occurring in the over 65s are unreported to medical services.[3] This is important because the estimated annual rates of total home accidents for this group (840/1000 (645/1000 unreported)) and falls (564/1000) are also high. Moreover, the incidence of home accidents rises markedly with age—from 6% at 65-74 years to 11% for the over 85s (p 30).[3]

It is common sense to reduce the risk of accidents and illness by prevention whenever possible. Screening by general practitioners of those aged over 75 years and opportunistic screening of younger elderly patients for vision, hearing, mobility, mental state, inappropriate drug treatment, and social difficulties are useful starting points. To assist this the elements associated with accidents and ill health in elderly people need to be more widely understood. Top of the list here for all ages must be adverse environmental factors including poor lighting, inadequate handrails on stairs, and loose objects on the floor, including mats.[4]

In elderly people personal factors are also important. Reduced body reserve (due to impaired elasticity in tissues and disuse atrophy of striped muscle—to name but two causes) affects the body's ability to compensate quickly enough to avoid impending accidents. Illnesses may present atypically in older patients—doctors have come to expect that patients of all ages will present with symptoms as described in medical texts first written when most patients were younger than they are today.[3] There are interactive problems of multiple pathology[5]; iatrogenic ill health caused by polypharmacy[6] (even in those aged over 50 years[7]); and socially adverse factors, which may compound the problems of daily living for elderly people. These include the loss of esteem because of problems arising from agism, grief, lack of social mobility, inadequate house design for indoor safety, the threat of living in a potentially violent community, and financial and other unrecognised anxieties.

Finally, who has the responsibility for reducing home accidents in elderly people? In a nutshell, we all do. Elderly people are not merely housebound opportunities for bigger armchairs or remote controlled television sets. People's abilities to respond effectively to the challenges of their environment depend on their physical and mental fitness. During the years of retirement the cumulative effect of physical, mental, and social disuse may result in increased frailty that may eventually predispose to accidents in the home. As one eminent but now also elderly person recently said to me, "When you have retired they no longer ask or expect you to do anything."

There is no doubt that general practitioners are in the key position[3] and can alert society to the value of elderly people. A meaningful older age can enhance the interest and determination of elderly people to find personal opportunities for their own wellbeing within a more active lifestyle. This necessarily includes an awareness of the environment, its hazards as well as its comforts. Positive attitudes from general practitioners can be quickly mirrored by paraclinical and other carers. Repeated home accidents, and certainly repeated falls in elderly people, may then be recognised for what they represent—signs of ill health requiring specific attention. At present the more usually accepted alternative is to increase the elderly person's dependency by merely seeking the provision of often inappropriate "social support services" rather than using the opportunity for a proper check on health and lifestyle.

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1 Department of Trade and Industry. *Home and leisure accidents report*. London: Department of Trade and Industry, 1988.
2 Livesley B. "Funny turns", episodic loss of consciousness, and falls. In Tallis R, ed. *The clinical neurology of old age*. Chichester: Wiley, 1989:433-42.
3 Graham HJ, Firth J. Home accidents in older people: role of primary health care team. *BMJ* 1992;305:30-2.
4 Gloag D. Strategies for accident prevention: a review of the present position. In: *Strategies for accident prevention: a colloquium*. London: HMSO, 1988:74-7.
5 Livesley B. *Caring for the elderly in the 1990s: the opportunities and challenges resulting from the fallibility of existing theories. Inaugural Lecture*. London: Research for Ageing Trust, 1989.
6 Cartwright A, Smith C. *Elderly people, their medicines, and their doctors*. London: Routledge, 1988:30-6.
7 Grymonpre RE, Mitenko PA, Sitar DS, Aoki FY, Montgomery PR. Drug-associated hospital admissions in older medical patients. *J Am Geriat Soc* 1988;36:1092-8.

Adenosine and cardiac arrhythmias

The preferred treatment for supraventricular tachycardia

Adenosine is an endogenous purine nucleoside that is capable of causing atrioventricular nodal conduction block in humans.[1] It is approved by the United States Food and Drug Administration for terminating paroxysmal supraventricular tachycardia, and it has recently gained a product licence in the United Kingdom for use as both a therapeutic agent for supraventricular tachycardia and a diagnostic agent in broad or narrow complex regular tachycardia of uncertain cause.[1,2] If adenosine does terminate a tachycardia the strong presumption is that the tachycardia was due to an arrhythmia involving the atrioventricular node,[3] whereas if the tachycardia continues unaffected it is highly likely to be of ventricular origin. Atrial arrhythmias (such as atrial flutter) will not be terminated, but the diagnosis should become apparent because of transient atrioventricular nodal block. The half life of adenosine in human plasma is as short as 0·6-1·5 seconds[4] so that any unwanted effects are transient.

The diagnostic information provided by atrioventricular nodal blockade in a patient with a broad complex tachycardia is independent of that provided by the patient's history and the electrocardiographic appearances—and, indeed, in the two studies in which the information was given the diagnostic value of adenosine compared favourably with the use of standard electrocardiographic criteria.[2,5] Adenosine is easy to administer, and in cases of diagnostic difficulty the interpretation of the response (termination or non-termination) is likely to be simpler for junior doctors than the use of complicated electrocardiographic rules.

The atrioventricular nodal blocking actions of adenosine have been known since 1929, so it may seem strange that the agent has taken so long to be used widely in Britain.[6] Part of the reason for the delay may have been the initial unsuccessful attempts to use it to terminate atrial fibrillation.[7] A related compound, adenosine triphosphate, has been widely used in France for many years, but its evaluation has been poorly documented even in the French literature. Another possible reason for the delay may have been pharmaceutical companies' lack of enthusiasm for development—for adenosine itself cannot be patented because it is a naturally occurring compound. Furthermore, there is no stable oral form of

adenosine, so that its use is restricted to the short term. Despite this a preparation of adenosine is now available commercially, and several hospitals are producing their own preparation.[8]

Adenosine is best given as a fast bolus into a large peripheral vein and followed by a saline flush. Its very short action allows several doses to be given one after the other without risk of a cumulative effect. A low dose should be given initially and then increased if atrioventricular nodal blockade is not achieved. This is preferable to giving a single large dose because of the transient side effects of flushing and dyspnoea. The datasheet in the United Kingdom recommends an initial dose of 3 mg for adults, and occasionally arrhythmias will be terminated by this low dose. Further doses of 6 mg and then 12 mg may be given if necessary at one to two minute intervals. The upper limit for any single dose of adenosine is 12 mg—based on the finding that just over 90% of paroxysmal supraventricular tachycardias are terminated by that dose.[9] Adenosine has, however, been used safely at higher doses, and a further increment to 18 mg may increase the likelihood of termination of a supraventricular arrhythmia, although this high dose is usually associated with an increase in the severity of side effects. The dose for children is calculated on a basis of 0·05 mg/kg, increasing by 0·05 mg/kg increments to a maximum bolus dose of 0·25 mg/kg.[10] The most important drug interactions are with aminophylline, which antagonises the effects of adenosine, and with dipyridamole, which accentuates and prolongs its effects. Adenosine should be used cautiously in patients with asthma, in whom bronchospasm may be exacerbated.[11]

Adenosine is the drug of first choice for terminating paroxysmal supraventricular tachycardia in the presence of left ventricular dysfunction, concomitant β blockade or severe hypotension because of its very short half life and proved safety in these circumstances. Intravenous verapamil may be preferred in patients with asthma. In terms of the treatment of uncomplicated paroxysmal supraventricular tachycardia there seems little to choose between adenosine and verapamil. The claim that termination with adenosine is faster than with verapamil (promotional literature for Adenocor, Sanofi Winthrop, 1992) has little relevance to clinical practice. The difference between 20 seconds to termination with adenosine and 60 seconds with verapamil (after injection over 15 seconds) is not important for a patient with a stable arrhythmia—and the total time to termination is likely to be longer for adenosine if several injections are required. Nevertheless the fact that adenosine may be used without precipitating haemodynamic deterioration in patients with broad complex tachycardia[2 5 13] means that both accident and emergency departments and cardiac care units would be safer places for patients with arrhythmias if the first instinct of the doctor on the spot was to reach for adenosine rather than verapamil.

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1 Faulds D, Chrisp P, Buckley MM-T. Adenosine: an evaluation of its use in cardiac diagnostic procedures, and in the treatment of paroxysmal supraventricular tachycardia. *Drugs* 1991;41:596-624.
2 Griffith MJ, Linker NJ, Ward DE, Camm AJ. Adenosine in the diagnosis of broad complex tachycardia. *Lancet* 1988;i:672-5.
3 DiMarco JP, Sellers TD, Lerman BB. Greenberg ML, Belardinelli L. Diagnostic and therapeutic use of adenosine in patients with supraventricular tachyarrhythmias. *J Am Coll Cardiol* 1985;6:417-25.
4 Moser GH, Schrader J, Deussen A. Turnover of adenosine in plasma of human and dog blood. *Am J Physiol* 1989;256:C799-806.
5 Rankin AC, Oldroyd KG, Chong E, Rae AP, Cobbe SM. Value and limitations of adenosine in the diagnosis and treatment of narrow and broad complex tachycardias. *Br Heart J* 1989;62:195-203.
6 Drury AN, Szent-Györgyi A. The physiological activity of adenine compounds with especial reference to their action upon the mammalian heart. *J Physiol* 1929;68:213-37.
7 Honey RM, Ritchie WT, Thomson WAR. The action of adenosine upon the human heart. *Q J Med* 1930;23:485-9.
8 Clarke C, Cairns CJ, Coutrouzas H, Terry D. The preparation and use of adenosine injection. *Pharmaceutical Journal* 1990;244:595-7.
9 DiMarco JP, Miles W, Akhtar M, Milstein S, Sharma AD, Platia E, *et al*. Adenosine for paroxysmal supraventricular tachycardia: dose ranging and comparison with verapamil. *Ann Intern Med* 1990;113:104-10.
10 Till J, Shinebourne EA, Rigby ML, Clarke B, Ward DE, Rowland E. Efficacy and safety of adenosine in the treatment of supraventricular tachycardia in infants and children. *Br Heart J* 1989;62:204-11.
11 Cushley MJ, Tattersfield AE, Holgate ST. Inhaled adenosine and guanosine on airway resistance in normal and asthmatic subject. *Br J Clin Pharmacol* 1983;15:161-5.
12 Adenocor®. Promotional literature. Sanofi Winthrop Ltd, 1992.
13 Garratt CJ, Griffith MJ, O'Nunain S, Ward DE, Camm AJ. The effects of intravenous adenosine on antegrade refractoriness of accessory AV connections. *Circulation* 1991;84:1962-8.

Juvenile myoclonic epilepsy

Underdiagnosed and treatment may have to be life long

Juvenile myoclonic epilepsy, formally recognised as a syndrome by the International League Against Epilepsy in 1985, accounts for about one in 10 cases of epilepsy.[1] Clinical awareness of the syndrome among non-neurologists, however, is low, resulting in delays of several years between the onset of symptoms and correct diagnosis.[1 2] Additionally, the best treatment, prognosis, and possible need to continue treatment long term are not widely appreciated.[2]

Typically the age at onset of juvenile myoclonic epilepsy ranges from 8 to 18 years. Patients experience single or repeated bilateral irregular jerks without loss of consciousness. The jerks occur more commonly on waking or in the mornings and are often but not always associated with generalised tonic-clonic seizures or absence seizures. Results of neurological examination are usually normal, but in 60-90% of cases the electroencephalogram shows the typical 4-6 Hz bilaterally symmetrical polyspike and wave pattern with normal background. Flickering light results in an epileptiform discharge in the electroencephalogram in up to 40% of patients.[3]

A typical history and electroencephalogram make the diagnosis straightforward, but some patients who experience only myoclonic seizures do not present to medical attention until they experience a generalised tonic-clonic seizure, having previously interpreted the myoclonic episodes as morning jitters or clumsiness.[1 4 5] Misinterpreted symptoms[2 4] or the mistaken reassurance provided by a normal electroencephalogram may lead to a delayed or incorrect diagnosis. Specifically seeking a history of myoclonic episodes significantly improves diagnostic accuracy.

Juvenile myoclonic epilepsy has a familial component. Two studies have suggested that a genetic component is located on