decision maker to have an extended dialogue with a computer.

Many explanations have been put forward for the apparently meagre use made of computers in decision making: some have blamed the poor interface, reliability, and design of systems, the lack of proved benefit, and the disruption to routine that can come about through thoughtless implementation[26]; such problems may also occur with computerised record systems.[27] A recent commentator has also blamed the pride and inertia of doctors and the teaching in medical schools.[28] Proponents of an artificial intelligence perspective sometimes claim that actuarial systems are inherently unacceptable as they do not follow accepted clinical reasoning processes.[24] To a non-clinician, it appears that the astonishing variety of clinical practice must also act against any innovation that tends to impose a degree of standardisation in clinical terms and procedures.

Clearly technological advances are revolutionising attitudes to computers, while greater awareness of costs, both monetary and in patient morbidity, is bringing clinical decision making under increasing scrutiny. Interest in the components of computer assisted decision making now appears to have caught up with established analytic techniques: between 1973 and 1983 the *Index Medicus* showed that the number of articles under "computers" rose from 370 to 850, those under "decision making" rose from 64 to 129, while those under "diagnosis: computer assisted" fell from 98 to 65. As microcomputers become familiar in routine practice we may expect many more applications of automatic interpretation of information—but, as is appropriate for any new technology, the medical profession would be right to be sceptical until benefit has been proved in rigorous evaluations. When that happens computer aids may well come to be seen as basic tools in the art of clinical medicine.

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Falls in older age

One of the common questions that has to be answered by physicians responsible for old people is: did the patient trip, slip, or just fall down? Indeed, so many body systems are concerned in the maintenance of posture that experienced physicians are not surprised when the multiple disorders accompanying aging produce not one but several weak links in the control of balance, precipitating a fall.

One of the additional contributing factors is the effect of age on the control of body "sway." This phenomenon was recognised by Sheldon[1] and has since been confirmed by Overstall and colleagues.[2] Though falls are common in the old they do not occur all the time, and acute disturbances may be required to aggravate the effects of sway. For an active elderly person such an event may be associated with minimal disease, may be described as an accident, and is likely to occur in the home (one third happen on the stairs) as Sheldon showed in his pioneering work almost 25 years ago.[3]

Medicolegal responsibilities make it all the more important to determine the factors associated with falls in institutions. In a recent investigation, underlying the grave problems faced in social service (part III) homes for the elderly, 59% of 201 residents were found by Woodhouse and colleagues to have had one or more falls in a year; 22% of these had resulted in some form of injury.[4] The purpose of this survey was to determine the prevalence and rate of falling in residents of these homes and to define the characteristics of those who fell. It did not set out to determine causes. Comparison with other surveys is difficult because (as the authors point out) both the methods of ascertainment of falls and the general level of disability of the groups that have been studied are variable. Further studies on falls should be population based, as has been suggested by Perry.[5]

Until these results are available, what advice can be offered to busy general practitioners about the methods that might be used to reduce the risk of morbidity from falls in individuals and the investigations that should be carried out in those not seriously injured? The need—still widely unrecognised—for health care of the elderly at home is more important now than when it was described 20 years ago, but the amount of work required need not be excessive if simple primary preventive care is placed in the hands of an interested and informed practice nurse or health visitor.[6] Screening to discover and treat diseases of eyes, ears, and feet should be accompanied by advice on avoiding home accidents.

Secondary prevention may require the equally simple measure of kind reassurance to restore confidence after a fall; sometimes a more clinical evaluation is warranted. An unexplained fall in old age may be associated with a fracture of the femur[7] or be the presenting feature of any acute illness. If there is no obvious or serious injury the practitioner should determine whether the event was due to an accident, an incident, or an illness. Accidents may be due to preventable hazards including visual deterioration and drug effects. Incidents are common in institutions during changes in posture or position, notably while using the toilet or transferring between bed and chair, and may be the early sign of an inevitably increasing dependency from unavoidable physical and mental decline. Unexpected falls, however, require further investigation. Obvious acute illness indicates its own treatment, but in other cases

remediable precipitating factors should be sought carefully. These include postural hypotension (arguably the only indication for using the sphygmomanometer in the elderly), transient cardiac arrhythmias, and the side effects of drugs.[8]

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Early posthaemorrhagic hydrocephalus

When does hydrocephalus occur after subarachnoid haemorrhage? How often is it clinically important, and how may it be recognised? What are the criteria for, and risks and benefits of, surgical intervention?

There are many reasons, both extracranial and intracranial, why patients may fail to improve or may deteriorate after a spontaneous haemorrhage into the subarachnoid space.[1,2] Dehydration, pneumonia, pulmonary oedema, hypoxia, hyponatraemia, unsuspected hyperglycaemia, undertreated and overtreated hypertension, cardiac arrhythmias, and alcohol withdrawal with or without vitamin deficiency must be considered, for once detected they may be treated readily. Intracranial complications include rebleeding, intracerebral haemorrhage, fits, brain swelling (oedema and engorgement), cerebral infarction, delayed cerebral ischaemia associated with cerebral vasospasm, and hydrocephalus. Faced with a patient with a proved subarachnoid haemorrhage and sudden or progressive decrease in conscious level, with headache, meningism, and restlessness, with or without signs of subhyaloid haemorrhage, papilloedema, compression of the brain stem, or a focal neurological deficit, within the first two weeks, it may be difficult—on the basis of clinical assessment alone—to decide what has happened intracranially. For example, even with good baseline clinical observations, computed tomography, and examination of the cerebrospinal fluid, the diagnosis of a rebleed may not be easy, and this has prompted reassessment of the pattern of early rebleeding.[3,4]

The various intracranial complications after subarachnoid haemorrhage are interdependent. Periarterial blood and blood in the basal cisterns predispose towards cerebral vasospasm[5,6] and communicating hydrocephalus.[7] The notoriously capricious relation between arterial narrowing (shown on an angiogram) and neurological deficit may reflect the ability of the cerebral circulation to compensate for such narrowed vessels. Cerebral ischaemia may be precipitated if additional stress occurs, such as hypotension or raised intracranial pressure secondary to hydrocephalus.[8,9]

Isotope cisternography shows that the circulation of cerebrospinal fluid is disturbed in most patients for the first few days after a subarachnoid haemorrhage.[10] Severe disturbances, defined by reflux of isotope into the ventricles and absence of activity over the hemispheres, occur in about 20% of patients. The incidence of demonstrable ventricular enlargement depends on the criteria used, the imaging technique, and the timing of the investigation: with computed tomography, the incidence is about 10% in the first week and 19% in the second week.[11]

After a subarachnoid haemorrhage about 7% of patients require treatment for progressive, symptomatic hydrocephalus.[7,11] In the long term the subarachnoid obstruction to the flow of cerebrospinal fluid is secondary to the leptomeningeal reaction to extravasated blood, and it is not clear whether antifibrinolytic treatment aggravates this problem or not. Acutely, hydrocephalus may be precipitated by blood obstructing the arachnoid villi, blood in the cisterns, or clot in the ventricles.[12]

Ventricular drainage for possible acute hydrocephalus in the acute stage after subarachnoid haemorrhage may be a lifesaving procedure,[18] but the degree of hydrocephalus may not be particularly dramatic, and the clinical picture is often unclear. Moderate ventricular enlargement is a poor prognostic sign, and Vassilouthis and Richardson make a convincing argument for the accurate measurement of ventricular size relative to skull size.[11] Cerebral blood flow and clinical grade tend to fall with increasing ventricular size.[19] Early shunting is contraindicated: firstly, because the high protein content of the cerebrospinal fluid might block the shunt, and, secondly, because only a minority of these patients will require a long term shunt. Intraventricular haemorrhage occurs in 35% of these patients and is often associated with more destruction to the brain—reflected in an overall mortality of 65%.[20] Thus uncritical use of ventricular drainage and even ventricular lavage to remove clots is deservedly unpopular because it helps relatively few patients. In the neonate, when the aetiology of intraventricular haemorrhage is hypoxia/ischaemia, ventricular drainage does not appear to help if there has been appreciable parenchymal haemorrhage.[21] Nor is it proved that obsessional attention to the management of early hydrocephalus improves the long term outcome for neonates with intraventricular haemorrhage alone. (It is possible that the risk of infection masks any benefits from ventricular drainage.)

Apart from the small risk of infection—ventriculitis or meningitis (6%)—one or more fits (16%), and intracerebral haematoma (less than 1%), there is a theoretical risk that overenthusiastic reduction of intracranial pressure may precipitate rebleeding of the aneurysm.[22] Aneurysms normally stop bleeding when intracranial pressure rises above arterial diastolic blood pressure. At this point there is sufficient reduction in the transmural pressure gradient during the cardiac cycle for platelet and fibrin deposition to occur and the intracranial pressure slowly subsides. In a minority of patients and experimentally after heparinisation, intracranial pressure continues to rise until it equals arterial blood pressure and death ensues.[22,23] In two separate series from Scandinavia drainage of cerebrospinal fluid down to pressures of 15 or 25 mm Hg did not greatly increase the risk of rebleeding.[24,25] If a rebleed occurred while a ventricular drain was open the intracranial haemorrhage did not appear to stop so readily, which resulted in a higher mortality. Thus drainage should be stopped immediately if a rebleed is suspected.

Because of these potential complications ventricular drainage is restricted to a minority of patients—those who are either failing to improve or actually deteriorating and