Talon noir.

Acral melanoma occurs on the soles and palms and under the nails (subungual melanoma). Benign pigmented naevi may also occur in these sites, but the seven points in the table will help to differentiate them. Talon noir, due to haemorrhage from trauma—for example, playing squash—may cause much concern but the distinct blood filled papillae seen on paring the lesion make the diagnosis plain.

The prognosis of malignant melanoma is related to the depth of invasion. Superficial lesions with a tumour thickness of less than 1 mm have a good prognosis if removed early (80-90% survival at five years). Histologically, the thickness of the tumour is measured from the granular layer (Breslow thickness). Deeper invasion carries a poor prognosis (only 40-50% survival for a tumour deeper than 3 mm).

It is therefore very important to remove any malignant melanoma while it is in the superficial stage.

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New Drugs

Thrombolytic treatment and new calcium antagonists

JOHN FEELY, TERENCE PRINGLE, DEREK MACLEAN

Injury to a blood vessel results in the formation of a haemostatic plug of platelets, and fibrin is laid down to reinforce it to form a thrombus. Once the repair process is established the fibrinolytic system is activated to remove fibrin. Tissue plasminogen activators produced at the site of the injury convert plasminogen to active plasmin, which lyses fibrin. The plasmin produced is active only within the thrombus, and any leakage is destroyed by circulating antiplasmins in the plasma. Thus the fibrinolytic system plays an important part in maintaining the patency of the vascular tree.

Thrombolytic treatment

Two thrombolytic agents—namely, streptokinase and urokinase—are available for clinical use.¹ Streptokinase, the metabolic product of β haemolytic streptococci, is, unlike urokinase (isolated

from human urine), antigenic in man. Streptokinase combines with plasminogen to form an activator complex, which converts plasminogen to plasmin, while urokinase activates plasminogen directly. The generalised lytic state caused by both of these drugs results in the dissolution of thrombi, but it also produces a haemostatic defect throughout the entire vascular system. For this reason these drugs should not be given to patients who are actively bleeding or who have had recent surgery, stroke, or cardiopulmonary resuscitation. Uncontrolled hypertension and active peptic ulceration are also contraindications to treatment.

In recent years other drugs have been developed that act locally at the site of thrombus formation and have a lesser systemic effect. Recombinant tissue plasminogen activator (t-PA) produced by recombinant deoxyribonucleic acid technology from human tissue cultures specifically activates plasminogen already bound to fibrin, and its action on circulating plasminogen is limited. A second drug, anisoylated plasminogen streptokinase activator complex (APSAC), is inactive in plasma. When it attaches to fibrin deacylation occurs, plasminogen activator complex is formed, and plasmin production is localised to the thrombus.

Myocardial infarction

Coronary angiography performed in patients within hours of the onset of pain has confirmed that coronary artery occlusion is the cause of myocardial infarction in most patients.² This has supported the view of pathologists that the rupture of an atheromatous plaque

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with subsequent thrombosis of the coronary artery is the precipitating event. Consequently there has been a resurgence of interest in thrombolysis for acute myocardial infarction despite the inconclusive results of earlier trials.

Streptokinase infused directly into the occluded coronary artery lyses the thrombus in up to four fifths of patients.³ In addition to the angiographic evidence of clot lysis and return of flow in the coronary artery other less exact variables—for example, the relief of pain, the resolution of ST elevation in the electrocardiograph, the emergence of predominantly benign arrhythmias, and the early release of creatine kinase⁴—have suggested that myocardial perfusion is effective. The re-establishment of coronary artery patency has not consistently been accompanied by an improvement in left ventricular function or a reduction in mortality.⁵ Though one placebo controlled randomised study of intracoronary streptokinase showed a decrease in mortality at 30 days after streptokinase treatment, this benefit was not evident at one year.⁶ The most likely cause of the failure to salvage myocardium is the delay from the onset of pain to intracoronary treatment with streptokinase and hence to reperfusion. It has been recognised that valuable time is lost during the transport of the patient to hospital, and a great limitation of intracoronary thrombolytic treatment is that it can be given only in specialised centres with facilities for cardiac catheterisation. Thus the widespread early application of intracoronary streptokinase to most patients who have myocardial infarction is not feasible.

Consequently the intravenous administration of streptokinase has been investigated. Angiographic studies have shown that high dose intravenous streptokinase lyses coronary thrombi (50-60% of patients), albeit not as often as by the intracoronary route. Intravenous streptokinase can be given very early in the course of the myocardial infarction because of the ease of administration. When streptokinase is given by this route it reduces the size of the infarction and consistently improves left ventricular contraction in the region of the infarction. Two large placebo controlled studies have examined the effects of 1.5 million units intravenous streptokinase in acute myocardial infarction. The ISAM study recruited 1741 patients.⁷ Though the size of the infarct was reduced in patients who received streptokinase, the power of the study was not great enough for the trend in the reduction in mortality to reach significance. The results of the GISSI study, in which 11 806 patients were randomised, showed that overall mortality at 21 days was 13.0% in the group given placebo compared with 10.7% in the group given streptokinase (a reduction of 18%).⁸ The largest reduction in mortality was in those patients who received treatment within three hours of the onset of pain. More favourable outcomes were found in patients who were treated with streptokinase and who were aged under 65 or had sustained an anterior infarction. The improvement in mortality persisted at the one year follow up. The prevalence of adverse reactions to streptokinase, including major bleeding episodes and anaphylaxis, was less than 3%.

Tissue plasminogen activator may have advantages over streptokinase. It is not antigenic as it is produced from tissue culture of human cells. Systemic hypotensive reactions, sometimes encountered when streptokinase is given too rapidly, do not occur. It has a short half life, approximately 10 minutes, and is also administered as an infusion. There is some evidence that tissue plasminogen activator is more effective as a thrombolytic agent than streptokinase. In one randomised study the coronary artery was reopened in 60% of patients who received tissue plasminogen activator compared with 35% who received streptokinase.⁹ Tissue plasminogen activator produced a lesser degree of systemic fibrinolysis than streptokinase, but this was not reflected in a lower prevalence of haemorrhagic complications at puncture sites. Though tissue plasminogen activator spares circulating fibrinogen, it has a specific lytic effect not only on thrombi within the arterial lumen but also on haemostatic plugs in the arterial wall.

Anisoylated plasminogen streptokinase activator complex has a fairly long half life of one to two hours and can be given as a single intravenous bolus injection. It is also highly effective in reopening coronary arteries.¹⁰ In doses required to lyse coronary thrombi, however, it produces as much systemic fibrolysis as non-anisoylated streptokinase, and the side effects are similar.

After successful thrombolysis high grade stenoses remain in 70% of the infarct related artery. Reocclusion of the coronary artery occurs in up to 20% of patients and may be more common with tissue plasminogen activator. Reinfarction is clinically apparent only in 5%,¹¹ which may reflect the lack of myocardial salvage despite successful thrombolysis. Nevertheless, various strategies have been advocated to maintain the blood supply to the myocardium at risk. These include anticoagulation, coronary angioplasty, and coronary artery bypass surgery. At present many doctors give anticoagulant drugs to patients after thrombolytic treatment and limit invasive investigations to those who have postinfarction angina or ischaemia induced by exercise.

Though expensive, thrombolysis is becoming established as initial treatment for myocardial infarction. It is most effective when given as soon as possible after the onset of the infarction. The ease and effectiveness of intravenous administration means that thrombolytic drugs can be given on first contact with the patient. The education of the public to seek help quickly when they experience chest pain, and the training of paramedics, general practitioners, or hospital based teams to administer thrombolytic treatment, will help to provide maximum benefit from this form of treatment.

Pulmonary embolism and deep vein thrombosis

Sequential pulmonary angiography and lung scans have shown that thrombolytic agents are highly effective in lysing large pulmonary emboli. In the first 24 hours after treatment both urokinase and streptokinase produce greater lung reperfusion and reduction in pulmonary artery pressure than prophylactic anticoagulation with heparin.^{12,13} By seven days this advantage is lost, and no difference is apparent between the lung scans of patients treated with thrombolytic agents followed by anticoagulation and those treated with anticoagulants alone. Tests of lung function, however, suggest that there is a continuing benefit for up to one year after thrombolysis. Presumably this reflects the lysis of small clots within the peripheral pulmonary microcirculation that are beyond the resolution of pulmonary angiograms or scans.¹⁴

Though the choice of treatment for pulmonary emboli will also determine the treatment for the source of the clot within the deep veins, anticoagulation remains the most popular treatment for isolated deep vein thrombosis. Thrombolysis is successful in dissolving thrombi in the deep veins of the legs in up to 60% of patients, and subsequent venograms show that venous valves are preserved.¹⁵ There is some doubt about whether the early lysis of a thrombus will prevent the postphlebotic syndrome, which causes considerable morbidity in these patients. Haemodynamic studies of the venous system after treatment indicate that the extent of the thrombus in the deep veins of the leg, not the type of treatment, is the most important determinant of the long term outcome.¹⁵

Thrombolysis is indicated in life threatening pulmonary embolism and may have advantages over anticoagulation in treating less severe episodes. Anticoagulation, however, remains the treatment of choice in deep vein thrombosis. No large studies have evaluated the use of tissue plasminogen activator or anisoylated plasminogen streptokinase activator complex in thromboembolic disease, and the results of such trials are awaited before an alteration to standard therapeutic policies is considered.

Peripheral arterial occlusion

Many studies have reported the effects of systemic thrombolysis on peripheral arterial occlusions and emboli.¹ Only up to two fifths of arteries are revascularised with high dose intravenous infusions. Success seems to depend on the duration of the symptoms, and arterial occlusions that have occurred less than 72 hours before treatment are most likely to be reopened. In recent years a low dose infusion of streptokinase has been delivered into the occluded artery close to the thrombus. Recanalisation of the artery has been reported in 70% of patients, including those who have had

symptoms for two years.¹⁶ Thrombolytic treatment is a useful alternative when surgery is contraindicated or in lesions not amenable to surgery. It may also be successful in lysing a thrombotic arterial occlusion that complicates peripheral arterial angioplasty.

Calcium antagonists

Since our review in 1983¹⁷ several new calcium antagonists purporting to have more selective effects than their older counterparts verapamil and nifedipine have been introduced. Agents such as perhexiline and prenylamine, which have complex pharmacological profiles, have been withdrawn or are rarely used. The increasing use of calcium antagonists has led to an expanded range of indications. In addition, to reduce the frequency of dosage sustained release formulations of both the commonly used calcium antagonists nifedipine and verapamil have been introduced. Thus nifedipine (10 mg, 20 mg Adalat Retard) can be given twice daily and verapamil (240 mg Securon SR) once daily with good antihypertensive effect. It should, however, be noted that not all patients, in part due to variation among different people in liver metabolism, are offered 24 hour control, and more frequent dosage may be necessary, particularly if used in angina.

DILTIAZEM

Diltiazem (Tildiem) is a benzothiazepine derivative similar to verapamil which has been in use in the United States and Japan for some years. After oral administration it undergoes extensive and variable first pass metabolism in the liver.¹⁸ In common with many drugs that undergo extensive presystemic elimination, such as verapamil and propranolol, first pass metabolism is saturated during chronic dosing, and plasma concentrations increase. Metabolites, some of which are pharmacologically active, are excreted largely into bile. Its pharmacokinetics, including elimination half life (four to six hours), are not appreciably altered by aging but have not been studied adequately in patients who have liver disease. As expected, kidney disease does not alter the kinetics of diltiazem.

Diltiazem has similar haemodynamic and electrophysiological effects to verapamil but is less potent at producing arterial vasodilatation and has a greater tendency to slow the heart rate. Sinus bradycardia is seen occasionally, and conduction through the atrioventricular node is slowed, suggesting caution in patients who have bradycardia or sick sinus syndrome or are receiving β blockers or digoxin. In addition to this pharmacodynamic interaction with digoxin there may also be a kinetic interaction, as seen with verapamil, reducing digoxin elimination.

Dosage is in the region of 180-360 mg daily, given in three or four doses, though 60 mg twice daily is often recommended initially for the elderly, as some patients show an enhanced response. As an antihypertensive or antianginal agent diltiazem does not seem to have any great advantage over verapamil.¹⁹ There is some evidence that it may prevent early reinfarction in patients who are recovering from subendocardial (non-Q wave) myocardial infarction.²⁰ The incidence of adverse effects, headache, dizziness, flushing, and gastrointestinal disturbance, is similar to that seen with verapamil.

NICARDIPINE

Nicardipine (Cardene), a dihydropyridine derivative, is similar to nifedipine in structure, pharmacological activity, and toxicity.²¹ It is well absorbed, peak concentrations occurring within an hour. It also undergoes extensive presystemic elimination after oral administration. It is not subject to the same problem of sensitivity to light as nifedipine and may be used intravenously. It is rapidly eliminated (half life one to two hours) by extensive hepatic metabolism, with inactive metabolites being excreted in bile and subsequently faeces. Elimination is not appreciably altered in patients who have renal disease. As information on patients who have liver disease is not yet available caution should be exercised in this group.

The primary effect of nicardipine is peripheral vasodilatation.²² This is accompanied by a reflex increase in myocardial pump activity. Though the heart rate may initially increase, it returns to normal during long term treatment. Coronary vasodilatation has been shown to occur. In clinical practice no appreciable effect on conduction tissue has been seen, making nicardipine, unlike verapamil and diltiazem, safe in patients who have impaired atrioventricular conduction. In angina its efficacy is comparable to other calcium antagonists and β blockers. Though nifedipine may decrease left ventricular contraction and relaxation in patients who have coronary heart disease, nicardipine does not.^{21, 23} As an antihypertensive it is of similar efficacy to diuretics or β blockers.

Adverse effects are largely related to vasodilatation—namely, flushing, palpitation, headache, peripheral oedema, and hot limbs—and are hence dose related and usually primarily a problem during the start of treatment. As with nifedipine, hypotension induced by exercise and, rarely, an increase in anginal symptoms, possibly due to reflex sympathetic activity, have been reported. Assessment of the quality of life with psychological variables showed no deterioration during treatment with nicardipine. Dosage should start with 20 mg (increasing if necessary to 40 mg) three times daily. To date nicardipine seems to be almost identical to nifedipine in its effects. If its lack of depressant effect on left ventricular function, particularly in patients who have compromised cardiac pumping ability, is confirmed it may prove advantageous. As with the other groups of calcium antagonists, however, we do not have sufficient comparative studies to make firm recommendations.

OTHER AGENTS

At least 20 new calcium antagonists are under development.^{23, 24} Most of these are dihydropyridone derivatives—for example, felodipine, amlodipine, nimodipine, nitrendipine, nisoldipine—that may have more selective effects on certain regional vasculature. Several phenylalkylamines (methoxyverapamil, tiapamil) similar to verapamil are also under study. Preliminary observations suggest that these drugs will not differ greatly from nifedipine and verapamil in terms of overall efficacy and toxicity.

Expanding indications for calcium antagonists

Calcium antagonists are increasingly being used in hypertension and angina, and verapamil has an established place in managing supraventricular tachycardia.¹⁷ By causing arteriolar vasodilatation nifedipine produces symptomatic relief in patients who have Raynaud's phenomenon,^{25, 26} particularly of the idiopathic variety rather than that secondary to collagen vascular disorders. There is less evidence to support the use of either verapamil or diltiazem in this condition.

By reducing the influx of calcium into cells calcium antagonists have a wide variety of effects, which may influence the choice of treatment in patients who have concomitant disorders. Both nifedipine and verapamil have been reported to reduce the frequency of migrainous headache.²⁶ There is some suggestion that tolerance may occur after a few months, and effective control may be restored by changing to another calcium antagonist. Preliminary studies suggest that calcium antagonists have a possible role in reducing neurological deficits after subarachnoid haemorrhage.^{26, 27} Experimental data also suggest that cell death due to calcium overload—for example, after cardiac arrest²⁸ and cyclosporin A nephrotoxicity²⁹—may be reduced by calcium antagonists.

In hypertrophic cardiomyopathy verapamil in particular improves cardiac symptoms and improves tolerance to exercise, even in patients who have not responded to β blockers.³⁰ Long term efficacy has not, however, been shown for these agents in patients who have pulmonary hypertension.

Nifedipine decreases the pressure in the lower oesophageal sphincter and has been used in patients who have diffuse oesophageal spasm and "nutcracker" oesophagus, which is a common cause of non-cardiac chest pain.³¹ Biting the nifedipine

capsule allows more rapid absorption for acute pain, and in patients who have symptoms related to food it may be taken 30 minutes before meals. Similarly, nifedipine relaxes the internal anal sphincter and may produce some symptomatic relief in proctalgia fugax. This effect on smooth muscle may extend to other systems—for example, myometrial contraction may be reduced in patients who have dysmenorrhoea and in premature labour.²⁴ Recently a combined calcium antagonist and anticholinergic preparation (terodiline) was introduced to treat unstable bladder and urinary incontinence.

To date calcium antagonists have not, in contrast with diuretics and β blockers, been associated with particular problems in managing patients who have concomitant peripheral vascular disease, gout, diabetes, or asthma. Many of the above observations are preliminary, and we await detailed confirmatory studies, including an evaluation of potential additional adverse effects. They may, however, be of relevance when considering whether to use a calcium antagonist or drugs of similar efficacy such as nitrates or β blockers in angina or diuretics or β blockers as drugs of choice in hypertension.

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Medicine and the Media

"EDDIE THE Eagle soars . . . into last place." Everyone loves a good loser. In 1986 Smith's Dock, the last shipyard on Teesside, closed. The losers here were 1400 workers made redundant. No one loved them then, and they remain an embarrassment a year later. Combining a few biographies with a large scale survey, *Shutdown* (ITV, 23 February) was a graphic social documentary of the stresses of redundancy and early unemployment.

Impact on the viewer is more likely to result from the tangible plight of the individual than from statistics, and the visual medium is very potent here—as with the famine in Ethiopia. The recitation of percentages by the Teesside Polytechnic sociologist were almost light relief among the unfolding tragedies. Watching early optimism turn into cataleptic helplessness was a poignant reminder that eloquence is not the prerogative of the skilled public speaker. The unrealistic ambitions of the budding entrepreneur were encouraged and then cruelly dashed after his fish and chip shop had been opened by the local member of parliament (whom we take not to be a certain junior health minister). The images of a derelict society in a derelict environment belied the assertions of an undivided nation emanating from Downing Street.

From the secure watchtower of general practice it is perfectly feasible to ignore the message of this programme. Of the few doctors motivated to tune in I am sure that many will have been aware already of the link between unemployment and health from treating the fallout of such mass redundancy. But what are the medical problems in such salients? From current knowledge a year is too

short a timespan for many of the exworkers to develop consequent ill health and the possibility of follow up was not indicated.

The production team's efforts may have been defeated by timing. The recent downturn in the numbers out of work has made unemployment into yesterday's news. The government would have us believe that unemployment is no longer a serious worry. This one programme could hardly have redressed the present balance.

I refer, finally, to just one of the quoted statistics. Less than half of the 200 employees who opted for training courses rather than for the dole found their new skills useful ammunition in the job market. Obviously too few of them had learnt to ski jump, but perhaps the government has this in mind for its new (compulsory) training schemes.—NORMAN BEALE, general practitioner, Wiltshire.

THE BBC'S PROGRAMME on television, *Network*, aimed last week (23 February) at reviewing how AIDS and infection with the human immunodeficiency virus (HIV) have been covered since last year's AIDS week. A short documentary on the year's broadcasting, by Simon Watney of the Terrence Higgins Trust, was followed by discussion with an invited audience. AIDS week was judged "uneven." Programmes, especially independent television's *AIDS Help*, were praised for factual content, but AIDS week was felt to have left many people bored and "saturated." Representatives of the networks agreed and said that they were now trying to limit coverage to one or two programmes each week.