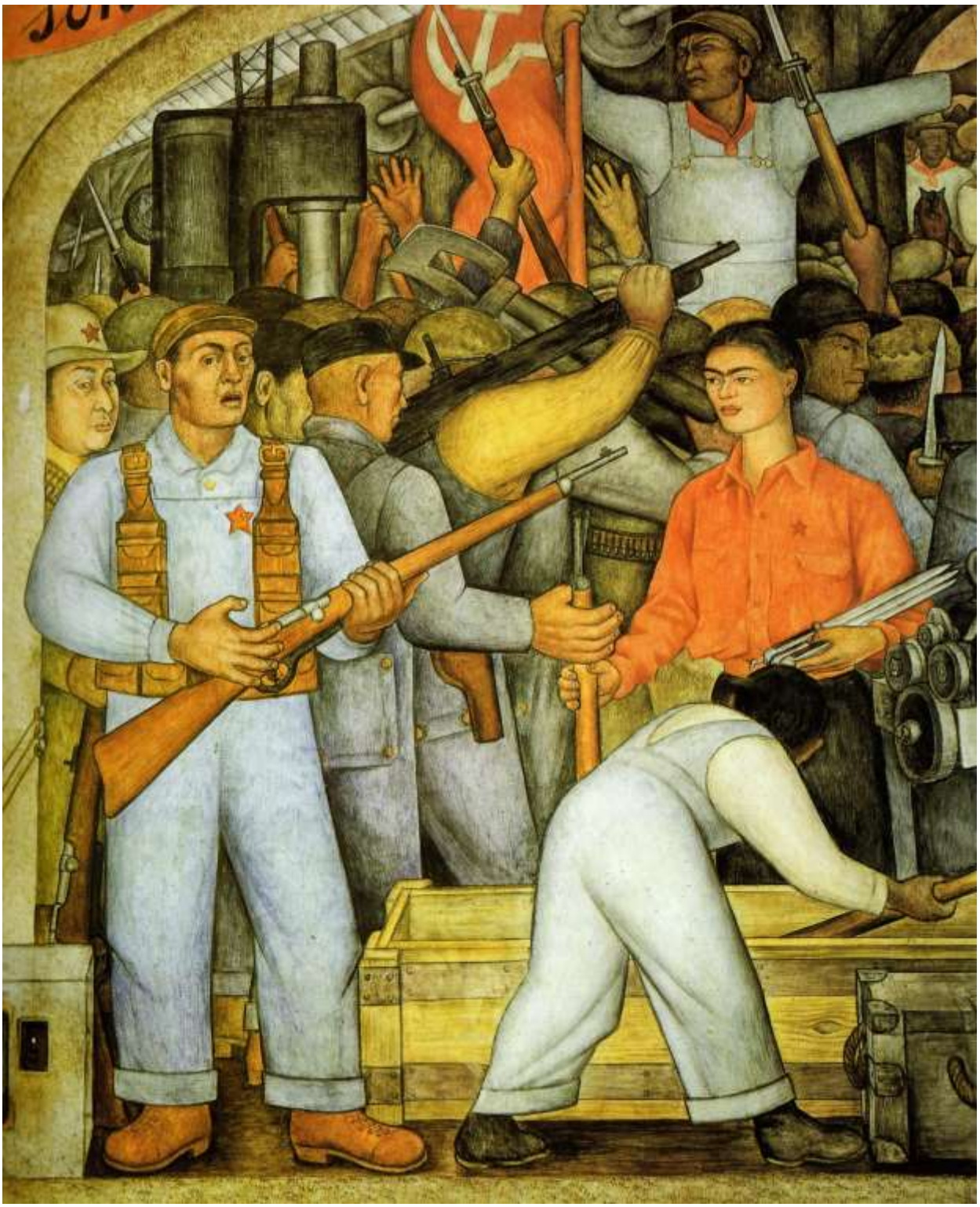
THE NORTHERN HOSPITAL

DEXAMETHASONE



"The Arsenal" - detail from "The History of Mexico" - mural fresco, 1929 - 35, Diego Rivera. Second floor, south wall, Ministry of Education, Mexico City.

Diego Rivera's second wife, Lupe Marin, recalled that when Frida first met Diego while he was working on one of his great murals, her face was strongly painted, she wore her hair bobbed and her dress was "décolleté a la flapper". But then, perhaps in part because of Diego's own Communist sympathies, Frida became intensely interested in politics, specifically in Communism, to the horror of her parents. She had by now begun to move in the circles of Mexico's Artists, all of whom were fanatical Socialists. Diego Rivera himself had become General Secretary of the Mexican Communist Party. In 1929 Frida joined the Young Communist League, and began to attend workers rallies, clandestine meetings, even began to make political speeches.

Frida's flamboyant dress sense, always very much reflected her philosophies. As a young girl in accordance with her strong bisexuality, she had dressed sometimes as a demure European woman, then startlingly at other times as a man. When her interests became political she began to dress as a Mexican worker. "She no longer wore white blouses (such as those beautifully depicted in her "Self-Portrait - Time Flies", 1929) her old boyfriend Alejnadro Gomez Arias wistfully once recalled, "Instead she wore black or red shirts and an enamel pin with a hammer and sickle". To identify with the workers she wore blue jeans and leather jackets covered in patches, and cut her hair very short. Now Frida was one of the "masses". Diego Rivera already strongly attracted to Frida on account of her Art, was now even more enchanted with her. Frida became his inspiration, as well as his lover. And in 1929, she became his muse.

In the Insurrection Panel of his "Ballad of the Proletarian Revolution" mural series in the Ministry of Education, he depicts Frida as the archetypal soldadera (female Mexican soldier) of the recent Mexican Revolution. She is flanked by other Socialist Artists such as Tina Modotti and David Alfaro Siqueiros, but Frida is most definitely center stage. She appears as a tom-boy, her hair shaved off, wearing a red workers shirt with a red Communist star on her pocket. She hands out rifles to the workers, soon to be soldiers of the Revolution. Though she was the very epitome of the soldadera, the look to Diego, who always had an eye for beauty, was not altogether the look her preferred in Frida. While she was modeling for his portrait, Diego quipped, unkindly, "You have a dog face!' Frida shot back instantly, "And you have the face of a fat frog!"

Though Frida took Diego's taunting in good humour, she actually gave back far, far worse than she ever got, she was happy enough to make herself plain for the cause of Socialism. But only up to a point.

Eager to keep Diego interested, after their marriage in August 1929, she soon discarded her unflattering workers clothes, but rather than going back to her previous European bourgeois style she chose another direction altogether - one that strongly appealed to both herself and her new husband - that of the Tehuana. Soon after they had married they left Mexico City for the southern city of Cuernavaca where Diego had received a lucrative commission from the US ambassador Dwight W. Morrow to paint a mural in the Palace of Cortes. The Tehuanas were the famous beautiful, brave, stately and strong willed women of the southwest Tehuantepec region of Mexico, and from them Frida developed her next great love, which Diego also strongly shared - a deep love of her Indian heritage and the ancient pre-Columbian culture.

The Tehuana would become Fridas identifying motif for the rest of her life, and the one that would make her instantly recognizable and famous first in all of Mexico, then the world, then to posterity. Only for one brief moment once in New York, did she discard her Mexican peasant dress, for the sophisticated chic of Fifth Avenue. But she soon tired of the pretentious capitalist "gringo-land" haute couture, going back to her native dress. In an age where to stand out in the crowd was simply not done - Frida with her brightly coloured dresses, masses of ribbons, flowers and pre-Columbian beads was quite a head turner wherever she went in New York - this in an age well before any sense of individuality was considered the norm - or even acceptable.

Her brief lover, Julien Levy, the New York Art dealer who exhibited Frida's work in his gallery for her famous solo New York Exhibition of 1938 described a visit with her to the Hanover Bank on Fifth Avenue. "Arriving inside the bank with her, I found we were surrounded by a flock of children who had followed us in, despite all the protests of the doorman. "Where is the circus?, they were calling! "Fiesta would have been more accurate. Frida was dressed in full Mexican costume!"

But there was also one other reason for Frida's elaborate Mexican dresses. She had a lifelong shame of her wasted leg, the result of polio which she had contracted at the age of six. Although she never hid the fact of her deformed leg in her Art, which was after all about her own suffering more often than not, she nonetheless was always careful to hide it in public. "She was beautiful and picturesque", Levy explained, "but sadly she did not wear bouffant native dress solely for effect". "I must have full skirts", Frida told him, "now that my sick leg is so ugly".

The agent dexamethasone is an exceptionally useful drug for the suppression of all forms of inflammation - but unfortunately it can, like Frida Kahlo's Tehuana dresses, really only ever mask a malady, rather than actually cure it!

Frida Kahlo with Diego Rivera - May Day Rally Mexico City, May 1929 (Photograph by Tina Modotti).

DEXAMETHASONE

Introduction

Dexamethasone is a synthetic corticosteroid, that is **highly potent** in its glucocorticoid effects.

It is **25 - 50** times more potent than hydrocortisone in its glucocorticoid effect, while having *minimal* mineralocorticoid effects.

Dexamethasone has strong **anti-inflammatory** and **immunosuppressant activity.**

Its principle uses in the Emergency Department include:

1. **Bronchospasm**
2. **Allergic reactions including anaphylaxis**
3. **Inflammatory edema of the CNS**
    - **Tumours of the brain and spinal chord**
4. **Croup**
5. **Bacterial meningitis (to reduce the incidence of sensorineural deafness).**
6. **Acute epiglottitis**
7. **SVC obstruction**

It is on the WHO List of Essential Medicines, the most important medications needed in a basic health system.

History

Cortisone was first identified by the American chemists **Edward Calvin Kendall** (1886 - 1972) and **Harold L. Mason** while researching at the Mayo Clinic.

Kendall was awarded the 1950 Nobel Prize for Physiology or Medicine along with **Philip S. Hench** and **Tadeus Reichstein** for the discovery of adrenal cortex hormones as well as their structures and functions.

Harold. Mason's contributions to the crystallization and characterization of cortisol have generally been forgotten outside of the Mayo Clinic.

Chemistry

Dexamethasone is a derivative of methylprednisolone.

Physiology

The principle physiological roles of the corticosteroids are widespread and complex, but essentially fall into three groups:

1. Enhancing the effects of circulating catecholamines

2. Metabolic effects

3. Global suppression of anti-inflammatory and immune responses

At the highest level, the corticosteroids are said to enable and enhance the complex cardiovascular and metabolic "fight or flight" survival responses of the organism to the presence of "stressors" (e.g. trauma, infection, surgery).

Classification

Naturally occurring adrenocortical steroids:

The **naturally occurring** adrenocortical steroids are:

- **Hydrocortisone (or cortisol)**
- **Cortisone**

These have both anti-inflammatory (glucocorticoid) and salt retaining properties, ie mineralocorticoid properties).

They are principally used as replacement therapy in adrenocortical deficiency states.

Synthetic corticosteroids:

The **synthetic** corticosteroid compounds include:

- **Prednisolone**
- **Prednisone**
- **Dexamethasone**
- **Methylprednisolone**
- **Fludrocortisone**

These are mainly used for their anti-inflammatory properties.

Classification according to predominant activity:

1. Synthetic agents with *marked glucocorticoid* activity and an *absence* of significant salt-retaining activity:

    - **Dexamethasone**
    - **Prednisolone**
    - **Methylprednisolone**
    - **Betamethasone**

    These drugs are primarily used for their potent anti-inflammatory effects.

2. Synthetic agents with predominant mineralocorticoid activity:

    - **Fludrocortisone**

    It is used as a mineralocorticoid replacement for patients with primary adrenal insufficiency and as a treatment for orthostatic hypotension.

3. Agents with a combination of both *glucocorticoid* activity and mineralocorticoid activity.

    - **Hydrocortisone**

Preparations

Dexamethasone as:

Tablets:

- 500 micrograms, 4 mgs.

Ampoules: (as dexamethasone sodium phosphate):

- 4 mg / 1 ml ampoule, 8 mg / 2 ml ampoule.

Mechanism of Action

The corticosteroids **regulate gene expression**.

When a corticosteroid enters a cell, it combines with corticosteroid receptors in the cytoplasm.

This drug-receptor complex enters the nucleus where it controls synthesis of protein, including enzymes that regulate cell activity such as inflammation.

Pharmacodynamics

Effects of the corticosteroids in general include:

1. Glucocorticoid effects including:
    - **Enhancing the vascular sensitivity to circulating catecholamines.**
    - Metabolic effects:
        - ♥ Gluconeogenesis:
            - ♥♥ This is the generation of glucose from *non-carbohydrate* carbon substrates such as pyruvate, lactate, glycerol, and glucogenic amino acids.
        - ♥ Proteolysis
        - ♥ Lipolysis
        - ♥ All corticosteroids can increase calcium excretion
    - Suppression of inflammation and immune responses.
2. Mineralocorticoid effects including:
    - Hypertension
    - Sodium and water retention
    - Potassium loss.

The corticosteroids as a group may have predominantly glucocorticoid effects (e.g. dexamethasone), mineralocorticoid effects (fludrocortisone), or a combination of both (e.g. **hydrocortisone**).

Note that *systemic effects* may result from all of oral, intramuscular, intravenous, inhaled, intra-articular and topical administration.

Pharmacokinetics

Absorption:

- Dexamethasone can be given IV and IM and orally

  Dexamethasone phosphate (as sodium) is rapidly absorbed following IM administration, however the IV route is generally preferred to the IV route.

Distribution:

- Synthetic corticosteroids such as dexamethasone are less extensively protein bound and more slowly metabolised than hydrocortisone.

  Only small amounts of dexamethasone are protein bound.

- Dexamethasone penetrates into tissue fluids and cerebrospinal fluids.

- Dexamethasone can cross the human placenta.

- Dexamethasone is excreted into human breast milk.

Metabolism and excretion:

- Metabolism occurs in most tissues, but primarily in the liver.

  The inactive metabolites are excreted in the urine, mainly as glucuronides and sulfates but also as unconjugated metabolites.

  Small amounts of unchanged drug are also excreted in the urine.

- Elimination half-life is around 1.88 - 2.23 hours

Indications

The principle indications for dexamethasone in the ED include:

1. **Bronchospasm**

2. **Allergic reactions including anaphylaxis**

3. **Inflammatory edema of the CNS**

   - **Tumours of the brain and spinal chord**

4. **Croup in children**

5. **Bacterial meningitis (to reduce the incidence of sensorineural deafness).**

6. **Acute epiglottitis.**

7. **SVC obstruction**

Other indications include:

8. **Prophylaxis and treatment for altitude sickness.**

9. **Nausea and vomiting,** (particularly postoperative or chemotherapy-induced).

10. **Dexamethasone may be given to women at risk of delivering prematurely to promote maturation of the fetus' lungs.**

11. Adjunctive treatment for some malignant diseases:

    - Multiple myeloma / lymphoma / some leukaemias

Contra-indications/precautions

These include:

1. Infections (caution):

   Immunosuppression can increase the risk and severity of infection and so caution is advised when prescribing hydrocortisone in a patient with active or latent infection.

   Some conditions require *both* corticosteroid and control of infection, (e.g. infective exacerbation of COPD or asthma) and this is usually safe so long as the infection can be effectively treated at the same time.

   In general terms, the decision to start or continue corticosteroids in a patient with infection depends on a number of factors including:

   - The type of infection:
     - ♥ Including whether active or latent
     - ♥ **Systemic fungal** infections generally contraindicate the use of corticosteroids.
   - Its severity
   - Whether the infection can be treated or controlled at the same time.
   - The specific indication for hydrocortisone.

2. Live vaccines:

   - Administration of live or live attenuated vaccines is contraindicated in patients receiving **immunosuppressive doses** of corticosteroids.

     Killed or **inactivated** vaccines may be administered to patients receiving immunosuppressive doses of corticosteroids, however the response to such vaccines may be diminished.

3. Known hypersensitivity including to the exipients such as to sulfites.

4. Intra-articular injection (corticosteroids in general):

    - Corticosteroids are contraindicated in patients with infective arthritis, skin or soft tissue infections near joint (risk of introducing bacteria into joint) or a prosthetic joint.

*The following **precautions** do not apply when used at **physiological** doses for **adrenal insufficiency**:* [2]

5. Latent TB:

    - May be reactivated; consider treatment with isoniazid.

6. Peptic ulcer disease:

    - Corticosteroids may increase the risk of peptic ulcers.

7. Diabetes:

    - Corticosteroids (including intra-articular injections) may cause hyperglycaemia and worsen diabetes control.

8. Hypertension/ heart failure:

    - May be worsened due to sodium and water retention (mineralocorticoid effect).

9. Psychiatric disorders:

    - May be exacerbated.

10. Glaucoma:

    - Intraocular pressure may increase.

11. Osteoporosis:

    - Long-term corticosteroid use increases the risk of osteoporotic fractures and accelerates bone loss.

12. Myasthenia gravis:

    - Increased muscle weakness may occur during the first few weeks of treatment with corticosteroids; (seek specialist advice).

Pregnancy

Dexamethasone is a category A drug with respect to pregnancy.

Category A drugs are those drugs which have been taken by a large number of pregnant women and women of childbearing age without any proven increase in the frequency of malformations or other direct or indirect harmful effects on the fetus having been observed.

Note that **hydrocortisone, prednisolone, prednisone** and **methylprednisolone** are the preferred agents for maternal disorders as placental transfer is limited, while **betamethasone** and **dexamethasone** are the preferred agents for fetal disorders as placental transfer is greater. [2]

Most studies have shown no increased risk of major congenital malformations following maternal use of corticosteroids. Early reports have suggested an association between corticosteroid use and a risk of cleft lip and palate . However, more recent data has shown no increased risk of orofacial clefts or preterm delivery.

One study has found that when pregnant women are exposed to potent or very potent corticosteroids, infants were at risk of intrauterine growth restriction.

In addition, prolonged use of systemic corticosteroids has been associated with maternal adverse effects, including hypertension, glucose intolerance, opportunistic infections, and ocular and bone side effects.

If possible, consider an alternative medicine to systemic corticosteroids during the first trimester.

Dexamethasone has been used as an antenatal corticosteroid for fetal lung immaturity when preterm delivery is anticipated.

Dexamethasone is safe to use in the second and third trimester if it is the medicine of choice. However, follow-up and monitoring of both maternal and fetal wellbeing by a multidisciplinary team is recommended.

Breast feeding

Published reports describing the use of dexamethasone during breastfeeding have not been located.

If dexamethasone is the treatment of choice, use the lowest effective dose for the shortest duration possible and observe the breastfed infant for potential adverse effects such as diarrhoea, vomiting and irritability.

Adverse Effects

Adverse effects are an inevitable result of **systemic** corticosteroid treatment if the **dose and/ or duration** of treatment are sufficient, because most are dose-related biological effects of the hormone.

Short courses of high dose systemic treatment cause fewer adverse effects than prolonged courses of lower doses.

The **longer - term** adverse effects of corticosteroids **in general** include:

Adverse glucocorticoid effects:

1. Adrenal suppression

   Manifests with longer term exogenous administration when:

   - Therapy is **abruptly ceased**

   Or

   - When a **stressor occurs**; due to secondary adrenocortical and pituitary unresponsiveness, particularly in trauma, surgery, infection or illness

   Adrenal hypofucntion can lead to Shock/ increased risk of shock states.

   The hypothalamic-pituitary-adrenal axis is suppressed by glucocorticoid therapy.

   The dose, duration of treatment and individual patient characteristics affect the onset and extent of this effect.

   Treatment with **prednisolone** at doses **greater than 10 mg** (or equivalent dose of glucocorticoid) **daily for more than three weeks** can be considered sufficient to cause clinically significant adrenal suppression that requires glucocorticoid replacement at a time of intercurrent illness, trauma or surgical stress. [1]

   Therefore, in this situation, tapering of the glucocorticoid dose is required to avoid both adrenal insufficiency and the rebound in symptoms that may occur with sudden cessation.

   After long-term use (more than 2 weeks), dose reduction must be slow to enable the hypothalamic-adrenal feedback system to re-equilibrate.

2. Immunosuppression:

   - Increased risk of infection.

   - Infections are more common in people treated with supraphysiological doses of corticosteroids, due to immunosuppressive effects.

   - **Corticosteroids may also mask the early symptoms and signs (by suppressing the inflammatory repose) of infection, resulting in later diagnosis, delay in treatment, and more severe clinical consequences.**

   - Reactivation of *Mycobacterium tuberculosis* infection occurs with such frequency that before commencing immunosuppressant treatment with

corticosteroids, screening for active or latent tuberculosis (TB) should be considered.

- Prophylaxis for *Pneumocystis jiroveci (carinii)* infection should be considered in patients who are at risk and taking high dose oral corticosteroids long term.

3. Metabolic effects:

   - Hyperglycaemia:

     ♥ Hyperglycaemia may accompany treatment with corticosteroids, particularly when higher doses are used, and blood glucose concentrations may require monitoring.

   - Hypertriglyceridaemia

   - Weight gain/ redistribution of fat centripetally, leading to the development of a "Cushingoid" state.

   - All corticosteroids increase calcium excretion

4. Dermatological effects:

   - Skin atrophy leading to thin fragile skin.

   - Purpura/ ecchymoses

   - **Poor wound healing**

   - Striae

   - Hirsutism

5. Gastrointestinal effects:

   - Dyspepsia

   - Peptic ulceration

   - Gastrointestinal bleeding

6. Myopathy:

   - Steroid myopathy with muscle weakness/ loss of muscle mass.

   - Tendon rupture can also occur, particularly of the Achilles tendon

7. Bone effects:

- Osteoporosis:
    - ♥ **Bone density loss** leading to osteoporosis is a risk for patients on continuous (> one month) corticosteroid therapy in doses greater than the equivalent of prednisolone **5 to 7.5 mg per day** or who are receiving frequent pulses of corticosteroid.
    - ♥ It can occur in men as well as women.
    - ♥ The risk of osteoporosis becomes greater at higher glucocorticoid doses.
    - ♥ Loss of bone mineral density occurs rapidly after corticosteroids are commenced and may exacerbate the osteoporosis associated with some rheumatological diseases, such as ankylosing spondylitis, and inflammatory bowel disease.
- Avascular (ischaemic) necrosis:
    - ♥ Most typically of the of the proximal femoral and humeral head
    - ♥ It is infrequent
    - ♥ It is idiosyncratic (i.e unpredictable).
    - ♥ It occurs more commonly following exposure to doses in excess of **20 mg** per day prednisolone (or equivalent dose of other corticosteroid).
    - ♥ The time between corticosteroid exposure and the development of avascular necrosis is variable, and can be up to *many years*, which makes diagnosis difficult.
    - ♥ Avascular necrosis should be considered in the differential diagnosis of hip and groin pain especially in patients who have been on high-dose and/or long-term corticosteroids at any time.
    - ♥ Both the pathogenesis and treatment of this condition remain controversial.

8. Ocular effects:
    - Increased intraocular pressure
    - Cataracts
9. Growth retardation in children:

- Chronic use of corticosteroids (at pharmacological doses) may retard growth in children.
- Follow growth and development carefully
- Catch-up growth may occur after corticosteroid withdrawal.

10. Menstrual irregularities:
    - Principally amenorrhoea
11. Psychiatric disturbances:
    - Euphoria
    - Depression
    - Paranoid psychosis

    Note that preexisting emotional instability or psychotic tendencies may also be *aggravated* by corticosteroids.

Adverse mineralocorticoid effects:

1. Sodium retaining effects:
    - Oedema
    - Hypertension
2. Hypokalaemic alkalosis.

Other effects:

1. Perineal / genital burning after IV bolus dexamethasone injection. [6]
    - This unusual reaction is not common, but well documented.

      It occurs much more commonly in females.

      The effect is almost immediate, but is only short lasting (several minutes).

      It may be mitigated in those susceptible by diluting the dose in saline and administering slowly.

      The reaction has also been reported for IV bolus, hydrocortisone.

      The mechanism of the reaction is unknown.

Note that most of the adverse effects relate to long term treatment and do not preclude *short* term use.

Corticosteroids applied topically can also cause adverse effects

Dosing

Exact dosing and the duration of dosing depends on the condition being treated as well as the severity of the condition and illness.

See latest Therapeutic Guidelines for full prescribing details.

In general terms:

Bronchospasm

Hydrocortisone is more commonly used

- For dexamethasone: 4 - 8 mg IV 4 - 6 - 8 - 12 hourly.

Allergic reactions

Hydrocortisone is more commonly used

- For dexamethasone: 4 - 8 mg IV 4 - 6 - 8 - 12 hourly.

Space occupying lesions of the CNS with inflammatory edema

In the ED:

- **16 -20 mg IV as a stat dose in the ED**

For ongoing therapy: [2]

- *Adult*, Oral/IV/IM, initially **4- 16 mg** daily in **2 - 4 divided** doses depending on severity of symptoms (higher doses have been used); gradually withdraw treatment or reduce to lowest effective dose.

SVC Obstruction: [1]

- **Dexamethasone 16 mg orally, SC or IV, as a single dose, followed by 16 mg orally, SC or IV, daily (in the morning to avoid sleep disturbance at night).** [1]

- **If no benefit is obtained within 5 days, it should be ceased.** [1]

Meningitis [1]

Adults and children 2 months or older, use:

- **Dexamethasone 10 mg (child: 0.15 mg/kg up to 10 mg) IV,**

  starting before or with the first dose of antibiotic,

  then 6-hourly for 4 days

Acute Epiglottitis: [1]

- **Dexamethasone 10 mg (child: 0.15 mg/kg up to 10 mg) IV, as a single dose; repeat at 24 hours if required.**

Croup: [3]

Mild - Moderate croup: **A single dose of oral Dexamethasone 0.15 mg/kg.**

Severe croup: **Give 0.6 mg/kg (max 12mg) IM/IV dexamethasone**

Altitude Illness:

Specifically for the treatment of severe **AMS** or **HACE** (it is not useful for HAPE)

- Give in a treatment dose of **4 mg 6 hourly** - this can be oral, IM or IV depending on the exact clinical scenario and availability

Chemotherapy-induced nausea and vomiting:

The dose depends on emetogenicity of chemotherapy (refer to treatment protocols).

The following doses may be used.

Adult, oral / IV 4 - 20 mg 30 minutes before chemotherapy.

If delayed emesis is anticipated, follow with 8 mg orally once or twice daily for 2 - 4 days.

Appendix 1

Approximate relative potencies and half-lives of the glucocorticoids:

Glucocorticoid	Relative glucocorticoid potency	Equivalent dose for glucocorticoid effect	Estimated biological half-life
Hydrocortisone	1	20 mg	8 to 12 hours
Cortisone acetate	0.8	25 mg	8 to 12 hours
Dexamethasone	25 to 50	400 to 800 micrograms	36 to 54 hours
Prednis(ol)one	4	5 mg	18 to 36 hours
Methylprednisolone	5	4 mg	12 to 36 hours

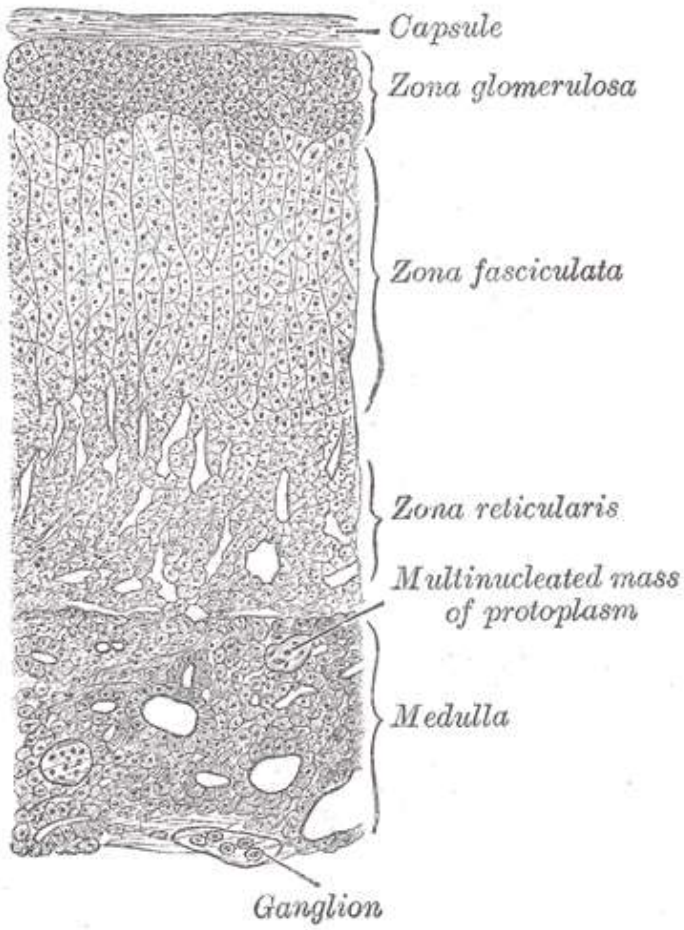
Appendix 2

Relative potencies of the topical preparations of the corticosteroids:

Mild	
Desonide	0.05%
Hydrocortisone	0.5%, 1%
Hydrocortisone acetate	0.5%, 1%
Moderate	
Betamethasone valerate	0.02%, 0.05%
Clobetasone butyrate	0.05%
Methylprednisolone aceponate	0.1%
Triamcinolone acetonide	0.02%
Potent	
Betamethasone dipropionate	0.05%
Betamethasone valerate	0.1%
Mometasone furoate	0.1%
Triamcinolone acetonide	0.1%
Very potent	
Betamethasone dipropionate	0.05% in optimised vehicle
Clobetasol propionate	0.05%

Appendix 3

Physiology of the Adrenal Gland:



Cross sectional anatomy of the adrenal gland, (Gray's Anatomy 1918).

The Adrenal Cortex:

This consists of **3 zones**:

- Zona glomerulosa:
  - ♥ This layer is the main site for production of the **mineralocorticoid** hormone, **aldosterone.**
- Zona fasciculata:
  - ♥ This layer is the main site for production of the **glucocorticoids** (mainly **cortisol**).
- Zona reticularis:

- ♥ This layer is the main site for production of **androgens**; mainly dehydroepiandrosterone (DHEA), DHEA sulfate (DHEA-S), and androstenedione (the precursor to testosterone) in humans.

The Adrenal Medulla:

- The chromaffin cells of the medulla are the body's main source of the circulating catecholamines; **adrenaline** (80 %) and **noradrenaline** (20 %).

Frida Kahlo aged nineteen, (far left) and family relatives, Coyoacan, 1926. (Photographed by Guillermo Kahlo). Frida at this age liked to dress as a male.

Frida Kahlo, New York City, 1946, photograph by Nickolas Muray

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Reviewed 6 July 2019.