enrichment. This is an important point, because there are suggestions that CHWs should become more formally integrated into the health system, possibly as enrolled nurses. Turning CHWs into community nurses would be a retrograde step not in line with the true concept and ideals of primary health care.

In our health wards we have come to appreciate the value of our motivated and energetic CHWs more and more. They are an inspiring group of women to work with. Community clinic nurses state that CHWs are more effective at delivering basic health education than they are, and clinics operating in areas without CHWs continually ask us when the CHW programme will be extended into their area. Their role in promoting healthy sexual behaviour, family planning and safe motherhood, preventing malnutrition and encouraging rational health seeking behaviour is potentially massive.

Anyone who doubts the effectiveness, value and relevance of CHWs to South Africa need only spend some time with them in our communities.

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Dr N. C. Dlamini Zuma, Minister of Health, comments: Dr McCoy *et al.* allege that I indicated that 'community health worker (CHW) programmes may be phased out, or at best, not further expanded'. This is not a true reflection of what I said.

Firstly I never addressed the topic of CHWs in my speech, but twice during meetings in Natal I was asked questions on CHWs after finishing it. Secondly I did not say that CHW programmes must be phased out or not further expanded. What I did say (and the audience agreed with this) was that we cannot build a health care system on CHWs. The basis on which to build the health care system is a clinic staffed by health professionals. In South Africa these are usually nurses, because so few of our primary health care facilities have full-time medical practitioners. Once we have the health professionals in place we can look at CHW programmes. I accept the complementary role CHWs can play in health care.

I fully agree with the authors that under no circumstances should CHWs become enrolled nurses. This will certainly erode their unique place in the health care team. Furthermore, CHWs function at a local or provincial level. It would be a mistake if the National Department were to try to be prescriptive in this regard. Defining the role of CHWs will therefore be a local and definitely not a national decision.

Recurrent herpes zoster and high-dose inhaled steroids for asthma

To the Editor: A 31-year-old woman who used beclomethasone 2 000 µg/d to control her severe asthma had 4 episodes of herpes zoster over a period of 18 months, all over the same dermatomal distribution at the right buttock. These episodes were not associated with booster doses of oral steroids. Investigations found no other causes of immune suppression. Her serum cortisol level was normal, as was urinary cortisol over a 24-hour period, thus excluding adrenal insufficiency.

On enquiring of the Medicines Control Council whether this is a recognised side-effect of high-dose inhaled steroids (it is well known to be a complication of parenteral or oral steroid use), we were told that no case has been reported so far.

We consider that there is a definite association between this patient's high-dose inhaled steroid use and her herpes zoster and that this should perhaps be included in a list of possible side-effects of high-dose inhaled steroid therapy, and have forwarded a copy of this letter to the Medicines Control Council.

The patient is now on high-dose inhaled fluticasone.

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Steroid-induced osteoporosis

To the Editor: Kalla *et al.*[1] recently published their work on the effect of glucocorticoid (GC) therapy on the bone mass of patients with rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE). They made the interesting observation that cortical bone mass was significantly lower in patients with RA than in those with SLE, although the latter required larger cumulative doses of GC for longer periods.

Individual sensitivity of skeletal tissue to GC is well known, and a number of 'risk factors' for steroid-induced osteoporosis have been suggested, including a high total cumulative steroid dose, long duration of GC therapy, age under 15 or over 50 years, postmenopausal status, general risk factors for osteoporosis, and disorders associated with increased interleukin-1, interleukin-6 or tumour necrosis factor (TNF) production, e.g. rheumatoid arthritis.[1-5] Kalla *et al.* suggest that their observation that patients with SLE are protected against the development of steroid-induced osteoporosis may be explained in part by the recent demonstration that patients with SLE have lower circulating levels of TNF — an interesting hypothesis deserving of further study.

The authors also conclude that the influence of GC therapy on bone is controversial and that bone loss in RA and SLE is more likely to represent an effect of the underlying disease than a complication of chronic GC therapy. Whereas an individual sensitivity to steroids as well as a primary disease effect on bone have been aptly documented, recent reports indicate that there can be no doubt as to the potential deleterious effects of long-term GC

therapy on skeletal integrity.[1-12] Moreover, it is now firmly established that GC, like numerous other causes of osteoporosis including early post-menopausal (type 1) osteoporosis, predominantly involve trabecular bone, with sparing of the cortical bone of the metacarpals and to a lesser degree the femoral neck. This may relate, at least in part, to the fact that trabecular bone is 8 times more metabolically active than cortical bone, and hence more prone to injury. Older techniques, including radiogrammetry, employed in the present study to measure appendicular bone mass, have therefore often underestimated steroid-induced bone loss.

Employing dual energy X-ray absorptiometry (DXA) to measure axial bone mass in 111 atopic asthmatic patients (60 on GC; 51 non-GC), we have also recently documented significant osteopenia, predominantly involving the spine, in 48% of steroid-treated subjects.[11] No correlation existed between bone mass and steroid dose/duration of therapy, gender, menopausal status, presence of conventional risk factors, cushingoid side-effects or serum biochemical measurements including cortisol binding globulin levels. In fact only basal and $1,25(OH)_2$ vitamin D-stimulated osteocalcin kinetics differed between steroid-treated patients with a normal and those with a decreased bone mass, suggesting individual sensitivity to steroids at an osteoblast level.

It is therefore fair to conclude that long-term GC therapy should be regarded as a common and important cause of osteoporosis. The development of bone loss is not necessarily dose-dependent or readily predictable; even low-dose GC therapy in RA has recently been shown to result in a 3-fold higher fracture rate than in RA patients not receiving GC.[12] Whereas patients who need it should certainly not be deprived of GC therapy, awareness that it may cause bone loss and early prophylaxis are required.

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Dr Kalla, Professor Meyers and Dr Laubscher reply: It was not our intention to encourage a cavalier approach to the use of GC therapy, but rather to report the interesting findings we observed in our study of two disease populations. We believe the following points need to be made in response to the above letter.

1. We agree that cortical bone may differ from trabecular bone in the manifestation of osteopenia owing to differences in their metabolic activities. However, some studies show equal effects of GC on cortical and trabecular bone.[1] It is also interesting that some diseases, like RA, may cause severe cortical bone loss with less impressive trabecular bone loss.[2] In the experimental situation, osteoclast and osteoblast responses are identical when subjected to local and systemic influences, irrespective of their origin.

2. The studies quoted above need to be interpreted with caution, since a large proportion of GC patients have been postmenopausal. The effects of GC seem to be worse in postmenopausal subjects (oestrogen deficiency), and it is not always clear how much is the additional effect of GC. Guyatt *et al.*[3] have elaborated the many problems with earlier studies, some of which have been addressed in subsequent research.

3. The author refers to experience with DXA in GC-dependent asthma patients whose bone mineral density (BMD) was significantly reduced. He does not provide information regarding menopausal status, physical activity or severity of the asthma, all of which could independently contribute to bone loss. We consider that it is extremely difficult to find adequate controls for this kind of research, since GC-treated diseases could interfere with BMD by several mechanisms. While asthma, SLE and RA may have in common the need for GC therapy, they differ significantly from each other in many other respects; in particular, cytokine activity is different.[4] Our selection of RA and SLE for comparison was based on their common auto-immune background and their wide difference in GC dosage requirements. They clearly differ significantly in the extent of disability they cause. It is interesting that Professor Hough was unable to show any relationship in asthma between GC daily dose or duration of cumulative therapy.

4. We are aware of the controversies relating to the effects of GC on cortical bone, and have extended our studies to include trabecular BMD measurement in SLE.[5] Again we were unable to show any significant effect on lumbar or femoral BMD in premenopausal SLE patients receiving high-dose (1 mg/kg/d in reducing doses for more than 6 months) GC therapy. The data showed that the underlying disease was likely to be the major cause of bone loss. Similar results in SLE have been reported by others.[6] It is possible that GC is simply a surrogate measure of severe disease rather than a pathogenetic factor in bone loss.

5. We would also caution against assuming that statistically significant differences necessarily translate to clinically significant differences. Sambrook *et al.*[7] are the only workers to have shown a prophylactic effect of calcitriol in GC osteoporosis.[7] Other work on postmenopausal subjects failed to show a beneficial effect of vitamin D and calcium supplementation,[8] but antiresorptive agents may be of some use. The use of deflazacort as a bone-sparing GC is encouraging, but controversy rages about the equivalent anti-inflammatory effect.[9] We would certainly encourage prophylactic therapy aimed at reducing fracture complications of osteoporosis.

6. We would argue, therefore, that it is *not* fair to conclude that GC causes bone loss under all circumstances. Unfortunately, experimental conditions are never able to

simulate the *in vivo* milieu in its entirety. Therefore, such results should be interpreted with caution. It is ironical that bone loss is thought to be due to osteoclast activating factors which are negatively influenced by GC, suggesting, in theory, that GC should protect against bone loss due to cytokine activity.

In conclusion, we agree that diligent awareness and early prophylaxis against bone loss are essential when initiating treatment with GC. Perhaps DXA measurement of BMD should be the reference standard for such decisions, particularly since rheumatic diseases may influence many of the metabolic markers of bone and cartilage metabolism.[10]

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Electrotherapy for snakebite

To the Editor: In 1988 the index catalogue of the library of the Surgeon General's office (US Army) listed over 500 references on the use of electrotherapy for bites and stings.

Guderian *et al.*[1] in a letter to the *Lancet* (1986) presented 34 cases of Ecuadorian snakebite. Twenty-five patients were treated within 30 minutes of the bite by high-voltage (25 mV), low-amperage (less than 1 mA) direct current, applied for 1 - 2 seconds at 5 - 10-second intervals for 4 - 5 shocks. Such a current is found in a modified stun gun or a spark plug of an outboard motor, lawnmower or auxiliary lighting plant.

Within 10 - 15 minutes there was no pain, no local or systemic poisoning and no morbidity or mortality. Most patients went home an hour later. Seven patients refused treatment. All developed clinical poisoning and 2 required amputations. A further 2 with established envenomation were treated 2 hours after the event. Pain relief occurred in 30 minutes, and swelling did not progress and subsided within 3 days.

These are very impressive results. Adverse comments would include unknown age and gender of patients and species of snake; lack of proper controls; natural selection of patients (the very sick could not get to hospital); and a highly selective group of patients, since snakebite is so common, causing high immunity in the community.[2] A possible mechanism of action was postulated. Venom has a short half-life, and electrospasm of local vessels contained the venom long enough for it to be degraded and hence inactive. However, Christensen[3] in 1955 showed that both wet and dry South African snake venom *in vitro* had an extremely long half-life, measured in months.

In vivo the half-life is much shorter. Venom action continues for 3 - 4 days if swelling is the clinical presentation, and for several days in Cape cobra (*Naja nivea*) and boomslang (*Dispholidus typus*) bites.

Reitz *et al.*[4] showed in 1987 that a similar electric current had no effect on morbidity and mortality in rats injected with pretreated or untreated venom of *N. haje* (Egyptian cobra) or *N. mossambica* (spitting cobra) compared with controls. No change in the composition of the venoms could be detected by electrophoresis after exposure to the current *in vitro*.

Likewise, Howe *et al.*[5] injected *Bothrops atrox* (Ecuadorian pit viper) venom subcutaneously in increasing doses into rats, half of which received electrotherapy. There was no difference in morbidity and mortality between the two groups.

Rats are not human, but it is unlikely that electrotherapy will be found more beneficial than placebo in the management of snakebite.

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Centre for behavioural sciences founded at the University of the Orange Free State

To the Editor: Although the Departments of Psychiatry and Psychology at the University of the Orange Free State have always set an example with their outstanding working relationship, it was felt that this collaboration should be formalised in order to increase research output and in the process also improve training. It was assumed that, since the two sciences have so much in common, not only the output but also the quality of research could be increased by founding a research centre. The final decision to go ahead with a Centre for Behavioural Sciences (a nationally and internationally recognised concept) was triggered by the Department of Psychiatry's prominent role in community service, which underlined the urgent need for research in this regard. Discussions between members of the Department of Psychology and community leaders emphasised this need.

In October 1993 the Centre for Behavioural Sciences — which can be regarded as a research bridge between the Departments of Psychiatry and Psychology — was finally approved by the authorities of the University. The Executive Committee consists of Professor C. A. Gagiano, head of the Department of Psychiatry, and Professors S. J. Wessels and D. A. Louw from the Department of Psychology. Professor Louw will be head of the Centre.

Since the main focus of the Centre will be on executing high-quality research in communities, several potential projects in this regard have already been identified. At present the Centre is conducting an epidemiological study (using the new DSM-IV) and a situation analysis in