

HEALTH EFFECTS OF RESPIRABLE DUST FROM OPENCAST COAL MINING

J.F. Munro*

G.K. Crompton†

INTRODUCTION

The recent ruling of the High Court in England that chronic obstructive pulmonary disease (COPD) is a compensatable condition in miners and ex-miners¹ has brought about renewed attention to the health hazards experienced by workers associated with the coal mining industry. At present a substantial and increasing proportion of the UK coal output is extracted by surface opencast methods, with a predicted output for the current financial year of 17 million tonnes, with 50% coming from England and almost 40% from Scotland.²

Respirable dust, i.e. inspired dust which reaches the alveoli, is generally considered to comprise particles of less than 2.5 microns aero-dynamic diameter. This article reviews the health effects of respirable dust from opencast coal mining on the workforce and in the community.

PM10 DUST

Most recent studies on the effects of air pollutants have measured ambient concentrations of PM10 dust, which can be defined as dust with an aero-dynamic diameter of 10 microns or less. Because PM10 dust is measured as a concentration or mass per cubic metre, the number of particles and the composition of the respirable dust aerosol may change considerably with little or no detectable effect on the overall concentration of PM10 dust. In 1995 the Committee of the Medical Effects on Air Pollutants (COMEAP) concluded that there was no clear evidence that associations with adverse health effects of PM10 dust were restricted to specific types of particles.³ COMEAP considered that it would be imprudent not to regard the acute associations as causal and that it would 'be prudent to consider the associations between long-term exposure to particles and chronic effects as causal'. There is no threshold level, however the lower the concentration, the lesser the effects; the greater the concentration, the more the effects.

COMEAP has recently quantified the acute effects of air pollution by PM10 particulate matter linking a change per $\mu\text{g}/\text{m}^3$ with alterations to acute mortality rates from all causes, respiratory hospital admissions and cardiovascular hospital admissions.⁴ Likewise, COMEAP has estimated the influence in asthmatics of changes in the ambient PM10 levels on symptoms and bronchodilator usage and evidence of a clear association has emerged.⁵ However, a European investigation⁶ found this association in some studies⁷⁻¹¹ but not in others; such an observation is in keeping with the possibility that the composition of PM10 dust may be at

least as important as the concentration. In other respects the evidence is more consistent; studies from the USA¹² and the UK¹³ indicate that an acute rise of $10 \mu\text{g}/\text{m}^3$ in PM10 will result in the number of deaths increasing by 1%. The possibility that PM10 dust may exert a chronic adverse effect on health and mortality is even more disquieting.¹⁴ In their summary, COMEAP concluded that 'while we accept that we may have understated the overall effects of air pollution in the UK we can say with reasonable confidence that the effects are likely to be at least as large as stated in this report.'³

DUST FROM OPENCAST MINING

The Quality of Urban Air Review Group has estimated the UK total PM10 emissions in 1995 at 232,000 tonnes, with 12% (29,000 tonnes) derived from mining and quarrying.¹⁵ Dust from opencast mining has two special features: it contains particles of shale and a substantial component of it is derived from diesel engine emissions.

Coal dust

Coal dust ranges from 1 to 10,000 microns. The larger particles are not respirable and comprise most of the mass. It seems improbable that opencast mining could produce concentrations exceeding the present UK standard of 2 milligrams/ m^3 of respirable dust. However, the mass of respired dust is a major factor in producing COPD¹⁶ and coal dust simple pneumoconiosis (CDSP), with the duration of exposure possibly being more important than dust concentration.¹⁷ Other contributory factors in COPD, but not in pneumoconiosis, include the quality of the coal and its quartz content, and associated cigarette smoking. A further major factor is attributable to individual susceptibility.

In CDSP, the inflammatory response produced by alveolar macrophage disintegration may ultimately result in progressive massive fibrosis (PMF) with the process becoming self-perpetuating. It follows that any respired coal dust is clinically relevant though the effect may not always be clinically detectable.

Diesel emission particles (DEP)

Diesel engine combustion from vehicles has been estimated to be the single largest source of PM10 emissions, comprising, in 1995, 18% (43,000 tonnes) of the total. This represents a fall of 6,000 tonnes from 1993.¹⁸ Indeed a principal objective of the UK national strategy for maintaining air quality is the progressive reduction of emission from diesel engines.¹⁹ Composition of PM2.5 dust (< 2.5 microns), which may be responsible for much of the adverse health effect of PM10 dust, is dominated by particles from secondary production and from vehicle exhaust emissions, DEPs in particular.

At a recent Public Local Inquiry to extract coal by opencast methods the appellants stated that the on-site

*Honorary Fellow, Faculty of Medicine, Edinburgh University

†Physician in Respiratory Medicine, Western General Hospital, Edinburgh

consumption of diesel would be 50,000 litres per week at a site where it was proposed to obtain 480,000 tonnes of coal over four years.²⁰ If these data are representative, it can be estimated that the Scottish opencast sites (with an annual production of not less than six million tonnes) will involve an on-site diesel consumption in excess of two million litres per week. This fuel is used by vehicles and other machinery which do not require a road licence and until recently has not been subject to any statutory regulations regarding the production of DEPs. However, a recent EC Directive has introduced a limit for new non-road mobile machinery of 0.54g/kWh though this has yet to be implemented in the UK. The proposal could contrast with the limit of 0.15 g/kWh for on-road vehicles from September 1998.²¹

It follows that although diesel consumption per square kilometre of open-cast activity may be roughly comparable to that in an urban setting, the actual DEP production in opencast mining could be substantially greater. This on-site DEP production is not included in the estimations of the 'total' PM10 emissions from mining operations. That DEPs could be a problem has now been recognised²² but as yet no attempt has been made to quantify the nature of the environmental load.

Seaton and his colleagues have suggested that DEPs below 1 micron in diameter may remain suspended for weeks and drift many miles.²³ They have proposed that this acidic ultra-fine particulate may not only cause acute respiratory illness in susceptible individuals, but may also change blood coagulability sufficiently to account for the increase in cardiovascular deaths and acute ischaemic episodes associated with particulate pollution. They have suggested that the numbers, composition and size, rather than mass, of particles may explain 'excess deaths occurring in people who may be indoors while similar serious effects cannot be seen in industrial workers exposed to much higher concentrations of dust measured by weight'.

In considering the health effects of respirable dust arising from opencasting it may be that these can be attributed as much to diesel emission particles as to other sources of increased PM10 production.

HEALTH HAZARDS IN THE WORK FORCE

Coal dust pneumoconiosis

The occurrence of CDSP in surface coal workers was first recognised in 1928,^{24,25} and it was initially described in opencast coal miners in the USA.²⁶⁻²⁸ It may occur even when the average respirable coal mine dust is less than 1 milligram/m³. At first this condition appeared to be specifically related to anthracite coal, but more recently the Institute of Occupational Medicine (IOM) has reported the results of a cross-sectional study involving 1,224 miners at nine different sites in the UK where other varieties of coal are mined:²⁹ 4.4% of the workforce had evidence of radiological changes of $\geq 1/0$, an incidence comparable to that found in the States. There was 'a strong effect of years worked in the dustiest opencast jobs, indicating the role of occupation in causing these radiological abnormalities.'

Five workers with CDSP were diagnosed. Two had PMF, one of whom also had CDSP. In view of the probable low concentrations of respirable coal dust it may seem remarkable that any significant changes were detected. There are a number of possible explanations. Those affected may have been unduly susceptible, or they may have been living in the vicinity of the opencast site and accordingly

subjected to a prolonged duration of exposure. Alternatively there may be an additive, or even synergistic, effect between coal particles and DEPs, a possibility which might not be encountered in deep miners.

Cross-sectional studies may fail to detect the prevalence of CDSP and especially of PMF. The latter may only be diagnosed after leaving the coal industry,³⁰ and may occur in workers or ex-workers who do not fulfil the radiological criteria of CDSP.³¹

Chronic bronchitis

In the USA study, chronic bronchitis was defined as either persistent phlegm production, regardless of complaints of cough, or a moderate phlegm production with persistent cough.²⁶ In miners who had never worked underground, the overall incidence was 25.5% varying between 20.3% in non-smokers and 37.9% in smokers. The incidence rose with age and with years of exposure. There was no control group.

In the IOM study chronic bronchitis was defined as a persistent cough with persistent sputum.²⁹ 21% of smokers had chronic bronchitis in contrast with 6% of non-smokers and ex-smokers. The incidence could not be statistically related to level of dustiness of the working conditions. The overall incidence in the work force was 13% in contrast with an incidence of 22% in deep miners and 5% in the control group of telecommunication workers³² provided in the reference given by the Institute. However, the reference which should have been quoted is a subsequent report³³ which identified an incidence of 6% in telecommunication workers and 9% in an albeit relatively small sample of postmen, a group that may also experience high occupational levels of DEPs.

That there appears to be an increased prevalence of chronic bronchitis in the opencast workforce is consistent with the extensive data available from deep pit miners.¹⁶

Asthma

The IOM study failed to find any increase in the incidence of asthma (5%). The significance of this requires to be interpreted with some caution. Subjects with respiratory symptoms, particularly those with asthma, may be forced to leave the employment if their symptoms are being aggravated by occupational exposure.

HEALTH EFFECTS IN THE COMMUNITY

General considerations

There are important differences between the health effects on a local community and on the workforce. Although the workforce will be exposed to a higher concentration of respirable dust, the duration of exposure will be greater in the community by a factor of 4. If, for example, people in the community respire 10% of the additional output of dust respired by opencast workers, in 10 years they will be exposed to the same load as that experienced by the workers in 4 years.

The workforce largely comprises healthy men aged 16-65 years. The community contains many outwith this age group including the elderly, and those of all ages suffering from cardiac and respiratory disease of such a severity as to prevent them from working. The latter are most at risk from an acute rise in atmospheric pollutants. The community also includes infants and young children, who might be most adversely affected by any chronic cumulative effect. Using the example given above, the child born

into a community which then experiences 20 years' opencast mining activity, will receive the same occupational exposure as a worker in eight years.

Specific studies

Three UK studies have related opencast mining to respiratory morbidity in the community. Temple and Sykes (1992) reported the experience of a General Practice in Glynneath.³⁴ Following the onset of mining operations the practice noted a significant difference in the weekly numbers of new episodes of asthma which rose from 4.4 per week to 7.9 per week. Commenting on this report, COMEAP considered that the results were consistent with an association between the opening of the opencast mine and an increase in asthma consultation but that further work would be needed to sustain a causal hypothesis.³⁵

In October 1997 the Welsh Combined Study of Public Health published the report on the acute effects of air pollutants and the respiratory health of children in West Glamorgan, involving children aged 8-11 who were attending four schools on three sites.³⁶ These were: (1) Swansea City Centre, 129 children with high levels of urban pollution; (2) Bishopton, 136 children, a village near the sea with anticipated high ozone levels, and (3) Glynneath, 161 children at two adjacent schools in a rural community with an active opencast site more than 1.5 km from the school.³⁷ The main clinical observations from the study were that adjusted respiratory function tests were significantly lower in Swansea (17 points) and at Glynneath (11 points) than at Bishopton. The reported prevalence of asthma varied considerably from 13% at Swansea to 23.4% at Glynneath but these differences were considered to be mainly due to differing diagnostic criteria. Despite PM10 levels being higher in Swansea than in Glynneath, there was no demonstrable effect of PM10 dust on respiratory function at Swansea. In contrast, at Glynneath during the summer but not in the winter increasing PM10 dust levels were associated with a decline in respiratory function. This occurred in children with and without asthmatic symptoms. The authors have suggested various explanations, one of which was that 'there is something different about the nature of the PM10 dust in Glynneath that makes it different and more likely to affect Peak Flow Rates'. The report concludes by stating 'the main finding of this study is that ambient PM10 dust levels at Glynneath during the summer of 1995 had a detectable adverse effect of respiratory function in local children'. Improvements in the technology of 'fingerprinting' PM dust are required before the contribution of any particular local industry can be assessed.

A further study using a Geographical Information System was undertaken in Lanarkshire.³⁸ This study showed a statistically significant association between hospital admissions with a primary respiratory disease and living close to an active opencast site. The study also indicated an association between the duration of the operations at a specific site and a rising level of respiratory disease in an isolated down-wind community. The authors recognised various limitations in the design of the study: for example it was only possible to incorporate 67 of the total of 102 opencast sites that were active during the 10-year study period; the overall effect of these limitations would be to under-report any association.

Inevitably these studies can be criticised. In contrast, the evidence that fails to show a relationship between

opencast mining and respiratory ill-health does not exist.

Ongoing research

At present the Department of Epidemiology and Public Health of the University of Newcastle is undertaking a prospective study funded by the Departments of the Environment and of Health.³⁹ The results of the study are expected in early 1999. The study is designed to test the hypothesis that exposure to dust of opencast coal mining may adversely influence respiratory symptoms and morbidity in pre-school and primary school children.

The study group comprises children from five communities centred within 750 metres of an active opencast site and a control group of children living in five matched communities. Although the protocol does not include an objective measurement of respiratory function, it is hoped that this cohort size will be sufficient to reach a conclusion regarding the nature and extent of any health effect in children. A longitudinal study would be required to assess the cumulative or chronic effects in children.

RECENT DEVELOPMENTS

The Government has now presented to Parliament as a White Paper the Review of Energy Sources for Power Generation.⁴⁰ One of the reports on which the Review was based states that, as a result of planning restraints, the opencast coal output from England is unlikely to exceed 3.0 million tonnes annually by 2003 but that Scotland 'has the capacity and currently the mineral planning climate to produce of the order of 9.0 million tonnes per year'.

Since this article was first submitted for publication, new draft guidelines have been published for England and Wales.⁴¹ The Scottish Office has also separately published, in draft form, new guidelines for opencast coal mining.⁴² These indicate that 4.0 million tonnes of the Scottish opencast coal output were railed South in 1997. The guidelines are aimed at providing much greater protection for communities with regard to both the acute and the chronic effects of opencasting. Major proposed changes include the introduction of a separation distance, or buffer zone, of 500 metres of opencast extraction and communities. This one measure should do much to mitigate the acute health effects of opencast mining. Likewise, there are a number of proposals to reduce the risk of chronic cumulative effects from respirable dust, including limiting the duration of opencasting on any one site to five years and restricting the number of sites in an area. The new guidelines also state that the planning advice notes on dust from mineral workings⁴³ will be reviewed once the result of the Newcastle study becomes available.

NEED FOR FURTHER RESEARCH

The new draft guidelines should provide communities with a considerable increase in protection from the health effects of opencast coal extraction. However, the nature of these effects remains poorly defined, particularly regarding any chronic effects.

COMEAP has identified a compelling need for more research including 'research for groups at special risk, the elderly and especially the chronic sick'.³ At present these health effects of opencast mining are not being addressed. There is a need to improve our understanding of the potential harmful affects of DEPs, especially emanating from off-road vehicles in a rural, as distinct from an urban, population. It is clear that much work requires to be done.

As a first step it may be prudent to assess the scale of the problem on those populations most at risk both with regard to the acute cardio-respiratory effects and also to determine if there is a quantifiable cumulative effect. A Geographical Information System approach might be valuable and might avoid the need to undertake a prospective study. Information could also be obtained by assessing the health status in communities that have been exposed to opencast coal extraction for a period of ten years or more, and comparing it with that of matched control communities.

As far as a cause is concerned, the possibility that DEPs and fine coal particles have an additive or synergistic effect requires to be considered, possibly using existing experimental techniques.⁴⁴⁻⁴⁷

In the meantime, Government is to be congratulated for drafting new guidelines which should help to protect communities from the acute and the cumulative effects of opencast coalmining.

ACKNOWLEDGEMENTS

We are very grateful to many colleagues who have assisted in the preparation of this paper. We are indebted to Ms Sonia Pagliari for providing the secretarial support so cheerfully.

REFERENCES

- 1 Rudd R. Coal miners' respiratory disease litigation. *Thorax* 1998; **53**:337-40.
- 2 *Opencast mining. Current opencast site operators* Bromley: Strata Publishing Limited, 1997; 16-23.
- 3 Department of Health Committee on the Medical Effects of Air Pollutants. *Non-biological particles in health* London: HMSO, 1995.
- 4 Department of Health Committee on the Medical Effects of Air Pollutants. *Quantification of the effects of air pollution on health in the United Kingdom* London: HMSO, 1998.
- 5 Vedal S, Petkau J, White R, Blair J. Acute effects of ambient inhalable particles on asthmatic and nonasthmatic children. *Am J Respir Crit Care Med* 1998; **157**:1034-43.
- 6 Roemer W, Hoek G, Brunekreef B *et al.* Effect of short-term changes in urban air pollution on the respiratory health of children with chronic respiratory symptoms: the PEACE project: Introduction. *Eur Respir Rev* 1998; **8**:52,4-11.
- 7 Timonen KL, Pekkanen J, Salonen RO *et al.* Air pollution and respiratory health of children: the PEACE panel study in Kuopio, Finland. *Eur Respir Rev* 1998; **8**:52, 27-35.
- 8 Beyer U, Franke K, Cyrus J *et al.* Air pollution and respiratory health of children: the PEACE panel study in Hettstedt and Zerbst, Eastern Germany. *Eur Respir Rev* 1998; **8**:52,61-9.
- 9 Vondra V, Branis M, Reisova M *et al.* Air pollution and respiratory health of children: the PEACE panel study in Prague, Czech Republic. *Eur Respir Rev* 1998; **8**:52,78-85.
- 10 Rudnia R, Vaskovi E, Palady A *et al.* Air Pollution and the respiratory health of children: the PEACE study in Hungary. *Eur Respir Rev* 1998; **8**:52,101-7.
- 11 Kalandidi A, Gratiou Ch, Katsouyanni K *et al.* Air pollution and respiratory health of children: the PEACE panel study in Athens, Greece *Eur Respir Rev* 1998; **8**:52,117-24.
- 12 Schwartz J. Air pollution and daily mortality: a review and meta-analysis. *Environ Res* 1994; **64**:36-52.
- 13 Wordley J, Walters S, Ayres J. Short term variations: hospital admissions and mortality and particulate air pollution. *Occup Environ Med* 1997; **54**:108-16.
- 14 Brunekreef B. Air pollution and life expectancy relation? *Occup Environ Med* 1997; **54**:781-4.
- 15 Department of the Environment. Quality of Urban Air Review Group. *Airborne particulate matter in the United Kingdom* London: HMSO, 1996.
- 16 Coggan D, Newman Taylor A. Coal mining and chronic obstructive pulmonary disease: a review of the evidence. *Thorax* 1998; **53**:398-407.
- 17 Hurler J, Burns J, Copland L *et al.* Coalworkers' simple pneumoconiosis and exposure to dust at 10 British coal mines. *Brit J Indus Med* 1982; **39**:120-7.
- 18 Department of the Environment. Expert Panel on Air Quality Standards. *Partides* London: HMSO, 1995.
- 19 Department of the Environment. *The United Kingdom national air quality strategy* London: HMSO, 1997.
- 20 Planning Appeal: Wester Torrance and Nethererton Farms. Harthill (PS/98/0034/Min) Reporter Mr D Hope (In Preparation).
- 21 Directive 97,68,EC. Relating to measures against the emission of gases and particulates. Pollutants from internal combustion engines to be installed in non-road mobile machinery. *Official Journal of the European Communities* 1998; **41**:1-85.
- 22 Department of the Environment. *The environmental effects of traffic associated with mineral workings* London: HMSO, 1998.
- 23 Seaton A, MacNee W, Donaldson K, Godden K. Particulate air pollution and acute health effects. *Lancet* 1995; **345**:176-8.
- 24 Collis E, Gilchrist J. Effects of dust upon coal trimmers. *J Industrial Hygiene* 1928; **No. 4**:101-9.
- 25 Gough J. Pneumoconiosis in coal trimmers. *J Pathol* 1940; **51**:277-85.
- 26 Fairman R, O'Brien R, Swecker S *et al.* Respiratory status of surface coal miners in the United States. *Archives of Environmental Health* 1977; 211.
- 27 Banks D, Bauer M, Castellan R, Lapp LN. Silicosis in surface coalmine drillers. *Thorax* 1983; **38**:275-8.
- 28 Amandus H, Hanke W, Kullman G, Reger R. A re-evaluation of radiological evidence from a study of U.S. strip coal miners. *Arch Environ Health* 1984; **39**(5):346-51.
- 29 Love R, Miller B, Groat S, Hagen S *et al.* Respiratory health effects of opencast coal mining: a cross sectional study of current workers. *Occup Environ Med* 1997; **54**(6):416-23.
- 30 Maclaren W, Soutar C. Progressive massive fibrosis and simple pneumoconiosis in ex-miners. *Brit J Indus Med* 1985; **42**:734-40.
- 31 Shennan D, Washington J, Thomas D, Dick J *et al.* Factors predisposing to the development of progressive massive fibrosis in coal miners. *Brit J Indus Med* 1981; **38**:321-6.
- 32 Lloyd MH, Gauld SJ, Soutar AC. Respiratory health among coal miners and telecommunication workers in South Wales. *Brit J Indus Med* 1986; **43**:177-81.
- 33 Soutar CA, Campbell SJ, Gurr DC, Lloyd MH *et al.* Cross sectional studies of respiratory diseases in British coal-miners. Institute of Occupational Medicine; TM/88/06; 1989.
- 34 Temple JMF, Sykes AM. Asthma and opencast mining. *BMJ* 1992; **305**:396-7.
- 35 Department of Health. Committee on the Medical Effects of Air Pollution. Report May 1992-December 1993. London: HMSO; 1994.
- 36 Report on the acute effects of the air pollution on the respiratory health of children in West Glamorgan. Welsh Combined Centre for Public Health (West Wales Centres); Swansea, 1997.
- 37 Temple JMF - Personal Communication.
- 38 Leng G, Douglas A. Does open-cast coal mining increase respiratory disease? *J Epidemiol Community Health* 1998; **52**:676.
- 39 Pless-Mullohi T, Howel D, Craven M, Tate J. Does living near opencast coalmining impair health? Design of an Environmental Epidemiology Study in Proceeding of an International Conference. Health Effects of Particulate Matter on Ambient Air. Prague 1997; 287-94.
- 40 White Paper - Department of Trade and Industry. Conclusions of the Review of Energy Sources for the Power Generation and Government Response to Fourth and Fifth Reports of the Trade and Industry Committee 1998.
- 41 Department of the Environment, Transport and the Regions. Draft of Mineral Planning Guidelines Note 3 (MPG3): Coal Mining and Colliery Spoil Disposal. 1998.

- ⁴² Scottish Office Development Department Draft of NPPG4. Annex A Opencast Coal and Related Minerals. 1998.
- ⁴³ The Scottish Office; The Development Department; Planning Advice Note Pan 50 Annex B The Control of Dust at Surface Mineral Workings. 1998.
- ⁴⁴ Hatch G, Boykin E, Graham J *et al.* Inhalable particles and pulmonary host defence: *in vivo* and *in vitro* of ambient air and combustion particles. *Environ Res* 1985; **36**:67-80.
- ⁴⁵ Brown G, Donaldson K. Inflammatory responses in lung of rats inhaling coalmine dust: enhanced proteolysis of fibronectin by bronchoalveolar leukocytes. *Brit J Indus Med* 1989; **46**:866-72.
- ⁴⁶ Kondo T, Takahashi S, Sato H *et al.* Cytotoxicity of size density fractionated coal fly ash in rat alveolar macrophages cultured *in vitro*. *Toxicol Vitro* 1993; **7(1)**:61-7.
- ⁴⁷ Rannels D, Lee YC. Effects of coal dust exposure and desposition of extracellular matrix by type II pulmonary epithelial cells in primary culture. *J Cellular Biochem* 1993; Suppl 17E:177.

SIGN Publications

SIGN publications available January 1999

- 4 Prevention of visual impairment in diabetes
- 5 Interface between hospital and the community: the immediate discharge document
- 6 Hospital inpatient management of acute asthma attacks
- 7 Helicobacter pylori: eradication therapy in dyspeptic disease
- 9 Management of diabetes in pregnancy
- 10 Report on good practice in the management of children and young people with diabetes
- 11 Management of diabetic renal disease
- 12 Management of diabetic foot disease
- 13 Management of patients with stroke part I: Assessment, investigation, immediate management and secondary prevention
- 14 Management of patients with stroke part II: Management of carotid stenosis and carotid endarterectomy
- 15 Management of elderly patients with fractured hip
- 16 Colorectal cancer
- 17 Investigation of asymptomatic microscopic haematuria in adults
- 18 Investigation of asymptomatic proteinuria in adults
- 19 Management of diabetic cardiovascular disease
- 20 Management of patients with stroke part III: Identification and management of dysphagia
- 21 Diagnosis and management of epilepsy in adults
- 22 Interventions in the management of behavioural and psychological aspects of dementia
- 23 Management of lung cancer
- 24 Management of patients with stroke part IV: Rehabilitation, prevention and management of complications, discharge planning
- 25 Report on a minimum data set for collection in people with diabetes
- 26 The care of patients with chronic leg ulcer
- 27 Drug therapy for peripheral vascular disease
- 28 Management of adult testicular germ cell tumours
- 29 Breast cancer in women
- 30 Psychosocial interventions in schizophrenia
- 31 Report on a recommended referral document
- 32 Coronary revascularisation in the management of stable angina pectoris

Due to be published by February 1999

- 33 Primary care management of asthma
- 34 Management of acute sore throat and indications for tonsillectomy
- 35 Diagnosis and treatment of heart failure due to left ventricular systolic dysfunction
- 36 Antithrombotic therapy

SIGN guidelines and reports are available free of charge within the NHS in Scotland. Elsewhere, a charge of £5.00 per copy for publications no. 1-24 and £7.50 per copy for publications no. 25 onwards applies. An additional charge is made for postage: 1-2 copies £1.00; 3-5 copies £3.00; 6-10 copies £5.00; 11-20 copies £6.00; 21-25 copies £7.00. For postage outside UK, please apply to the SIGN Secretariat for details.

To order publications please contact the SIGN Secretariat, Royal College of Physicians, 9 Queen Street, Edinburgh EH2 1JQ. (Tel 0131 225 7324).

Payment is required with order. Please make cheques payable to Royal College of Physicians of Edinburgh.

SIGN guidelines and reports can also be downloaded free of charge from the SIGN website: www.show.scot.nhs.uk/sign/home.htm