

Inhaled and intranasal corticosteroids

Topically active glucocorticoids delivered straight to the bronchial or nasal mucosa to suppress airway inflammation over days and weeks; they are maintenance agents, they open no airway acutely, and almost everything that limits them follows from the fraction of drug that misses the target and reaches the rest of the body.

Online guide: <https://www.medguideglobal.com/pharmacology/respiratory-and-allergy/inhaled-corticosteroids>

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Quick revision

One Liner: An inhaled corticosteroid prevents the next attack rather than treating the present one, and every important adverse effect is either local damage where the drug lands or systemic exposure from the fraction that escapes the airway.

1. These are maintenance anti-inflammatory agents. The fluticasone propionate inhalation aerosol label states plainly that the product "is NOT indicated for the relief of acute bronchospasm". [1]
2. The benefit builds slowly. The budesonide inhalation powder label says improvement can occur within 24 hours of starting, "although maximum benefit may not be achieved for 1 to 2 weeks, or longer". [2]
3. During an acute attack a short-acting bronchodilator is the agent that acts; the fluticasone label directs the patient to "always use a short-acting bronchodilator (rescue medicine)" at that moment. [1]
4. Oropharyngeal candidiasis and dysphonia are the signature local effects of the inhaled route, and both appear among the most common adverse reactions in the fluticasone propionate aerosol label. [1]
5. Rinsing after each dose is the countermeasure the labels themselves specify: the beclometasone label says to rinse the mouth with water without swallowing "to help reduce the risk of oropharyngeal candidiasis". [3]
6. Systemic effects are real but dose- and duration-related: hypercorticism and adrenal suppression, reduced bone mineral density with long-term use, glaucoma and cataract, and slowed growth in children. [1][3]
7. Paediatric growth is monitored routinely, by stadiometry, for as long as an inhaled or intranasal corticosteroid is continued. [3][4]
8. Strong CYP3A4 inhibitors such as ritonavir and ketoconazole raise systemic corticosteroid exposure far enough to have produced Cushing's syndrome and adrenal suppression after marketing. [4][1]
9. By the intranasal route the local problems change: epistaxis, nasal ulceration, nasal septal perforation and impaired wound healing replace thrush and hoarseness as the things to watch for. [4][6]
10. UK regulators have advised vigilance for pneumonia in patients with chronic obstructive pulmonary disease treated with inhaled medicines containing a steroid. [7]

Overview

1. Corticosteroids given by inhaler or nasal spray exploit a simple pharmacological trick: put a highly potent, poorly absorbed glucocorticoid directly onto the inflamed mucosa, and the anti-inflammatory effect is obtained locally while systemic exposure stays low. Budesonide, for example, has roughly two hundred times the glucocorticoid receptor affinity of cortisol and about a thousand times its topical anti-inflammatory potency, so very little material needs to arrive to do the work. [2]
2. The consequence students must fix first is that nothing in this mechanism relaxes bronchial smooth muscle. Suppressing inflammation changes the airway over days, so a preventer inhaler is useless in the minutes of an attack, and the fluticasone label reserves that role for a short-acting bronchodilator. Treating a preventer as a rescue inhaler is the commonest and most dangerous misunderstanding attached to this class. [1]
3. Because the drug is deposited rather than injected, the two ends of the dose-response curve behave differently.

What lands on the mucosa causes the local trouble: thrush, hoarseness, nosebleeds. What is swallowed or absorbed across the lung causes the systemic trouble, and the labels tie that arm firmly to how much is given and for how long, warning that the benefit of these agents in minimising adrenal axis dysfunction can only be expected when recommended dosages are not exceeded. [2][1]

4. Device and technique therefore sit inside the pharmacology rather than beside it. A valved holding chamber with a face mask is offered in the fluticasone aerosol label as a way of delivering the drug to young patients, while the breath-actuated beclometasone inhaler must not be used with one at all, and the NHS recommends a spacer for anyone on high steroid doses from a pressurised metered-dose inhaler and for every child aged fifteen or under. [1][3][8]

Classification and drug examples

Intro: Grouped by the route the drug is delivered to, because the route decides the indication, the local adverse effects and the size of the systemic dose. The molecules overlap freely across the two groups: fluticasone and beclometasone each appear as an inhaler and as a nasal spray.

1. Name: Orally inhaled corticosteroids for the lower airway

1. Note: Delivered by metered-dose inhaler, dry-powder device or nebuliser for asthma maintenance. Their shared local burden is oropharyngeal candidiasis and dysphonia; their shared systemic burden is adrenal, skeletal, ocular and growth-related.

1. Name: Fluticasone propionate

Flovent

1. Route: Oral inhalation

1. Note: Described in its label as a synthetic trifluorinated corticosteroid with potent anti-inflammatory activity. Oral systemic bioavailability is negligible, under one per cent, so the swallowed fraction contributes little and most systemic exposure comes from the lung itself. [1]

2. Name: Budesonide

Pulmicort

2. Route: Oral inhalation, as dry powder or nebuliser suspension

2. Note: An anti-inflammatory corticosteroid with potent glucocorticoid and weak mineralocorticoid activity. Named here without a link because the only released page for it on this site covers a budesonide-formoterol combination inhaler, which is a different product. [2]

3. Name: Beclometasone dipropionate

1. Beclomethasone dipropionate

2. QVAR

3. Route: Oral inhalation

3. Note: A prodrug, rapidly activated by hydrolysis to the active monoester 17-monopropionate, with lung tissue itself performing much of that conversion. Excretion is mainly faecal. [3]

4. Name: Mometasone furoate and ciclesonide

4. Route: Oral inhalation

4. Note: Further members of the same group used as asthma preventers. Neither has a medicine page on this site, so both are named rather than linked; they share the class warnings on candidiasis, adrenal suppression and growth that the fluticasone and beclometasone labels carry. [1][3] [1][3]

2. Name: Intranasal corticosteroids for the upper airway

2. Note: Aqueous pump sprays for rhinitis and nasal polyps. The systemic warnings are the same in kind, but the local ones are mucosal injury rather than infection of the throat.

1. Name: Beclometasone dipropionate, intranasal

Beconase AQ

1. Route: Intranasal spray

1. Note: Its label covers seasonal and perennial allergic rhinitis, nonallergic vasomotor rhinitis, and prevention of nasal polyps recurring after surgery. Significant relief was obtained within three days in trials, but some patients waited as long as two weeks. [6]

2. Name: Fluticasone propionate, intranasal

Flonase

2. Route: Intranasal spray

2. Note: Available both on prescription and over the counter. The prescription generic reviewed here is licensed for the nasal symptoms of perennial nonallergic rhinitis from four years of age, while the over-the-counter presentation is sold for hay fever and other upper respiratory allergies. [4][5] [6][4]

Mechanism of action

Summary: A glucocorticoid receptor agonist is deposited on the airway mucosa, where it shuts down a wide range of inflammatory cells and the mediators they release. The molecular chemistry is chosen for high receptor affinity and poor oral absorption, so the anti-inflammatory effect concentrates at the site of deposition. The labels are candid that the step from that receptor event to the clinical improvement in asthma is not fully worked out.

Target: The cytosolic glucocorticoid receptor in bronchial and nasal mucosal cells and in the inflammatory cells infiltrating them

Cidal Static: Inflammation-suppressing and preventive, not bronchodilating and not curative

Kill Kinetics: Effect accumulates over days to weeks of continuous use and recedes after withdrawal

1. Step: 1

1. Title: Deposition on the mucosa and binding of the glucocorticoid receptor

1. Detail: The inhaled or sprayed particle lands on bronchial or nasal epithelium and the drug enters local cells to engage the glucocorticoid receptor. Potency at that receptor is deliberately extreme: budesonide binds it around two hundred times more avidly than cortisol and is roughly a thousand times more potent as a topical anti-inflammatory. Beclometasone dipropionate arrives as a prodrug and is hydrolysed rapidly to its active monoester, a conversion that lung tissue performs quickly. [2][3]

2. Step: 2

2. Title: Broad suppression of inflammatory cells and their mediators

2. Detail: Receptor occupancy alters transcription so that many arms of the inflammatory response fall together. The budesonide label records inhibitory activity against mast cells, eosinophils, neutrophils, macrophages and lymphocytes, and against histamine, eicosanoids, leukotrienes and cytokines. The fluticasone label describes the same breadth, listing basophils as well and noting inhibition of mediator production or secretion. [2][1]

3. Step: 3

3. Title: Reduced mucosal oedema and vascular leak in the nose

3. Detail: Applied topically to the nasal mucosa, beclometasone dipropionate produces anti-inflammatory and vasoconstrictor effects. Shrinking the congested, leaking mucosa is what relieves blockage in rhinitis, and the same suppression of inflammation is what discourages nasal polyps from re-forming after they have been removed surgically. [6]

4. Step: 4

4. Title: No bronchodilator step exists anywhere in this sequence

4. Detail: Nothing above relaxes airway smooth muscle, which is why relief is measured in days rather than minutes and why every one of these labels excludes acute bronchospasm from the indication. A patient in an attack needs a short-acting bronchodilator; adding extra puffs of a preventer does not substitute for one, and the fluticasone label warns against inhaling more doses or using the inhaler more often than prescribed. [1][2]

5. Step: 5

5. Title: The escaping fraction produces the systemic effects

5. Detail: Most of what reaches the lung is absorbed from it, and fluticasone propionate is then bound about ninety-nine

per cent to plasma proteins and cleared through CYP3A4 to a single circulating carboxylic acid metabolite. That absorbed fraction is what suppresses the hypothalamic-pituitary-adrenal axis, thins bone and slows growth, and it is why the labels anchor those risks to dose and to duration of exposure. [1]

6. Step: 6

6. Title: An honest gap at the end of the chain

6. Detail: The beclometasone label states that the precise mechanism of corticosteroid action on asthma is not known, and the budesonide label says the same of corticosteroid actions on inflammation in asthma. The receptor pharmacology is well described; the causal path from it to a fall in exacerbation rate is inferred rather than demonstrated, and the page does not claim otherwise. [3][2]

Major clinical uses

1. Drug: Fluticasone propionate, inhaled

1. Organism: Not applicable

1. Indication: Maintenance treatment of asthma as prophylactic therapy

1. Line: first-line preventer

1. Note: Prescribed for continuous daily use rather than for symptoms. Maximum benefit, the label says, may not be achieved for one to two weeks or longer after treatment begins, which is why early judgements about whether it is working are unreliable. [1]

2. Drug: Budesonide, inhaled

2. Organism: Not applicable

2. Indication: Maintenance treatment of asthma as prophylactic therapy from six years of age

2. Line: first-line preventer

2. Note: Available as a dry-powder inhaler and as a nebuliser suspension, which extends the class into younger children and into patients who cannot coordinate a hand-held device. Named without a link because the released page here is for a combination inhaler. [2]

3. Drug: Beclometasone dipropionate, inhaled

3. Organism: Not applicable

3. Indication: Maintenance treatment of asthma as prophylactic therapy in adults and children from four years of age

3. Line: first-line preventer

3. Note: The breath-actuated presentation removes the need to coordinate actuation with inspiration, but its label specifically forbids using it with a spacer or volume holding chamber, so device instructions are not transferable between products. [3]

4. Drug: Beclometasone dipropionate, intranasal

4. Organism: Not applicable

4. Indication: Relief of the symptoms of seasonal or perennial allergic and nonallergic vasomotor rhinitis

4. Line: first-line for persistent rhinitis

4. Note: Onset is faster than in the lung but still not immediate: significant symptomatic relief was obtained within three days in the trials reported, yet relief may not appear in some patients for as long as two weeks. [6]

5. Drug: Beclometasone dipropionate, intranasal

5. Organism: Not applicable

5. Indication: Prevention of recurrence of nasal polyps following surgical removal

5. Line: established

5. Note: A distinct licensed indication rather than an off-label extension of rhinitis treatment, and a reminder that the class treats mucosal inflammation wherever it can be reached topically. [6]

6. Drug: Fluticasone propionate, intranasal

6. Organism: Not applicable

6. Indication: Management of nasal symptoms of rhinitis, including over-the-counter use for hay fever and other upper

respiratory allergies

6. Line: widely used

6. Note: The prescription generic examined here is licensed for perennial nonallergic rhinitis from four years of age. The over-the-counter presentation is sold for temporary relief of nasal congestion, runny nose, sneezing and itchy or watery eyes. [4][5]

7. Drug: Inhaled corticosteroid added to bronchodilator therapy

7. Organism: Not applicable

7. Indication: Chronic obstructive pulmonary disease, as an addition to existing bronchodilator treatment

7. Line: selected patients, guideline-directed

7. Note: UK regulators state that inhaled steroids may be added to ongoing bronchodilator therapy in COPD according to treatment guidelines, while advising vigilance for pneumonia. Any patient with severe COPD who develops pneumonia on such treatment should have that treatment reconsidered. [7]

Pharmacokinetics

1. Drug: Fluticasone propionate, inhaled

1. Route: Oral inhalation

1. Absorption: The majority of what reaches the lung is systemically absorbed; oral systemic bioavailability is negligible, below one per cent, because of incomplete absorption and presystemic metabolism

1. Csf Penetration: Not applicable

1. Metabolism: CYP3A4, producing the 17-beta-carboxylic acid derivative as the only circulating metabolite detected in man

1. Elimination: Hepatic clearance of the CYP3A4 metabolite

1. Half Life: Terminal elimination half-life of approximately 7.8 hours after intravenous dosing

1. Dose Adjust: Strong CYP3A4 inhibitors are the interaction that matters, because clearance depends on that single pathway [1]

2. Drug: Beclometasone dipropionate, inhaled

2. Route: Oral inhalation

2. Absorption: Absorbed as the parent prodrug and as its active monoester; lung tissue itself hydrolyses the dipropionate rapidly

2. Csf Penetration: Not applicable

2. Metabolism: Ester hydrolysis to three major metabolites, of which 17-monopropionate is the active one

2. Elimination: Mainly faecal; under ten per cent of drug and metabolites appear in urine

2. Half Life: Terminal half-life of the active monoester of about four hours

2. Dose Adjust: Plasma protein binding of the active monoester is roughly 94 to 96 per cent across the measured concentration range [3]

3. Drug: Budesonide, inhaled

3. Route: Oral inhalation

3. Absorption: Delivered topically to the airway, with the systemic contribution governed by the dose given

3. Csf Penetration: Not applicable

3. Metabolism: Hepatic; the label frames the systemic corticosteroid burden as a function of staying within recommended dosages

3. Elimination: Hepatic

3. Half Life: Short relative to the duration of the anti-inflammatory effect on the mucosa

3. Dose Adjust: The label states that the benefit in minimising adrenal axis dysfunction can be expected only when recommended dosages are not exceeded [2]

4. Drug: Fluticasone propionate, intranasal

4. Route: Intranasal

- 4. Absorption: Topical application to the nasal mucosa, with a smaller absorbed fraction than the inhaled route but not a negligible one
- 4. Csf Penetration: Not applicable
- 4. Metabolism: CYP3A4
- 4. Elimination: Hepatic
- 4. Half Life: As for the inhaled molecule
- 4. Dose Adjust: Ketoconazole coadministration raised plasma fluticasone propionate exposure 1.9-fold and lowered plasma cortisol exposure by 45 per cent [4]
- 1. No dose regimens appear on this page. Choice of molecule, device, strength and step-up or step-down belongs to the treating clinician working from a current prescribing reference and the individual patient's control, age and comorbidity.
- 2. The pharmacokinetic idea that unifies the class is deliberately poor oral bioavailability. A swallowed fraction that is barely absorbed, or is destroyed before it reaches the circulation, is what allows a potent glucocorticoid to be given daily for years. [1]
- 3. Because fluticasone propionate depends on one cytochrome pathway for clearance, its interaction profile is narrow but sharp, and it is shared by the nasal presentation as well as the inhaler. [1][4]
- 4. Device performance sits alongside these figures rather than beneath them. How much drug is actually delivered depends on technique, and a spacer is recommended by the NHS for anyone taking high steroid doses from a pressurised metered-dose inhaler. [8]

Adverse effects

- 1. Effect: Oropharyngeal candidiasis
 - 1. Detail: Localised infection of the mouth and pharynx with *Candida albicans* has occurred in patients treated with these inhalers, and candidiasis sits in the most common adverse reactions list of the fluticasone propionate aerosol label. It follows directly from depositing an immunosuppressive drug on the oropharyngeal mucosa on the way past. [1][2]
- 2. Effect: Dysphonia and throat irritation
 - 2. Detail: Hoarseness reflects a local effect of the steroid on the laryngeal muscles and mucosa in the path of the aerosol. The fluticasone propionate aerosol label lists dysphonia and throat irritation among the most common adverse reactions, alongside upper respiratory infection or inflammation, sinusitis, cough, bronchitis and headache. [1]
- 3. Effect: Local nasal effects of the intranasal route
 - 3. Detail: The nasal sprays trade thrush and hoarseness for mucosal injury. Epistaxis, nasal ulceration, *Candida albicans* infection, nasal septal perforation and impaired wound healing are grouped together in the fluticasone nasal label as the local nasal effects to expect and to look for. [4]
- 1. Effect: Hypercorticism and adrenal suppression
 - 1. Detail: Both labels reviewed here carry the same sentence: it is possible that systemic corticosteroid effects such as hypercorticism and adrenal suppression, including adrenal crisis, may appear in a small number of patients. Deaths from adrenal insufficiency have occurred in asthmatic patients during and after transfer from systemic corticosteroids to an inhaled agent.
 - 1. Action: The risk tracks dose and duration; recognition of adrenal insufficiency and any adjustment of therapy rest with the treating clinician. [1][3]
- 2. Effect: Reduced growth velocity in children
 - 2. Detail: Orally inhaled corticosteroids may cause a reduction in growth velocity in paediatric patients. In the studies cited by the fluticasone propionate aerosol label the mean reduction was approximately one centimetre per year, ranging from 0.3 to 1.8 centimetres per year, and it appears to depend upon dose and duration of exposure. Intranasal corticosteroids carry the same warning.
 - 2. Action: Growth is monitored routinely, for example by stadiometry, throughout treatment; interpretation and any

change of therapy belong to the treating clinician. [1][3][4]

3. Effect: Reduced bone mineral density

3. Detail: Decreases in bone mineral density have been observed with long-term administration of products containing inhaled corticosteroids. This is a duration-dependent effect rather than an acute one, and it matters most for patients kept on higher strengths for years.

3. Action: Relevant to anyone with additional risk factors for osteoporosis; assessment of bone health rests with the treating clinician. [1][3]

4. Effect: Glaucoma, raised intraocular pressure and cataract

4. Detail: Glaucoma, increased intraocular pressure, blurred vision and cataracts have been reported following long-term administration of inhaled corticosteroids, and the fluticasone nasal label states that intranasal and inhaled use may result in the development of glaucoma or cataracts. The beclometasone nasal label records the same events as rare after intranasal use.

4. Action: Close monitoring is warranted, and the labels direct that patients with a change in vision are considered for ophthalmological review. [1][3][4][6]

5. Effect: Pneumonia in chronic obstructive pulmonary disease

5. Detail: This signal comes from COPD populations rather than from asthma labels. A case-control study cited by UK regulators found that current use of an inhaled corticosteroid was associated with a significant increase of 70 per cent in the frequency of hospitalisation for pneumonia, with the risk dose-dependent. Clinicians are advised to remain vigilant for pneumonia and other lower respiratory tract infection in COPD patients treated with inhaled medicines containing a steroid, because the clinical features of such infections and of exacerbations frequently overlap.

5. Action: Regulators advise that any patient with severe COPD who has had pneumonia during such treatment should have their treatment reconsidered. [7]

6. Effect: Failure of asthma control mistaken for a tolerable symptom

6. Detail: Because the class does not act quickly, deteriorating control can be masked by increasing reliever use rather than being recognised. The fluticasone label warns against inhaling more doses or using the inhaler more often than prescribed, and against stopping it on the grounds of feeling better.

6. Action: Worsening symptoms or rising reliever use warrant medical reassessment rather than self-adjustment of the preventer. [1]

1. Drug: Fluticasone propionate

1. Effect: The narrowest interaction profile problem in the class, because clearance runs through CYP3A4 alone; postmarketing reports with ritonavir describe systemic corticosteroid effects including Cushing's syndrome and adrenal suppression. [4][1]

2. Drug: Beclometasone dipropionate, inhaled

2. Effect: Its most common adverse reactions are listed as oral candidiasis, upper respiratory tract infection, nasopharyngitis, allergic rhinitis, oropharyngeal pain and sinusitis; dysphonia does not appear in that list. [3]

3. Drug: Beclometasone dipropionate, intranasal

3. Effect: Rare instances of nasal septum perforation have been reported spontaneously, nosebleeds occurred in fewer than three per hundred patients, and localised *Candida albicans* infection of the nose and pharynx has occurred only rarely. [6]

4. Drug: Beclometasone dipropionate, intranasal

4. Effect: Rare instances of wheezing have been reported after intranasal use, alongside cataract, glaucoma and raised intraocular pressure. [6]

5. Drug: Fluticasone propionate, intranasal

5. Effect: Epistaxis was observed more frequently than with placebo in trials lasting from two to twenty-six weeks, and postmarketing cases of nasal septal perforation have been reported. [4]

Contraindications, precautions and interactions

1. Hypersensitivity to any ingredient of the product contraindicates it, as the fluticasone propionate nasal spray label states directly. [4]
2. These agents are excluded from the treatment of an acute asthma attack: the fluticasone propionate inhalation aerosol is not indicated for the relief of acute bronchospasm, and neither is the budesonide inhalation powder. [1][2]
1. A nasal corticosteroid is withheld until healing has occurred in patients who have had recent nasal septal ulcers, nasal surgery or nasal trauma, because corticosteroids inhibit wound healing. [6][4]
2. Transfer from an oral corticosteroid to an inhaled one is the moment of greatest hazard in this class, since the inhaled drug does not replace the systemic steroid the adrenal glands have stopped producing. [1]
3. Children on inhaled or intranasal corticosteroids have their growth followed routinely by stadiometry, and the over-the-counter nasal presentation advises that children use it for the shortest time needed to relieve symptoms. [3][5]
4. Existing glaucoma or cataract is a reason for particular care, and the over-the-counter nasal spray tells purchasers with either condition to consult a doctor before using it. [5][1]
5. In chronic obstructive pulmonary disease the pneumonia signal justifies a lower threshold for investigating new respiratory symptoms rather than assuming an exacerbation. [7]
1. With: Strong CYP3A4 inhibitors, including ritonavir and ketoconazole
1. Effect: Use with fluticasone propionate is not recommended, because increased systemic corticosteroid adverse effects may occur. Coadministration of ketoconazole produced a 1.9-fold increase in plasma fluticasone propionate exposure and a 45 per cent decrease in plasma cortisol exposure. [1][4]
2. With: Ritonavir specifically
2. Effect: Postmarketing reports describe clinically significant interactions in patients receiving fluticasone propionate products with ritonavir, resulting in systemic corticosteroid effects including Cushing's syndrome and adrenal suppression. The inhaler label says the combination is not recommended unless the potential benefit outweighs the risk of systemic corticosteroid side effects. [4][1]
3. With: Other strong CYP3A4 inhibitors named in the labels
3. Effect: Atazanavir, clarithromycin, indinavir, itraconazole, nefazodone, nelfinavir, saquinavir, telithromycin, conivaptan, lopinavir and voriconazole appear in the same list, so the problem is not confined to antiretrovirals and azole antifungals in the abstract. [4]
4. With: Systemic corticosteroids being withdrawn
4. Effect: Deaths due to adrenal insufficiency have occurred in asthmatic patients during and after transfer from systemic corticosteroids, so the two are not interchangeable on a milligram-for-milligram basis. [1]
5. With: Spacers and holding chambers as a device variable
5. Effect: Not a pharmacological interaction but a delivery one. A valved holding chamber with face mask may be used with the fluticasone aerosol in young patients, whereas the breath-actuated beclometasone inhaler must not be used with a spacer or volume holding chamber at all. [1][3]

Comparison tables

1. Title: Inhaled versus intranasal: what changes with the route
1. Caption: Same receptor, same systemic warnings, different local hazards and different indications. Choice of route and product belongs to the treating clinician.
1. Key: feature
1. Label: Feature
1. Is Row Header: true
2. Key: inhaled
2. Label: Orally inhaled
3. Key: nasal

3. Label: Intranasal

1. Feature: Licensed purpose

1. Inhaled: Maintenance treatment of asthma as prophylactic therapy

1. Nasal: Rhinitis, allergic and vasomotor, plus prevention of nasal polyp recurrence after surgery [1][6]

2. Feature: Time to full effect

2. Inhaled: Maximum benefit may take one to two weeks or longer

2. Nasal: Significant relief within three days for many, up to two weeks for some [1][6]

3. Feature: Characteristic local problem

3. Inhaled: Oropharyngeal candidiasis and dysphonia

3. Nasal: Epistaxis, nasal ulceration and rare septal perforation [1][4]

4. Feature: Local countermeasure in the label

4. Inhaled: Rinse the mouth with water without swallowing after each dose

4. Nasal: Withhold after nasal surgery, ulcer or trauma until healing is complete [3][6]

5. Feature: Growth in children

5. Inhaled: Reduction in growth velocity; routine stadiometry

5. Nasal: Same warning and the same monitoring instruction [3][4]

6. Feature: CYP3A4 interaction

6. Inhaled: Strong inhibitors not recommended with fluticasone propionate

6. Nasal: Identical restriction on the nasal presentation [1][4]

7. Feature: Device dependence

7. Inhaled: Delivered dose depends on technique; spacer advice differs by product

7. Nasal: Pump priming and aim matter; the spray is not a systemic depot [8][6]

2. Title: Preventer against reliever, side by side

2. Caption: The single distinction most worth getting right about respiratory medicines.

1. Key: question

1. Label: Question

1. Is Row Header: true

2. Key: ics

2. Label: Inhaled corticosteroid

3. Key: saba

3. Label: Short-acting bronchodilator

1. Question: What it does to the airway

1. Ics: Suppresses mucosal inflammation over days

1. Saba: Relaxes bronchial smooth muscle within minutes [2][1]

2. Question: Role during an attack

2. Ics: None; explicitly not indicated for relief of acute bronchospasm

2. Saba: The agent the label names as rescue medicine [1]

3. Question: How it is taken

3. Ics: Regularly, including on days without symptoms

3. Saba: When symptoms occur [8]

4. Question: Main long-term concern

4. Ics: Local candidiasis, and dose-related adrenal, skeletal and ocular effects

4. Saba: Rising use is a marker that control is being lost [1]

High-yield exam pearls

1. Pearl: A preventer inhaler has no place in the management of an acute attack, and the labels say so in the indication itself rather than burying it in a warning.

1. Why: Examiners test this because the error is clinically lethal, and because a patient reaching for the brown inhaler in a crisis is a recognisable scenario. [1][2]
2. Pearl: Rinsing the mouth with water without swallowing after inhalation exists to reduce oropharyngeal candidiasis, not for taste or hygiene.
2. Why: The mechanism, removing deposited steroid from the mucosa, is the answer being looked for, and it explains why a spacer helps for the same reason. [3][8]
3. Pearl: The growth effect in children is quantified: a mean reduction of about one centimetre per year, dependent upon dose and duration of exposure.
3. Why: It is the standard example of a systemic effect from a topical drug, and the number lets a candidate answer without either dismissing or exaggerating the risk. [1]
4. Pearl: Ketoconazole given with fluticasone propionate raised its plasma exposure 1.9-fold and cut plasma cortisol exposure by 45 per cent.
4. Why: It converts an abstract CYP3A4 warning into a measured effect on the adrenal axis, which is exactly how the interaction is usually asked about. [4]
5. Pearl: Nasal septal perforation and epistaxis belong to the intranasal route, not to the inhaled one, and recent nasal surgery or trauma defers treatment until healing.
5. Why: Route-specific adverse effects are a favourite discriminator between two otherwise identical corticosteroid presentations. [4][6]
6. Pearl: Beclometasone dipropionate is a prodrug hydrolysed to its active 17-monopropionate metabolite, with lung tissue doing much of the conversion.
6. Why: Prodrug status is easy to ask and easy to forget, and it distinguishes beclometasone from fluticasone, which is active as given. [3]
7. Pearl: The pneumonia signal in this class is a COPD finding, associated with a 70 per cent increase in hospitalisation for pneumonia and dose-dependent.
7. Why: Candidates often transplant it into asthma, where the asthma labels do not carry it; knowing which population it belongs to is the point. [7]

Common exam traps

1. Trap: Calling an inhaled corticosteroid a bronchodilator, or expecting it to abort wheeze.
1. Correction: It suppresses inflammation and does nothing to airway smooth muscle. The fluticasone label directs that a short-acting bronchodilator is always used during a sudden asthma attack. [1]
2. Trap: Concluding after a few days that a preventer has failed.
2. Correction: Improvement may begin within a day, but maximum benefit may not be reached for one to two weeks or longer, so a short trial proves nothing either way. [2]
3. Trap: Assuming a topical steroid cannot cause systemic corticosteroid effects.
3. Correction: Hypercorticism, adrenal suppression including adrenal crisis, falling bone mineral density, cataract and glaucoma are all in the labels, tied to higher doses and prolonged use. [3][1]
4. Trap: Treating spacer advice as universal across inhalers.
4. Correction: The fluticasone aerosol permits a valved holding chamber with a face mask in young patients, while the breath-actuated beclometasone product must not be used with a spacer or volume holding chamber. [1][3]
5. Trap: Believing an inhaled corticosteroid can safely replace an oral one dose for dose during a taper.
5. Correction: Deaths from adrenal insufficiency have occurred during and after transfer from systemic corticosteroids in asthmatic patients, which is why the changeover is a supervised process. [1]
6. Trap: Reading the CYP3A4 warning as theoretical.
6. Correction: Postmarketing reports of fluticasone propionate with ritonavir describe systemic corticosteroid effects including Cushing's syndrome and adrenal suppression, so the interaction has already produced clinical harm. [4]

Self-test questions

1. Question: A patient with asthma develops acute breathlessness and audible wheeze. Which statement about their inhaled corticosteroid is correct?

1. It has no role in relieving the acute episode and a short-acting bronchodilator is required
2. Doubling the number of puffs will produce bronchodilation within minutes
3. It relaxes airway smooth muscle faster than a beta-2 agonist
4. It is licensed specifically for the relief of acute bronchospasm

1. Explanation: The fluticasone propionate inhalation aerosol is not indicated for the relief of acute bronchospasm, and its patient information directs that a short-acting bronchodilator is always used as the rescue medicine during a sudden asthma attack. [1]

2. Question: Why do the labels for these inhalers direct that the mouth is rinsed with water without swallowing after each dose?

1. To help reduce the risk of oropharyngeal candidiasis
2. To prevent tooth enamel erosion from the propellant
3. To improve absorption of the drug from the lung
4. To avoid an unpleasant taste that reduces adherence

2. Explanation: The beclometasone dipropionate inhalation aerosol label gives that exact reason for the instruction, and localised *Candida albicans* infection of the mouth and pharynx is a recognised consequence of steroid deposited on the oropharyngeal mucosa. [3]

3. Question: A child on a long-term inhaled corticosteroid is reviewed in clinic. Which monitoring do the labels specifically direct?

1. Routine measurement of growth, for example by stadiometry
2. Annual bone marrow examination
3. Three-monthly serum potassium
4. Routine pulmonary function testing after each dose

3. Explanation: Orally inhaled corticosteroids may cause a reduction in growth velocity in paediatric patients, and the labels instruct that the growth of such patients is monitored routinely, with stadiometry named as the method. [3][1]

4. Question: A patient taking fluticasone propionate is started on ritonavir. What is the principal concern?

1. Raised systemic fluticasone exposure causing Cushing's syndrome and adrenal suppression
2. Loss of asthma control from accelerated fluticasone clearance
3. Precipitation of acute bronchospasm on first dose
4. Irreversible reduction in inhaler device output

4. Explanation: Ritonavir is a strong CYP3A4 inhibitor, and postmarketing reports describe clinically significant interactions with fluticasone propionate products resulting in systemic corticosteroid effects including Cushing's syndrome and adrenal suppression. [4]

5. Question: Which adverse effect is characteristic of the intranasal rather than the orally inhaled route?

1. Nasal septal perforation
2. Dysphonia
3. Oropharyngeal candidiasis
4. Cough on inhalation

5. Explanation: The fluticasone propionate nasal spray label groups epistaxis, nasal ulceration, *Candida albicans* infection, nasal septal perforation and impaired wound healing as local nasal effects, and postmarketing cases of septal perforation have been reported. [4]

6. Question: In which population have regulators specifically advised vigilance for pneumonia during treatment with inhaled medicines containing a steroid?

1. Patients with chronic obstructive pulmonary disease

2. Children with seasonal allergic rhinitis
3. Adults using a nasal spray for vasomotor rhinitis
4. Patients after surgical removal of nasal polyps
6. Explanation: UK regulators advise physicians to remain vigilant for pneumonia and other lower respiratory tract infection in COPD patients treated with inhaled drugs that contain steroids, because the clinical features of such infections and of exacerbations frequently overlap. [7]
7. Question: Which of these correctly describes beclometasone dipropionate?
 1. A prodrug rapidly hydrolysed to the active monoester 17-monopropionate
 2. An active drug cleared solely by renal excretion of unchanged parent
 3. A long-acting beta-2 agonist used for symptom relief
 4. A leukotriene receptor antagonist taken by mouth
7. Explanation: Its label states that beclometasone dipropionate is a prodrug rapidly activated by hydrolysis to the active monoester, with lung slices metabolising the parent compound rapidly, and excretion is mainly faecal. [3]

Frequently asked questions

1. Question: Why is an inhaled corticosteroid useless during an asthma attack?
 1. Answer: Because it works on inflammation, not on muscle. Relief in an attack requires the bronchial smooth muscle to relax, and no step in the corticosteroid mechanism does that. The effect of a preventer accumulates over days, so extra puffs during a crisis change nothing while delaying the bronchodilator that would help. The labels put this in the indication itself rather than leaving it to be inferred. [1][2]
 2. Question: How quickly does a preventer inhaler start to work?
 2. Answer: Some improvement can appear within a day of starting budesonide, but the labels are consistent that the full effect takes considerably longer, on the order of one to two weeks and sometimes more. Nasal sprays are quicker: significant relief within three days was reported for intranasal beclometasone, though some patients waited as long as a fortnight. [2][6]
 3. Question: Why does thrush happen, and does rinsing actually prevent it?
 3. Answer: Only a fraction of an inhaled dose reaches the lung; the rest is deposited in the mouth and throat, where a potent local immunosuppressant encourages *Candida albicans* to overgrow. Rinsing removes that deposit before it can act, and the beclometasone label states the instruction exists to help reduce the risk of oropharyngeal candidiasis. A spacer helps by the same logic, by capturing large particles that would otherwise strike the oropharynx. [3][8]
 4. Question: If the drug is topical, how can it suppress the adrenal glands?
 4. Answer: Topical does not mean unabsorbed. The majority of fluticasone propionate delivered to the lung is systemically absorbed, and that circulating fraction reaches the hypothalamic-pituitary-adrenal axis like any other glucocorticoid. The labels therefore state that hypercorticism and adrenal suppression may appear in a small number of patients, and tie the protective margin to not exceeding the recommended dosage. [1][2]
 5. Question: Do these medicines stunt children's growth?
 5. Answer: They can slow growth velocity, and the effect is measurable rather than hypothetical: a mean reduction of about one centimetre per year in the studies cited, with a range from 0.3 to 1.8 centimetres per year, depending upon dose and duration of exposure. That is why routine stadiometry is written into the labels for both the inhaled and the intranasal route. Balancing it against the risk of uncontrolled asthma is a judgement for the treating clinician. [1][4]
 6. Question: Which medicines make an inhaled corticosteroid dangerous?
 6. Answer: Strong inhibitors of CYP3A4. Fluticasone propionate is cleared through that single pathway, so ritonavir, ketoconazole and the other strong inhibitors named in its label raise systemic exposure sharply. Ketoconazole raised plasma exposure 1.9-fold while cutting plasma cortisol exposure by 45 per cent, and real cases of Cushing's syndrome and adrenal suppression have followed the ritonavir combination. [4][1]
 7. Question: Does the device really change the dose?
 7. Answer: It changes what is delivered, which is what matters. The fluticasone aerosol offers a valved holding chamber

with a face mask for young patients, whereas the breath-actuated beclometasone inhaler must not be used with a spacer at all. The NHS recommends a spacer for anyone taking high steroid doses from a pressurised metered-dose inhaler and always for children aged fifteen and under, partly to reduce steroid effects on the mouth and throat. [1][3][8]

8. Question: Is it safe to stop a preventer once the asthma feels settled?

8. Answer: Feeling well on treatment is the expected result of treatment, not evidence that it is no longer needed, and the NHS advice for beclometasone inhalers is to keep using them regularly even without symptoms. Stopping or changing a preventer is a decision for the treating clinician, who can step therapy down in a controlled way rather than removing it abruptly. [8][1]

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