offered advice only on the basis of the unique features of the family history.

To put the problem into practical terms, a patient with cancer of the breast whose mother or sister also has this disease wants to know if her daughters are at risk. The lifetime risk of a woman developing breast cancer in Britain is about one in 17.⁸ The probability of one first degree relative of any woman developing breast cancer by chance is therefore quite high (about one in six, given that there are three first degree relatives). Thus it would be reasonable to reassure a post-menopausal woman with unilateral cancer of the breast with one other close relative affected at about the same age that there is little if any excess risk for other members of the family. On the other hand, if both breast cancers occur in young women, especially if there is evidence of pre-existing benign breast disease⁸ or if one of the cases is bilateral,⁹ then the risk to other family members may be considerable.

Families where there is a clear excess of other common cancers¹⁰ may be characterised by the occurrence of the same type of cancer—for example, cancer of the colon—but some families have an excess of adenocarcinomas of different tissues¹¹ or even cancers of diverse types.¹⁹ An unusual familial risk would be indicated either by several cases of the same type or by exceptionally early ages at onset or both. By bearing in mind the incidence of the common cancers the doctor will be able to reassure most families after taking a careful family history, but it is equally important for him to ensure that the rare high risk families are screened appropriately.

Carcinoma of the bronchus raises a totally different problem. Though there is some evidence of a genetic component, the environmental cause is clear and overwhelming. Even if there were evidence that smokers who develop lung cancer were genetically susceptible, the only reasonable advice to give to patients anxious about lung cancer in the family is to avoid smoking of any kind.

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¹ Newhouse ML, Thompson H. Mesothelioma of pleura and peritoneum following exposure to asbestos in the London area. *Br J Ind Med* 1965;22:261-9.

² Lips KJM, Van der Sluis Veer JAAP, Struyvenberg A, Geerdinka RA. Genetic predisposition to cancer in man. Advantages and problems of central registration and screening of families at risk. *Am J Med* 1982; 73:305-7.

³ Spector BD, Filipovitch AH, Perry GS, Kersey JH. Epidemiology of cancer in ataxia-telangiectasia. In: Bridges BA, Harnden DG, eds. *Ataxia-telangiectasia—a cellular and molecular link between cancer, neuropathology and immune deficiency*. Chichester: Wiley, 1982:103-38.

⁴ Taylor AMR, Harnden DG, Arlett CF, et al. Ataxia telangiectasia: a human mutation with abnormal radiation sensitivity. *Nature* 1975; 258:427-9.

⁵ Alderson M. The extent of the cancer problem. In: Alderson M, ed. *The prevention of cancer*. London: Edward Arnold, 1982:3-19.

⁶ Anderson DE. Familial susceptibility. In: Fraumeni JF, ed. *Persons at high risk of cancer*. New York: Academic Press, 1975:39-54.

⁷ Lynch HT, ed. *Genetics and breast cancer*. New York: Van Nostrand Reinhold, 1981.

⁸ Kalache A, Vesey M. Risk factors for breast cancer. *Clinics in Oncology* 1982;1:661-78.

⁹ Anderson DE. Genetic study of breast cancer: identification of a high risk group. *Cancer* 1974;34:1090-7.

¹⁰ Fraumeni JF Jr. Clinical patterns of familial cancer. In: Mulvihill JJ, Miller RW, Fraumeni JF Jr, eds. *Genetics of human cancer*. New York: Raven Press, 1977:223-56. (*Progress in cancer research and therapy*. Vol 3.)

¹¹ Lynch HT, Krush AJ, Thomas RJ, Lynch J. Cancer family syndrome. In: Lynch HT, ed. *Cancer genetics*. Springfield, Ill: Charles C Thomas, 1976:355-88.

¹² Li FP, Fraumeni JF. Soft-tissue sarcomas, breast cancer, and other neoplasms. A familial syndrome? *Ann Intern Med* 1969;71:747-52.

The librarian's changing role

The traditionally custodial role of the librarian is changing. The use of computers for cataloguing, information retrieval, and certain housekeeping procedures is commonplace, and the emphasis is increasingly on exploitation, not only of books and journals in the library but of information available elsewhere and in non-print form. A report by Matheson and Cooper, from the Association of American Medical Colleges, describes the library of the future in three stages: at 5-10 years, 10-20 years, and beyond 20 years.¹ Ultimately they envisage an almost paperless library, relying on machinery to gather, retrieve, and transfer information. They foresee a time when research will be carried out by teams of researchers linked by computer irrespective of where they work individually.

Pessimistic librarians have nightmares that the technological age will cause redundancy, but the optimists will be encouraged by the American report. They will dream that they are the centre of the hospital's information system—teaching students how to formulate their own knowledge data bases, acting as information brokers for researchers and clinicians in assessing the results retrieved from numerous automated sources, and helping the administrators by contributing to the institute's data base on management information.

Those who make management and financial decisions must acknowledge that the technology exists to provide advanced systems for providing the right information to the right person at the right time. There is a danger that individuals, departments, or libraries will buy equipment for a specific project without considering whether a multipurpose machine, capable of linking with locally, nationally, and internationally available data bases, would be more efficient. There are networks for the dissemination of automated information—for example, several libraries within the University of London have access to the records of each other's stocks. Health regions have similar links for the transfer of more general information, and there is software for minicomputers to access on line information retrieval data bases.

Computers can provide three particularly valuable services. Firstly, to remove dull routines—for example, automated systems which record and cancel loans, reserve material, send overdue notes, and order books. Secondly, to increase efficiency, such as using systems to transmit interlibrary loans. Thirdly, to extend the service of the "home" library by using data bases—not only Medline, Cancerline, Toxline, and so on but also statistical and management information.

Soon it will not matter that the library does not stock a certain journal: the text will be available on line and the library's or the individual's terminal will provide the relevant article immediately. It would be a pity, however, if libraries no longer provided printed journals and books, as browsing is important and many major discoveries in medicine must have been initiated or helped by serendipity.

Today's readers must find it hard to imagine a completely automated library, equipped with terminals and visual display units instead of books and journals, with workers at home or in their offices or laboratories tapping keys and reading from screens. The computer must be treated as a useful slave, not a master. Should we be aiming at a completely automated society, where information is king, where every home and office has access to the whole sum of knowledge? Can too much on line information be provided? Librarians

know that many potential library users manage very well without such help.

Librarians and information scientists can be trained to cope with the technicalities of retrieving and transferring information from the present commercially available data bases as well as from those generated by research workers, academics, students, and administrators. Conceivably, however, the automated information explosion will be so vast that a specially trained intermediary will have to evaluate the results of the search, not the person who has initiated the query.

Automation has a very important part to play in the library of the future, but it must not be thought that economies will automatically follow. The cost of terminals, the use of data bases, and the training of staff in different techniques will need capital funds as well as a continuing financial commitment. Nevertheless, the availability of more information and its use for the benefit of students, research workers, clinicians, and, ultimately, the patient will eventually justify the cost.

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¹ Matheson NW, Cooper JAD. Academic information in the Academic Health Sciences Center; roles for the library in information management. *J Med Educ* 1982;**57**, 10, part 2:1-93.

Digitalis intoxication: a new approach to an old problem

Since the use of the foxglove was first described by Withering almost 200 years ago several digitalis glycosides have been isolated and purified. Of these, two (digoxin and digitoxin) are in common use. They share several properties, including a positive inotropic effect and an action to delay atrioventricular conduction, most of which is mediated by the vagus nerves. In addition, both drugs have a narrow therapeutic index—that is, the dose that produces toxicity lies close to that which produces clinical benefit. They vary, however, in their chemical structure, a feature that leads to differences in their pharmacokinetics: digoxin is excreted mainly unchanged in the urine, whereas digitoxin undergoes metabolism in the liver, excretion in the bile, and enterohepatic recirculation. Digitalis intoxication may occur whenever the excretory pathways are compromised or an inappropriately large dose of drug is given, or both. Because the glycoside in most common use in Britain is digoxin, the feature that predisposes to intoxication is renal failure, usually consequent on old age.¹ Occasionally, however, digitalis glycosides are taken in deliberate overdose, and rarely an error in the dose given is made by either medical or nursing staff.

The response to digitalis glycosides varies with many factors, including the type and duration of the heart disease, accompanying anoxia, and the concentrations of sodium, potassium, calcium, and magnesium in the tissues and blood.² The glycosides inhibit the action of the membrane bound

enzyme sodium/potassium ATPase,³ and similar effects occur with hypokalaemia. Hence the latter is an important contributory factor in digitalis intoxication, being particularly likely to occur in patients who are receiving either a benzothiadiazine or a loop type diuretic⁴ such as frusemide or bumetanide (which also causes magnesium depletion). Hypothyroidism is another predisposing factor for digitalis intoxication, partly because glomerular filtration is depressed.⁵ In addition, the activity of the sympathetic nervous system may determine whether or not a patient develops a cardiac arrhythmia. Besides diuretics, many other drugs may interact to increase the risks of digitalis intoxication.⁶ Notable among these are other cardioactive drugs, particularly antiarrhythmic agents.⁷

Whatever its underlying aetiology, digitalis intoxication is common. Of the various estimates of its prevalence, the highest is from the United States, where almost a third of patients receiving cardiac glycosides when admitted to hospitals in Boston were definitely or possibly intoxicated.⁸ In Britain, a study from Belfast showed that about a fifth of patients receiving digitalis glycosides developed adverse effects.⁴ In contrast, of 391 patients attending general practices in Liverpool, only 37 had serum concentrations exceeding the "therapeutic range."⁹ Nevertheless, intoxication may occur at serum concentrations within the "therapeutic range," especially if the plasma potassium concentration falls below normal.

Most of the side effects of digitalis glycosides were recognised by Withering. General symptoms include anorexia, nausea, vomiting, and diarrhoea, but these are non-specific.¹⁰ The manifestations in the central nervous system include mistiness or yellowed vision, confusion, and a "psychotic" state in the elderly.¹¹ Cardiac manifestations of toxicity include worsening of heart failure, but almost any disturbance of rhythm and conduction may occur (with the exception of Mobitz type II atrioventricular block, parasystole, and junctional tachycardia).¹⁰ The most frequent abnormalities are ventricular extrasystoles (coupling) and either first or second degree atrioventricular block. Complete atrioventricular dissociation may occur, and atrial tachycardia with atrioventricular block (usually 2:1) is common.

The diagnosis of digitalis toxicity is usually simple. The development of malaise, gastrointestinal upset, or a new arrhythmia in someone who is receiving digitalis glycosides should provoke suspicion. If these features get better after the drug has been withdrawn or its dose reduced then this is further support for the diagnosis. Measurement of the concentration of the glycoside in the plasma or serum, however, together with estimation of the plasma potassium concentration may be very helpful. If the latter is normal toxicity is unlikely with plasma digoxin concentrations below 2.5 nmol/l (2 ng/ml), whereas toxicity is very likely with values above 5 nmol/l (4 ng/ml).¹² Nevertheless, in patients whose plasma potassium concentrations are below normal plasma glycoside concentrations between 1.3 and 2.5 nmol/l may be associated with toxicity. Assessment of colour vision with the Farnsworth-Munsell test may be helpful in confirming the diagnosis in some patients,¹³ but in my view pharmacological tests such as the administration of acetylthiocholine, edrophonium, or edetic acid are not only unreliable but also potentially dangerous.

The principles of treatment of digitalis toxicity are similar to those underlying the management of all drug overdoses. Firstly, the treatment should be stopped—at least temporarily. Secondly, for digitoxin, absorption from the gastrointestinal