supplying the "reciprocal" territory in addition to the disease in the coronary artery that was responsible for the infarct. Nearly a quarter of these patients suffered non-fatal reinfarction and late pulmonary oedema or death, compared with only 10% of those patients without such electrocardiographic changes.

Persistent ventricular ectopic beats after myocardial infarction suggest extensive underlying myocardial fibrosis with poor left ventricular function, features which are associated with a high mortality. It is not known whether long term use of antiarrhythmic agents in these patients will prolong life and thus justify subsequent bypass surgery.

Of the various drugs that have been given after myocardial infarction—agents which influence platelet behaviour,²⁰ antiarrhythmic drugs,²¹ and beta adrenergic blockers^{22–25}—only beta blockers have been convincingly shown to reduce mortality. The delay in documenting the effect of beta blockers is explained by the exclusion of many high risk patients from the drug trials, which increased the proportion of patients at low risk in whom therapeutic intervention would be unlikely to prolong survival.¹³ Beta blockers are not effective antiarrhythmic agents, but (like sympathectomy) they prevent ventricular fibrillation after acute coronary occlusion in animals²⁵ and so probably they exert their beneficial effects by reducing ischaemia and hence ventricular fibrillation, which is provoked by ischaemia. The patients most likely to benefit from them are those with continuing ischaemia.

A strategy for managing patients after myocardial infarction has emerged.²⁶ The patient who will do well may be recognised by his good exercise tolerance, good left ventricular function, and absence of ventricular arrhythmia. There is no evidence to suggest that treatment with beta adrenergic blockers will be of benefit.¹³ Patients with recurrent angina or evidence of inducible ischaemia should undergo prompt coronary angiography and those with left main stem or triple vessel coronary artery disease and adequate left ventricular function should be considered for coronary bypass surgery; those with a stenosis of the left anterior descending artery should be considered for balloon angioplasty. Patients with severe impairment of left ventricular function should have medical treatment to control their ventricular arrhythmias. The results of prospective studies comparing medical with medical plus surgical management are awaited.

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- ¹ Cobb LA, Baum RS, Alvarez H, Schaffer WA. Resuscitation from out-of-hospital ventricular fibrillation: 4 years' follow-up. *Circulation* 1975; **52**, suppl III:223-35.
- ² Kannel WB, Doyle JT, McNamara PM, Quickenton P, Gordon T. Factors related to incidence of sudden death. *Circulation* 1975; **51**: 606-13.
- ³ Perper JA, Kuller LH, Cooper M. Arteriosclerosis of coronary arteries in sudden, unexpected deaths. *Circulation* 1975; **52**, suppl III:27-33.
- ⁴ Chaitman BR, Fisher LD, Bourassa MG, et al. Effect of coronary bypass surgery on survival patterns in subsets of patients with left main coronary artery disease: report of the Collaborative Study in Coronary Artery Surgery (CASS). *Am J Cardiol* 1981; **48**:765-77.
- ⁵ European Coronary Surgery Study Group. Long-term results of prospective randomised study of coronary artery bypass surgery in stable angina pectoris. *Lancet* 1982; **ii**:1173-80.
- ⁶ Mathur VS, Hall RJ, Garcia E, de Castro CM, Cooley DA. Prolonging life with coronary bypass surgery in patients with three-vessel disease. *Circulation* 1980; **62**, suppl I:90-8.
- ⁷ DeRouen TA, Hammermeister KE, Dodge HT. Comparisons of the effects on survival after coronary artery surgery in subgroups of patients from the Seattle Heart Watch. *Circulation* 1981; **63**:537-45.
- ⁸ Betriu A, Castaner A, Sanz GA, et al. Angiographic findings one month after myocardial infarction: a prospective study of 259 survivors. *Circulation* 1982; **55**:1099-105.
- ⁹ Bertrand ME, Lefebvre JM, Laisne CL, Rousseau MF, Carre AG, Lekieffre JP. Coronary arteriography in acute transmural myocardial infarction. *Am Heart J* 1979; **97**:61-9.
- ¹⁰ Madigan NP, Rutherford BD, Frye RL. The clinical course, early prognosis and coronary anatomy of subendocardial infarction. *Am J Med* 1976; **60**:634-41.
- ¹¹ Petch MC. Unblocking coronary arteries. *Br Med J* 1982; **284**:683-4.
- ¹² Pantridge JF, Geddes JS. A mobile intensive-care unit in the management of myocardial infarction. *Lancet* 1967; **ii**:271-3.
- ¹³ Griggs TR, Wagner GS, Gettes LS. Beta adrenergic blocking agents after myocardial infarction: an undocumented need in patients at lowest risk. *Journal of the American College of Cardiology* 1983; **1**:1530-3.
- ¹⁴ Moss AJ, Davis HT, DeCamilla J, Bayer LW. Ventricular ectopic beats and their relation to sudden and nonsudden cardiac death after myocardial infarction. *Circulation* 1979; **60**:998-1003.
- ¹⁵ Davis HT, DeCamilla J, Bayer LW, Moss AJ. Survivorship patterns in the posthospital phase of myocardial infarction. *Circulation* 1979; **60**: 1252-8.
- ¹⁶ Weld FM, Chu K-L, Bigger JT Jr, Roinitzky LM. Risk stratification with low-level exercise testing 2 weeks after acute myocardial infarction. *Circulation* 1981; **64**:306-14.
- ¹⁷ Thérault P, Waters DD, Halphen C, Debaisieux J-C, Mizgala HF. Prognostic value of exercise testing soon after myocardial infarction. *N Engl J Med* 1979; **301**:341-5.
- ¹⁸ Rapaport E, Remedios P. The high risk patient after recovery from myocardial infarction. *Journal of the American College of Cardiology* 1983; **1**:391-400.
- ¹⁹ Palmeri ST, Harrison DG, Cobb FR, et al. A QRS scoring system for assessing left ventricular function after myocardial infarction. *N Engl J Med* 1982; **306**:4-9.
- ²⁰ The Anturane Reinfarction Trial Research Group. Sulfapyrazone in the prevention of cardiac death after myocardial infarction. The anturane reinfarction trial. *N Engl J Med* 1978; **298**:289-95.
- ²¹ Chamberlain DA, Jewitt DE, Julian DG, Campbell RWF, Boyle D McC, Shanks RG. Oral mexiletine in high-risk patients after myocardial infarction. *Lancet* 1980; **ii**:1324-7.
- ²² Multicentre International Study. Improvement in prognosis of myocardial infarction by long-term beta-adrenoreceptor blockade using practolol. *Br Med J* 1975; **iii**:735-40.
- ²³ Hjalmarson A, Elmfeldt D, Herlitz J, et al. Effect on mortality of metoprolol in acute myocardial infarction: a double-blind randomised trial. *Lancet* 1981; **ii**:823-6, 7.
- ²⁴ Norwegian Multicenter Study Group. Timolol-induced reduction in mortality and reinfarction in patients surviving acute myocardial infarction. *N Engl J Med* 1981; **304**:801-7.
- ²⁵ Khan MI, Hamilton JT, Manning GW. Protective effect of beta adrenoceptor blockade in experimental coronary occlusion in conscious dogs. *Am J Cardiol* 1972; **30**:832-7.
- ²⁶ Epstein SE, Palmer ST, Patterson RE. Current concepts: evaluation of patients after acute myocardial infarction: indications for cardiac catheterization and surgical intervention. *N Engl J Med* 1982; **307**: 1487-91.

Road blindness

Far back in 1972 the Commission of the European Communities assembled a dossier¹ showing the advantages of laminated glass windscreens over those made of toughened glass. With bureaucratic thoroughness the European Economic Community committees sifted the evidence, assessed legal, social, economic, and monetary implications, and considered the effects on employment, transport, and regional policy. With surprising unanimity these committees recommended the replacement of toughened by laminated glass in the windscreens of all new vehicles. Yet, when in 1976 the European Parliament accepted these recommendations and urged the European Economic Community Council to act on them, Germany and Britain opposed this legislation. As a result no directive has been issued—an ironic outcome, since Germany had been the first country to report serious eye injuries resulting from shattered toughened windscreens,² and Britain had been first in setting up a study of the relative safety of toughened windscreens versus laminated windscreens with high resistance to penetration.³

When a head on collision occurs at moderate speed the motorist—if unrestrained—flexes forward; his legs press against the floor, his knees strike the dashboard, and his head hits the windscreen. What happens next depends on the composition of the glass. On impact the monolithic toughened glass windscreen, in common use in Britain, may shatter into multiple polygonal fragments; it then offers little resistance to the head, which ploughs through the windscreen all the way to the lower frame with serious risk of laceration of the face and eyes. When the eye is damaged the typical injuries are corneoscleral perforation, uveal prolapse, and opacification or dislocation of the lens. Even with modern microsurgical techniques the initial damage is often so severe that many of these eyes are permanently blinded or may even have to be enucleated.⁴ By contrast, high penetration resistance laminated windscreens are composed of two sheets of lightly tempered glass enclosing a plastic interlayer; they offer a cushioning effect to the head and high resistance to penetration, so that eye injury is rare. Other designs of non-splintering passenger-retaining windscreens are available which are equally safe.

When the motorist is wearing a safety belt the risk to the eye from windscreens, though greatly reduced, is not eliminated.⁵ In a collision the body cannot move forwards, but the impact may cause a toughened windscreen to shatter and then disintegrate; or the windscreen may be broken by direct external force such as a stone or animal, so that 15–20 lb (7–9 kg) of compressed glass explodes into the vehicle.⁶ In either case car occupants wearing belts may have their eyes penetrated by flying glass. In similar conditions laminated windscreens crack in a web like fashion but generally remain intact, so that the risk of eye perforation is negligible.

Comparative⁷ and experimental⁸ studies testify to the superiority of laminated over toughened windscreens from the standpoint of eye safety; but perhaps the most convincing evidence derives from the parallel development of safety glass for windscreens on both sides of the Atlantic. In the United States and Canada laminated windscreens with high resistance to penetration have been used in all vehicles since 1966; the virtual absence of reported ocular injuries in North America contrasts sharply with the frequency of such reports in Europe, where toughened glass windscreens remain in common use.

In Britain legislation on seat belts has been followed by a welcome decrease in the number of perforating eye injuries, but even if seat belts were worn universally some motorists would continue to suffer damage to their eyes from toughened glass windscreens. As a profession we should strive to persuade the government to withdraw its opposition to the European Economic Community proposals: the mandatory fitting of non-splintering passenger-retaining windscreens in all new vehicles is now long overdue.

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¹ Commission of the European Communities. *Official Journal of the European Communities* 1972;C119:66.

² Holland G. Analyse von 2309 verletzungen der augen und lider. *Klin Monatsbl Augenheilkd* 1964;45:915–27.

³ Mackay GM, Siegel AW, Hight PV. Tempered versus HPR laminated windshields. A comparative study of United Kingdom and United States collisions. In: *Stapp car crash conference*. Warrendale, PA: SAE Publishers, 1970:369–87. (Proceedings 14th Stapp conference. Paper No 700911.)

⁴ Blake J, Kelly G, Fahey C, Khan MA. Eye injuries in road traffic accidents. *Ir Med J* 1983;76:120–4.

⁵ Piffaretti JM. Augenärztliche aspekkte der autounfalle in der Schweiz. *Praxis* 1979;68:1264–6.

⁶ Patrick LM, Trosien KR, DuPont FT. Safety performance comparison of 30 mil HPR laminated and monolithic differentially tempered windshields. *International Automobile Safety Conference Compendium*. Brussels, June 1970, No 700427.

⁷ Hass U, Chapman-Smith JS. The role of vehicle safety glass in eye injuries. *NZ Med J* 1976;84:53–5.

⁸ Kimura C. Experimental study on perforating eye injuries caused by the shattering of windshields, using a sled-dummy test device. *Keio J Med* 1978;27:111–21.

Incontinence in women with neuropathic bladders

Urinary incontinence is rarely fatal but may cause social death. The involuntary leakage restricts the sufferer's mobility, and the odour of urine on the person and clothing makes her feel a social outcast. Many women do not seek help, unaware of the methods available to alleviate their distress.

Urinary incontinence is caused by a fistula, an overactive detrusor, or weakness of the sphincter. Effective treatment must depend on an accurate diagnosis. Unfortunately, the bladder is an unreliable witness: a history alone cannot detect the cause of incontinence, and urodynamic tests are necessary. Complex equipment is not needed for every case: the bridge test of Sutherst,¹ using a catheter to detect urethral shortening and lowered resistance in stress incontinence,² and the Lapedes test³ for bladder neuropathy may be carried out in clinics or at the bedside.⁴

Inserting a urethral catheter to control incontinence without first determining its cause is intellectual sloth. Attempts to control urethral leakage by putting in even larger sizes of catheter adds incompetence to ignorance. When faced with a demoralised woman with leakage around her urethral catheter the size of a garden hose, the doctor should remove the catheter. Often there may be urethral ulceration and severe bladder spasms associated with too large a retaining balloon. Eggshell calculus may have formed around the balloon and may need removal by litholapaxy, or the debris may need removal by bladder washout. If the iatrogenic damage is severe, a temporary plastic suprapubic cystostomy catheter may be needed. In some patients the double balloon Portsmouth catheter will maintain continence.⁵ Oestrogens and alpha stimulants help the urethra to heal.

Any woman who needs a catheter should have a urethral antiseptic-anaesthetic jelly applied before instrumentation.^{6–8} Why is it that so many nursing schools teach inferior methods of female catheterisation? Do they really believe that the antiseptic jelly works better on a sterile swab on the trolley than inside the female urethra? It is as though nurses were taught that when giving injections the needle need not be clean if it is short but that a long needle must be sterile. A urethral antiseptic jelly is mandatory for every patient—for we nearly all carry potential pathogens in the distal urethra. By no means every patient, however, will need a permanent catheter. The uninhibited bladder is common in old age, senile dementia, and Parkinson's syndrome and after cerebrovascular lesions.^{9–10} It may be controlled by antispasmodics, regular voiding by the clock ("bladder drill"), marsupial pads, or bladder stretching under anaesthesia.¹¹ Oestrogens may help in older women.^{12–13} Urethral hypotonia may be improved by