



IV phenobarbitone shock

To the Editor: It has been brought to the attention of the Executive Committee of the South African Paediatric Association that the intravenous form of phenobarbitone is no longer available in South Africa.

In August 2004 Aventis informed all provincial authorities that the worldwide production of sodium gardenal would be stopped and that it would no longer be available once stocks had been depleted.

This is a matter of great concern in terms of treating children in South Africa, especially those who present with epilepsy, in particular status epilepticus. In general, as far as developing countries are concerned, the action of Aventis cannot be defended. It would have been far better had they made sure that alternative arrangements were available in Africa before unilaterally withdrawing sodium gardenal.

Intravenous phenobarbitone has proved to be highly effective, it is safe and cheap, it can be given in repeated doses by rapid push-in, and it is currently recommended on all the international APLS guidelines for the treatment of status epilepticus. We have been informed that intravenous phenytoin or lorazepam are proposed alternatives. These drugs would not pose a problem in tertiary settings, but at primary and secondary level intravenous phenobarbitone is easy to administer with relatively few complications, and needs to be available.

The decision by the World Health Organization (WHO) to remove intravenous phenobarbitone extensively without consultation in developing countries, especially in Africa, is also disconcerting. It is strongly advised that this matter be reconsidered and that dialogue be initiated with the WHO on this issue.

Phenytoin and lorazepam have been suggested as alternatives. Intravenous phenytoin has to be administered over a long period of time via a syringe driver and requires an intravenous line, which may not always be possible in rural settings. Cardiac monitoring is recommended because of cardiac arrhythmias. It cannot be repeated once given and it may not be as affective as phenobarbitone.

Lorazepam, on the other hand, is dangerous as a follow-up after 2 doses of short-acting benzodiazepine because respiratory depression is very likely. Again, this would be a problem in primary and secondary settings where there are no facilities to ventilate children. It is also markedly expensive compared with intravenous phenobarbitone.

It is therefore clear that intravenous phenobarbitone remains the mainstay of first-line treatment for status epilepticus, especially in the primary and secondary health care settings, where the majority of children in South Africa are managed. Phenobarbitone is still manufactured by alternative companies internationally and we would support efforts to have these

products registered and distributed in South Africa as soon as possible.

Currently intravenous phenobarbitone is available as a Section 21 medication, but this is not effective or useful for the future use of intravenous phenobarbitone for the children at risk.

We urge the Department of Health to take cognisance of the problem, and we would support any initiative from the Central Department of Health to address this medical crisis in the management of status epilepticus in children.

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'Found guilty' – an unjust outcome?

To the Editor: A well-respected surgical colleague was recently found guilty by the Health Professions Council of South Africa (HPCSA) on 5 of 8 charges after complications arose from a laparoscopic procedure for gastric reflux. Sentence was delivered on Friday 14 October, where he was cautioned and discharged.

As an anaesthesiologist I have witnessed many of these procedures by a wide variety of surgeons and my comments are based on personal experience. Looking at those who made up the bench for this hearing (a general practitioner, a community medicine doctor and a retired surgeon), I'm surprised that they did not include a surgeon *actively* involved in this type of surgery.

Together with all my colleagues currently engaged in laparoscopic surgery in Cape Town, I am devastated by the outcome of the hearing. Knowing what happened, and the steps taken to manage events, we can only assume that inexperienced people are, unfairly to themselves, being appointed to sit at these hearings.

The complications that arose in this case are well known to those involved in laparoscopic surgery. There is nothing disgraceful about a wrong clinical decision ... it is human. The unfortunate surgeon, who is highly experienced in laparoscopic surgery and well respected by colleagues, both academic and private, acted in the best interests of the patient. He sought advice and the problem was eventually resolved. The patient had a traumatic postoperative course but fortunately survived the ordeal and I believe is now fit and healthy. I have a sneaking suspicion that this case represents an attack on laparoscopic surgery by those who seem to have very little insight into the specialty.

A surgeon's decision may not always be correct, but to be found guilty of unprofessional conduct and to be accused of belated surgical action, failing to recognise the clinical course



in the postoperative period and bringing the profession of specialist surgeon into disrepute is provocative and laughable. I'm surprised that this hearing favoured the 'expertise' presented by a retired surgical academic, who by his own admission had done very little laparoscopic surgery, over that of a professor and a specialist intensivist currently at the top of their careers. Surely experienced medical personnel should be appointed to hear public grievances? Those of us who are members of the Medical Protection Society (MPS) are concerned that in a case like this the legal representatives failed dismally. This case should have won hands down.

There is a perception among the lay public and litigation lawyers that as most of us have some form of medical protection there's no harm in 'having a go'! MPS reports suggest that medicolegal claims in South Africa have escalated way above rates in the rest of the world. It is my impression that we in clinical medicine are seen as an easily milked cash cow. We are under continual pressure from medical aids, hospital groups and the media – and now our very own HPCSA.

I sincerely hope that the colleague in question has the stamina to exercise his rights and appeal against the findings of the HPCSA, and that his surgical association reacts strongly to this disgraceful decision.

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Well done, SAMA's Industrial Relations Unit!

To the Editor: It is reassuring to know that the South African Medical Association, through its Industrial Relations Unit, has the capacity to assist doctors, especially hospital doctors, should any have reason to believe that they have been subjected to unfair labour practices.

My own experience is that about 3 years after retirement I was phoned by the hospital concerned and told that I had received a salary increase some 2 or 3 years before retirement for which I had not been paid. I was told that if I supplied my bank details I would be paid. Having heard nothing for a year I made further enquiries, only to be told that the provincial health department concern had no money.

I had no recourse other than through the SAMA Industrial Labour Unit, which was entirely successful in obtaining my back pay.

I don't hesitate to recommend to all doctors that they should become SAMA members, for this and many other reasons!

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Selective serotonin reuptake inhibitors in children and adolescents

To the Editor: The introduction of the selective serotonin reuptake inhibitors (SSRIs) was widely viewed as an important advance in clinical psychopharmacology, not only because of their broad-spectrum efficacy but also because of their tolerability and safety advantages, particularly compared with the older tricyclic antidepressants (TCAs) and monoamine oxidase inhibitors (MAOIs). Subsequently there has been considerable controversy about this class of agents, partly because of concerns about the extent to which they have been injudiciously prescribed for 'cosmetic' problems rather than for genuine psychopathology,¹ and partly because of concerns regarding their adverse effects. Most recently, attention has been paid to the appropriate use of SSRIs in children and adolescents.

The 'Drug Alert' published by the National Adverse Drug Event Monitoring Centre in the September 2005 SAMJ² is singularly unhelpful in this regard. The report takes a far more conservative stance than that taken by regulators in the USA, the UK and the EU; it may be misleading by implication and omission; and (if followed to the letter) it may cause child and adolescent psychiatric patients significant harm.

The 'Drug Alert' warns practitioners on four points. First, 'None of the SSRIs are currently approved in South Africa for any indication in children and adolescents.' It should be pointed out, however, that fluoxetine is registered with the US Food and Drug Administration (FDA) for child and adolescent depression and several of the SSRIs (fluvoxamine, sertraline, and fluoxetine) are also FDA-registered for child and adolescent obsessive-compulsive disorder (OCD).³ Practitioners should also be aware that decisions about whether to submit pharmaceutical agents to the Medicines Control Council for registration of particular indications may often be made on the basis of cost rather than scientific or clinical considerations.

Second, 'SSRIs have been associated with an increase in the risk of suicidal thinking and behaviour (suicidality) in children and adolescents with MDD [major depressive disorder] and other psychiatric disorders.' However, as the drug alert also states, 'no suicides occurred' in the 24 trials involving over 4 400 patients. In addition, a systematic review⁴ published recently found no significant difference in the risk of suicide in patients taking SSRIs compared with those taking TCAs. As several commentators have pointed out, patients with overt suicidal ideation are excluded from clinical trials and the heterogeneous nature of the trial designs employed (use of different definitions and assessments of self-harm in different study populations) further contributes to the difficulty of interpreting the data. The trials quoted were not designed to address the question of whether SSRIs increase suicidal ideation, and cannot in fact do so.⁵



Third, products ‘containing citalopram, escitalopram, fluvoxamine, paroxetine, sertraline and venlafaxine are contraindicated in children under 18 years of age’. While many regulatory authorities have cautioned practitioners about possible adverse events of these agents, so far none have taken so inflexible an approach as to say that they are all ‘contraindicated’ in children and adolescents. The European Medicines Agency in the same press release referred to by the MCC concedes that: ‘It is recognised that a doctor may sometimes take a decision based on the individual clinical needs of a child or an adolescent to use these products for the treatment of depression or anxiety. The CHMP [Committee for Medicinal Products for Human Use] is recommending that in these cases patients be monitored carefully for the appearance of suicidal behaviour, self-harm or hostility, particularly at the beginning of treatment.’⁶

It must be remembered that TCAs are not effective in child and adolescent depression, while SSRIs have been shown to be effective (on their own and in combination with cognitive behavioural therapy) for child and adolescent depression, and as a sole intervention in some childhood anxiety disorders, especially OCD.⁷⁻¹⁰ Fluoxetine, the only SSRI not on the MCC’s ‘contraindicated’ list, is not always tolerated by younger patients, especially those who have severe anxiety, and is also not available in any form except 20 mg capsules in most of the state sector, making appropriate dosing difficult. The effect

of implementing the MCC recommendations as published would be to deprive child and adolescent patients with these disorders of available, effective and potentially life-saving alternative medications.

Depression is a common, disabling, and potentially fatal disorder that is substantially and unequivocally associated with suicide and deliberate self-harm in children and adolescents. A large recent Swedish forensic study¹¹ showed that only 13% and 4% respectively of children and adolescents who actually committed suicide had received any antidepressant medication at all; youth suicide is far more likely to occur due to untreated depression than the adverse effects of any antidepressant. It should also be noted that since the advent of the SSRIs in 1988, suicide rates have decreased significantly in many countries.^{12,13}

Fourth, ‘Discontinuation of SSRIs, especially abrupt discontinuation, commonly leads to significant withdrawal symptoms.’ While it is true that discontinuation symptoms can be seen with some SSRIs after abrupt discontinuation, this warning runs the risk of conflating SSRIs, which are not associated with dependence, with medication classes such as the benzodiazepines, where dependence is an acknowledged risk.

In view of the above points we urge practitioners to note that these warnings from the MCC do not seem to reflect a balanced view of current scientific thinking, and may have significant adverse consequences for patients if implemented



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,¹ 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*¹ 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.²

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,³ 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.³ 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).'⁴ 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.⁴

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,⁵ but in another study this benefit was only limited to those aged between 3 and 6 months.⁶ In view of the above controversy it is worrying that the document goes



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 study⁷ 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,⁸ 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 study⁹ 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.⁸ 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 guidelines¹ state that although atopy can be suggested by a family history, or certain physical features, e.g. Dennie's 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'.¹ 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 studies² 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 study³ 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.*⁴ 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.*⁴ 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'.¹ In my opinion, and also that of others,² 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.³

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