CHAPTER 4

Old Age Psychiatry

Age changes

Brain weight decreases by 20% by the age of 90 years, there is selective neuronal loss of between 5% and 50% and the cells tend to shrink. There is also a 15–20% reduction in synapses in the frontal lobes. Lipofuscin accumulates in some cells, but its significance is uncertain. Plaques and tangles are found in aged brains but seldom in middle-aged ones. Granulovacuolar degeneration can often be found in the hippocampus and occasional vascular amyloid deposits are seen in cortical blood vessels. All these changes are more pronounced in Alzheimer's disease (AD).

Performance in intelligence testing, learning ability, short-term memory and reaction time tend to decline with age but often not significantly until about the age of 75 years.

Sleep

There is a positive correlation between increasing age and complaints of poor sleep. Studies indicate that sleep becomes shorter, lighter and more broken, with greater difficulty getting back to sleep again. Stages 3 and 4 of sleep are rarely attained and periods of rapid-eye-movement sleep are also infrequent. Apnoeic episodes are commoner. The worst sleep patterns are found in demented patients, who often also become much more confused in the evening or night ('sundowning'). If simple corrective measures do not help, a short course of a hypnotic may be justified.

Factors that disturb sleep patterns

- Anxiety
- Depression
- Pain
- Discomfort due to constipation
- Urgency, frequency, nocturia
- Restless legs
- Cramps
- Nocturnal cough or breathlessness
- Daytime napping
- Unrealistic expectations
- Drugs (theophylline, sympathomimetics)
- Drug withdrawal (sedatives, hypnotics)

Simple advice for poor sleepers

- Rise at a regular and early hour
- Maintain activity during the day
- Avoid coffee or tea during the evening
- Do not go to bed hungry
- Wind down before trying to get to sleep
- Take a warm milky drink in the evening
- Do not go to bed too early

Problem drinking

• Repeated ingestion leading to dependency, physical disease or other harm.

• Consumption peaks at age 55 and declines thereafter. One survey has shown that 17% of

over 60s have a problem and 12% are heavy drinkers. The usual problem is daily dosing rather than bingeing—often concealed. Older people may have particular problems with alcohol if their balance or cognition is already impaired, with obesity or malnutrition and alcohol predisposes to hypothermia.

- Treatment entails total withdrawal: delirium tremens is controlled with chlordiazepoxide.

The Institute of Alcohol Studies produces a useful fact sheet.

'Graduate drinkers'

M = F
- Falls, confusion, gastrointestinal effects, self-neglect, anxiety, depression, hallucinations, Wernicke's encephalopathy, dementia, liver and heart complications

'Late-onset drinkers'

F > M
- Attempt to assuage loneliness and sadness
- Depression common
- Complications similar to group 1

Anxiety

Anxiety is very common in older people and may accompany depression, dementia and physical illness or may cause physical symptoms (palpitations, breathlessness, giddiness, abdominal discomfort, bowel fixation). Always consider anxiety or depression in recurrent attenders in a GP or hospital setting. Treatment is by reassurance or cognitive therapy, but if severe and amounting to panic attacks, SSRIs are the drugs of choice.

Paraphrenia (persistent delusional disorder)

This is a late-life schizophreniform paranoid psychosis in which personality and affect are well preserved and there is no thought disorder. It most often affects unmarried women who live alone, especially those who suffer from deafness. There is often a highly structured system of delusions and hallucinations, which may have a sexual content or which may relate to electrical appliances, for example. The response to antipsychotic drugs is good if concordance can be achieved. Newer agents, such as low-dose risperidone, cause fewer long-term side-effects but increase the drug bill.

Causes of hallucinations

- Paraphrenia
- Poor vision
- Bereavement
- Depression
- Acute brain syndrome (including drugs, e.g. dopaminergic treatment for Parkinson's disease)
- Dementia

Depression

Prevalence

Depression occurs in around 10–15% of people aged over 65 years and is severe in 3%. The most important thing is to consider the possibility. If you are not sure, ask the patient—most will tell you and there is a surprisingly good correlation between a yes/no answer to that question and a full psychiatric assessment. For an intermediate approach, screening tools such as the Geriatric Depression Scale (15-point version) or the BASDEC (Brief Assessment Schedule Depression *Card*s) may be helpful. The latter, which was developed on a medicine for the elderly ward, has the questions in large print on cards so you don't have to yell 'are you so depressed you have thought of killing yourself?' to a deaf patient on an open ward. Many old, ill people in hospital are anxious, lose their appetite, can't sleep or concentrate and so in the list of features below, physical aspects are least helpful and anhedonia, perhaps the most.

Features

• Association with physical illness. Most chronic illness is associated with depression. Growing evidence suggests that there may be a subtype of depression in later life, characterized by a distinct clinical presentation and an association with cerebrovascular disease.
• Somatization of symptoms, hypochondriasis.
• Pervasive anhedonia ('when did you last enjoy anything?').
• Guilt, worthlessness, low self-esteem.
• Hopelessness and helplessness.
• Apathy or agitation, anxiety, delusions.
• Sleep disturbance.
• Withdrawal, poor concentration and memory ('pseudodementia').
• Self-neglect, malnutrition, dehydration.
• Suicide risk.

In almost all industrialized countries, men aged 75 years and older have the highest suicide rate among all age groups. Whereas in younger age groups suicide attempts are often impulsive acts, suicide attempts in older people are often long planned and involve high-lethality methods. These characteristics, in addition to the fact that elderly are more fragile and frequently live alone, more often lead to fatal outcome. In later life, in both sexes, major depression is the most common diagnosis in those who attempt or complete suicide. A previous serious attempt, bereavement and isolation all point to high risk.

Treatment

Supportive

This involves counselling, relieving loneliness and practical measures, e.g. benefits check. Depression is often best managed with help of the local old age psychiatry service. In most areas this is a multi-professional group, with Community Psychiatric Nurses (CPNs), social workers and a consultant. The team will carry out further assessment if necessary—usually in the patient's own home and will support them to continue with medication, etc. The old age psychiatry service may run a day hospital—many have different days for clients with depression or psychosis and dementia. Other options might include referral to Cruse Bereavement Care or arranging a day centre.

Sometimes the focus is on a patient but the health needs of their carer are overlooked. Depression is extremely common amongst carers and it is essential to recognize this and offer support, such as arranging respite care or a sitting service such as Crossroads, before deterioration in the carer's mental health precipitates a crisis.

Drugs

SSRIs are the drugs of choice, having fewer sedating and anticholinergic effects than the tricyclic antidepressants and being relatively safe in overdose. Nausea, diarrhoea and restlessness can occur. To minimize nausea, start at a very low dose for the 1st week and gradually increase. Give a simple explanation of the chemical basis of depression and explain that depression can't just be shaken off by 'counting your blessings', or having a bit more moral fibre! Patients may have had bad experiences with benzodiazepines in the past and so stress that these drugs are different, that they do not usually make them feel dopey but will need to be stopped gradually when no longer needed. Explain to the patient that any nausea will wear off and strongly reinforce the need to stick with the tablets for at least 6 weeks before expecting the cloud to lift. Information sheets can be useful. Treatment should be continued for a year or possibly even for life in severe cases.

There is no clear evidence that one SSRI is more efficacious or better tolerated by elderly patients than another. Other features may influence the choice of agent. For example, fluoxetine, fluvoxamine and paroxetine are more likely to be involved in significant drug–drug interactions than citalopram or sertraline. Everyone has their own favourites but our current practice for most patients is citalopram starting with 10 mg. In special situation, the following are used: mirtazapine (a pre-synaptic α_2-antagonist which increases noradrenergic and serotinergic transmission) where appetite stimulation is needed, trazadone (tricyclic with few anti-muscarinic effects) if sedation is needed and

venlafaxine (a serotonin and noradrenaline reuptake inhibitor) for resistant depression. If nausea is a major problem on SSRIs, lofepramine (a tricyclic with few antimuscarinic side effects), building from 70 mg may be helpful.

Electroconvulsive therapy

Electroconvulsive therapy (ECT) is comparatively sure, quick and safe in severe cases but most psychiatrists are now very reluctant to consider ECT because of the bad press it has received. This is a great pity as patients who were previously 'brought back' to a useful life now sometimes linger and die on their medication.

Dementia

Dementia, of which AD is the commonest cause, is a public health problem of enormous magnitude.

What is dementia?

Dementia is a **syndrome** (*lots of causes*) of **acquired** (*not learning difficulties*), **chronic** (*lasts months to years*), **global** (*not **just** memory or **just** language problems*) impairment of higher brain function, in an **alert patient** (*not drowsy*), which **interferes with the ability to cope** with daily living (it doesn't usually matter if an old person doesn't know 'it's Tuesday' but if he or she doesn't know 'it's winter' he or she might freeze).

Remember:

My (memory)
Old (orientation)
Grandmother (grasp)
Converses (communication)
Pretty (personality change)
Badly (behaviour disorder)

(from Brice Pitt, Emeritus Professor of Old Age Psychiatry at St Mary's, London).

Dementia contrasts with **delirium**, an acute confusional state with impaired consciousness. A person can become delirious at any age, but frail older people often become confused when they are ill. An acute confusional state resolves as the underlying illness (e.g. chest or urinary infection) gets better. However, delirium is particularly common on a background of dementia, in which case the confusion will improve but only to a limited extent.

Clinical features of acute confusion

- Onset typically abrupt
- Marked fluctuation: lucid intervals
- Altered consciousness
- Inability to sustain, focus or shift attention
- Disturbed cognition
- Delusions and hallucinations
- Fear, bewilderment, restlessness or hypoactivity
- Possibly signs of underlying cause

Causes of acute confusion

Intracranial

- Infarction—'silent'; often frontal
- Infection—meningoencephalitis
- Injury—head injury, fat embolism
- Iatrogenic—drugs acting on CNS (including abrupt withdrawal, e.g. tranquillizers)

Extracranial

- Infection—especially chest and urine
- Metabolic—fluid and electrolyte imbalance, hypoglycaemia, hypothermia
- Anoxia—cardiac or respiratory failure, 'silent' myocardial infarction, carbon monoxide poisoning
- Toxic—alcohol, drugs
- Nutritional—Wernicke's encephalopathy

Treatment is summarized as follows:

1 Plentiful reassurance and explanation: avoid confrontation.

2 Treat underlying cause, correct fluid and electrolyte imbalance, correct nutritional deficiencies.

3 Environmental—use a dim light overnight.

4 Restlessness, agitation: haloperidol (cheap) risperidone (better side-effect profile but more

expensive) or if neuroleptics are to be avoided try chlormethiazole or lorazepam.

Causes of the dementia syndrome

The **primary dementias**, where the disease mainly affects the neurons in the brain, include AD, Lewy body disease, other frontotemporal lobar atrophies including Pick's disease and frontotemporal dementia and Creutzfeldt—Jakob disease.

The commonest **secondary dementia,** in which the neuronal damage is secondary to pathology in other tissues, is vascular dementia (which includes multiple small infarcts and white matter ischaemia). CADASIL (see below) is a familial microangiopathy that usually presents in middle age with recurrent TIAs or stroke. The mean age of onset for TIAs and/or stroke is 45 years, but the range extends from the early 20s to the 60s. Other important causes are drugs and alcohol, endocrine and metabolic problems such as thyroid dysfunction, recurrent or severe hypoglycaemia, post-hypoxia, nutritional problems such as vitamin B_{12} deficiency, brain tumours, trauma and infections including syphilis and HIV.

Remember:
Drugs and alcohol
Eyes and ears
Metabolic
Emotional (really, psychiatric problems)
Nutritional
Trauma and tumours
Infections
Atheroma—vascular dementia.

How common is dementia?

Dementia is rare below the age of 55 years but the prevalence of dementia increases dramatically with age to about **3% in the over 65s** and rising to about **20% in the over 80s**. There is a slight female preponderance. In elderly people, AD probably accounts for half to two-thirds of cases of dementia. About 700 000 people in England and Wales have dementia.

What happens in dementia?

The onset of dementia is insidious with gradual changes in memory and concentration, thinking processes, language use, personality, behaviour and orientation. Short-term memory is impaired early—long-term recall is often much better. Thinking becomes rigid and concrete. The condition progresses to obvious problems with short-term memory and managing basic activities of daily living, increasing disorientation and sometimes difficult or distressing behaviour such as night-time wandering, aggression or apathy. A tendency to lose things easily turns into paranoia and even delusions. Constant repetition of the same questions can be very trying for carers. Eventually, the patient is completely disorientated, no longer recognizes close family members, ceases to communicate and becomes doubly incontinent, bed-bound and totally dependent. Sadly survival is often 8–10 years.

Why does dementia matter?

Dementia is a devastating condition for the **patient,** while insight is preserved, and their **family** who witness the progressive deterioration. For the spouse this has been likened to 'being bereaved without being widowed'. Dementia also has major **economic** consequences. Demographic changes are resulting in marked increases in the oldest old, one in five of whom may have dementia, a major cause of dependency and institutional care. Politicians and **society** are beginning to grapple with the issues and the cost of providing health and social care for patients with AD. In England, the direct costs of AD have been estimated at between £7.06 billion and £14.93 billion (2001), greater than the costs of stroke, heart disease and cancer. In addition to the considerable **morbidity**, it is believed that AD is the fourth leading cause of **death** in the West. However, 'bronchopneumonia' usually appears on the death certificate. Despite this burden, dementia is only just beginning to command the attention it deserves.

How is a diagnosis of dementia made?

The **GP** is usually the first port of call, but a survey performed by the Alzheimer's Disease Society suggests that there is often difficulty

in obtaining a diagnosis. Many old people are slightly forgetful and it can be difficult to distinguish ageing changes from early dementia. The term **age-associated memory impairment** is applied to a subjective complaint of forgetfulness in those over 50 years of age, with a performance on memory testing one standard deviation below the normal for a young adult. Almost 20% of people over 50 years of age meet these criteria and the significance is uncertain.

GPs may be reluctant to diagnose an 'untreatable' condition. If the patient lives alone there may be no one to give a history and unless a simple test of cognition is performed, it is easy to be misled by 'a good social front'. Suitable quick screening tests include Hodkinson's Abbreviated Mental Test Score. However, if there are family members and they are concerned there is usually a problem, whereas if only the patient is complaining the diagnosis is often anxiety, depression or 'worried well'. Although dementia may have been developing for months, the patient often presents acutely because of a social crisis (e.g. death of caring spouse) or physical crisis (any illness, often a chest or urine infection, which worsens the confusion).

Having identified possible dementia, the GP may manage the patient or **refer** to a geriatrician, an old age psychiatrist, a neurologist, or in some areas, a specialist memory clinic.

What are the aims of a clinical assessment?

Is it dementia?

A full history, with more detailed cognitive function testing, including assessment of language, visuospatial skills and reasoning (e.g. Mini-Mental State Examination (see Appendix 4), Alzheimer's Disease Assessment Scale) usually answers this question. At this stage, other conditions must be ruled out.

The **differential diagnosis** includes an acute confusional state, depression, communication difficulties due to deafness, poor vision, or language deficits, PD, schizophrenia and mania.

What type of dementia is it?

The next step is to identify the cause. The dementia may be reversible (e.g. hypothyroidism), treatment may slow disease progression (e.g. treating hypertension in vascular dementia), specific treatment may be available (e.g. AD), genetic counselling may be required (e.g. familial AD) or it may be important to avoid certain medication (e.g. neuroleptics in Lewy body disease).

There is no diagnostic test for most of the primary dementias until a post-mortem examination, so the *likely* cause is determined by the **clinical features** and the results of **investigations.** Common conditions such as vascular dementia and AD may co-exist.

Progressive deterioration is common in AD whereas step-wise deterioration is characteristic of vascular dementia. Neuropsychiatric phenomena such as delusions and hallucinations and extreme sensitivity to major tranquillizers are a feature of Lewy body disease. Parkinsonian features on examination would suggest vascular dementia or Lewy body disease. Most patients with dementia show some fluctuation, known as 'sundowning' because the confusion worsens in the evening, but this can be surprisingly marked in Lewy body disease, even affecting conscious level. Weighted scores, such as the Hachinski ischaemia score, may improve diagnostic accuracy and work is in progress to determine whether patterns of change found on neuropsychological and language testing add to diagnosis.

Investigations typically include blood tests to exclude reversible causes or other major pathology (blood count, biochemical profile, ESR, thyroid function, B_{12} and folate and syphilis serology) CXR and ECG and a CT scan or MRI. In the late stages, a CT scan usually shows cerebral atrophy but many patients with AD have a normal looking scan initially. The main purpose of the scan is to rule out a space-occupying lesion and identify major vascular disease. Genetic tests, such as determining the apolipoprotein E alleles that predispose to AD, are not routine.

Management

Management depends on the severity of the dementia, whether the patient lives alone and comprises a multi-disciplinary, multi-agency package of care. The package needs to be well co-ordinated and to evolve as the needs of the patient and carer change. Options include:

- Coping strategies and psychological techniques, reminiscence work and validation therapy.
- Optimize hearing, vision and improve general health.
- Treat other conditions which may impair cognition (e.g. anaemia, heart failure).
- Treat risk factors (e.g. hypertension in vascular dementia).
- Treat specific symptoms and behaviours (major tranquillizers, unfortunately, are often the only option).
- Education and support for carers (Alzheimer's Disease Society, Carers' National Association).
- Genetic counselling (only in rare early-onset dementias).
- Legal advice (e.g. an Enduring Power of Attorney may obviate the need for the Court of Protection at a later date, advice about driving, advance directives, etc).
- Therapy assessments (occupational therapy, speech and language therapy (for swallowing and communication) and physiotherapy; the aim is usually assessment to plan appropriate care and advise carers, rather than treat the patient).
- Assessment by social services (financial entitlements, especially attendance allowance, provision of services like home help and access to 'care management', the process by which frail old people are assessed for substantial packages of care at home or residential care).
- Regular district nurse/community psychiatric nurse support.
- Sitting services (Crossroads), day hospitals, respite care.
- Proper provision of long-term care.

This list demonstrates just how much can be done in dementia. Until recently, no specific treatment was available. Drug management focused on the effects of the disease (e.g. major tranquillizers for disturbed behaviour) but advances in our understanding of the pathological processes in AD have led to the development of drugs to ameliorate the underlying biochemical changes.

Notes on Alzheimer's disease

AD is divided into early-onset familial AD (EOFAD) and the usually sporadic late-onset form (LOAD). The pathology of both is identical with characteristic amyloid-containing extracellular **plaques** and the abnormal material which develops inside the neurons, the **neurofibrillary tangles**. It has been more than 10 years since it was first proposed that the neuronal degeneration in AD may be caused by deposition of amyloid beta-peptide (Aβ) in plaques in brain tissue. According to the amyloid hypothesis, accumulation of Aβ in the brain drives the pathogenesis of AD. The rest of the disease process, including formation of neurofibrillary tangles containing tau protein, is proposed to result from an imbalance between Aβ production and Aβ clearance.

Three genes have been linked with EOFAD and all probably increase the brain levels of the amyloid precursor protein (see Table 4.1).

Risk factors for Alzheimer's disease

- Down's syndrome. Essentially all people with trisomy 21 develop the neuropathological hallmarks of AD after 40 years of age. More than half such individuals also show clinical evidence of cognitive decline. The presumed reason is the lifelong over-expression of the amyloid precursor protein and resultant over production of the Aβ-amyloid.
- Age.
- Female sex.
- Apolipoprotein E4 genotype.
- Head injury.
- Elevated homocysteine levels (can be decreased by folate*).

GENES AND ALZHEIMER'S DISEASE

		Chromosome	
EOFAD			
β-amyloid	APP	21	Associated with Down's syndrome
Presenilin 1	*PSEN1*	14q	Commonest gene defect in EOFAD
Presenilin 2	*PSEN'*	1q	
LOAD			
Apolipoprotein E	ApoE	19q	Polymorphic: e2 e3 e4 are the common isoforms. e4 is associated with atherosclerosis, coronary heart disease, vascular dementia, early and late onset AD
?		10, 12	At least four other loci are postulated

EOFAD, early-onset familial Alzheimer's disease; LOAD, late-onset Alzheimer's disease.

Table 4.1 Genes associated with familial and sporadic AD.

Protective factors for Alzheimer's disease

- Education (partly a threshold effect, but ? confounding with other associations with social class, e.g. diet high in antioxidants*).
- Continued brain activity (keep reading!).
- Tobacco (may be the nicotinic effect on cholinergic transmission, but not worth it).
- Wine and coffee (so it's not all bad news!).
- Exercise.
- Diet rich in foods containing vitamin E but not vitamin E supplements*.
- Non-steroidal anti-inflammatory drugs and aspirin.
- Hormone replacement therapy (observational data but as hormone replacement therapy users are fitter than average, trials are needed).
- Treatment with a statin (could be reduction in vascular damage but trials are needed).

The metabolism of acetylcholine
(see Fig. 4.1)

Cholinergic transmission could be enhanced by increasing the availability of the precursor, via direct stimulation of the receptors or by preventing the breakdown of endogenous acetylcholine. Three drugs, which are all acetylcholinesterase inhibitors, are available (Table 4.2). Acetylcholinesterase inhibitors are widely available in some countries but in the UK should be prescribed according to NICE guidelines (2001):

- Diagnosis of AD made in a specialist clinic.
- MMSE ≥ 12.
- Carer's views considered and feasible to expect compliance.
- Drug continued if cognitive or behavioural benefit (stable MMSE indicates benefit as decline is expected).
- Six-monthly review and drug stopped if benefit no longer apparent or MMSE falls below 12.

*Large trials are in progress with vitamins B_6, B_{12}, C, β-carotene and folic acid, oestrogens, celecoxib, simvastatin, selenium and ginkgo and will report between 2003 and 2008. Other options include β- and γ-secretase inhibitors (to modulate amyloid plaque formation) or vaccination with Aβ following successful studies in a mouse model of AD. However, the specific treatments for AD which are currently in use are based on earlier work showing that the neurons which bear the brunt of the damage are those which use acetylcholine to transmit messages.

ALZHEIMER'S DISEASE—CHOLINESTERASE INHIBITORS

Drug name	Recommended dose	Common side-effects	Possible drug interactions
Galantamine Prevents the breakdown of acetylcholine and stimulates nicotinic receptors to release more acetylcholine in the brain	4 mg, twice a day Increase after 4 weeks to 8 mg twice a day After another 4 weeks, increase to 12 mg twice a day if well tolerated	Nausea, vomiting, diarrhoea, weight loss	Prescription with inhibitors of cytochrome P450, e.g. paroxetine, amitriptyline or ketoconazole may cause cholinergic toxicity due to decreased metabolism of galantamine. Caution with all three drugs in sick sinus syndrome, peptic ulcer and hence NSAIDs, COPD, urine retention
Rivastigmine Prevents the breakdown of acetylcholine and butyrylcholine (a brain chemical similar to acetylcholine) in the brain	1.5 mg, twice a day Increase by 3 mg/day every 2 weeks to 6 mg twice a day Continue up to 6 mg twice a day if well tolerated	Nausea, vomiting, weight loss, upset stomach, muscle weakness	
Donepezil Prevents the breakdown of acetylcholine in the brain	5 mg, once a day (nocte to minimize nausea) Increase after 4–6 weeks to 10 mg once a day	Nausea, diarrhoea, vomiting	

Table 4.2

Although the licensed indication is AD, trials have shown benefit in Lewy body dementia and vascular dementia, and so the lack of diagnostic precision is not a danger.

Although the cholinergic system is primarily affected other neurotransmitter systems are involved and memantine, an *N*-methyl-D-aspartate receptor antagonist (which reduces glutamate-induced neurotoxicity) has just been licensed in the UK for the treatment of moderate to severe AD. The fact that it may also be useful in vascular dementia, neuropathic pain and glaucoma indicates that this is a rather non-specific approach.

Frontotemporal dementia

Frontotemporal dementia is characterized by gradual changes in personality, social behavior, and language ability. Symptoms depend on whether the damage has primarily affected the right (behavioral problems) or left side (language deficits) of the frontal and anterior temporal lobes that control executive functioning. Frontotemporal dementia usually develops between 35 and 75 years of age and so, although it is rare (about 3% of dementia cases), it may present at an age when patients are labelled with AD. Orientation and memory are better preserved than in AD.

Pathologically, it is characterized by neurofibrillary tangles. These are known to be abnormally processed microtubule proteins. The microtubule-associated protein tau promotes tubulin polymerization and has a role in stabilizing the microtubules that are responsible for neuronal architecture and transport. A mu-

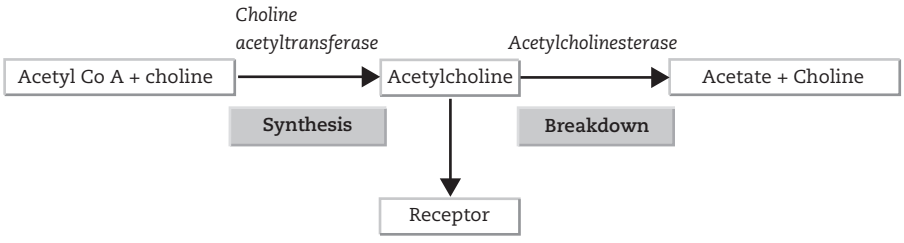

Fig. 4.1 The metabolism of acetylcholine.

tation in the *tau* gene causes a form of frontotemporal dementia called frontotemporal dementia with Parkinsonism linked to chromosome 17 (FTDP-17). Mutations in the *tau* gene impair the binding of tau protein to the microtubule and so frontotemporal dementia is one of the 'tauopathies'.

CADASIL

Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy is characterized by a history of migraine, mid-adult (30s–60s) onset of cerebrovascular disease progressing to dementia, and diffuse white matter lesions and subcortical infarcts on neuroimaging. The pathological hallmark of CADASIL is electron dense granules in the media of arterioles that can often be identified by electron microscopic evaluation of skin biopsies. More than 90% of patients have mutations in the *Notch3* gene (chromosomal locus 19p). Molecular genetic testing is available.

Transient global amnesia

A curious episodic disorder predominantly affecting older people. It is of unknown cause and is not predictive of stroke or dementia. In an episode the person remains alert and capable of high-level intellectual activity (e.g. driving), but if questioned may be perplexed and has impaired memory for past and present events.

Features

- Sudden-onset amnesia—retrograde for recent events, anterograde preventing new memories being laid down.
- Bemusement, perplexity, disorientation, repetitive questioning.
- Preservation of alertness, verbal fluency, motor activity.
- Duration a few hours, although complete recovery may take a few days; low recurrence rate.

Self-neglect

Old people are not infrequently encountered living in conditions of extreme degradation with total disregard for hygiene and self-care, the 'senile squalor syndrome'. Some will be found to have mental illness but others appear normal despite hoarding vast quantities of rubbish. This has been termed the Diogenes syndrome after Diogenes of Sinope, the ancient Greek philosopher who showed his contempt for material things by living in a barrel. He believed that happiness is attained by satisfying one's natural needs in the cheapest and easiest ways possible. In this context, the perpetrator is seen to have

Risk factors associated with self-neglect

- Dementia.
- Depression.
- Bereavement and isolation.
- Disability.
- Alcohol.
- Previous psychiatric disorder.
- Mental subnormality.
- Obsessive-compulsive disorder.
- Lifelong difficult personality/eccentricity.

made a bizarre lifestyle choice, rather than having an illness, but the condition may lead to hypothermia, malnutrition and infections as well as vigorous protests from the neighbours!

Further information

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