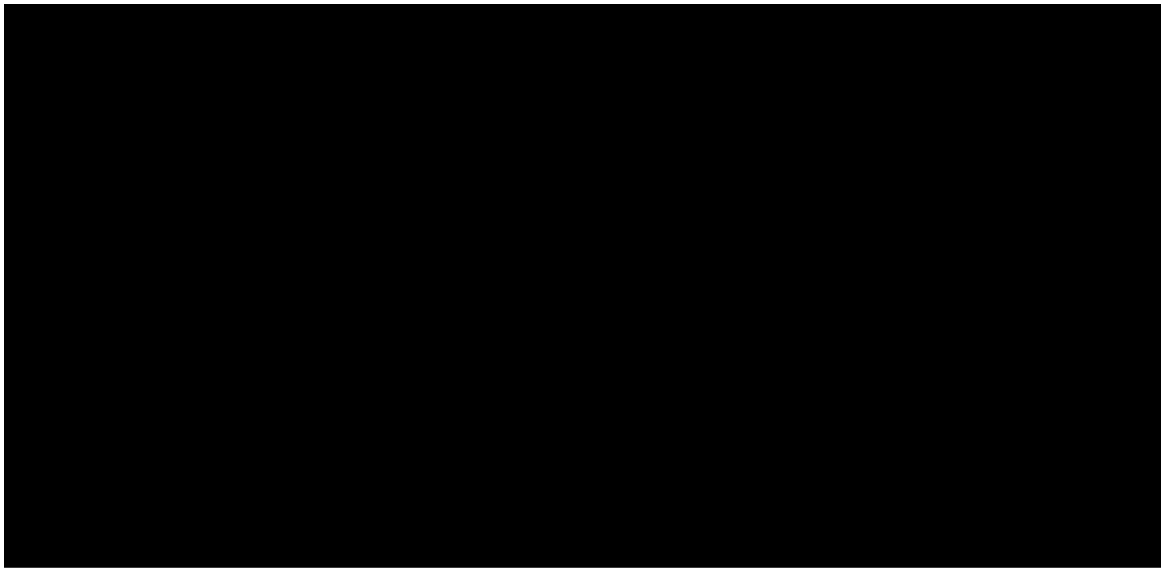
- Acute,generalized,severe type I reaction &may cause death.
- Clinically presents with bronchospasm,facial&laryngeal edema, hypotension, nausea/vomiting/diarrhea.
- Management:-
- ABCDE protocol
- Position pt. lying flat & feet raised
- Give oxygen
- Monitor BP
- Establish venous access
- 0.5ml of 1:1000(1mg/ml)epinephrine IM &repeat after 5 min. if shock persists
- Administer antihistamines IV(10-20mg chlorpheniramine)
- Administer 100mg IV hydrocortisone
- If hypotension persists>1.2L of fluid





TYPE 2 HYPERSENSITIVITY

- Cytotoxic hypersensitivity
- Antibody mediated causing destruction of healthy cells
- Specific to tissue/organ
- Central tolerance prevents reaction of immune cells to normal healthy cells/self antigens.
- B cells release IgM &IgG>react to self antigens on surface of cells>can be intrinsic or extrinsic e.g, penicillin & activates complement system.
- Involves 3 cytotoxic mechanisms.



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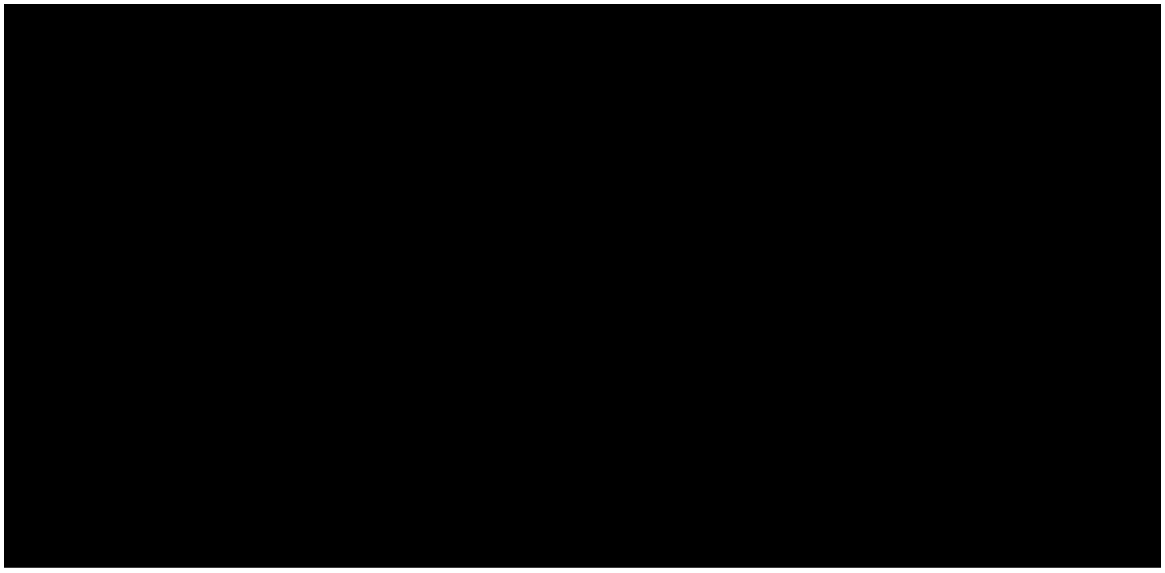
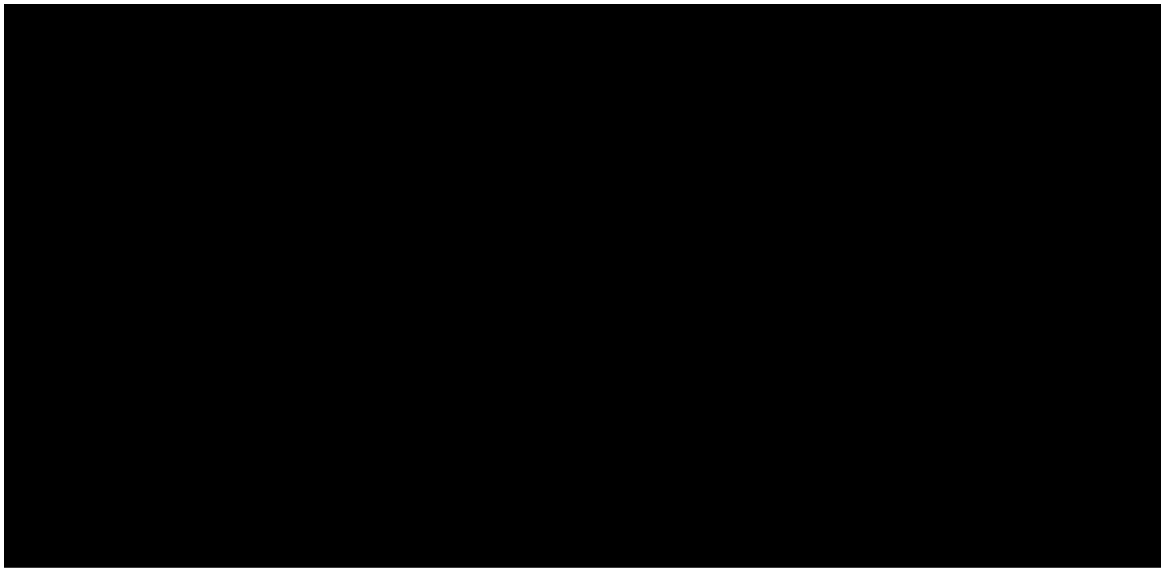
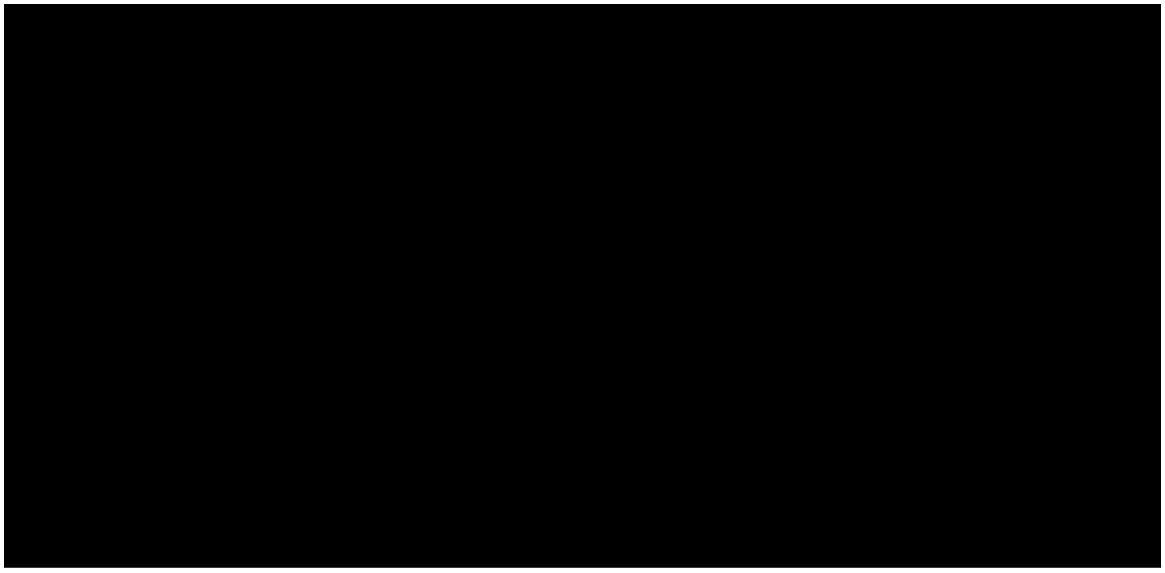


Cont'd.

- cytotoxic mechanism 1:-
- Neutrophils recruited>enzymes released>oxygen free radicals>destroy cells.
- extrinsic>drug reactions>antigens mostly blood bound> hemolytic anemia, thrombocytopenia, neutropenia
- Intrinsic>GoodPasture syndrome>antibodies against basement membrane in kidneys & alveoli

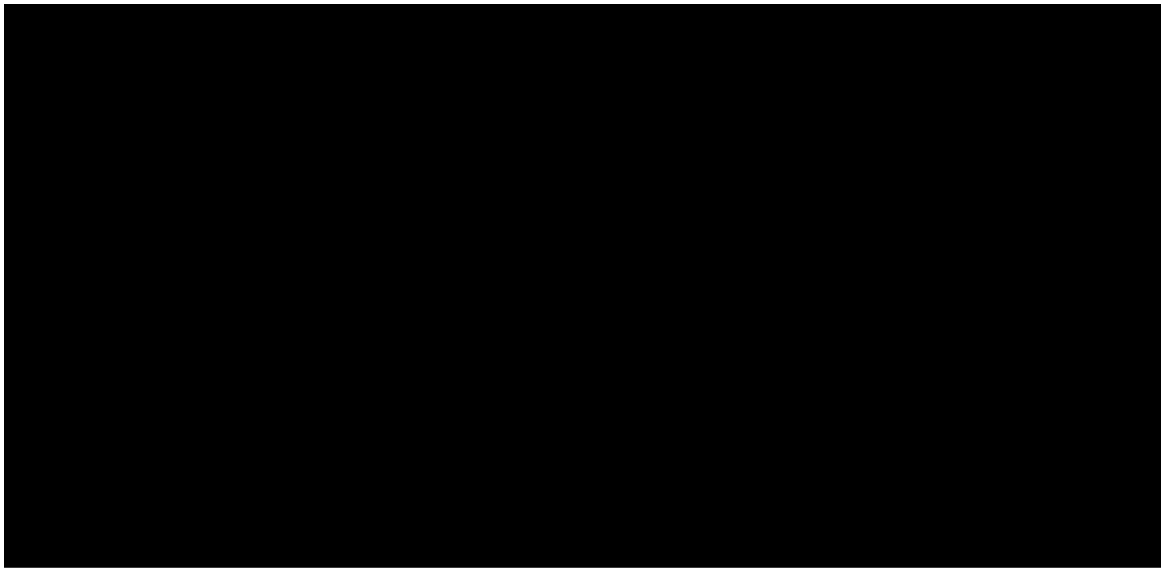


- Cytotoxic mechanism 2:-
- Last complement proteins of complement cascade bind together and form membrane attack complex(MAC)
- Attaches to antigen on cell>opens membrane>cell lysis
- E.g, ABO incompatibility
- Cytotoxic mechanism 3:-
- C3b binds to IgG on cells>opsonized>phagocytosis in the spleen



TYPE 3 HYPERSENSITIVITY

- Antigen-Ab complexes bind to blood vessel walls>tissue damage
- Plasma cells typically switch from IgM to IgG.
- Differs from type 2 that immune complexes are present in blood rather than on cell surface
- Immune complexes bound to blood vessels>complement cascade(on a larger scale than type 2)>inc. capillary permeability>edema
- Neutro`phils recruited>degranulation>cell damage>vasculitis(in kidneys,joints)
- E.g, SLE, serum sickness



TYPE 4 HYPERSENSITVITY

- cell mediated(CD4+ & CD8+ T cells)
- Antigen+APC>lymphnode> CD4+ T cell binds and matures>releases IL-2 &IFN>macrophages release further cytokines>edema/urticaria
- Delayed 6 to 8 hrs.
- CD8+ binds to APC & directly kills/damages the host cell.
- E.g, atopic dermatitis,tuberculosis,multiple sclerosis, IBD