three months after myoblast transfer in 18 subjects showed an increase in 30 of the 69 muscle groups measured, no change in 26, and a reduction in 13. Interpreting these results is difficult given the uncontrolled nature of the study and the short period of follow up.

Blau and colleagues studied the tibialis anterior muscle from eight patients with Duchenne dystrophy, with a documented deletion in the dystrophin gene, one month after the injection of 100m cultured myoblasts into up to 100 injection sites.[10] Using the polymerase chain reaction they showed that in three patients dystrophin messenger RNA derived from the donor myoblast DNA was being expressed. Given the extreme sensitivity of this technique, this result presumably reflects the persistence of donor DNA from a few of the implanted myoblasts. The number of dystrophin positive fibres was only about 10 per 1000 fibres—no greater than the frequency of spontaneously occurring dystrophin positive "revertant" fibres in muscles on the control side. Despite being hailed by *Nature* as a transplant success it was an unequivocal clinical failure.

So where do we stand in relation to myoblast transfer in Duchenne dystrophy? Could the essentially negative results merely reflect the birth pangs reminiscent of earlier transplantation procedures such as kidney, bone marrow, and heart? Simple technical problems may explain the failure, which further studies could resolve. Alternatively, cell transfer may turn out to be a non-starter in the treatment of muscular dystrophy, and other experimental approaches, such as introducing gene constructs either directly into the muscle[11,12] or using viral vectors, may need to be pursued further. Meanwhile, it is imperative that the Duchenne boys are given optimal supportive care, including the provision of orthoses to maintain ambulation. We should also continue the search for drugs to influence the course of the disease.[13,14]

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Where are the guidelines for treating hypertension in elderly patients?

No longer any doubts that it should be treated

Raised blood pressure, particularly systolic pressure, has long been recognised as a potent risk factor for coronary artery disease, stroke, and congestive heart failure in elderly patients. This risk factor has a greater impact in elderly than in middle aged patients. Two interventional studies published in the mid-1980s provided strong evidence in favour of treating raised blood pressure in elderly patients,[1,2] and three further studies published within the past year have confirmed these earlier findings[3,4]; one also addressed the issue of isolated systolic hypertension.[5]

Despite differences in study design and drugs used the consistency across these studies is impressive.[6] Diuretics reduced blood pressure, morbidity, and mortality. For example, the incidence of stroke fell by between 25% and 47%. As the incidence of stroke in elderly patients is high the savings in absolute terms are far greater in elderly patients than in young patients. Low doses of diuretics or thiazide diuretics combined with potassium sparing agents were remarkably free of side effects, and in the Medical Research Council trial a thiazide diuretic was better than atenolol.[4] The newer classes of antihypertensive drugs—angiotensin converting enzyme inhibitors, calcium channel blockers, and α blockers—have not been studied in this context, but as in the MRC study outcome may vary with drug class.

Is there an age limit beyond which drug treatment should be withheld? The systolic hypertension in the elderly trial (SHEP) and the Swedish trial in older patients with hypertension (STOP hypertension)[3] both show benefit into the ninth decade, while the European working party on hypertension in the elderly (EWPHE) did not.[1] In very old patients the impact of risk factors may be more complicated than in younger patients. The systolic hypertension in the elderly trial suggested that treating isolated systolic hypertension is rewarding, and another large study (Syst-Eur) is currently investigating this.[7]

Last month Fotherby and colleagues reported on general practitioners' management of hypertension in elderly patient and expressed concern at the lack of consistency given the published data.[8] But is it appropriate to expect general practitioners to incorporate the results of the current plethora of clinical trials into their clinical practice forthwith? Even among doctors whose specialty is hypertension it takes time for a consensus to emerge. An ideal clinical trial does not exist, and no one trial provides the definitive information on which to base practice. Thus we welcome the confirmatory evidence from recent trials for treating combined systolic and diastolic hypertension in elderly patients and the systolic hypertension in the elderly trial,[3] which provides a rationale for treating isolated systolic hypertension. The Syst-Eur study will provide further guidance on managing systolic hypertension.

Each new study builds on its predecessors, providing new information, which aids the fine tuning of treatment. In the case of hypertension in elderly patients this includes the blood pressure levels at which to treat, the target blood pressure, upper age limits, how to manage other cardiovascular risk factors, and the use of newer drugs such as calcium antagonists and angiotensin converting enzyme inhibitors.

For combined systolic and diastolic hypertension the trial data suggest that treatment should be started at pressures above 160/90 mm Hg, and the target should be to lower both systolic and diastolic pressures below these levels. Similarly, in isolated systolic hypertension 160 mm Hg should be taken as the threshold, though recommendations regarding isolated systolic hypertension must be tentative pending confirmation from the Syst-Eur trial. Although a cautious approach to implementing the results of recent landmark studies may be wise, inaction cannot be justified in the face of the substantial body of information pointing towards the need to treat raised blood pressure in elderly patients.

One is led inevitably to the conclusion of Fotherby and colleagues that authoritative, clear guidelines for managing this common disorder are needed and that such guidelines should be revised regularly. Who should be responsible? The British Hypertension Society has produced useful guidelines on treating mild hypertension and is currently reviewing them. Perhaps it should set itself the task of doing this both for hypertension in general and hypertension in elderly patients in particular, reviewing its guidelines biennially.

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Thrombolytic treatment for elderly patients

Age is not a contraindication

The risk of having a myocardial infarction and of dying as a result increases with age: about 80% of fatal myocardial infarctions in Britain occur in patients over 65.[1] A 75 year old with an acute myocardial infarction is seven times more likely to die in hospital than a patient aged 50, and mortality remains twice that of younger patients after discharge.[2]

Why is the mortality so much greater in elderly people? Conditions such as heart failure, angina, diabetes, and hypertension coexist more frequently, and all contribute to a poorer outcome.[2] So does increased age itself[2] and possibly the altered cardiac and systemic responses to myocardial infarction described in elderly patients.[3] Another reason, however, is that they are often treated differently from younger patients.[3,4]

Several studies have shown that thrombolytic treatment reduces mortality and morbidity after acute myocardial infarction[5-9] and, although not designed to assess the efficacy or safety of treatment in elderly patients, their results agree: thrombolytic agents produce the greatest reductions in absolute mortality in those at highest risk of death—particularly older patients. For example, in the second international study of infarct survival (ISIS-2) combined treatment with streptokinase and aspirin saved 10 lives for every 1000 patients treated aged under 60 but 47 lives for every 1000 patients over 70.[7]

Such benefit from thrombolysis depends on prompt administration, ideally within six hours of the onset of symptoms. Compared with other patients with myocardial infarction, elderly patients are more likely to present late, be difficult to diagnose, and have absolute contraindications to thrombolysis. Some must therefore be excluded from treatment,[10,11] and the prescription rate of thrombolytic agents in elderly patients should not be expected to equal that in younger patients. Evidence exists, however, that some older patients are left untreated for less clearly justifiable reasons.

A recent survey of coronary care units in Britain suggested that 40% set an upper age limit for the use of thrombolysis and 20% excluded patients from coronary care, where thrombolysis is usually given, on the grounds of age alone.[12] Even in coronary care units not operating a formal age policy, thrombolysis is used less than in younger patients, often for poorly defined reasons. Experience in North America seems similar: one study showed that a patient aged 75 with no contraindications to thrombolysis had only half the chance of a similar patient aged 40 of receiving treatment.[4] Perhaps audit, which has already been used to identify and minimise inappropriate underuse, can improve matters.[13]

Why is the use of thrombolysis apparently restricted on the grounds of age? Cost effectiveness is at least as good as in younger patients.[14,15] Apprehension regarding the risk of haemorrhagic complications persists and may discourage some doctors from giving thrombolytic drugs to older patients. The risk of stroke at or around the time of myocardial infarction increases with age—to 1·1% in patients aged over 75 who are untreated and to 1·7% in those of similar age who receive thrombolysis.[16] This level of risk—six strokes per 1000 patients treated in this age group—is, however, far from that required to negate the overall benefits of thrombolytic drugs on mortality and morbidity in elderly patients.

Other concerns may exist. How might thrombolysis affect other important end points such as symptoms, function, and dependence? Will the early benefits on mortality be offset by a greater requirement for relatively high risk interventional treatment? Current research suggests not.[17]

Ultimately, decisions regarding the appropriate use of thrombolysis in older patients can become rational only if