

## GUIDELINES FOR CLINICAL PRACTICE BASED ON TRIAL RESULTS

The major clinical trials discussed in this review have not covered all the problems and possibilities of hypotensive treatment. Their results—though not those obtained by analysis of subgroups of patients defined after the trial was completed—must be used as guidelines for clinical practice. Bearing in mind the limitations of the trials, and also other published work, we suggest the following six guidelines.

Firstly, patients with malignant (accelerated) hypertension need treatment, but for the vast majority of patients who have non-malignant hypertension there is no "cut off" pressure above which treatment is mandatory. Adequate clinical trials have not been conducted in patients with diastolic pressures greater than 115 mm Hg, but such evidence as there is suggests that treatment is necessary when the diastolic pressure exceeds 115-120 mm Hg. There will be no appreciable benefit to an individual patient from treating a diastolic pressure of less than 100 mm Hg.

Secondly, there is no evidence that any particular level of systolic pressure should be treated, and thus there is no reason to treat patients with isolated systolic hypertension, however that might be defined.

Thirdly, blood pressure varies, and in an appreciable proportion of patients with diastolic pressures up to 115 mm Hg these will revert to normal values over a few months. It is prudent to check the blood pressure on several occasions before deciding whether to treat and also to measure it yearly in any patient found at any time to have a diastolic pressure greater than 100-105 mm Hg.

Fourthly, it is probably more important to stop a patient smoking than to treat his mildly raised blood pressure. Any pharmacological treatment causes side effects, so non-pharmacological methods should always be considered; thus weight control is essential as it may be sufficient to control raised pressure.

Fifthly there is no evidence that a different treatment policy is needed in any particular race, sex, or age group, although the effects of different treatments in very elderly patients have not been adequately studied.

Finally, there is no evidence that any particular form of drug treatment is superior to any other, except for a suggestion that  $\beta$  blockers are less effective in preventing the complications of high blood pressure in smokers. The choice of initial treatment therefore depends mainly on the expected side effects, which dictate patient compliance, and on cost. On all counts, initial drug treatment

should be bendrofluazide. A dose of 2.5 mg daily is as effective as higher doses and although this low dose has not been tested in a large scale trial designed to evaluate its effect on mortality, it would seem logical not to use the higher doses, which probably carry a higher risk of unwanted effects.

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*A 3 year old boy has been diagnosed as having hypertrophic muscular dystrophy. There is no family history and his maternal uncle is not afflicted. The mother's creatine phosphokinase is normal as is that of her sister. When the sister marries is there any possibility of her sons being afflicted?*

Between a half and two thirds of mothers of boys with hypertrophic (Duchenne) muscular dystrophy are carriers. Unfortunately, neither a normal creatinine phosphokinase in the mother nor in her sister excludes the carrier status. Three normal creatinine phosphokinases might, however, reduce the probability, and most genetic centres have a curve constructed from obligate carriers and a control population from which probabilities may be calculated. In addition an assessment of the pedigree, especially looking at the number of normal brothers and sons, will also help to alter the probability of being a carrier. Finally, new DNA probes are proving extremely useful in detecting carriers and a combined assessment using all three methods should be attempted at an appropriate genetic centre.—M BARAITSER, consultant in clinical genetics, London.

*What drugs and physical measures enhance the resolution of post-traumatic myositis ossificans?*

Regrettably there are no really effective measures known which will accomplish this. Traumatic myositis ossificans may follow rupture or bruising of, or frank haemorrhage into, muscle, or follow repetitive minor trauma, for example, in athletes. A similar clinical picture of ossification of muscle, and hence loss of mobility, may also occur in association with

paraplegia.<sup>1</sup> The only measure advocated recently is the diphosphonate etidronate, but results have been disappointing. Strenuous mobilising activity should be avoided lest more trauma, and hence more ossification, results. Large plaques of ossified tissue may be surgically removed when the condition is considered to be stable and not progressing, but the resultant surgical trauma may lead to further ossification. Irradiation has also been suggested in the early stages of the development of the myositis ossificans with unproved results.—C G BARNES, consultant rheumatologist, London.

1 Foster JB. Myositis ossificans. In: Walton J, ed. *Disorders of voluntary muscle*. 4th ed. Edinburgh: Churchill Livingstone, 1981:817-8.

## Correction

## Child sex rings

In the table of this paper by Wild and Wynne (19 July, p 184) the numbers of boys in rings 9, 10, and 11 have moved to the left. The table should read:

	Independent rings		
	9	10	11
Boys	?	0	4

We apologise for this error.