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Autistic disorder in nineteenth-century London

Three case reports

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ABSTRACT This article examines the existence, description, perception, treatment, and outcome of symptoms consistent with autistic disorder in nineteenth-century London, England, based on case histories from the notes of Dr William Howship Dickinson at Great Ormond Street Hospital for Children. Three cases meeting the DSM-IV criteria for autistic disorder are described in detail. Other cases in which autistic traits are described are briefly summarized. The article explores the environment of contemporary medical practice, beliefs about childhood brain disorders, and social practice regarding children with brain disorders, and the impact of these factors on assessment and treatment. It correlates Dickinson's observations with current research on autism, providing information about children with autism before the condition was formally named in 1943.

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Introduction and methodology

This article examines medical practice, beliefs about childhood brain disorders, and social practice as applied to a small sample of children with autism in nineteenth-century England.

Case notes by Dr William Howship Dickinson, Great Ormond Street Hospital for Children, were compared with DSM-IV criteria for autistic disorder. Three cases that met these criteria were examined in detail, and others were summarized. Information on these cases was examined in light of current research on autistic spectrum disorders.

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Historical background

In 1852, a spacious townhouse in London was converted into a hospital for impoverished children with serious illness or injury. Founded with beds for only 10 patients, the Great Ormond Street Hospital for Children expanded rapidly. By 1858 it housed 75 children in two wings; a larger purpose-built facility was constructed between 1871 and 1875 (Baldwin, 2001).

Previously, there had been no paediatric hospital provision in England, so only a handful of children were ever admitted to hospital for inpatient care (Baldwin, 2001). Poor and working-class children were cared for at home by family members. Wealthier families could procure the services of 'nurses' (generally women with no formal medical training) and home visits from physicians (Hopkins, 1994, pp. 113–16).

Great Ormond Street Hospital for Children emerged as a favourite cause among the upper classes of Victorian London society, its most famous patron being Charles Dickens. As a charitable institution, it turned no child away for lack of money. The great majority of children in London lived in poverty at this time, leaving lack of information, inclination, transportation, and time as the main barriers to hospital treatment for those in need. Both outpatient and inpatient care were available at Great Ormond Street (Baldwin, 2001).

Dr William Howship Dickinson (1832–1913) served at Great Ormond Street as an assistant physician from 1861 to 1869, and as a physician from 1869 to 1874. This work was performed on a voluntary basis, in addition to his primary service at St George's Hospital, where he progressed through a series of posts following his graduation from Caius College, Cambridge, in 1859 (Royal College of Physicians, 1955, pp. 144–5).

While best known for his accomplishments in adult nephrology, Dickinson developed a keen interest in the effects of ill-health on brain function and behaviour in children. In the absence of a codified system of diagnosis or treatment, he relied on his prodigious powers of observation: 'He was a meticulously careful observer and a man of immense industry . . . and his example, in the elicitation of patients' histories and methodological observation, probably had a permanent influence on the crowds of students that invariably accompanied him' (Royal College of Physicians, 1955, pp. 144–5).

Three volumes of Dickinson's handwritten case notes, dictated to nurses or assistants, have been bound for Great Ormond Street Hospital's Museum and Archives. The collection of unnumbered papers describes 398 children with a wide variety of conditions affecting neurological function. These include 24 cases in which children presented with symptoms characteristic of autistic spectrum disorders (Dickinson, 1869–82).

Dickinson generally saw autistic symptoms as secondary to other conditions, particularly epilepsy (eight cases) or infectious illness with seizures (four cases), but also infectious illness without known seizures (one case), physical symptoms consistent with cerebral palsy (four cases), and symptoms consistent with Tourette syndrome (one case). This is consistent with modern findings, although the terminology Dickinson used to describe primary conditions is that of the Victorian era. Bailey et al. (1996) produced a comprehensive review of the literature on autism and comorbid medical disorders, establishing that seizure disorders occur in up to one-third of persons with autism, and that the rate of cerebral palsy is also elevated in this population. Baron-Cohen et al. (1999) cite comorbidity of Tourette syndrome and autistic spectrum disorders at 4.3 to 6.4 percent.

In seven cases described by Dickinson, autistic symptoms appear to have been the primary reason for admission, and the focus of any diagnostic efforts and treatment. Four sets of case notes, for two boys and two girls, do not present a picture of symptoms that is clear enough to warrant inclusion in this article. All cases described in which onset of symptoms occurred after 36 months of age were also excluded, as required by current diagnostic criteria. Onset of symptoms after age 3 has been observed and remarked upon by expert practitioners, however, particularly when symptoms follow a known illness (Wing, 1996, pp. 62–3).

Two descriptions, both of male children, fit the DSM-IV criteria for autistic disorder. A third, for a female child, also fits DSM-IV criteria, but her clinical picture includes a comorbid diagnosis of epilepsy. Because epilepsy is common among children with autism, this case has also been included, although it differs from the other two in several respects.

The existence of three relatively clear-cut cases of autism and numerous descriptions of autistic symptoms within a set of nineteenth-century medical notes will not surprise veteran researchers, many of whom have mentioned pre-1943 cases that are consistent with autism (Frith, 1989; Wing, 1996). However, autism was not formally defined and named until 1943, and was believed for decades to be a novel, unusual, and rare condition. Kanner (1943) himself called it a ‘new syndrome’.

By the 1960s, autism prevalence was calculated as 4–5 children per 10,000, and it continued to be considered a rare disorder (Lotter, 1966). Forty years later, conditions on the autistic spectrum are among the most common childhood neurological disorders, with prevalence statistics as high as 1 in 160 children (Chakrabarti and Fombonne, 2001). Whether there has been an actual increase in the number of children with autistic spectrum disorders or simply improved recognition of these conditions is a matter of current debate (Gillberg and Wing, 1999; Shattock et al., 2001).

The cause of autism is still unknown, although research indicates that

it is a multigenic disorder in which a varied combination of genetic differences is expressed in different levels of severity, perhaps influenced by environmental factors (Fombonne, 1998). Diagnosis is performed by using formal test instruments, observations, and interviews assessed according to published criteria: either those set out by the World Health Organization's ICD-10 (1992), or those in the DSM-IV, published by the American Psychiatric Association (1994; revised 2000).

Working in the Victorian era, Dickinson could not rely on peer-reviewed diagnostic aids. Following the path of contemporary generalist investigators, such as Sir William Osler, he simply observed, asked questions, shared what he saw in the form of case notes, and occasionally recommended treatment based on empirical evidence.

Dr Dickinson dictated meticulous case notes for his neurology patients at Great Ormond Street Hospital between 1869 and 1882. These writings were based on his own observations, reports by nurses and other hospital personnel, and sometimes interviews with parents or with the young patients themselves. Three bound volumes of his case notes specific to neurological conditions have been archived by the hospital (Dickinson, 1869–82).

Each case is described in a single report; a few also include reports on follow-up visits, readmissions, or later communications from outside physicians or families. Reports include the child's name, age, and address; admission, diagnosis, discharge, and outcome data; and notes concerning illness history, possible aetiology, observations while on the ward, treatment, and treatment response. The level of detail varies: Dickinson had an apparent interest in discerning the differences between various forms of epilepsy, for example, so pre-ictal, ictal, and post-ictal observations tend to be thorough. On the other hand, the course of infectious encephalitis is briefly described, as no treatment was available and the condition almost always ended in death.

For the modern reader, many of Dickinson's case descriptions are a litany of unnecessary death and suffering. Although theories of infectious disease were spreading, they were not doing so as quickly as epidemic illness itself. Febrile seizures and brain damage were depressingly common outcomes of unchecked infection – if the young patient was strong enough to survive at all. Non-infectious neurological conditions, such as epilepsy, hydrocephalus, and brain tumours, were poorly understood. Without appropriate medications or surgical procedures, practitioners and carers could often do no more than try to comfort the affected child while awaiting the inevitable outcome.

Once installed in a bed at Great Ormond Street Hospital for Children, patients with neurological problems received little medical care. However,

this brief respite from the grinding poverty and filth of their customary lives may have benefited many greatly: a hospital stay meant receiving adequate food, a warm bed, and clean clothing, often for the first time in a child's life (Baldwin, 2001).

Medical equipment for diagnosing and treating neurological disorders was primitive by modern standards, but reflected the latest innovations of the time. Some children with cerebral palsy or various forms of paralysis were treated using electrical stimulation, or Faradism. Early forms of adaptive equipment were also in use to improve the lives of these patients. Children with rheumatic illnesses, such as chorea, might be sent to Cromwell House, a rural convalescent home operated by Great Ormond Street (Dickinson, 1869–82). Epilepsy could be diagnosed only through observation, as electroencephalography was not applied to humans until 1924 (Malmivuo, 1999).

Although not explicitly mentioned in Dickinson's notes, therapeutic baths (hydrotherapy) using plain water and sometimes mineral salts were widely used to treat adults with neurological conditions during this era, and may have been tendered to some patients at Great Ormond Street (Commissioners in Lunacy, 1847, p. 213).

As for medication, 'bromide salts' (potassium bromide and related compounds) had only recently supplanted herbal remedies as the treatment of choice for seizures (Locock, 1857). Potassium bromide is still used as an animal anticonvulsant, and is occasionally prescribed for human use when more modern drugs are ineffective for seizure control. Although it is a powerful anticonvulsant, potassium bromide can also cause a constellation of serious side effects known as bromine toxicity, or bromism. These include stupor, muscle pain, loss of coordination, depression, psychosis, rash, and gastrointestinal problems (Erdmann, 1995).

Many of the children with autistic symptoms described by Dickinson also presented with serious bowel disturbances. In one of three cases described in this article, senna syrup, calomel (mercury chloride), and cod liver oil were used to address this issue with some success.

It should be noted that mercury chloride came into popular use as a patent-medicine sedative for teething babies in the later years of the nineteenth century, and was revealed several decades later to cause widespread developmental and physical health problems, including the condition known as pink disease (Dally, 1997). Indeed, developmental delay and muscle weakness may be more pronounced in some children described by Dickinson owing to popular child-rearing practices, as well as the effects of poverty. Malnutrition, chronic diarrhoea, and vitamin deficiency have a detrimental effect on the developing child's brain and body. The use of dangerous home remedies like calomel or the opiate syrup laudanum, used

to quiet fussy babies, could also be responsible for neurotoxic effects and poor developmental outcomes. Violent physical punishment was widely practised, with an inherent potential for brain injury. If a child became ill, enforced bed rest and isolation were common remedies, which could lead to muscle atrophy and lack of mental stimulation (Edgar, 1999).

Case 1: Ralph

Ralph, age 2.5 years, was brought to Great Ormond Street from the Islington neighbourhood of London on 10 December 1877. He remained at the hospital for only 4 days.

Based on a parent interview, Dickinson concluded that both mother and father were in good health, although Ralph's mother is described as 'rather nervous'. The parents told Dickinson that they had one other child with no health problems. Neither parent's occupation is listed. Islington was in transition during the end of the nineteenth century, so occupation and level of income cannot be deduced by location. It is noted that Ralph's father was a foundling, so this family's income was probably low. Ralph's birth is described as normal and rapid rather than prolonged.

On examination by Dickinson, the child appeared to be physically healthy, other than a persistent cough, a somewhat 'rickety' (sunken) chest, and constipation. He had contracted whooping cough at the age of 2, but there is no other report of infectious illness. Ralph was well nourished, had a good appetite, and did not have palsy. Dickinson reported his head circumference as within normal limits at 18⅝ inches. His central fontanel was still slightly open, but the membranes appeared pink and healthy.

Despite his general look of health, Ralph had severe feeding and gastrointestinal problems from an early age as well as developmental delay. His parents reported multiple bouts of diarrhoea, starting at 8 months of age. His first attack of severe diarrhoea lasted for 5 weeks. He had been weaned early and then 'brought up by hand' (fed with a bottle – Dickinson noted that Ralph required feeding assistance), and was not toilet trained. He still had oral-motor problems at age 2.5, with difficulty in swallowing solids and frequent drooling.

Although Ralph did not have grand mal seizures, hospital staff observed that 'when asleep [he] often twitches in the arms and legs and eyes'. This may indicate the presence of undiagnosed nocturnal seizures, or of nocturnal myoclonus.

It was Ralph's development and behaviour that were of concern to his family and the medical staff, with speech the most evident problem. Ralph is described as almost entirely non-verbal: 'has never said more than "mum, mum" . . . apparently sees and hears [but] does not understand when

spoken to, or speak,' wrote Dickinson. Ralph had learned to stand at 12 months of age, but had not yet walked when admitted.

Ralph's parents reported that they had first noticed problems at around 3 months of age, when Ralph would fix his gaze on something for long periods of time, to the exclusion of other stimuli. At age 2.5, he was said to '[take] no notice . . . follows things with his eyes, but they often roll'. Dickinson notes a gaze preference for the left eye. He describes the child's eye movements as nystagmus, and they are consistent with this diagnosis. This may have impaired Ralph's vision, and contributed to developmental and behavioural differences. Nystagmus has been observed in a number of children with autism, although it can also occur independently or as a marker of other conditions (Scharre and Creedon, 1992).

Other repetitive movements were described, including rubbing his fists on his eyes, striking his eyes, kneading his hands, watching his hands in movement, pulling his toes, rolling and jerking his head, and stretching his neck in an odd way. That many of these repetitive movements involved his eyes could indicate that he was responding to sensations related to his nystagmus, although Dickinson does note that Ralph did not seem to be experiencing ocular pain.

Ralph had no normal play. When given a toy or other object to hold, he would only strike himself in the face with it. He was also given to fits of screaming, although Dickinson wrote that his 'temper [is] not bad'. No meaningful interaction with staff or other children at the hospital was noted.

One of the most interesting parts of the case notes on Ralph is a brief story reported from the parent interview: 'When carrying this child mother saw an idiot boy with paralysis one day . . . [who] used to rub his hands together and look at them as this boy does.' This may indicate that the mother was simply comparing her son to the boy she had seen, perhaps wondering if Ralph, too, would develop paralysis. However, it could mean she held the persistent folk belief that seeing a disabled person while pregnant could cause one's unborn child to have the same disability.

Dickinson gave Ralph a diagnosis of 'dementia, congenital'. No treatment was offered. The reason for Ralph's early departure from the hospital was not noted. Perhaps staff felt he could not be helped owing to the nature of his difficulties.

Based on the case notes, Ralph met the DSM-IV criteria for autistic disorder. He displayed marked impairment in the use of multiple nonverbal behaviours, failure to develop peer relationships, lack of spontaneous seeking to share enjoyment, interests, or accomplishments, and lack of social and emotional reciprocity; evinced a delay in, or total lack of, spoken language, and lack of imaginative or imitative play; displayed

stereotyped and repetitive motor mannerisms; and was developmentally delayed.

Case 2: George

In September 1877, 3-year-old George's parents brought him to Great Ormond Street from Dartford in Kent, at that time a village of just under 10,000 inhabitants. A brief parent interview turned up no clues as to aetiology, as both mother and father were healthy, with 'no neurotic history' in the mother's family. The mother's childbearing history was not unusual for the time: two living children, one deceased child, and one miscarriage.

George started life as a weak infant, born prematurely and with a poor sucking reflex. When the boy proved unable to nurse properly by the end of his first month, he was fed by bottle on cow's milk and water. By 9 months of age George could stand and walk with support, but he had marked hypotonia and did not progress to standing or walking unaided. Dr Dickinson notes that the child's parents said the boy was 'never able to sit up well, gradually slips down'.

George was left-handed, and Dickinson observed that he also favoured his left leg over his right. His head circumference was 18⅞ inches. The head shape was roughly normal, although Dickinson felt the forehead was somewhat narrow.

The child had not developed normal speech. His parents said he had 'never said anything but mumma and this only for three months', meaning that his first word appeared around 2.7 years. George did appear to have some receptive speech, and was able to communicate by means of sounds or gestures when he needed toileting. He had no history of seizures.

George's affect was unusual. 'Lies in bed with either an idiotic grin on his face or opening and closing his mouth to show his teeth and drawing the angles out,' wrote Dickinson, seeming to indicate odd, purposeless facial expressions. Although on examination George's eyes seemed to function normally, the child did not retain eye contact or show much interest in the examining doctor. According to Dickinson, the child could follow an examiner's finger with his eyes, but his gaze was irregular and easily distracted. George was not noted to play, attend the playschool sessions available at the hospital, or interact with either children or adults, other than to indicate a need to move his bowels.

The doctor added that George engaged in repetitive movements. 'Both arms [are] in almost constant movement; of the character of the meaningless movement of an infant,' Dickinson wrote.

> The movement affects all the joints from the shoulder to the fingers and includes all the movements at those joints but most remarked near the body,

> that is at shoulder. In attempting to use this arm to grasp anything movements become very violent and coarse; often when hand is close up against the object it is dashed away. Tries to hold the large joint rigid when the fingers are brought near the object and then makes a snatch on it. Fingers while this effort is being made are mostly rigidly extended and all separated spread out like a fan.

This description is consistent with stereotypical arm-flapping and hand-flapping movements frequently seen in people with autism. Undiagnosed visual problems may have also affected the boy's motor development.

A note that he had sometimes suffered from diarrhoea accompanied George's history of feeding problems. The most recent attack, 12 months previous to admission, lasted for 4 days. He had also caught measles recently, recovering just 4 weeks before admission. Although it is not explicitly stated, it is possible that his symptoms worsened following this bout with measles, causing his parents to make the costly journey to London for medical treatment. As George's symptoms had been evident for a long time, it is reasonable to expect that something triggered his admission at this time.

After observation, Dr Dickinson labelled George's symptoms with the term 'dementia'. George remained at Great Ormond Street for 2 months, when he was released to his parents. No drug treatment is mentioned in his case file, nor was improvement in his symptoms noted. Indeed, George's stay in the hospital may have harmed his health, as he contracted a fever 3 weeks after admission, with an elevated temperature recorded thereafter for almost 2 weeks.

George's symptoms meet the DSM-IV criteria for autistic disorder.

Case 3: Ida

Ida was a 2.9-year-old girl and, like Ralph, lived in Islington. She was admitted to Great Ormond Street Hospital in November 1872. Her family had been caring for her at home with increasing difficulty since the onset of seizures at 3 months of age. Dickinson's notes indicate that Ida's parents brought her in when the character of her seizures seemed to worsen, no doubt causing them greater worry about her long-term prognosis.

Ida's development prior to her first seizure was apparently normal, but infantile epilepsy struck hard. 'Ever since then she has had fits – not a day has passed without one or two occurring – sometimes she has had as many as 30,' the records state. The parent interview turned up no family history of epilepsy 'or other neurosis', and although Ida had contracted a full-blown case of measles previous to hospitalization, there was no change in

her epileptic symptoms while ill. She had one healthy sibling, and another who had died of a respiratory illness.

Her seizures were described as varying in type, number, and severity, with most affecting the left side. Duration could be as short as a minute or, reportedly, as long as 24 hours. In the absence of effective seizure control, these events took a terrible toll on the child's development.

Many children with epilepsy were seen at Great Ormond Street, but something about the way Ida presented grabbed the attention of both Dickinson and the staff. His observations of this child were even more detailed than most of his other case notes. Although the nature and frequency of Ida's seizures are described, her affect and behaviour were seen as particularly unusual. Dickinson describes her sleep pattern as disturbed, and her reaction to people as odd. 'When awake she sits up and looks about her in a half unconscious way, or else lies rolling about in bed, moving restlessly from side to side,' he said. '[Ida] cannot speak, but if moved in bed contrary to her wishes, or if anything is done that she does not like, she makes a half-screeching noise. If toys be given her to play with she takes no notice of them.' She was seen to rock in her bed frequently, 'lurching herself against the bedstead'.

As well as being completely non-verbal, Ida did not have adequate receptive speech. Her physical development and general health were relatively normal: she was described as 'a well-nourished child . . . with a fresh colour', and could stand and walk with minimal support. Dickinson's neurological examination revealed no major problems, other than a minor squint affecting her right eye. Dickinson wrote that there was no history of a blow on the head, or of worms, which were then widely believed responsible for causing seizures in young children.

Ida did suffer from marked and severe constipation. This was probably responsible for her nocturnal screaming fits, and became a focus of her medical treatment at Great Ormond Street.

Seizure control was, of course, the first order of business. Dickinson prescribed 3 grains of potassium bromide to be taken in a fluid mixture four times daily. He added to this a daily dose of three fluid ounces of senna syrup to address the child's constipation.

When Ida's bowels had still not moved by her fourth day at Great Ormond Street, Dickinson wrote an even more powerful prescription. She was to be given 11 grains of calomel (mercury chloride), with sugar if required to get it down; three fluid ounces of senna syrup; and three fluid ounces of cod liver oil twice daily. Her potassium bromide dosage was also increased.

This combination produced a remarkable change in Ida. The ward staff

reported that following four bowel movements, her screaming and rocking quieted noticeably, her sleep pattern improved, and she became more engaged with the world around her.

By 29 November, the situation was very much improved. Dickinson's notes read: 'Bowels regular. No screaming. Appears to notice things rather more than she did. A book was given her today and after a while she tore a picture out of it.'

Getting Ida to eat proved to be more difficult than expected, however. She would not feed herself properly, biting the middle out of a piece of buttered bread rather than eating the whole piece, and showing evidence of swallowing problems. Hospital staff members were able to feed her bread soaked in beef tea.

Feeding problems may have contributed to a dramatic worsening of symptoms within the first week of December. Ida began screaming and rocking again, and her constipation returned. It took 2 very difficult weeks of treatment changes before improvements returned. 'Still restless. Screams and gets in a passion beating herself and the bed with no apparent cause,' Dickinson wrote on one visit. 'In much the same condition as last note – Bites her jacket, stuffing it into her mouth,' he noted a few days later. But by 21 December Ida was again moving her bowels normally, and no longer rocking in her bed and screaming. Dickinson remarked that for the first time she exhibited normal play behaviour for a child her age, playing with a doll.

Ida went home with her family three days later, on Christmas Eve. It is likely that she continued as an outpatient, receiving medication at the Great Ormond Street day clinic. There is no record of readmission.

Of the three children described in this article, Ida stands out as unique in several ways. Her autistic symptoms were in some ways the most marked, but they were also closely linked to the onset and severity of her seizures. Because Ida's epilepsy was of early onset, it could be that her autistic traits were purely a form of acquired epileptic aphasia – the sequelae of uncontrolled epilepsy.

Dickinson's diagnosis was simply 'convulsive fits, epileptiform', so he seems to have believed that Ida's developmental, communication, and movement differences were due largely to epilepsy. The use of antiseizure medication does appear to have made a big difference. It is interesting to note, however, that potassium bromide alone did not guarantee improvement: Ida's symptoms showed persistent improvement only when her bowel problems were under control.

At the time of admission, Ida met the DSM-IV criteria for diagnosis of autistic disorder. At discharge, she was still a severely disabled child, but

appeared to be improving, as evinced by appearance of some normal play and increased attentiveness, and lessening of repetitive movements and discomfort.

Discussion

One can deduce Dickinson's frame of reference for autistic symptoms by examining what he chose to focus on in his observations and interviews. His notes indicate that he employed some basic neurological tests: measuring head circumference to check for the possibility of hydrocephalus, checking eye function and motility, and observing movement patterns and gait for disturbance. Although these tests were not systemized, they were used to compare these three patients against either putative norms or other children he had examined. Had their responses more closely matched those of children with another known neurological condition, such as Duchenne's muscular dystrophy or cerebral palsy, Dickinson's diagnosis would have reflected this fact, as it did in case notes on other children.

More importantly, Dickinson's reliance on these measures, as well as on collecting data about seizures, bowel function, and developmental milestones, indicate that he saw the autistic symptoms of these three children as organic in origin. Dickinson was aware that similar symptoms could have a psychological basis, as he made diagnoses of hysteria and hysterical mutism in other cases: something about these three children was different.

Dickinson also did not diagnose these patients as idiots or imbeciles, despite the severity of their developmental delays. He examined a number of other children who were given no label other than 'idiot' or 'idiocy', but his hand seems to have been stayed for some reason in these three cases. One might assume that the difference was affect and sociability: as Kanner and others observed in the early years of research into autism, the primary difference between children with both mental retardation and autism and those with mental retardation alone is the quality and character of their affective contact and play.

It is always difficult to pass judgement on medical conditions affecting individuals one has not examined in person, no matter how detailed the records about the patient may be, and regardless of one's own scope of knowledge. Just as other researchers have attributed autistic traits to historical figures ranging from Victor of Aveyron (Wing, 1976) to Albert Einstein (Katz, 2000), there is a strong case for correlation between the symptoms exhibited by Ralph, George, and Ida and modern diagnostic criteria for autism. In the absence of further information, however, alternative explanations are always possible.

These cases do illuminate the need for research into the history of

autism, not only to build a clear picture of its aetiology and epidemiology, but also better to understand the social context of beliefs about neurological conditions as regards diagnosis and treatment.

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