Education and debate

North of England evidence based guideline development project: guideline on the use of aspirin as secondary prophylaxis for vascular disease in primary care

Martin Eccles, Nick Freemantle, James Mason and the North of England Aspirin Guideline Development Group

Patients who have had cardiovascular disease and stroke are treated with aspirin to reduce their subsequent risk of vascular events or death and thereby to increase the length and quality of their life. This guideline aims to provide general practitioners with evidence linked recommendations on the use of aspirin as secondary prophylaxis for cardiovascular disease and stroke in patients at high risk of these disorders. It is assumed that doctors will use their knowledge and clinical judgment in managing individual patients in the light of available resources. Recommendations may not be appropriate for use in all circumstances. This is a summary of the full version of the guideline.¹

Incidence

In general practice, patients with a raised risk of vascular disease present with several disorders—acute or previous myocardial infarction, unstable or stable angina, transient ischaemic attacks, and peripheral vascular disease. The incidence and prevalence of these conditions and the workload associated with them in general practice can be estimated from the recent national morbidity survey in general practice for England and Wales, and is shown in the table.²

Categorising evidence

Throughout this guideline the strength of statements on evidence and of recommendations is categorised according to the scheme discussed in the first paper in

Summary points

The use of aspirin in the secondary prophylaxis of vascular disease is cost effective

Aspirin should be used in patients with acute myocardial infarction, prior myocardial infarction, stable and unstable angina, and prior stroke or transient ischaemic attack

In acute myocardial infarction a dose of $150\ \mathrm{mg}$ daily should be used

In the other indications a dose of 75 mg daily should be used

the series.³ The box below shows these categories in descending order of importance.

Use of aspirin

Aspirin as an antiplatelet agent

The potential importance of aspirin treatment in patients with a raised risk of vascular diseases has been well described in a recent meta-analysis, the latest update of work reported by the Antiplatelet Trialists' Collaborative Group.⁴ The treatment recommendations in this guideline draw on that work and, where necessary, develop it further by including subsequent

Centre for Health Services Research, University of Newcastle upon Tyne, Newcastle upon Tyne NE2 4AA Martin Eccles,

Martin Eccles, professor of clinical effectiveness

Centre for Health Economics, University of York, York YO1 5DD Nick Freemantle, senior research fellow James Mason, research fellow Other members of the guideline development group are listed in the

Correspondence to: Professor Eccles Martin.Eccles@

appendix

BMJ 1998;316:1303-9

Diseases associated with a raised risk of vascular events—incidence, prevalence, and workload in a general practice, assuming a list size of 2000 patients

Clinical condition	Classification category*	Incidence (new patients/GP/year)	Prevalence (patients consulting/GP/year)	Workload (condition related consultations/GP/year)
Acute myocardial infarction	Acute myocardial infarction	4.6	5.8	10.6
Previous myocardial infarction	Old myocardial infarction	1.0	6.0	2.2
Unstable or stable angina	Angina pectoris	10.4	22.8	52.8
Transient ischaemic attack and prior stroke	Transient cerebral ischaemia	5.0	6.0	9.4
Peripheral vascular disease (intermittent claudication)	Other peripheral vascular disease	4.8	8.0	14.2
All conditions		25.8	48.6	89.2

^{*}As morbidity statistics categories do not match the clinical groups exactly, classification details are presented

Categories of strength used in statements

Strength of evidence

Ia—Evidence from meta-analysis of randomised controlled trials

Ib—Evidence from at least one randomised controlled trial

IIa—Evidence from at least one controlled study without randomisation

IIb—Evidence from at least one other type of quasi-experimental study

III—Evidence from descriptive studies, such as comparative studies, correlation studies, and case-control studies

IV—Evidence from expert committee reports or opinions or clinical experience of respected authorities, or both

Strength of recommendations

A—Directly based on category I evidence B—Directly based on category II evidence or extrapolated recommendation from category I evidence

C—Directly based on category III evidence or extrapolated recommendation from category I or II evidence

D—Directly based on category IV evidence or extrapolated recommendation from category I, II, or III evidence

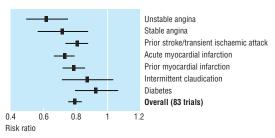


Fig 1 Risk ratio of non-fatal myocardial infarction, stroke, or death from vascular causes in patients at high risk of a vascular event. Meta-analysis of 83 trials

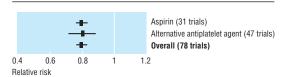


Fig 2 Relative risk of non-fatal myocardial infarction, stroke, or death from vascular causes in patients at high risk of a vascular event who were taking aspirin or an alternative antiplatelet drug. Meta-analysis of 78 trials

trials. We have calculated a pooled risk ratio in relation to clinical subgroups and overall for the impact of antiplatelet treatment on subsequent myocardial infarction, stroke, and death from vascular causes.

This meta-analysis of 83 randomised trials on antiplatelet treatment is summarised in figure 1, which shows that the overall pooled risk ratio is 0.79 (95% confidence interval 0.76% to 0.82%). Tests for heterogeneity provide good evidence of homogeneity between the trials included in this analysis (Q = 74.81, df = 82, P = 0.70). Thus, strong evidence exists that antiplatelet treatment has a protective effect in patients

at raised vascular risk. Trials used several different antiplatelet drugs, including aspirin. Indirect comparison of trials in which aspirin or an alternative antiplatelet drug was used provides no evidence of systematic differences in effect (fig 2).

Substantially different risk reductions in mortality and morbidity are found for different disorders. This may be confounded by differences in the design of studies (particularly the length of follow up), but nevertheless reflects true variation in the potential benefits of treatment in diverse groups of patients. Pooled risk differences for each disorder are described in figure 3.

While 5% of patients in the unstable angina trials benefit from aspirin, the mean benefit in patients with diabetes is much smaller, and the confidence intervals around it are wide. The overall estimate of effect is 3.4% (2.9% to 4.0%) in the fixed effects model. The substantial heterogeneity of effect between studies is unlikely to have occurred by chance (Q=107.38, df=82, P=0.032). The pooled estimate of overall effect in relation to the random effects model is 2.7% (1.7% to 3.7%).

Duration of treatment

The major trials of aspirin have used different study durations ranging from 1 to 48 months. Those recommendations that are made for treatment over a time period covered by the evidence from clinical trials are designated "A"; those that result from extrapolation beyond the period covered by a trial are designated "D." However, in the case of unstable angina, a "D" recommendation was upgraded. This is because the evidence from trials in these patients extends for 18 months, and the guideline development group felt that after this time patients could be regarded as having stable angina and be treated under the recommendations for that condition.

Dosage of aspirin

Trials have used different doses of aspirin; more recent trials have tended to use lower doses. There is no evidence that aspirin in doses greater than 75 mg provides greater benefit, and three recent major trials have used this regimen.⁵⁻⁸ Comparison of the effects of treatment in studies examined here, or in the broader range of comparisons reported by the antiplatelet trialists' collaboration, shows no evidence of differences (fig 4).⁴ We have therefore recommended that the dosage for antiplatelet treatment should be 75 mg

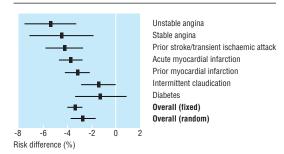


Fig 3 Risk differences (%) for non-fatal myocardial infarction, stroke, or death from vascular causes in patients at high risk of a vascular event treated or not treated with an antiplatelet agent

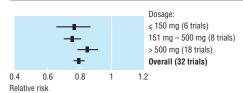


Fig 4 Relative risk of non-fatal myocardial infarction, stroke, or death from vascular causes in relation to treatment with aspirin or an alternative antiplatelet agent. Meta-analysis of 32 trials

of aspirin daily, except in the case of acute myocardial infarction, where most of the evidence is provided by a single trial using twice that dose.⁹

Aspirin and vascular disease

Acute myocardial infarction

Statement: giving aspirin within 24 hours of acute myocardial infarction lowers the risk of a vascular event over the subsequent month (Ia)

Nine trials have examined the role of antiplatelet treatment after acute myocardial infarction. These trials provide a pooled risk difference of 3.8% (2.8% to 4.7%). The pooled incidence rate difference, shown by random effects model, estimates that antiplatelet treatment for one month results in a 3.3% reduction in the risk of myocardial infarction, stroke, or death from vascular causes—or a number needed to treat value of 30. Limited follow up from the second international study of infarct survival indicates that the benefit of one month's treatment with aspirin may be maintained for four years. The second international study of the second international study of infarct survival indicates that the benefit of one month's treatment with aspirin may be maintained for four years.

Recommendations: acute myocardial infarction

- Patients with a suspected acute myocardial infarction should be treated with 150 mg of aspirin daily (A)
- Patients with proved acute myocardial infarction should be given 150 mg of aspirin daily for one month (A)
- After one month, patients should be treated according to the section on previous myocardial infarction (A)

Previous myocardial infarction

Statement: aspirin given to patients who have previously had a myocardial infarction lowers their risk of a subsequent vascular event (Ia)

Within this area of investigation there are 11 trials, and average treatment periods vary from 12 to 41 months. 21-40 Overall, these trials give a risk difference of 3.2% (2.2% to 4.2%) for myocardial infarction, stroke, or vascular death. The pooled incidence rate difference, shown by random effects model, estimates that antiplatelet treatment for one year results in a 1.5% reduction in the risk of myocardial infarction, stroke, or death from vascular causes—or a number needed to treat of 65.

Recommendations: previous myocardial infarction

- Patients who have previously had a myocardial infarction should be treated with 75 mg of aspirin daily for three years (A)
- After three years, aspirin should be continued long term at a dose of 75 mg daily (D)

Stable angina

Statement: aspirin given to patients with stable angina lowers their risk of having a subsequent vascular event (Ia) In the six studies examining the effectiveness of antiplatelet treatment for patients with stable angina, the overall risk difference was 4.5% (1.9% to 7.1%).^{5 40-46} The pooled incidence rate difference, shown by random effects model, estimates that antiplatelet treatment for one year results in a 0.7% reduction in the risk of myocardial infarction, stroke, or death from vascular causes—or a number needed to treat of 150.

Recommendations: stable angina

- Patients who have stable angina should be treated with aspirin 75 mg daily for four years (A)
- After four years, aspirin should be continued long term at a dose of 75 mg daily (D)

Unstable angina

Aspirin given to patients with unstable angina lowers their risk of having a subsequent vascular event (Ia)

Seven trials provide evidence of the effectiveness of antiplatelet treatment in unstable angina. ⁷ ⁴⁷⁻⁵⁴ Overall, a risk difference of 5.5% (3.4% to 7.5%) in the incidence of myocardial infarction, stroke, or death from vascular causes was achieved. There is no evidence of heterogeneity between estimates of treatment effect (Q=8.72, df=6, P=0.19). The pooled incidence rate difference, shown by random effects model, estimates that antiplatelet treatment for one year results in a 6.6% reduction in the risk of myocardial infarction, stroke, or death from vascular causes—or a number needed to treat of 15.

Recommendations: unstable angina

- Patients with suspected unstable angina should be treated with 75 mg of aspirin daily for 18 months (A)
- After 18 months, patients with a history of unstable angina should be treated in accordance with the recommendation for stable angina (A)

Previous stroke or transient ischaemic attack

Statement: aspirin given to patients with a history of transient ischaemic attack or mild to moderate stroke lowers their risk of a subsequent vascular event (Ia)

Within this area of investigation there are 19 trials, showing an overall risk reduction of 4.3% (2.8% to 5.8%).⁶ ⁵⁵⁻⁸³ The pooled incidence rate difference, shown by random effects model, estimates that

antiplatelet treatment for one year brings a 1.4% reduction in the risk of myocardial infarction, stroke, or death from vascular causes—or a number needed to treat of 69.

Recommendations: previous stroke or transient ischaemic attack

- Patients with a history of a stroke or transient ischaemic attack should be treated with 75 mg of aspirin daily for four years (A)
- Computed tomography is unnecessary before starting treatment in these patients (D)
- After four years, aspirin should be continued long term at a dose of 75 mg daily (D)

Intermittent claudication

Statement: aspirin given to patients with intermittent claudication seems to have a small and statistically uncertain effect on the risk of a vascular event (Ia) Substantial evidence from 23 randomised trials shows that antiplatelet treatment for intermittent claudication is unlikely to have a beneficial effect on the subsequent incidence of non-fatal myocardial infarction, non-fatal stroke, and death from vascular causes. 84-110 Overall, the risk difference in favour of treatment is 1.3% (-0.1% to 2.7%), a difference of borderline significance. The use of antiplatelet treatment in patients with intermittent claudication for reasons other than secondary prophylaxis of vascular events is discussed elsewhere. 4-111

Recommendations: intermittent claudication

- Patients with intermittent claudication who have additional indications of raised vascular risk should be treated in line with the recommendations for that indication (D)
- There is insufficient evidence to support use prophylactic aspirin in patients with intermittent claudication but no additional vascular risk factors (A)

Diabetes

Statement: aspirin given to patients with diabetes seems to have a small and statistically uncertain effect on the risk of a vascular event (Ia)

Within this area of investigation there are eight trials. $^{112-122}$ These show an overall estimate of risk difference of 1.2% (-0.9% to 3.3%), which is of uncertain significance. There is no evidence of heterogeneity of treatment effect between these trials (Q=7.66, df=7, P=0.36). The pooled incidence rate difference, shown by random effects model, estimates that antiplatelet treatment for one year brings a non-significant reduction in the risk of myocardial infarction, stroke, or death from vascular causes of 0.3%—or a number needed to treat of 360.

Recommendations: diabetes

- Diabetic patients with additional indications of a raised risk of vascular events should be treated in line with the recommendations for those indications (D)
- There is insufficient evidence to support the use of prophylactic aspirin in patients with diabetes but no additional risk factors (A)

Safety and cost effectiveness of aspirin Side effects and costs of aspirin

Statement: the benefits of using aspirin in the secondary prophylaxis of vascular disease considerably outweigh the attributable risks of gastrointestinal or cerebrovascular bleeding (Ib)

Statement: the use of aspirin is likely to be cost saving or cost neutral (IV)

The dosages, cautions, contraindications, and side effects of aspirin as an antiplatelet drug are described in the *British National Formulary*, section 2.9.¹²⁸ All guideline recommendations for treatment apply only in the absence of recognised cautions, contraindications, side effects, or interactions as documented in the latest version of the formulary.

The net value of aspirin to individual patients must balance the increased risk of haemorrhage against the reduced likelihood of a cardiovascular event. A recent review examined all trials listed in the antiplatelet trialists' collaboration for information on toxicity. 124 When patients receiving aspirin and placebo were compared, the pooled odds ratio for all forms of gastrointestinal bleeding was 2.0 (1.5 to 2.8) and for bleeding leading to hospital admission was 1.9 (1.1 to 3.1). Similarly, when these groups were compared for either peptic ulcer or gastrointestinal symptoms leading to withdrawal of treatment, the pooled odds ratios were 1.3 (1.1 to 1.6) and 1.5 (1.1 to 1.9). The review found a consistent tendency of lower rates of adverse events in lower dose

Two major trials in which a dosage of 75 mg aspirin daily was used have been reported. ⁵ ⁶ It is possible to project the major benefits and risks attributable to this treatment regimen, but estimates should be treated with caution, since the trials did not have enough power to measure adverse effects at conventional levels of statistical significance. Assuming 1000 person years of treatment, the following effects are attributable to 75 mg of aspirin daily:

- (1) Ten patients with stable angina will avoid vascular events (non-fatal or fatal myocardial infarction or sudden death). However, one patient will have a fatal bleed, one will have a major non-fatal bleed, and one patient will experience a minor bleed (where "bleed" includes stroke and gastrointestinal haemorrhage).
- (2) Twenty nine patients who have previously had a transient ischaemic attack or minor stroke will avoid a vascular event (non-fatal or fatal stroke or other vascular death). However, six patients will have a serious bleed (possibly fatal) and eight patients will suffer less serious bleeds.

Economic aspects

We found no adequate cost-benefit analyses of aspirin as an antiplatelet drug. The cost of aspirin itself is negligible. Generic aspirin, in 75 mg dispersible tablets, costs approximately £1 per year to prescribe, although proprietary brands may cost 10-20 times more. From a health service perspective, the net cost includes the cost of aspirin, treatment for attributable adverse events, and savings from fewer vascular events. Patients with vascular disease tend to consult their general practitioner regularly, and any increase in consultation because of treatment with aspirin would probably be small. Since the reduction in vascular events exceeds considerably the attributable adverse events, and given the nature of the medical interventions for both, aspirin treatment probably results in a net cost saving to the health service. The balance of costs could shift adversely if it were necessary to provide expensive H₂ agonists to ameliorate gastrointestinal symptoms in a number of patients. The trials reported above, however, do not indicate that this would be the case. Aspirin is probably cost saving or cost neutral, although formal cost calculation has not proved possible because of inadequate hospital cost data.

Recommendation: secondary prophylaxis of vascular disease

· Use of aspirin in the secondary prophylaxis of vascular disease is cost effective (D)

Research needs

The following research needs were identified by the guideline development group during the preparation of this document:

- (1) Many of the trials of antiplatelet treatment were conducted before the introduction of other important treatments for some groups of patients with ischaemic heart disease (for example, angiotensin converting enzyme inhibitors for heart failure): all potential interactions between the actions of relevant drugs have not been explored.
- (2) Further research on the appropriate duration of treatment is required.
- (3) A formal evaluation of the cost effectiveness of aspirin and other antiplatelet agents is required.
- (4) Further trials are needed to examine the effect of 75 mg of aspirin daily in patients with intermittent claudication or diabetes as the only risk factor for vascular disease.

We thank the following for reviewing the full version of the draft guideline: Dr Phil Ayres, Dr Richard Baker, Professor Stuart Cobbe, Dr Chris Griffiths, and Dr Andrew Herxheimer. Janette Boynton, Julie Glanville, Susan Mottram, and Anne Burton are thanked for their contribution to the functioning of the guideline development group and the development of the practice guideline.

Appendix

The guideline development group comprises the following members, in addition to the authors: Mr Mark Campbell, prescribing manager, Wolfson Unit of Clinical Pharmacology, University of Newcastle upon Tyne; Dr David Graham, general practitioner, Hexham; Dr Keith MacDermott, general practitioner, York; Dr Tony McKenna, general practitioner, Stockton-on-Tees; Dr Maureen Norrie, general practitioner, Eston,

Middlesborough; Dr Colin Pollock, medical director, Wakefield Health Authority; Dr Helen Rogers, senior lecturer and consultant in stroke medicine, Centre for Health Services Research, University of Newcastle; Dr Jeff Rudman, general practitioner, Distington.

The project steering group comprises: Professor Michael Drummond, Centre for Health Economics, University of York; Professor Andrew Haines, Department of Primary Care and Population Sciences, University College, London Medical School and Royal Free Hospital School of Medicine; Professor Ian Russell, Department of Health Sciences and Clinical Evaluation, University of York; Professor Tom Walley, Department of Pharmacology and Therapeutics, University of Liverpool.

Funding: The work was funded by the Prescribing Research Initiative of the UK Department of Health.

Conflict of interest: None.

- 1 North of England Evidence Based Guidelines Development Project. Evidence based guideline for the use of aspirin for the secondary prophylaxis of vas-cular disease in primary care. Newcastle upon Tyne: Centre for Health Services Research, 1997.
- McCormick A, Fleming D, Charlton J. Morbidity statistics from general practice. Fourth national study 1991-1992. London: HMSO, 1995.

 Eccles M, Freemantle N, Mason J. Methods of developing guidelines for
- efficient drug use in primary care. *BMJ* 1998;316:1232-5.

 Antiplatelet Trialists' Collaboration. Collaborative overview of ran-
- domised trials of antiplatelet treatment. I. Prevention of death, myocardial infarction and stroke by prolonged antiplatelet therapy in various categories of patients. *BMJ* 1994;308:81-106.
- Juul-Möller S, Edvardsson N, Jahnmatz B, Rosen A, Sorensen S, Ömblus R, for the Swedish Angina Pectoris Aspirin Trial Group. Double blind trial of aspirin in primary prevention of myocardial infarction in patients with stable chronic angina pectoris. Lancet 1992;340:1421-5.
- SALT Collaborative Group. Swedish aspirin low-dose trial (SALT) of 75 mg aspirin as secondary prophylaxis after cerebrovascular ischaemic events. *Lancet* 1991;338:1345-9.
- Wallentin LC for the Research Group on Instability in Coronary Artery Disease in Southeast Sweden. Aspirin (75 mg/day) after an episode of unstable coronary artery disease: long-term effects on the risk for myocardial infarction, occurrence of severe angina and the need for revascularization. J Am Coll Cardiol 1991;18:1587-93.
- Nyman I, Larsson H, Wallentin L for the Research Group on Instability in Coronary Artery Disease in Southeast Sweden (RISC). Prevention of serious cardiac events by low-dose aspirin in patients with silent myocardial ischaemia. Lancet 1992;340:497-501.
- ISIS-2 (Second International Study of Infarct Survival) Collaborative Group. Randomised trial of intravenous streptokinase, oral aspirin, both or neither among 17,187 cases of suspected acute myocardial infarction: ISIS-2. Lancet 1988:ii:349-60.
- 10 Rasmanis G, Vesterqvist O, Green K, Edhag O, Henriksson P. Effects of intermittent treatment with aspirin on thromboxane and prostacyclin formation in patients with acute myocardial infarction. Lancet 1988;ii:245-7
- 11 Bjerre Knudsen J, Kjøller E, Skagen K, Gormsen J. The effect of ticlopidine on platelet functions in acute myocardial infarction., A double blind controlled trial. *Thromb Haemost* 1985;53:3326.
- 12 Funke Küpper AJ, Verheugt FWA, Jaarsma W, Roos JP. Failure of sulphinpyrazone to prevent left ventricular thrombosis in patients with AMI treated with oral anticoagulants. In: Proceedings of the X world congress on cardiology, Washington, DC, 1986:419. (Abstract 2414.)
- 13 Funke Küpper AJ, Verheugt FWA, Peels CH, Galema TW. Effect of low dose acetyl salicylic acid on the frequency and hematologic activity of left ventricular thrombus in anterior wall acute myocardial infarction. Am j Cardiol 1989;63:917-20.
- 14 Verheugt FWA, Funke Küpper AJ, Galema TW, Roos JP. Low dose aspirin after early thrombolysis in anterior wall acute myocardial infarction. Am J Cardiol 1988:61:904-6.
- 15 Verheugt FWA, van-der-Laarse A, Funke Küpper AJ, Sterkman LGW, Galema TW, Roos JP. Effects of early intervention with low-dose aspirin (100 mg) on infarct size, reinfarction and mortality in anterior wall acute myocardial infarction. Am J Cardiol 1990;66:267-70.
 Gent AK, Brook CG, Foley TH, Miller TN. Dipyridamole: a controlled
- trial of its effect in acute myocardial infarction. BMJ 1968;iv:366-8.

 17 Jones EW. A study of dazoxiben in the prevention of venous thrombosis after suspected myocardial infarction [dissertation]. Nottingham: University of Nottingham, 1985.
- 18 Brochier ML for the Flurbiprofen French Trial. Evaluation of flurbiprofen for prevention of reinfarction and re-occlusion after successful thrombolysis or angioplasty in acute myocardial infarction. Eur Heart J 1993;14:951-7.
- 19 ISIS Pilot Study Investigators. Randomized factorial trial of high-dose intravenous streptokinase, of oral aspirin and of intravenous heparin in acute myocardial infarction. Eur Heart J 1987;8:634-42.
- 20 Baigent C, Collins R. ISIS-2: 4 year mortality follow up of 17,187 patients after fibrinolytic and antiplatelet therapy in suspected acute myocardial infarction. *Circulation* 1993;88:1-291.

- 21 Gentile R, Lagana B, Calcagni S. Borgia MC, Baratta L. Efficacy of plate-let inhibiting agents in the prevention of reinfarction in smoker patients. In: Proceedings of the X world congress on cardiology, Washington, DC, 1986:302. (Abstract 1724)
- 22 Breddin K, Loew D, Lechner K, Uberla KK, Walter E, on behalf of the German-Austrian Myocardial Infarction (GAMIS) Study Group. The German-Austrian aspirin trial: a comparison of acetylsalicylic acid, placebo and phenprocoumon in secondary prevention of myocardial infarction. Circulation 1980;62(suppl V):63-72.
- 23 Anturane Reinfarction Italian Study (ARIS) Research Group. Sulphinpyrazone in post-myocardial infarction. *Lancet* 1982;i:237-42.
- 24 Elwood PC, Cochrane AL, Burr MI, Sweetnam PM, Williams GH, Welsby E, et al. A randomized controlled trial of acetylsalicylic acid in the secondary prevention of mortality from myocardial infarction. BMJ 1974;i:436-40.
- 25 Elwood PC. Trial of acetylsalicylic acid in the secondary prevention of mortality from myocardial infarction. BMJ 1981;282:481.
- 26 Persantine-Aspirin Reinfarction Study (PARIS) Research Group. Persantine and aspirin in coronary heart disease. Circulation 1980;62:449-61.
- 27 Persantine-Aspirin Reinfarction Study (PARIS) Research Group. The persantine-aspirin reinfarction study. Circulation 1980;62 (suppl V):V85-8.
- 28 Vogel G, Fischer C, Huyke R. Prevention of reinfarction with acetylsalicylic acid. In: Breddin HK, Loew D, Uberla K, Dorndoff W, Marx R, eds. Prophylaxis of venous peripheral cardiac and cerebral vascular diseases with acetylsalicylic acid. Stuttgart: Shattauer, 1981:123-8.
- 29 Vogel G. Fischer C, Huyke R. Reinfarktprophylaxe mit Acetylsalizylsäure. Folia Haematol (Leipz) 1979;106:797-803.
- 30 Coronary Drug Project (CDP) Research Group. Aspirin in coronary heart disease. J Chronic Dis 1976;29:625-42.
- 31 Coronary Drug Project (CDP) Research Group. Aspirin in coronary heart disease. Circulation 1980;62(suppl V):59-62.
- 32 Coronary Drug Project (CDP) Research Group. The coronary drug project: design, methods and baseline results. *Circulation* 1973;47(suppl 1):149.
- 33 Anturane Reinfarction Trial (ART) Research Group. Sulfinpyrazone in the prevention of sudden death after myocardial infarction. N Engl J Med 1980:302:250-6.
- 34 Anturane Reinfarction Trial (ART) Research Group. The anturane reinfarction trial: re-evaluation of outcome. N Engl J Med 1982;306:1005-8.
- Sherry S. The anturane reinfarction trial. Circulation 1980;62(suppl V):73-8.
- 36 Temple BA, Pledger GW. The FDA's critique of the anturane reinfarction trial. N Engl J Med 1980;303:1488-92.
- 37 Elwood PC, Sweetnam PM. Aspirin and secondary mortality after myocardial infarction. *Lancet* 1979;ii:1313-5.
- 38 Elwood PC, Sweetnam PM. Aspirin and secondary mortality after myocardial infarction. *Circulation* 1980;62(suppl V):53-8.
- 39 Klimt CR, Knatterud GL, Stamler J, Meier P. Persantine-aspirin reinfarction study. Part II. Secondary coronary prevention with persantine and aspirin. J Am Coll Cardiol 1986;7:251-69.
- 40 Aspirin Myocardial Infarction Study (AMIS) Research Group. AMIS: a randomized controlled trial of aspirin in persons recovered from myocardial infarction. JAMA 1980;243:661-9.
- 41 Becker MC. Angina pectoris: a double blind study with dipyridamole. *J Newark Beth Israel Hospital* 1967;18:88-94.
 42 Berglund U, Lassvik C, Wallentin I. Effects of the platelet inhibitor ticlopi-
- 42 Berglund U, Lassvik C, Wallentin I. Effects of the platelet inhibitor ticlopidine on exercise tolerance in stable angina pectoris. Eur Heart J 1987;8:25-30.
- 43 Berglund U, von Schenck H, Wallentin I. Effects of the platelet inhibitor ticlopidine on platelet function in men with stable angina pectoris. *Thromb Haemost* 1985;54:808-12.
- 44 Wirecki M. Treatment of angina pectoris with dipyridamole: a long-term double blind study. *J Chronic Dis* 1967;20:139-45.
 45 Shar S, Schlant RC. Dipyridamole in the treatment of angina pectoris.
- 45 Shar S, Schlant RC. Dipyridamole in the treatment of angina pectoris JAMA 1967;201:865-7.
 46 Chemical Market MM Society MC From DL Helmon DD. Prochanges
- 46 Chesebro JH, Webster MW, Smith HC, Frye RI, Holmes DR, Reeder GS, et al. Antiplatelet therapy in coronary disease progression: reduced infarction and new lesion formation. *Circulation* 1989;80(suppl II):266.
- 47 Lewis HD, for the Veterans Administration Co-operative Study Group. Unstable angina: status of aspirin and other forms of therapy. *Circulation* 1985;72(suppl V):155-60.
- 48 ALDUSA (aspirin at low dose in unstable angina) pilot study. Report from the coordinating center. Lyon: Unite de Pharmacologie Clinique, 1987.
- 49 Aspirin Myocardial Infarction Study (AMIS) Research Group. AMIS: the aspirin myocardial infarction study: final results. *Circulation* 1980;62(suppl V):79-84.
- 50 Prandoni P, Milani L, Barbiero M, Cardaioli P, Sanson A, Barbaresi F, et al. A combination of dipyridamole with low-dose aspirin in the treatment of unstable angina. *Minerva Cardioangiol* 1991;39:267-73.
- 51 Cairns JA, Gent M, Singer J. Finnie KJ, Froggatt GM, Holder DA, et al. Aspirin, sulfinpyrazone, or both in unstable angina. Results of a Canadian multicentre trial. N Engl J Med 1985;313:1369-75.
 52 Balsano F, Rizzon P, Violi F, Scrutinio D, Cimmkiniello C, Aguglia F, et al
- 52 Balsano F, Rizzon P, Vtoli F, Scruttinio D, Cimmkiniello C, Aguglia F, et al for the Studio della Ticlopidina nell'Angina Instabile Group. Antiplatelet treatment with ticlopidine in unstable angina: a controlled multicentre clinical trial. Circulation 1990;82:17-26.
- 53 Scrutinio D, Lagioia R, Rizzon P on behalf of Studio della Ticlopidina nell'Angina Instabile Group. Ticlopidine treatment for patients with unstable angina at rest. A further analysis of the study of ticlopidine in unstable angina. Eur Heart J 1991;12(suppl G):27-9.
- 54 Lewis HD, Davis JW, Archibald DG, Steinke WE, Smitherman TC, Doherty J, et al. Protective effects of aspirin against acute myocardial infarction and death in men with unstable angina. Results of a Veterans' Administration co-operative study. N Engl J Med 1983;309:396-403.

- 55 Ross Russell RW. The effect of ticlopidine in patients with amaurosis fugax. Guildford: Sanofi Winthrop, 1985. (Sanofi internal report 105062/0051.)
- 56 Gawel M, Rose FC. Use of sulphinpyrazone in the prevention of restroke and stroke in man. In: Rose FC, ed. Advances in stroke therapy. New York: Raven Press, 1982;158.
- 57 Reuther R, Dorndorf W, Loew D. Behandlung Transitorisch-ischamer Attacker mit Acetylsalicylsäure. Münchener Medizinische Wochenschrift 1980:122:795-8.
- 58 Reuther R. Dorndorf W. Aspirin in patients with cerebral ischaemia and normal angiograms or nonsurgical lesions. In: Breddin K, Dorndorf W. Loew D, Marx R, eds. Acetylsalicylic acid in cerebral ischaemia and coronary heart disease. Stuttgart: Schattauer, 1978:97-106.
- 59 Roden S, Low-Beer T, Carmalt M, Cockel R, Green I. Transient cerebral ischaemic attacks—management and prognosis. *Postgrad Med J* 1981;57:275-8.
- 60 Robertson JT, Dugdale M, Salky N, Robinson H. The effect of a platelet inhibiting drug (sulfinpyrazone) in the therapy of patients with transient ischaemic attacks (TIAs) and minor strokes. *Thrombosis et Diathesis Haem-orrhagica* 1975;34:598.
- 61 Acheson J, Danta G, Hutchinson EC. Controlled trial of dipyridamole in cerebral vascular disease. *BMJ* 1969;i:614-5.
- 62 Acheson J, Danta G, Hutchinson EC. Platelet adhesiveness in patients with cerebral vascular disease. Atherosclerosis 1972;15:123-7.
- 63 Sorensen PS, Pedersen H, Marquardsen J, Petersson H, Heltberg A, Simonsen N, et al. Acetylsalicylic acid in the prevention of stroke in patients with reversible cerebral ischaemic attacks. A Danish co-operative study. Stroke 1983:14:15-22.
- 64 Blakely JA. A prospective trial of sulfinpyrazone and survival after thrombotic stroke. Proceedings of VII international congress on thrombosis and haemostasis, 1979:161. (Abstract 42.)
- 65 Boysen G, Soelberg-Sørensen P, Juhler M, Andersen AR, Boas J. Olsen JS, et al. Danish very-low-dose aspirin after carotid endarterectomy trial. Stroke 1988:19:1211-5.
- 66 Fields WS, Lemak NA, Frankowski RF, Hardy RJ. Controlled trial of aspirin in cerebral ischaemia. Stroke 1977;8:301-14.
- 67 Fields WS, Lemak NA, Frankowski RF, Hardy RJ. Controlled trial of aspirin in cerebral ischaemia. Part II. Surgical group. Stroke 1978;9:309-18.
- 68 Lemak NA, Fields WS, Frankowski RF, Hardy RJ. Controlled trial of aspirin in cerebral ischaemia: an addendum. Neurology 1986;36:705-10.
- 69 Guiraud-Chaumeil B, Rascol A, David J, Boneu B, Clanet M, Bierme R. Prevention des récidives des accidents vasculaires cérébraux ischemiques par les anti-agrégants plaquettaires. Rev Neurol (Paris) 1982;138:367-85.
- 70 Rascol A, Guiraud-Chaumeil B, Boneu B, David J, Clanet M. A long-term randomized trial of antiaggregating drugs in threatened stroke. In: Rose FC, ed. Advances in stroke therapy. New York: Raven Press, 1982:147-53.
- 71 Canadian Co-operative Study Group. A randomized trial of aspirin and sulfinpyrazone in threatened stroke. N Engl J Med 1978;299:53-9.
- 72 Gent M, Barnett JHM, Sackett DL, Taylor DW. A randomized trial of aspirin and sulfinpyrazone in patients with threatened stroke. Results and methodologic issues. *Circulation* 1980;62(suppl V):97-105.
- 73 Whisnant JP, Matsumoto N, Elveback LR. The Canadian trial of aspirin and sulphinpyrazone in threatened stroke. Am Heart J 1980;99:129-30.
- 74 Gent M, Blakely JA, Hachinski V, Roberts RS, Barnett HJM, Bayer NH, et al. A secondary prevention randomized trial of suloctidil in patients with a recent history of thromboembolic stroke. Stroke 1985;16:416-24.
- 75 Britton M, Helmers C, Samuelsson K. High-dose acetylsalicylic acid after cerebral infarction—a Swedish co-operative study. Stroke 1987;18:325-34.
 76 Bousser MG, Eschwege E, Haguenau M, Lefauconnier JM, Thibult N,
- 76 Bousser MG, Eschwege E, Haguenau M, Lefauconnier JM, Thibult N, Touboul D, et al. AICLA controlled trial of aspirin and dipyridamole in the secondary prevention of athero- thrombotic cerebral ischaemia. Stroke 1983;14:5-14.
- 77 Bousser MG, Eschwege E, Haguenau M, Lefauconnier JM, Thibult N, Touboul D, et al. Essai coopératif contrôlé de prévention secondaire des accidents ischémiques cérébraux liés à athérosclérose par l'aspirine et le dipyridamole. Presse Med 1983;12:3049-57.
- 78 Gent M, Blakely JA, Easton JD, Ellis DJ, Hachinski VC, Harbison JW, et al. The Canadian American ticlopidine study (CATS) in thromboembolic stroke. Stroke 1988;19:1203-10.
- 79 Gent M, Easton JD, Hachinski VC, Panak E, Sicurella J. Blakely JA, et al. The Canadian American ticlopidine study (CATS) in thromboembolic stroke. *Lancet* 1989;i:1215-20.
- UK-TIA Study Group. United Kingdom transient ischaemic attack (UK-TM) aspirin trial: interim results. BMJ 1988;296:316-20.
- 81 UK-TIA Study Group. United Kingdom transient ischaemic attack (UK-TM) aspirin trial: final results. J Neurol Neurosurg Psychiatry 1991;54:1044-54.
- 82 ESPS Group. The European stroke prevention study (ESPS). Principal endpoints. *Lancet* 1987;ii:1351-4.
- 83 ESPS Group. The European stroke prevention study (ESPS). Stroke 1990;21:122-30.
- 84 Bourde C, Eschwege E, Verry M. Controlled clinical trial of an antiaggregating agent, ticlopidine, in vascular ulcers of the leg. *Thomb Haemost* 1981;46:91. (Abstract 0271.)
- 85 Bourde C, Giraud D. Correlations cliniques et téléthermographiques dans les ulcères de jambes d'origine vasculaire traités par un anti-aggrégant plaquettaire: la ticlopidine. J Mal Vasc 1982;4:31-7.
- 86 Stuart J, Aukland A, Hurlow RA, George AJ, Davies AJ. Ticlopidine in peripheral vascular disease. Proceedings of VI international congress of the Mediterranean League Against Thrombosism 1980:75-87. (Abstract 73.)
- 87 Adriansen H. Medical treatment of intermittent claudication: a comparative double-blind study of suloctidil, dihydroergotoxine, and placebo. Curr Med Res Opin 1976;4:395-401.
- 88 Shaw K. Assessment of the effect of ticlopidine on diabetic pre-gangrene. Guildford: Sanofi Winthrop, 1983. (Sanofi internal report 001.6.241.)

- 89 Jones NAG, De Haas H, Zahavi J, Kakkar VV. A double blind trial of suloctidil v placebo in intermittent claudication. Br J Surg 1982;69:
- 90 Krause D. Double blind study-ticlopidine versus placebo-in intermittent claudication. Guildford: Sanofi Winthrop, 1983. (Sanofi internal report
- 91 Holm J, Lindblad L, Schersten T, Sunrkula M. Intermittent claudication: suloctidil vs placebo treatment. Vasa 1984;13:175-8.
- 92 Verhaeghe R, Van Hoof A, Beyens G. Controlled trial of suloctidil in intermittent claudication. *J Cardivasc Pharmacol* 1981;3:279-86. 93 Signorini GP, Salmistraro G, Maraglino G. Efficacy of indobufen in the
- treatment of intermittent claudication. Angiology 1988;39:742-5.
- 94 Aukland A, Hurlow RA, George AJ, Stuart J. Platelet inhibition with ticlopidine in atherosclerotic intermittent claudication. J Clin Pathol 1982;35:740-3.
- 95 Hess H, Keil-Kuri E. Theoretische grundlagen der Prophylaxe Obliterinerender Arteriopathien mit Aggregationshemmern und Ergebnisse einer Langzeitstudie mit ASS (Colfarit). In: Proceedings of the colfarit sym-
- posion III. Cologne, 1975:80-87. 96 Stiegler H, Hess H, Mietaschk A, Trampisch HJ, Ingrisch H. Einfluss von Ticlopidin auf die Periphere Obliterierende Arteriophie. Dtsch Med Wochenschr 1984;109:1240-3.
- 97 Cloarec M, Caillard P, Mouren X. Double blind clinical trial of ticlopidine versus placebo in peripheral atherosclerotic disease of the legs. *Thromb Res* 1986;suppl VI:160.

 98 Balsano F, Coccheri S, Libretti A, Nenci GG, Catalano M, Fortunato G,
- et al. Ticlopidine in the treatment of intermittent claudication: a
- 21-month double blind trial. *J Lab Clin Med* 1989;114:84-91. 99 Arcan JC, Blanchard J, Boissel JP, Destors JM, Panak E. Multicentre double blind study of ticlopidine in the treatment of intermittent claudica
- tion and the prevention of its complications. *Angiology* 1988;39:802-11.

 100 Destors JM, Arcan JC. Evaluation des médicaments par voie orale de la claudication intermittente des membres inférieurs à la phase III des essais cliniques. Choix retenus dans l'étude AĈT. Thérapie 1985;40:451-8
- 101 Katsumura T, Mishima Y, Kamiya K, Sakaguchi S, Tanabe T, Sakuma A. Therapeutic effect of ticlopidine, a new inhibitor of platelet aggregation, on chronic arterial occlusive diseases, a double blind study versus placebo. Angiology 1982;33:357-67.
- 102 Ellis DJ. Treatment of intermittent claudication with ticlopidine. In: Proceedings of International Committee on Thrombosis and Haemostasis. 32nd Meeting, 1986:63:60. (Abstract addendum.)
- 103 Hess H, Mietaschk A, Deichsel G. Drug-induced inhibition of platelet function delays progression of peripheral occlusive arterial disease. A prospective double-blind arteriographically controlled trial. Lancet 1985:i:415-9.
- 104 Colwell JA, Bingham SF, Abraira C, Anderson JW, Kwaan HC, et al for the Co-operative Study Group. VA co-operative study on antiplatelet agents in diabetic patients after amputation for gangrene: I. Design, methods and baseline characteristics. Controlled Clin Trials 1984;5:
- 105 Colwell JA, Bingham SF, Abraira C, Anderson JW, Comstock JP, Kwaan HC, et al. Veterans Administration co-operative study on antiplatelet agents in diabetic patients after amputation for gangrene: II. Effects of aspirin and dipyridamole on atherosclerotic vascular disease rates Diabetes Care 1986;9:140-8.
- 106 Schoop W, Levy H, Schoop B, Gaentzsch A. Experimentelle und Klinische Studien zu der sekundaren Prevention der Peripheren Arteri-

- osklerose. In: Bollinger A, Rhyner K, eds. Thrombozytenfunktionshemmer, Wirkungsmechanismen, Dosierung und Praktische. Stuttgart: Thieme, 1983:49-58.
- Schoop W. Levy H. Prevention of peripheral arterial occlusive disease with antiagreggants. Thromb Haemost 1983;50:137.
- Schoop W. Spatergebnisse bei Konservitiver Therapie der Arteriellen
- Verschlusskrankheit. *Der Internist* 1984;25:429-33.

 109 Janzon L, Berqvist D, Boberg J. Boberg M, Eriksson I, Lindgarde F, et al.

 Prevention of myocardial infarction and stroke in patients with intermittent claudication, effects of ticlopidine. Results from STIMS, the Swedish ticlopidine multicentre study. I Intern Med 1990;227:301-8.
- 110 Balsano F. Violi F. and ADEP Group. Effect of picotamide on the clinical progression of peripheral vascular disease. A double blind placebo controlled study. *Circulation* 1993;87:1563-9.
- Moher M, Lancaster T. Who needs antiplatelet therapy? Br J Gen Pract 1996;46:367-70.
- 112 Pollock A, Wright AD. The effect of ticlopidine on platelet function in patients with diabetic peripheral arterial disease. Guildford: Sanofi Winthrop, 1979. (Sanofi internal report 105062/0019.)
- 113 Nyberg G, Larsson O, Westberg NG, Aurell M, Jagenburg R, Blohme G. A platelet aggregation inhibitor—ticlopidine—in diabetic nephropathy: a randomized double blind study. Clin Nephrol 1984;21:184-7.
- 114 Pannebakker MAUI, Jonker JJC, Den Ottolander GJH. Influence of sulphinpyrazone on diabetic vascular complications. In: Proceedings of VI international congress of Mediterranean League Against Thrombosis. Monte Carlo, 1980. (Abstract 167.)
- 115 Oakley NW, Dormandy JA, Flute PT. Investigation of the effect of ticlopidine in the incidence of cardiovascular events in selected high risk patients with diabetes. Guildford: Sanofi Winthrop 1983. (Sanofi internal report 105062/0019.)
- 116 Belgian Ticlopidine Retinopathy Study Group (BTRS). Clinical study of
- ticlopidine in diabetic retinopathy. Ophthalmologica 1999;204:4-12.

 117 DAMAD Study Group. Effect of aspirin alone and aspirin plus dipyridamole in early diabetic retinopathy—a multi-centre randomized controlled clinical trial. *Diabetes* 1989;38:491-8.
- 118 Mirouze J on behalf of TIMAD Study Group. Ticlopidine in the secondary prevention of early diabetes-related microangiopathy: protocol of a multicentre therapeutic study (TIMAD study). Agents Actions 1984;
- multicentre and applications and morbidity in ETDRS Investigators. Aspirin effects on mortality and morbidity in ETDRS Investigators. Aspirin effects on mortality and morbidity in study report 14. *JAMA* 1992;268:1292-300.
- 120 ETDRS Research Group. Effects of aspirin treatment on diabetic retin-opathy: ETDRS report number 8. Ophthalmology 1991;98(suppl):757-65.
- Breddin K, Loew D, Lechner K, Uberla KK, Walter E. Secondary prevention of myocardial infarction: a comparison of acetylsalicylic acid, placebo and phenprocoumon. *Haemostasis* 1980;9:325-44.
- 122 Uberla K. Multicentre two year prospective study on the prevention of secondary myocardial infarction by ASA in comparison with phenprocoumon and placebo. In: Breddin K, ed. Acetylsalicylic acid in cerebral ischaemia and coronary heart disease. Stuttgart: Schattauer, 1978:159-69.
- 123 British National Formulary. No 32. London: British Medical Association and Royal Pharmaceutical Society of Great Britain, 1996.
- Roderick PJ, Wilkes HC, Meade TW. The gastrointestinal toxicity of aspirin: an overview of randomised controlled trials. Br J Clin Pharm 1993;35:219-26.

(Accepted 16 December 1997)

A memorable patient

Truth drug

It was Remembrance Day poppies that brought him to mind and made me seek out his picture. He was my last first world war veteran patient and he had kindly allowed me to record his wound for posterity.

I found the photos and looked on a dignified elderly man with a ramrod back and military moustache and a foreshortened upper limb that hung strangely and limply by his side. I remember his whole persona exuded a rigid, traditional, regimental career and yet it was founded on just six months of soldiering. As old as the century he had, as was typical, lied about his age and volunteered. After some square bashing and rudimentary combat training he had been landed in France, picked his regiment on site from the lists of the decimated, and, as he had chosen a Scottish regiment, was put in a kilt and sent to the front. There, a German shell had bisected his arm at the elbow. Even though he was slipping in and out of consciousness, he was ordered to make his own way back, unaided, through the lines and mud, under fire. At some point he was picked up by a horse drawn ambulance and taken to a casualty clearing station run by the French. The military doctor felt that the limb was salvageable because the injury was bone and soft tissue, and the hand was neurologically intact.

Many operations and months later he did indeed come out with a limb which, as I had discovered when I shook his hand, he could make perfectly functional with some tricks of movement and positioning, and it had served him well for 70 years. He discoursed the fate of his comrades of the day while I conducted the anaesthetic preliminaries for his prostatectomy. As he felt the influence of the thiopentone sapping his consciousness, he began to overemphasise his words, but clearly, coherently, and very determinedly, he expressed a last thought: "That bastard Haig should have been hung."

Ian D Conacher, consultant anaesthetist, Newcastle upon Tyne

We welcome articles up to 600 words on topics such as A memorable patient, A paper that changed my practice, My most unfortunate mistake, or any other piece conveying instruction, pathos, or humour. If possible the article should be supplied on a disk. Permission is needed from the patient or a relative if an identifiable patient is referred to. We also welcome contributions for "Endpieces," consisting of quotations of up to 80 words (but most are considerably shorter) from any source, ancient or modern, which have appealed to the reader.

Health needs assessment

Development and importance of health needs assessment

John Wright, Rhys Williams, John R Wilkinson

This is the first in a series of six articles describing approaches to and topics for health needs assessment, and how the results can be used effectively

Bradford Hospitals NHS Trust, Bradford Royal Infirmary, Bradford BD9 6RJ John Wright, consultant in epidemiology and public health medicine

Nuffield Institute for Health, Leeds LS2 9PL Rhys Williams,

Rhys Williams, professor of epidemiology and public health

North Yorkshire Health Authority, York YO1 1PE John R Wilkinson, deputy director of public health

Correspondence to: Dr Wright wrightj@brihosp.mhs. compuserve.com

Series editor: John Wright

BMJ 1998;316:1310-3

Most doctors are used to assessing the health needs of their individual patients. Through professional training and clinical experience we have developed a systematic approach to this assessment and we use it before we start a treatment that we believe to be effective. Such a systematic approach has often been missing when it comes to assessing the health needs of a local or practice population.

The health needs of individual patients coming through the consulting room door may not reflect the wider health needs of the community. If people have a health problem that they believe cannot be helped by the health service, then they will not attend. For example, many people with angina or multiple sclerosis are not known to either their local general practitioner or to a hospital specialist. Other groups of patients who may need health care but do not demand it include homeless people and people with chronic mental illness.

Distinguishing between individual needs and the wider needs of the community is important in the planning and provision of local health services. If these needs are ignored then there is a danger of a top-down approach to providing health services, which relies too heavily on what a few people perceive to be the needs of the population rather than what they actually are.

What is health needs assessment?

Health needs assessment is a new phrase to describe the development and refinement of well established approaches to understanding the needs of a local population. In the 19th century the first medical officers for health were responsible for assessing the needs of their local populations. More recently, in the 1970s the Resource Allocation Working Party assessed relative health needs on the basis of standardised mortality ratios and socioeconomic deprivation in different populations, and it used this formula to recommend fairer redistribution of health service resources. The 1992 Health of the Nation initiative was a government attempt to assess national health needs and determine priorities for improving health. Health



Summary points

Health needs assessment is the systematic approach to ensuring that the health service uses its resources to improve the health of the population in the most efficient way

It involves epidemiological, qualitative, and comparative methods to describe health problems of a population; identify inequalities in health and access to services; and determine priorities for the most effective use of resources

Health needs are those that can benefit from health care or from wider social and environmental changes

Successful health needs assessments require a practical understanding of what is involved, the time and resources necessary to undertake assessments, and sufficient integration of the results into planning and commissioning of local services

needs assessment has come to mean an objective and valid method of tailoring health services—an evidence based approach to commissioning and planning health services.

Although health needs assessments have traditionally been undertaken by public health professionals looking at their local population, these local health needs should be paramount to all health professionals. Hospitals and primary care teams should both aim to develop services to match the needs of their local populations. Combining population needs assessment with personal knowledge of patients' needs may help to meet this goal.⁵

Why has needs assessment become important?

The costs of health care are rising. Over the past 30 years expenditure on health care has risen much faster than the cost increases reported in other sectors of the economy, and health care is now one of the largest sectors in most developed countries. Medical advances and demographic changes will continue the upward pressure on costs.

At the same time the resources available for health care are limited. Many people have inequitable access to adequate health care, and many governments are unable to provide such care universally. In addition there is a large variation in availability and use of health care by geographical area and point of provision.⁸ Availability tends to be inversely related to the need of the population served.⁹

Another force for change is consumerism. The expectations of members of the public have led to greater concerns about the quality of the services they receive—from access and equity to appropriateness and effectiveness.

These factors have triggered reforms of health services in both developed and developing countries. In Britain these reforms resulted in the separation of the responsibility for financing health care from its provision and in the establishment of a purchasing role for health authorities and general practitioners. Health authorities had greater opportunities to try to tailor local services to their own populations, and the 1990 National Health Service Act required health authorities to assess health needs of their populations and to use these assessments to set priorities to improve the health of their local population.¹⁰ This has been reinforced by more recent work on inequalities in health, suggesting that health authorities should undertake "equity audits" to determine if healthcare resources are being used in accordance with need.12

At a primary care level, through fundholding, locality commissioning, and total purchasing projects, general practitioners have become more central to strategic planning and development of health services. With this increased commissioning power has come the increased expectation from patients and politicians that decision making would reflect local and national priorities, promoting effective and equitable care on the basis of need.¹³ The Labour government has committed itself to ensuring access to treatment according to "need and need alone," and the key functions of primary care groups will be to plan, commission, and monitor local health services to meet identified local needs.¹⁴ ¹⁵

Needs

Doctors, sociologists, philosophers, and economists all have different views of what needs are. ^{16–20} In recognition of the scarcity of resources available to meet these needs, health needs are often differentiated as needs, demands, and supply (fig 1).

Need in health care is commonly defined as the capacity to benefit. If health needs are to be identified then an effective intervention should be available to meet these needs and improve health. There will be no benefit from an intervention that is not effective or if there are no resources available.

Demand is what patients ask for; it is the needs that most doctors encounter. General practitioners have a key role as gatekeepers in controlling this demand, and waiting lists become a surrogate marker and an influence on this demand. Demand from patients for a service can depend on the characteristics of the patient or on the media's interest in the service. Demand can also be induced by supply: geographical variation in hospital admission rates is explained more by the supply of hospital beds than by indicators of mortality^{21 22}; referral rates of general practitioners owe more to the characteristics of individual doctors than to the health of their populations.²³

Supply is the health care provided. This will depend on the interests of health professionals, the priorities of politicians, and the amount of money available. National health technology assessment programmes have developed in recognition of the importance of assessing the supply of new services and treatments before their widespread introduction.

Need, demand, and supply overlap, and this relation is important to consider when assessing health needs (fig 2).²⁰

Health needs

The World Health Organisation's definition of health is often used: "Health is a state of complete physical, psychological, and social wellbeing and not simply the absence of disease or infirmity." A more romantic definition would be Freud's: "Health is the ability to work and to love."

Healthcare needs are those that can benefit from health care (health education, disease prevention, diagnosis, treatment, rehabilitation, terminal care). Most doctors will consider needs in terms of healthcare services that they can supply. Patients, however, may have a different view of what would make them healthier—for example, a job, a bus route to the hospital or health centre, or decent housing.

Health needs incorporate the wider social and environmental determinants of health, such as deprivation, housing, diet, education, employment. This wider definition allows us to look beyond the confines of the medical model based on health services, to the wider influences on health (box). Health needs of a population will be constantly changing, and many will not be amenable to medical intervention.

Influences on health

- Environment: housing, education, socioeconomic status, pollution
- Behaviour: diet, smoking, exercise
- Genes: inherited health potential
- Health care: including primary, secondary, and tertiary prevention

Health needs assessment

Assessment of health needs is not simply a process of listening to patients or relying on personal experience. It is a systematic method of identifying unmet health and healthcare needs of a population and making changes to meet these unmet needs. It involves an epidemiological and qualitative approach to determining priorities which incorporates clinical and cost effectiveness and patients' perspectives. This approach must balance clinical, ethical, and economic considerations of need—that is, what should be done, what can be done, and what can be afforded.²⁵

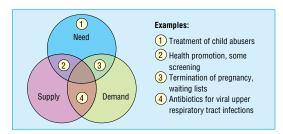


Fig 2 Relation between need, supply, and demand—central area shows ideal relation. Modified from Stevens and Raferty.²⁴

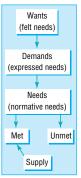


Fig 1 Different aspects of needs

Health needs assessment should not just be a method of measuring ill health, as this assumes that something can be done to tackle it. Incorporating the concept of a capacity to benefit introduces the importance of effectiveness of health interventions and attempts to make explicit what benefits are being pursued. Economists argue that the capacity to benefit is always going to be greater than available resources and that health needs assessment should also incorporate questions of priority setting,²⁶ suggesting that many needs assessments are simply distractions from the difficult decisions of rationing.²⁷

For individual practices and health professionals, health needs assessment provides the opportunity for:

- Describing the patterns of disease in the local population and the differences from district, regional, or national disease patterns;
- Learning more about the needs and priorities of their patients and the local population;
- Highlighting the areas of unmet need and providing a clear set of objectives to work towards to meet these needs;
- Deciding rationally how to use resources to improve their local population's health in the most effective and efficient way;
- Influencing policy, interagency collaboration, or research and development priorities.

Importantly, health needs assessment also provides a method of monitoring and promoting equity in the provision and use of health services and addressing inequalities in health.²⁸

The importance of assessing health needs rather than reacting to health demands is widely recognised, and there are many examples of needs assessment in primary and secondary care.^{21 30 31}

There is no easy, quick-fix recipe for health needs assessment. Different topics will require different approaches. These may involve a combination of qualitative and quantitative research methods to collect original information, or adapting and transferring what is already known or available.

The stimulus for these assessments is often the personal interest of an individual or the availability of new funding for the development of health services. However, assessments should also be prompted by the importance of the health problem (in terms of frequency, impact, or cost), the occurrence of critical incidents (the death of a patient turned away because the intensive care unit is full), evidence of effectiveness of an intervention, or publication of new research findings about the burden of a disease.

Why do projects fail?

Some needs assessments have been more successful than others. Projects may fail for several reasons. 31-33

Firstly, what is involved in assessing health needs and how it should be undertaken may not be understood. Educational strategies can improve the understanding and necessary skills of health professionals, and local public health teams can provide valuable support and guidance. Common sense can be a more important asset than detailed methodological understanding.³⁴ Starting with a simple and well defined health topic can provide experience and encourage success.

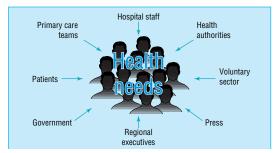
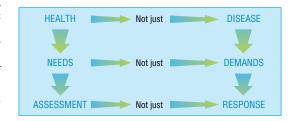


Fig 3 Contributors to needs assessment



Secondly, projects may fail because of a lack of time, resources, or commitment. The time and resources required can be small when shared among professionals in a team, and such sharing has the potential to be team building. Involving other organisations such as social services, local authorities, or voluntary groups can provide similar advantages and encourage multiagency working. Integration of needs assessment into audit and education can also provide better use of scarce time. Such investment of time and effort is likely to become increasingly necessary in order to justify extra resources.

A third reason is the failure to integrate the results with planning and purchasing intentions to ensure change. The planning cycle should begin with the assessment of need.28 Objectives must be clearly defined (box) and relevant stakeholders or agencies-be they primary care teams, hospital staff, health authorities, the voluntary sector, the media, regional executives, government, or patients-must be involved appropriately (fig 3). Although such an assessment may produce a multitude of needs, criteria can be used to prioritise these needs-for example, the importance of a problem in terms of frequency or severity, the evidence of effectiveness of interventions, or the feasibility for change. Needs assessments that do not include sufficient attention to implementation will become little more than academic or public relations

This series will describe the different approaches to assessing health needs, how to identify topics for health

Questions to ask when assessing health needs

- What is the problem?
- What is the size and nature of the problem?
- What are the current services?
- What do patients want?
- What are the most appropriate and effective (clinical and cost) solutions?
- What are the resource implications?
- What are the outcomes to evaluate change and the criteria to audit success?

needs assessments, which practical approaches can be taken, and how the results can be used effectively to improve the health of local populations. It will give examples of needs assessment from primary care but will also cover the specific problems of needs assessment for hard to reach groups. Many of the techniques of community appraisals used in needs assessment originate from experience in developing countries, and some of the lessons from this experience will be described.

We are grateful to John Bibby and Dee Kyle for their valuable contributions and to Margaret Haigh for secretarial support.

Funding: None.

Conflict of interest: None.

- Smith R. Rationing: the search for sunlight. BMJ 1991;303:1561-2.
- Ford HL, Gerry E, Airey CM, Johnson MH, Williams DRR. The prevalence of multiple sclerosis in the Leeds District. J Neurol Neurosurg Psychiatry 1998 (in press).
- Department of Health and Social Security. Sharing resources for health in England: report of the Resource Allocation Working Party. London: HMSO,
- Department of Health. The Health of the Nation: a strategy for health in England. London: HMSO, 1992. (Cm 1986.)
- Shanks J, Kheraj S, Fish S. Better ways of assessing health needs in primary care. BMJ 1995;310:480-1.
- Organisation for Economic Cooperation and Development. Health care systems in transition: the search for efficiency. Paris: OECD, 1990. (Social
- policy studies No 7.) Harrison A, Dixon J, New B, Judge K. Funding the NHS. Can the NHS cope in future. BMJ 1997;314:139-42.
- Anderson TV, Mooney G. The challenge of medical practice variations. London: McMillan, 1990.
- Tudor Hart J. The inverse care law. Lancet 1971;i:405-12.
- 10 Department of Health. Working for patients. London: HMSO, 1989. (Cm
- 11 National Health Service Management Executive. Assessing health care
- needs. Leeds: NHSME, 1991. (DHA project discussion paper.)
 Variations Subgroup of the Chief Medical Officer's Health of the Nation
 Working Group. Variations in health. What can the Department of Health and the NHS do? London: Department of Health, 1995.

- 13 National Health Service Executive. An accountability framework for GP fundholding: towards a primary care led NHS. Leeds: NHSE, 1994. (EL (94)54)
- 14 Secretary of State for Scotland. Designed to care. Edinburgh: Department of Home and Health, Scottish Office, 1997.
- 15 NHS Executive. The new NHS. London: Stationery Office, 1997. (Cm 3807.)
- 16 Culyer A J. Need and the National Health Service. London: Martin Robert-
- 17 Bradshaw J. A taxonomy of social need. In: McLachlan G, ed. Problems and progress in medical care. 7th series. London: Oxford University Press, 1972.
- 18 Frankel S. Health needs, health-care requirements and the myth of infinite demand. Lancet 1991:337:1588-9.
- 19 Williams. Priorities not needs. In: Corden A, Robertson G, Tolley K, eds. Meeting needs. Aldershot: Avebury Gower, 1992.
- 20 Stevens A, Gabbay J. Needs assessment needs assessment. Health Trends 1991;23:20-3.
- 21 Feldstein MS. Effects of differences in hospital bed scarcity on type of use. BMJ 1964;ii:562-5.
- 22 Kirkup B, Forster D. How will health needs be measured in districts? Implications of variations in hospital use. J Public Health Med 1990;12:
- 23 Wilkin D. Patterns of referral: explaining variation. In: Roland M, Coulter A, eds. Hospital referrals. Oxford: Oxford University Press, 1992.
- 24 Stevens A, Raferty J, eds. Health care needs assessment—the epidemiologically based needs assessment reviews. Oxford: Radcliffe Medical Press, 1994.
- 25 Black D. A doctor looks at health economics. Office of Health Economics annual lecture. London: OHE, 1994.
- 26 Donaldson C, Mooney G. Needs assessment, priority setting, and contracts for health care: an economic view. *BMJ* 1991;303:1529-30.
- 27 Mooney G. Key issues in health economics. Hemel Hempstead: Harvester Wheatsheaf, 1994.
- 28 Womersley J, McCauley D. Tailoring health services to the needs of individual communities. J Publ Health Med 1987;41:190-5.
- 29 Majeed FA, Chaturvedi N, Reading R, Ben-Shlomo Y. Monitoring and
- promoting equity in primary and secondary care. BMJ 1994;308:1426-9.
 30 Gillam SJ, Murray SA. Needs assessment in general practice. London: Royal College of General Practitioners, 1996. (Occasional paper 73.)
- 31 Jordan J, Wright J, Wilkinson J, Williams DRR. Health needs assessment in primary care: a study of the understanding and experience in three districts. Leeds: Nuffield Institute for Health, 1996.
- 32 London Health Economics Consortium. Local health and the vocal community, a review of developing practice in community based health needs assessment. London: London Primary Health Care Forum, 1996.
- 33 Jordan J, Wright J. Making sense of health needs assessment. Br J Gen Pract 1997;48:695-6.
- 34 Gillam S. Assessing the health care needs of populations-the general practitioner's contribution [editorial]. Br J Gen Pract 1992;42:404-5.

These articles have been adapted from Health Needs Assessment in Practice, edited by John Wright, which will be published in July

Coping with loss

The dying adult

Colin Murray Parkes

This paper focuses on two common problems that arise when people come close to death, fear and grief. Fear is the psychological reaction to danger; grief the reaction to the numerous losses that are likely to occur in the course of an illness that is approaching a fatal outcome. Both can be expected to arise in patients, their families, and-though we are reluctant to admit it-in their doctors and other carers. Both fear and grief need to be taken into account if we are to mitigate the psychological pains of dying.

Fear

Though it may seem obvious that people who are dying are likely to be afraid, we should not assume that we know what they fear. The box shows the fears, in approximate order of frequency, expressed to me by patients in a hospice. It is clear that fears of death itself come quite far down on the list. Difficult to quantify but of particular importance is reflected fear, the fear that people see in the eyes of those around them or hear in the questions that are not asked. Many problems in

Summary points

We should never assume that we know what people with terminal illness fear

Most patients will benefit if we can help them to feel secure enough to share their fears

Fear can aggravate pain, and pain fear

Patients with life threatening illnesses experience a series of losses as the illness progresses

Grief is natural and needs to be acknowledged and expressed

communication arise out of fear, and we may need to take time to create trust and a safe place in which people can begin to talk about the things that make them feel unsafe.

This is the seventh in a series of 10 articles dealing with the different types of loss that doctors will meet in their practice

St Christopher's Hospice, Sydenham, London SÉ26 6DZ Colin Murray consultant psychiatrist

cmparkes@aol.com

Series editors: Colin Murray Parkes and Andrew Markus

BMJ 1998;316:1313-5

Causes of fear in people with life threatening illness

- Fear of separation from loved people, homes, jobs, etc.
- Fear of becoming a burden to others
- Fear of losing control
- Fear for dependents
- Fear of pain or other worsening symptoms
- Fear of being unable to complete life tasks or responsibilities
- Fear of dying
- · Fear of being dead
- · Fear of the fears of others (reflected fear)

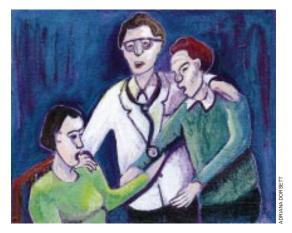
Knowing about dying

Few people in our society know how people die. Their image of death comes from horror comics, dramatic representations in the media, and the scare stories that get passed around a family when someone dies a painful death. To most people a little illness is bad; as the illness progresses they expect the symptoms to get worse, and it is logical to expect that, at the moment of death, every symptom will be as dreadful as it can be. This image of death as the peak of suffering bears little resemblance to the quiet slipping away of many a patient in the late stage of cancer whose symptoms are being relieved and who is surrounded by a loving family.

Patients who have learned to trust the doctor may well be able to share these fears and be reassured. But words like "cancer" and "death" are tainted with so many horrific accretions that it may be hard for the patient to use them. We may need to show by our own matter of fact manner that we are not afraid to speak the unspeakable. This does not mean that we should force people to face facts that they are not yet ready to face, but there are many ways in which we can ease things along. By inviting questions, answering honestly the questions that are asked (but not necessarily the ones that are not asked), and giving reassurance where reassurance is possible and emotional support to grieving when it is not, we shall often help patients and their families to make something good of the time that remains to them.

Fear builds on fear

Fear often aggravates itself. Thus people with cancer may lie in bed worrying about their cancer. We do not



need to know about the gate theory of pain² to know that a minor ache or pain will get worse if we worry about it. Though many of the pains of cancer patients are not directly caused by cancer, if we are to relieve them we need to take them seriously, discover their cause, and give an explanation and reassurance that will convince the patient.

The list of physical symptoms that can be caused by fear is long, ranging from the physiological effects of disturbance of the autonomic nervous system to the secondary effects of overbreathing. When they occur in people who are already physically ill the resulting tangle of physical and psychosomatic symptoms is not always easy to unravel.

Although we often know that a particular case of cancer is likely to end fatally we can seldom predict how long it will take.³ One of the hardest problems is living with uncertainty, and patients will often demand to be told how long they have to live. Doctors should not make predictions that will probably be wrong, and they need to be prepared to support their patients while they wait for the situation to clarify itself.

It is often wise to resist pressure to carry out another operation or another battery of tests that will be unlikely to leave the patient any better off. Clearly, if we are to be of help we must tread a line between alarmist overinvestigation and facile reassurance.

Something can be done

There is always something that can be done to help people through the long periods of waiting, be it a regular chat with a trusted doctor with whom patients can air their fears or a minor tranquilliser that may break the vicious circle of fear and symptoms. Diazepam still has a place in the short term treatment of anxiety, particularly in patients whose life is not likely to be long enough for habituation to become a problem. Several antidepressants, including 5-HT reuptake inhibitors (such as fluoxetine) as well as the more sedative of the tricyclics (such as amitriptyline and dothiepin), have anxiolytic properties that may benefit people who are anxious and depressed.

Cancer invades families, and it is important to reach out to all of those whose lives are touched by it. Support given to a patient's spouse indirectly helps the patient. That said, it is common to find that, as long as the patient is alive, members of the family will minimise their needs for help and support. We should be aware that things are not always as satisfactory as they seem. One way to tap into the needs of the family is to invite them to help us to draw a family tree. This not only tells us who exists, it also shows our interest and allows family members to share their fears and other feelings about each other.

The dying patient's griefs

Although the course of cancers and other fatal diseases can seldom be predicted, these diseases do tend to progress in a stepwise way. Initially the prognosis may not be bad—an operation and a course of radiotherapy or chemotherapy may offer the hope of cure, and most patients and doctors prefer to adopt an optimistic attitude. This does not mean that nothing has been lost. Quite apart from the physical mutilation and loss of function that can result from drastic treatments for

drastic diseases, patients who have suffered a life threatening illness will never again be as secure as once they were. We need to be at hand and to encourage them to share their perception of the implications of the illness for their life.⁴

If people have been helped to express their grief at the losses that have occurred at an early stage of an illness, they will be more likely to be able to cope effectively when they are faced with another set of losses

Expressing grief

If people have been helped to express their grief at the real losses that have occurred at an early stage of an illness they will be more likely to be able to cope effectively when they are faced with the next set of losses. After a period of relative quiescence a new symptom may arise; it is investigated and is found to indicate that the cancer has spread. This time it is more difficult to deny the fact that things are not going the way they should. Perhaps another course of chemotherapy is given, but the benefits are less and the patient's general condition is likely to be deteriorating. It becomes obvious that this person will never be able to return to work, and this may be a real cause for grief.

One of the most disturbing losses is the loss of the respect of others that is reflected in their expressions of pity, for pity, unlike sympathy, demeans the person pitied. To some extent this is counteracted if we and the patient's family continue to treat the patient with respect. The loss of respect will be aggravated if we patronise, infantilise, or denigrate the patient.

The stages of dying described by Elizabeth Kubler Ross-denial, anger, bargaining, depression, and acceptance5-have rightly been criticised: they do not correspond to the unpredictable way in which most cancers progress.6 Even so there is a tendency for people to move, in fits and starts, from a state of relative denial of the true situation to some kind of acceptance. Some never accept the situation and continue to expect to get better; others seem to embrace the prospect of death. In between there is a majority who oscillate back and forth between courageous attempts to face facts and episodes of optimism that are quite unrealistic. These fluctuations make it difficult to get reliable measures of "insight," and most research in this field is of dubious value. Anger and depression, which are frequent accompaniments of grief, and "bargaining," by which Ross means the attempts that cancer patients often make to accept one sacrifice in the expectation of a reward ("I don't mind losing my hair

Losses of patients with life threatening illness

- · Loss of security
- · Loss of physical functions
- Loss of body image
- Loss of power or strength
- Loss of independence
- Loss of self esteem
- Loss of the respect of others
- · Loss of future

as long as I can be kept alive until my daughter's wedding"), are often found.

Coping and letting go

In the later stages the loss of all of the appetites, including the appetite for life, makes acceptance easier. Many patients find it easier to "let go" of life because of this.

In all cases the patient's previous personality and their accustomed ways of viewing the world and coping with problems will colour the way they cope with illness and death. Those whose experience of life has left them confident in their own worth and trusting in the love of others usually seem to feel secure in the face of death; others, who may be less secure in both their confidence in themselves and their trust in others (and ultimately in God), may find it hard to step into the unknown. Spiritual values that arise from having found meaning in life make it easier to find meaning in death, but this should not be confused with religiosity, which is often an attempt to propitiate God and seek God's protection. This kind of faith often breaks down when God fails to keep his side of the supposed bargain.

Whatever our own faith, it is important to respect the faiths of others and to resist the temptation to proselytise. Each person has their own religious language, and we must learn that language if we are to communicate successfully on spiritual issues. Many patients will enjoy the opportunity to share with us their attempts to make sense of their lives, and it is our privilege to be a part of this search.

The doctor's grief

To help those who are dying we must be prepared to share their griefs and stay with them in their fear. Sometimes we have the satisfaction of knowing that the pain we have shared has been followed by a peaceful and even a triumphant end and this makes it easier to bear, but there are no guarantees. Sometimes death is a messy and a bitter business which leaves us harrowed and ashamed. Maybe the person who died has triggered off our own most dreaded fears; maybe we feel responsible for their suffering or their death.

At such times we too will need the support of someone we can trust—and we should not feel ashamed to ask for it. We too will need to grieve; if it is all right for our patients to cry, it should be all right for us too. We are not supermen and superwomen who can always be counted on to give help but never need it for ourselves, and we must be prepared to let others take over, for a while, the daily routines in order to give us space to grieve. It is a sign of maturity to know when to ask for help, and the wise doctor will have worked out systems of support to meet a range of needs.⁷

Funding: No additional funding. Conflict of interest: None.

- Parkes CM.Attachment and autonomy at the end of life. In: Gosling R, ed. Support, innovation, and autonomy. London: Tavistock, 1973:151-66.
- Melzack R, Wall PD. Pain mechanisms. Science 1965;150:971-9.
- 3 Parkes CM. Accuracy of predictions of survival in later stages of cancer. BMJ 1972;i:29-31.
- Maguire P, Parkes CM. Loss and the reaction to physical disablement and surgery. BMJ 1998;316:1086-8.
- Ross EK. On death and dying. London: Tavistock, 1970.
- 6 Schultz R, Aderman D. Clinical research and the stages of dying. *Omega* 1974;5:137-43.
- 7 Bennet G. The doctor's losses. BMJ (in press).

The articles in this series are adapted from *Coping with Loss*, edited by Colin Murray Parkes and Andrew Markus, which will be published in July.