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What every doctor needs to know about 11 November

From 11 November, with only a few exceptions, patients will have the right to see computer records that doctors or others may hold on them. The Data Protection Act, which has been introduced in stages, will become fully operational on that day, and the Data Protection Registrar will have powers to enforce compliance. "Data subjects" (patients) will have the right, after making a written request and paying a fee of not more than £10, to be told by the registered "data user" (a doctor, practice, or health authority with computer records) whether any personal information about them is held on computer files; they then have the right to be supplied with a copy of that information within 40 days. Before 11 November Parliament is to be asked to approve an order that will enable doctors to prevent access being given to information they consider "likely to cause serious harm" to the physical or mental health of the patient or another person.

What are the implications for doctors? Firstly, they will need to think more carefully about what is entered into computer files. Secondly, those who recorded personal health data on disc or tape before the idea of patients having a right to see their records was contemplated should consider reviewing and editing such records before 11 November. No such action is necessary with records held solely for research as these are exempt from access rights; and information held solely for word processing falls outside the act. Thirdly, doctors identified as having overall clinical responsibility for the patient who is seeking access to records will be asked by whoever is acting as data protection coordinator to scrutinise within a week or so the applicant's computer record and manual case notes to decide whether any data in the computer record need to be withheld or made more understandable. These doctors will usually need to consult with the other people who have contributed substantially to the patient's record. On the rare occasions when modified access is considered essential the doctor must indicate exactly what part of the computer record should be withheld and why. He should also decide whether the patient should be counselled about the record when it is made available.

How much extra work this will mean is not known. There may well be an initial surge of requests stimulated by the media and the freedom of information lobby. Subsequently requests may correlate with litigation and the numbers of patients under psychiatric care. Distrust of computers may lead some patients to request access, but generally such requests should be viewed as reflecting a breakdown in the mutual trust that should exist between patient and doctor.

Time spent fostering this relationship may reduce the time spent in dealing with requests to see records.

Those who want to know more should consult the excellent booklets explaining the legislation in practical terms that are available free from the Office of the Data Protection Registrar¹⁻⁸; and specific guidance on how requests for access to personal health data should be handled has recently been issued by the Department of Health and Social Security.⁹ At least those who are to function as data protection coordinators should now be familiar with these documents.

In the future the illogicality of treating computer held medical records differently from manual records will undoubtedly lead to pleas for patients to be granted rights of access to their whole medical record. Such a policy of openness was recommended by the Steering Group on Health Services Information,¹⁰ but the practical difficulties of implementing it are formidable.

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Should bronchodilators be combined in chronic bronchitis and emphysema?

There is a new enthusiasm for combining oral theophyllines with inhaled β_2 agonists to achieve better bronchodilatation in patients with chronic obstructive airways disease. Ipratropium bromide and high dose topical steroids may even be added. We need to consider carefully the value of this moderately expensive polypharmacy.

Theophylline has been used for many years, but how it achieves bronchodilatation remains unclear. Earlier notions of phosphodiesterase inhibition appear unlikely,^{1,2} at least at therapeutic plasma concentrations of 10-20 mg/l, and so do intracellular calcium translocation³ or antagonism of either adenosine receptor receptors⁴ or prostaglandins.⁵ In patients with asthma oral theophylline may potentiate the bronchodilatation from an inhaled β_2 agonist without increasing muscle tremor.⁶ A similar useful interaction has now been shown in patients with chronic bronchitis who show some bronchodilatation from salbutamol alone.⁷ In some (but not all) such patients bronchodilatation may be increased by adding ipratropium bromide to an inhaled β_2 agonist.⁸ This

leads to the proposal that combining a slow release oral theophylline with both an inhaled β_2 agonist and ipratropium may achieve better bronchodilatation.⁹⁻¹⁰ Many patients fail to use a metered dose inhaled properly, despite repeated instruction, so that inhalation through a reservoir device—for example, Nebuhaler, Ventamatic—which ensures most efficient delivery of the inhaled drugs to the lungs,¹¹ also seems sensible. Adding a high dose topical steroid by a reservoir inhaler may yield further bronchodilatation¹² and spare the need for oral steroids—as in patients with asthma.¹³ In patients with severe airflow obstruction 15-7.5 mg/day of oral prednisolone appeared to prolong survival over 14-18 years of follow up.¹⁴ Can a similar benefit be obtained by inhaled steroids, with less risk of side effects? Clearly this polypharmacy (which has not yet been proved by controlled trial) can be justified only if bronchodilatation is shown objectively, as by a rise in peak expiratory flow rate measured four times daily over several weeks.¹²

Much attention has recently focused on respiratory muscle function in patients with chronic airflow obstruction, and it has been proposed that theophylline may improve diaphragmatic contractility and delay the onset of diaphragmatic fatigue.¹⁵ Animal¹⁶ and some human studies¹⁵ report improvements in transdiaphragmatic pressure generation at therapeutic concentrations of theophylline, but other studies do not.¹⁷ Any such effects are, however, less obvious in patients with chronic bronchitis and emphysema: thus in 15 such patients 30 days of theophylline increased maximal transdiaphragmatic pressure and reduced diaphragmatic fatigue, and the mean forced expiratory volume in one second (FEV₁) also rose from 0.6 to 0.8 l.¹⁸ In contrast, in 14 similar patients maximal inspiratory pressures were not changed by theophylline, and the six minute walking distance increased on average by only 6%, but the FEV₁ again rose from 0.8 to 0.9 l.¹⁹ The respiratory muscles are most stressed in exercise, yet most double blind studies using objective assessment of exercise tolerance show no (or only minor) improvement from theophylline in patients with chronic bronchitis and emphysema.²⁰⁻²⁴

Thus an oral theophylline combined with an inhaled β_2 agonist can yield useful bronchodilatation in patients with chronic bronchitis and emphysema, but any improvement in respiratory muscle function²⁵ in such patients is probably only a fringe benefit—and like other such benefits even this may be taxed. Oral theophylline passes into lung lining fluids and can then halve the bactericidal activity of alveolar macrophages.²⁶ As acute exacerbations of respiratory failure most often arise from infection in these patients,²⁷ using theophylline to improve respiratory muscle function in such exacerbations may be unwise.

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Acetazolamide in acute mountain sickness

During a 1969 Korean expedition to climb Lhotse in the Himalaya most of the climbers were comfortable at 4000 m. Some had mild headache and anorexia, but one man became unconscious and remained so for three weeks. He was my introduction to acute mountain sickness. This story illustrates both the range of severity of acute mountain sickness and its idiosyncratic nature.

About half of those passing through 4243 m on the Everest route experience symptoms of acute mountain sickness: headache, anorexia, nausea, vomiting, insomnia, dizziness, breathlessness, and weakness.¹ Some 4% suffer life threatening disease. As over 5000 visits are made each year to the high Nepal Himalaya² this is an important problem.

The severe forms of acute mountain sickness affect the lungs, the brain, or both. We do not know whether the mechanisms of the mild and severe forms differ in nature or merely in degree.³ Does the headache of acute mountain sickness, for example, represent an early stage of cerebral oedema? Much uncertainty surrounds these mechanisms, though the main underlying cause is hypoxia rather than hypobaria.⁴ Oedema certainly plays a part: in the lungs it has the characteristics of a permeability oedema⁵ and venous pressure is not raised; but in the brain there is no consensus on whether the oedema is vasogenic or cytotoxic.⁴ Impairment of the membrane pump has been postulated,² and vascular changes have been invoked to explain pulmonary⁶ and cerebral⁷ disease. Vascular thrombosis occurs at high

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