Dementia will develop in 10% of people over the age of sixty five years. This cruel illness exacts a high psychological price and creates a severe burden for society. An increasing number of drugs are used in its management, some of them among the most widely prescribed in the world.

The drugs used can be classified into

i. those affecting neurotransmitter systems;
ii. cerebral metabolism enhancers;
iii. vasodilators; and
iv. antiembolic, antithrombotic and antihypertensive drugs

There is much scientific evidence for changes in a number of neurotransmitter systems in dementia. Drugs affecting the cholinergic system have been the most widely studied. Acetylcholine precursors are not effective. Acetyl-1-carnitine hypothesised to have cholinomimetic properties has recently been demonstrated to show some improvement and prevention of deterioration. Tetrahydroaminoacridine, an acetylcholinesterase inhibitor, showed initial promise but later trials gave negative results. Manipulation of the serotonergic, dopaminergic, noradrenergic and gabaergic systems have not proven fruitful as yet. Peptides are not useful but animal studies with nerve growth factor are promising.

Codergocrine mesylate (Hydergine) improves neuronal metabolism and is the only drug for dementia approved by the FDA in the United
States of America. Although shown to provide benefit particularly in mood and behaviour a recent well designed trial demonstrated no improvement and possible deleterious effect. Oxiracetam also enhances neuronal metabolism and a number of recent trials have reported significant improvement. Further research is necessary with both drugs.

Vasodilators had been widely used previously but results have been equivocal overall. There are risks of postural hypotension with concomitant decrease in cerebral blood flow and further deterioration.

Multi infarct dementia constitutes about 15-20% of dementias and can often be clinically differentiated from the rest. A major contributing factor is hypertension. Maintenance of systolic blood pressure in the upper normal range has been shown to improve cognition while a decrease of systolic blood pressure to low normal may be deleterious. Angiotensin converting enzyme inhibitors may be the drug of choice because they improve cerebral blood flow autoregulation. Low dose aspirin (375 mg daily) in multi infarct dementia has also been shown to delay cognitive decline.

A large number of other drugs have been tried with equivocal or negative results.

In conclusion, although many trials are promising there is as yet no consistently effective drug for the prevention or relief of dementia.