In summary, patients who present with a suspected acute myocardial infarction should receive an infusion of intravenous nitrate. If myocardial ischaemia persists and symptoms begin within four hours the patient should be given thrombolytic treatment and possibly a β blocker. Subsequent short term anticoagulation with heparin is justified, though long term treatment with warfarin has little scientific basis; aspirin may be a suitable alternative. Patients should be referred to a cardiac unit if they develop surgical complications or episodes of postinfarct angina.

DAVID P LIPKIN
Consultant cardiologist

COLIN J REID
Senior registrar

Cardiology Department,
Royal Free Hospital,
London NW3 2QG

1 De Bono D. Coronary thrombolysis. Br Heart J 1987;57:301-5.
2 Van der Laarse A, Vermeer F, Hermens WT, et al. Effects of early intracoronary streptokinase on infarct size estimated from cumulative enzyme release and on enzyme release rate: a randomised trial of 533 patients with acute myocardial infarction. Am Heart J 1986;112:672-81.
3 Grupo Italiano per lo Studio della Streptochinasi nell 'Infarto Miocardioco (GISSI): Effectiveness of intravenous thrombolytic treatment in acute myocardial infarction. Lancet 1986;i:397-401.
4 Verstraete M. Thrombolysis in the management of acute myocardial infarction. Current Opinion in Cardiology 1987;2:615-21.
5 Gold HK, Leinbach RC, Garabedian HD, et al. Acute coronary reocclusion after thrombolysis
6 Timmis GC, Mammen EF, Ramos RG, et al. Haemorrhage versus rethrombosis after thrombolysis for acute myocardial infarction. Arch Intern Med 1986;146:642-67.
7 Topel EJ, Califf RM, George BS, et al. Immediate versus delayed angioplasty in acute myocardial infarction. N Engl J Med 1987;317:581-8.
8 Simoons ML, Arnold AER, Betriu A, et al. Thrombolysis with tissue plasminogen activator in acute myocardial infarction: No additional benefit from immediate percutaneous coronary angioplasty. Lancet 1988;i:197-202.
9 Hackett D, Davies G, Chierchia S, Maseri A. Intermittent coronary occlusion in acute myocardial infarction: Value of combined thrombolytic and vasodilator therapy. N Engl J Med 1987;317:1055-9.
10 Yusef S, Collins R. Intravenous nitroglycerin and nitroprusside therapy in acute myocardial infarction reduces mortality. Evidence from randomised controlled trials. Circulation 1985;72 (suppl III):111-224.
11 ISIS-1 Group. Randomised trial of intravenous atenolol among 16027 cases of suspected acute myocardial infarction. Lancet 1986;ii:57-65.
12 MIAMI Trial Research Group. Metoprolol in acute myocardial infarction (MIAMI). A randomised placebo controlled trial. Eur Heart J 1985;6:199-226.
13 Yusef S, Peto R, Lewis JA, Collins R, Sleight P. Beta blockade during and after myocardial infarction: an overview of the randomised trials. Prog Cardiovasc Dis 1985;27:335-71.
14 Boyle D. Intravenous beta-blockade in acute myocardial infarction. Current Medical Literature 1985;4:153-6.
15 Wilcox RG, Hampton JR, Banks DC, et al. Trial of early nifedipine in acute myocardial infarction: the TRENT STUDY. Br Med J 1986;293:1204-8.
16 Gibson RS, Boden WE, Theroux P, et al. The diltiazem reinfarction group, Diltiazem and reinfarction in patients with non-Q wave myocardial infarction. Results of a double-blind, randomised, multicenter trial. N Engl J Med 1986;315:423-9.
17 Moss AJ and the multicenter diltiazem post-infarction research group. Long term effect of Diltiazem on mortality and reinfarction after myocardial infarction (MI)—MDPIT study. J Am Coll Cardiol 1988;11 (suppl A):27A.
18 De Silva RA, Hennekens CH, Lown B, Casscells W. Lignocaine prophylaxis in acute myocardial infarction: an evaluation of randomised trials. Lancet 1981;ii:855-8.
19 Lie KI, Willens HJ, Van Capelle FJ, Durrer D. Lignocaine in the prevention of primary ventricular fibrillation. N Engl J Med 1974;291:1324-6.
20 Adams PC, Cohen M, Chesebro JH, Fuster V. Thrombosis versus embolism from cardiac chambers and rejected valves. J Am Coll Cardiol 1986;8 (suppl B):76-87B.
21 Kakkar VV, Adams PC. Preventive and therapeutic approach to venous thromboembolic disease and pulmonary embolism. J Am Coll Cardiol 1986;8 (suppl B):146-58B.

Delayed effects of head injuries in children

Head injuries in childhood are common and account for about a quarter of all traumatic deaths in children under 15. The devastating effects of severe brain damage in survivors of major injuries are well known, and much help for them and their families has been and is provided through the efforts of Headway, the National Head Injuries Association. The effects of lesser degrees of brain damage resulting from minor trauma are, however, less well recognised, yet they may have serious developmental and educational consequences in children.[1] A conference aimed at increasing awareness of these developmental and educational effects was held last month in London, jointly organised by the department of neuropsychology at Atkinson Morley's Hospital and Headway. A Children's Head Injury Trust is being set up to further these aims.

Improved imaging techniques have shown that minor head injuries, particularly swirling injuries, can cause obvious intracerebral lesions. Neuronal damage may be extensive without prolonged coma or other stigmata of severe brain trauma. Interrupted neurones have a capacity for recovery in children's brains that is greater the younger the child. But such recovery may result in incorrect connections being formed, which may produce persistent sensory abnormalities and major difficulties in processing multiple stimuli—for example, concentrating on a lesson when there is much ambient noise. An additional problem for the young after head injuries is that new learning processes are more affected than memory for already learnt facts and skills, so the smaller the store of such memories the harder it may be to catch up.

Many apparently minor abnormalities in children who have suffered head injuries, particularly those affecting their speech and language abilities, comprehension, behaviour, and drive, may be overlooked or never even considered after a seemingly good recovery from a serious accident. Children return home and to school without further thought about their ability to cope, but two thirds may have persisting difficulties. The most common abnormality is slowing of information processing, which results in difficulty in concentration and learning and so impairment of school progress. There may also be problems with socialisation, social disinhibition, and secondary emotional disturbances. Pressure may be put on an apparently "lazy" child, who in reality cannot cope.

Although some children have neurodevelopmental difficulties before their injury (perhaps constituting risk factors for trauma), there is strong evidence that the effects of head injuries on later performance may be serious; they may lead to a spiral of reduced performance in the face of unchanged expectations, backsliding at school, loss of friends and morale, behaviour disturbance, and ultimate failure. Attaching labels to children is often unhelpful, but there is a good case for skilled and repeated assessment of children who have recovered from head injuries. These assessments should be done preferably by a neuropsychologist and should ensure that the children's social and educational progress is satisfactory and that due consideration is given to the need for remedial help. This does not necessarily have to mean special schooling since with a proper understanding of the particular problems, such as attention deficit, much may be done in normal schools.

O G BROOKE

Medical Scientist,
BMA House,
London WC1H 9JP

1 Johnson DA, Roethig-Johston K. Stopping the slide of head injured children. Special children 1987 Nov:18-20.