GLUCORTICOSTEROIDS

DR SHAMS SULEMAN

LEARNING OBJECTIVES

- Classify Glucocorticoids
- Describe the mechanism of action, pharmacological effects, clinical uses, and adverse effects of glucocorticoids
- Describe dexamethasone suppression test

ADRENOCORTICAL HORMONES

Basal Secretion

Group	Hormone	Daily
Glucocorticoids	Cortisol	5 – 30 mg
	Corticosterone	2 – 5 mg
Mineralocorticoids	Aldosterone	5 – 150 mcg
	11- deoxycorticosterone	Trace
Sex Hormones		
Androgen	DHEA	15 – 30 mg
Progestogen	Progesterone	0.4 – 0.8 mg
Oestrogen	Oestradiol	Trace

ADRENAL HORMONES: SOURCES

Natural

- Glucocorticoids
  - Cortisol (Hydrocortisone)
  - Corticosterone
- Mineralocorticoids
  - v11-Desoxycorticosterone Acetate (Doca)
  - Aldosterone

Synthetic

GLUCOCORTICOIDS

Signs and Symptoms of Adrenal Crisis

- Headache / dizziness
- Low back pain
- Stomach / leg pain
- Pale skin/shivering
- Severe vomiting / diarrhea
- Lethargy / listlessness
- Loss of appetite
- Neurological deficits
- Confusion
- Low blood sugar
- Low blood pressure
- Seizures
- Cardiovascular collapse
- May present with shock-like symptoms

CLASSIFICATION

1. Short acting
2. Intermediate acting
3. Long acting
4. Inhalational

CLASSIFICATION

Short acting (8-12 hours)

- Hydrocortisone (cortisol)
- Cortisone

CLASSIFICATION

Intermediate acting (12-36 hours)

- Prednisone
- Prednisolone
- Methylprednisolone
- Fluprednisolone
- Paramethasone
- Triamcinolone

CLASSIFICATION

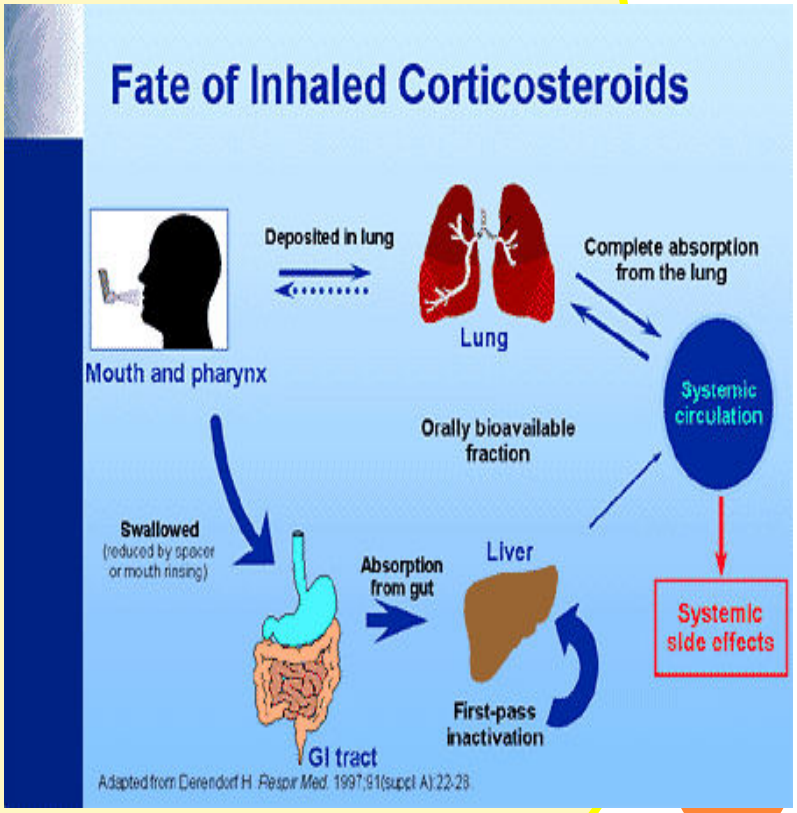
Long acting (36-72 hours)

- Betamethasone
- Dexamethasone
- Beclomethasone
- Budesonide
- Fluticasone

CLASSIFICATION

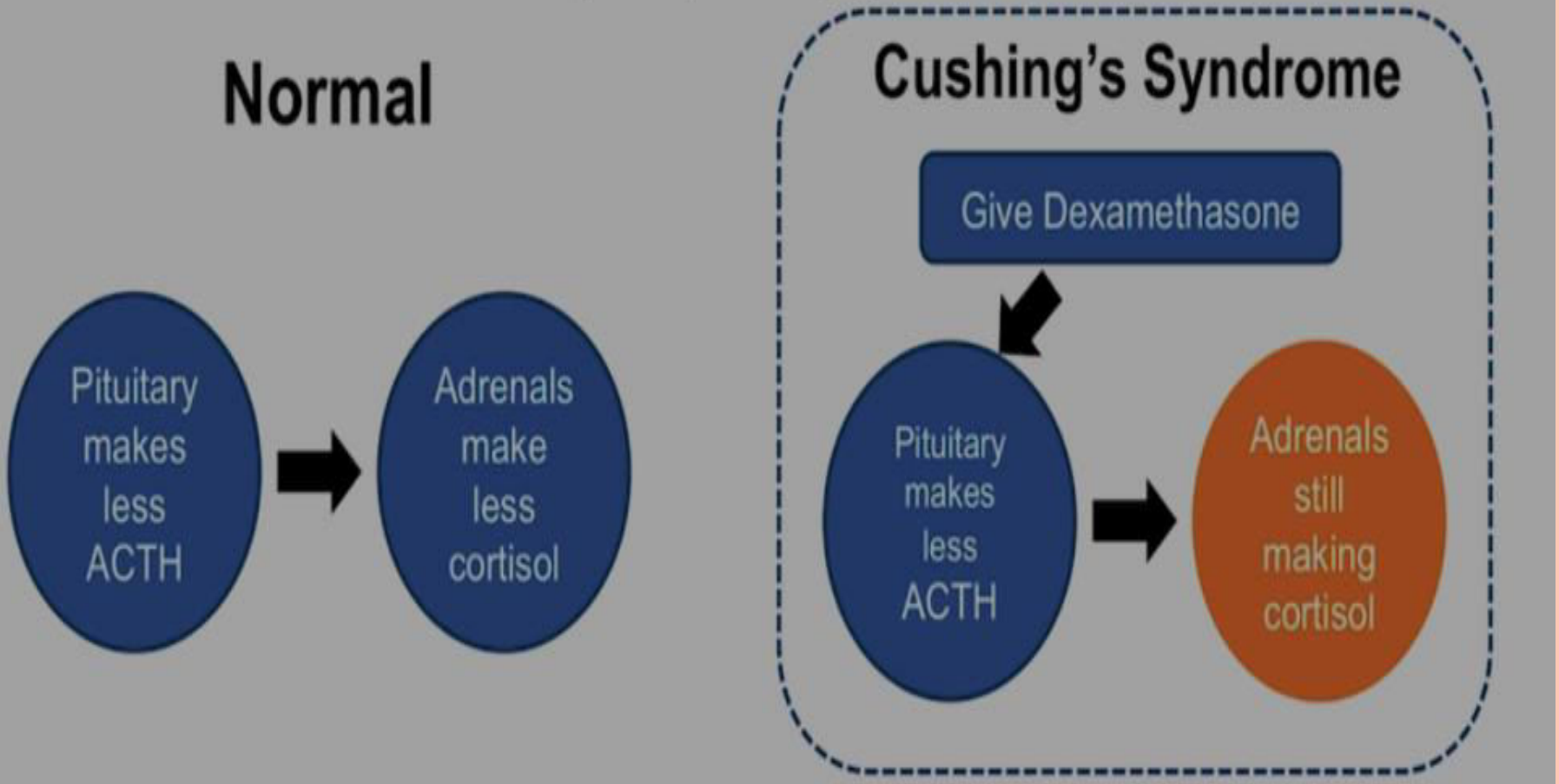
Inhalational

- Triamcinolone
- Beclomethasone
- Budesonide
- Fluticasone
- Ciclesonide
- Flunisolide
- Mometasone

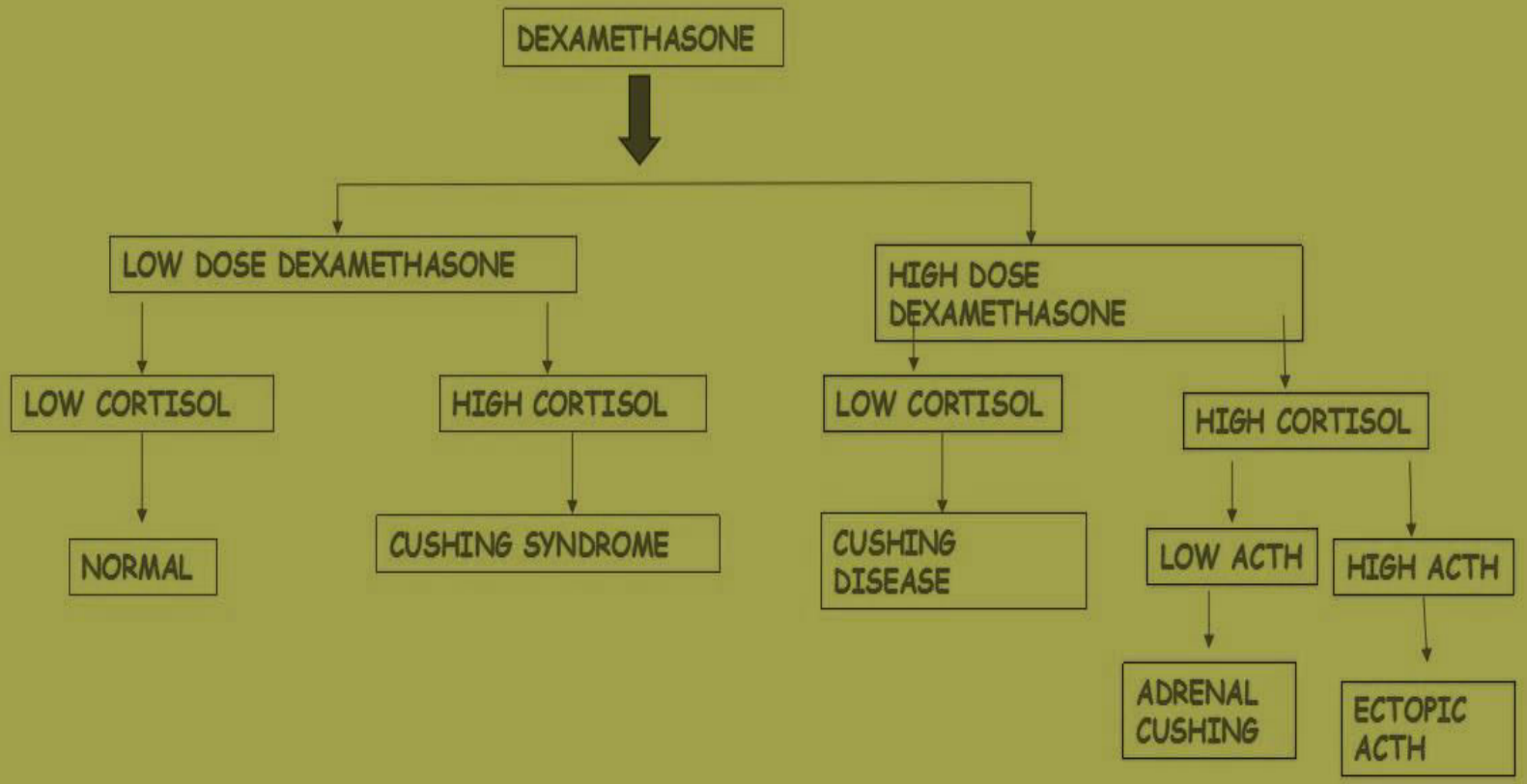


Dexamethasone Suppression Test

Dexamethasone acts like cortisol, lowers the amount of ACTH released by the pituitary gland



Dexamethasone Suppression Test



medcrine

DEXAMETHASONE SUPPRESSION TEST (DST)*		
Types of DST		**Indication**
Low-dose DST	**Overnight 1 mg test**	Diagnosis of endogenous CS
	2 mg- two day test	
High-dose DST	**Overnight 8 mg test**	Differentiate etiology of ACTH-dependent CS (pituitary vs ectopic)
	8 mg- two day test	
Intravenous DST		Diagnosis of endogenous CS
		Differentiate CD from adrenal/ectopic CS
Dexamethasone-CRH		Differentiate between CD & pseudo-CS

CS: Cushing's syndrome (endogenous); CD: Cushing's disease

*See text for details on test interpretation

PHARMACOLOGICAL EFFECTS

- **Corticosteroids** are a class of steroid hormones that are produced in the adrenal cortex.
- Corticosteroids are involved in a wide range of physiologic systems such as
- stress response,
- immune response and regulation of inflammation,
- carbohydrate metabolism,
- protein catabolism,
- blood electrolyte levels, and
- behavior.

PHARMACOLOGICAL EFFECTS

(Extensions of physiological effects)

1. **PHYSIOLOGIC EFFECTS**
   - Direct effects
   - Permissive effects
     - Response of vascular & bronchial smooth muscle to catecholamines
     - Lipolytic response of fat cells to ACTH, catecholamines, growth hormones

PHARMACOLOGIC EFFECTS

2. **METABOLIC EFFECTS**
   - Carbohydrate – gluconeogenesis, glycogen, decrease glucose utilization, increase blood glucose levels
   - Protein (catabolic & anti-anabolic effects) – in lymphoid tissue, muscle, fat, skin
   - Lipid – lipolysis, redistribution of body fat (different tissue sensitivity

PHARMACOLOGIC EFFECTS

3. **ANTI-INFLAMMATORY EFFECTS**
4. **IMMUNOSUPPRESSIVE EFFECTS**
5. **CNS** – sense of well being, insomnia, restlessness, depression, psychosis
6. **Skeletal muscle**: required for normal function
7. **CVS**: mineralcorticosteroid –induced changes, enhance vascular reactivity to other vasoactive substances

ANTI-INFLAMMATORY & IMMUNOSUPPRESIVE ACTION

Reduced manifestations of inflammation

- Decreased release of vasoactive & chemo attractive factors
- Decreased secretion of lipolytic & proteolytic enzymes
- Decreased extravasation of leukocytes (increased neutrophils in blood, decreased lymphocytes)
- Decreased fibrosis
- Decreased expression of pro-inflammatory cytokines: COX2

Immunosuppressive & anti-allergic actions

- Suppresses all types of hypersensitivity & allergic phenomenon
- At High dose: Interfere with all steps of immunological response
- Causes greater suppression of CMI (graft rejection & delayed hypersensitivity)
- Transplant rejection: ↓ antigen expression from grafted tissues, delay revascularization, ↓ sensitisation of T lymphocytes etc.

CELL TYPE	FACTOR	COMMENTS
Macrophages and monocytes	Arachidonic acid and its metabolites (prostaglandins and leukotrienes)	Mediated by glucocorticoid inhibition of COX-2 and PLA_2.
	Cytokines, including: interleukin (IL)-1, IL-6, and tumor necrosis factor-α (TNF-α)	Production and release are blocked. The cytokines exert multiple effects on inflammation (e.g., activation of T cells, stimulation of fibroblast proliferation).
	Acute phase reactants	These include the third component of complement.
Endothelial cells	ELAM-1 and ICAM-1	ELAM-1 and ICAM-1: critical for leukocyte localization.
	Acute phase reactants Cytokines (e.g., IL-1) Arachidonic acid derivatives	Same as above, for macrophages and monocytes.
Basophils	Histamine, LTC_4	IgE-dependent release inhibited by glucocorticoids.
Fibroblasts	Arachidonic acid metabolites	Same as above for macrophages and monocytes. Glucocorticoids also suppress growth factor–induced DNA synthesis and fibroblast proliferation.
Lymphocytes	Cytokines (IL-1, IL-2, IL-3, IL-6, TNF-α, GM-CSF, interferon-γ)	Same as above for macrophages and monocytes.

Glucocorticoids - Pharmacokinetics

- Therapeutically given by various routes – orally, IM, IV, topically
- Hydrocortisone undergoes high first pass metabolism
- Oral bioavailability of synthetic corticoids is high
- Both, endogenous and therapeutically administered GC are bound to Corticosteroid Binding Globulin (CBG)
- Synthetic steroids have to undergo reduction in liver to active compounds
- Metabolized in liver and excreted in urine
- Exogenously administered hydrocortisone has t1/2 of 1.5 Hrs

Preparations

- **Oral:** Tablets
- **Parenteral:** Dexamethasone
  Hydrocortisone
  Pulse therapy =
  Methyl prednisolone
  1 gm IV OD * 3 days
  Depot = Triamcinolone Acetonate
- **Respiratory:**
  Nebulizers
  Revolizers
  Inhalers
- **Topical:**
  Creams/Lotions/Solutions
- **Eye, ear & nasal**
  Creams/Lotions/Solutions

POSOLOGY & BIO EQUIVALENCE

- Usual dosage ==== 1 mg/kg/day
- Preferably in a single morning dose
- _____ Impractical !!!!!!!!!!
- Interconversions

1 mg Dexamethasone = 10 mg Prednisolone

1 mg Prednisolone = 25 mg Hydrocortisone

- Injection Hydrocortisone == 100mg/250mg/500mg
- Tab. Prednisolone == 5 mg
- Tab. Dexamethasone == 0.5 mg
- Injection 1ml Dexamethasone == 4 mg

  = 8 Tablets each of Prednisolone & Dexamethasone

Glucocorticoids - MOA

- Not stored:
  - rate of synthesis = rate of release
- Synthesize rhythmically and controlled by irregular pulses of ACTH, influenced by light and major pulses occur early in the morning and after meals
- Glucocorticoids act via their receptors located in nucleus (GR)
- GRs are widely distributed and located almost in all cells of the body
- They are made up of almost 800 amino acids

Glucocorticoids - MOA

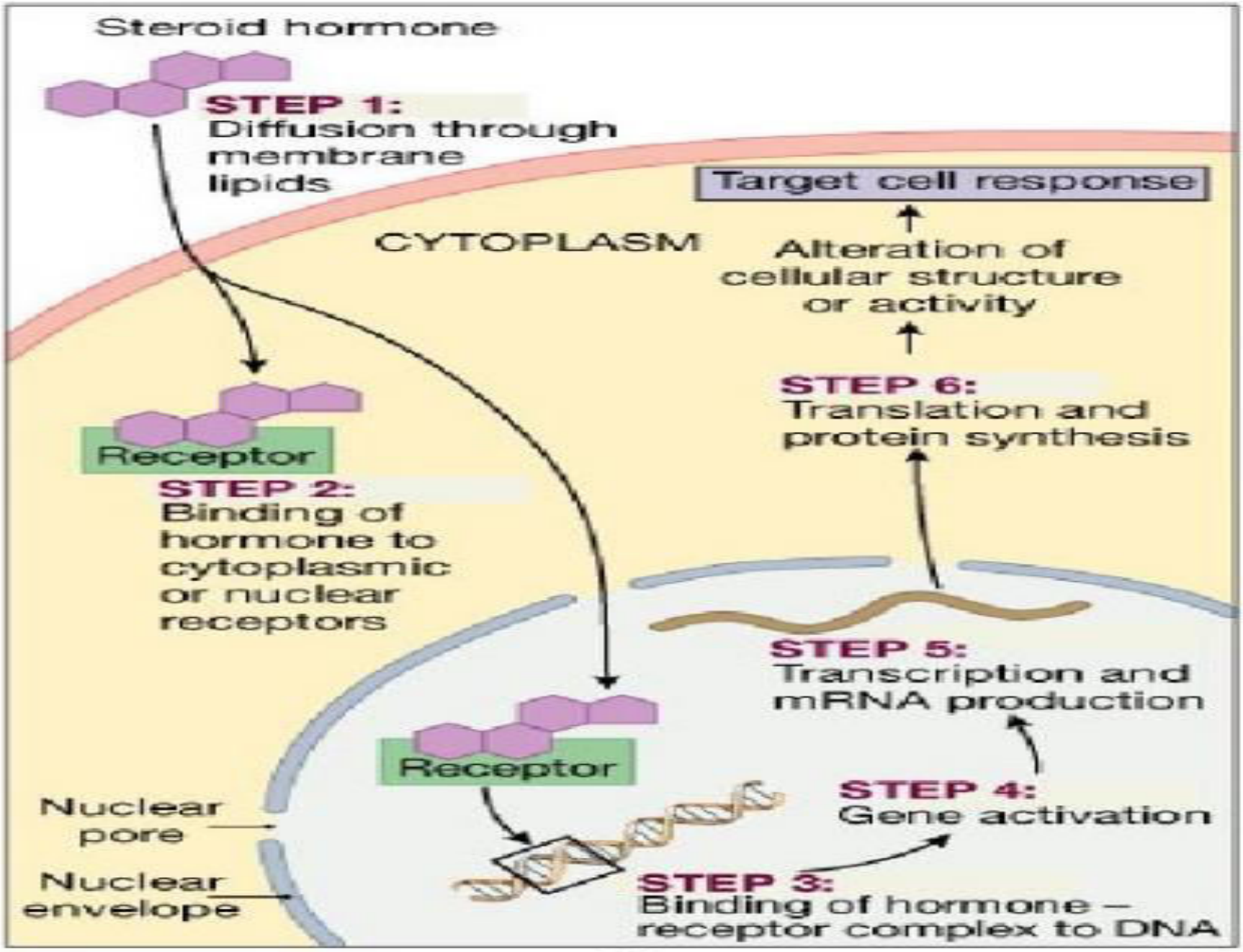
- GR receptors are located in the cytoplasm
- One GR receptor has a DNA binding domain and a ligand binding domain along with stabilizing proteins (HSP 90 and HSP 70)
- This receptor is incapable of activating transcription
- Binding of free steroid molecule to GR forms an unstable compound
- Therefore HSP and other proteins get dissociated
- The S+GR complex enters the nucleus and binds to Glucocorticoids response element (GRE) on gene and regulate transcription by RNA polymerase II and others
- The resulting mRNA is transported to cytoplasm for production of protein and bring about final response

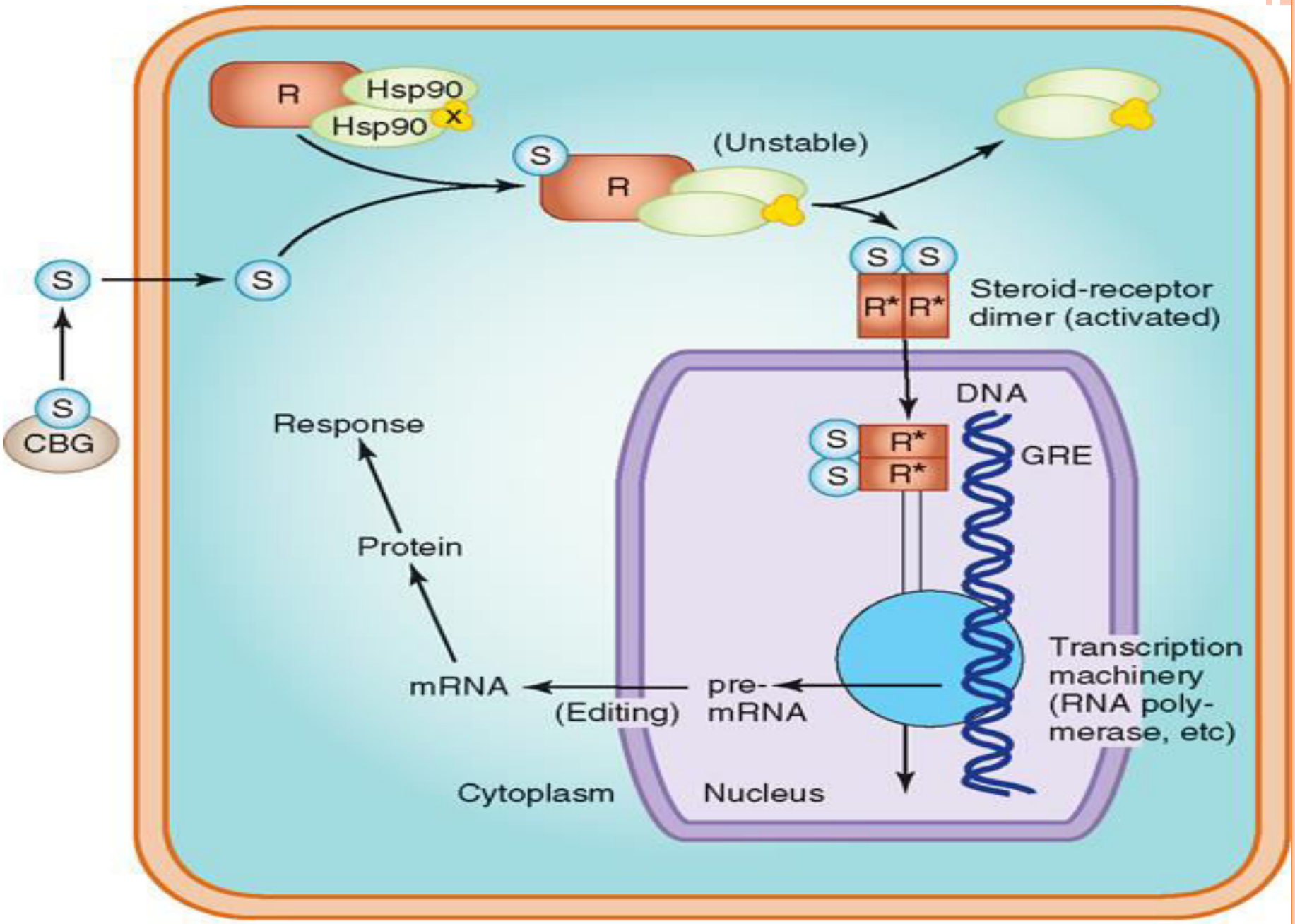
Hormone enters cell & binds to glucocorticoid receptor

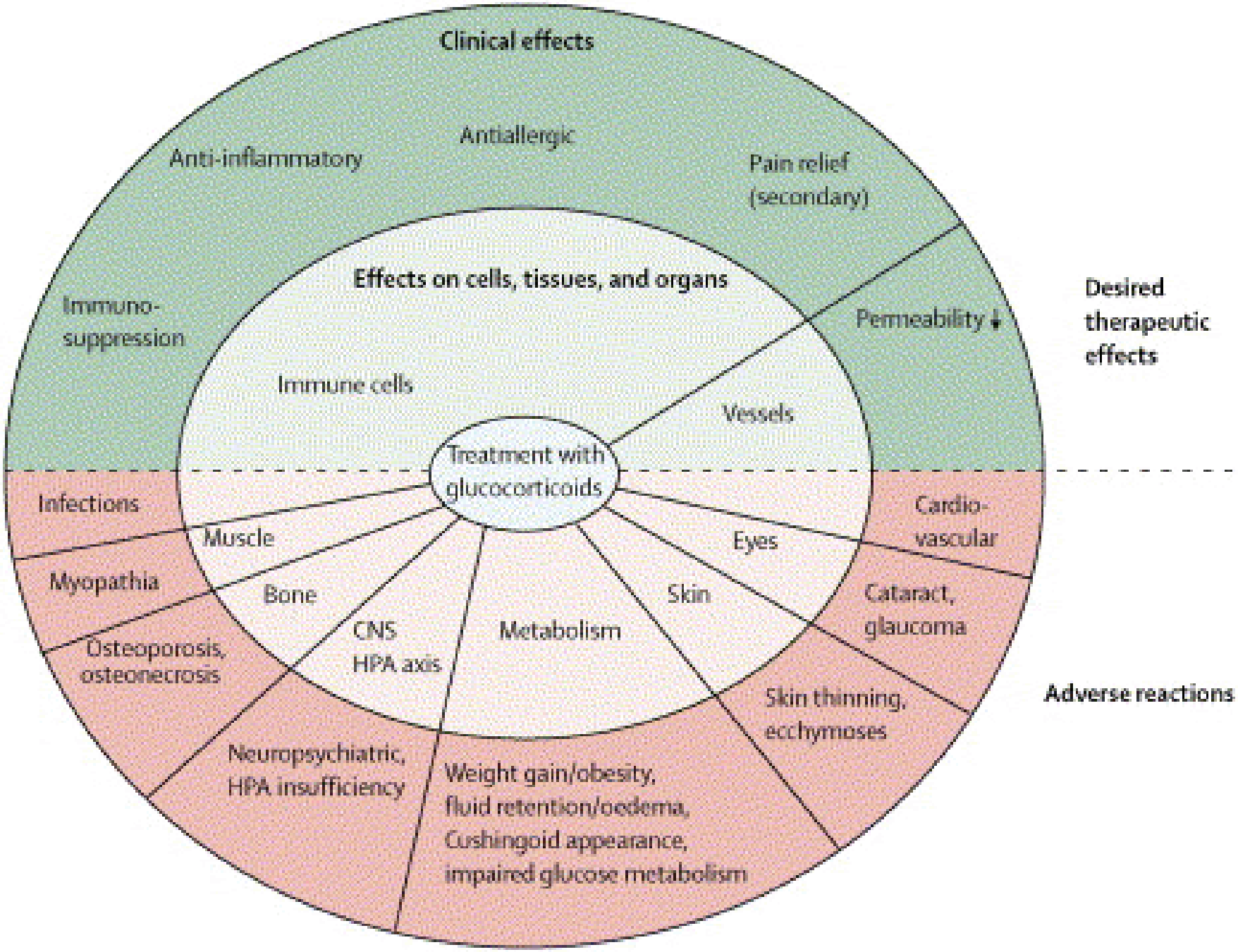
Conformational changes that allow it to dissociate from the Heat shock protein(HSP)

Dimerization - dimeric ligand-bound receptor complex is transported into the nucleus

Interacts with DNA & nuclear proteins through GREs in the promoter of responsive genes


Steroid hormone
STEP 1:
Diffusion through membrane lipids
Target cell response
CYTOPLASM
Alteration of cellular structure or activity
STEP 6:
Translation and protein synthesis
Receptor
STEP 2:
Binding of hormone to cytoplasmic or nuclear receptors
STEP 5:
Transcription and mRNA production
Receptor
Nuclear pore
STEP 4:
Gene activation
Nuclear envelope
STEP 3:
Binding of hormone – receptor complex to DNA


R
Hsp90
Hsp90
x
S
R
(Unstable)
S
S
S
S
R*
R*
Steroid-receptor
dimer (activated)
S
CBG
DNA
Response
S
R*
S
R*
GRE
Protein
Transcription
machinery
(RNA poly-
merase, etc)
mRNA
(Editing)
pre-
mRNA
Cytoplasm
Nucleus

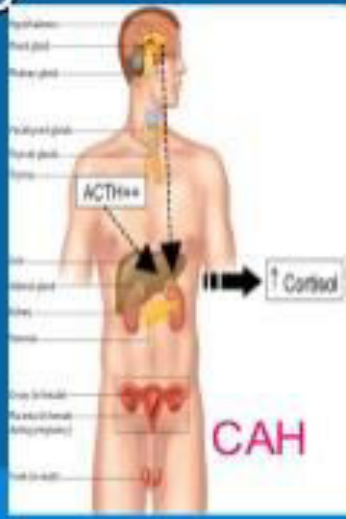

Clinical effects
Antiallergic
Anti-inflammatory
Pain relief (secondary)
Effects on cells, tissues, and organs
Immuno-suppression
Permeability ↓
Desired therapeutic effects
Immune cells
Vessels
Treatment with glucocorticoids
Infections
Muscle
Eyes
Cardio-vascular
Myopathia
Bone
Skin
Cataract, glaucoma
CNS HPA axis
Metabolism
Osteoporosis, osteonecrosis
Skin thinning, ecchymoses
Neuropsychiatric, HPA insufficiency
Weight gain/obesity, fluid retention/oedema, Cushingoid appearance, impaired glucose metabolism
Adverse reactions

THERAPEUTIC USES

1. Diagnosis & treatment of disturbed adrenal function
2. Prevention of infant respiratory distress syndrome
3. Non-adrenal disorders

Replacement Therapy

- Adrenal insufficiency – acute/chronic
  - Abrupt withdrawal of steroid therapy
  - Chronic infections – Tuberculosis
  - Autoimmune adrenal disease
  - Surgery, Hemorrhage and AIDS
- Congenital adrenal hyperplasia
  - Congenital disorder due to deficiency of 21-hydroxylse enzyme – no cortisol but ACTH – increased androgen production



Addisonian Crisis

Features:

- Severe shock – hypotension, tachycardia
- Fever, abdominal pain, nausea & vomiting
- Hyponatraemia/hyperkalaemia ±hypercalcaemia, hypoglycaemia

Management:

ABCDE assessment

- Correct volume depletion
- Replace glucocorticoids
- Correct metabolic abnormalities
- Treat underlying cause

100mg hydrocortisone I/V every 8hrs

Replacement Therapy

- Acute adrenal insufficiency
  - IV replacement of sodium chloride and fluid
  - IV hydrocortisone 100 mg stat followed by100 mg every 8 Hrs – maximal daily rate of secretion

(alternatively, dexamethasone can be used)

- Chronic adrenal insufficiency
  - Hydrocortisone
  - Prednisolone or dexamethasone – long acting
  - Fludrocortisone for mineralocorticoid effects
- Congenital adrenal hyperplasia
  - Hydrocortisone 0.6 mg/kg in divided doses – to maintain feedback suppression

EMERGENCY USES

- Acute severe asthma
- Raised intracranial pressure
- Septic shock
- Acute adrenal insufficiency
- Anaphylactic shock – 2nd choice

NON-ADRENAL DISORDERS

1. **Allergic reactions:**

- Urticaria
- Angio -neurotic edema
- Contact dermatitis
- Bee stings
- Serum sickness,
- Drug reactions

Allergic Disorders

- Exhibit a delayed response in allergies (1-2 hrs even in IV injection)
- In anaphylaxis, angioneurotic oedema and serum sickness etc. – adrenaline is the choice
- Seasonal allergies, bee sting, drug allergies –
  - Allergic reactions can be suppressed by corticosteroids as supplements
- Intranasal administration in allergic rhinitis - budesonide and flunisolide

NON-ADRENAL DISORDERS

2. **Anti-Inflammatory& Immunosuppression**:

- Inflammatory Arthritides; RA, SLE, Gout, Reactive Arthritis
- Bronchial Asthma
- Inflammatory Bowel Disease
- Bechet's syndrome
- Nephrotic syndrome
- Organ transplant
- Subacute thyroiditis
- Sarcoidosis, asthma

Anti-inflammatory Uses

- For suppression of inflammatory components in –
  - Rheumatoid arthritis – as adjuvant with NSAIDs in severe cases
  - Osteoarthritis – NSAIDs, intra-articular injection
  - Rheumatic fever – severe cases with carditis and CHF
  - Gout – NSAID failed cases and colchicine failed cases – intra-articular injection
  - Vasculitic disorders: Polyarteritis nodosa

Intra-articular Steroids

Can be used in inflammatory
Non-inflammatory diseases

- Knee joint
- Shoulder joint
- Tennis elbow
- Carpal tunnel syndrome

Bronchial Asthma

- The increased recognition of the immunological and inflammatory nature of Bronchial asthma has led to the use of corticosteroids
- In severe asthma attacks

  IV hydrocortisone Methylprednisolone

  Oral prednisolone
- Acute attacks:

  *Inhaled beclmethasone, budesonide, flunisolide alone or combined with beta-2 agonists/ipratropium

  *Oral steroids

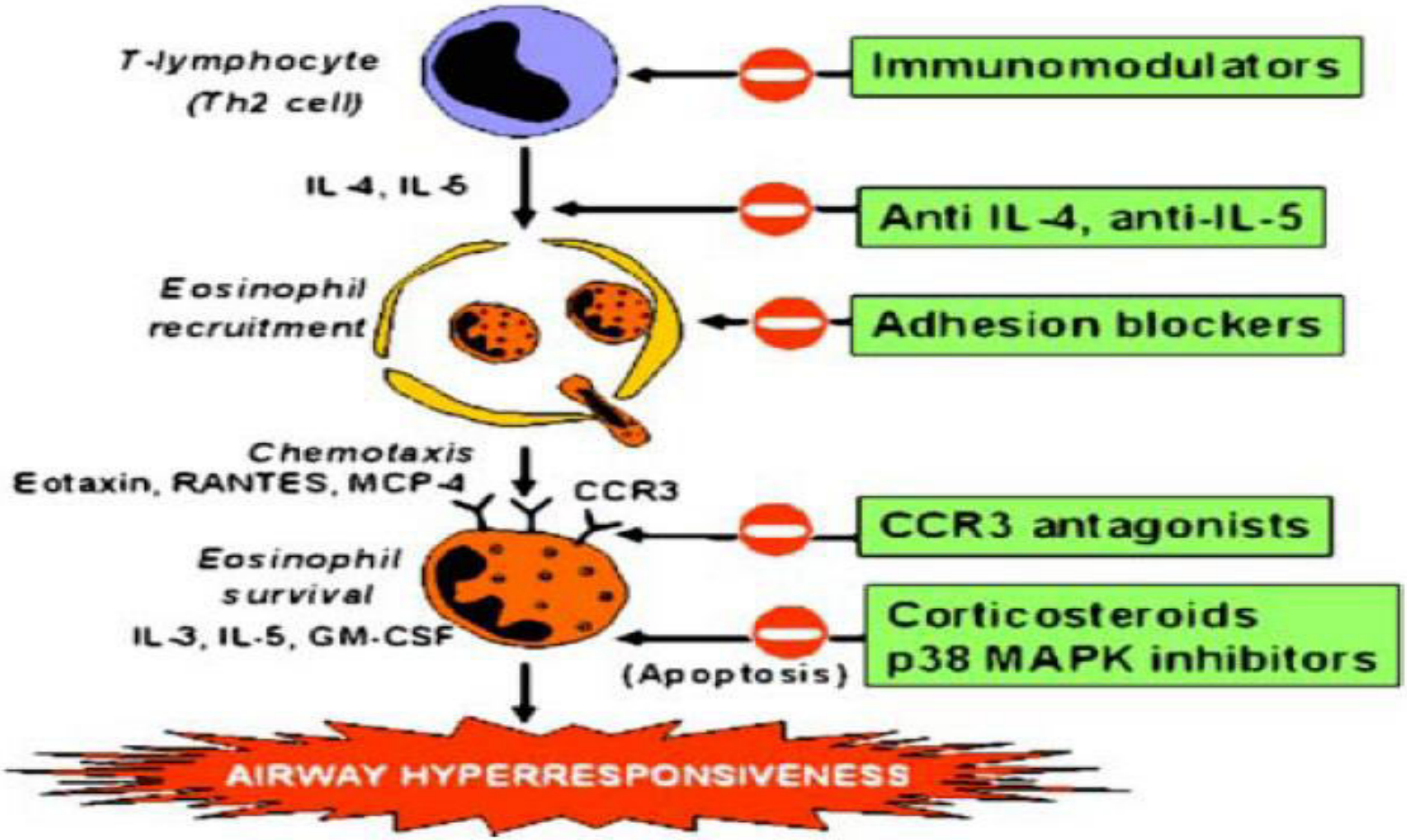
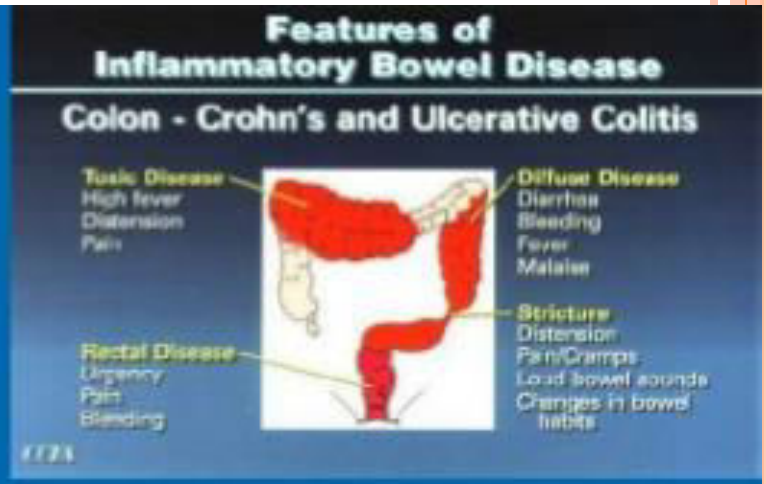


Figure 1. Inhibition of eosinophilic inflammation. Several strategies are possible to inhibit eosinophil inflammation in tissues, including immunomodulators (e.g., cyclosporin), inhibitors of proinflammatory cytokines (e.g., IL-4 and IL-5), inhibition of critical adhesion molecules (e.g., VLA4), blockade of chemokine receptors on eosinophils (e.g., CCR3) and induction of apoptosis (e.g., by corticosteroids and p38 MAP kinase inhibitors).

GIT



- Inflammatory conditions of intestine like
  - Ulcerative colitis
  - Crohn`s disease
  - Coeliac disease

  (oral therapy or retention enema with hydrocortisone)
- May mask the major complications like perforation and peritonitis

Autoimmune diseases

- Autoimmune haemolytic anaemia
- Idiopathic thrombocytopenic purpura
- Active chronic hepatitis, alcoholic hepatitis

(Prednisolone 1-2 mg/kg/day given till remission followed by gradual withdrawal or low dose maintenance)

Renal diseases

- Nephrotic syndrome in children
- Renal disease secondary to SLE
- Renal sarcoidosis
- Glomerulonephritis – membranous type

(Life saving importance – usually given in large doses followed by tapering to maintenance dose)

Ocular Diseases

- Important drug therapy for suppressing inflammation in eye and preservation of sight
- Topical instillations are used for conditions of the anterior chamber – allergic conjunctivitis, iritis, iridocyclitis and keratitis etc.
- Systemic steroids for the posterior chamber
- Dexamethasone topical 0.1%
- Prednisolone oral
- Contraindicated in viral, fulminant bacterial infections, fungal infections and injuries

NON-ADRENAL USES

3. Stimulation of fetal lung maturation (RESPIRATORY DISTRESS SYNDROME)

- Treatment of mother with large dose of glucocorticoids
- BETAMETHASONE I/M to mother

NON-ADRENAL USES

4. Infections: Gram negative septicemia
5. Leukemia, lymphoma
6. Pemphigus and other skin diseases
7. Use in raised intracranial pressure
8. Mountain sickness
9. Hypercalcemia
10. Multiple sclerosis

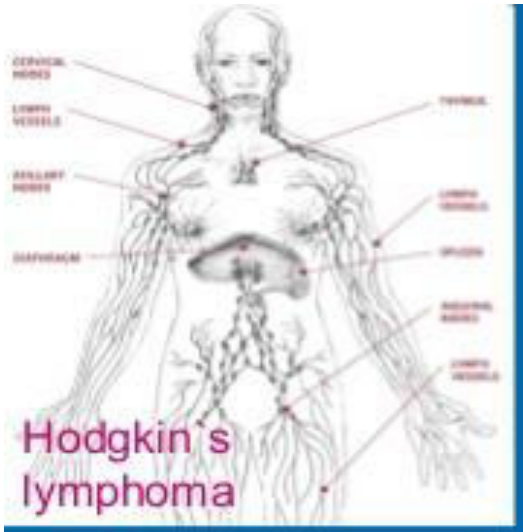
Infectious Diseases

- Indicated only in severe infective diseases to tide over crisis or prebent complictions
  - AIDS and pneumocystis carinii pneumonia
  - In haemophilus influenza meningitis to reduce neurological complications
  - Tubercular meningitis
  - Lepra reaction
  - Scepticaemia

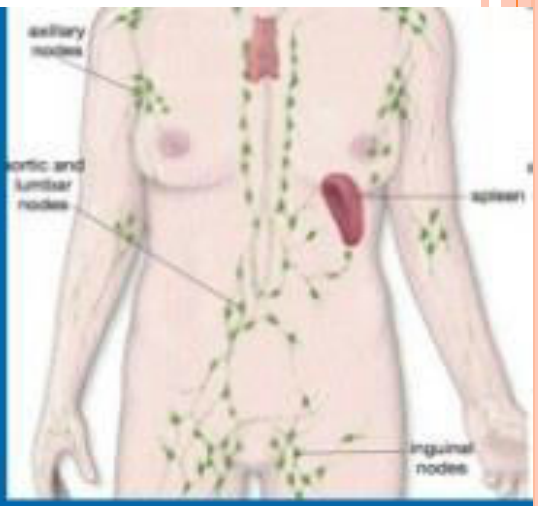

Lepra reaction

Indications of Corticosteroids (along with ATT) in T.B

- Massive T.B pleural effusion
- Massive T.B pericardial effusion
- T.B ascites (massive)
- T.B meningitis
- T.B choroiditis



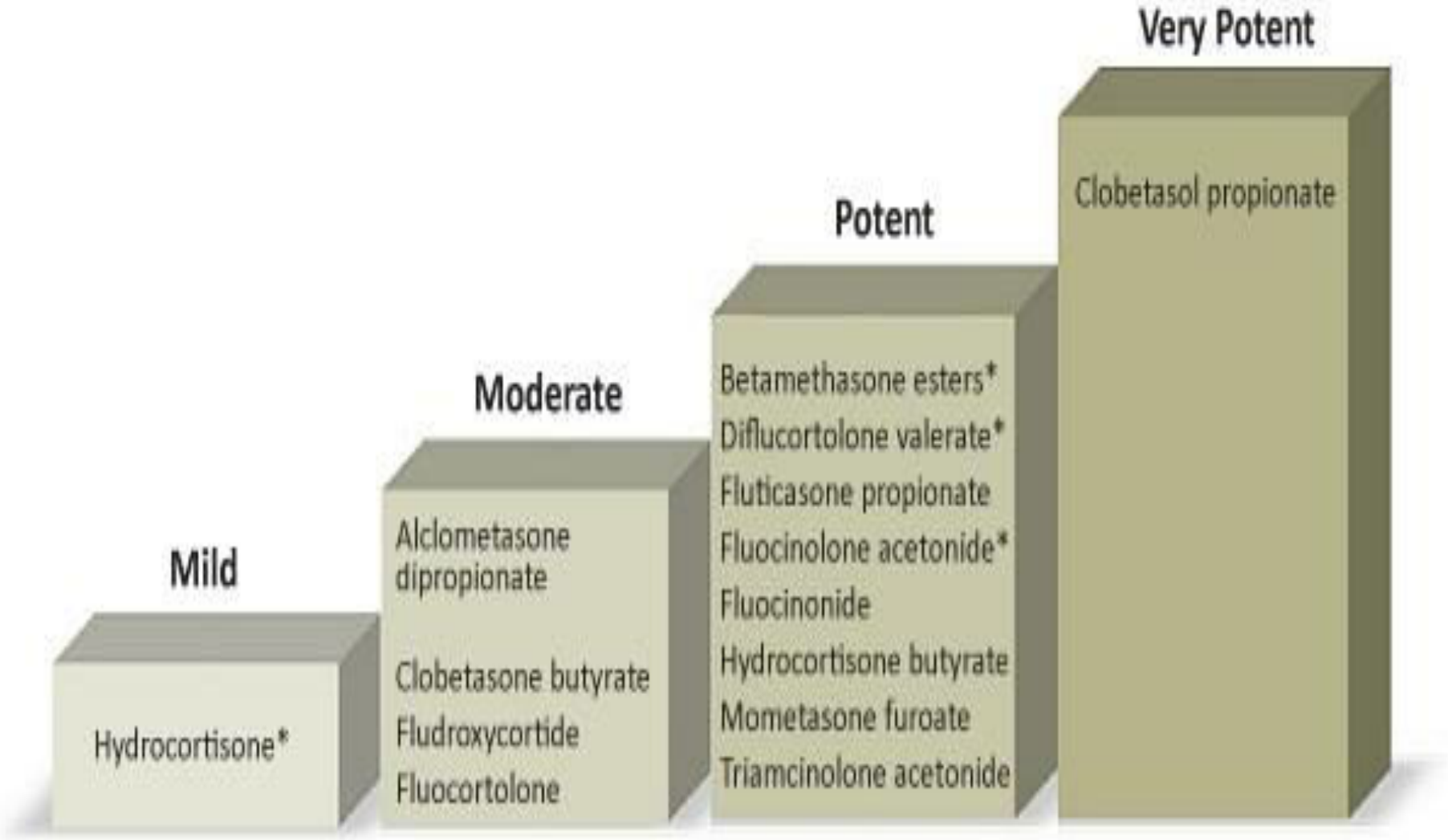
Malignancy



- Essential for combined chemotherapy of
  - Acute lymphatic leukemia
  - Hodgkin's and other lymphomas
  - Hormone responsive breast carcinoma
- Symptomatic relief in other advance malignancies by improving appetite and controlling secondary hypercalcaemia

Skin Diseases

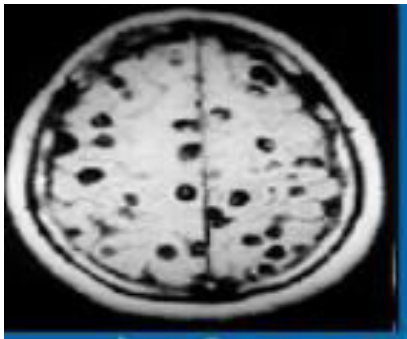
- The largest application of steroid therapy
- Topical forms are widely used in many eczematous skin diseases
- Systemic therapy are also required and may be life saving in
  - Pemphigus vulgaris
  - Exfoliative dermatitis
  - Stevens-Johnson syndrome



Potency of steroid creams

Potency	Generic name	Delivery vehicle and trade name
Class 1: Weak	Hydrocortisone acetate 1%	cream (Biocort®[f]; Stopitch®[g])
	Hydrocortisone acetate 0,5%	cream, ointment (Dilucort®[h])
	Hydrocortisone 1%	cream (Procutan®[c];Vari-Hydrocortisone®[b])
	Hydrocortisone 0.5%	cream (Skincalm®[h])
Class 2: Moderately Potent	Betamethasone valerate 0.05%	cream (Betnovate Half Strength®[i])
Class 3: Potent	Fluticasone propionate 0.05%	Cream (Cutivate®[k])
	Fluticasone propionate 0.005%	Ointment (Cutivate®[k])
	Hydrocortisone butyrate 0.1%	cream, ointment, lotion (Locoid®[l]) topical (Locoid Crelo®[l])
	Betametasone valerate 0.1%	cream (Betnovate®[i]; Repivate®e; Adco-Betamethasone®[e]; Vari-Betamethasone®[b]) ointment, lotion (Betnovate®[i]; Lenovate®[h]; Persivate®[h])
	Fluocinolone acetonide 0,025%	ointment, gel, cream (Synalar®[i]) cream, ointment (Cortoderm®[h])
	Beclomethasone dipropionate 0.025%	cream (Beclate®[e])
	Diflucortolone valerate 0.1%	ointment, cream (Nerisone®[d])
	Methylprednisolone aceponate 0.1%	cream, ointment, milk (Advantan®[d])
	Betamethasone dipropionate 0.05%	cream, ointment (Diprosone®[c])
	Mometasone furoate 0.1%	cream, ointment, lotion (Elocon®[c]) cream (Aspen Mometasone®[h]; Mometagen®[p])
Class 4: Very Potent	Clobetasol propionate 0.05%	cream, ointment (Dermovate®[i]; Dovate®[h]; Xenovate®[h])

[f]Akacia HealthCare, Isando, South Africa; [g]Adcock Ingram, Bryanston, South Africa; [h]Aspen Pharmacare, Woodmead, South Africa; [b]Specpharm Holdings, Halfway House, South Africa; [c]MSD/Schering-Plough South Africa, Midrand, South Africa; [i]Sekpharma, Sandton, South Africa; [k]GlaxoSmithKline South Africa, Bryanston, South Africa; [l]Astellas Pharma, Bedfordview South Africa; [e]Cipla SA, Bellville, South Africa; [i]Glenmark Pharmaceuticals South Africa, Midrand, South Africa; [d]Bayer, Isando, South Africa; [p]Mylan South Africa, Modderfontein, South Africa

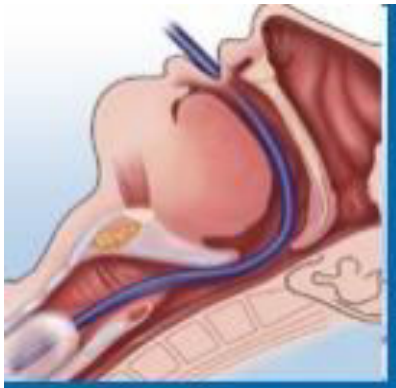


Cerebral Oedema

- Cerebral oedema due to tumors (neoplasms)
- Traumatic and poststroke oedema (?)

(Dexamethasone or betamethasone is preferred because no Na+ retaining activity)

- Other CNS conditions - spinal chord injury, Bell`s palsy and neurocysticercosis
- (Oral Prednisolone is the preferred drug)



Other Uses

HACE on MRI

- Antiemetic – with ondansetron
- Acute mountain sickness
- Aspiration pneumonia, pulmonary oedema from drowning
- Hyperthyroidism – thyroid storm

Adverse Effects

- Two types:
  - From abrupt withdrawal
  - Chronic therapeutic use of high dose
- Withdrawal
  - Flare up of underlying disease
  - Suppression of HPA axis and acute adrenal insufficiency
  - Increased ICT and papilloedema

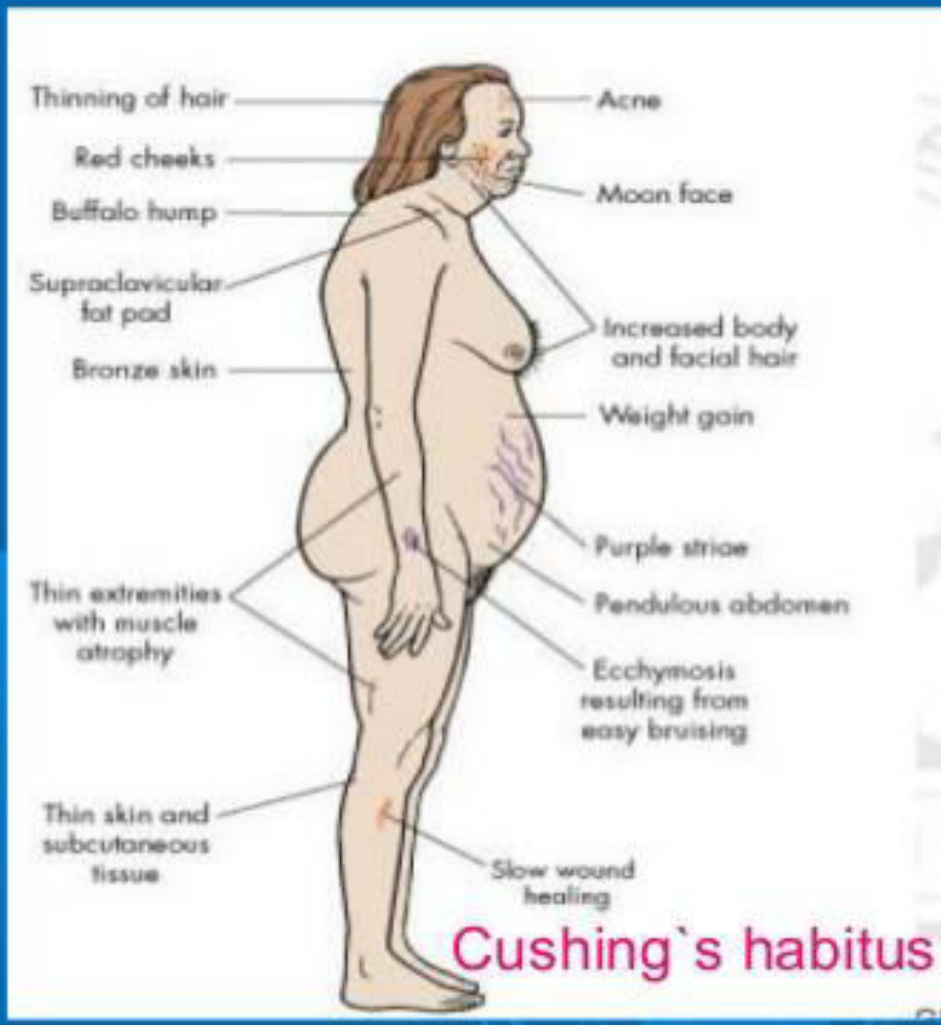
WITHDRAWAL

- Not to be stopped abruptly if patient is on hydrocortisone [>25 mg/day] or for > 2-3 weeks
- Severe adrenal crisis & death of patient due to suppression of HPA axis
- Use short acting steroids with lowest possible doses form
- Prescribe the whole daily dose of the drugs at once in the morning
- Switch to alternate day therapy if possible
- After the long term use intermediate acting steroids allow for more flexible withdrawal

ADVERSE EFFECTS

- Long term therapy – immunosuppression
- HPA axis suppression
- Addison-like symptoms
- Tapering the dose
- Physiological daily replacement (5mg prednisone) until adrenal function is restored

Adverse Effects



Cushing`s habitus

- High blood sugar
- High blood pressure
- Vertigo
- Blurred vision
- Female balding
- Menstrual irregularities
- Hirsutism
- Severe depression
- Cognitive impairment
- Emotional instability
- Easy fatiguability

Major Side Effects Associated with Corticosteroid Therapy

Dermatologic and soft tissue
- Skin thinning and purpura
- Cushingoid appearance
- Alopecia
- Acne
- Hirsutism
- Striae
- Hypertrichosis

Eye
- Posterior subcapsular cataract
- Elevated intraocular pressure/glaucoma
- Exophthalmos

Cardiovascular
- Hypertension
- Perturbations of serum lipoproteins
- Premature atherosclerotic disease
- Arrhythmias with pulse infusions

Gastrointestinal
- Gastritis
- Peptic ulcer disease
- Pancreatitis
- Steatohepatitis
- Visceral perforation

Renal
- Hypokalemia
- Fluid volume shifts

Genitourinary and reproductive
- Amenorrhea/Infertility
- Intrauterine growth retardation

Bone
- Osteoporosis
- Avascular necrosis

Muscle
- Myopathy

Neuropsychiatric
- Euphoria
- Dysphoria/depression
- Insomnia/akathisia
- Psychosis
- Pseudo tumor cerebri

Endocrine
- Diabetes mellitus
- Hypothalamic-pituitary-adrenal insufficiency

Infectious disease
- Heightened risk of typical infections
- Opportunistic infections
- Herpes zoster

ADVERSE EFFECTS

- Supraphysiological doses for > 2-3 wks
- Extensions of pharmacological effects
  - Metabolic
  - Cushing's syndrome
  - GIT
  - Muscle
  - Bone
  - Eye
  - Electrolytes
  - Water retention
  - CNS
  - Growth
- Withdrawal of therapy
  - HPA axis suppression

ADVERSE EFFECTS

- Metabolic
  - Hyperglycemia, glycosuria
  - Moon facies, obesity, hyperlipidemia
  - Increased appetite
- Skin – Increases growth of fine hair, thinning of skin, with striae & bruising, punctate acne
- GIT – peptic ulcer, pancreatitis, NV
- Muscle - muscle wasting, myopathy, growth retardation in children

ADVERSE EFFECTS

- CNS - insomnia, depression, acute psychosis
- Eye - cataract, raised IOP
- Bone - Osteoporosis, aseptic necrosis of hip
- Fluid & electrolytes - Water retention, hypokalemia, alkalosis, hypertension
- BLOOD – Lymphopenia, leukocytosis

CONTRAINDICATIONS

- Diabetes mellitus
- Peptic ulcer
- Hypertension
- Pregnancy
- Osteoporosis
- Tuberculosis & other infection
- Epilepsy
- Renal failure
- CCF

- Glaucoma
- Cushing's syndrome
- Herpes simplex keratitis
- Psychosis

MISUSE IN SPORTS

- Normal dose ___ relaxation effects on the respiratory tract
- In larger dose___ analgesic effects.

- Enables athletes for better training and sporting performance.
- Glucocorticoids are catabolic steroids.
- **VS** ___ anabolic steroids = increase muscle mass and strength.

Clinical pharmacology

Adrenocorticosteroid Inhibitors

- Metyrapone: 11 beta-hydroxylase enzyme inhibitor – used in Cushing`s syndrome and test of pituitary efficiency
- Aminoglutethemide: Stops conversion of cholesterol to pregnelone (Medical adrenalectomy) – Breast cancers
- Mifepristone: Progesterone antagonist
- Spironolactone: Aldosterone antagonist
- Ketoconazole: Inhibits synthesis of all hormones in testes and adrenal cortex – used in Cushing`s syndrome and also in hirsutism in female

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