women at high risk—very young and older women, and those from social classes IV and V.4 21 Neural tube defects might have been prevented by periconceptional vitamin supplementation,22 increasing awareness of the importance of diet both before and during pregnancy, and the wider availability of nutritious foodstuffs. But this is not consistent with observations in Sheffield¹⁸ and Paisley²³ that the dramatic decline has occurred concurrently with massive local unemployment and a deteriorating social climate.

So the cause of neural tube defects and their decline remains tantalisingly elusive: we have a plethora of data but no explanation.

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- 1 Holmes LB, Driscoll SG, Atkins L. Etiologic heterogeneity of neural-tube defects. N Engl J Med 1976;294:365-9.
- 2 Leck I. Causation of neural tube defects: clues from epidemiology. Br Med Bull 1974;30:158-63. 3 Elwood JM, Elwood JH. Epidemiology of anencephalus and spina bifida. Oxford: Oxford University Press, 1980.
- Bradshaw J, Weale J, Weatherall J. Congenital malformations of the central nervous system. London: HMSO, 1980:13-8. (Population Trends, No 19.)
- 5 Office of Population Censuses and Surveys. Congenital malformations 1984. OPCS Monitor, MB3 85/2. London: HMSO, 1985.
- 6 Romijn JA, Treffers PE. Anencephaly in the Netherlands: A remarkable decline. Lancet
- 7 International Clearinghouse for Birth Defects monitoring systems. Annual report. San Francisco: International Clearinghouse, 1981.
- 8 Czeizel A. Spina bifida and anencephaly. Br Med J 1983;287:429.
 9 Danks DM, Halliday JL. Incidence of neural tube defects in Victoria, Australia. Lancet 1983;i:65.
- 10 Creasy MR, Alberman ED. Congenital malformations of the central nervous system in spontaneous abortions. J Med Genet 1976;13:9-16.
- 11 Bell JE, Gosden CM. Central nervous system abnormalities-pregnancy. Clin Genet 1978;13:387-96. -contrasting patterns in early and late
- 12 MacHenry JCRM, Nevin NC, Merrett JD. Comparison of central nervous system malformations in spontaneous abortions in Northern Ireland and south-east England Br Med J 1979;i: 1395-7.
- 13 Roberts CJ, Lloyd S. Area differences in spontaneous abortion rates in South Wales and their relation to neural tube defect incidence. Br Med J 1973;iv:20-2.
- 14 Byrne J, Warburton D. Neural tube defects in spontaneous abortions. Am J Med Genet, in press.
 15 Verma IC. High frequency of neural-tube defects in north India. Lancet 1978;i:879-80.
- 16 Carter CO, Evans K. Spina bifida and anencephalus in Greater London. J Med Genet 1973;10:
- 17 Ferguson-Smith MA. Spina bifida and anencephaly. Br Med J 1983;287:428-9.
 18 Lorber J, Ward AM. Spina bifida—a vanishing nightmare? Arch Dis Child 1986;60: 1086-81.
 19 Carstairs V, Cole S. Spina bifida and anencephaly in Scotland. Br Med J 1984;289:1182-4.
 20 Kirke PN, Elwood JH. Anencephaly in the United Kingdom and Republic of Ireland. Br Med J
- 1984:289:1621.
- 21 Owens JR, Harris F, McAllister E, West L. 19-year incidence of neural tube defects in area under constant surveillance. Lancet 1981;ii:1032-5
- 22 Smithells RW, Sheppard S, Schorah CJ, et al. Apparent prevention of neural tube defects by periconceptional vitamin supplementation. Arch Dis Child 1981;56:911-8.
 23 Shepherd RC. Spina bifida and anencephaly. Br Med J 1983;287:59.

Medicine and managers

Better Management, Better Health, the new report from the National Health Service Training Authority, deserves the attention of doctors not just because it offers to improve health but also because it seeks to involve all professions in management.1 Furthermore, the training authority wants responses from those working in the National Health Service.

The report is aimed at the health departments of England and Wales, the NHS, and the training authority itself and is based on the conclusions of several working groups made up of educators and managers. It begins with the premise that all elements of the service should be actively managed and that "tribal" groups should not be left to regulate their own affairs. Doctors are encouraged to initiate proposals to improve performance rather than merely respond to the ideas and directives of management.

Changes are recommended not only from the haphazard or non-existent assessment of staff but also from the inflexible \square and didactic traditional methods of management education. ≤ 0 Managers must know more about the "core technology" of $\stackrel{>}{\sim}$ the service, and doctors should learn effective management. and apply it to their own work. To this end the training authority seeks to work with interested professional bodies 2 (it does so with the BMA) and to publish general recommendations on management development for doctors.

Inevitably this will cost time, energy, and money, and the report recommends that 0.5% of payroll expenditure should be on management development. Currently, despite being the largest employer in Britain, the NHS has no quantifiable $\frac{\Box}{\Box}$ national budget for this—nor is any new money suggested. $\overline{\omega}$

This brave manifesto is presented in refreshing style with $\frac{\vec{0}}{2}$ colourful cameos of staff (looking industrious and happy) a and, of course, patients. The assumption implicit in the title—that better management will lead to better healthis appealing but not proved. Yet investment will have $\vec{\omega}$ to compete with other "good things" such as health promotion—not to mention patient services. None the less, the 3 need to use resources effectively cannot be denied—and the & principles proposed by the training authority deserve general 50 support.

The report is directed mainly at the development of full $_{\aleph}^{\omega}$ time managers but acknowledges the role of others. Its o implications for doctors will become clearer when more $\vec{\omega}$ specific proposals are presented by the training authority. These will no doubt be based on the response to the authority's recently published discussion document Developing the Role of Doctors in the Management of the NHS.2

The authority must recognise that doctors manage staff \mathfrak{S} and commit resources as well as provide services. Their management contribution ranges from clinical practice through functional management of clinical departments to working in the medical advisory structure or even being a general manager. At all levels there is a common need to practise principles of planning, organisation, direction, and control. The message from the training authority should not $\stackrel{\sim}{\Rightarrow}$ therefore be alien to doctors, but it does imply further questions about undergraduate and postgraduate training and about how doctors organise themselves locally.

Firstly, at what stage in a doctor's career should management development begin? Better Management, Better Health focused on the established practitioner, and many courses are already available—especially to senior registrars, junior consultants, and new principals in general practice. In some regions participation in an approved management course is $\stackrel{\circ}{ ext{--}}$ already a condition of consultant appointment, but should $^{\circ}_{\omega}$ the principles not be introduced to all medical students?

Secondly, much "management" is already inherent in \(\Sigma\) good clinical practice; doctors should define the purpose of investigation and treatment, choose the best options, and $\stackrel{\triangle}{\rightarrow}$ review quality and effectiveness. So it would be logical to \subseteq develop links between clinical and management education 9 and to hope for the active cooperation of the royal colleges, the professional bodies, and those responsible for postgraduate training.

Thirdly, the current management revolution begs a reappraisal of the medical advisory structure. If doctors are to have a say in management they must have their own 5 corporate, responsive, and effective local mechanisms. Now is a good time to re-examine and overhaul the cogwheel machinery (now rusting in parts) so it will meet the challenge to provide advice to health authorities and to maintain a negotiated independence.

Responsibility for effective provision of the service includes all doctors. Who is not a manager?

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- National Health Service Training Authority. Better management, better health. Bristol: National Health Service Training Authority, 1986. (Chairman: J Donne.)
 National Health Service Training Authority. Developing the role of doctors in the management of the National Health Service: a discussion document. Bristol: National Health Service Training Authority, 1986.

Bronchioloalveolar carcinoma

Bronchioloalyeolar carcinoma accounts for less than 3% of all primary lung cancers but has attracted disproportionate interest because of its intriguing clinical behaviour and unusual pathological appearance. Indeed, pathologists have been so exercised by this tumour that some have given it 36 different names while others claim that it does not exist at all.

Tumour cells of many different origins may adopt a similar histological pattern in the distal airway, and the term bronchioloalveolar carcinoma is confined to those originating in bronchioles or alveoli; it excludes metastasising adenocarcinomas from the bronchus or distant sites.² The tumour may arise from ciliated, mucinous, or clara cells in the bronchiolar epithelium or from type II pneumocytes in the alveolus. The cells all grow as a single layer along the walls of the airspaces, and the unusual environment of the alveolus with a plentiful supply of food, oxygen, water, and space probably encourages many different cells to grow in the same

The cause of bronchioalveolar carcinoma is equally uncertain and interesting. Smoking and gender appear to be unimportant, while pre-existing lung damage-either local scarring or diffuse pulmonary fibrosis—is definitely associated. The strange case report of a man who developed the carcinoma after habitually going to bed with his mouth full of olive oil led to suggestions of a link with the inhalation of oil,3 although such a history is rare. The pathological similarity between bronchioloalveolar carcinoma and the viral disease of sheep jaagsiekte suggests an infectious cause, but epidemiological evidence does not support this hypothesis.

Clinically bronchioloalveolar carcinoma has two distinct forms.45 More common is an unremarkable peripheral lung tumour that may be diagnosed by needle biopsy: metastases are unusual, and five year survival after surgery is 70%. The second form is more distinctive and presents radiographically as consolidation affecting one or more separate lobes or segments. About 10% of these patients have bronchorrhoea, and spread is assumed to be airborne, although a multifocal origin cannot be ruled out. Regional and distant metastases occur less commonly than in other lung cancers, and death may therefore be from respiratory failure as more and more of the lung becomes occupied by the tumour. Because this type of bronchioloalveolar carcinoma is widespread surgery is ineffective, and neither radiotherapy nor chemotherapy help. Nevertheless, the tumour may grow slowly, and some patients live for three years after the diagnosis. Both forms of the carcinoma (as well as the metastatic adenocarcinoma) may progress to widespread pulmonary nodules, and at this stage it is uncertain whether spread occurs by blood or airways.

Recently attempts have been made to classify bronchioloalveolar carcinoma cytologically into mucinous (secretory), non-mucinous (non-secretory), and undifferentiated groups.67 The mucinous variety tends to associate with multifocal disease, and the non-mucinous with peripheral nodules, while distant metastases are more common in the undifferentiated group; but these correlations are imprecise. Since bronchioloalyeolar carcinoma represents many tumours with similar histological appearance clinicopathological correlations will remain difficult until the cell of origin can be identified more reliably.

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- Carter D, Eggleston JC. Atlas of tumour pathology. Second series, fascicle 17. Washington DC: Armed Forces Institute of Pathology 1980:127-147.
 Schraufnagel D, Peloquin A, Pare JAP, et al. Differentiating bronchiolo-alveolar carcinoma from
- adenocarcinoma. Am Rev Respir Dis 1982;15:74-9.
- 3 Maesen FPV, Lawes JH, van den Tweel JG. Bronchiolo-alveolar carcinoma after inhalation of vegetable oil. Eur J Respir Dis 1985;67:136-40.
- vegetable oil. Eur J Respir Dis 1985;67:136-40.
 Miller WT, Husted J, Freiman D, et al. Bronchiolo-alveolar carcinoma: two entities with one pathologic diagnosis. Am J Roenigenol 1978;130:905-12.
 Manning JT, Spiurt HJ, Tschen JA. Bronchiolo-alveolar carcinoma. Cancer 1984;54:525-34.
 Clayton F. Bronchiolo-alveolar carcinoma. Cancer 1986;57:1555-64.
 Tas L, Weisbrod GL, Pearson FG, et al. Gytologic diagnosis of bronchiolo-alveolar carcinoma by fine needle aspiration biopsy. Cancer 1986;57:1565-70.

Haematology, ethnography, and thrombosis

The activities of the coagulation and fibrinolytic systems and the reactivity of platelets vary as widely as the haematological indices measured by a "full blood count." Such differences are of interest to those who wish to understand thrombosis: if those at high risk of thrombosis could be identified by examining factors that promote or control fibrin and platelet deposition steps might be taken to prevent the thrombosis.

Identifying relations between blood values and thrombosis has not proved easy. In certain individuals thrombosis is linked to a congenital deficiency of certain blood factorsfor example, antithrombin III, protein C, or plasminogen activator—but such deficiency states are rare and in most people the reason for a thrombosis is unknown. The main reason for our slowness in getting to grips with the problem may be that blood values change dramatically after a thrombosis.

The prospective approach is most likely to yield information, and several studies of patients at high risk of venous thromboembolism—for example, surgical patients—have suggested a relation between haematological values and thrombosis. For example, evidence of "hypercoagulability" (short activated partial thromboplastin time, higher factor VIII activity, higher concentrations of fibrinogen and fibrin degradation products, and prolonged euglobin lysis time) was found preoperatively in plasma from surgical patients who went on to develop venous thromboembolism.13

The information that we have for arterial thrombosis is derived from a few very large studies in which haematological