Language and memory disorder in the case of Jonathan Swift: considerations on retrospective diagnosis

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The cause of behavioural changes described by Alzheimer for his original case, Auguste D., has been recently reconfirmed by histological examination. However, there has been active speculation regarding the cause of behavioural changes exhibited by the political satirist Jonathan Swift (1667–1745) during the final three years of his life for over 250 years. Swift’s symptoms of cognitive changes, memory impairment, personality alterations, language disorder and facial paralysis have all been apportioned differing levels of significance in various attempts at retrospective diagnosis. The various medical arguments put forward from the 18th through 20th centuries will be critically examined. The diagnoses considered refer to evolving theories of insanity, phrenology, localization of cortical function, hydrocephalus, psychoanalysis, aphasia, dementia and depression in ageing. Re-consideration of the attempts to re-diagnose Swift’s final mental state by the leading neurological thinkers of the day, including Wilde (The Closing Years of Dean Swift’s Life. Dublin: Hodges and Smith, 1849), Bucknill (1882), Osler [Osler’s textbook Principles and Practice of Medicine (1892); published in St Thomas’s Hospital Gazette (London) 1902; 12: 59–60], Brain (Irish Med J 1952: 320–1 and 337–346) and Boller and Forbes (J Neurol Sci 1998; 158: 125–133) reveal the changing attitudes regarding the significance of behavioural symptoms to neurological diagnosis from the 18th century to the present day.

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Introduction

In 1906, Alois Alzheimer presented the case of Auguste D. at a meeting of the South-West German Psychiatrist at Tubingen. He provided a 5-year case history, with details of behavioural testing (Maurer et al., 1997). The diagnosis was determined by microscopic examination of nervous tissue at necropsy. Retrospective confirmation of Auguste D.’s diagnosis as formulated by Alzheimer 100 years ago was provided in 1999 by Graeber and Mehraein. This verification was achieved by carrying out modern histopathological techniques on the preserved brain of Auguste D. Morphological examination revealed the presence of numerous amyloid plaques, or miliar foci, as Alzheimer called them, in the upper cortical layers along with neurofibrillary tangles. The findings of Graeber and Mehraein (1999) completely match Alzheimer’s original description (Alzheimer, 1907). This is a remarkable instance in which an historical diagnostic category was upheld using modern assays (Enserink, 1998).

What is generally required for definitive diagnosis of many diseases affecting cognitive functions is the conjunction of behavioural performance patterns on a particular set of standardized test batteries, and pathological findings. There are a number of circumstances in which diagnosis remains indeterminate; lack of detailed description of behavioural impairment by a trained clinician using validated assessment tools, lack of information about pre-morbid abilities and...
history of illness, or lack of evidence of aetiology from imaging, tissue biopsy or autopsy.

In the case of Auguste D., there was both clinical and pathological data for what became the eponym of Alzheimer’s disease: a detailed case description and history of illness recorded by Alzheimer, containing repeated observation by (the same) trained and experienced clinician using recognized assessments, and a pathological examination at autopsy. Nevertheless, there have been questions raised about the diagnosis of Auguste D. recently, with speculation that she may have been suffering from dementia with a different aetiology. Amaducci and co-workers (1991, 1996) have recently suggested that metachromatic leucodystrophy might be the cause. O’Brien (1996) hypothesized that vascular dementia was the source of Auguste D.’s behavioural disorder. These recent debates appearing in the journal Science echo questions raised by descriptions of this brain by Alzheimer’s contemporaries. In his 1907 publication, Alzheimer did note that, along with generalized atrophy, the larger blood vessels were altered by arteriosclerosis. However, later reports on the same brain clearly state that there were no signs of vascular disease (Graeber and Mehraien, 1999). Graeber and co-workers (1998) verified both the absence of vascular pathology and markers for metachromatic leucodystrophy. Thus, Alzheimer’s original diagnosis, which had been called into question, was retrospectively confirmed by modern pathological examination of the brain.

In other instances of retrospective diagnosis, the discussion about a case reveals more about the varying status of confirming evidence, rather than the debated nosological category. This paper considers the final illness of the satirical writer Jonathan Swift (1667–1745). For over 250 years, the diagnosis of the changes in his cognitive abilities and social behaviour has been debated by the leading clinicians of the day. In addition, the description of behavioural changes seen in ageing that appears in Swift’s *Gulliver’s Travels* (1726) has been the source of interest to modern neurologists.

Recently, a series of letters on dementia as described in *Gulliver’s Travels* appeared in the *Lancet*. There is argument about what Swift’s immortals, the Struldbruggs, are suffering from—Alzheimer’s disease or Pick’s disease. One author raised the question of whether this might not also apply to Swift’s last years as well. Lewis (1993) presents Swift as the archetypical example of Alzheimer’s disease:

Swift’s last years were marked by protracted bouts of walking, a progressive aphasia, and the inability to recognise anyone . . . to any alert clinician of the 1990’s, the signs and symptoms of Alzheimer’s disease are clear . . . Ralph Waldo Emerson and Jonathan Swift were probable Alzheimer’s sufferers and wrote about their advancing conditions with great poignancy and insight . . .

*Gulliver’s Travels* was written by Swift when he was 59 years old. In Chapter X, Gulliver meets the Luggnaggians, among whom lives the immortal race of Struldbruggs. These individuals are destined never to die, but suffer the ravages of age and infirmity. Gulliver assumed these people must be particularly wise, given their decades of learning and cultivation. In fact, they were socially isolated and depressed, exhibiting all the negative consequences of extreme senescence:

. . . [the Struldbruggs] commonly acted like Mortals, till about thirty Years old, after which by degrees they grew melancholy and dejected, increasing in both till they came to four-score . . . When they came to fourscore Years, which is reckoned the Extremity of living in this Country, they had not only all the Follies and Infirmities of other old Men, but many more which arose from the dreadful Prospects of never dying . . . Opinionative, Peevish, Covetous, Morose, Vain, Talkative, but incapable of Friendship, and dead to all natural Affection . . . They have no Remembrance of any thing but what they learned and observed in their Youth and middle Age, and even that is very imperfect . . . AT Ninety . . . they have at that age no Distinction of Taste, but eat and drink whatever they can get, without Relish or Appetite . . . In talking they forgot the common Appellation of things, and the Names of Persons, even of those who are their nearest Friends and Relations. For the same reason they never can amuse themselves with reading, because their Memory will not serve to carry them from the beginning of a Sentence to the end . . . neither are they able . . . to hold any conversation (farther than a few general words) with their neighbours . . . THEY are deprived and hated by all sort of People . . . the usual way of computing how old they are is by asking them what Kings or great Persons they can remember, and then consulting History, for infallibly the last Prince in their Mind did not begin his Reign after they were Fourscore Years old . . . THEY were the most mortifying Sight I ever beheld . . .

Lewis (1993) suggested that this portrait of senility is drawn from Swift’s childhood observation of his uncle Godwin, who is said to have suffered memory loss with ageing. Lewis goes on to assert that Swift was being prescient about his own final mental state:

Anyone acquainted with Alzheimer’s disease will recognize a familiar ring in words written well over two centuries ago . . . Since it seems unlikely that such a picture could have been drawn other than from reality, it is probable that Swift was describing in 1726 what we now know as Alzheimer’s disease . . . It is shocking to realise that Swift may have been afflicted with the same disease himself . . . Swift’s last years were marked by protracted bouts of walking, a progressive aphasia and the inability to recognize anyone. In 1952 it was postulated that his illness was the result of ‘cerebral arteriosclerosis’ and ‘involu-
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clinician of the 1990’s, the signs and symptoms of Alzheimer’s disease are clear.

This article prompted a letter to the editor of the Lancet by Crichton (1993). He states that Lewis’s suggestion that Swift died of Alzheimer’s disease himself 20 years after first describing its clinical features is ‘imaginative, but probably untrue’. Crichton argues that,

Although Swift is clearly describing the signs and symptoms of a dementing process, the prominent and early personality changes and absence of cortical deficits typical of Alzheimer’s disease, such as apraxias and visuospatial disorientation, makes Pick’s disease… seem more likely.

Boller and Forbes (1998) also reviewed references to the passage describing the Struldbriggs. Of this description, they note, ‘The diagnosis inferred by astute readers has varied according to the times.’ They suggest that in the 19th century this might have been identified as ‘General Paresis of the Insane’ or ‘Neurosyphilis’, while in the mid 20th century the diagnosis would have been arteriosclerotic dementia. The authors also refer to the discussions of Lewis (1993) and Crichton (1993), mentioned above, regarding Alzheimer’s disease and Pick’s disease. Boller and Forbes offer, ‘Perhaps one could go further and speculate on the diagnosis of Primary Progressive Aphasia…’ (Boller and Forbes, 1998, p. 128) as the description of the disease of the Struldbriggs, but they concur with Lewis’s diagnosis of Alzheimer’s disease for Swift himself.

One of the most celebrated writers of political satire of his age, Jonathan Swift was concerned with psychological, psychiatric and linguistic themes throughout his literary career (Lorch, 2006b). He was, in turn, the focus of both lay and professional interest regarding his mental state throughout his life, and for centuries after his death, speculation continues. Born in 1667, he suffered lifelong illness, which included intermittent dizziness, nausea and deafness (subsequently identified as Ménière’s disease), and his personality was notorious for being both irritable and voluble. However, the focus of the present paper is the final years of his life, about which there was talk of his ‘madness’, referring not only to his political views, but also to his personal behaviour.

Today, Jonathan Swift is perhaps best remembered as the literary author of Gulliver’s Travels (1726) but he also held the influential position as Dean of St Patrick’s Cathedral, Dublin from 1713 to the time of his death in 1745. Swift was generally interested in the workings of the body in health and disease as well as social issues (e.g. A Modest Proposal, 1729). His own state of health figured heavily in his letters and writings throughout his life, and he had many close friends who were eminent physicians in London and Dublin including John Radcliffe, John Arbuthnot, James Grattan and Richard Helsham (Le Fanu, 1978). Swift’s writings reflect a lifelong interest in how people spoke, thought, behaved and reasoned (Lorch, 2006b). However, Swift himself exhibited behaviour during the final 3 years of his life that has been the cause of much speculation among biographers and medical historians for the past 250 years since his death.

The most recent opinion on Jonathan Swift was put forward in 1999 by Lawlor. In a meeting of the Irish Heart Foundation Council on Stroke, he gave a paper on depression following stroke. Lawlor presented the behaviour of Jonathan Swift as a clear illustration of this symptom:

His rage increased to a degree of madness in a miserable state and the first proper inhabitant of his own hospital. He sank into a quiet speechless condition and dropped out of the term of his life in this helpless state… It appears that Dean Swift suffered a stroke and subsequently post stroke depression. Post stroke depression is not a new phenomenon, having been first described in 1752.

In this, Lawlor (1999) was not a careful historian however. Swift was not in fact an inmate at the lunatic asylum that he helped to found, nor could he possibly have been. Swift died in 1745, while St Patrick’s Hospital only opened 3 years later.

This issue celebrates the case Alzheimer presented at a meeting of the South-West German Society of Alienists in November 1906. Alzheimer described ‘eine eigenartige Erkrankung der Hirnrinde’ (a peculiar disease of the cerebral cortex)—the clinical and neuropathological features of a woman referred to as Auguste D. The clinical features were of a 5-year history of mental decline with progressive difficulty in personal care, disorientation, impaired memory, as well as, troubles in reading and writing (Maurer et al., 1997). These symptoms echo those that have been recorded for the final years of Jonathan Swift.

While definitive histopathology is available for Auguste D., thereby ending speculation (Graeber et al., 1998), the cause of Jonathan Swift’s cognitive decline cannot be verified. There is little evidence available of either detailed observational records of his behavioural changes, or pathological evidence subsequent to his death. The evidence that does exist—letters, autopsy report, death mask, skull and brain cast—are tantalizing but indeterminate. However, this has not prevented physicians and medical historians from considering various retrospective diagnoses for Swift since his death in 1745. These successive interpretations are critically reviewed in the present paper. They chart over two centuries of developing knowledge in neurology regarding memory and language disorders, affective changes in the elderly and the impact of chronic disease on mental function.

The mind of Jonathan Swift

Evidence taken as revealing the mind of Swift can be taken from a variety of sources. In the various commentaries that have been produced have drawn upon: (i) self-report in Swift’s letters; (ii) letters of family and acquaintances
describing visits with Swift; (iii) paintings and busts made at different stages of his life; (iv) The report of the autopsy at his death; and (v) subsequent examinations of his death mask, skull and brain cast.

That Swift suffered 50 years of intermittent illness, which had as its major features deafness, vertigo and nausea, is well documented in Swift’s own writings. His letters are filled with descriptions of these symptoms throughout his life. This chronic and progressive illness was the focus of the medical treatments that he received from various physicians throughout his life. Swift himself attributed his illness to diet. He believed that a ‘surfeit of green apples’ was the original cause, and refused to eat fruit for the rest of his life. His doctors prescribed various remedies, e.g. Peruvian bark, which provided little relief from his symptoms.

A description of vertigo by Clutterbuck (1827) that appeared in a prominent 19th century British medical encyclopaedia cited Swift as the prototypical case. A direct causal link is drawn between the vertigo and cognitive decline:

Vertigo is apt to recur, and thus often becomes frequent and habitual. After a time the mental powers become impaired, and complete idiocy often follows; as was the case in the celebrated Dean Swift. It frequently terminates in apoplexy or palsy, from the extension of disease in the brain.

Victorian physicians subsequently identified Swift’s complaint as Ménière’s disease (Lecky, 1861). Present day sufferers of Ménière’s disease report difficulties in cognitive function, and the mistaken fear that they have Alzheimer’s disease. The history given by ‘Patient 4’, a female, age 32, with a 22-year history of symptoms on a patient information website for this disease posted by The Department of Otolaryngology, Washington University School of Medicine in St Louis is a clear example:

You can get confused easily and your memory and concentration aren’t reliable. It’s what some people with the disease refer to as ‘brain fog’. Many of them originally were afraid that they may have a brain tumor or Alzheimer’s because it can sometimes gets so bad. …Now try to imagine living with this disease never knowing when one of these periods of tinnitus, vertigo, hearing loss, double vision, lack of coordination, recruitment, disequilibrium, or ‘brain fog’ is going to hit, or how bad it will be…. Understandably, anxiety and depression seem to go hand-in-hand with Ménière’s for many sufferers. (http://oto.wustl.edu/men/mn1.htm)

Swift wrote a surprising poem, composed and revised over a long period of time, in which he specifically refers to the symptom of memory loss in conjunction with vertigo. In the ‘Verses on the Death of Dr Swift Written by Himself’ finally published in 1731, when he was 64 years old, he includes the stanzas:

For poetry he’s past his prime
He takes an hour to find a rhyme…
See how the Dean begins to break
Poor gentleman, he droops apace,
You plainly find it in his face;
That old vertigo in his head
Will never leave him till he’s dead;
Besides, his memory decays,
He recollects not what he says.
He cannot call his friends to mind;
Forgets the place where last he dined;
Plies you with stories o’er and o’er;
He told them fifty times before.

While these protests about difficulties in word finding and memory have typically been viewed as reflecting cognitive changes with ageing, a close review of Swift’s early letters, some written in his 20s when his vertigo and deafness were first recorded, also contain similar complaints.

Swift’s later years
In 1740, now aged 73, Swift (Fig. 1) made a will which testified him to be ‘of sound mind although weak in body.’ The soundness of not only his mind, but also his linguistic faculty, is attested by this sample from a letter written at age 75 on church business:

… I do further intreat my… Chapter that, the infirmaties of age disabling me to attend personally and take the care
which I have much at heart, they will keep up the honour and dignity of the Chapter, and not let it suffer in its rights and privileges by the encroachments of any persons nor by the neglects, disobedience, or perfidy of those who are subject to it... 

This is the final piece of writing documented to have been produced by Swift. Although he lived for five more years, there are no other literary works known to have been written by him.

Five months after making his will, Swift was found by the courts to be ‘not capable of taking care of his person or fortune’. His estate was valued at £10,000; an enormous sum at that time (Malcolm, 1989). Later that year, friends’ letters record that Swift suffered an eye disorder. This was vividly described in subsequent biographies as a turning point in Swift’s health. His quality of life after recovering from the acute infection is described by Samuel Johnson in 1781 (i.e. 36 years after Swift’s death):

Next year (1742), he had an inflammation in his left eye... The tumour at last subsided; and a short interval of reason ensuing, in which he knew his physician and his family, gave hopes of his recovery; but in a few days he sunk into lethargick stupidity, motionless, heedless, and speechless. But it is said, that, after a year of total silence, when [told it] was his birth-day, he answered, It is all folly; they had better let it alone. It is remembered that he afterwards spoke now and then, or gave some intimation of a meaning; but at last sunk into perfect silence, which continued till about the end of October, 1745, when, in his seventy-eighth year, he expired without a struggle.

A later Victorian biographer, William Lecky (1861), described the episode thus:

At length the evil day arrived. A tumour, accompanied by excruciating pain, arose over one of his eyes. For a month he never gained a moment of repose. For a week he was with difficulty restrained by force from tearing out his eye. The agony was too great for human endurance. It subsided at last, but his mind had wholly ebbed away. It was not madness; it was absolute idiocy that ensued. He remained passive in the hands of his attendants without speaking, or moving, or betraying the slightest emotion. Once, indeed, when someone spoke of the illuminations by which the people were celebrating the anniversary of his birthday, he muttered, ‘It is all folly; they had better leave it alone.’ Occasionally he endeavoured to rouse himself from his torpor, but could not find words to form a sentence, and with a deep sigh he relapsed into his former condition.

Compare this with the description given by Henry Craik in 1892:

... it was in 1736, while he was engaged on a poem satirising the Irish Parliament... that his illness, in its final and most crushing form, overtook him. A few years more of almost unbroken gloom were left to Swift: but his state at last fell into one of utter helplessness and isolation, interrupted by fits of frenzy and violence, and by attacks of terrible agony of pain. Finally, he sank into absolute mental apathy: and death released him only on the 19th of October, 1745.

Some biographers seemed to use visual evidence from portraits of Swift painted at various ages for their diagnosis of the change in his last years. For example, Lecky (1861) offered this description of his last portrait: '[Swift’s] harsh [facial] expression had passed away... every spark of intelligence had disappeared. It was not till he had continued in this state for two years that he exchanged the sleep of idiocy for the sleep of death.'

All of these descriptions are based on remarkably little documentary evidence. There are few observations drawn from contemporary eyewitnesses of Swift’s mental and physical state during the last 3 years of his life. All that can be identified as pertinent to this question is contained in two letters written by and to family or members of the household. These very same people had a hand in taking over control of his finances and the running of the church. The passages that discuss Swift’s language and behaviour are given below:

He would attempt to speak his mind, but could not recollect words to express his meaning.

... [He] endeavoured, with a good deal of pain, to find the words to speak to me; at last, not being able after many efforts, he gave a heavy sigh, and, I think, was afterwards silent.

Sometimes he will not utter a syllable, at other times he will speak incoherent words; but he never yet as far as I could hear, talked nonsense, or said a foolish thing.

As far as this author can ascertain, they are the only documentary evidence of Swift’s behaviour as reported by eyewitnesses published to date. No records have been found from any attending physician. Swift was under the care of the recently qualified Dr John Whiteway. He was the nephew of Swift’s housekeeper and cousin, Mrs Whiteway. Swift had paid for John Whiteway’s medical training at Steevens’ Hospital where he was a governor. Swift had outlived all of his other, more illustrious, physician friends.

His death was reported in the Gentleman’s Magazine in October 1745:

At three o’clock in the afternoon dyed the great and eminent patriot the Rev. Dr. Jonathan Swift, Dean of St Patrick’s, Dublin, in the 78th year of his age, whose genius, works, learning and charity are universally admired. He bequeathed the bulk of his fortune, which is about £12,000 to build and endow an hospital for lunatics, idiots and incurables...

An autopsy was performed by John Whiteway, and a death mask was cast. Upon opening the skull, the brain was found to be ‘loaded with water’. Swift was buried in St Patrick’s Cathedral and the commemorative marker bears a Latin epitaph that he wrote for himself.
18th century madness

In the 18th century, the concepts of madness and rationality were a major social concern of the day. Thus, it should not be surprising that Swift’s behaviour in his last years were described by his many biographers as reflecting madness, insanity and imbecility in old age. For example Samuel Johnson (1781) wrote:

...his ideas, therefore, being neither renovated by discourse, nor increased by reading, wore gradually away, and left his mind vacant to the vexations of the hour... till at last his anger was heightened into madness... He now lost distinction. His madness was compounded of rage and fatuity.

Sir Walter Scott (1814) described Swift’s behaviour during his final years as marked by ‘violent and furious lunacy’; ‘frantic fits of passion’; ‘[having the] situation of a helpless changeling’. However, these descriptions can be seen as reflections of the writers’ own concepts and beliefs about mental illness as well as their feelings towards their subject. It is essential to stress at this point that while Samuel Johnson (1709–1784) was living at somewhat the same time as Swift, Scott (1771–1832) was born almost three decades after Swift’s death. Both of their biographies are based on extracts of letters and reminiscences, rumours and hearsay handed down over decades. They had in fact never met Swift, and were openly declared his political and literary rivals (Anonymous, 1883).

In the first half of the 19th century, Swift became the focus of a serious debate about the methodology employed by phrenology, which was played out in the pages of the Lancet (Lorch, 2006a). An examination of Swift’s skull was carried out in 1835, under the auspices of the British Association for the Advancement of Science meeting held in Dublin that summer. There was great phrenological interest in his case. The chief medical officer (Houston, 1835) reported:

It would appear from the depression on the anterior part of the head that the man must have been apparently idiot. The bones must have undergone considerable change during the 10 or 12 last years of his life, while in a state of lunacy.

The illness of Swift’s final years was next considered by Dr William Wilde, renowned Dublin eye specialist and father of Oscar. Prompted by a letter from a colleague inquiring about the nature of Swift’s reported eye infection, Wilde (1849) wrote a long journal article that was reprinted as the book The Closing Years of Dean Swift’s Life. Wilde pointed to several sources for his final illness: ‘overloading the stomach’, ‘catching cold by sitting on a damp exposed seat’, ‘a system so nervous’, ‘a temper so irritable’ and ‘a mind so excessively active’. He concluded that these factors ‘...led to cerebral congestion with periodic attacks which increased in duration and intensity throughout his life.’

Wilde considered Swift’s death mask (Fig. 2) as providing significant evidence of motor weakness and the signs of chronic eye infection: ‘There is an evident drag in the left side of the mouth, exhibiting a paralysis of the facial muscles on the right side... The left eye... is comparatively sunken and collapsed within the orbit.’ His final diagnosis of Swift’s last illness was:

Exophthalmus of the left eye... produced by an internal abscess, or intense inflammation of the anterior lobe... by producing effusion, &c., destroyed his memory, and rendered him at times un governable in his anger, as well as produced paralysis... That his not speaking was not the result either of insanity or imbecility, but arose either from paralysis of the muscles by which the mechanism of speech is produced, or from loss of memory of the things which he wished to express, as frequently occurs in cases of cerebral disease...

Wilde was at pains to dispel the impression created by Swift’s earlier biographers that he was insane:

... neither in his expression, nor the tone of his writing, nor from an examination of any of his acts, have we been able to discover a single symptom of insanity, nor aught but the effects of physical disease, and the natural wearing and decay of a mind such as Swift’s.

Thirty-five years later, Dr John Charles Bucknill was also prompted to reconsider the illness of Swift by a letter from a curious medical colleague. Bucknill had been the founding editor of the Asylum Journal of Mental Science (later to become the Journal of Mental Science and now the British Journal of Psychiatry) in 1853 and of Brain in 1879. He was one of Britain’s leading experts in mental diseases in the 19th century. Bucknill (1882) reviewed the evidence of Swift’s autopsy, and the arguments put forward by Wilde, in light of the developments that had taken place in the understanding
of the cortical organization of motor and language functions in the intervening years:

At the opening of the skull there was much water in the brain, which was probably subarachnoid effusion, is sufficient evidence of dementia . . . . The knowledge of the importance of [the right facial paralysis] has been acquired since Sir William Wilde wrote his work, and it is therefore not surprising that . . . he does not connect the right sided hemiplegia with the very peculiar affection of speech recorded by . . . the authentic witnesses . . . It is enough now that we can diagnose . . . his insanity as dementia with aphasia. The dementia arising from general decay of the brain from age and disease, the paralysis and aphasia from disease of one particular part of the brain.

At the end of the 19th century, Dr William Osler (1892) chose to use Jonathan Swift’s final illness to illustrate one of his sections in his textbook Principles and Practice of Medicine.

Dean Swift is said to have died of hydrocephalus, but this seems very unlikely. It is based upon the statement the ‘he (Mr. Whiteway) opened the skull and found much water in the brain,’ a condition no doubt of h. ex vacuo, due to the wasting associated with his prolonged illness and paralysis. In nearly all cases there is either a tumor at the base of the brain or in the third ventricle, which compresses the venae Galeni. The passage from the third to the fourth ventricle may be closed, either by a tumor or by parasites. More rarely the foramen of Magendie, through which the ventricles communicate with the cerebrospinal meninges, becomes closed by meningitis.

A question on a humorous examination paper based on Osler’s textbook was published in the St. Thomas’s Hospital Gazette (London) in 1902 and reprinted in American Medicine, 1902: ‘Who made an autopsy on Dean Swift and what did he report?’

Forty years later, the leading Irish medical historian T. G. Wilson (1940) again re-examined the evidence accounting for the behaviour exhibited by Swift during his final years of life. His is an excellent example of a rigorous scientific approach to artefacts as evidence, which can provide an interesting, 20th century juxtaposition to Wilde’s scientific approach to artefacts as evidence, which can provide an interesting, 20th century juxtaposition to Wilde’s ‘fitts’ and subsequent death were complications of uremic poisoning, and that the water on the brain found at autopsy was due to cerebral atrophy. Dale concluded that Swift’s fits and subsequent death were complications of uremic poisoning, and that the water on the brain found at autopsy was due to cerebral atrophy. Dale also was the only biographer writing after the 1860s who challenged the diagnosis of Ménière’s disease. He attributed Swift’s auditory and vestibular symptoms to allergy rather than Ménière’s disease:

. . . recently described by Drs. Sheldon and Horton of the Mayo Clinic. These workers have established that recurrent episodes of acute vertigo, ringing in the ears and sudden deafness are commonly due to histamine sensitisation (allergy). . . .

In the same year, the English neurologist Sir Walter Russell Brain received an Honorary Fellowship from the Royal College of Physicians of Ireland. He chose as the topic of his formal address ‘The illness of Dean Swift’. Brain does not take into account Wilson’s (1940) measurements of the death mask, instead returning to the 19th century opinion that it shows evidence of paralysis of the right face and sunken left eye. Brain based his insights into Swift’s personality and social behaviour on the highly biased
cases of motor aphasia were quite well known in the 18th century. One well-documented case was the famous English satirist Jonathan Swift (1667–1745), who was almost completely mute during the last years of his life despite the fact that he seemed to understand everything that was said to him. His only known utterances occurred in emotion-laden situations... Never once during his illness was Swift able to utter an ordinary declarative sentence.

Discussion

These various attempts to re-diagnose Swift’s final mental state with the re-examination of his letters, portraits, skull, death mask and brain cast reveal much about the changing issues of interest to clinicians and theorists regarding interpretations of behaviour and cognition from the 18th century to the present day. Sir Walter Scott (1814) makes this insightful point on declaiming the suggestion that Swift died of tertiary syphilis:

‘...I do not believe myself to be a laborious dry writer, because if the fit comes not immediately I never heed it but think of something else...’

Swift repeatedly renegated his ability to write ‘What I writ was not worth transcribing to you... but igad I can not write anything easy to be understood...’ In a letter to Miss Jane Waring dated 29 April 1696 he is also seen to make self-deprecating excuses: ‘If you will pardon the ill hand and spelling, the reason and sense of it you will find very well and proper’. In a letter to Archbishop King, dated 6 January 1709, Swift says that he is delayed in replying due to a ‘cruel distemper, a giddiness in my head, that would not suffer me to write or think of anything and of which I am now recovering’. Again writing to Archbishop King on 8 March 1710: ‘I have read over what I writ, and find it confused and incorrect which your Grace must impute to the violent Pain of Mind I am in, greater than ever I felt in my Life.’ In this letter, it might be suggested that Swift is describing emotional rather than physical pain, as Swift is recounting...
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The value of considering the various retrospective diagnoses offered for Jonathan Swift is not in trying to determine a definitive attribution (for this is ultimately impossible), but rather to throw light on the argumentation proposed by those various writers and seen as reflections of their historical period. Their concepts of behaviour, illness and ageing are revealed through the manner in which they interpreted the evidence afforded by Swift’s case. This critical review illuminates the style of reasoning that was employed, and provides examples of what was considered compelling argumentation for explaining behaviour at a given period. In this way, examining a line of retrospective diagnoses can be of value (Arrizabalaga, 2002; Karenberg and Moog, 2004). It may serve as a kind of projection test, much like a Rorschach...

On the use of retrospective diagnosis

The intention of the preceding review of the case of Jonathan Swift is to bring into relief the boundary shifts and re-mapping of the terrain of mind, thought, language and memory. It provides an historical perspective to the taxonomic distinctions of affect, mood and emotion, rationality and mental illness. This paper plots the changes in attribution of signs and symptoms as evidence, and their significance in changing nosologies. This author’s conclusion is that the evidence that exists for Swift’s final years cannot provide an adequate basis for diagnosis to current-day practitioners. Subtle qualitative analysis of elicited behaviours, and quantitative techniques of standardized assessment are now employed for making crucial diagnostic distinctions. Modern imaging, molecular genetics, histology and other techniques for determining pathology are now considered crucial to determining diagnosis.

Unlike Alzheimer’s case of Auguste D., there is no record of any medical assessment made during this period Swift’s life, nor was there any detailed pathological examination at the time of his death. No determination of the illness of Jonathan Swift can ever be reached. This is necessarily due to the limitations of the documentary evidence that exists. There were no recorded observations by attending physicians, no detailed descriptions of behavioural testing, no detailed history of illness for the crucial period, no samples of speech, writing, or reading, no evidence of memory abilities or activities of daily living. There is little value in the autopsy report, as there was no direct inspection of the brain but only opening of the skull case. The only physical evidence that exists is the death mask, skull and perhaps the brain case. These objects cannot provide information about the aetiology of any neuropathology. It must be concluded that what is required for the determination of a differential diagnosis is unobtainable. We cannot even be confident of accurately labelling the behavioural symptoms Jonathan Swift displayed in his final years. Were these symptoms of aphasia, depression, bradykinesia, or merely the natural result of chronic disability and social isolation?

This analysis of how clinicians considered the evidence available for Jonathan Swift’s final years provides a systematic review of changing diagnostic methods—from lay observations provided by caretakers and friends, to descriptions by trained physicians. More specialist labels and categories were employed with the development of nosological systems in the 19th century. This approach was refined by the use of clinical–pathological correlation methods, in which the co-occurrence of behavioural symptoms was linked to examination at autopsy from the late 19th century onwards.

The value of considering the various retrospective diagnoses offered for Jonathan Swift is not in trying to determine a definitive attribution (for this is ultimately impossible), but rather to throw light on the argumentation proposed by those various writers and seen as reflections of their historical period. Their concepts of behaviour, illness and ageing are revealed through the manner in which they interpreted the evidence afforded by Swift’s case. This critical review illuminates the style of reasoning that was employed, and provides examples of what was considered compelling argumentation for explaining behaviour at a given period. In this way, examining a line of retrospective diagnoses can be of value (Arrizabalaga, 2002; Karenberg and Moog, 2004). It may serve as a kind of projection test, much like a Rorschach...
longevity. In Swift’s day, life expectancy averaged 40 years, O’Brien (1996) suggested that Alzheimer’s original patient Auguste D. may have had vascular dementia, and Amaducci (1991, 1996) proposed the diagnosis of metachromatic leucodystrophy. While the original case notes bring detail to the behavioural description of the disorder, the definitive answer to this speculation has been successfully determined by Graeber and colleagues (1998) with the publication of the histopathology of the original slides. In the case of Jonathan Swift, there has also been renewed speculation regarding retrospective diagnosis. However in his case, this review concludes that there is no behavioural or pathological evidence available that could be employed to determine a definitive answer.

It is useful to compare the behavioural description offered by Alzheimer with what is known of Swift. While Auguste D. was in her early 50s when she showed evidence of cognitive decline, Swift was already well into his late 60s before any signs of difficulty were documented. The comparison between these two cases also raised the issue of ageing. The relative youth of Auguste D. was significant for Alzheimer. He emphasized its ‘pre-senile’ occurrence, which allows us to plot changing concepts on diseases of language and memory in ageing over a 250-year period.

Conclusion

There has been recent discussion of the retrospective diagnosis of Alzheimer’s original case Auguste D. published 100 years ago. O’Brien (1996) suggested that Alzheimer’s original patient Auguste D. may have had vascular dementia, and Amaducci (1991, 1996) proposed the diagnosis of metachromatic leucodystrophy. While the original case notes bring detail to the behavioural description of the disorder, the definitive answer to this speculation has been successfully determined by Graeber and colleagues (1998) with the publication of the histopathology of the original slides. In the case of Jonathan Swift, there has also been renewed speculation regarding retrospective diagnosis. However in his case, this review concludes that there is no behavioural or pathological evidence available that could be employed to determine a definitive answer.

It is useful to compare the behavioural description offered by Alzheimer with what is known of Swift. While Auguste D. was in her early 50s when she showed evidence of cognitive decline, Swift was already well into his late 60s before any signs of difficulty were documented. The comparison between these two cases also raised the issue of ageing. The relative youth of Auguste D. was significant for Alzheimer. He emphasized its ‘pre-senile’ occurrence, arguing that the behaviours associated with senility could occur at a younger age. For Alzheimer, this was evidence in support of a continuity model of ageing (Cheston and Bender, 2003). Age is now considered a major factor in developing Alzheimer’s disease that is linked to current longevity. In Swift’s day, life expectancy averaged 40 years, while he did not show signs of memory or language difficulties until he reached his 70s. The poem ‘When I come to be old’ written by Swift in 1699, when he was 32 years old, contains the wish to avoid certain behaviours that he believed to be typical failings in the elderly, including ‘...Not to be peevish or morose, or suspicious... Not to tell the same story over and over to the same People... Not to neglect decency, or cleanness, for fear of falling into Nastyness...’. It is interesting that none of Swift’s biographers attributed his perceived changes in behaviour simply to ageing. However, as early as 1685, Willis recognized that some people ‘become by degrees dull...by the mere declining of age’. Towards the end of the century, Cullen (1776) first classified senile dementia as a medical entity, amentia senilis (cited in Roma´n, 1999).

Current considerations of dementia include questions about the relative significance of non-organic contributory factors such as sensory depravation due to institutionalization. Cheston and Bender (2003) point out that there is little information on Auguste D.’s pre-morbid history, or details about the onset of illness. These factors may also be relevant in Swift’s case, but have not been considered in any of the retrospective diagnoses. Nor has there been any discussion of the effect of chronic disability from his intermittent vertigo and deafness on Swift’s mental status. The symptoms attributed to Swift—cognitive changes, memory impairment, personality alterations, language disorder and facial paralysis—have all been apportioned differing levels of significance by various writers in the 18th, 19th and 20th centuries. The evidence that exists must be deemed inadequate today for the determination of any one of the many competing hypotheses. That being said, there can be great value in the discussion of the way in which different signs and symptoms were given status and significance by different writers at different historical periods. What was previously considered as evidence typical of ageing has been increasingly redefined through the development of medical knowledge (Cunningham, 2002). Personality, social context, and stress factors are all now considered relevant to explanations of alterations in a person’s behaviour patterns. The case of Jonathan Swift allows us to plot changing concepts on diseases of language and memory in ageing over a 250-year period.

References

Memory, ageing and Swift’s brain


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