Doctors and the medical aid industry

To the Editor: I respond to the comments of Dr Groenveld¹ from what is so unfortunately branded 'the other side'.

How sad that SAMA, the HPCSA, government, medical aids, and the public all get berated by the learned writer. How naïve (with respect) to maintain that in a R50 billion private health care industry, stakeholders as vital as doctors 'should not be required to be businessmen'. If you aren't, then don't be resentful if other stakeholders do treat it as a business.

This does not mean that the stakeholders should question each other's right to existence either, or treat health care as a zero sum game where we all fight for a bigger slice. We shouldn't aspire in South Africa to have the unregulated USA model which consumes 15% of GNP and still leaves 40 million uninsured, or the UK National Health where doctors are told exactly what to do.

So, we don't have a perfect system in South Africa, but let's at least move forward from adversarial finger-pointing, without hankering after an unrealistic utopia of unbridled freedom regardless of cost, which is not grounded in the reality of working South Africans.

Solution? Nothing simple — no right and wrong, but pragmatic stakeholders forging partnership relationships to work out a range of possibilities that give us greater access to

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Let us give credit to those pioneering funders and practitioners who have risked sacrificing the comfort of armchair critic status in favour of the partly successful but promising models upon which we can base our future successes. A decade after the early acrimonious engagements we now find clinicians on both sides of the table representing IPAs and managed care organisations — talks are still tough, but no longer as naïve, arrogant or petulant.

I am not sure that our health care system can afford anything other than all of us rolling up our sleeves and jointly evolving workable answers.

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 Groenveld PWL. Doctors and the medical aid industry (Briewe). S Afr Med J 2004; 94: 140-141

Infant feeding and prevention of mother-to-child HIV transmission

To the Editor: I fail to understand how your reviewer was unable to recognise the fairly obvious flaws in the paper by Hilderbrand *et al.*,¹ and did not grasp the seriousness and impact of such faulty data on public perceptions of infant feeding. This error of judgement is compounded by the *Journal*'s egregious running title on the cover stating: 'HIV — formula feeds increase child survival'. In fact the paper does not have child survival or mortality as an outcome, and you are merely trading on the terminology of the global revolution in child health promotion. This prominent display is both disingenuous and misleading.

I have criticisms of the methods employed to answer questions on infant feeding in HIV, and the authors' interpretation of their results. I will deal only with the latter. The single most important inference they draw from their findings is that formula feeding for infants of HIV-infected women is feasible and safe in urban environments with sufficient potable water.

However the evaluation of 'urban environments' is far more complicated than their methods allow,² and one may have rural settings where there is access to 'sufficient potable' water. Their data do not assist in deciding what is 'sufficient', so the key resource they identify is 'potable water'.

The Nairobi (urban) randomised controlled trial of breastversus formula-feeding in infants of HIV-infected mothers³ shows the weakness of their postulate. Mortality, and the incidence of diarrhoea, pneumonia and other illnesses were similar in both the breast- and formula-fed arms; nutrition was better in the breast-fed arm, significantly so in the first 6 months. The cardinal point to remember is that these similarities in outcome, and the nutritional benefit of breast-feeding, occurred after 24 months (median duration of breast-feeding 17 months), when the HIV infection rate in the breast-feeding arm was a whopping 37% compared with 21% in the formula arm. Accordingly breast-feeding over this prolonged period remained effective for these outcomes despite the very high HIV infection rate; the obvious answer is to reduce HIV transmission through breast-feeding while retaining its advantages! Six months provides adequate benefits of breast-feeding, and the transmission risk of HIV is at worst about 5% (the latter is from a meta-analysis of nine African trials involving 4 085 children). If our group's hypothesis is correct this figure may be even lower with exclusive breast-feeding.

What is critical to the thrust of this letter is that in the Nairobi trial all women 'had access to potable water, extensive health education regarding safe preparation of formula, a reliable supply of formula, and access to medical care for their infants'. So breast-feeding stood up to comparison with formula in a developing country setting, which is as good as it can get for minimising the disadvantages of formula.

There are other examples of breast-feeding in urban environments. In Durban and Harare (an extremely large trial) studies are detecting substantial benefits in children of HIV-infected mothers who breast-feed in preference to formula-feeding.

We have to promote solutions that are not abstracted from this continent's priorities and that respect the durability and strength of African traditions; breast-feeding is more than just about infant feeding, it affirms a wider public good.

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Auto-antibody testing in obstetric patients

To the Editor: We read with interest the article by Afman *et al.*¹ on the relationship of auto-antibodies and obstetric outcome in a tertiary high-risk obstetric unit. In our view the lack of an association between antinuclear antibodies (ANAs) and adverse pregnancy outcome may be due to a fundamental error in interpretation of the ANA results. The authors have grouped the 33 true ANA-positive patients with the 13 patients

who had only an anti-cytoplasmic pattern on indirect immunofluorescence (IIF) testing. The latter staining pattern is a 'by-product' of the IIF test using HEp-2 cells, and in this study is likely to be due to anti-parietal and anti-smooth muscle antibodies that were presumably confirmed on tissue substrate (method not given in paper). By definition, antibodies directed at cytoplasmic components cannot be considered to be ANAs. Hence, the comparison should have been between the 33 true ANA- positive patients and relevant control patients. A secondary issue is that negative IIF does not rule out the presence of anti-Ro antibodies even when HEp-2 cells are used as substrate. Anti-Ro antibodies are highly associated with the rare event of neonatal lupus and need to be sought by other methods if there is any suspicion of this condition.

Secondly, it would have been helpful to know the total number of patients in the two groups who were HIV-positive, and the frequencies of the respective auto-antibodies. We also note the finding that anticardiolipin (ACL) antibodies were more frequent in women with severe pre-eclampsia. In a previous study based on our own experience in a routine antenatal clinic, ACL antibodies were poorly predictive for pre-eclampsia, so the test may be more useful where there is a higher pretest risk of an associated event. Finally, we feel the title of the paper is not an accurate reflection of the study. ACL antibodies are not directed against nuclear antigens. 'Auto-antibody testing in obstetric patients' might be a more accurate title and a better reflection of the nature of the study.

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Pressure chamber explosion — Southern African Underwater and Hyperbaric Medical Association statement

To the Editor: Members of the Southern African Underwater and Hyperbaric Medical Association (SAUHMA), a special interest group of the South African Medical Association, were shocked to learn of the death of the Eloff brothers when a chamber they apparently built and used exploded, killing

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