S. pneumoniae is an uncommon, but potentially serious neonatal pathogen. Earlier literature stressed early-onset disease with S. pneumoniae, similar to neonatal group B streptococcal infections. Most cases of early-onset disease are associated with prematurity and prolonged rupture of membranes. Infection is presumed to be acquired from the mother intra partum, either transplacentally or via ascending infection, as is likely in both the cases reported here, given the rapidity of onset of disease and that both mothers were clinically well. While S. pneumoniae is not considered to be part of the normal vaginal flora, transient pelvic colonisation can occur. Pneumococcal pelvic infections may occur in association with surgery, foreign bodies or delivery, and mothers may have invasive pneumoccal disease, e.g. meningitis.

Late-onset pneumococcal disease among neonates has been reported. In a study of *S. pneumoniae* infections in the neonate (SPIN) the mean age of onset was 18.1 days and 90% of the infants were term babies (≥ 38 weeks' gestation). The source of acquisition of pneumococci in late-onset infections is not clear. Vertical transmission from the mother and horizontal transmission from siblings and other adults may occur.

Because of difficulties in diagnosis, the true incidence of *S. pneumoniae* infection in neonates is unknown. In developing countries *S. pneumoniae* probably causes about 25% of neonatal pneumonia and this may be becoming more frequent, particularly with HIV co-infection.

Neonatal diagnosis of *S. pneumoniae* sepsis has had limited significance since the infection would be adequately treated by empiric regimens for neonatal sepsis including penicillin. This may change with the increasing prevalence of non-susceptibility of *S. pneumoniae* to penicillin, particularly where meningitis cannot be excluded. In these settings a third-generation cephalosporin (such as cefotaxime or ceftriaxone) would be more appropriate. If resistance to the third-generation cephalosporins is prevalent, vancomycin may be indicated.

C Bamford A Whitelaw S Haffejee

Department of Medical Microbiology Groote Schuur Hospital/National Health Laboratory Service Cape Town cbamford@sun.ac.za

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Educating bonesetters

To the Editor: We appreciate the noble work of Dr Onuminya in his community.¹ However, the attempt to bridge the gap between orthodox and traditional medicine is not new. Shah *et al.*² undertook training programmes for rural health practitioners in Nepal. At evaluation after 6 years they found significant improvements in the knowledge base and working skills of these practitioners after undergoing training programmes. In a 2-year prospective study, Eshete³ found a reduction in amputation rates after a one-day instructional course offered to bonesetters in Ethiopia.

Because of local high patronage it seems that the traditional system of bone setting is here to stay. The above pilot reports¹⁻³ indicate that it is possible to educate bone setters and reduce morbidity. Because of widespread prevalence of bonesetters in developing countries (in India it is estimated that there are 70 000 traditional healers and bonesetters who treat 60% of all trauma patients), a national initiative is required to include them in the mainstream health care systems of developing countries. These bonesetters often work in remote places and villages where there are no trained doctors. With some basic education and training in the field of orthopaedic care they can become a most effective vehicle for patient care and referral.

Anil Agarwal Manoj Kumar Goyal

Department of Orthopaedics UCMS and GTB Hospital Shahdara Delhi India rachna.anila@yahoo.co.in

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Does access to better housing affect personal quality of life and well-being?

To the Editor: Since 1994 a concerted effort has been underway to improve the living conditions of all South Africans through providing/upgrading housing. Although over 1 million houses had been delivered or were under construction by the end of 2000, there is a paucity of information on the short- and long-term effects of housing delivery on quality of life and wellbeing. A study was therefore conducted involving 334 adult residents of an informal settlement in Soweto in 1999 (before relocation to a new housing estate or site tenure allocated), and in 2001 and 2003 (after relocation). Comparisons were made

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between three groups, namely new housing estate residents (N = 73), site tenure residents (N = 158), and a group living in informal housing (N = 103).

From the repeated measures analyses, estimated marginal means showed significant group and time effects for overall quality of life (Fig. 1). Although there were no differences between new housing estate and site tenure residents with regard to quality of life (p > 0.05), both these groups had a better quality of life than informal housing residents (p < 0.05). This finding was not unexpected as previous research has shown that quality of life is very poor in the latter.³ In 2001, quality of life was significantly better than in 1999 (p < 0.01), but returned to similar levels to 1999 in 2003 (p > 0.05). It is possible that changes in living circumstances in 2001 were responsible for this effect. A more likely explanation is provided by the classic study of the effects of fortune and misfortune on quality of life.⁴ The study found that lottery winners' quality of life shot up, while that of accident victims decreased dramatically immediately after the event, only to return to previous levels at a later date.4

There were no significant group or time effects (p > 0.05) for wellbeing (Fig. 1). This lack of group or time effects provides support for the model of homeostatic wellbeing. ⁵ Analogous to the homeostatic maintenance of blood pressure and temperature, this model considers that wellbeing is actively controlled and maintained by psychological factors that function under the influence of personality. ⁵

Overall findings revealed that access to better housing positively affects quality of life; there is habituation of quality of life in the long term, as people adapt to both positive and negative

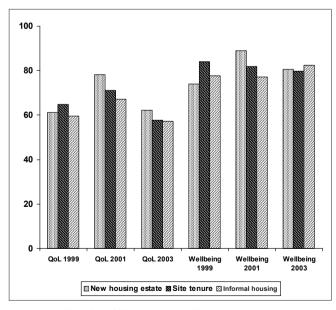


Fig. 1. Overall quality of life (QoL) and wellbeing in 1999, 2001 and 2003 among new housing estate, site tenure and informal housing residents.

life events; and, irrespective of living conditions, wellbeing is maintained at a homeostatic level.

Margaret S Westaway

Health and Development Research Group Medical Research Council and School of Health Systems and Public Health University of Pretoria mwestawa@mrc.ac.za

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Post-exposure prophylaxis for rape survivors

To the Editor: In a recent incident in the Eastern Cape a cocaine addict was arrested on drug possession charges and put in a holding cell. Sadly, during his time in the cells he was raped. Naturally the victim was very concerned about the possibility of having contracted HIV. However, the attending district surgeon refused to provide antiretrovirals (ARVs) as part of post-exposure prohylaxis (PEP). SANCA drug counsellors were faced with this distressed and traumatised victim when he turned to them for help after his release and were quite perplexed by the failure of the district surgeon to prescribe PEP for 28 days.

Why were the ARVs not provided? One possibility is that there may have been a supposition of potential poor medication adherence. Predicting a person's adherence based on speculation would seem to be unfair. Is there in fact proof that substance dependence causes poor adherence to ARV medication? The evidence is mixed, with a review of the issue showing that 11 of 26 studies found no association with substance abuse.¹

The second possibility is that there may have been a concern around the pharmacological interaction between cocaine and ARVs. One review on the issue does mention an increased risk of a cocaine overdose with antiretroviral therapy, citing that the administration of a potent CYP 3A4 inhibitor could result in a cocaine overdose. ARVs that induce CYP 3A4 activity, such as nevirapine, may shift the metabolism of cocaine from hydroxylation to *N*-demethylation and create a higher level of the potentially toxic metabolite.² However the article does not mention the likelihood of this event occurring, and nevirapine is in any case not used in any PEP regimens.

But are any of these issues relevant considerations in this case? Shouldn't all rape survivors have the right to ARVs,

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