THE DIAGNOSIS AND MANAGEMENT OF ALLERGIC RHINITIS: SUMMARY OF RECOMMENDATIONS BY THE SOUTH AFRICAN ALLERGIC RHINITIS WORKING GROUP (SAARWG) 2015

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On Behalf of the SAARWG, participating members in addition to authors

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ABSTRACT
Background: Allergic rhinitis (AR) is a common chronic condition which is often unsatisfactorily diagnosed or treated. Relevant allergens are area-specific hence the need for careful selection for relevance and cost-effectiveness. In this document, the South African Allergic Rhinitis Working Group (SAARWG) aims to address several diagnostic and therapeutic issues related to AR in the South African context.

Recommendations: The diagnosis of AR relies on a sound clinical assessment together with laboratory tests for allergic sensitisation. Tailored screening panels for relevant allergens in South Africa have been devised and are discussed. X-rays and CT scans are generally unnecessary in uncomplicated AR. Component testing can differentiate between allergen-specific and cross-reactive components and aid in the selection of suitable patients for immunotherapy.

Management: The management of AR entails allergen reduction, pharmacotherapy and immunotherapy. Intranasal corticosteroids are the pharmacological treatment of choice for AR and alleviate immediate as well as delayed symptoms. The role of second generation antihistamines and montelukast as add-on treatment for AR is discussed. First generation antihistamines are generally discouraged owing to their unfavourable side-effect profile. Systemic steroids and food restrictions are not recommended in the routine management of AR. Referral to an ear, nose and throat surgeon is recommended in treatment-resistant or complex cases to rule out anatomical or inflammatory complicating factors.

Immunotherapy is the only treatment modality which can alter the natural course of the disease, but is currently unlicensed in South Africa and supply issues remain a concern.
**1. INTRODUCTION**

Allergic rhinitis (AR) remains one of the most common and expensive chronic conditions in South Africa and internationally, but as yet remains largely underdiagnosed and inadequately treated.

Although AR per se is not a serious condition, its consequences can be serious, including exacerbation of asthma symptoms, increased susceptibility to viral illnesses, comorbidities such as rhinosinusitis and otitis media, disturbance in taste and smell, sleep deprivation and marked reduction in quality of life with reduced productivity at work and school.

In March 2015, the SAARWG met to discuss diagnostic and therapeutic issues related to AR, with the aim of improving overall diagnosis and management of this condition in the South African context. Despite diagnosing and treating patients according to best practice guidelines, both locally and internationally, a significant number of patients remain dissatisfied with clinical outcomes. This meeting aimed to explore this problem.

**2. CLINICAL DIAGNOSIS OF ALLERGIC RHINITIS, ASSESSMENT OF SEVERITY AND TREATMENT RESPONSE**

**2.1 CLINICAL DIAGNOSIS OF ALLERGIC RHINITIS**

The diagnosis of AR relies chiefly on a sound clinical assessment together with laboratory tests for allergic sensitisation. The clinical assessment should include a thorough history, taking into account frequency, duration, seasonality and severity of symptoms. In South Africa, the ARIA (Allergic Rhinitis and its Impact on Asthma) guidelines are widely applied, despite a recognition of some of its weaknesses. These guidelines divide symptoms into intermittent or persistent and severity into mild, moderate or severe (Figure 1).

A clinical examination should assess for signs of atopy, nasal signs of inferior turbinate swelling/pallor and concomitant allergic diseases such as eczema and asthma. Comorbidities such as rhinosinusitis, otitis media and hearing loss should be assessed for. Equally important is examining for co-existing factors that can also cause nasal obstruction such as nasal polyposis, nasal septal deviation, other internal and external nasal deformities or midfacial hypoplasia. Such factors will be discussed further in Section 7. It is also important to note that nasal obstruction is a late chronic symptom in AR.

Plain film sinus X-rays have no place in the routine diagnosis of AR. Imaging in the form of CT scanning should be reserved for those with poor response to treatment or those with suspected chronic sinus disease and those in whom surgery may be contemplated.

**2.2 ASSESSING RESPONSE TO TREATMENT**

In assessing the response to treatment, assessments such as visual analogue scales or allergic rhinitis control tests could be applied. Ideal control tests need to be reproducible, quick and easy to perform in routine clinical practice and focus on the disease’s impact on everyday life.

The aim of control tests is to monitor treatment response, detect adverse effects and gauge the need to adjust treatment. Several control tests are available, including the “Control of Allergic Rhinitis and Asthma Test” (CARAT), “Rhinitis Control Assessment Test” (RCAT) and “Allergic Rhinitis Control Test” (ARCT). All are validated but have not been compared in head-to-head trials. The clinician should use the same validated control test consistently and regularly in a patient to monitor the AR.

Nasal patency is a complex clinical issue that can involve mucosal, structural and psychological factors. The perception of nasal obstruction is subjective and does not always correlate with nasal examination. Therefore, a number of more objective measures of nasal patency are available, including:

- Peak Nasal Inspiratory Flow Rate (PNIFR);
- Rhinomanometry;
- Acoustic Rhinometry;
- Optical Rhinometry;
- Allergen-specific Nasal Provocation Testing (ASNPT);
- Mucociliary clearance.

The above tests are generally not easily reproducible or widely applied.

**3. LABORATORY TESTING FOR ALLERGIC RHINITIS**

Careful history taking remains essential to the diagnosis of allergy and can identify specific triggers in the patient’s environment. However, in many patients there may be uncertainty about the allergen or triggers involved in an allergic reaction and a rational and cost-effective approach to laboratory allergy testing should be adopted.

**3.1 LABORATORY TESTS AVAILABLE FOR ALLERGEN DETECTION IN ALLERGIC RHINITIS**

Clinicians may use the following laboratory tests to assist in the diagnosis of allergic conditions:

- Skin prick testing (SPT);
- Specific IgE ELISA tests/“ImmunoCAP” tests (originally referred to as “RAST” testing or radioallergosorbent tests but this terminology is no longer used as the methods have changed to normal ELISA platforms);
- Component testing by specific IgE or microarray (see Section 4);
- Cellular allergen stimulation tests (CAST) for non IgE-mediated allergies (see Section 5).

Total IgE is not a relevant screening test for AR and its use is to be discouraged. The choice of SPT versus specific IgE (ImmunoCAP) tests depends on availability, cost and other factors such as concomitant medications. SPT are cost-effective and provide a point-of-care answer, yet need an antihistamine and steroid washout period and are operator- and technique-dependent. For this reason, SPT should be performed by trained clinical personnel.
or accredited laboratories in a standardised fashion after adequate antihistamine washout. Specific IgE by ImmunoCAP test are more expensive and results may take a few days or even weeks to be available, yet are standardised, do not require a medication washout period and may be added on to an existing sample within 48 hours if necessary. Both SPT and specific IgE, at cut-off levels of 3 mm and 0.35 kU/L respectively, are considered sensitive for allergy detection, but are not specific (specificity 50%), hence the relevance of low-positive results should be interpreted in the light of history of symptoms.

3.2 SCREENING PANELS FOR ALLERGIC RHINITIS

Previously published local South African allergy guidelines10-13 have emphasized the regional variability of prevailing aero-allergens and the cost effective use of blood tests, especially when looking for groups or mixes of allergens to assist clinicians and patients to reach a diagnosis and effective treatment as quickly as possible.

The traditionally used “Phadiatop®” blood test is a screening test for aeroallergens designed to differentiate between atopic and non-atopic patients by screening for IgE antibodies to common inhalant allergens. The Phadiatop® test is designed for international use and has not been modified to include the most relevant allergens in South Africa.

The South African Rhinitis Working Group (SAARWG) recognised the need to provide new updated panels of inhalant allergies in the South African setting and created a task force to do this. This task force, the Allergy Diagnostic Working Group (ADWG), consists of representatives from the Allergy Society of South Africa (ALLSA), the National Pathology Group (NPG) and the SAARWG. The taskforce set up a recommended basic screening panel, based on countrywide statistical analysis of laboratory test outcomes and available aeroallergen data.14 The basic screen should include:

- Bermuda grass;
- Rye grass;
- Dermatophagoides pteronyssinus;
- Blomia tropicais;
- Alternaria alternata;
- Cladosporium herbarum;
- Aspergillus fumigatus;
- Cat;
- Dog.

This approach should logically replace the Phadiatop®.

Further testing should be symptom-driven and based on specific geographical regions. For example, in the Western Cape, Epicoccum mould and the German cockroach should be considered and in KwaZulu-Natal the oriental cockroach. In the Highveld, Free State and Northwest regions consider adding maize pollen, eucalyptus and weed pollen mix (cosmos and khaki-bush). If patients have seasonal exacerbations during springtime, additional testing for tree pollen IgE may be requested (appropriate tree mix screen or the individual tree pollens allergens in the environment).

Food allergy testing should not routinely be performed for allergic rhinitis, unless the patient gives a history of acute reactions to foods involving multiple systems, not just nasal. The clinician should be alerted to the fact that false positive food allergy tests may occur secondary to cross-reactivity between various foods and aeroallergens (e.g. grass pollen can cross-react with wheat, soya or peanut).

3.3 RECENT ADVANCES IN LABORATORY TESTING FOR ALLERGIC RHINITIS AND RELATED CONDITIONS

3.3.1 South African Tree Pollen test

A cost effective tree pollen test strip has been developed by Lancet Laboratories in consultation with one of the leading allergy test manufacturers in Germany (Euroimmun). Advice on which South African trees should be included was obtained from clinical specialists and the SAARWG. One test blot or strip allows a screen of 18 different trees including alder, birch, oak, elm, olive, plane, willow, poplar, ash, white pine, eucalyptus, acacia (salinga), cypress, mulberry, lilac syringe, jacaranda, karee and stinkwood.

3.3.2 Mast Cell Tryptase

Tryptase is a protease released by mast cells and basophils during degranulation. Measuring acutely elevated levels of serum mast cell tryptase (in the active β-form) is diagnostic of anaphylaxis and can help distinguish it from other disorders with similar clinical symptoms.14,15

Typically, serum mast cell tryptase peaks 1-2 hours after

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Figure 1: ARIA Classification of allergic rhinitis
onset of symptoms and remains elevated for 4-6 hours but sometimes up to 12 hours after an episode. Serial levels should be performed. Mast cell tryptase is stable and may be ordered on stored blood to confirm the diagnosis of anaphylaxis.

3.3.3 Molecular Allergology
The role of molecular allergology/component-resolved diagnostics in AR is discussed in detail in Section 4.

4. ROLE OF COMPONENT TESTING IN ALLERGIC RHINITIS
Component tests are available in the form of ImmunoCAP tests for specific components - these are to be requested specifically - or as a microarray test such as the ISAC test (Immuno Solid Phase Allergen Chip test), which tests for 112 food and aeroallergen components simultaneously. These should not form part of screening panels. This testing should stay in the hands of the trained allergists and clinicians with the relevant expertise. Component testing can differentiate between allergen-specific components and cross-reactive components, which in the context of AR, can serve one of the following purposes:10,16,17

- Identify and manage cross-reactivity syndromes, e.g. pollen fruit syndrome;
- Identify true positive versus false positive food allergy tests in patients with aeroallergen sensitisation;
- Help in the selection of patients most likely to respond to immunotherapy.

These situations will be elaborated on below.

4.1 IDENTIFICATION OF CROSS-REACTIVITY SYNDROMES, E.G. POLLEN FRUIT SYNDROME16
Cross-reactivity may occur when a particular antigen causes an allergic reaction to an unrelated antigen because of amino acid sequence similarities. Patients with pollen allergies are often sensitised to cross-reactive components that occur in pollens as well as foods of plant origin. This can be clinically relevant. Such components include, in increasing order of allergenicity: CCD (cross-reactive carbohydrate determinants), profilins, PR-10 proteins and LTPs (lipid transfer proteins). These components are the cause of the oral allergy syndrome, otherwise known as pollen-fruit syndrome. In this syndrome, the patient usually experiences local oral symptoms upon ingestion of the food which has a cross-reactive component with a pollen, however, systemic symptoms may occur in some cases.

4.2 IDENTIFYING TRUE POSITIVE VERSUS FALSE POSITIVE FOOD ALLERGY TESTS IN PATIENTS WITH AEROALLERGEN SENSITISATION16
Certain foods such as peanut, soya and wheat can also share cross-reactive components with pollen allergens and thus come up as a false positive in food allergy testing in patient with AR. In this situation, a good history is critical. Tolerance on regular consumption of the food in question will exclude a true food allergy and the patient should be advised to continue eating the food despite a positive food-specific IgE test.

If there is uncertainty about tolerance to the food,
component testing may help, e.g. for peanut allergy, the storage components Ara h 1,2,3,6 are more likely to reflect a true allergy, whereas the cross-reactive components Ara h 5,8,9 may represent a false positive cross-reactivity. For soya, positive Gly m 5 and 6 are more likely to reflect true allergy than the component Gly m 4, a PR-10 protein. For wheat, omega-5 gliadin is most likely to reflect a true wheat allergy.

4.3 HELPING IN THE SELECTION OF PATIENTS MOST LIKELY TO RESPOND TO IMMUNOTHERAPY

Grass pollen immunotherapy: Specific markers indicate the likelihood of success of grass pollen immunotherapy:
- Cyn d 1 or grass group 1 allergen for Bermuda grass allergy;
- Phl p 1 or grass group 1 and Phl p 5 or grass group 5 for Timothy grass allergy. However, there is a lot of similarity between the group 1 and group 5 grass pollen allergen components in other grass species, especially those belonging to the Pooidae subfamily, e.g. Timothy and Rye grass. Patients sensitised to Phl p 1 only and not to any of the other allergen components in Timothy grass, are probably sensitised to Bermuda grass and/or maize pollen and not to Timothy or Rye grass.

Cat: Fel d 1, cat uertoglobin is a marker of primary sensitisation and predicts successful immunotherapy. Fel d 2 (cat serum albumin) is likely to cross-react with most other mammalian albumins and Fel d 4, a lipocalin, cross-reacts with horse, dog and cow. If a patient is Fel d 2 or 4 positive in the absence of Fel d 1 positivity, immunotherapy is not recommended.

Dog: Can f 1 and Can f 2 are lipocalins and are associated with primary sensitisation to dog. Can f 1 sensitisation predicts successful immunotherapy. Can f 5, a prostatic allergen secreted by male dogs only, is also an indicator of primary sensitizer to male dogs, but immunotherapy is not recommended in the absence of Can f 1 positivity.

Horse: Equ c 1, a horse lipocalin, is the major allergen in horse dander, but results should be interpreted in correlation with the clinical history, as there is some cross-reactivity with mouse and cat lipocalin.

House dust mite: Sensitisation to D pter 1 (cysteine protease) and D pter 2 (NPC protein) indicate true house dust mite sensitisation. D pter 10 (tropomyosin) cross-reacts with shellfish and other insects - immunotherapy is not recommended if D pter 10 is positive but D pter 1 and 2 are negative.

Blomia tropicalis does not cross-react significantly with other house dust mites, hence a separate B. Tropicalis vaccine is needed for Blomia tropicalis positive patients. The component Blo t 5 is currently only available on ISAC testing.

5. ROLE OF OTHER TESTS IN AEROALLERGEN SENSITISATION

The use of alternative basophil-activation based tests such as the CAST (Cellular Antigen Stimulation Test) may be considered for non IgE-mediated mechanisms of aeroallergen sensitisation. However, these tests need further validation on a larger scale level before they can be routinely recommended.

6. MANAGEMENT OF ALLERGIC RHINITIS

Management of AR consists of the 3 pillars of:
- Allergen reduction strategies;
- Pharmacological treatment:
  - Topical intranasal corticosteroids;
  - Antihistamines;
  - Leukotriene inhibitors;
  - Antibiotics;
  - Other.
- Immunotherapy.

Allergen reduction strategies have previously been described and are not further discussed in this document.

6.1 PHARMACOLOGICAL TREATMENT OF ALLERGIC RHINITIS

6.1.1. Intranasal corticosteroids

Intranasal corticosteroids are recommended as first line treatment for AR in both adults and children. This recommendation comes from the ARIA revised guidelines (2010) as well as more recent guidelines such as the American “Guidelines on the Management of Allergic Rhinitis” published in February 2015.

Intranasal corticosteroids are effective for both intermittent and persistent AR, as well as for other chronic nasal conditions. They have a profound effect on inflammatory response by suppressing many elements of the inflammatory cascade, reducing eosinophil infiltration and suppressing cytokine production. Their clinical efficacy begins after 1-3 days of use and peaks after 2-3 weeks. Intranasal corticosteroids inhibit both the immediate and delayed phases of the allergic response in AR. Placebo-controlled clinical trials demonstrate their effectiveness in the reduction of nasal symptoms including sneezing, itching, rhinorrhea and congestion, in both adults and children with AR. They are the treatment of choice in patients with any element of nasal congestion. Combination therapy is generally not much more effective than monotherapy with intranasal corticosteroids, but can be tried in patients with severe or persistent symptoms.

The efficacy of available intranasal corticosteroid molecules is generally comparable, with reduction in symptoms of 40-80% achieved in most clinical studies.

Intranasal corticosteroid molecules available in South
Africa include:

- Beclomethasone;
- Budesonide;
- Triamcinolone;
- Mometasone;
- Fluticasone propionate;
- Fluticasone furoate;
- Ciclesonide.

No single molecule has been consistently deemed better than another. All are consistently superior to placebo.\(^2^0\) Many clinicians and patients are reluctant to use intranasal steroids because of the concern of local and systemic side effects. The available molecules are all considered generally safe with minimal local side effects (e.g. stinging, nose bleeds occurring in 5-10% of patients) or systemic side effects.\(^2^1\) The more lipophilic the molecule, the more increased the local binding and reduced systemic availability is. Systemic bioavailability of swallowed topical intranasal steroid is low (<1%) for fluticasone, mometasone and ciclesonide and higher (30-40%) for beclomethasone and budesonide.\(^2^2\)

Suppression of the HPA axis is generally considered negligible with intranasal steroid sprays, although the molecule beclomethasone may have an effect on growth velocity.\(^2^2\) Safety aspects should be taken into consideration especially in children and in those on multiple sources of topical steroids (such as inhaled corticosteroids for asthma, topical corticosteroids for the skin) which may have a cumulative effect.

Cushing’s syndrome has been described with the chronic use of beclomethasone drops.\(^2^3\) The long-term use of intranasal beclomethasone, particularly in the drop form with its higher nasal and pharyngeal absorption and bioavailability, should be discouraged.

Higher dose intranasal corticosteroid “nasules” with extremely low reported bioavailability, e.g. fluticasone, may be used where rhinitis is difficult to control and anatomical issues have been excluded.

6.1.2 Antihistamines

Histamine is pivotal in the allergic response and antihistamines act as inverse agonists to dampen the effects of released histamine.\(^2^4\)

The first generation antihistamines (chlorphenamine, promethazine, hydroxyzine) have poor receptor selectivity and a high propensity towards central nervous system side effects. They have no place in routine management of AR.\(^2^4,2^5\) Recently their anti-muscarinic effect has been identified as a factor accelerating cognitive decline in Alzheimer’s disease.\(^2^6\)

The second generation antihistamines have fewer side effects, better receptor selectivity and no tachyphylaxis and are the antihistamines of choice in the management of AR.\(^2^7,2^8\) Antihistamines are particularly useful when pruritis, sneezing and rhinorhoea are predominant symptoms. Generally, the reduction in symptoms achieved by antihistamines for congestion (nasal obstruction) is significantly less than that achieved by intranasal corticosteroids.\(^2^9\)

Antihistamines can be used as add-on treatment to intranasal corticosteroids to aid in the pruritis and rhinorrhoea control.\(^3^0\) Antihistamines may be needed as a first line treatment in certain situations such as when there is reluctance to take intranasal corticosteroids (e.g. in children), predominant itch/sneeze symptoms and also as rescue therapy when symptoms are severe. Their onset of action is generally fast (between 1-3 hours), however, maximal effect is reached only after 2 weeks of continuous use.

Most second generation antihistamines have a prolonged action and need once daily dosing. The exception is in young children <6 years age, who have faster elimination and may need twice daily dosing for certain antihistamines such as cetirizine and levocetirizine.\(^3^1\)

Available second generation antihistamines in South Africa are:

- Cetirizine;
- Levocetirizine;
- Loratadine;
- Desloratadine;
- Fexofenadine;
- Rupatadine.

Efficacy studies show a clear benefit over placebo for all of the above antihistamines. Currently, no one antihistamine has been found to be consistently more efficacious than another.\(^3^2\) Rupatadine, a molecule newly available in SA, has a dual antihistamine as well as antiplatelet activating factor (anti-PAF) effect. A theoretical advantage is reduced nasal congestion but in clinical practice this has not been proven as superior to other antihistamines.\(^3^3\)

If the patient has an inadequate response to a particular antihistamine, another second generation antihistamine may be tried.

Choice of antihistamine should be affected by:\(^2^7,2^8,3^2\)

- Side effects profile: In the second generation antihistamines up to 30% of drug may pass through the blood brain barrier, so somnolence may still be an issue. This is greater for cetirizine and lower for fexofenadine. Patient variability is high with regards to central nervous system side effects.
- Metabolic interactions: Loratadine, desloratadine and rupatadine are metabolised in the liver utilising the CYP450 enzyme system and are thus more prone to
drug interactions with enzyme inhibitors. Fexofenadine can be bound by aluminium and magnesium-containing antacids.

- Cost and reimbursement.
- Special situations: All of the above-listed antihistamines are licensed for use in children in South Africa,34,36 some for younger children (e.g. from 6 months onwards for fexofenadine) and some currently for older children (e.g. over 12 years for rupatadine).
- In pregnancy, cetirizine, loratadine and levocetirizine are considered safe but more studies are required. Fexofenadine and desloratadine have been shown to cause reduction in size in animal foetuses, hence, are currently not recommended during pregnancy.

Antihistamines do not prevent asthma in children with AR and/or eczema. Antihistamines should not be used in the treatment of viral upper respiratory tract infections. The SA Allergic Rhinitis Working Group particularly discourages the use of older generation antihistamines in combination with oral corticosteroids or systemic decongestants for the treatment of “colds.” These carry a serious risk of side effects.

6.1.3 Montelukast
The leukotriene receptor antagonists are licensed in South Africa for the use of AR in patients with concomitant asthma. In general, montelukast is better than placebo but not as effective as intranasal corticosteroids or antihistamines.36,37 Therefore, they should not be used as first line agents for AR, or as mono-therapy. They do play a potential role as an add-on therapy in patients with predominant symptoms of nasal congestion, with some studies showing better improvement in night-time symptoms and congestion compared with antihistamines.38-41 In this situation, they should be tried for periods of at least 4-6 weeks to assess their effect. Antihistamine plus montelukast combination is very costly with minimal benefit over the individual therapies.

Recent concerns have been published about central nervous system (e.g. sleep disturbance) and psychiatric side effects (e.g. depression) with montelukast in a small percentage of paediatric patients.35 This is rare but the clinician should be aware of such side effects. Cost-benefit ratio also needs to be taken into consideration.

6.1.4 The role of antibiotics in the management of allergic rhinitis and its complications

Uncomplicated AR does not benefit from antibiotic treatment. However, antibiotics are recommended for the co-morbidity of acute bacterial rhinosinusitis. In adults, antibiotic treatment is recommended for a minimum of 5 days and for children 7-10 days. In adults, the number needed to treat for clear benefit with antibiotics is 13 for acute rhinosinusitis. The diagnostic features and guidelines for antibiotics of choice for acute bacterial rhinosinusitis are depicted in Tables I-III.43

Antibiotics, specifically macrolides, can be used for their anti-inflammatory effect, anti-neutrophil effect as well as for promotion of mucociliary clearance in the following situations:44

### TABLE I: DIAGNOSTIC CRITERIA FOR ACUTE BACTERIAL RHINOSINUSITIS

| Anterior/Post nasal discharge . OR |
| Nasal obstruction ± |
| Facial pain/pressure ± |
| Change in sense of smell |
| Together with: |
| Severe lasting purulence or fever; |
| Worsening of symptoms (2nd sickening) starting usually within 10 days of onset of illness; |
| With symptoms lasting a total of >10 days and <3 months. |

### TABLE II: ANTIBIOTICS RECOMMENDATIONS FOR ADULTS WITH ACUTE BACTERIAL RHINOSINUSITIS

<table>
<thead>
<tr>
<th>IMMEDIATE CHOICE</th>
<th>FAILURE OF TREATMENT (AFTER 48-72 HOURS)</th>
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<tbody>
<tr>
<td><strong>RECOMMENDED DRUG OF CHOICE</strong></td>
<td><strong>ALTERNATIVE IF PENICILLIN ALLERGIC</strong></td>
</tr>
<tr>
<td>Amoxicillin (1 g twice daily for 5 days)</td>
<td>Telithromycin (800 mg daily) Gemiﬂoxacin (320 mg daily) Levoﬂoxacin (500 mg twice daily or 750 mg daily) Moxifloxacin (400 mg daily) ALL 5 DAYS</td>
</tr>
<tr>
<td>Amoxicillin-clavulanate (2g SR twice daily OR 1 g amoxicillin-clavulanate+1g amoxicillin BD) Cefuroxime (1 g twice daily) Cefpodoxime (400 mg twice daily) ALL FOR 5 DAYS</td>
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OR
- Recurrent upper respiratory infections (e.g. in those with immune deficiencies);
- Those at high risk of lower respiratory tract infections (e.g. cystic fibrosis, recurrent bronchitis);
- Chronic rhinosinusitis;
- Nasal polyps.

Dosing regimens in these situations are not standardised and the minimum duration of therapy should be 6 weeks. The impact of long term macrolide use on antibiotic resistance warrants careful monitoring.

6.1.5 Other treatment modalities in allergic rhinitis

Nasal douching or use of saline nasal sprays is encouraged in both AR and acute or chronic rhinosinusitis. Saline use is of particular benefit to rinse the nose before the use of intranasal corticosteroids.

Any form of systemic steroids or over-the-counter first generation antihistamine combinations is to be strongly discouraged in the routine use for AR treatment. Avoidance of certain foods such as wheat or dairy is generally discouraged for AR in the absence of other clear signs of food allergy involving other systems.

6.2 IMMUNOTHERAPY

Immunotherapy to aeroallergens is the only possible treatment with disease-altering effects in AR. Immunotherapy should be considered in patients who have an inadequate response to pharmacotherapy and avoidance techniques and in whom a thorough nasal examination has failed to reveal structural issues that could be hampering management. It is especially useful in mono-sensitised patients. Immunotherapy for aeroallergens is available in the subcutaneous and (increasingly popular) sublingual forms. Sublingual drops/sprays are currently not licensed in South Africa but available with special Medicines Control Council (MCC) approval.

Patients on immunotherapy should be monitored regularly for efficacy and side effects. Side effects are generally mild and localised and the treatment can usually be continued. However, recent local supply problems with sublingual immunotherapy as well as subcutaneous immunotherapy have hampered their use and threatened the continuity of treatment. The SAARWG endeavours to promote the continued availability of sublingual immunotherapy in South Africa. Of promise is oral immunotherapy tablets for grass and house dust mite. The ideal situation would be obtaining MCC licensing status in South Africa for immunotherapy, which will make immunotherapy much more readily available.

7. POSSIBLE REASONS FOR TREATMENT FAILURE IN ALLERGIC RHINITIS

Poor compliance, incorrect choice of medications, incorrect administration of medications, incorrect diagnosis or confounding diagnostic factors should be considered in poor treatment response in AR. A vital part of AR diagnosis and follow-up is a thorough nasal examination as well as examination of facial features and shape. Poor response to a good treatment regime should prompt referral to an ENT specialist for further evaluation and nasal endoscopic examination of the nose and adenoids. Lateral plain film X-rays of the neck are unreliable for nasal and adenoidal

<table>
<thead>
<tr>
<th>TABLE III: ANTIBIOTIC RECOMMENDATIONS IN CHILDREN WITH ACUTE BACTERIAL RHINOSINUSITIS (± ACUTE OTITIS MEDIA)</th>
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</thead>
<tbody>
<tr>
<td><strong>IMMEDIATE CHOICE</strong></td>
</tr>
<tr>
<td><strong>RECOMMENDED DRUG OF CHOICE</strong></td>
</tr>
<tr>
<td>Amoxicillin (80-90 mg/kg/day in 2 divided doses)</td>
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<tr>
<td>&lt;2 yr: 7-10 days</td>
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<tr>
<td>&gt;2 yr: 5-7 days</td>
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<tr>
<td>OR</td>
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<tr>
<td>Amoxicillin-clavulanate (90 mg/kg/day amoxicillin + 6.4 mg/kg/day clavulanate) in 2 divided doses</td>
</tr>
<tr>
<td>&lt;2 yr: 7-10 days</td>
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