Invited Review

Causes and Progression of Dementia

Alzheimer's Study Group

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ABSTRACT: Senile Dementia is a form of general intellectual impairment observed in elderly people. Approximately 10 percent of all people more than 65 years of age have clinically important intellectual impairment. Although about 20 percent of these cases may be due to treatable causes such as toxic drug reactions, most cases are what is known as Alzheimer's disease. This condition begins with failing attention and memory, loss of mathematical ability, irritability and loss of sense of humour, and poor orientation in space and time. Alzheimer's disease is relentlessly progressive and leads to death in 5 to 15 years. Examinations of the brains of persons who have died of Alzheimer's disease show characteristic twisted fibers, called neurofibrillary tangles, in certain areas of the brain, and cores of abnormal protein, called neuritic plaques, interspersed among nerve cells. No cure is known for Alzheimer's disease. A form of this disease strikes younger persons, in whom it is known as presenile dementia.

Introduction

Dementia (a word taken from Latin, originally meaning "madness", from *de-* "without" + *ment*, the root of *mens* "mind") is a serious loss of cognitive ability in a previously unimpaired person, beyond what might be expected from normal aging. It may be static, the result of a unique global brain injury, or progressive, resulting in long-term decline due to damage or disease in the body. Although dementia is far more common in the geriatric population, it can occur before the age of 65, in which case it is termed "early onset dementia".[1]

Dementia is not a single disease, but rather a non-specific illness syndrome (i.e., set of signs and symptoms) in which affected areas of cognition may be memory, attention, language, and problem solving. It is normally required to be present for at least 6 months to be diagnosed;[2] cognitive dysfunction that has been seen only over shorter times, in particular less than weeks, must be termed delirium. In all types of general cognitive dysfunction, higher mental functions are affected first in the process.

Especially in the later stages of the condition, affected persons may be disoriented in time (not knowing what day of the week, day of the month, or even what year it is), in place (not knowing where they are), and in person (not knowing who they, or others around them, are). Dementia, though often treatable to some degree, is usually due to causes that are progressive and incurable.

Symptoms of dementia can be classified as either reversible or irreversible, depending upon the aetiology of the disease. Less than 10% of cases of dementia are due to causes that may presently be reversed with treatment. Causes include many different specific disease processes, in the same way that symptoms of organ dysfunction such as shortness of breath, jaundice, or pain are attributable to many aetiologies.

Without careful assessment of history, the short-term syndrome of delirium (often lasting days to weeks) can easily be confused with dementia, because they have all symptoms in common, save duration. Some mental illnesses, including depression and psychosis, may produce symptoms that must be differentiated from both delirium and dementia.[3]

There are many specific types (causes) of dementia, often showing slightly different symptoms. However, the symptom overlap is such that it is impossible to diagnose the type of dementia by symptomatology alone, and in only a few cases are symptoms enough to give a high probability of some specific cause. Diagnosis is therefore aided by nuclear medicine brain scanning techniques. Certainty cannot be attained except with brain biopsy during life, or at necropsy in death.

Some of the most common forms of dementia are: Alzheimer's disease, vascular dementia, frontotemporal dementia, semantic dementia and dementia with Lewy bodies. It is possible for a patient to exhibit two or more dementing processes at the same time, as none of the known types of dementia protects against the others.

History

Up to the end of the 19th century, dementia was a much broader clinical concept, which included mental illness and any type of psychosocial incapacity, including those which could be reversed.[4] Dementia at this time simply referred to anyone who had lost the ability to reason, and was applied equally to psychosis of mental illness, "organic" diseases like syphilis which could destroy the brain, and to the dementia associated with old age, which was held to be caused by "hardening of the arteries."

Dementia when seen in the elderly was called **senile dementia** or **senility** and viewed as a normal and somewhat inevitable aspect of growing old, rather than as being caused by any specific diseases. At the same time, in 1907, a specific organic dementing process of early onset, called Alzheimer's disease, had been described. This was associated with particular microscopic changes in the brain, but was seen as a rare disease of middle age.

Much like other diseases associated with aging, dementia was rare before the 20th century, although by no means unknown, due to the fact that people seldom lived long enough to experience it.
By the period of 1913-20, schizophrenia had been well-defined in a way similar to today, and also the term dementia praecox had been used to suggest the development of senile-type dementia at a younger age. Eventually the two terms fused, so that until 1952 physicians used the terms dementia praecox ("precocious dementia") and schizophrenia interchangeably. The term "precocious dementia" for a mental illness suggested that a type of mental illness like schizophrenia (including paranoia and decreased cognitive capacity) could be expected to arrive normally in all persons with greater age. At the same time, the beginning use of dementia to describe both what we know understand as schizophrenia and senile dementia, after about 1920, acted to give the word "dementia" a more limited role, as one of describing a type of permanent mental deterioration which was not expected to be reversible. This is the beginning of the more recognizable use of the term today.

In 1976, neurologist Robert Katzmann suggested a link between "senile dementia" and Alzheimer's disease.[5] Katzmann suggested that much of the senile dementia occurring (by definition) after the age of 65, was pathologically identical with Alzheimer's disease occurring before age 65 and therefore should not be treated differently. He noted that the fact the "senile dementia" was not considered a disease, but rather part of aging, and this fact was keeping millions of aged patients with what otherwise was identical with Alzheimer's disease, from being diagnosed as having a disease process, rather than simply considered as aging normally.[6] Katzmann thus suggested that Alzheimer's disease, if taken to occur over age 65, is actually common, not rare, and was the 4th or 5th leading cause of death, even though rarely being reported on death certificates in 1976.

This suggestion opened the view that dementia is never normal, and must always be the result of a particular disease process, and is not part of the normal healthy aging process, *per se*. The ensuing debate led for a time to the proposed disease diagnosis of "senile dementia of the Alzheimer's type" (SDAT) in persons over the age of 65, with "Alzheimer's disease" diagnosed in persons younger than 65 who had the same pathology. Eventually, however, it was agreed that the age limit was artificial, and that Alzheimer's disease was the appropriate term for persons with the particular brain pathology seen in this disease, regardless of the age of the sufferer. A helpful finding was that although the incidence of Alzheimer's disease increased with age (from 5-10% of 75 year olds to as many as 40-50%

of 90 year olds), there was no age at which all persons developed it, so it was not an inevitable consequence of aging, no matter how great an age a person attained.

Also, after 1952, mental illnesses like schizophrenia were removed from the category of "organic brain syndromes," and thus (by definition) removed from possible causes of "dementing illnesses" (dementias). At the same, however, the traditional cause of senile dementia – "hardening of the arteries" – now returned as a set of dementias of vascular cause (small strokes). These were now termed "multi-infarct dementias" or vascular dementias.

In the 21st century, a number of other types of dementia have been differentiated from Alzheimer's disease and vascular dementias (these two being the most common types). This differentiation is on the basis of pathological examination of brain tissues, symptomatology, and by different patterns of brain metabolic activity in nuclear medical imaging tests such as SPECT and PET scans of the brain. The various forms of dementia have differing prognoses (cause of illness), and also differing sets of epidemologic risk factors. The causal etiology of many of them, including Alzheimer's disease, remains unknown, although many theories exist such as accumulation of protein plaques as part of normal aging, inflammation, inadequate blood sugar, and traumatic brain injury.

Signs, Symptoms and Comorbidities

Dementia is not merely a problem of memory. It reduces the ability to learn, reason, retain or recall past experience and there is also loss of patterns of thoughts, feelings and activities (Gelder *et al.* 2005). Additional mental and behavioural problems often affect people who have dementia, and may influence quality of life, caregivers, and the need for institutionalization. As dementia worsens individuals may neglect themselves and may become disinhibited and may become incontinent. (Gelder *et al.* 2005).

Depression affects 20–30% of people who have dementia, and about 20% have anxiety.[7] Psychosis (often delusions of persecution) and agitation/aggression also often accompany dementia. Each of these needs to be assessed and treated independent of the underlying dementia.[8]

The Canadian Medical Association Journal has reported that driving with dementia could lead to severe injury or even death to self and others. Doctors should advise appropriate testing on when to quit driving.[9]

In the United States, Florida's Baker Act allows law enforcement and the judiciary to force mental evaluation for those suspected of suffering from dementia or other mental incapacities.

In the United Kingdom, as with all mental disorders, where a sufferer could potentially be a danger to themselves or others, they can be detained under the Mental Health Act of 1983 for the purposes of assessment, care and treatment. This is a last resort, and usually avoided if the patient has family or friends who can ensure care.

The United Kingdom DVLA (Driving & Vehicle Licensing Agency) states that dementia sufferers who specifically suffer with poor short term memory, disorientation, lack of insight or judgment are almost certainly not fit to drive—and in these instances, the DVLA must be informed so said license can be revoked. They do however acknowledge low-severity cases and early sufferers, and those drivers may be permitted to drive pending medical report.

Behaviour may be disorganized, restless or inappropriate. Some people become restless or wander about by day and sometimes at night. When people suffering from dementia are put in circumstances beyond their abilities, there may be a sudden change to tears or anger (a *"catastrophic reaction"*).[10]

Causes

Fixed cognitive impairment

Various types of brain injury, occurring as a single event, may cause irreversible but fixed cognitive impairment. Traumatic brain injury may cause generalized damage to the white matter of the brain (diffuse axonal injury), or more localized damage (as also may neurosurgery). A temporary reduction in the brain's supply of blood or oxygen may lead to hypoxic-ischemic injury. Strokes (ischemic stroke, or intracerebral, subarachnoid, subdural or extradural haemorrhage) or infections (meningitis and/or encephalitis) affecting the brain, prolonged epileptic seizures and acute hydrocephalus may also have long-term effects on cognition. Excessive alcohol use may cause alcohol dementia, Wernicke's encephalopathy and/or Korsakoff's psychosis, and certain other recreational drugs may cause substance-induced persisting dementia; once overuse ceases, the cognitive impairment is persistent but not progressive.

Slowly progressive dementia

Dementia which begins gradually and worsens progressively over several years is usually caused by neurodegenerative disease; that is, by conditions affecting only or primarily the neurons of the brain and causing gradual but irreversible loss of function of these cells. Less commonly, a non-degenerative condition may have secondary effects on brain cells, which may or may not be reversible if the condition is treated.

The causes of dementia depend on the age at which symptoms begin. In the elderly population (usually defined in this context as over 65 years of age), a large majority of cases of dementia are caused by Alzheimer's disease, vascular dementia or both. Dementia with Lewy bodies is another fairly common cause, which again may occur alongside either or both of the other causes.[11][12][13] Hypothyroidism sometimes causes slowly progressive cognitive impairment as the main symptom, and this may be fully reversible with treatment. Normal pressure hydrocephalus, though relatively rare, is important to recognize since treatment may prevent progression and improve other symptoms of the condition. However, significant cognitive improvement is unusual.

Dementia is much less common under 65 years of age. Alzheimer's disease is still the most frequent cause, but inherited forms of the disease account for a higher proportion of cases in this age group. Frontotemporal lobar degeneration and Huntington's disease account for most of the remaining cases.[14] Vascular dementia also occurs, but this in turn may be due to underlying conditions (including antiphospholipid syndrome, CADASIL, MELAS, homocystinuria, moyamoya and Binswanger's disease). People who receive frequent head trauma, such as boxers or football players, are at risk of chronic traumatic encephalopathy[15] (also called dementia pugilistica in boxers).

In young adults (up to 40 years of age) who were previously of normal intelligence, it is very rare to develop dementia without other features of neurological disease, or without features of disease elsewhere in the body. Most cases of progressive cognitive disturbance in this age group are caused by psychiatric illness, alcohol or other drugs, or metabolic disturbance. However, certain genetic disorders can cause true neurodegenerative dementia at this age. These include familial Alzheimer's disease, SCA17 (dominant inheritance); adrenoleukodystrophy (X-linked); Gaucher's disease type 3, metachromatic leukodystrophy, Niemann-Pick disease type C, pantothenate kinase-associated neurodegeneration, Tay-Sachs disease and Wilson's disease (all recessive). Wilson's disease is particularly important since cognition can improve with treatment.

At all ages, a substantial proportion of patients who complain of memory difficulty or other cognitive symptoms are suffering from depression rather than a neurodegenerative disease. Vitamin deficiencies and chronic infections may also occur at any age; they usually cause other symptoms before dementia occurs, but occasionally mimic degenerative dementia. These include deficiencies of vitamin B_{12}, folate or niacin, and infective causes including cryptococcal meningitis, HIV, Lyme disease, progressive multifocal leukoencephalopathy, subacute sclerosing panencephalitis, syphilis and Whipple's disease.

Rapidly progressive dementia

Creutzfeldt-Jakob disease typically causes a dementia which worsens over weeks to months, being caused by prions. The common causes of slowly progressive dementia also sometimes present with rapid progression: Alzheimer's disease, dementia with Lewy bodies, frontotemporal lobar degeneration (including corticobasal degeneration and progressive supranuclear palsy).

On the other hand, encephalopathy or delirium may develop relatively slowly and resemble dementia. Possible causes include brain infection (viral encephalitis, subacute sclerosing panencephalitis, Whipple's disease) or inflammation (limbic encephalitis, Hashimoto's encephalopathy, cerebral vasculitis); tumors such as lymphoma or glioma; drug toxicity (e.g. anticonvulsant drugs); metabolic causes such as liver failure or kidney failure; and chronic subdural hematoma.

Dementia as a feature of other conditions

There are many other medical and neurological conditions in which dementia only occurs late in the illness, or as a minor feature. For example, a proportion of patients with Parkinson's disease develop dementia, though widely varying figures are quoted for this proportion.[*citation needed*] When dementia occurs in Parkinson's disease, the underlying cause may be dementia with Lewy bodies or Alzheimer's disease, or both.[16] Cognitive impairment also occurs in the Parkinson-plus syndromes of progressive supranuclear palsy and corticobasal degeneration (and the same underlying pathology may cause the clinical syndromes of frontotemporal lobar degeneration). Chronic inflammatory conditions of the brain may affect cognition in the long term, including Behçet's disease, multiple sclerosis, sarcoidosis, Sjögren's syndrome and systemic lupus erythematosus. Although the acute porphyrias may cause episodes of confusion and psychiatric disturbance, dementia is a rare feature of these rare diseases.[17]

Apart from those mentioned above, inherited conditions which may cause dementia alongside other features include:[18]

- Alexander disease
- Canavan disease
- Cerebrotendinous xanthomatosis
- DRPLA
- Fragile X-associated tremor/ataxia syndrome
- Glutaric aciduria type 1
- Krabbe's disease
- Maple syrup urine disease
- Niemann Pick disease type C
- Kufs' disease
- Neuroacanthocytosis
- Organic acidemias
- Pelizaeus-Merzbacher disease
- Urea cycle disorders
- Sanfilippo syndrome type B
- Spinocerebellar ataxia type 2

Diagnosis

Proper differential diagnosis between the types of dementia (cortical and subcortical) will require, at the least, referral to a specialist, e.g., a geriatric internist, geriatric psychiatrist, neurologist, neuropsychologist or geropsychologist. Duration of symptoms must evident for at least six months for a diagnosis of dementia or organic brain syndrome to be made (ICD-10).

Cognitive testing

There exist some brief tests (5–15 minutes) that have reasonable reliability and can be used in the office or other setting to screen cognitive status (Table 1). Examples of such tests include the abbreviated mental test score (AMTS), the mini mental state examination (MMSE), *Modified Mini-Mental State Examination* (3MS),[21] the *Cognitive Abilities Screening Instrument* (CASI),[22] and the clock drawing test.[23] Scores must be interpreted in the context of the person's educational and other background, and the particular circumstances; for example, a person highly depressed or in great pain will not be expected to do well on many tests of mental ability.

Table 1: Sensitivity and specificity of common tests for dementia

Test	Sensitivity	Specificity	Reference
MMSE	71%-92%	56%-96%	[19]
3MS	83%-93.5%	85%-90%	[20]
AMTS	73%-100%	71%-100%	[20]

While many tests have been studied,[24][25][26] and some may emerge as better alternatives to the MMSE, presently the MMSE is the best studied and most commonly used. Another approach to screening for dementia is to ask an informant (relative or other supporter) to fill out a questionnaire about the person's everyday cognitive functioning. Informant questionnaires provide complementary information to brief cognitive tests. Probably the best known questionnaire of this sort is the *Informant Questionnaire on Cognitive Decline in the Elderly (IQCODE)*.[27]

On the other hand the *General Practitioner Assessment Of Cognition* combines both, a patient assessment and an informant interview. It was specifically designed for the use in the primary care setting and is also available as a web-based test. Further evaluation includes retesting at another date, and administration of other tests of mental function.

Laboratory tests

Routine blood tests are also usually performed to rule out treatable causes. These tests include vitamin B_{12}, folic acid, thyroid-stimulating hormone (TSH), C-reactive protein, full blood count, electrolytes, calcium, renal function, and liver enzymes. Abnormalities may suggest vitamin deficiency, infection or other problems that commonly cause confusion or disorientation in the elderly. The problem is complicated by the fact that these cause confusion more often in persons who have early dementia, so that "reversal" of such problems may ultimately only be temporary. Testing for alcohol and other known dementia-inducing drugs may be indicated.

Imaging

A CT scan or magnetic resonance imaging (MRI scan) is commonly performed, although these modalities do not have optimal sensitivity for the diffuse metabolic changes associated with dementia in a patient that shows no gross neurological problems (such as paralysis or weakness) on neurological exam. CT or MRI may suggest normal pressure hydrocephalus, a potentially reversible cause of dementia, and can yield information relevant to other types of dementia, such as infarction (stroke) that would point at a vascular type of dementia.
The functional neuroimaging modalities of SPECT and PET are more useful in assessing long-standing cognitive dysfunction, since they have shown similar ability to diagnose dementia as a clinical exam.[28] The ability of SPECT to differentiate the vascular cause from the Alzheimer's disease cause of dementias, appears to be superior to differentiation by clinical exam.[29]
Recent research has established the value of PET imaging using carbon-11 Pittsburgh Compound B as a radiotracer (PIB-PET) in predictive diagnosis of various kinds of dementia, in particular Alzheimer's disease. Studies from Australia have found PIB-PET to be 86% accurate in predicting which patients with mild cognitive impairment would develop Alzheimer's disease within two years. In another study, carried out using 66 patients seen at the University of Michigan, PET studies using either PIB or another radiotracer, carbon-11 dihydrotetrabenazine (DTBZ), led to more accurate diagnosis for more than one-fourth of patients with mild cognitive impairment or mild dementia.[30]

Prevention

It appears that the regular moderate consumption of alcohol (beer, wine, or distilled spirits) and a Mediterranean diet may reduce risk.[31][32][33][34] A study failed to show a conclusive link between high blood pressure and developing dementia. The study, published in the *Lancet Neurology* journal July 2008, found that blood pressure lowering medication did not reduce dementia but that meta analysis of the study data combined with other data suggested that further study could be warranted.[35]
Brain-derived neurotrophic factor (BDNF) expression is associated with some dementia types.[36][37][38]
A Canadian study found that a lifetime of bilingualism delays the onset of dementia by an average of four years when compared to monolingual patients.[39]

Management

Except for the treatable types listed above, there is no cure to this illness. Cholinesterase inhibitors are often used early in the disease course. Cognitive and behavioral interventions may also be appropriate. Educating and providing emotional support to the caregiver (or carer) is of importance as well (*see also elderly care*).

Pain and dementia

As people age, they experience more health problems, and most health problems associated with aging carry a substantial burden of pain; so, between 25% and 50% of older adults experience persistent pain. Seniors with dementia experience the same prevalence of conditions likely to cause pain as seniors without dementia.[40] Pain is often overlooked in older adults and, when screened for, often poorly assessed, especially among those with

dementia since they become incapable of informing others that they're in pain.[40][41] Beyond the issue of humane care, unrelieved pain has functional implications. Persistent pain can lead to decreased ambulation, depressed mood, sleep disturbances, impaired appetite and exacerbation of cognitive impairment,[41] and pain-related interference with activity is a factor contributing to falls in the elderly.[40][42]

Although persistent pain in the person with dementia is difficult to communicate, diagnose and treat, failure to address persistent pain has profound functional, psychosocial and quality of life implications for this vulnerable population. Health professionals often lack the skills and usually lack the time needed to recognize, accurately assess and adequately monitor pain in people with dementia.[40][43] Family members and friends can make a valuable contribution to the care of a person with dementia by learning to recognize and assess their pain. Educational resources (such as the Understand Pain and Dementia tutorial) and observational assessment tools are available.[40][44][45]

Medications

Currently, there are no medications that are clinically proven to be preventative or curative of dementia.[46] Although some medications are approved for use in the treatment of dementia, these treat the behavioural and cognitive symptoms of dementia, but have no effect on the underlying pathophysiology.[47]

- Acetylcholinesterase inhibitors: Tacrine (Cognex), donepezil (Aricept), galantamine (Razadyne), and rivastigmine (Exelon) are approved by the United States Food and Drug Administration (FDA) for treatment of dementia induced by Alzheimer's disease. They may be useful for other similar diseases causing dementia such as Parkinsons or vascular dementia.[47] Acetylcholinesterase inhibitors aim to increase the amount of the neurotransmitter acetylcholine, which is deficient in people with dementia.[48] This is done by inhibiting the action of the enzyme acetylcholinesterase, which breaksdown acetylcholine as part of normal brain function.[49] Though these medications are commonly prescribed, in a minority of patients these drugs can cause side effects including bradycardia and syncope.[50]

- N-methyl-D-aspartate (NMDA) receptor blockers: Memantine is marketed under several names by different pharmaceutical companies including: Abixa, Akatinol, Axura, Ebixa, Memox and Namenda.[51] In dementia, NMDA receptors are over-stimulated by glutamate, which creates problems for neurotransmission (and thus cognition) and also leads to damage to neurons through excitotoxicity. Memantine is thought to work by improving the "signal-to-noise" ratio and preventing excitotoxic damage.[52] Hence, due to their differing mechanisms of action memantine and acetylcholinesterase inhibitors can be used in combination with each other.[53][54]

Off label

- Amyloid deposit inhibitors: Minocycline and Clioquinoline, antibiotics, may help reduce amyloid deposits in the brains of persons with Alzheimer's disease.[55]

- Antidepressant drugs: Depression is frequently associated with dementia and generally worsens the degree of cognitive and behavioral impairment. Antidepressants effectively treat the cognitive and behavioral symptoms of depression in patients with Alzheimer's disease,[56] but evidence for their use in other forms of dementia is weak.[57]

- Anxiolytic drugs: Many patients with dementia experience anxiety symptoms. Although benzodiazepines like diazepam (Valium) have been used for treating anxiety in other situations, they are often avoided because they may increase agitation in persons with dementia and are likely to worsen cognitive problems or are too sedating. Buspirone (Buspar) is often initially tried for mild-to-moderate anxiety.[citation needed] There is little evidence for the effectiveness of benzodiazepines in dementia, whereas there is evidence for the effectivess of antipsychotics (at low doses).[58]

- Selegiline, a drug used primarily in the treatment of Parkinson's disease, appears to slow the development of dementia. Selegiline is thought to act as an antioxidant, preventing free radical damage. However, it also acts as a stimulant, making it difficult to determine whether the delay in onset of dementia symptoms is due to protection from free radicals or to the general elevation of brain activity from the stimulant effect.[59]

- Antipsychotic drugs: Both typical antipsychotics (such as Haloperidol) and atypical antipsychotics such as (risperidone) increase the risk of death in dementia-associated psychosis.[60] This means that any use of antipsychotic medication for dementia-associated psychosis is off-label and should only be considered after discussing the risks and benefits of treatment with these drugs, and after other treatment modalities have failed. In the UK around 144,000 dementia sufferers are unnecessarily prescribed antipsychotic drugs, around 2000 patients die as a result of taking the drugs each year.[61]

Services

Adult daycare centres as well as special care units in nursing homes often provide specialized care for dementia patients. Adult daycare centres offer supervision, recreation, meals, and limited health care to participants, as well as providing respite for caregivers. In addition, Home care can provide one-on-one support and care in the home allowing for more individualized attention that is needed as the disease progresses. While some preliminary studies have found that music therapy may be useful in helping patients with dementia, their quality has been low and no reliable conclusions can be drawn from them.[62]

Nursing people with dementia

 Psychiatric nurses can make a distinctive contribution to people's mental health. The four main premises upon which psychiatric nursing is based are:

- The nursing is an interactive, developmental human activity that is more concerned with the future development of the person than the origins.

- The experience of mental distress related to the psychiatric disorder is represented through disturbances or reports of private events that are known only to the person concerned.

- Nurses and the people in care are engaged in a relationship based on mutual influence.

- The experience of psychiatric disorder is translated into problems of everyday living and the nurse notes the human responses to the psychiatric distress, not the disorder.[63]

Feeding tubes

There is little evidence, even after widespread use, that feeding tubes help patients with advanced dementia gain weight, regain strength or function, prevent aspiration pneumonias, or improve quality of life. The risks associated with the use of tubes are not well known.[64]

Epidemiology

Evidence from well-planned, representative epidemiological surveys is scarce in many regions, particularly in low-income countries. However, estimates from 2005 suggest a global dementia prevalence of 24.3 million, with 4.6 million new cases of dementia every year. The number of people affected will double every 20 years to 81.1 million by 2040.[65]

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