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Corticosteroids: implications for nursing practice

Aims and intended learning outcomes

Several corticosteroids, such as prednisolone, dexamethasone and hydrocortisone/cortisol, are prescribed to suppress inflammation in a range of conditions. This article aims to update knowledge and encourage a review of practice. The emphasis will be on monitoring the adverse effects of medication and on patient education. After reading this article you should be able to:

- Explain the rationale for the prescription of topical and systemic corticosteroids.
- Detect and minimise the adverse effects associated with long-term administration of oral and topical corticosteroids.
- Discuss the importance of adherence to prescribed corticosteroid regimens.
- Recognise the drugs that interact with corticosteroids.
- Consider strategies to optimise medication management of corticosteroids.

Introduction

Box 1 provides a glossary of some of the terms used in this article. Corticosteroids comprise the glucocorticoid and mineralocorticoid hormones synthesised in the adrenal cortex and the drugs derived from them. Commonly prescribed corticosteroids are listed in Table 1.

Corticosteroids have revolutionised the management of several disabling conditions and have saved many lives. However, they feature prominently in reviews of adverse drug reactions and suboptimal prescribing (Chyka 2000). In the UK, it is estimated that more than 250,000 people are taking continuous oral corticosteroids (Walsh et al 1996), and more than half of all children being treated for asthma in primary care are exposed to chronic high doses of corticosteroids via a combination of inhaler and intra-nasal devices (Ekins-Daukes et al 2002).

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Corticosteroids suppress the immune system, inflammatory processes and hypersensitivity responses, and provide symptom relief in many acute and chronic conditions, including asthma and ulcerative colitis (Table 2). The doses prescribed for these conditions are often higher than in replacement regimens. At these 'pharmacological' doses, adverse effects are very likely and careful monitoring is required.

How the body handles corticosteroids

Corticosteroids are lipid soluble and rapidly cross cell membranes. They can therefore be administered by many routes (Table 3). Topical administration reduces,
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Box 1. Glossary

Addison's disease A condition due to deficiency of hormones of the adrenal cortex. This arises when the adrenal cortex is destroyed by autoimmune processes or infection (eg, tuberculat, HIV).

Congenital adrenal hyperplasia A group of congenital disorders in which the synthesis of adrenal steroid hormones is dysregulated. Over-production of androgens may occur.

Cortisol The main glucocorticoid hormone secreted by the adrenal glands. Hydrocortisone is the synthetic form.

Dyslipidemia Abnormal lipid profile, usually an excess of cholesterol, fatty acids or triglycerides. This is a risk factor for cardiovascular disease.

Endogenous Produced within the body, rather than administered as a medication.

Fibroblasts Cells that synthesise collagen and the ground substance of connective tissues.

Hirsutism Abnormally high concentration of red blood cells (haemoglobin concentration >20g/dl)

Hypokalaemia Abnormally low potassium concentration in the blood (<3.5mmol/l).

Macrophages Large white blood cells that can ingest debris and bacteria. Essential for immunity.

Necrosis A bone disorder in which blood supply to the bone decreases, resulting in bone death.

Parathyroid hormone A hormone that increases plasma calcium concentration by absorbing calcium from bone, gut and urine.

Polyarthaemia Abnormally high concentration of red blood cells (haemoglobin concentration >20g/dl).

S frauds Skin defects that look like red lines, associated with rapid growth, for example, during pregnancy or steroid medication.

Systemic Pertaining to the whole body. Systemic administration allows the drug to pass to the entire body. It includes the oral, intravenous and intramuscular routes.

Telangiectases Red spots on the skin, which form as capillaries or small arteries dilate.

Thrombocytopenia Abnormally low number of platelets in the blood (<130 x 10^9/l).

Ulcerative colitis A chronic, episodic inflammatory disease of the large intestine and rectum.

TIME OUT 1

Reflect on the last time you cared for a patient receiving intramuscular corticosteroid injections. Did you have a system of recording injection sites? How could this be useful?

Table 1. Classification of corticosteroids

<table>
<thead>
<tr>
<th>Oral corticosteroids</th>
<th>Glucocorticoid effect (dose equivalent)</th>
<th>Mineralocorticoid effect</th>
<th>Duration of effect (hours)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Short-acting</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cortisone</td>
<td>20mg</td>
<td>+++</td>
<td>8-12</td>
</tr>
<tr>
<td>Hydrocortisone</td>
<td>20mg</td>
<td>+++</td>
<td>8-12</td>
</tr>
<tr>
<td>Intermediate-acting</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prednisone</td>
<td>5mg</td>
<td>++</td>
<td>18-36</td>
</tr>
<tr>
<td>Triamcinolone</td>
<td>4mg</td>
<td>-</td>
<td>18-36</td>
</tr>
<tr>
<td>Methylprednisolone</td>
<td>4mg</td>
<td>-</td>
<td>24-36</td>
</tr>
<tr>
<td>Prednisolone</td>
<td></td>
<td>+++</td>
<td>36-54</td>
</tr>
<tr>
<td>Long-acting</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dexamethasone</td>
<td>750µg</td>
<td>-</td>
<td>36-54</td>
</tr>
<tr>
<td>Betamethasone</td>
<td>750µg</td>
<td>-</td>
<td>36-54</td>
</tr>
</tbody>
</table>

Inhaled corticosteroids

<table>
<thead>
<tr>
<th>(dose equivalent for adverse effects)</th>
<th>Time (hours) in circulation*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Budesonide</td>
<td>6.9</td>
</tr>
<tr>
<td>Fluticasone</td>
<td>43.2</td>
</tr>
<tr>
<td>Mometasone</td>
<td>500µg</td>
</tr>
</tbody>
</table>

*Calculated as three times the terminal half-life. ++++ Marked effect. ** Moderate effect. - No effect.

Inhaled corticosteroids The type of inhaler device used may alter drug absorption by up to 500 per cent (Wilson et al 1999). Therefore, a change in inhaler, including switching from chlorofluorocarbon propellants to the more environmentally friendly hydrofluorokanes, requires careful patient monitoring. Spacers and dry powder devices have higher rates of drug delivery than other devices. However, patients with reduced inspiratory function, young children and people with severe lung disease may be unable to activate dry powder inhalers (Cave et al 1999).

Sixty per cent of children using corticosteroid inhalers report at least one local adverse effect, most commonly cough on inhalation, hoarseness, speech difficulty or oral candidiasis (Dubus et al 2001). Fluticasone propionate has a greater potential for local adverse effects (Adams et al 2002) and accumulation (Cave et al 1999) than other inhaled corticosteroids. Systemic adverse effects are unlikely with doses equivalent to beclomethasone below 800µg in adults and 400µg in children (Lipworth and Wilson 2002). Rinsing the mouth and expectorating reduces the quantity of beclomethasone (but not budesonide or fluticasone) absorbed into the general circulation (Toogood 1998). Patients may need reminding that inhaled corticosteroids improve asthma symptoms only after three to seven days, but does not abolish systemic absorption and the associated adverse effects.

Systemic administration Orally administered corticosteroids pass to the liver, where they are extensively metabolised into active and inactive metabolites, which then pass into the general circulation. When drugs are administered by other routes they bypass the liver and are not metabolised, which may enhance their potency. In medical emergencies, high plasma concentrations of corticosteroids are rapidly achieved by intravenous administration. Intramuscular injection carries a risk of muscle atrophy, which is minimised by administration deep into the gluteal muscle (McKenny and Salerno 1998). The deltoid muscle is too small for steroid injections (Fowler 1998). Intramuscular injections are inadvisable for children, older people, patients with low muscle mass and those with purpura due to thrombocytopenia. Care of the injection site is essential, as steroid injections are associated with swelling, tingling, numbness, pain and sterile abscess (McKenny and Salerno 1998). Each injection site should be used only once and documented, as repeated injections may cause scarring, induration, necrosis and tissue atrophy (Fowler 1998).

TIME OUT 1

Reflect on the last time you cared for a patient receiving intramuscular corticosteroid injections. Did you have a system of recording injection sites? How could this be useful?
Topical corticosteroids

Table 2. Clinical indications for use of corticosteroids

<table>
<thead>
<tr>
<th>Condition</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Systemic therapy</strong></td>
<td></td>
</tr>
<tr>
<td>Replacement therapy</td>
<td>In these conditions, replacement therapy is prescribed as a combination of 20-30mg hydrocortisone plus 50-300µg fludrocortisone daily (BNF 2002)</td>
</tr>
<tr>
<td>Suppressing the immune system</td>
<td>Hydrocortisone is administered intravenously</td>
</tr>
<tr>
<td>Autoimmune diseases</td>
<td>Over-activity of the immune system is suppressed by relatively high doses, calculated in relation to body weight</td>
</tr>
<tr>
<td>Managing inflammation</td>
<td>Prompt administration can save sight</td>
</tr>
<tr>
<td>Rheumatology</td>
<td>Betamethasone or dexamethasone reduce swelling</td>
</tr>
<tr>
<td>Palliative care and oncology</td>
<td>Corticosteroids have anti-tumour activity in these cancers</td>
</tr>
<tr>
<td>Respiratory distress syndrome of the newborn</td>
<td>Corticosteroids are indicated when preterm delivery is likely</td>
</tr>
<tr>
<td>Topical therapy</td>
<td></td>
</tr>
<tr>
<td>Atopia</td>
<td>Inhaled steroids provide long-term control and prophylaxis. They reduce inflammation, oedema and mucus secretion in the airways. Systemic therapy is indicated in emergencies</td>
</tr>
<tr>
<td>May fever, allergic rhinitis</td>
<td>Inflammation and mucus secretion are suppressed by intranasal applications. Systemic absorption is greater with nose drops than with nasal sprays</td>
</tr>
<tr>
<td>Inflammatory skin conditions ‘drug rashes’, contact dermatitis, psoriasis, nappy rash, eczema, pemphigus (only with specialist supervision)</td>
<td>Hydrocortisone is a useful topical corticosteroid, due to its relatively moderate anti-inflammatory activity</td>
</tr>
<tr>
<td>Ulcerative inflammation (under specialist supervision)</td>
<td>Corticosteroids reduce inflammation in the mucosal skin</td>
</tr>
<tr>
<td>Oral ulceration, refractory to other measures</td>
<td>Hydrocortisone lozenges or triamcinolone dental paste may be useful if no local infection is present</td>
</tr>
</tbody>
</table>

BNF 2002, RCOG 2002

... days, and are useful only as prophylaxis (BNF 2002).

**Topical corticosteroids** These are applied to treat inflammatory skin conditions and are available in many forms, such as lotions, creams, ointments and solutions. For example, 1% and 0.1% hydrocortisone creams are used to relieve symptoms of pruritus, nappy rash and eczema. Some preparations contain other active ingredients or additives, which may induce hypersensitivity responses, for example antimicrobials and wool fat.

Systemic absorption of topical corticosteroids is increased if the skin is thin, broken or inflamed, and if an occlusive dressing is applied. Tight fitting nappies act as exclusive dressings and should not be placed over topical corticosteroids. Rapid absorption occurs in babies and children, and through mucous membranes or eyelids. Systemic adverse effects are likely when potent corticosteroids (for example, betamethasone 0.1%, clobetasol propionate 0.05%) are applied to extensive areas of skin for prolonged periods (ABN 2000).

Systemic absorption following rectal administration can be erratic. For example, if the rectum is inflamed, absorption increases from 20 per cent to 50 per cent (McKernan and Salerno 1998).

**Distribution and elimination** Corticosteroids are 90 per cent bound to plasma proteins. Liver enzymes metabolise corticosteroids into soluble forms, which can then be excreted by the kidneys. Adverse effects are more likely if the activity of liver enzymes is reduced by prolonged use of steroid medication, congenital adrenal hyperplasia, or by severe stress, eg surgery.
### Table 3. Possible local adverse reactions with corticosteroids

<table>
<thead>
<tr>
<th>Route of application</th>
<th>Local adverse effects</th>
<th>Suggested management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral</td>
<td>Irritation to lining of upper gastrointestinal tract</td>
<td>Take medication with food or milk and a full glass of water. Remain in an upright position for 30 minutes</td>
</tr>
<tr>
<td>Intranasal injection</td>
<td>Injection of hydrocortisone as sodium phosphate ester is often accompanied by pain and a ‘tingling’ sensation in the perinasal area</td>
<td>Inform patients</td>
</tr>
<tr>
<td>Intramuscular injection</td>
<td>Muscle atrophy at the injection site. Local reactions (see text)</td>
<td>Use each site only once and document</td>
</tr>
<tr>
<td>Inhaler devices</td>
<td>Oral candidiasis (thrush)</td>
<td>Oral, laryngeal and pharyngeal irritation may cause cough or hoarseness. Special dentists may decrease oral candidiasis, but increase cough (Dohus et al 2001). Cough can be minimised by pre-treatment with an inhaled beta₂-agonist (BNF 2002)</td>
</tr>
<tr>
<td>Intranasal applications</td>
<td>Headache, nausea, epistaxis, rebound congestion, perforation of the nasal septum, loss of sense of smell, nose bleeds, due to atrophy of the lining of the nose.</td>
<td>Restrict administration to recommended doses, particularly if patients are using beclomethasone and budesonide nasal sprays without prescription</td>
</tr>
<tr>
<td>Skin</td>
<td>Spread of infection, acne, dermal atrophy, depigmentation, perioral dermatitis, telangiectases and striae</td>
<td>Avoid application to the face and broken skin without specialist advice. Adverse effects can be fully assessed (Helms 2000), and a maximum of five to seven days</td>
</tr>
<tr>
<td>Intrarticular injections</td>
<td>Osteonecrosis, tendon rupture, infection</td>
<td>Corticosteroid injections into the Achillestendon are contraindicated because of the risk of tendon rupture (BNF 2002). Consult specialist regarding exercises and avoidance of weight-bearing</td>
</tr>
<tr>
<td>Rectal administration</td>
<td>Local pain, burning, rectal bleeding, delayed healing</td>
<td>Avoid if patient has bowel obstruction, recent gastrointestinal surgery, bleeding tendencies or infection</td>
</tr>
<tr>
<td>Ophthalmic application</td>
<td>Infections, thinning or perforation of cornea and sclera, glaucoma and cataracts</td>
<td>Specialist supervision recommended (BNF 2002)</td>
</tr>
<tr>
<td>Ear drops</td>
<td>Sensitivity reactions</td>
<td>Avoid prolonged use</td>
</tr>
</tbody>
</table>

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**Actions of corticosteroids**

Without corticosteroids, the body cannot resist the slightest stress or infection, and the chemical mediators released in the inflammatory response will cause a catastrophic fall in blood pressure (Scheinman and Parker 2001). This is a constant danger in Addison’s disease and adrenal suppression (see below). Corticosteroids control the immune response, metabolic pathways, fluid and electrolyte balance, the cardiovascular and central nervous systems. They have two groups of actions:

- **Glucocorticoid effects**, including metabolic changes and anti-inflammatory actions.
- **Mineralocorticoid effects** – mainly retention of salt and water, together with loss of potassium and hydrogen ions.

Some drugs, such as dexamethasone, have predominantly glucocorticoid actions, whereas others are predominantly mineralocorticoid. Many drugs, such as hydrocortisone, have both (Table 1).

Corticosteroids act on intracellular receptors, thus modulating gene expression and protein synthesis in target tissues. Mineralocorticoids are essential for the maintenance of fluid and electrolyte balance. Glucocorticoids are important for Saei-Parsy et al (1999), Schimmer and Parker (2001):

- **Strengthening biological membranes by reducing capillary permeability; this maintains blood pressure and prevents oedema.**
- **Increasing responsiveness to vasoactive hormones, particularly noradrenaline (norepinephrine) and adrenaline (epinephrine) – corticosteroids are therefore useful in maintaining responsiveness to beta₂-agonists in asthma (Jordan and White 2001a).**
- **Coping with starvation.**
- **Maintaining stability of the central nervous system.**
- **Curtailing release of inflammatory mediators, such as prostaglandins, bradykinin and histamine.**
- **Suppressing activity of leucocytes (white blood cells).**

**Adverse effects of corticosteroids**

In view of the many potential adverse effects of corticosteroids (Figure 1), it is suggested that they offer little clinical benefit to children with mild symptoms of asthma and vaso-induced wheezing (Baxter-Jones et al 2002). They should be withheld until severity can be fully assessed (Helms 2000), and only prescribed if beta₂-agonists are needed for symptom relief more than once each day and a four- to six-week trial of cromoglycate prophylaxis fails, particularly in children aged under five years (BNF 2002). Although problems can arise with any dose or route of administration, the incidence and severity of adverse effects increases with cumulative dose and drug potency (Walsh et al 2001). Furfurose propionate may cause more adverse effects than either budesonide or budesonide, even when increased potency is considered (Lipworth and Wilson 2002). To summarise, corticosteroids may affect the:

- **Inflammatory and immune responses.**
- **Metabolic pathways.**
- **Skin.**
- **Gastrointestinal tract.**
- **Bones.**
- **Muscles.**
- **Cardiovascular system.**
- **Central nervous system.**
- **Eyes.**
Reproductive system.

Adrenal glands.

Inflammation and immunity Corticosteroids depress all manifestations of the inflammatory response – appropriate and inappropriate. Their actions include:

- Decreased production and release of inflammatory mediators, particularly prostaglandins, reducing pain and swelling (Jordan and White 2001b).
- Failure of neutrophils and macrophages to migrate to sites of tissue damage and microbial invasion, increasing the risk of infections.
- Failure to activate macrophages, increasing the risk of reactivation of dormant infections, particularly tuberculosis, amoebiasis and fungal or viral eye infections.
- Suppression of the immune response, increasing the severity of infections.
- Shrinkage of lymphoid tissue.
- Decreased proliferation of fibroblasts, collagen and new blood vessels, reducing healing and scar tissue formation.

Corticosteroids increase susceptibility to infections, including those associated with live vaccines (Box 2). Infections can spread rapidly, without signs and symptoms, because the natural indicators of infection are suppressed (Neal 1997). For example, a chest infection can rapidly develop into lobar pneumonia. Inhibition of fibroblasts impedes healing; wounds such as lower leg ulcers and pressure areas take longer to heal. Delayed or interrupted healing may be the only indication of wound infection. Where this occurs, specimens should be sent for microbiological investigation. Antimicrobial agents may be rendered ineffective, allowing resistant micro-organisms to emerge, therefore, appropriate antimicrobial therapy can only be determined by wound culture and sensitivity. Isolated reports suggest that retinol may restore wound healing capacity (Stockley 1999).

**TIME OUT 2**

Daily assessment of body temperature at 5-6pm could be useful to detect the first signs of infection, as pyrexia is most easily detected at this time of day. How could this be organised for patients in the community? How would you explain to patients the rationale for this?

**TIME OUT 3**

A high index of suspicion is needed if infections are to be detected in patients receiving pharmacological doses of corticosteroids and treated before they become serious. How would the knowledge that a patient was prescribed prednisolone 40mg/day influence your response to:

- An older patient who refuses food or starts finger-fingering his mouth?
- A patient who declines to visit the dentist, saying that she has never needed to before?
- An older patient who develops confusion over two to three days?
- A lower leg ulcer that is gradually enlarging?

Vulnerability to infection increases the risk of dental caries. Patients using oral corticosteroids were found to have fewer teeth, in worse condition than matched controls (Walsh et al 2001). Patients prescribed oral corticosteroids, either currently or in the past three months, should avoid contact with people who have measles, chickenpox or shingles. Because of the anti-inflammatory actions of corticosteroids, serious systemic illness may develop without the familiar rash. Where exposure has occurred, it is essential that medical staff are notified without delay, as the infection can be abated if immunoglobulins are administered within three days. Risks are lower with inhaled and topical corticosteroids (BNF 2002).

**Metabolic disturbances** Corticosteroids affect all
the body’s metabolic pathways. When the body is starving, endogenous corticosteroids increase the availability of glucose for the brain and heart, even at the cost of destroying other tissues, such as skin. Corticosteroids elevate plasma glucose concentrations by preventing uptake and use of glucose in most tissues and aiding formation of new glucose from amino acids and glycerol in the liver (gluconeogenesis). The resulting hyperglycaemia stimulates insulin production and reduces the body’s sensitivity to insulin, which is a risk factor for cardiovascular disease. Type 2 diabetes and even ketosis can develop. Patients receiving oral corticosteroids, or more than 800µg inhaled corticosteroids per day, should be monitored for hyperglycaemia. Elevated glucose concentrations in secretions such as saliva or sweat may promote the growth of Candida species (thrush). To supply amino acids for gluconeogenesis, corticosteroids increase protein breakdown and decrease protein synthesis in muscle, skin and bone. This delays wound healing and eventually causes skin and muscle atrophy. Protein breakdown reduces the collagen content of all tissues, making them more fragile. Skin, membranes, capillaries, tendons and bones are all vulnerable. Corticosteroids adversely affect plasma lipid concentrations, which should be monitored. The liver produces more lipids, while the breakdown of fat in adipose tissue is enhanced. This elevates the plasma concentration of lipids, particularly cholesterol, fatty acids and triglycerides; fat embolus is a rare complication. Alternate-day therapy may ameliorate these problems (Sholter and Armstrong 2000). Corticosteroids redistribute carbohydrate, fat and protein reserves. Eventually this causes central obesity, ‘moon face’ and thin limbs. This central distribution of body fat may be due to increased insulin concentrations and is associated with an increased risk of cardiovascular disease (Despres et al 2001). Corticosteroids increase appetite and promote weight gain, unless intake is monitored carefully. Appetite stimulation is often beneficial in palliative care (BNF 2002). Corticosteroids redistribute carbohydrate, fat and protein reserves. Eventually this causes central obesity, ‘moon face’ and thin limbs. This central distribution of body fat may be due to increased insulin concentrations and is associated with an increased risk of cardiovascular disease (Despres et al 2001). Corticosteroids increase appetite and promote weight gain, unless intake is monitored carefully. Appetite stimulation is often beneficial in palliative care (BNF 2002).

**Skin**

Corticosteroids can adversely affect the skin. As protein is broken down, the skin thins and becomes vulnerable to shearing forces. Local infection cannot be ‘walled off’ and may spread rapidly because fibroblasts are inhibited. Patients with plaster casts, and those on skin traction or on bed rest are particularly vulnerable. Capillary walls become more fragile, so bruises, petechiae and blood blisters form readily. Extra care is needed when undertaking nursing care such as moving and handling, cannulation and venepuncture. Increased susceptibility to infections and sweat can cause or exacerbate acne.

**Gastrointestinal tract**

Increased protein breakdown thins the gut lining. This predisposes to indigestion, ulcerative oesophagitis, peptic ulceration and gastric bleeding, particularly if non-steroidal anti-inflammatory drugs or alcohol are also taken. Stools should be observed for signs of gastrointestinal bleeding, and samples may be required for faecal occult blood testing. Nausea, vomiting, anorexia, abdominal distension and pancreatitis are less common (Karch 2000). As nausea is also a symptom of adrenal suppression, it is important that this is investigated, with a venous blood sample taken for electrolytes and glucose.

**Bone**

Corticosteroids can adversely affect bone growth and strength. Bone mineral density decreases as the cumulative dose of inhaled corticosteroids increases (Wong et al 2000). Even at the lowest doses, corticosteroids alter bone metabolism, inhibit osteoblasts and upset the balance between bone formation and reabsorption. Corticosteroids also reduce absorption of calcium from the gut, and increase its excretion from the kidneys. The resulting hypocalcaemia increases parathyroid hormone secretion, which exacerbates osteoporosis and bone loss (Saebl-Pang et al 1999). Some bone loss reverses on discontinuation (Cave et al 1999). Corticosteroid users are vulnerable to fractures in vertebrae and long bones. In a UK primary care case-control study, the risk of hip fracture increased six fold with oral corticosteroids (n=1,101) (Walsh et al 2001); it doubled in a US survey of post-menopausal women (n=8,068) (Baltzan et al 1999). Risk of vertebral fracture increased ten fold (Walsh et al 2001). For people using corticosteroids, there is little evidence that bisphosphonates either strengthen the femoral neck or reduce the incidence of fractures (Blair et al 2002). However, strategies to minimise the risk of steroid-induced osteoporosis include:...
Monitoring risk factors for osteoporosis. These include high doses, prolonged therapy, previous fractures, hypogonadism, age, low calcium intake and family history of osteoporosis (Lambinoudaki and Kung 2000).

- A high-calcium diet plus vitamin D supplements (Toogood 1998), together with monitoring for vitamin D intoxication (Arrin et al 2002).

- Bone densitometry on starting treatment and after six months (Schimmer and Parker 2001), as most bone loss occurs in the first six months of therapy. However, results should be interpreted cautiously, as fractures may occur at higher values of bone mineral density than in people who have never taken corticosteroids (Walsh et al 1996).

- Hormone replacement therapy for post-menopausal women (Lipworth 1999, Toogood 1998). Osteonecrosis of the head of femur or humerus is a serious and inevitable adverse effect of high-dose therapy. Therefore, joint pain and stiffness in a patient prescribed corticosteroids should trigger urgent investigations. Tendon rupture is a rare adverse effect (BNF 2002).

- Permanent growth suppression rarely occurs with less than six months’ treatment, but prolonged use of oral corticosteroids may reduce final height and delay puberty. However, untreated asthma or antritis also impairs growth. Research on inhaled cortico-steroids indicates that they may slow growth, but final adult height is not usually affected, unless high doses (for example, 1mg fluticasone propion-ate/day) are administered (Cave et al 1999, Lipworth 1999). Growth monitoring on centile charts is important for all children using any steroid preparation, as there is considerable individual vari-ation in response (BNF 2002, Toogood 1998).

- Muscle Protein loss from skeletal muscles contributes to muscle wasting and weakness, which is reported by 60 per cent of users (Walsh et al 2001). This cannot be remedied by increased protein intake, because corticosteroids inhibit protein synthesis (Neal 1997). Muscles of the arm and thigh are particularly affected, and get out of chairs. Myopathy, or muscle wasting, is particularly serious in older women, who already have low muscle mass.

- Respiratory myopathy in patients with respiratory disorders further diminishes respiratory function; has low muscle mass. and get out of chairs. Myopathy, or muscle wasting, is particularly serious in older women, who already have low muscle mass.

- Cardiovascular problems Long-term corticosteroids intensify coronary atherosclerosis. Much of the increased risk is due to dyslipidaemia, hyperglycaemia (see above) and hypertension. Steroids increase sodium ion reabsorption in the kidneys, in exchange for potassium or hydrogen ions. This promotes fluid retention, weight gain, hypertension and congestive heart failure. Pulmonary oedema and hypokalaemia are of particular con-cern where corticosteroids are co-administered with beta-agonists in the management of acute asthma (Jordan and White 2001a). Twenty per cent of patients taking corticosteroids have hypertension (Sholter and Armstrong 2000). Steroid-induced hypertension is attributable to: Fluid retention. Enhanced vascular responses to vasoconstrictors, particularly noradrenaline (norepinephrine).

- Diminished responses to vasoconstrictors, such as nitrates. This impairs the ability of the blood ves-sels to dilate in hot environments, predisposing users to hyperthermia.

- Increased cardiac contractility. For most corticosteroids, restriction of dietary sodium reduces, but does not abolish, the rise in blood pressure. However, sodium restriction may have no effect on hypertension caused by drugs with no mineralocorticoid actions, such as dexamethasone. Corticosteroids increase erythropoietin produc-tion, which stimulates red blood cell production. The resulting polycythaemia is responsible for the characteristic red face and the possible association with thromboembolism (BNF 2002).

- Prolonged exposure to corticosteroids causes loss of muscle protein and fibrosis of the heart muscle. Corticosteroids administered after myocardial infarc-tion reduce healing; this increases infarct size and makes the heart wall vulnerable to rupture and aneurysm formation (Sholter and Armstrong 2000).

- Central nervous system Euphoria, irrespective of the underlying disease severity, is the usual initial response to corticosteroids, sometimes accompanied by agitation, restlessness, insomnia, rapid speech or even mania. Steroid psychosis, with hallucinations, delusional beliefs, thought disorders and suicidal ideation, is relatively rare. Long-term therapy is asso-ciated with mood swings, depression and anxiety (Brown et al 1999; Pre-existing mental illness may be aggravated (BNF 2002). Corticosteroids are asso-ciated with cognitive impairment and deficits in verbal memory, possibly as a result of steroid-induced atrophy of the hippocampus and cerebral cortex. Even after long-term use, discontinuation of med-ication often reverses memory loss (Brown et al 1999).

- Corticosteroids lower the seizure threshold, wors-ening epilepsy and Occasionally causing convul-sions, particularly with intravenous administration.

- Eyes Severity-five per cent of patients develop cataracts after several years of 15mg/day oral pred-nisolone (BNF 2002). Cessation of therapy may not resolve the condition or prevent progression (Schimmer and Parker 2001). Cataracts have been reported as a serious and irreversible adverse effect of high-dose corticosteroids among asthmatic patients. Chest. 115, 3, 587-592. British National Formulary (2002) British National Formulary No. 44. London, British Medical Association and the Royal Pharmaceutical Society of Great Britain.


- Beta2-agonists in the management of acute asthma (Last accessed June 24 2002).


- British Medical Association and the Royal Pharmaceutical Society of Great Britain. 51, RR-2, 1-36.


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CONTINUING PROFESSIONAL DEVELOPMENT

Table 4. Conditions requiring cautious use of corticosteroids

<table>
<thead>
<tr>
<th>Medical condition</th>
<th>Potential problems</th>
</tr>
</thead>
<tbody>
<tr>
<td>AIDS/HIV, tuberculosis, fungal infection, herpes simplex infection or infection at site of recent surgery or serious injury</td>
<td>Slow healing, worsening of existing infections, or increased susceptibility to new infections. Monitor very carefully if there is history of tuberculosis. Corticosteroids induce fluid retention, worsening those conditions. Deleterious effects on risk factors.</td>
</tr>
<tr>
<td>Congestive heart failure, hypertension, kidney disease</td>
<td>Deterioration of diabetes. Loss of control and even ketoacidosis may follow.</td>
</tr>
<tr>
<td>Coronary heart disease</td>
<td>Masking of symptoms may allow complications to develop quickly.</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>Increased risk of complications.</td>
</tr>
<tr>
<td>Diverticulitis, peptic ulcer or ulcerative colitis</td>
<td>Glaucoma triggered or exacerbated.</td>
</tr>
<tr>
<td>Epilepsy</td>
<td>Glaucoma or family history of glaucoma.</td>
</tr>
<tr>
<td>Glaucoma or family history of glaucoma</td>
<td>First dose of corticosteroids may cause muscle weakness, which may cause problems with breathing. Close supervision required.</td>
</tr>
<tr>
<td>Joint damage</td>
<td>Corticosteroids may be eliminated slowly, which may accentuate adverse effects.</td>
</tr>
<tr>
<td>Liver disease/underactive thyroid</td>
<td>Corticosteroids increase bone loss and risk of fractures.</td>
</tr>
<tr>
<td>Myasthenia gravis</td>
<td>Hirsutism, amenorrhoea and impotence, as a result of inhibition of oestrogens and androgens (Hoffmeister and Tietze 2000).</td>
</tr>
<tr>
<td>Myasthenia gravis</td>
<td>Corticosteroids induce fluid retention, worsening those conditions. Deleterious effects on risk factors.</td>
</tr>
<tr>
<td>Osteoporosis, low bone density</td>
<td>Corticosteroids induce fluid retention, worsening those conditions. Deleterious effects on risk factors.</td>
</tr>
<tr>
<td>Ovarian hyperstimulation</td>
<td>Corticosteroids may be eliminated slowly, which may accentuate adverse effects.</td>
</tr>
<tr>
<td>Psychosis, pre-existing mental illness, previous steroid-induced psychosis</td>
<td>Corticosteroids increase bone loss and risk of fractures.</td>
</tr>
<tr>
<td>Recent myocardial infarction</td>
<td>Corticosteroids induce fluid retention, worsening those conditions. Deleterious effects on risk factors.</td>
</tr>
<tr>
<td>Systemic lupus erythematosus</td>
<td>Corticosteroids induce fluid retention, worsening those conditions. Deleterious effects on risk factors.</td>
</tr>
</tbody>
</table>

(BNF 2002, MDI 2002)

Figure 2. Control of glucocorticoid secretion – hypothalamic/pituitary/adrenal (HPA) axis

Corticosteroids administered as medications constantly inhibit CRH and ACTH secretion. The adrenal cortex eventually shrinks and fails to synthesise any hormones, even in response to extreme stress.

Systemic therapy has been associated with hirsutism, amenorrhoea and impotence, as a result of inhibition of oestrogens and androgens (Hoffmeister and Tietze 2000).

Adrenal suppression and insufficiency The potentially life-threatening condition is due to disruption of the hypothalamic/pituitary/adrenal (HPA) axis, which normally controls the production of steroids from the adrenal glands (Figure 2). The hypothalamus secretes corticotrophin-releasing hormone (CRH), which stimulates the anterior pituitary to secrete adrenocorticotrophic hormone (ACTH). This stimulates the adrenal cortex to release corticosteroids. Secretion of CRH is controlled by:

Stressors acting on the hypothalamus – for example, trauma, pain, illness, infection, labour.

Circadian (24-hour) rhythm. With normal sleep-wake patterns, CRH peaks around midnight, causing ACTH and corticosteroid hormones to peak in the early morning (6-9am). Disruption to the circadian cycle of corticosteroid secretion and adrenal suppression is minimised if oral corticosteroids are given in the morning, as a single dose before 9am, or on alternate days (ICSM 1998).

Negative feedback control by circulating corticosteroids, endogenous or artificial, suppressing CRH and ACTH secretion. Corticosteroid drugs disrupt the regular feedback system and remove the normal stimulation to the adrenals. Eventually, the adrenals atrophy and are no longer able to increase hormone secretion in response to stress. Adrenal suppression shows individual variation, but may occur after two weeks’ of oral, topical, inhaled or intranasal corticosteroids within the recommended dose range (Cave et al 1999). However, in adults, clinical consequences are unlikely at doses below 2000µg/day for inhaled beclomethasone (Clark and Lipworth 1997). Patients and professionals need to be alert for symptoms and signs of adrenal insufficiency, which include weakness, nausea, weight loss, hypoglycaemia, dehydration, electrolyte imbalance and hypotension.

There is some correlation between the severity of bruising and adrenal suppression. Therefore, detection of bruises should alert professionals to the need for investigations (Lipworth 1999). Adrenal crises arise when individuals with adrenal suppression and insufficiency are exposed to severe stress. The adrenals are unable to respond to stress in the usual way by secreting corticosteroids. The resulting medical emergency is characterised by nausea, vomiting, abdominal pain, exhaustion, dehydration, hypotension and shock. In severe cases, blood pressure falls rapidly, fatalities have occurred (BNF 2002). To aid emergency admission, it is important that steroid cards be given to patients receiving corticosteroids for more than three weeks (ICSM 1998), including those using high-dose inhalers.
I

Withdrawal of therapy

If administration has continued for less than a week, problems with withdrawal are unlikely (BNF 2002). Otherwise, doses should be gradually tapered to topical routes.

Cautions and contraindications

Before therapy is started, specific enquiries should be made regarding cautions and contraindications. Corticosteroids exacerbate several long-term conditions, such as diabetes mellitus, peptic ulcer disease, epilepsy, hypertension, congestive cardiac failure and mental illness (Abrams 1995) (Table 4). Experts feel that corticosteroids are ill-advised for older people with diabetes (Hanlot et al 2001) or heart failure (Remme and Swedberg 2001). Caution is advised in patients at risk of infection, as resistance is lowered, or with current infections, as signs and symptoms will be masked, allowing silent progression. Corticosteroids are contraindicated for patients with:

- Systemic infections, particularly fungal infections, tuberculosis, measles or chickenpox, including recent exposure and illness – severe infection may result.
- Eye infections, particularly herpes simplex – risk of corneal perforation with eye drops (BNF 2002).
- Hypersensitivity to corticosteroids.
- Oesophagitis, gastritis or peptic ulcer – these conditions may progress silently, and clients may bleed without warning (McKerny and Salerno 1998).

Pregnancy and breastfeeding

The relative importance of disease control and adverse effects requires careful evaluation. Corticosteroids cross the placenta to a variable extent. Intrauterine growth retardation and reduced head circumference may occur if corticosteroids are administered for prolonged periods or short courses are repeated. Prednisolone (administered orally) passes into breast milk. Doses above 40mg/day may cause adverse effects in breastfed neonates, who should be carefully monitored (BNF 1998). Topical corticosteroids should not be applied to the breasts of breastfeeding mothers (Jordan 2000).

Interactions

The effects of corticosteroids are modified by co-administration with many drugs (Table 5). Drugs such as cimetidine and rifampicin, which increase the activity of liver enzymes, accelerate the elimination of corticosteroids, thereby decreasing their effectiveness. Other drugs, such as ketorolac and erythromycin, inhibit liver enzymes, decreasing the elimination of corticosteroids and increasing adverse effects. Corticosteroids worsen many common disorders, such as hypertension and diabetes. The doses of medications prescribed to manage these conditions may therefore need to be increased (Stockley 1999).

Medication management and patient monitoring

When initiating steroid therapy, certain ‘baseline’ observations will be needed: weight, blood pressure, heart rate, temperature, respiration rate and depth, assessment of peripheral perfusion, cognitive orientation and grip strength. A venous blood sample will also be needed to assess blood glucose, electrolyte balance and full blood count. Pre-existing infection should be investigated through enquiry and examination (Karch 2000). Patients and families should be supplied with information leaflets, advice and, where appropriate, steroid cards. Many patients are concerned about the adverse effects of corticosteroids, particularly weight gain, infections, fractures, stunted growth and ocular complications, to the extent that more than one third would refuse any corticosteroid medication (Roulet 1998). Nurses


Leavy B (1999) Systemic adverse effects of inhaled corticosteroids: a systematic review and meta-analysis. Archives of Internal Medicine, 159, 9, 941-945.

Table 5. Drug-drug interactions

<table>
<thead>
<tr>
<th>Drug</th>
<th>Results of drug-drug interaction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcohol, non-steroidal anti-inflammatory drugs (NSAIDs), aspirin</td>
<td>Gastrointestinal bleeding is an important consideration</td>
</tr>
<tr>
<td>Estradiol vaginal cream</td>
<td>Absorption of oral medication is reduced by co-administration. Separate administration by a period of two hours may increase the elimination of corticosteroids (see text)</td>
</tr>
</tbody>
</table>

Both increased and decreased coagulation have been reported. Careful attention to postmenstrual times is important. Corticosteroids may increase blood glucose concentration, necessitating increased doses of antidiabetic medications. Corticosteroids may lower the threshold for convulsions, necessitating higher doses of antiepileptic drugs.

Exacerbate sodium retention and potassium loss. Where high doses are administered in medical emergencies, fluid retention can lead to fluid overload and pulmonary oedema. Concentrations of both ciclosporin and corticosteroids are increased. While these drugs are commonly co-prescribed for patients who have received organ transplants, their combined actions may increase the incidence of convulsions and hyperglycaemia (Stockley 1999). Metothrexate toxicity may also be increased. Use of ciclosporin with methylprednisolone may cause convulsions (BNF 2002).

Plasma corticosteroid concentrations are increased in patients taking oestro-gen-containing contraceptives, but the clinical significance of this is uncertain. A dosage reduction of corticosteroids may be required. Intravenous device failure has occasionally been attributed to anti-inflammatory agents, including corticosteroids.

Hypokalaemia, which may cause respiratory muscle weakness, bradycardia, heart block or cardiac arrest. This is important where drugs are administered in high doses to manage medical emergencies.

Co-administration with diuretics may make the diuretic less effective. Reduce the elimination of corticosteroids (see text).

Seek medical advice (see Box 3). There may be a marked rise in blood pressure and fall in potassium concentration. Suggest patients do not eat liquorice.

Exacerbate fluid retention, hypertension and congestive heart failure. Isolated reports of antagonism by corticosteroids.

Corticosteroids may invalidate results of skin tests.

Corticosteroids and growth hormone have opposing effects on growth.

Theophylline concentrations may be increased or decreased. Steroid-induced agitation and psychosis may be worsened, but withdrawal symptoms may be eased.

need to explain the potential complications and adverse effects of corticosteroid medication, as well as the rationale for prescription. Some patients may require psychological or emotional support to cope with the effect of corticosteroids on body image. The efficacy of prescribed corticosteroid regimens can be optimised by regular monitoring of the response to the drug, adverse effects (Table 6), adherence to the prescribed regimen and the effectiveness of patient education. Some practitioners may formulate nursing diagnoses in relation to corticosteroid therapy, for example:

- Altered cardiac output related to fluid retention.
- Fluid volume excess related to water retention.
- Infection related to immunosuppression.
- Ineffective individual coping related to body change and body image.
- Knowledge deficit regarding drug therapy (McKerney and Salerno 1998).

Conclusion

Inadequate treatment and follow-up remain important causes of preventable asthma deaths (Burr et al 1999). However, corticosteroid use is complicated by numerous adverse drug reactions, both during therapy and following withdrawal; any of these may affect patient concordance. Apart from replacement...
regimens, authorities are agreed that corticosteroids should be prescribed at the lowest effective dose for the shortest possible time (BNF 2002, Cave et al 1999). Nurses play important roles in monitoring disease, adherence and adverse effects. Unless professionals make specific enquiries regarding adverse effects, patients may not divulge this information; rather, they will miss doses or withhold their medication altogether (White and Sander 1999). The guidelines in Tables 4, 5 and 6 may help to minimise risks and enhance adherence.

### Table 6. Managing the common adverse effects of corticosteroids

<table>
<thead>
<tr>
<th>Potential problem</th>
<th>Suggested preventive measures</th>
</tr>
</thead>
</table>
| Increased risk of infections | ■ Teach good hand-washing techniques  
 ■ Monitor body temperature daily at 5-6pm  
 ■ Avoid exposure to infectious disease  
 ■ Contact doctor on exposure to chickenpox or measles  
 ■ Adjust immunisations (Box 2) |
| Nutrition | ■ Encourage a well-balanced, low-calorie diet  
 ■ Ask dietician to provide diet plan  
 ■ Monitor diet by asking patient to record intake for 24-hour periods  
 ■ Weigh patient weekly  
 ■ Regularly measure waist circumference  
 ■ Encourage snuspltus dental hygiene and low-sugar diet  
 ■ Arrange six-monthly dental inspections  
 ■ Suggest patient uses a mouthwash  
 ■ Foods rich in salt should be avoided, except with replacement regimens. Condiments and processed foods are high in sodium. Avoid salt-containing medicines such as antacids. Avoid liqueurs |
| Risk of dental caries | ■ Monitor blood pressure regularly  
 ■ Encourage patient to eat foods high in calcium; low-fat dairy products suggested  
 ■ Recommend the patient takes vitamin D and possibly calcium supplements  
 ■ Venous blood samples to monitor electrolytes  
 ■ Encourage foods that are high in potassium, such as raisins, bananas, meat |
| Risk of hypertension | ■ Limit salt intake  
 ■ Fluid balance records and daily weighing are important during initiation of therapy  
 ■ Regular eye examinations are important to detect changes before permanent eye damage occurs – arrange appointments on initiation of therapy, after six months, then at least yearly |
| Risk of osteoporosis | ■ Monitor blood pressure regularly  
 ■ Encourage patient to eat foods high in calcium, low-fat dairy products suggested  
 ■ Recommend the patient takes vitamin D and possibly calcium supplements  
 ■ Venous blood samples to monitor electrolytes  
 ■ Encourage foods that are high in potassium, such as raisins, bananas, meat |
| Loss of potassium, causing muscle weakness, depression, constipation, cardiac complications, Salt and water retention | ■ Monitor blood glucose concentrations regularly and if thrush appears on the skin  
 ■ Monitor lipid profile  
 ■ Observe for breathlessness; monitor fluid retention; minimise salt intake  
 ■ Monitor full blood count  
 ■ Encourage moderate exercise  
 ■ Bone densitometry assessments  
 ■ Consider hormone replacement therapy  
 ■ Regularly plot height/weight on centile charts |
| Cardiovascular disease | ■ Monitor blood glucose concentrations regularly and if thrush appears on the skin  
 ■ Monitor lipid profile  
 ■ Observe for breathlessness; monitor fluid retention; minimise salt intake  
 ■ Monitor full blood count  
 ■ Monitor body temperature daily at 5-6pm  
 ■ Avoid exposure to infectious disease  
 ■ Contact doctor on exposure to chickenpox or measles  
 ■ Adjust immunisations (Box 2) |
| Hypertension | ■ Monitor blood glucose concentrations regularly and if thrush appears on the skin  
 ■ Monitor lipid profile  
 ■ Observe for breathlessness; monitor fluid retention; minimise salt intake  
 ■ Monitor full blood count  
 ■ Encourage moderate exercise  
 ■ Bone densitometry assessments  
 ■ Consider hormone replacement therapy  
 ■ Regularly plot height/weight on centile charts |
| Diabetes | ■ Monitor blood glucose concentrations regularly and if thrush appears on the skin  
 ■ Monitor lipid profile  
 ■ Observe for breathlessness; monitor fluid retention; minimise salt intake  
 ■ Monitor full blood count  
 ■ Encourage moderate exercise  
 ■ Bone densitometry assessments  
 ■ Consider hormone replacement therapy  
 ■ Regularly plot height/weight on centile charts |
| Gastrointestinal tract | ■ Increased intracocular pressure and glaucoma; cataracts or clouding of vision; infections |
| Skin | ■ Take oral corticosteroids with food or milk  
 ■ Observe and test stools for blood loss  
 ■ No smoking  
 ■ No alcohol  
 ■ Keep patient cool and dry  
 ■ Encourage foods that are high in salt should be avoided, except with replacement regimens. Condiments and processed foods are high in sodium. Avoid salt-containing medicines such as antacids. Avoid liqueurs |
| Hair | ■ Increased risks of thrombosis  
 ■ Increased cholesterol  
 ■ Hyperglycaemia/diabetes  
 ■ Cardiovascular disease  
 ■ Loss of potassium, causing muscle weakness, depression, constipation, cardiac complications, Salt and water retention |
| Increased risk of infections | ■ Provide advice on managing acne  
 ■ Consult podiatrist regarding foot care  
 ■ Anticipate poor healing, contact wound care specialists promptly  
 ■ Take swabs if healing delayed  
 ■ Increase vigilance of pressure areas; regularly evaluate pressure damage risk score  
 ■ Avoid friction and shearing forces on the skin – teach patients correct use of moving and handling aids  
 ■ Allow extra time for procedures involving tissue handling  
 ■ Ensure good communication within the multidisciplinary team. For example, orthopaedic surgeons and plaster technicians need to be aware that the patient is prescribed corticosteroids and adjust treatment, if possible |
| Potential problem | Suggested preventive measures |
| Thinning of the skin | ■ Encourage moderate exercise  
 ■ Bone densitometry assessments  
 ■ Consider hormone replacement therapy  
 ■ Regularly plot height/weight on centile charts |
| Potential problem | Suggested preventive measures |
| Poor wound healing | ■ Monitor blood glucose concentrations regularly and if thrush appears on the skin  
 ■ Monitor lipid profile  
 ■ Observe for breathlessness; monitor fluid retention; minimise salt intake  
 ■ Monitor full blood count  
 ■ Encourage moderate exercise  
 ■ Bone densitometry assessments  
 ■ Consider hormone replacement therapy  
 ■ Regularly plot height/weight on centile charts |
| Gastrointestinal tract | ■ Increased intracocular pressure and glaucoma; cataracts or clouding of vision; infections |
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 ■ Allow extra time for procedures involving tissue handling  
 ■ Ensure good communication within the multidisciplinary team. For example, orthopaedic surgeons and plaster technicians need to be aware that the patient is prescribed corticosteroids and adjust treatment, if possible |
| Potential problem | Suggested preventive measures |
| Mental health | ■ Increased intracocular pressure and glaucoma; cataracts or clouding of vision; infections |
| Reproductive system | ■ Regular eye examinations are important to detect changes before permanent eye damage occurs – arrange appointments on initiation of therapy, after six months, then at least yearly |
| Adrenal suppression/insufficiency | ■ Regular eye examinations are important to detect changes before permanent eye damage occurs – arrange appointments on initiation of therapy, after six months, then at least yearly |
| Withdrawal of therapy | ■ Regular eye examinations are important to detect changes before permanent eye damage occurs – arrange appointments on initiation of therapy, after six months, then at least yearly |

**TIME OUT 6**

Now that you have completed the article, you might like to write a practice profile. Guidelines to help you are on page 55.