



inflexibly. We would argue that the SSRIs do have an important role to play in psychiatric practice, including that of child and adolescent psychiatry, and that clinicians should, as always, balance the benefits and risks for any particular patient and keep the interests of the patient paramount. Obtaining an expert opinion from a child and adolescent psychiatrist would be useful in situations where a practitioner is unsure. We hope that the MCC will urgently reconsider the wording of this directive, in the interests of the many young patients who may otherwise be deprived of necessary and effective treatments.

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## Rocephin – the thin end of the wedge

**To the Editor:** I am sure I speak for many colleagues who have been cajoled, squeezed, begged and pressurised by medical aids, hospital administrators, pharmacists and representatives to stick to formularies to the point at which, backs against the wall, we say, 'No more!' The 'thin edge of the wedge' for me is the Rocephin issue.

I have grown up with Rocephin. For me, rightly or wrongly, Rocephin is the drug that diminishes my anxiety, oh so slightly, over the safety of my patients; that may prevent meningitis

secondary to sinus surgery; and that may stop a child's otorrhoea when other drugs have failed and prevent him or her from developing mastoiditis.

But despite evidence that many generics are inferior,<sup>1</sup> the pressure is on me not to use Rocephin, but to prescribe a generic, for the vast saving of about R5 per dose.

When will this pressure stop? If I surrender on the Rocephin issue, what comes next? Are the medical aid administrators, the hospital managers and the pharmaceutical buyers willing to share the medical risk that I face on a daily basis? Will they stand in the dock with me one day, and admit to using medications that are not proven to be equal in efficacy?

We hear so much about 'sharing risk' nowadays. The best way those pressurising me to use 'their' not 'my' choice of drugs can share risk, is by sharing my risk. What about, as a suggestion, paying all or part of my medical indemnity insurance?

I'm not looking for handouts. I'm not looking for perverse incentives. I don't even know, or care, whether the 'local' generic ceftriaxone is equivalent to Rocephin. For a saving of only R5 a dose? I learn from one generic manufacturer that their local factory manufacturing quality is excellent, and then I hear that the drug I am interested in is allegedly manufactured in Turkey, shipped to Germany, and then to South Africa where it is distributed.

For me there is a line I dare not cross, on the other side of which my autonomy stands for nothing. Where I still have a choice, I must fight to maintain it, lest that thin edge of the wedge be pushed in further and further, until it becomes a thick edge, and then a wall. 'Rocephin' for me is that issue, and I will not budge. It is time we as a collective organisation of medical professionals stand up and say to those who would manipulate us 'This far and no further'.

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## Problematic childhood atopic eczema consensus document?

**To the Editor:** The childhood atopic eczema consensus document published as part 2 of the June issue of the *SAMJ*<sup>1</sup> is problematic and necessitates the following comments.

A discussion of the controversy over the definition of atopic eczema is necessary, particularly in view of the recommendations that pertain to allergy testing. Without this the rest of the article is open to misinterpretation. When dermatologists speak of atopic eczema they mean a clinical diagnosis. As



mentioned by the authors, the diagnosis of atopic eczema according to the World Allergy Organization's Nomenclature Review Committee cannot be made without a positive immunoglobulin E (IgE) antibody or skin-prick test.<sup>2</sup>

While allergy specialists believe that specific IgE sensitisation (food and environment) is central to development of atopic eczema, the role of allergy in the pathogenesis of clinical atopic eczema is at best controversial. A systematic review of 65 studies,<sup>3</sup> where children with clinical features of atopic eczema were tested for serum IgE antibodies or had a skin-prick test, found a prevalence of 7 - 78% in population studies and 47 - 75% in hospital-based studies. This means that as few as 7% of clinical atopic eczema children could have positive allergy tests. Although dermatologists would diagnose all these children as having atopic eczema, allergologists would exclude those with negative IgE or skin-prick tests. Another important finding of this systematic review was that of the 8 studies that identified a link between atopy and atopic dermatitis severity, 7 studies concluded that the number of skin-prick tests or IgE antibody specificities were significantly associated with atopic eczema severity.<sup>3</sup> This would concur with current dermatology practice, to consider 'allergy' in severe (and non-responsive) disease.

When it comes specifically to food allergy, the document mentions only one reference that quotes 'up to 80% of infants

with atopic eczema will have positive food allergy tests', without mentioning other studies with lower prevalence figures. A comprehensive epidemiological study found that in infancy, 'As atopic dermatitis severity increased so did the prevalence of IgE-mediated food allergy (Group 0, 40/346 vs. Group 1, 6/36 vs. Group 2, 8/35 vs. Group 3, 12/35 vs. Group 4, 24/35).'<sup>4</sup> This was a birth cohort from 'atopic families' where group 0 had no atopic eczema and groups 1 - 4 had increasing disease severity. Therefore the prevalence of food allergies in that study increased from 11.5% (in the no eczema group) to 16.5%, 23%, 34% and 69% in those with more severe atopic eczema.<sup>4</sup>

As (clinical) atopic eczema is not severe in the majority of infants, the prevalence of food allergy would be expected to be much lower in most patients. In addition, the previously mentioned statement ('up to 80% of infants with atopic eczema will have positive food allergy tests') is not qualified by mentioning that at least in some patients the diagnosed food allergies do not correlate with the severity of atopic eczema, as evidenced by the variable response to exclusion diets. For example, in one study a diet that excluded eggs was found to improve atopic eczema in children who tested positive for egg allergy,<sup>5</sup> but in another study this benefit was only limited to those aged between 3 and 6 months.<sup>6</sup> In view of the above controversy it is worrying that the document goes

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on to state that 'testing for food allergy is an essential part of the management ...' This is indeed misleading and would not only have horrendous cost implications but also not improve the care of most of our patients. General practitioners and dermatologists treat the majority of 'clinical' atopic eczema patients who respond well to standard treatment.

What about the rest of the world? An American study<sup>7</sup> concluded that '... at present, there is scant evidence that allergy is central to the development of atopic dermatitis, although it may be an aggravating factor in a few patients. Hence there is little rationale for the routine use of allergy testing ... in the management of this disease.' The most recent guidelines, the Canadian Atopic Eczema Guidelines,<sup>8</sup> which use a clinical definition of atopic eczema, state: 'Some physicians send selected patients (i.e., those who fail to respond to standard therapy) for allergy testing to try to identify specific environmental or food allergies.' In a questionnaire-based study<sup>9</sup> investigators found that with effective topical treatment of atopic eczema 'Parental concern of food allergy decreased significantly from 7.7 to 4.0 on a 10 point scale ( $P < .001$ ).' The latter is a helpful study to bear in mind when dealing with parents who insist on allergy testing.

Finally, the take-home message for the management of clinical atopic eczema at any age should be to aim for adequate topical care, referring poor responders to or discussing such cases with dermatologists who have a wide armamentarium that includes in-hospital care, ultraviolet light and systemic treatment. It is only in a few, mainly younger patients with unresponsive disease, that allergy testing and exclusion diets may be helpful. There is no agreement between dermatologists and allergy specialists on the definition of atopic eczema, as evidenced by the latest guidelines.<sup>8</sup> Interdisciplinary discussions and such an agreement would help to reduce confusion.

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**P C Potter, A I Manjra, R Weiss, P du Plessis, N Rabobee, N Ndlova, M Davies and E Weinberg (Members of the SA Childhood Working Group of the Allergy Society of South Africa) reply:** We welcome the opportunity to respond to Dr Khumalo's letter. We emphasise that these are guidelines for paediatric eczema. Not all cases of eczema are 'atopic', and the term 'intrinsic' or 'constitutional dermatitis' was applied to the subgroup that has no IgE elevation. Those with IgE elevation are in the 'atopic' eczema group. Eczema has always been understood as being atopic or non-atopic.

The new nomenclature guidelines<sup>1</sup> state that although atopy can be suggested by a family history, or certain physical features, e.g. Dennies lines, associated rhinitis or asthma, it can only be confirmed by the documentation of positive specific IgE tests to environmental allergens. Atopy is therefore defined as '... a personal or familial tendency to produce IgE antibodies in response to low dose of allergens, usually proteins and to develop typical symptoms such as asthma, rhinoconjunctivitis or eczema/dermatitis'.<sup>1</sup> The name 'atopic' implies that the patient is sensitised to allergens that can result in exacerbation of symptoms and should therefore be avoided.

Some patients with eczema have significant food allergy. Several studies<sup>2</sup> have proved that food allergens exacerbate symptoms in children with atopic dermatitis. Implications of distinguishing atopic from non-atopic eczema include prevention of adverse reactions to food, preventing asthma development, indications for probiotics, and the prevention of eczema flares. These interventions require specific confirmation of elevation specific IgE in children with eczema. In a large study<sup>3</sup> of infants with eczema, asthma prevention of more than 50% was possible in those children sensitive to house dust mites and grass pollen.

Dr Khumalo addresses the question of prevalence of IgE sensitisation in patients with eczema, quoting studies of Flohr *et al.*<sup>4</sup> The low rate of 7% that he quotes was derived from unvalidated questionnaire data from Kota Kinabala, Borneo, Malaysia, where the overall percentage of allergy present in the community was estimated to be only 4% in the non-atopic dermatitis group.

In June 2005 data were presented at the World Allergy Organization Congress in Munich from the EPAAC study (WAO-EAACI congress proceedings) on allergy prevalence in infants with eczema. In this study from Europe, Australia and South Africa, 60% of 2 184 infants aged 1 - 2 years with mild to moderate eczema (atopic dermatitis) had an elevated IgE. In the South African subgroup of 161 children, 47% were sensitised to egg, 28.4% to milk, 26.8% to peanut and 39.9% to house dust mites.

Dr Khumalo is incorrect in stating that testing for food allergy would have 'horrendous cost implications' as skin tests for the 5 common allergens can be performed for R75 in private



practice (and at less cost for state patients), less than the cost of a small tube of most topical corticosteroids. Our eczema guidelines recommend testing for 4 - 5 food allergens and possibly for house dust mites using skin tests. RAST tests are inexpensive if one restricts them to the 4 - 5 relevant allergens (egg, milk, peanut, soya, wheat, and house dust mites), based on a history of the patient's diet and geographical location.

The role of IgE Fcε receptor, mast cell, basophil and T-cell responses in the pathogenesis of eczema is complex and in some cases depends on environmental exposure. The role of IgE in non-atopic dermatitis, which mainly involves a TH-1 response, is less clear.

One cannot call all cases of this heterogeneous disease atopic dermatitis without even doing allergy diagnostic tests. In citing the paper by Flohr *et al.*<sup>4</sup> Dr Khumalo did not refer to their conclusion, namely that 'continued use of the term atopic dermatitis is problematic'.

Dr Khumalo concurs that those eczema children who are 'poor responders' have a chance of up to 69% of being allergic, but in his 'take-home message' suggests that they should receive hospital care, ultraviolet light and systemic treatment! This contradicts his previous statement, as the cost of such treatment would be very expensive, whereas exclusion diets are free. These recommendations should not be considered before food allergies have been excluded. Patients who do not respond to simple conventional topical treatments should receive a panel of inexpensive allergy skin tests or selected RAST testing. They would also benefit from an assessment by a practitioner with an interest in allergy.

Dr Khumalo is incorrect in asserting that there is 'no agreement between dermatologists and allergy specialists on the definition of atopic eczema' as dermatologists and allergists have embraced the new nomenclature worldwide. His statement reflects the sentiments of a few old-school practitioners who continue with the incorrect use of the term 'atopic dermatitis' in eczema patients who have no evidence of atopy! This should be called 'non-atopic eczema'.

Having recognised the importance of allergy in children with eczema it is likely that working relationships between dermatologists and allergy specialists will continue to strengthen.

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## Minimal access or minimal invasive surgery

**To the Editor:** In an excellent paper at the 2005 Congress of the South African Society of Obstetricians and Gynaecologists, Dr E Rosemann stated that 'laparoscopy is the leading cause for legal action against gynaecologists in South Africa, and ... it is a growing industry'.<sup>1</sup> In my opinion, and also that of others,<sup>2</sup> a major reason is that laparoscopic surgery has been industry-driven, with the consequence that major procedures have been performed without proper prior research or training.

A second reason is that the terms 'minimal access' and 'minimal invasive surgery' are both used to indicate that an operation is associated with minimal trauma, and therefore minimal risk of complications and early discharge from hospital. This concept is wrong and misleading, since minimal access does not necessarily imply that minimal invasive surgery will be performed. The term minimal access surgery means that small incisions are used to gain access to the abdomen or pelvis, usually for diagnostic laparoscopy or laparoscopic surgery. This term can also be used for mini-laparotomy. The term minimal invasive surgery means that a minor operative procedure has been performed inside the abdomen or pelvis, e.g. a partial salpingectomy for an early tubal pregnancy.

However, minimal access (or minimal entrance) can also be used for maximal invasive surgery, e.g. radical hysterectomy and lymphadenectomy for cervical cancer. This requires a highly trained and skilled laparoscopic surgeon, or the operation will be fraught with danger.

In conclusion, the terms minimal access and minimal invasive surgery are not synonymous. This implies that even where a minimal access route of entrance to the abdomen or pelvis is used, the degree of invasion of the surgery to be performed will determine the degree of surgical expertise required. The type of surgery should be based on the findings of well-designed prospective clinical trials. In the absence of such trials, the safest and most beneficial approach should be used. An example is early ovarian cancer, where the role of laparoscopy is undefined, and laparotomy is still the gold-standard surgical procedure for diagnosis and staging.<sup>3</sup>

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