DEMENTIA

A survey of the syndrome of dementia

by

B. Mahendra

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Preface

Three points must strike anyone who has embarked on a study of dementia over a period of time. *Firstly,* that our conception of the syndrome is in a state of flux. Gone, for instance, in the past decade or two, is the requirement of a chronic, progressive, irreversible disorder for the diagnosis. I remember the surgeon who, when I was a student, returned a referral saying he would operate on the man when his dementia got better. Feeling superior, and encouraged by the consultant psychiatrist, we students laughed a good deal at this. Before we finished clerking on that Unit a visiting Professor of Psychiatry had demonstrated the reversibility of the symptoms of dementia in a patient with a rare metabolic disorder. Perhaps ignorance is sometimes an advance on received wisdom. The lesson is the concept of dementia must always reflect the state of knowledge and is therefore in a sense *ad hoc.*

Secondly, what the criteria for, and also who the arbiters of, the diagnosis might be is not always clear. It is traditional to think that expressing opinions and making diagnosis of mental illness is almost a civic right, i.e. virtually everyone's business. It would be an exaggeration to claim the same situation existed for dementia but over the years, despite the availability of generally well-known definitions of the concept, dementia has meant all things to all men and women. As I sit through learned discussions on the minutiae of chemical, immunological and radiological technique I fear there is danger of forgetting the simple clinical formulation of dementia and with it the prospect of clear understanding of the concept. It will emerge in the book that what has been written, for instance, about alcoholic and metabolic dementias, has sometimes been the result of misapprehension and, in every sense, confusion about what the changes of dementia might be in those conditions. Rigorously applied and explicitly stated criteria were thought to be necessary for the schizophrenias and perhaps we must insist likewise for dementia.

Thirdly, one is always struck by the neglect of the subject of dementia.

Despite the torrent of statistical evidence and projections about the numbers of demented patients and the growing social importance of the syndrome, the subject still has not caught the popular imagination, medical or lay. I think a major obstacle to a serious study of dementia is the entrenched attitudes displayed by the traditional medical specialities and their preoccupations with demarcation disputes. A brief glance at the chapter headings will show a great variety of approaches to the subject. A speciality which refuses to acknowledge the value of scientific investigation or, on the other hand, washes its hands of the psychological and social aspects of the condition or refuses to soil them with the (for the present) often difficult management of hopelessly demented patients cannot hope to seriously contribute to advancement of knowledge. I suspect ideologies and doctrines of specialism are more important reasons for this neglect of dementia, though there may be something to be said for the claim that lack of concern might also owe a little to our unconscious fears of ageing and decrepitude, a point usually made by those of a metaphysical bent who can reach those parts of the soul that the rest of us cannot.

This book is an attempt to provide a critical account of the more important aspects of dementia as conceived at the present time. It aims to provide neurologists and psychiatrists in training and in preparation for the membership examinations, specialists in one or another area of investigation who wish to obtain a global overview of the syndrome, interested senior undergraduates, the hard-pressed general practitioner who might find himself in the vanguard of community care, nurses, social workers, occupational therapists and relatives of demented patients with an account which, it is hoped, will be manageably comprehensive. The works given for further reading are general and more accessible accounts for those who wish to know a little more, and those who want to pursue any subject at greater length have a set of references at the back of the book.

An incidental benefit in assembling these several conditions, which give the syndrome of dementia its rich variety, is the opportunity to describe their historical aspects. These stories are oft-told but are not always readily available. It will be clear that a historical perspective is vital to the understanding of dementia.

I make no apology for the presence of sections on social, ethical and pathographic aspects of the condition. It is plain that doctors will lead an increasingly sterile existence if they do not acknowledge those wider areas of life and living which might have a bearing on the course of illness and the practice of medicine. In any case, the points that have been made are of a very basic kind or are in the form of simple narrative. I hope they will serve as starting points for discussion, controversial if need be, and also encourage a more serious study of those individuals in public or artistic life who have become afflicted by dementia of one kind or another.

B MAHENDRA

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1
Dementia: a brief history of the concept

The best way to suppose what may come is to remember what is past.
George Savile (1633–1695)

The concept of dementia that we know today, and which forms the subject-matter of this book, has evolved over many centuries. The problem has always been what to make of the term. Its derivation has not helped matters for 'dementia' corresponds to Latin *dementatus* – that is, out of one's mind, crazed, applicable to any and all abnormal, unusual, incomprehensible or bizarre behaviour.

In tracing the course of its distilled meaning – from the vantage point of over twenty centuries of hindsight – we shall see the term being adapted to suit conditions that were current in any given society. It is easy to understand that a concept now employed in most instances in relation to the elderly sick in the 20th century might not have had much meaning for the very different populations, patients and practitioners of the past.

For a start, living to be old was a distinctly unusual phenomenon in past centuries. Very few in the population survived to the senium, the period of life in which dementia becomes a calculable prospect rather than a chance occurrence. An estimate of those reaching the age of 65 in primitive society is around 3%; the life-span, on the average, of a subject in the Roman Empire was less than 30; an Englishman in 1700 could hope to live to a mean of 35 years; in 1840 the span of life had risen to 40–43 and in the 1980s it is 70–76, women living to the higher age.

In those early days, while the small numbers of the elderly would have shown the common cognitive changes associated with normal old age, only a minute proportion would have become demented in a modern sense. It is easy to see how the features of pathologic old age in the few would have been swamped by the relatively larger number of the elderly showing subtler physiological changes. This explanation would take care of growing old as an aetiological factor; the ancient role of such modern toxins as radiation,

transient viruses and metallic poisons we may never come to know.

This account of earlier approaches to senility and attitudes to old age owes much to George Rosen's excellent paper (1961). He quotes Cicero's essay on old age in the 2nd century BC. The major preoccupation in this age was the practice of mental hygiene. Poets and philosophers, at least, wondered if an active mental life might not forestall or postpone the decrepitude of old age. Cicero wrote:

> It is our duty to resist old age; to compensate for its defects by a watchful care; to fight against it as we would fight against disease. . . . Much greater care is is due to the mind and soul; for they, too, like lamps, grow dim with time, unless we keep them supplied with oil. . . . Intellectual activity gives buoyancy to the mind. . . . Old men retain their mental faculties, provided their interest and application continue . . . the aged remember everything that interests them. . . . (Rosen, 1961)

This passage, which would not be out of place in a modern book on alternative health and mind care, also trails a familiar preoccupation of the next several centuries – mental hygiene for the prevention or postponement of old age. Cicero observes both the physical and mental effects of senility but there is no indication he attributed the changes to pathology. And the idea that intellectual activity could stave off the mental debilitation of the senium would have appeared quaint if some modern evidence did not lend support to it.

Celsus (30 BC–AD 50) probably first used the term 'dementia' in a medical context in the 1st century AD. In his encyclopaedia he is said to mention in passing that paralysis occurs in old age, but in his discussion of insanity there is no reference to old people. A century later the term 'senile dementia' itself seems to have been first used by Aretaeus, the physician of Cappadocia, but whether the concept had evolved to the point where it could be differentiated from the changes of normal old age is not known. Aretaeus commented on the mental decay of old age in discussing the differential diagnosis of a reversible form of excitement. He remarked this condition bore no resemblance to 'the dotage which is the calamity of old age . . . dotage commencing with old age never intermits, but accompanies the patient until death; while mania intermits, and with care ceases altogether' (Alexander, 1972). Mania was, of course, not necessarily the modern affective disorder but presumably any form of reversible mental illness with a component of excitement. This undoubtedly included delirium of organic causation.

In the 2nd century AD, when only a fraction of humanity was still destined to reach old age, Juvenal (AD 60–130) dealt with the subject with pessimism and bitterness in his tenth satire. This was the work that denounced the vanity of human wishes and warned of the tragedy of human hopes. Old age is ugly, deaf, blind, crippled by disease and deprived of reason.

> But worse than all bodily failing is the weakening mind which cannot remember names of slaves, nor the face of a friend he dined with last

> evening, cannot remember the names of offspring begotten and reared. . . .

If you must pray for something, *orandum est ut sit mens sana in corpore sano*, which in a truncated form became a tag popularized by generations of schoolchildren.

Rosen then describes the work of Caelius Aurelianus a century later, who was influenced by Soranus of Ephesus, a physician in Rome at the time of the Emperors Trajan (AD 53–117) and Hadrian (AD 76–138). It appears that Caelius made several references to old age and mental illness and by this time, a full century after Celsus and Aretaeus, the pathological basis to some of the changes in the senium was being appreciated.

A couple of centuries later, in the 4th century AD, Oribasius, physician to the Emperor Julian, wrote of cerebral atrophy in his digest of medicine and surgery. This disease was thought to manifest itself in a loss of intellectual capacity and in weakness of movement. Oribasius tried to relate the condition to the ageing process and this was probably one of the earlier attempts to speculate as to aetiology of a more specific nature than mere old age.

By the 7th century AD the Byzantine physician, Paul of Aegina, was able to deal with the problems of loss of memory and reason in his writings. Rosen has said his discussion is obscure and it is uncertain as to whether senile dementia, feeblemindedness or aphasia were being dealt with. This is no reflection on Paul of Aegina as it was not until the 19th century that the distinction between the cognitive impairement due to dementia could be separated from that caused by mental handicap. The point to note, however, is that by this time loss of memory and reason had become subjects for legitimate study by physicians, as opposed to poets and philosophers. And as for the confusion between dementia and feeble-mindedness, it might well suggest that features, till then associated with the senium, were being observed in a younger age group.

As we have seen, states of excitement occasionally needed sorting out from dementia, but a more common cause of difficulty is likely to have been depression. Rosen notes that in the 9th century AD Rhazes the Persian physician mentions melancholy as an inevitable condition in the lives of old and decrepit persons. The Moslem writers of the Middle Ages seem to have had ideas similar to those of the Graeco-Romans on the nature of old age. The inevitable decrepitude and melancholic character of old age were commonplace ideas during the Renaissance period but the principal sources remained the ancient authors or mediaeval writers indebted to classical thought (Rosen, 1961).

Robert Burton (1577–1640), the author of *Anatomy of Melancholy*, observed that 'after seventy years all is trouble and sorrow'. He had views on the relationship between old age and melancholy too, saying that old age was 'natural to all . . . being cold and dry, and of the same quality as Melancholy is, must needs cause it, by diminution of spirits and substance. . . .'

Most people remember Shakespeare's (1564–1616) description of

> . . . lean and slippered pantaloon,
> With spectacles on nose and pouch on side
> His youthful hose, well saved, a world too wide
> For his shrunk shank; and his big manly voice,
> Turning again toward childish treble, pipes
> And whistles in his sound. Last scene of all
> That ends this strange eventful history,
> Is second childishness and mere oblivion,
> Sans teeth, sans eyes, sans taste, sans everything. (*As You Like It*)

but the bard has also to say:

> An old man is twice a child (*Hamlet*)

and

> You see me here, you gods, a poor old man,
> As full of grief as age; wretched in both! (*King Lear*)

which as the plot reveals is likely to have been something more than mere understandable reaction to a domestic dispute. Lear's senile peregrinations are well contrasted with the lunatic ravings of poor Tom O'Bedlam. The distinction between plain madness and senile decay is made clear and, like Burton, Shakespeare notes the association between cognitive and affective change.

We now move to the period between 1535 and 1860, the era covered by Hunter and Macalpine's excellent source book (1963). The authors include accounts of the involvement of several practitioners who involved themselves with cases of cognitive disorder. Philip Barrough (1560–1590), a licensed practitioner in Cambridge and author of a widely read textbook, seems to have drawn a distinction between loss of memory and loss of reason in cognitive disturbance.

> If reason be lost together with the memorie, then the affect is called *Fatuitas* or *stultitia* [that is] folishnes or doltishnes, and both these do come of one disposition, but that is more vehement wher both are hurte.

Richard Cosin, who lived in the later part of the sixteenth century, wrote an early account of the legal implications of insanity. *Inter alia* he considered six categories of 'wants of understanding and reason'. His 'dementia' refers to

> A passion of the minde, bereaving it of the light of understanding: Or . . . when a man's perceivance and understanding of all things is taken away, and may be englished distracted of wit, or being beside himself. (Hunter and Macalpine, 1963)

This, of course, is too wide-ranging and general to be a definition of mere cognitive change and suggests that while the features of the dementing

process could be recognized and described, the term 'dementia' was not necessarily appended to it. In fact, Cosin's other legal categories, *lethargie*–' . . . a notable forgetfulnes of all things almost, that hereto fore a man hath knowen . . . '; *delirium*–' . . . that weaknes of conceite and consideration, which we calle dotage: when a man, through age or infirmitie, falleth to be a childe again in discretion'; *stultitia*–' . . . that follie which is seen in such, as albeit they be but simple and grosse witted, yet are not to be accounted Idiotes, or Naturals . . . ', all contain elements of cognitive impairment amounting to dementia in its modern conception. The multiplicity of terms probably reflected the confusion in the ranks of the practitioners who were, as Cosin's interest in these matters suggests, not all medical men. Perhaps this attempt at a legal interpretation of the confused state of cognitive functioning in the 16th century was a foretaste of the legal profession's attempts to wrestle with the elusive concept of psychopathy four centuries later.

As Hunter and Macalpine note, early in the 17th century, Bedlam (Bethlem Hospital) was able to make its contribution to dementia. The Puritan divine, Thomas Adams, in his book *Mystical Bedlam OR The World of Mad-men* (1615) wrote of ' . . . some, that be hurt in both imagination and reason, and they necessarily therewithall doe lose their memories'. His other two categories refer to what in modern terms would be called delusions (erring in cogitation and reason) and hallucinations (problems with Phantasie and Imagination). These were subdivisions of the 'three internal senses or faculties . . . Imagination, Reason and Memorie'. They are clearly not mutually exclusive categories and one wonders how a demented patient with delusions and hallucinations might have fared. As melancholy in the sense of depressed affect was recognized and very likely given pride of place in any diagnostic hierarchy, it seems probable that problems of cognition continued to be understood and explained in terms of disordered affect, thought and perception except in the elderly when a primary disorder of higher functions could be invoked.

Hunter and Macalpine also include an account of one William Salmon (1644–1713), a practitioner who made his living apparently from patients turned away from St Bartholomew's Hospital, an early indication perhaps of attitudes to the less glamorous branches of the medical profession. He described a patient with senile dementia under the heading, 'Defects of Imagination, Reason and Memory in a Man superannuated' whom he diagnosed as 'not mad, or distracted like a man in Bedlam' but 'decayed in his Intellectuals'. He observed the early stages when such patients complain of depression and hypochondriacal symptoms and drew attention to the diagnostic triad of emotionality leading to involuntary laughing and crying associated particularly with arteriosclerotic dementia; loss of memory for recent events and perseveration (Hunter and Macalpine, 1963).

This brings us near the 18th century and the time is about right to embark on one of the more romantic stories in all medicine, the saga of general paresis. But before we, so to speak, take a break, it might be helpful to summarize what we have learned, with due warning that what we now say might well be conjecture and no more.

The earliest meaning of dementia is likely to have meant insane and seriously irrational behaviour. No distinction was presumably made between the normal and abnormal changes of old age until there were numbers of elderly in the population exhibiting this behaviour. At some point it must have been noted that the difficulties of some of the elderly were significantly different, and greater than that of the majority of their peers. Associated with this serious impairment in cognitive function was change in affect, mostly in the direction of depression, but cause and effect were probably beyond the understanding of observers. Whilst distinction could be drawn between the insanity of old age with a clear cognitive bias and the madness of younger patients with a predominant loss of reason and sense, there was almost certainly no concept of pre-senile dementia, no attempt apparently being made to distinguish between the cognitive failure of this group and those who were intellectually handicapped from a very early age.

AN INTERLUDE: THE STORY OF GENERAL PARESIS

Henry (1941) has given a straightforward account of the conventional view of the origins and discovery of general paresis. The evidence for the existence of general paresis prior to the early 19th century is thought to be inconclusive. Syphilis did not appear, at least in epidemic form, until after the return of Columbus from Haiti in 1493. Within a few years it had spread through Europe like a plague.

The recognition of general paresis as a separate disease entity was slow: conventional wisdom attributes this to Thomas Willis (1672). Willis wrote:

> In . . . cases . . . when, the brain being previously undisposed, they were visited with dullness of mind and forgetfulnes and then with stupidity and foolishness, they would afterwards fall into paralysis . . .

Hare (1959) has disputed the claims that Thomas Willis actually describes general paresis. Willis' passages in *De Anima Brutorum* are reviewed by Hare who believes much of the description would fit the case for arteriosclerotic dementia. He cites Robertson (1923), who had also expressed the view that it was 'much more likely that Willis refers to senile or arteriosclerotic dementia'.

After Willis one had to wait for more than a century for a communication which is possibly on the same subject, by John Haslam in 1798. Hare believes Haslam's claims to have observed general paresis to have been based on sounder evidence than Willis'. Haslam wrote:

> The paralytic affections are a more frequent cause of insanity than is believed. . . . As a rule the paralytics present disorders of motion, which are wholly independent of the mental disease . . . in the majority of patients memory is materially weakened. These patients, as a rule, fail to recognize their condition . . . [and] still maintain they are extremely strong and capable of the greatest deeds. (Haslam, 1798)

However no satisfactory or unequivocal accounts of a disease corres-

ponding to general paresis were given before those of the Parisian alienists in the early 19th century when anything from a sixth to a quarter of admissions to mental hospitals were for this illness (Hare, 1959). Esquirol in 1805 referred to the '. . . incurability of insanity complicated with paralysis', similar in prognostic hopelessness to the dementia of old age, but it had not occurred to Esquirol or anyone else that they were dealing with a separate and distinct disease entity (Henry, 1941).

In 1822 Bayle began presenting the conclusions to his clinical and pathological studies. He stated that the mental and physical symptoms are an expression of a single disease which is based upon a chronic inflammation of the meninges. Bayle was thus the first writer to affirm that general paresis was a disease entity characterized by disturbances of intellectual functions, by a variety of grandiose ideas peculiar to the disease and by progressive muscular inco-ordination and enfeeblement. He stated further that general paresis could not be considered as a complication of a mental illness and the symptoms did not occur as a part of any other form of mental illness (Henry, 1941).

Not deterred by all this, even as late as 1838, Esquirol continued to regard general paresis as a complication of various forms of mental disorder. (It is easy to see why clinicians cling to ideas like this. Cognitive disturbance was also probably considered for a long time as a complication of affective, melancholic or other 'mental' processes; perhaps it follows from the reductionist notion in medicine of trying to fit all symptomatology into a plausible, *single* diagnosis, most often a safe, well-established one. It is not simply the perversity of practitioners but a consequence of their upbringing.)

The specific aetiology for general paresis was not proposed until Esmarch suggested in 1857 that syphilis was the essential cause. Fournier had considered tabes dorsalis syphilitic in 1875 and in 1894 offered statistical evidence for the syphilitic origin of general paresis. But it was 1904 before a full description of the histopathological changes was given by Alzheimer.

In that same year Kraepelin had stated that 'syphilitic infection is essential for the later appearance of paresis'. Cerebrospinal fluid had been made practically available by Quinke's method in 1890 and when Wassermann's diagnostic test came into practice in 1906, an important link in the chain of evidence substantiating the theory of syphilitic origin of general paresis was forged. Further laboratory evidence was forthcoming with Lange's colloidal gold test discovered in 1912 (Henry, 1941).

Progress was being made on other fronts as well. In 1905 Schaudinn found the spirochaete in primary lesions and in 1913 *Treponema pallidum* was demonstrated in paretic brains by Noguchi and Moore. Ehrlich had discovered 'Salvarsan' (neoarsphenamine), the first of the modern pharmaceuticals, and this was pressed into service in 1910. In 1917 Wagner Von Jauregg was using fever therapy and, two world wars later, penicillin became the definitive treatment.

Hare (1959) has wondered why general paresis might have suddenly appeared on the firmament and then faded away only slightly less dramatically. From being a rare or non-existent disease it had become very

prevalent in Paris in the early 19th century. From its origins in northern France the disease spread across Europe, then to America and later still to less highly industrialized countries. Hare believes there is evidence that during the subsequent 140 years the disease had shown gradual modifications in clinical form and a recent natural decline in prevalence, and that these changes are comparable with those that took place in the clinical course of syphilis during the years that followed the 15th century epidemic.

Hare further believes these points in the evidence will support the hypothesis that general paresis is due to a special 'neurotropic' strain of the syphilitic spirochaete, for they allow us to put forward the view that a mutation giving rise to the neurotropic strain occurred in northern Europe towards the end of the 18th century; that the spread of the mutant strain explains the time lapse before the disease was recognized in other countries; and that the new disease slowly changed in its prevalence and clinical manifestations (Hare, 1959).

Hare's thesis is an important one inasmuch as it considers the mutability of illness. If general paresis can arise relatively suddenly, so can other causes of dementia and their eclipse, for secular reasons, can be equally rapid. In a later interlude I shall discuss the recognition of Huntington's chorea, another illness that rose quickly to public prominence in the late 19th century after years of neglect. When one considers also the possibility of a kind of viral aetiology for Alzheimer's disease which has been mooted in a few quarters, one becomes cautious about commenting on diseases outside one's era. Not only concepts but diseases themselves may undergo change and break out of the Linnaean chains we like to impose upon them. Hare (1974) has further discussed the comings and goings of some other psychiatric illnesses including schizophrenia in a similar vein.

No account of any disorder with psychiatric manifestations in the 17th and 18th centuries would be complete without some mention of witchcraft, its alleged practitioners and the reaction of the public to them. There is little doubt now that the majority of persecuted witches were strange and solitary women given to various displays of eccentric behaviour. Some of them were old, and Rosen describes the last witch to be burnt in Scotland in 1722 as having been a very old woman. However, there seems little evidence that the psychiatric difficulties of these women were purely or mainly cognitive in nature. In fact, one would have thought that the pathetic mental and decrepit physical state of a truly demented woman would have evoked pity rather that the fear and loathing which seem to have fuelled much of the persecution. It would have been a different matter, though, if old women with cognitive defects had also exhibited physical 'stigmata' and the fate of female Huntington's choreics, as we shall see later, may well have been very different.

Modern psychiatry often traces its roots to Philippe Pinel (1745–1826), the humanitarian reformer of asylum practices who released the patients of the Bicetre and Salpetriere from their chains. He did not have much to say about dementia but uses the word 'demence' to designate one of the five

classes of mental derangement and featuring 'the abolition of the thinking faculty'. However, he must have influenced his favourite, and most illustrious, pupil, Jean Etienne Dominique Esquirol (1772–1840), the very same as tried to make sense out of the signs of general paresis, who in 1838 published *Traite des Malades Mentales*, the fruit of 40 years' study of patients at the mental hospitals of Salpetriere and Charenton and in private practice. He defined dementia as a cerebral affection marked by a weakening of sensibility, understanding and will, and by the impairment of memory, reasoning and attention.

Esquirol distinguished three varieties of dementia: acute, chronic and senile. The acute variety could be caused by fever or haemorrhage and it was curable. The chronic form could be due to such factors as masturbation and drunkenness, or it could follow mania or epilepsy, and it was seldom cured. Clearly, Esquirol included a wide range of psychiatric disorders including delirium and functional illness in this conception. '*Demence senile*' is described as follows:

> Senile dementia results from the progress of age. There is . . . loss of sensibility along with . . . the faculty of understanding, before reaching an extreme state of decrepitude. Senile dementia . . . commences with feebleness of memory, particularly recent memory; attention . . . becomes impossible; the will is uncertain, the movements are slow . . . (Esquirol, 1838)

However, after this passage, which would seem unexceptionable as a modern description of dementia, Esquirol goes on to treat the origins of senile dementia in certain other cases. These were not rare instances, by any means. In fact Esquirol has said senile dementia 'began rather often' in this fashion. The passage that follows – which is an account of general excitement and hyperactivity, irritability, an increase in appetite for food, drink and sex – is clearly one that would sound like mania to modern ears and the 'dementia' that followed was very likely what we would call depression. Esquirol, then, was over-inclusive in his use of the term 'senile dementia' but his description of cognitive changes in some of those he considered demented in the senium brings him close to the modern conception.

Esquirol made a further contribution to our growing understanding of the concept. We have already seen that at least from the 7th century AD physicians had been confusing the cognitive impairment that follows a disturbance in the fully grown brain with that which arose in the undeveloped or partially developed brain. In other words, demented individuals were being confused with the intellectually subnormal. It is not clear why in the early 19th century attention should become focused on this difference. It may be, as so often is the case, that social pressures caused the minds of clinicians to be exercised. There was industrialization in the air, disturbing rustic life, and the subnormal who could be contained in villages, when the whole land was a village and life was uniformly simple, had now to be dealt with in new ways. The distinction between dementing intellectual impairment and subnormal intellectual deficit was no longer

academic. It was also the heady years following the French Revolution and the other side of the coin to the mindless savagery of those times was a new humanitarian spirit that was abroad in the land. The treatment of the mentally ill and handicapped, who had no privileges worth speaking of, was no doubt under favourable review, not least by Pinel and Esquirol.

In 1838 Esquirol was able to sum up the difference between the demented and the mentally handicapped in an epigram: 'The dement is a man deprived of the possessions he once enjoyed, he is a rich man who has become poor. But the defective has been penniless and wretched all his life.' It could not have been put more succintly.

James Cowles Prichard (1786–1848), physician and commissioner in Lunacy, described four stages of dementia that reflected its progression: impairment of recent memory, loss of reason, incomprehension and loss of instinctive action. He used the term 'Incoherency or Dementia' to denote a depression or a loss of the intellectual faculties. But views as to aetiology remained speculative. Prichard could add in 1842 that

> senile decay is occasionally the consequence of various disorders affecting the brain, such as long continued mania, or melancholia, or attack of apoplexy, or paralysis or severe and often repeated attacks of epilepsy or typhoid fevers in which the brain has been much affected. (Hunter and Macalpine, 1963)

Whilst the general idea seems to have been that organic change in the brain was needed to precipitate or sustain the cognitive impairment of old age, the early 19th-century practitioners were unwilling or unable still to separate the disorders of consciousness caused by acute injury and infection from the chronic course of dementia. Affective features could have been symptoms of organic disorder; also chronic, intractable depression could give a picture of persistent cognitive change, especially in that period a century before the availability of effective treatment, indistinguishable from dementia.

Conceptual advance was now not going to be rapid. Fifty years after Prichard, as the century turned, Tuke still managed to distinguish four types of dementia in the following fashion: primary or acute; secondary, which could follow mania or melancholia; senile; and paralytic. As with almost everyone on the continent in those early Kraepelinian days Tuke included cases of 'schizophrenia' under the heading of dementia.

In 1845 Wilhelm Griesinger's textbook on psychiatry had included a classification of 'apathetic dementia' under the general label of 'states of mental weakness'. Senile dementia was one example of apathetic dementia and thought to be due to disease of the cerebral arteries, a view that persisted until the time of Alzheimer. Griesinger was the quintessential exponent of the organic school of psychiatry. The phrase 'all mind diseases are brain diseases' has been attributed to him and he believed in the unitary nature of psychotic illness, i.e. mental illness is due to a single process and their manifestations are reflections of the stage and severity of the process.

A significant contribution to the mental pathology of old age was provided by Wille in 1873–1874. He endeavoured to clarify the aetiological, pathogenetic and pathologo-anatomical aspects of this area of