"A medical revolution has extended the life of our elder citizens without providing the dignity and security those later years deserve."

1. Introduction

From an evolutionary point of view there must be some advantage for the continued survival of mankind following the end of reproductive life. Since humans are unique in both the richness and complexity of their memories and experiences, and have the ability to share this knowledge with others, one presumes this to be a major reason. While each generation adds to the gifts it has received from its ancestors, the lessons taught are unfortunately not always fully integrated. Hence, history repeats itself unnecessarily and painfully, the elderly being perceived at a very primitive level akin to a “problem” rather than for the contribution they could make. Going by the dictum that “in the old days parents had many children, and now children have lots of parents” this “problem” has uniquely become our problem. The challenge, therefore, is to change this paradigm, allowing the elderly to continue to contribute to society within the social model of the extended family, in spite of the increasing number of nuclear families. One of the first steps in this direction is to ensure their psychological and physical health and the promotion of a sense of well-being; so that they become an asset and not a burden to society.

2. Ageing

Definitions

Ageing is the progressive decline in function and performance, which accompanies advancing years. It is multifactorial in origin, partly inborn (primary ageing) involving concepts such as apoptosis or programmed cell death, biological clock and other genetic factors, and partly environmental (secondary ageing) due to a “wearing out” by stress and strain and the accumulation of “toxins”. Gerontology covers all knowledge pertaining to the ageing process; Geriatrics deals mainly with the care and treatment of physical illness in the elderly; while Psychogeriatrics involves the socio-psychiatric and cognitive disorders and their management in the elderly. It is the fastest growing field in psychiatry.

Demographic effects

The United Nations predicts a world population of 9 billion for the year 2070, of whom 34% will be over the age of 60. The international trend is to describe as “elderly” people aged 65 and over, while lesser developed countries would include those over the age of 60 years in order to highlight the current needs of the aged. South Africa’s “Rainbow Nation” reflects a demographic diversity, in that elderly blacks constitute 6.5% of their population group, compared to whites at 16%, with Indians and coloureds in between. The current 3.3 million (7.3%) elderly aged 60 years and older will more than double within the next 25 years. In general, this increase has been due to reduced fertility rates and decreased mortality rates in the young, rather than any marked extension in life expectancy. Thus, future demographic trends are dependent on measures of population control and improved health services. Under normal circumstances, 2.2 children per family unit are required to maintain steady population numbers.

From a variety of data a theoretical lifespan of about 120 years has been calculated in humans. Owing to mental and physical factors, however, we not only die at an earlier age but also live a portion of our lives disabled. This amounts to 18% of our given life-years in lesser developed countries as opposed to 8% in developed countries. At present, in lesser developed
countries in the poorer socio-economic sectors, the life expectancy of a female is roughly 64 years and that of a male roughly 60 years; contrasting with 77 and 72 years respectively in the more affluent sectors. Though AIDS in South Africa is steadily reducing the life expectancy of certain population groups; it should be noted that persons of any population group currently approaching the 60-year mark can look forward to an average of another 18 years of life. The causes of death in those over the age of 65 years in developed countries are generally considered to be cardiovascular disease 53%; neoplasms 17%, and respiratory disease 14%. In fourth place are the neurodegenerative disorders (such as Alzheimer’s disease), which are becoming notably more prevalent as other causes are brought under control.4 There is a mutual relationship between old age and disease: disease hastens ageing and age renders the old person more vulnerable to diseases, especially of the degenerative kind.1

Psychosocial implications

The increase of elderly in the upper age brackets has enormous financial implications. Approximately 30% of the elderly suffer from psychiatric symptoms while 80% suffer from some physical illness, and many from both.1,4 Individuals in this group are on at least one medication, thus the need for setting up effective services for the elderly becomes quite clear. The most common psychiatric illness in the elderly is depression, at a prevalence of about 18% in women and 12% in men. Dementia, depending on age category, has a much lower prevalence rate of 5-10%, frequently complicated by bouts of delirium. Anxiety disorder (often comorbid with depression) is present in some 15% of elderly while a third will complain of sleep disturbance.5 Prior to the mid 1980’s there was a tendency to keep psychiatically disturbed elderly patients in hospital. Following a change in policy, mainly because of more effective medication, these patients are now increasingly being placed back in the community, resulting in an ever-increasing burden on outpatient health services, non-governmental organisations and individual caregivers.

With regard to community care, one has to remember that in the last 50 years in more developed countries the proportion of “middle-aged” (between 35 and 65 years) to elderly, has dwindled from 10 to 1 roughly 4 to 1. This means that less people are supporting elderly pension funds and providing financial aid towards their health requirements.1 Over the same period the proportion of middle aged women (the caregivers of the elderly) going out to work has risen from some 10 to 60%. Lesser developed countries are now pursuing this trend. In lesser developed countries up to 50% of the population is aged less than 25 years.5 These young in turn compete with the elderly for support from the high income 25 to 45 year age group. The latter group, though, is contending with problems of its own. They have a very high HIV-infection rate due to their projected “steady state” of 32% within a decade. Other problems include dwindling natural and economic resources, competition with the more advanced nations on an industrial and technological level, the high rate of unemployment and the effects of migration.6 Some 25% of potential workers are unemployed in South Africa, and most of these originate from the poorer socio-economic groups. In these instances a carer is available for the elderly, but the latter’s pension is often now the chief source of income for that particular family. In addition, the care required for the mental illness is often beyond the scope of most families. All these factors need to be taken into account by policy-makers.

Generally the elderly are easy victims of poor housing and poverty, pensions that cannot keep pace with the daily cost of living and a decreasing intellect that gives rise to an inability of using financial resources to best advantage. All this requires an increase in the number of services for the elderly, for example primary health care, home visiting, day centres, liaison services, day hospitals, hospital based services as well as residential and old age homes, council houses and villages.1

Physical changes

Height decreases by some 8cms by the age of 80 years owing to a decrease in bone mass and an increased curvature of the spine. Body weight steadily decreases, while joints stiffen and osteoarthritis becomes more common. The skin becomes dry, thin and wrinkled; and joints stiffen and osteoarthritis becomes more common. The enlarged prostate is common and troublesome. The enlarged prostate gland in males and the atrophic vagina in females may lead to urinary tract infections, contributing to incontinence and impaired health. Other physical ailments to which the elderly are especially prone include diabetes, dehydration, anaemia, cancer and hypothermia.1,7

Central nervous system

Mental function in the elderly is spared more than the dramatic microscopic changes in the brain would suggest. Cell loss approaches 50% in some cortical areas while fewer dendritic interconnections remain between neurons. All senses decline - deafness and
failing vision leading to social isolation, while loss of sense of smell and taste lead to a decrease in appetite.

Intellectual function

A small percentage of elderly persons maintain a stable IQ even at advanced ages, although the great majority demonstrate a gradual decline. Memory, contrary to belief, is not static throughout one's lifetime, nor is it dramatically altered in healthy, emotionally stable elderly persons. “Fast mapping” (in young children) is followed by “fluid intelligence” (rote memory and the ability to acquire new information), which is more susceptible to the effects of ageing than “crystallised intelligence” (which synthesises and integrates acquired information with the person’s educational and past experience). The latter increases with age and offsets the former. Responses may be slowed but are again offset by experience and knowledge.1,7

Sexual function

Although all phases of the sexual act tend to be slowed down and prolonged in the elderly, decreased sexual interest and activity in this age group - though in part hormonal in aetiology and due to a lack of partner opportunity - is generally psychological rather than physiological. While some are sexually active others find intimacy sufficient.1

Personality changes

There is a natural tendency from extroversion towards introversion with ageing and as the circle of friends diminishes, so does the interest in social and current affairs.1 The elderly become more set in their ways and are often preoccupied with bodily functions (such as the bowels), which may lead to hypochondriasis.

Life changes

Specific life changes and “losses” are associated with this stage of life.1 Contemporary western society with a tendency towards materialism holds “senior citizens” in lower status than their working counterparts. Thus, retirement can “age” a person, and those with a high investment in a work role more easily lose self-esteem and become depressed. Income drops substantially following retirement requiring careful budgeting and adjustment to inflation. Health deteriorates often accompanied by discomfort or pain, which apart from restricting mobility and reducing social interaction generally impairs the enjoyment of life. This increasingly leads to dependency. Colleagues, friends and spouse (company) are lost with advancing years resulting in progressive isolation with the loss of meaningful communication with others and especially the support of confidants. Elderly men living alone are the most vulnerable to suicide. As the standard of accommodation declines because of dwindling financial resources, the elderly may find themselves placed considerable distances away from suitable shopping areas, access to transport and health services. The above contributes to a loss of independence as family and other social support systems increasingly have to be called in to maintain a semblance of independent living. This results in a role reversal that is frequently uncomfortable or painful to the elderly as well as those near to them. With impending closure of life there is a re-evaluation of past experiences and achievements, their meaning and purpose. The unfulfilled frequently become very difficult and bitter in temperament.1

Styles of ageing

Some maintain that the elderly cope best if they accept the inevitability of ageing, a quieter life and reduced social contact. Others, again, stress that the elderly, aware of certain failing skills, must make all the more effort to counteract this deterioration in order to maintain a sense of purpose and satisfaction. The answer probably lies between the two, dependent on factors such as personality type, cultural background and former interests. In practice, the mental health worker will frequently find the style of ageing dominated by: anxiety and hypochondriasis, bewilderment and indecision, irritability and frustration, defiance, denial and dependency.1 Research has shown that any fieldworker acting as a close friend and “sounding board” to the elderly has a pivotal role to play in relieving the above. Some 70% of elderly, however, will adapt constructively.4,5

Needs of the elderly

The basic needs of the elderly are those of people generally, but unfortunately, are usually not met. They consist of physical needs such as nutrition, shelter, warmth, comfort and cleanliness, while their psychological needs encompass respect, security and self-determination.1

3. Principles of assessment

History taking

Firstly, it must be established what illness is in fact present. This may be difficult since the individual’s reaction to the illness, the presence of drugs, physical illness, emotional upheavals and depression may all complicate the picture. Collateral information from a spouse, family or friends is essential and patients need to be reminded to bring along their glasses and/or
hearing-aids, medications, and medical reports to the first interview.

Functional and social assessment

This is mandatory in order to plan future care. The aim is to assess the degree of impairment as well as the retained abilities, in order to maintain the person in the community. It involves the assessment of: mobility, ability to communicate needs, ability to relate to others, ability to wash/dress self, ability to feed self, control of bladder and bowels and the presence of aggression and other socially unacceptable behaviour. The patient’s present social functioning and ability for self-care need to be evaluated with regards to: supervision required, ability to prepare meals, ability to go shopping, ability to do housework and compliance with medication. The social assessment takes into consideration accommodation, employment, economic resources, and evaluates the degree of available social support. The social assessment also incorporates financial and medico-legal matters such as Testamentary capacity, Wills, Power of Attorney and need for curatorship. With the above-mentioned knowledge, each patient’s future can be projected and planned with regard to care and placement.

Examinations and investigations

The illness in question determines the scope and depth of examinations and investigations required. Often this entails both a physical and neuropsychological assessment concentrating on neurological deficits. The Mini-Mental Status Examination (MMSE) should be administered routinely (see elsewhere in this chapter). Generally, in typical or advanced cases of dementia, investigations may have little to offer towards clinical diagnosis, treatment and health benefit. Cost restraints often dictate that investigations cannot routinely be performed. However, a positive result is more likely to be obtained when; the patient is below 65 years of age; the dementia has been of recent and rapid onset; the course of the disease fluctuates markedly and the physical examination reveals a neurological deficit. Should the cause of a delirium not be found more investigations are mandatory.

Baseline investigations consist of the following (the so-called “organic work-up”): Full blood count and when indicated plasma viscosity; urea and electrolytes; thyroid function tests; liver function tests; random blood sugar; calcium and phosphate; total cholesterol; niacin; vitamin B12 and red cell folate; tests for syphilis and HIV; chest X-ray and mid-stream specimen of urine depending on dip-stick urine (e.g., Combur 9 Test) result. More specialised investigations would include CT scan and/or psychometric testing when there are difficulties with clinical assessment or for medico-legal purposes. A CT or MRI scan measuring the width of the medial temporal lobes is a useful adjunct in the assessment of Alzheimer’s disease.

4. Depression

In the elderly, depression is the commonest psychiatric disorder, affecting 18% of women and 12% of men. It is more common in the urban than in rural areas and in the working classes. It is usually multifactorial in aetiology with a broad range of causes being responsible at any given time.

Physical stresses that may precipitate depression include viral brain infections (occurring especially during the post-influenza period) any condition affecting the brain (Parkinson’s disease, early Alzheimer’s disease or vascular dementia), anaemia, cancer, endocrine disturbances, hypothryoidism, and drugs (L-dopa, Aldomet®, reserpine).

However, psychological stresses are usually more important in depression. Predisposing factors consist of a variable hereditary contribution, traumatic childhood experiences, bereavement during childhood and personality. Obsessional, conscientious personalities, who are unable to display their emotions are more at risk. So are individuals lacking a close friend or confidant.

Depression in the elderly appears in numerous guises, ranging from frank psychotic symptoms to the more common, vague, somatic or neurotic complaints with little or no overt sadness. In practice one or more of the following features will dominate the picture: (Figure 1) anxiety, irritability/aggression, and depression. The physical symptoms are often vague or hypochondriacally in nature. More commonly, we find the elderly patient’s underlying physical ailments exacerbated by depression, and the “unpopular” patient who goes “doctor shopping” is often a patient suffering from depression. Alternatively the patient may merely be extremely agitated, anxious or tense, and are then incorrectly treated with benzodiazepines and other tranquillisers. Others again present with irritability or in its extreme, aggression.
Lastly, some patients will only admit to feeling depressed or "down" on questioning. The reason for this may be the ability of the elderly to compartmentalise their mood state, thus making it possible for them to continue with their daily tasks, rather than a lack of insight into their depressed condition. Others again, see depression as a sign of moral weakness and will not readily admit to being depressed. The core criteria for the presentation of major depression are (Table 1):\textsuperscript{10}

<table>
<thead>
<tr>
<th>Table 1 Core criteria for major depressive episode: DSM-IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Five or more of the following symptoms have been present over a 2-week period (most of the day on most days) including one or both of the first two:</td>
</tr>
<tr>
<td>* Depressed mood (usually worse in the mornings)</td>
</tr>
<tr>
<td>* Loss of interest or pleasure in activities</td>
</tr>
<tr>
<td>* Significant weight loss or gain with corresponding appetite</td>
</tr>
<tr>
<td>* Insomnia (usually with premature awakening) or hypersomnia</td>
</tr>
<tr>
<td>* Psychomotor agitation or retardation</td>
</tr>
<tr>
<td>* Fatigue or loss of energy</td>
</tr>
<tr>
<td>* Feelings of worthlessness or excessive or inappropriate guilt</td>
</tr>
<tr>
<td>* Diminished ability to think and concentrate or indecisiveness</td>
</tr>
<tr>
<td>* Recurrent thoughts of death or suicide</td>
</tr>
</tbody>
</table>

Regardless of the presentation, a common finding is a decompensation in function; the person no longer seems to care about the state of his/her dwelling, bodily hygiene, pastimes or friends. In addition, the elderly will frequently express excessive concern regarding their bodily health or financial state. The more usual case seen in general practice will be suffering from depression as depicted below (Table 2):\textsuperscript{5,10}

<table>
<thead>
<tr>
<th>Table 2 Core criteria for ‘minor’ or ‘neurotic’ depression of the elderly (depression not otherwise specified): DSM-IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>The mood may be depressed, but more commonly there is a feeling of dissatisfaction or an inability to enjoy life (usually worse in the evenings). Sadness may alternate with periods of normal mood. At least three of the following symptoms must be present:</td>
</tr>
<tr>
<td>* Bouts of tearfulness or crying (often worse in the evenings)</td>
</tr>
<tr>
<td>* Irritability or bouts of anger</td>
</tr>
<tr>
<td>* Loss of interest or pleasure in activities</td>
</tr>
<tr>
<td>* Mild appetite disturbances, often with weight gain</td>
</tr>
<tr>
<td>* Sleep difficulties (usually an inability to fall asleep because of excessive worrying)</td>
</tr>
<tr>
<td>* Decompensation in function with neglect of home and bodily hygiene</td>
</tr>
<tr>
<td>* Fatigue or loss of energy</td>
</tr>
<tr>
<td>* Social withdrawal, avoidance of contact with friends</td>
</tr>
<tr>
<td>* Gloomy outlook on the future accompanied by brooding on the past and feelings of self-pity.</td>
</tr>
</tbody>
</table>

Depression is responsible for the majority of suicides in the elderly. Though this group only constitutes a small proportion of the population, they account for a third of all suicides. Most at risk is an elderly, single or recently bereaved male with a poor social support system. Agitated depressives are also at greater risks than apathetic or retarded depressives.

The treatment of depression ranges from the manipulation of social circumstances and resources, psychotherapy, antidepressants and tranquillisers, to electroconvulsive therapy and psychosurgery.

Following an empathic hearing, active and firm intervention is usually indicated, preferably involving the patient’s spouse, family members and friends. Guidance and pragmatic advice must be spelled out clearly. The choice of antidepressant is individual, based on experience with the drug, contraindications, cost, tolerability and availability of the drug. A suggested schedule is outlined in Table 3. Though more costly, the newer antidepressants in general, exhibit a more favourable side-effect profile. Should the aggression, agitation or insomnia not settle on antidepressant medication alone, additional treatment may be indicated. Benzodiazepines are not generally employed owing to (1) the greater degree of control necessary to avoid possible abuse (2) the possible occurrence of anxiety, emotional vulnerability and “morning hangovers” (3) excess sedation (4) amnestic episodes (5) ataxia with a predisposition to falls, and (6) problems with derealisation and delirium.
Table 3 Treatment schedule for depression

1. Selective Serotonin Reuptake Inhibitors (SSRI’S):

1.1 Fluoxetine (Prozac®), paroxetine (Aropax®), citalopram (Cipramil®)
   * 10mg mornings (citalopram preferably at night)
   * Review at 4 weeks
   * Increase to 20mg if necessary

1.2 Sertraline (Zoloft®), fluvoxamine (Luvox®)
   * 50mg mornings or evenings (fluvoxamine preferably at night)
   * Review at 4 weeks
   * Increase in 4-weekly increments of 50mg (to 150mg maximum) if necessary

Side-effects include: headache, nausea, agitation, anxiety, tremor, insomnia, akathisia, sexual dysfunction.

1.3 Escitalopram (Cipralex®)
   * 5mg at night
   * Review at 4 weeks
   * Increase to 10mg if necessary

2. Other agents are:

- Tetracyclic Antidepressants: mianserin (Lantan®)
- Modified Tricyclic Antidepressants: lofepramine (Emdal®)
- Reversible Monoamine Oxidase Inhibitors: moclobemide (Aurorix®)
- Serotonin and Noradrenaline Reuptake Inhibitors (SNRI): venlafaxine (Efecto®)

3. Noradrenergic and Specific Serotonergic Antidepressants (NaSS A):

- Mirtazapine (Remeron®)
  * 15mg at night
  * Review at 4 weeks
  * Increase in 4-weekly increments of 15mg (to 45mg maximum) if necessary

On reaching the highest level of tolerance or maximum dose, treatment should be continued for at least three months before deciding that the patient has not responded. Should the depressed elderly person not respond adequately to the above regimens, consultation with or referral to a more specialised centre should be sought. Electroconvulsive therapy (ECT) is still one of the most potent treatment applications in our armamentarium. Depression in the elderly is eminently treatable, but owing to the high relapse rate (80% in the following two years) prolonged after-care and maintenance therapy for this period is mandatory.¹ ²

5. Bipolar disorder

Most of these patients have a past history of manic or depressive illness and thus “graduate” into this age group.

First onset mania can occur in this age group but is rare. Exclude an underlying physical illness for example stroke, early dementia, or substance abuse such as alcohol, as the precipitating cause. In many manics the features of mania are replaced by lability of mood, irritability and often some paranoia. Often, both hypomania and depression co-exist ("mixed bipolar"). Both the therapeutic and toxic effects of lithium occur at lower blood levels in old age. This necessitates closer monitoring, and lithium levels of no higher than 0.4 – 0.6 mmol/L are recommended¹ ² for maintenance therapy while higher levels may be required in the acute phase. Anticonvulsants such as carbamazepine (Tegretol®), sodium valproate (Epilim®) and lamotrigine (Lamictin®) may yield better results than lithium.

6. Anxiety and agitation

Anxiety is prevalent in 15% of elderly and as a symptom often co-exists with another disorder such as depression or dementia. Anxiety disorders such as panic disorder, phobia, obsessive-compulsive disorders, generalised anxiety disorders, acute stress reactions and posttraumatic stress disorder do occur in the elderly. Anxiety disorders may make their first appearance after the age of 60 years. Treatments of choice are antidepressants (Table 3) and less commonly major tranquillisers when dementia is present. The elderly are susceptible to the occurrence of sedation, confusion, impaired psychomotor performance and amnestic episodes when given benzodiazepines.¹

Sustained agitation in the elderly occurs in more than 70% of patients suffering from dementia or delirium and is thus treated with major tranquillisers and the appropriate medication for any associated medical condition. Though a third of elderly depressed patients present with “agitated depression”, antidepressants remain the treatment of choice in this situation.

7. Sleep disturbances

A third of elderly suffer from sleep disorders. Insomnia, drowsiness during the day, daytime naps and the use of hypnotics are common. The causes of sleep disturbances include primary sleep disorders, mood disorders (e.g., depression), medical conditions and environmental factors. Of the primary sleep disturbances the dysomnias are the most common including primary insomnia, nocturnal myoclonus, restless leg syndrome and sleep apnoea. Pain, nocturia, dyspnœa and heartburn are the medical symptoms that most affect sleep. The absence of a daily rhythm may disturb sleep such as occurs in institutions where
elderly go to bed very early, (e.g., 17h00) and then find themselves insomniac in the early morning hours. More commonly, extended afternoon naps (in excess of 20 minutes) lead to disturbed sleep cycle rhythms with somnolence during the day, mild confusion and incontinence at night. Also check for premorbid sleepwalking (in the patient’s youth) as a cause of nocturnal confusion as this condition is prone to recur in old age. Sleepwalking and restless leg syndrome can be treated with benzodiazepines which pre-empt their occurrence. Alcohol even in small quantities may affect the quality of sleep through fragmentation and early morning wakening. It may also precipitate or worsen sleep apnoea. When prescribing hypnotics attention must be given to side-effects such as cognitive impairment, drowsiness, psychomotor retardation, ataxia and falls, morning “hangovers” and blurring of biological day/night rhythms. In long-standing insomnia low dose antidepressants (e.g., citalopram) are the treatment of choice (Table 3).

8. Paraphrenia

Also known as late onset schizophrenia or persistent persecutory states in the elderly. As a symptom, paranoia, ranging from vague suspiciousness to delusions of persecution, may be seen in the elderly and are not uncommonly associated with mood disorder (such as depression) and acute or chronic brain disorder (delirium and dementia), or are found to have been present as a personality trait (personality disorder), throughout life.¹

Clinical features

Paraphrenia is a distinct psychiatric disorder occurring for the first time in life in 1% of the population over the age of 60. It is characterised by systematized, that is highly circumscribed paranoid (usually persecutory) delusions, and hallucinations, which may occur in a variety of sensory modalities, in the absence of a primary affective illness or obvious organic aetiology.⁵,¹²

The paraphrenic patient is typically female, solitary, partially deaf and eccentric (hostile and prickly), but without a past history of serious psychiatric illness.¹² There may, however, be an excess family history of psychiatric illness as a whole and particularly schizophrenia.⁵ The clinical picture is one in which an old person becomes convinced that her neighbours or family are interfering with her and are attempting to harm her. Delusions usually involve “high-tech” devices such as satellites and M-NET, which spy on her, but the exact mechanisms remain unclear. Misidentification syndromes are common, as are partition delusions, in which the walls or other boundaries of the home are inexplicably breached by observers and tormentors. Persecutory hallucinations may occur, the situation deteriorates, and ultimately the patient is referred for treatment by long-suffering neighbours and health staff. By virtue of omission in the DSM-IV, paraphrenia would straddle the diagnostic categories of delusional disorder, schizophreniform disorder and schizophrenia (paranoid type), depending on the severity and range of symptoms of the presenting illness, respectively:¹¹

1. Delusional disorder where the patient’s delusions are strictly localised to one particular neighbour who is heard talking sometimes to her and is believed to enter the patient’s dwelling and interfere with her belongings.

2. A schizophreniform type where delusions are more widespread, often extending into the neighbourhood and the street. There are ideas of reference so that people seen talking together are thought to be talking about the patient, car lights are flashed ominously, and special optical and bugging devices are used to spy on the patient. There may also be delusions of jealousy about the spouse and erotic delusions occur.

3. A schizophrenic type, almost identical with paranoid schizophrenia in the younger patients, in which the subject hears herself discussed in the third person and experiences passivity feelings for example being influenced from a distance or having her thoughts read. Personality tends to be better preserved than in schizophrenia and thought disorder is confined to occasional neologisms (private words) and metonyms (ordinary words used with a private meaning). These differences and the more appropriate emotional and behavioural responses are explicable by the paraphrenic's more established and mature personality at the age when the disease first develops.

Treatment

Major tranquillisers/neuroleptics are indicated. Do not overtreat - delusions or delusional activity will “stop” but not disappear as it does in the younger age group. Lack of insight and judgement usually necessitate depot neuroleptics if not placementaffording greater supervision and care. Watch out for neuroleptic sensitivity and/or emerging depression after 6-12 weeks of treatment and treat accordingly (Table 3).¹

9. Abuse of the elderly

In the USA it is estimated that 10% of the elderly are abused.¹² Figures for South Africa are not available. Abuse of elderly includes physical abuse as well as “acts of omission” or negligence, leading to the detriment of the health and well-being of the person. This would include physical, psychological, financial and material aspects. Examples would be the denial of food, visits, medication, clothing and other essentials. Note that sexual abuse and incest also occurs.
10. Hypersexuality

A form of sexual disinhibition characterised by excessive, socially inappropriate sexual behaviour. It may occur in both the very early and more advanced stages of dementia. Control is rapidly obtained within a few days with the use of an anti-androgen cyproterone acetate (Androcur®) available in both oral (50-100mg 3x daily) tablets and depot injection (300mg/2 – 4 wks). Initiate and maintain treatment for a period of approximately 6 months. If, on cessation of oral therapy symptoms recur within a few days, further treatment for another 3 – 6 months is indicated.

References


Recommended reading

2. Lishman WA. Organic Psychiatry: The Psychological

disturbances:
* Aphasia
* Apraxia
* Agnosia

Disturbance in executive functioning

The cause is a medical condition, post-substance abuse or a combination of both.

Epidemiology

The prevalence of dementia in the general population is 5%-10%. Starting at 1% for 60 year olds the incidence of dementia doubles every five years, rising to 30-40% for those over the age of 85 years.

Aetiology

The syndrome of dementia becomes a diagnosis once the cause of the dementia has been established, often only definitively at post-mortem examination. In developed countries Alzheimer’s disease accounts for some 50% of all cases of dementia, vascular dementia for 20%, and mixed causes (i.e., both Alzheimer’s and vascular) for 15%. The latter group is expanding on the grounds of definition, aetiology and number of elderly. The remainder consists of dementia associated with alcoholism (5%) and dementia associated with other causes (such as HIV dementia, Parkinson’s disease, brain injury, intracranial irradiation, Huntington’s disease, Creutzfeld Jakob disease, frontotemporal dementias including Pick’s disease, neurosyphilis, hypothyroidism, tumours, pellagra, folate and vitamin B12 deficiencies). Note that HIV infection is steadily on the increase in sub-Saharan Africa and has reached epidemic proportions accounting for the highest number of dementias. HIV dementia gives rise to dementia with Lewy Bodies (DLB).

Dementia is mostly progressive and usually irreversible (cures are often misdiagnosed cases of delirium), but its profound psychosocial effects may be amenable to intervention, as may the causes hastening or complicating the disease process (e.g., depression). Hence early diagnosis is very important. Reversible dementias encompass conditions such as normal pressure hydrocephalus, tumours of the brain, neurosyphilis and hypothyroidism. In many of these, however, function is not fully restored to previous levels following treatment.

Diagnostic assessment

Establish whether dementia is in fact present. Explain to relatives that memory is not static mentioning the changes from “fluid intelligence” to “crystallised intelligence” with only a proportion (roughly one third depending on age group) becoming “dodderly”. Questions addressed to informants should centre around the following points:

1. History

1.1 Memory impairment: In excess of age-related cognitive decline, that is, poor memory must interfere with daily functioning. Articles are mislaid, faces not recognised and disorientation regarding time or place occurs. Statements need to be repeated and the patient is forgetful. Initially there is difficulty in learning new information (short-term memory), but later on long-term memory is also affected. Increasingly the person lives in the past.

1.2 Personality and emotional changes: Usually this involves either an accentuation, or less commonly, an alteration (a “different side” or “shadow” comes to the fore) of pre-morbid traits, for example impulsive, aggressive or paranoid tendencies. There is a loss of initiative and the individual becomes increasingly apathetic and withdrawn. The patient becomes self-centred, hypochondriacal, argumentative and lacking in self-care, and can be said to have entered into his “second childhood”. Impaired impulse control may be observed for example social disinhibition, clumsy shoplifting, inappropriate spending of money and exhibitionism. Emotional changes occur in that sensitivity, interest and affection may disappear with decreasing rapport. The mood is not usually depressed and an emotional shallowness is more common. Bouts of irritability are common.

1.3 Intellectual impairment: Thinking becomes more primitive and the person cannot cope with novel tasks. The higher cortical functions are affected, namely:

Dysphasia manifests itself in the patient’s inability to read, listen to the radio, watch TV and their language may become vague, stereotyped, imprecise and dysphasic (jargon dysphasia). They may struggle to understand social communication. “Simple” language has to be employed.

Agnosia presents in the patient’s inability to recognise familial faces, a struggle to identify objects and difficulties in finding his way in a known area.

Apraxia shows itself in the inability to perform acquired motor functions. The patient can no longer dress her-/himself, struggles to close and open doors, fold a letter and place it in an envelope. Others will struggle with crocheting and needle work.

Impairment in executive function may be one of the first symptoms of Alzheimer’s disease. The patient struggles with complex tasks such as using...
As the disease progresses, that mimic. This determines whether s/he can wash, dress and feed and toilet her-/himself (ADL – Activities of Daily Living) and are they coping. Where does the person live, under what circumstances, who takes care of the person and confusion at night.

2. Functional and social evaluation

Broadly speaking, the demented patients clinically present with overlapping cognitive (memory and intellect), behavioural and psychological symptoms. The neurological examination must be undertaken in all patients.

1.4 Physical changes: As the disease progresses, the patient appears unduly frail and weak, looks older than his or her chronological age, and is stooped in posture with a slow, shuffling gait. Sphincter control is reduced. There is usually poor appetite during the day, with bouts of restlessness and confusion at night.

3. Mini-Mental Status Examination (MMSE)

The MMSE is done routinely in elderly to evaluate mental function. In literate patients (7 years of schooling) cognitive aspects of mental function can be tested by using the MMSE (which was originally designed to distinguish dementia from the 10% of patients with depressive pseudo-dementia that mimic dementia). While depressed patients will obtain a high MMSE score, moderately demented patients will score 26 or less out of a maximum score of 30. There is no time limit for the completion of the test and the results are very useful for monitoring progress. AD patients lose an average of 2 – 3 points per year.

4. Physical examination

A physical examination with the emphasis on the neurological examination must be undertaken in all patients.

5. Special investigations

The cause of the cognitive impairment should be established. Special investigations will help to improve or rule out treatable causes of dementia. The "organic work-up" entails a full blood count, plasma viscosity, urea and electrolytes; thyroid, liver and parathyroid function tests; random blood sugar, niacin, vitamin B12 and red cell folate, VDRL, HIV and urine dipstick. More specialised investigations would include a CT or MRI scan (with measurements of the medial temporal lobe) as well as psychometric testing.

Cost restraints and other practicalities often dictate the number of investigations that can be performed. A "positive" result is more likely to be obtained when a patient is below 65 years of age, the dementia has been of recent and rapid onset, the course of the disease fluctuates markedly and the physical examination reveals a neurological deficit.

Types of dementia

1. Alzheimer’s disease

The neuropathological hallmarks of Alzheimer’s disease (AD) are: amyloid plaques, neurofibrillary tangles, synaptic and neuronal loss with subsequent brain atrophy. Pathology at micro-vascular level has increasingly been implicated in the aetiology of AD, blurring the boundaries with vascular dementia (VaD). AD and most other dementias tend to follow a sinusoidal course in that the initial slow, progressive deterioration accelerates rapidly before flattening out towards the end (Figure 1). The illness may be as short as six months or as long as 20 years with an average of 10 years.

AD patients’ brains at macroscopic level (MRI and CT scan) are shrunken with flattening of gyri, widening of sulci, atrophied medial temporal lobes and enlarged ventricles. Microscopically there is synaptic and neuronal loss, and the presence (especially in the cortex and hippocampus) of senile plaques containing amyloid protein as well as intracellular neurofibrillary tangles containing paired helical filaments of tau protein. Neurochemically there are deficits in neurotransmitters including acetylcholine, noradrenaline, serotonin and somatostatin.

Specific mutations on chromosomes 21, 14 and 1 inherited as a familial autosomal dominant trait with full penetrance are found in less than 1-5% of all AD patients. Here the illness starts in the late 40's or early 50's and is essentially "pre-senile" in onset (i.e., before the age of 65 years). In the remaining 95% of cases the exact role played by the apolipoprotein genes on
# MINI-MENTAL STATUS EXAMINATION

<table>
<thead>
<tr>
<th>1. What is the:</th>
<th>Year?</th>
<th>Attention &amp; concentration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Season?</td>
<td></td>
<td>Memory</td>
</tr>
<tr>
<td>Month?</td>
<td></td>
<td>Speech</td>
</tr>
<tr>
<td>Day?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Date?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Where are we:</td>
<td>Country?</td>
<td>Working memory</td>
</tr>
<tr>
<td></td>
<td>Province?</td>
<td>Attention &amp; concentration</td>
</tr>
<tr>
<td></td>
<td>City?</td>
<td>Speech</td>
</tr>
<tr>
<td></td>
<td>Hospital?</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ward or Floor?</td>
<td></td>
</tr>
<tr>
<td>3. I am going to name 3 items. (One second each). The words must have no semantic or phonetic (same sound) connection. Repeat them please. Give one point for each correct answer. After this repeat the words until the patient can repeat all 3 (stop if unable to learn them after 6 attempts).</td>
<td></td>
<td>Attention &amp; concentration</td>
</tr>
<tr>
<td>4. Do serial 7’s and the backwards spelling of the 5-letter word. The final score is the higher score of the two tests. Use “World” or “Herfs” (for Afrikaans-speaking patients).</td>
<td></td>
<td>Attention &amp; concentration</td>
</tr>
<tr>
<td>5. Ask the patient to repeat the words from question 3. Give one point for each correct answer.</td>
<td></td>
<td>Memory</td>
</tr>
<tr>
<td>6. Point at a pencil and a watch. The patient must name them.</td>
<td></td>
<td>Speech: Nominal dysphasia</td>
</tr>
<tr>
<td>7. The patient must repeat after you. “No ifs, ands or buts” or “Nog vis, nog vlees, nog voël” (for Afrikaans-speaking patients).</td>
<td></td>
<td>Speech: conducting dysphasia</td>
</tr>
<tr>
<td>8. The patient must perform a 3-stage task: “Take this piece of paper with your right hand. Fold it in half with both hands. Place it on the floor”.</td>
<td></td>
<td>Speech: sensory dysphasia</td>
</tr>
<tr>
<td>9. The patient must read the following sentence and execute the instruction so that you can see that s/he understands it: “CLOSE YOUR EYES”. (Write in capital letters). For Afrikaans-speaking patients “MAAK TOE JOU OË”.</td>
<td></td>
<td>Speech: Dyslexia</td>
</tr>
<tr>
<td>10. The patient must write a sentence of his/her own. (It must make sense, spelling mistakes can be ignored).</td>
<td></td>
<td>Speech: Dysgraphia</td>
</tr>
<tr>
<td>11. The patient must copy the diagram below. (Give one point if the corners and sides are drawn correctly and if the sides cross in the shape of a diamond).</td>
<td></td>
<td>Input: Agnosia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Execution: Apraxia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Constructional ability</td>
</tr>
</tbody>
</table>
## MINI-MENTAL STATUS EXAMINATION

<table>
<thead>
<tr>
<th>PATIENT NAME:</th>
<th>DATE:</th>
<th>SCORE ACHIEVED</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. What is the:</td>
<td>Year?</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Season?</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Month?</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Day?</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Date?</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>2. Where are we:</td>
<td>Country?</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Province?</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>City?</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hospital?</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ward or Floor?</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>3. I am going to name 3 items. (One second each). The words must have no semantic or phonetic (same sound) connection. Repeat them please. Give one point for each correct answer. After this repeat the words until the patient can repeat all 3 (stop if unable to learn them after 6 attempts).</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Do serial 7's and the backwards spelling of the 5-letter word. The final score is the higher score of the two tests. Use &quot;World&quot; or &quot;Herfs&quot; (for Afrikaans-speaking patients).</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Ask the patient to repeat the words from question 3. Give one point for each correct answer.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Point at a pencil and a watch. The patient must name them.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. The patient must repeat after you. &quot;No ifs, ands or buts&quot; or &quot;Nog vis, nog vlees, nog voël&quot; (for Afrikaans-speaking patients).</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. The patient must perform a 3-stage task: &quot;Take this piece of paper with your right hand. Fold it in half with both hands. Place it on the floor&quot;.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. The patient must read the following sentence and execute the instruction so that you can see that s/he understands it: &quot;CLOSE YOUR EYES&quot;. (Write in capital letters). For Afrikaans-speaking patients &quot;MAAK TOE JOU OË&quot;.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. The patient must write a sentence of his/her own. (It must make sense, spelling mistakes can be ignored).</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. The patient must copy the diagram below. (Give one point if the corners and sides are drawn correctly and if the sides cross in the shape of a diamond).</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
chromosome 19 in late-onset familial and sporadic patients still has to be determined as well as the other genetic influences and environmental factors. Recently a mutation on chromosome 10 has become increasingly important in late-onset AD.

2. Vascular dementia

Among the vascular dementias, multi-infarct dementia associated with multiple areas of cortical infarction, patchy cognitive impairment, focal neurological signs and a "stepwise" rather than a steady continuous deterioration as in Alzheimer's disease is more easily diagnosed than dementia due to vascular damage of the deep white matter (Figure 2). After each shower of "mini-strokes" producing a sudden deterioration in the individual's functioning, there is a partial recovery which stabilises within approximately 3 - 12 weeks, until the next stroke or "step" occurs several weeks or months later (Figure 2). In both vascular and alcohol-induced dementias temporary arterial spasms may result in intermittent or fluctuating intellectual and personality changes with unpredictable bouts of irritability and mood swings. In both types of vascular dementias the risk factors for stroke such as hypertension, arrhythmias, hypercholesterolaemia, diabetes, smoking and alcohol are involved.

3. Alcohol-induced dementia

Paramount are deep white matter changes blurred with alcohol-induced vasculopathy clinically indistinguishable from vascular dementia and with the same risk factors as precipitating and perpetuating causes. Note that both vascular and alcohol-induced dementia patients have relatively well-preserved personalities compared to the degree of dementia present. Their excellent social skills or verbal ability may be misleading unless one tests for the presence of dementia using the MMSE.

4. Huntington's disease

Huntington's disease is an inherited disease (autosomal dominant gene on chromosome 4) characterised by degeneration of the basal ganglia and cerebral cortex. Age of onset is between 35 and 50 years when choreaform movements and progressive dementia are noted. The dementia initially shows features of a sub-cortical dementia before affecting the cortex as the illness progresses. No treatment is available and death results in 15-20 years. Psychiatric disorders especially depression may be the presenting features.

5. Parkinson's disease

The primary features of this disease are tremor, muscular rigidity, hypokinesia and postural
abnormality. Though a movement disorder, cognitive impairment occurs in 10-40% of patients during the course of the disease. Impairment of memory and executive functions can occur in approximately 10% of patients.

6. Dementia with Lewy bodies (DLB)

Dementia with Lewy bodies usually exhibits fluctuations in cognition (mimicking multi-infarct dementia) with pronounced variations in attention and alertness, recurrent well-formed visual hallucinations (especially sun-downing) and motor features of Parkinsonism. The course of the illness tends to be rapidly progressive, interspersed with repeated falls, syncope, transient loss of consciousness, hallucinations in other modalities and congruent delusions. There is sensitivity to neuroleptic side-effects (requiring utilisation of drugs like clozapine or the newer neuroleptic agents) and a possibility that these patients may be particularly responsive to cholinesterase inhibitors.

7. Pick’s disease and fronto-temporal dementia

Pick’s disease is a progressive dementia that chiefly affects the frontal cortex. The disorder most commonly manifests itself between the ages of 50-60 years and is distinguished from frontotemporal dementia (FTD) by the presence of characteristic intraneuronal argentophilic Pick inclusion bodies found at autopsy. It presents with prominent personality changes and impaired executive function. In frontotemporal dementia diagnostic criteria range across four domains; 1. Behavioural disorder: Insidious onset and slow progression; early loss of personal and social awareness; early signs of disinhibition; mental rigidity and inflexibility; and hyperorality, stereotyped and perseverative behaviour. 2. Affective symptoms: Depression and anxiety; somatic preoccupation; emotional unconcern. 3. Speech disorder: Reduction and stereotypy of speech; echolalia and perseveration. 4. Physical signs: Early primitive reflexes and incontinence; late akinesia, rigidity and tremor.

8. Creutzfeldt-Jakob’s disease (CJD)

CJD is brought about by a virus-like infective agent called a prion. It causes a rapid progressive dementia also affecting the pyramidal and extrapyramidal systems. A new variant of CJD described in England in 1995 appears to express itself under certain conditions in individuals under the age of 40 years, leading to their death within a year. This variant is associated with bovine spongiform encephalopathy (BSE) or “Mad Cow Disease”.

9. Dementia associated with normal pressure hydrocephalus

Normal pressure hydrocephalus occurs in elderly and is characterised by a triad of ataxia (wide-based gait), urinary incontinence and dementia. CT scans of the brain show prominent enlargement of the ventricles out of keeping with the widening of the sulci. Ventricular peritoneal shunts may improve cognitive functions in 10-30% of patients. Note, usually a demented person only becomes incontinent on attaining a MMSE score of 8-10/30.

10. Dementia secondary to head injury

11. AIDS dementia complex/HIV dementia

HIV infection currently affects 5 million people in South Africa and is set to reach a steady state of 32% within less than a decade. The course of the illness may vary considerably but in general the patient converts to AIDS after 9 years of illness and dies a year later from systemic complications. Nearly 90% of AIDS brains are histopathologically abnormal, more than half of them uniquely due to HIV infection. Though referred to as the AIDS dementia complex it contributes significantly to the morbidity of HIV patients, causing varying degrees of cognitive, motor or behavioural impairment.

<table>
<thead>
<tr>
<th>Table 2 Clinical features of aids dementia complex/ HIV dementia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cognition: memory, concentration, word-finding and processing difficulties</td>
</tr>
<tr>
<td>Motor: fine hand tremor, slowness and clumsiness, affecting handwriting, loss of balance</td>
</tr>
<tr>
<td>Behaviour: loss of spontaneity and social withdrawal, apathy and socially inappropriate behaviour, acute mania or psychosis</td>
</tr>
</tbody>
</table>

Management

Management of dementia involves a quadrangle, viz. doctor- patient- caregiver- community. Take the time to fully discuss the illness with the caregiver and patient. Not only is this therapeutic but it also prevents doctor-shopping. Relatives may blame the dementia on a non-causal incident such as; a severe flu, a mugging or motor vehicle accident (without head injury), a son or daughter leaving home, or some other emotional or financial stressor. Attempt to establish that the onset of dementia occurred prior to the event and that the latter incident may at most have acted as a possible...
contributing factor in exposing the underlying illness. Be sympathetic but firm in handling a stubborn patient who is no longer coping and requires relocation to premises offering more supervision and care. Be prepared to act the role of “bad wolf” by firmly recommending admission to an old age home when it is necessary, since families are more often than not both guilt-ridden and intimidated by this decision. Discuss the genetics of the illness. Treatment of dementia is aimed at reversible factors and symptom alleviation.

Course: Explain to caregivers that dementia is a progressive disease ultimately leading to death, commonly within eight to twelve years. Death is usually from a stroke or myocardial infarct in vascular and alcohol-induced dementias, while bronchopneumonia and urinary tract infections, giving rise to septicaemia, are the common causes of death in AD and other dementias. Note that the patient in a “persistent vegetative state” does not appreciate the sensation of hunger or thirst. For this reason nasogastric tubes in these terminal patients are not recommended. Involve and discuss with family in all cases.

Genetics: If there is a history of AD in the family the illness is said to be “familial” as opposed to “sporadic” (where it occurs out of the blue). The term “familial” is in part misleading in that AD is becoming more common with the increase in the number of elderly and advancing age. To date, autosomal dominant inheritance with full penetrance AD (e.g., chromosomes 21, 14 and 1) affects less than 1 - 5% of all cases of AD, and here invariably the family members know of it and usually develop the illness by their early fifties. The genetics of late-onset AD (e.g., apolipoprotein gene on 19) occurring in the 70’s and 80’s as well as the influence of environmental factors still has to be clarified. Because results are inconclusive, blood tests for genetic testing are not recommended.

Driving and firearms: Driving a car relies on implicit memory, praxis and executive functioning. In the early phases of the illness patients can usually still drive a car because these abilities are still relatively intact. With time however, they are unable to pay attention to all aspects of driving, become impulsive and exercise the wrong options. Note that if the dementia renders the person incapable of safe driving and controlling a vehicle, he/she is medically disqualified from driving. Similarly, gun licences should be revoked for the same reason.

Financial affairs and Wills: Should the patient be incapable of handling her/his own financial affairs, urge a reliable and trustworthy member of the family to take control of the situation. Transfer of authority by means of Power of Attorney forms work well in “early” dementia where competency is still preserved. Failing this, or in situations where disputes are likely, or where no family members are available, curatorship should be sought. The forms are obtainable from the court who will appoint a curator bonis to attend to the patient’s affairs, through a curator ad litem. Social workers who are well-versed in these matters may have to be called in for advice and help with these cases. Note that if the patient has not yet written a Will or Testament and now wishes to do so, referral to a psychiatrist is necessary to establish testamentary capacity.

Non-governmental organisations: All families with a member suffering from dementia must be referred to support organisations such as Alzheimer’s South Africa. These are available nation wide and extremely active in psycho-education, advice and counselling, support of the family and research.

Non-pharmacological treatment: This should be implemented prior to attempting drug treatment and involves:

(i) the psycho-education, support – and if necessary, treatment – of the carer, as well as arranging periods of respite.

(ii) the assessment of the patient’s environment with particular emphasis on optimising orientation and handling by caregivers. Establish a safe and familiar routine, restricting the patient to a maximum of 20 minutes afternoon nap. Remove precipitating factors and restrict the area of wanderers. Avoid restraints if possible.

(iii) implementing validation/affirmative therapy, reminiscence therapy and 24-hour reality orientation with the patient. Diversion tactics are very important; keep the patient occupied. It thus involves assessing the patient’s environment with particular emphasis on factors concerning their

Figure 3 Course of dementia (with memory enhancer)
orientation and handling by caregivers; as well as attending to the patient’s sensory modalities such as sight and hearing, taking into account the patient’s diminishing cognitive abilities.

Pharmacological treatment is indicated when non-pharmacological intervention has failed. The choice of drug is individual, based on experience, contraindications, tolerability, availability and especially cost of the drug. Under ideal conditions the following regimens should be implemented (Table 3 & 4, Figure 3):

### Table 3 Pharmacological management of Alzheimer’s disease

1. **Vitamin E - 1000iu 2x daily**
2. **Acetylcholinesterase inhibitors (AChEIs) - donepezil (Aricept®) 5-10mg at night, rivastigmine (Exelon®) 3-6mg twice daily and galantamine (Reminyl®) 8-12mg twice daily.**
3. **Psychotropic agents for residual symptoms i.e., mood and behavioural disturbances (depression, restlessness, agitation, psychotic symptoms, insomnia).**

Three acetylcholinesterase inhibitors are currently available for AD: donepezil (Aricept®), rivastigmine (Exelon®) and galantamine (Reminyl®). These drugs are acetylcholine esterase inhibitors resulting in increased acetylcholine levels in the brain. They improve memory, behaviour and function initially above baseline for approximately the first nine months, where after the deterioration of the disease appears to be slowed down by roughly half for the remainder of its course. In general terms patients lose 2-3 points on MMSE per year. On treatment they can expect to have lost this amount after 2 years. Patients need to adapt to the drugs, thus slow titration is recommended to the maximum dose tolerated. The higher the latter the better the results. Use the maxim “start low, go slow, end high”. Increase donepezil from 5mg – 10mg after 8 weeks, and rivastigmine in increments of 1.5mg twice daily, every 4 weeks, to a total of 6mg – 12mg per day. Galantamine is similarly titrated upwards in twice daily dosages of 4mg, 8 mg and 12mg to acquire an effective dose of 16mg – 24mg per day. Side-effects are usually transitory and include nausea, vomiting, bouts of diarrhoea, gastric discomfort, sedation, agitation, sweating, dizziness and headache.

### Table 4 Treatment schedule for restlessness, psychotic symptoms, agitation and insomnia

<table>
<thead>
<tr>
<th>Drug</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Haloperidol</strong> (Serenace®)</td>
<td>* 0.5mg twice daily</td>
</tr>
<tr>
<td></td>
<td>* Increase the dose to 0.75mg, 1.0mg and 1.5mg twice daily if necessary, for daytime control. Wait a day or two between increases.</td>
</tr>
</tbody>
</table>

Together with thioridazine (Melleril®) – depending on QTc interval (ECG)

- 25mg at night
- Increase the dose to 50mg, 75mg and 100mg at night, if necessary, for nocturnal control. Wait a day or two between increases.

Where budgetary constraints apply, the combination of haloperidol and thioridazine empirically yield the best response in cases of persistent agitation or psychosis. Though a degree of caution is indicated in the use of the latter drug because of prolongation of the QTc interval (i.e., >450 msec).

Start with the lowest possible dose of neuroleptics. Other agents that can be substituted for the above are: zuclopenthixol (Clopixol®) 1mg twice daily; olanzapine (Zyprexa®) 2.5mg at 17h00; risperidone (Risperdal®) 0.5mg twice daily; quetiapine (Seroquel®) 25mg in the mornings and 50mg at night; and ziprasidone (Geodon®) 20mg twice daily.

Occasionally lorazepam (Ativan®) 2.4mg, intramuscular chlorpromazine (Largactil®) 25-50mg; haloperidol (Serenace®) 2.5 mg; zuclopenthixol acetate (Clopixol Acuphase®) 50-100mg or olanzapine (Zyprexa®) may initially be required for control. Alternatively, the oral solution of risperidone (Risperdal®) 0.25mg should be administered.

Depot agents are indicated in most paranoid and non-compliant patients and comprise fluphenazine decanoate (Modicate®) 12.5 – 25mg/4 wks and zuclopenthixol decanoate (Clopixol Depot®) 200-400mg/4 wks.

The inherent symptoms of apathy and social withdrawal mimic depression in the AD patient, making it difficult to distinguish between the two. In addition, depression frequently accompanies some 20% of AD patients in the early stages. Generally the diagnosis of depression is clinically relevant in the higher ranges of the MMSE, while the benefit of doubt should swing towards the patient being socially withdrawn and not depressed as one approaches a MMSE of 20-22/30.

Prevention: To date the following agents have been found to either delay the onset of AD or slow down its course: Apolipoprotein E 2 or 3, hormonal replacement therapy (instituted perimenopausally when indicated), anti-inflammatories (low-dose), vitamin E (1000iu once daily), vitamin C (600-900mg daily), red wine (approximately 250-500 ml per day because of ingredients such as resveratrol), and intellectual stimulation and higher education (improves the number of synapses). Most important is the control of vascular factors as these are closely linked as risk factors to the pathophysiology of AD. These factors comprise the management of transient ischaemic attacks (TIAs) and vascular dementia (Table 5):
Table 5 Management of vascular dementia

<table>
<thead>
<tr>
<th>Treatment of risk factors:</th>
</tr>
</thead>
<tbody>
<tr>
<td>* hypertension - taking raised age-acceptable blood pressures into account.</td>
</tr>
<tr>
<td>* diabetes - noting that in asymptomatic diabetics random blood-glucose levels up to 15mmol/l are acceptable.</td>
</tr>
<tr>
<td>* smoking - should be stopped.</td>
</tr>
<tr>
<td>* alcohol - reduce consumption to an equivalent maximum of three tots of spirits a day.</td>
</tr>
<tr>
<td>* prevent platelet aggregation - with Disprin® 80-150mg per day.</td>
</tr>
<tr>
<td>* hyperlipidaemia - control via diet and statins</td>
</tr>
<tr>
<td>* body mass index – dietary modification and regular exercise</td>
</tr>
<tr>
<td>* endothelial stress and inflammation – thiamine supplementation</td>
</tr>
<tr>
<td>* hyperhomocysteinaemia – folic acid and vitamins B6 and B12 supplementation</td>
</tr>
<tr>
<td>* psychotropic medication – may be indicated for behavioural problems or mood disorders</td>
</tr>
</tbody>
</table>

In conclusion, dementia is often far more catastrophic for the relatives of an affected individual than for the patient and ongoing support for those who provide the care is essential in order to maintain a patient in the community.

References


SECTION III

DELIRIUM

Definition

The key feature of delirium is a disturbance of consciousness accompanied by a change in cognition. Delirium is an acute, fluctuating, temporary disorder resulting in: reduced awareness of one’s surroundings and an inability to attend to any given task or conversation; as well as disorientation and memory impairment. In addition, at least two of the following symptoms are present; perceptual disturbances (misinterpretations and hallucinations), delusions (usually paranoid in nature), incoherent speech, disturbance of sleep-wakefulness cycle, and a change in energy levels or motor activity.1,2,3

The disturbance of consciousness (reduced clarity of awareness of the environment) results in the person being oblivious of their surroundings, imagining that they are, for example, at home instead of at the hospital. The attention deficit (reduced ability to focus, sustain or shift attention) renders them unable to attend to any given task or conversation. The change in cognition (memory deficit, disorientation, language disturbance) may mimic dementia as may the development of perceptual disturbances (misinterpretations and hallucinations - usually visual) and delusions (poorly systematised), but these tend to be more fragmented in delirium. Delirium is also known as acute confusion, acute brain syndrome, metabolic encephalopathy, toxic psychosis and acute brain failure. DSM-IV in order to avoid confusion uses the term “delirium”.

Delirium constitutes a medical emergency since the illness causing it may be life-threatening.1,2,3
Table 1 Core criteria for delirium: DSM-IV

Delirium is distinguished by a disturbance of consciousness. It may be caused by a general medical condition, a substance, and/or multiples of both.

1. Disturbance of consciousness and attention deficit
2. A change in cognition and/or perceptual disturbance
3. Short period of onset and fluctuating course
4. History, physical examination or laboratory findings indicate the physiological consequence of a substance or general medical condition as the cause

Epidemiology

Cases of delirium increase in direct proportion to the increase in number of elderly in the population. Of all age groups the elderly, and especially the demented, are uniquely prone to delirium. Some 10 to 15% of elderly general surgical patients become delirious after operation, and between 30 to 50% of all elderly admissions are likely to be delirious at some point during their stay in hospital. The death rate for delirium varies from 10 to 30%, and is as high as 50% within the year that the patient has had delirium. The presence of delirium is a medical emergency, no matter what the patient’s age.

Predisposing factors

Predisposing factors for the development of delirium consist of the stressors that accompany ageing, extremes of age (the elderly and children), previous brain insults (head injuries, stroke, Parkinson’s disease), a history of delirium, alcohol dependence, diabetes, cancer, sensory impairment (poor vision or hearing) and malnutrition.

Aetiology

Organic aetiology is identifiable in more than 95% of cases, while the non-organic causes (pseudo-delirium) usually blend in with the latter. The aetiology is typically multi-factorial in origin, involving organic factors, psychosocial stressors, unfamiliar environments, and excessive or diminished sensory input (e.g., from reduced visual acuity and hearing). The causes involved in post-operative delirium include the stress of surgery, post-operative pain, foreign setting, insomnia, medication, metabolic disturbance, infection, fever and blood loss.

A useful checklist for diagnosing the causes of delirium is provided by the mnemonic DIMTOP:

D - Drugs
I - Infection
M - Metabolic
T - Trauma
O - Oxygen deficit
P - Psychological, perceptual and postictal

Of these the commonest causes are:

Drugs: It is generally conceded that iatrogenics are responsible for up to 20% of cases of delirium in the elderly

1. Sedatives and tranquillisers – benzodiazepines
2. Antidepressants - less commonly the anticholinergic effects of tricyclic antidepressants
3. Oral hypoglycaemic agents (note that in asymptomatic diabetics random blood glucose levels of up to 15mmol/l are acceptable)
4. Steroids
5. L-dopa
6. Antihypertensives and digoxin (note that in the elderly mildly higher blood pressure readings are acceptable)
7. Alcohol – “delirium tremens” usually occurs on the second to third day after cessation or a marked reduction in drinking
8. Heavy metal and other toxins

Infection: Anywhere in the body, but commonly the cause is a urinary tract infection or bronchopneumonia. Acute psychotically ill elderly are frequently dehydrated predisposing to urinary tract infections.

Metabolic: Uraemia, hypo- and hyperglycaemia. Also dehydration due to inadequate fluid intake, diuretics, laxatives or diarrhoea.

Trauma: Both psychological and organic (posttraumatic stress, concussion or subdural haematoma in origin). Head injuries with diffuse axonal degeneration. Post-operative complications.

Oxygen deficit: As occurs in cardiovascular and respiratory disorders, especially strokes.

Psychological, perceptual and postictal: Anything lessening the person’s contact with familiar surroundings or visual cues can cause confusion (reality testing), for example, moving to a new room, an emotional shock, dim lights and the loss of glasses or a hearing aid. Postictal confusion presents as delirium and requires medical attention.

Pathophysiology of delirium

Acetylcholine is the principal neurotransmitter of the reticular formation, which is the key area involved in...
delirium. A shortage of acetylcholine in the brain is associated with delirium. It thus follows that drugs that reduce the action of acetylcholine may precipitate or worsen delirium. These drugs (usually in higher dosages) are antidepressants, antipsychotics (chlorpromazine) and anticholinergic agents (biperiden and orphenadrine). The reticular formation is the most important brain system regulating consciousness and includes the dorsal segmental pathways.

**Diagnostic assessment**

**History**

In order to diagnose delirium, good collateral information is vital, which means obtaining a history of nocturnal behaviour as well as that seen during the day. As a rough guideline while nocturnal and daytime restlessness is present in both delirious and demented patients - nocturnal restlessness is more pronounced in delirium, while daytime restlessness is more pronounced in dementia. Thus your most reliable informants are roommates, night nurses and domestics doing night shift rather than senior staff on call during the day.

**Clinical features**

Note that delirium can be envisaged as a “symptom” of an underlying disorder. It is a very sensitive indicator of a disease in progress and is usually present before fever, pain or tachycardia, that is, delirium can precede any sign of an acute illness by up to two days. The clinical features usually develop over a short period of time (hours to days) and tend to fluctuate during the course of the day or night. The usual history is that of an elderly person suddenly becoming increasingly psychotic and unmanageable, especially during the night, while being quiet and drowsy during the day (reversal of sleep-wake cycle). The patient may then become increasingly restless in the late afternoon, referred to as “sun downing”. In more subtle cases the relatives will claim that the patient is just not him/herself, or the staff may report that the patient has become a “little odd”. A sudden and recent onset of incontinence may be the only sign. A sudden decompensation in function and change in psychomotor activity are good indicators of delirium. In the latter the patient is either hypoactive, stuporous and apathetic or hyperactive and restless; though a mixed picture may often be present. Restlessness occurs especially during substance withdrawal when it is also associated with autonomic hyperactivity such as tachycardia, flushing, tremor, perspiration, pupillary dilatation, pallor, nausea, vomiting and hyperthermia.

A key feature of delirium is a shortened attention span and concentration deficit that fluctuates markedly. A degree of lack of awareness of surroundings (clouding of consciousness) occurs in all patients with delirium and expresses itself as impaired orientation to time and place. Speech disturbance may consist of aphasia, fragmented phrases or incoherence. Visual hallucinations may include coloured patterns, visions of insects, people or scenery. Emotional changes consist of fear, anger, anxiety, depression and euphoria. Once the cause is found and correctly treated the response is rapid and the patient will usually be well within a week (and at most 4 weeks), unlike the demented patient whose intellectual and behavioural deficits will persist. Once a patient has recovered, his recall of events during the delirious episode is usually patchy.

**Principles of assessment**

Refer to the section dealing with: Examinations And Investigations.

The assessment of delirium is made at the bedside and relies on clinical skills.

**Mini-Mental Status Examination (MMSE):** Characteristically one needs to rally the patient to do the test, which frequently cannot be completed. This is due to the patient’s lack of attention and ease of distractibility. Use the MMSE to monitor response to treatment.

**Differential diagnosis**

1. **Dementia**

The key features that distinguish a delirium from a dementia is the acute onset, fluctuating, reversible course of delirium coupled with disturbed consciousness, attention deficit, reversal of sleep-wakefulness cycle and altered motor activity.

2. **Schizophrenia**

Some schizophrenic patients demonstrate severe disordered behaviour which may be confused with the hyperactivity found in delirium. These patients being floridly psychotic will also have impaired attention and concentration spans.

3. **Depression**

Patients who are severely depressed with psychomotor retardation need to be distinguished from hypoactive patients suffering from delirium.

4. **Factitious disorder and malingering**

These patients demonstrate contradictions in symptoms and signs.
Table 2 Clinical differentiation of delirium and dementia

<table>
<thead>
<tr>
<th>Criterion</th>
<th>Dementia</th>
<th>Delirium</th>
</tr>
</thead>
<tbody>
<tr>
<td>History</td>
<td>Chronic disease</td>
<td>Acute disease</td>
</tr>
<tr>
<td>Onset</td>
<td>Insidious (months to years)</td>
<td>Rapid (hours to days)</td>
</tr>
<tr>
<td>Course</td>
<td>Progressive</td>
<td>Fluctuating</td>
</tr>
<tr>
<td>Duration</td>
<td>Months to years</td>
<td>Days to weeks</td>
</tr>
<tr>
<td>Level of Consciousness</td>
<td>Alert</td>
<td>Clouded, fluctuating</td>
</tr>
<tr>
<td>Orientation</td>
<td>Initially intact - later impaired</td>
<td>Impaired</td>
</tr>
<tr>
<td>Attention/Concentration</td>
<td>Initially intact - later impaired</td>
<td>Prominently impaired</td>
</tr>
<tr>
<td>Sleep</td>
<td>Occasional confusion at night</td>
<td>Disrupted sleep/wake cycle (often reversed)</td>
</tr>
<tr>
<td>Restlessness</td>
<td>Worse in the day</td>
<td>Worse in the night</td>
</tr>
<tr>
<td>Psychomotor</td>
<td>Initially unchanged - later impaired</td>
<td>Agitated, retarded or mixed</td>
</tr>
<tr>
<td>Executive function</td>
<td>Relatively preserved</td>
<td>Relatively preserved</td>
</tr>
<tr>
<td>Affect</td>
<td>Various but stable - bouts of irritability</td>
<td>Anxious, agitated - labile</td>
</tr>
<tr>
<td>Thinking</td>
<td>Decreased in amount</td>
<td>Disordered</td>
</tr>
<tr>
<td>Memory</td>
<td>Impaired, in keeping with stage of dementia</td>
<td>Markedly impaired, out of keeping</td>
</tr>
<tr>
<td>Perception</td>
<td>Hallucinations less common (except sundowning)</td>
<td>Hallucinations common (especially visual)</td>
</tr>
<tr>
<td>Reversibility</td>
<td>Not reversible</td>
<td>Reversible</td>
</tr>
<tr>
<td>Prognosis</td>
<td>Poor</td>
<td>Relatively good</td>
</tr>
</tbody>
</table>

Management

Find and treat the causative factor. Do a full mental and physical examination. In the elderly, among the above-mentioned multi-factorial causes of delirium, this often turns out to be a urinary tract infection. Because of the sensitivity of delirium as a sign of the underlying disorder, the initial one or two mid-stream urinary specimens for testing may be normal, only to be followed by a positive result within a further day or two. Patients in this age group tend not to report dysuria, frequency of micturition or pelvic pain and the only clue may be the sudden occurrence of incontinence or smelly urine. A urine dipstick test that includes testing for nitrates and leucocytes is helpful in these cases. Immediately start treatment with an appropriate antibiotic. The nocturnal upheaval to family, roommates or ward staff caused by an untreated patient is not justified. Cotrimoxazole (Bactrim®) is readily available and is administered in a dosage of 2 tablets twice daily for 10 days to combat the often- accompanying pyelonephritis. Alternatively, ciprofloxacin (Ciprobay®) 500mg stat, or 500mg daily over 3 days will resolve most urinary tract infections. With repeated infections, laboratory results obtained from microscopy, culture and sensitivity may serve as a further guide to treatment.

If further investigations are necessary, they usually follow along the lines of the "organic" work-up or refer to a secondary (medical) care centre.

Restore the normal nutritional and metabolic state. Enforce the day/night cycle rhythm.

Attend to the surroundings by reducing noise levels and providing some lighting at night.

Sedation consists of the implementation of the treatment schedule for restlessness, psychotic symptoms, agitation and insomnia for dementia, though higher doses may initially be necessary. A sharp cutback in this medication will then be required once the cause of the delirium is brought under control. In severe cases the following regimen may initially be necessary (Table 3):

<table>
<thead>
<tr>
<th>Drugs used in severe delirium</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haloperidol (Serenace®)</td>
</tr>
<tr>
<td>Lorazepam (Ativan®)</td>
</tr>
</tbody>
</table>

Recall of events from the time of the delirium is characteristically patchy. Once the delirium has settled and the patient recovered, detailed investigation for possible underlying dementia is advisable in order to pre-empt future potential problems.

Summary: Steps in evaluating delirium

1. Collateral history
2. Psychiatric examination
3. MMSE – if possible
4. Physical examination
5. Differential diagnosis
6. Investigations e.g., urine dipstick
7. Treat cause
8. Establish homeostasis
9. Follow-up
References


Recommended reading


CATATONIA

Catatonic disorder due to a general medical condition

A small percentage of all admissions present with catatonia; a non-specific syndrome of multiple aetiologies. It may be secondary to substance abuse (alcohol, cocaine), iatrogenic (neuroleptic induced extrapyramidal syndromes and neuroleptic malignant syndrome – NMS) or psychiatric (mood disorders, schizophrenia, dementias).

The DSM-IV criteria for catatonia are the presence of two or more of the following:

1. motor immobility (catalepsy, stupor)
2. excessive motor activity
3. extreme negativism or mutism
4. peculiarities of voluntary movement (posturing, stereotypies, mannerisms, or grimacing)
5. echolalia or echopraxia

Patients are usually immobile, mute, withdrawn, refusing to eat or drink, and staring; but on responding to treatment may later describe having experienced intense anxiety and fear usually related to psychotic ideation.

Between 70-80% of catatonics will respond dramatically within 1-3 hours to low dose benzodiazepines, ideally lorazepam (Ativan®) 1-2mg given intramuscularly or intravenously (least preferred orally). Occasionally a second dose is required as well as maintenance medication of carbamazepine (Tegretol®) 400-600mg yielding variable results.

References


Recommended reading