Therefore, when Mbewu writes about 'black South Africans' it is not clear what he is referring to. However, despite the terminology Dr Mbewu has misunderstood our message. We do not say that Africans either alone or together with Indians are immune to atherosclerosis. Our paper[2] is in fact saying that atherosclerotic disease is well established among African patients (as well as in the other population groups). This finding is supported by other studies from our institution.[3,4] We also observed a difference in presentation and risk profile between Indian and white patients on the one hand and African patients on the other. However, for some reason that we cannot understand we do not see African patients with peripheral atherosclerosis presenting with ischaemic heart disease as an anaesthetic risk factor.

We have an established protocol for the pre-operative management of patients undergoing aortobifemoral bypass. A comprehensive history is always taken, all patients undergo an electrocardiogram, and those patients with a history of ischaemic heart disease or an abnormal electrocardiogram undergo a stress electrocardiogram. This protocol is well established and cost effective. We do not use the Minnesota classification and, furthermore, Seedat *et al.*[5] demonstrated that this classification was unreliable for eliciting ischaemic heart disease in Africans.

In our study[2] there was no history of ischaemic heart disease and no evidence of this condition on pre-operative electrocardiograms. None of our patients from all groups had morbidity related to ischaemic heart disease. Mbewu attributes our findings to limited access of 'black South Africans' to electrocardiography. This does not apply to our study because all patients were submitted to the same protocol in the same vascular unit by the same surgical team and all the patients had access to electrocardiography as part of pre-operative management strategy.

A point must be made, however, that we were looking at anaesthetic risk factors for this type of surgery — we were not looking for subtle signs and symptoms of ischaemic heart disease.

We do not dispute the findings of Mayosi *et al.* (as quoted by Mbewu[1]) who noted high prevalences of risk factors such as smoking, diabetes and hypertension in black South Africans admitted to a coronary care unit. Again, it is not clear from the correspondence by Mbewu whether that study included Indians and Africans or whether it looked at Africans only. The presence of risk factors such as smoking, diabetes and hypertension was also observed among African patients in our study, but despite that there was no history of ischaemic heart disease or abnormal electrocardiograms in African patients in our study. In the study by Seedat *et al.*[5] the prevalence of ischaemic heart disease in Africans was 2.4%. In that study 'black' denoted 'African'.

In conclusion, we agree with Dr Mbewu that further studies of ischaemic heart disease need to be done on black South Africans, with special emphasis on African patients, in order to look at the incidence of ischaemic heart disease.

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MORPHINE AND CHRONIC CANCER PAIN

To the Editor: Cancer pain can be broken down into the following categories: (*i*) pain caused by the cancer process itself, i.e. bony invasion or nerve compression or infiltration that is likely to present with the features of acute pain; (*ii*) pain arising from the treatment for the cancer process and likely to present as chronic pain — this might include pain resulting from radical surgery, chemotherapy or radiation therapy; and (*iii*) bedsores, chronic infection or constipation, which may become part of the pain syndrome.

Chronic pain is not easy to assess, but will affect attitude and lifestyle, will need long-term medication, and will involve many individuals besides the patient. Furthermore, expectations of the results of medication need to be realistic.

The International Association for the Study of Pain (IASP) created the first multi-axial system based on the region of the body involved in chronic pain, the organ system affected, the temporal characteristics and pattern of the pain, its duration and intensity and the source of pain, and a taxonomic classification has been made. Thoracic pain, for example, is '300' (region) and respiratory system pain is '20'.

All the above have been efforts to make pain relief as 'logical' as possible!

The management of pain is typically pyramidal: mild pain is treated with non-opioid analgesics (e.g. paracetamol or non-steroidal anti-inflammatory agents), moderate pain with opioid analgesics of weak to moderate strength (e.g. codeine), and severe pain is treated with strong opioid analgesics, e.g. morphine.

Morphine is the most frequently used of the strong opioid analgesics. For chronic pain management oral administration is

favoured, either as an elixir or an immediate-release or slow-release tablet. Usually the elixir is used to titrate the dose required for effective analgesia. Dosage intervals of 2 - 6 hours are needed (usually 4 hours) Once the correct (effective) dosage rate is obtained, the patient can be switched to the equivalent in controlled release form, if desired.

An article in *Time* magazine (28 April 1997, pp. 56 - 57) describes the 'mailbox effect' of morphine (morphine blocks the pain signals to the brain in the spinal cord). This article is well worth reading, as it makes a strong case for the chronic use of morphine in patients with chronic severe pain. It also warns about the prejudice of doctors who are not really familiar with the problems of chronic pain with regard to the prescription of morphine. A photo summarises the essential point: 'a patient finds relief — not addiction'.

Disadvantages of opioids are constipation (sometimes), nausea and vomiting, sedation, respiratory depression, tolerance and dependence. In my experience the latter is of little trouble in the terminally ill patient, and morphine does not really seem to be addictive when it is direly needed to keep pain under control.

Respiratory depression following opioid administration at the appropriate dose level is minimal in those patients who have pain.

Tricyclic antidepressants may further help and sometimes anticonvulsants like carbamazepine may be of further help in the patient with pain, and steroids can relieve nerve and bone pain.

The bottom line, however, is that we oncologists use litres and litres of the elixir, and we are extremely frustrated by the 'fear' of day clinic doctors, general practitioners, etc. to prescribe adequate quantities to terminal cancer patients with pain.

The usual elixir is 20 mg per 5 ml, and the lowest effective dose is usually 2.5 ml of this 4-hourly, which means a dose of 15 ml daily, or 500 ml monthly. This could easily reach levels 2 - 3 times higher than this! So to issue 50 ml or 100 ml is totally useless.

I wish this message could for once be understood!
(Don't forget the laxative with the morphine!)

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flatlining?