readers, we discover that 19 of the "normal" survivors had a reading age two years below average, a "finding consistent with their verbal IQ." If, just for the sake of argument, we include these 19 children in the minor handicap group, we reach a new—dare we say truer?—figure of 68 handicapped children among 131 survivors: a handicap rate of 51·1%.

Much as we enjoyed these authors' novel approach to the presentation of data, we feel impelled to offer their potential followers a more depressing analysis. On the basis of the results from Sutton-in-Ashfield, the denial of medical care to babies under 1500 g at birth is likely to result in death for more than half, with half of the survivors handicapped, one in seven severely so. Perhaps this paper should have carried a Government health warning?

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**We sent this letter to the authors, who reply below.—ED, *BMJ*.

SIR,—As indicated in our report of the Mansfield study, we used the same definitions (including those for handicap), standards, and assessment methods as those in the previously reported review of a community-derived sample of very low birth weight children born in Hammersmith Hospital.[1] Reasons for presenting handicap as a percentage of the total number of liveborn infants were clearly explained in that review. We merely followed the same enlightened reasoning and method of reporting.

Handicap has, of course, been defined differently elsewhere, in both narrower and broader terms, and reported also as a percentage of survivors examined.[2] In the narrower view of handicap, rates for the total Mansfield and Hammersmith community-derived samples are the same (18% of examined survivors). If we take the broader view, the handicap rate of 51·1% as computed for Mansfield's surviving very low-birthweight children falls between rates for other samples of very low-birthweight children reared by intensive care in Montreal (59·4%) and at the Hammersmith Hospital (46·7%). The outcome defined in these terms, however, has not yet been systematically examined in the entire community-derived Hammersmith sample born during 1961-75. Because of differences between communities in respect of social class and other factors, direct comparison of outcomes broadly defined has been difficult or inappropriate, and presumably will remain so whenever data include the incidence of learning difficulties and impaired attention span.

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[1] Jones RAK, Cummins M, Davies PA. *Lancet* 1979;i: 1332-5.
[2] Davies PA, Hull D, ed. In: *Recent advances in paediatrics*. No 5. London: Churchill Livingstone, 1976: 117-8.

Adverse interaction between nifedipine and beta-blockade

SIR,—With regard to the paper by Professor L H Opie and Dr Denise A White (29 November, p 1462) concerning the occasional severe hypotensive effect of nifedipine when given in conjunction with a beta-adrenergic blocking drug, we wish to report a case where this effect probably led to fatal myocardial infarction.

A man, aged 47, with longstanding hypertension, renal failure, and type IIB hyperlipidaemia had had five episodes of myocardial infarction between 1967 and 1978. In 1978 coronary angiography showed disease of three coronary arteries and a dyskinetic segment in the left ventricle. Operation was not advised, and he stayed at work with relatively few symptoms from angina taking propranolol 160 mg four times daily, isosorbide 10 mg four times daily, clofibrate 1 g four times daily, and Moduretic 1 tablet (amiloride 5 mg; hydrochlorathiazide 50 mg) in the morning.

In October 1980 he had further exacerbation of the angina; his blood pressure was 130/80 mm Hg, serum urea 11·9 mmol/l (71·5 mg/100 ml), and serum creatinine 189 μmol/l (2·1 mg/100 ml). In an attempt to improve his symptoms nifedipine 10 mg three times daily was substituted for the isosorbide, the other treatments being continued. Eighteen days later he was admitted to hospital with postural dizziness but no further chest pain. The blood pressure was initially unrecordable but two hours later was 60 mm Hg systolic, pulse rate 48/min, serum urea 30 mmol/l (180 mg/100 ml), and serum creatinine 366 μmol/l (4·2 mg/100 ml). An electrocardiograph was unchanged and the cardiac enzymes remained normal over the next three days. The nifedipine was stopped immediately and the propranolol on the next day as he remained severely hypotensive. Although oliguria was a feature for the first few days, his blood pressure and general condition slowly improved and on the fourth day after admission, three days after propranolol was stopped, the blood pressure was 100/40 mm Hg and serum creatinine 420 μmol/l (4·75 mg/100 ml) (from a peak of 495 (5·6 mg/100 ml). He then suffered further acute chest pain with cardiogenic shock from an undoubted myocardial infarction from which he ultimately died.

We feel sure that this severe hypotension, which was not readily reversible, was due to the nifedipine and that the consequent prolonged hypotension was the factor that led to the fatal infarction, although clearly his middle-term prognosis was very poor.

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Treatment of hypertension in black South Africans

SIR,—Professor Y K Seedat's article (8 November, p 1241) on the use of beta-blockers in hypertension in blacks has many clinical implications and perhaps some unjustified conclusions.

More black hypertensives seem to have low renin levels. This does not necessarily imply a different form of hypertension and the concept that this low-renin hypertensive group will benefit from diuretics rather than beta-blocker therapy has already been challenged and disproved time and again. The age of the group studied was not mentioned in the article, and this is of importance in connection with the duration of hypertension, which in turn has a bearing on the response to therapy.

My experience of the use of beta-blockers in blacks in this city (who are invariably West Indians and over 70% from Jamaica) is at variance with that reported in your article. My observations show a significant reduction not only in systolic and diastolic blood pressure at rest but also in systolic blood pressure during dynamic exercise. This is more pronounced following acute beta-blockade than in long-term administration.

This brings me to the assessment of compliance, which is understandably difficult to evaluate. The fact that your patients had a lower heart rate does not prove that they took their medication daily between visits—they may have done so for only one or two days before the visit, thus not getting optimal benefit in blood pressure reduction but a sufficient lowering in heart rate.

Although I have used a different beta-blocker, metoprolol, this is also cardioselective and has similar properties to atenolol, and there is no scientific reason why the response should be different. More relevant perhaps is the origin of our blacks—as West Indians their ancestors were mostly from the African West Coast and are therefore West African Negroes. Is hypertension in blacks really different? We still lack this basic information. Is the skin colour the only common factor among blacks, and are we correct in classifying them as one separate group?

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SIR,—I read with interest the clinical study performed by Professor Y K Seedat (8 November, p 1241). However, I find his conclusions somewhat misleading. While he highlights the lack of effect of atenolol, there was only brief mention of the lack of effect of chlorthalidone. Furthermore, there was no statistical difference between the blood pressures on each of the two drugs used separately. I feel that the conclusion should read that neither atenolol 100 mg daily alone nor chlorthalidone 25 mg daily alone should have been the baseline treatment of hypertension in his small group.

Further, he suggests that beta-blockers, not just atenolol, should not be regarded as the baseline treatment in blacks. This is really extending a very small study to an enormous population. He is assuming that all beta-blockers have effects similar to those of the low dose of atenolol that was used and further assumes that all blacks have physiological responses similar to those of his sample of 24 Zulus. These statements, taken out of context, are extremely misleading and I strongly feel that they should be withdrawn.

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SIR,—I wish to thank Dr S W P Mhlongo (6 December, p 1569) for his comments on my paper (8 November, p 1241). Dr Mhlongo has misconstrued my article as at no stage have I implied that race may be an important factor in an individual's response to beta-blocker therapy for hypertension. The mode of hypotensive action of beta-blocking agents is not known, and various theories have been postulated.[1] Thus the reason why beta-blocking agents do not act in black South Africans will only be understood once we know how beta-blocking agents produce a hypotensive effect.

The statement by Dr Mhlongo that "salt (NaCl) consumption among the Zulus appears empirically to be much higher than that of whites or Asians" is a clinical impression which I share. However, in the final analysis scientific data rather than clinical impressions are