



## BIROn - Birkbeck Institutional Research Online

Hellal, Paula and Lorch, Marjorie (2007) The validity of Barlow's 1877 case of acquired childhood aphasia: case notes versus published reports. *Journal of the History of the Neurosciences* 16 (4), pp. 378-394. ISSN 0964-704X.

Downloaded from: <http://eprints.bbk.ac.uk/908/>

*Usage Guidelines:*

Please refer to usage guidelines at <http://eprints.bbk.ac.uk/policies.html> or alternatively contact [lib-eprints@bbk.ac.uk](mailto:lib-eprints@bbk.ac.uk).

Author's Accepted Manuscript – do not cite without permission

To appear in Journal of the History of the Neurosciences.

The validity of Barlow's 1877 case of acquired childhood aphasia: Case notes versus published reports.

Paula Hellal and Marjorie Perlman Lorch

Birkbeck College, University of London

Birkbeck College, University of London,

43 Gordon Square, London WC1H 0PD England

Email: [m.lorch@bbk.ac.uk](mailto:m.lorch@bbk.ac.uk)

Phone: 44-207-631-6119

Fax: 44-207-383-3729

## Abstract

In 1877, Barlow described a 10-year-old boy with right hemiplegia and aphasia, quick recovery of language function, and subsequent left hemiplegia and aphasia, who was shown to have symmetrical left and right Broca's area lesions at autopsy. The report of this case motivated many writers in the second half of the 19th century to develop theories on localization, laterality, equipotentiality and development of specialization, recovery of function, and the role of the right hemisphere (see Finger et al., 2003, for review). This paper presents an analysis of the original archived case notes that have recently come to light. Examination reveals discrepancies in significant details of the history of the case and raises questions about the degree of impairment and recovery throughout his illness as reported in the published article. Consideration of these differences between the presentation of the case in the British Medical Journal publication and the documentation in the original patient records raises issues about the validity of this case as evidence for the many arguments it was to support that have persisted to the present.

Keywords: child aphasia, Barlow, localization, recovery of function, Broca's area, motor function, Great Ormond Street Hospital

## Introduction

A unique case of acquired childhood aphasia was published in the British Medical Journal (BMJ) in 1877 by Dr. Thomas Barlow (1845-1945). This case was discussed by a great many of the major British physicians writing on aphasia in the later half of the 19th century. The case was used as evidence to motivate theories of localization and lateralization of language in the brain, patterns of impairment, and recovery that have been perpetuated to the present day. It was also instrumental in getting age at symptom onset to be considered as an important factor in proposed mechanisms of recovery. However, these various interpretations were based on the publication of the case as represented in Barlow's BMJ article. Recently, the first author (Hellal) discovered the original and unpublished archive case notes made while the boy was an inpatient at Great Ormond Street Hospital for Sick Children (GOSH) in London. While carrying out research into the 19<sup>th</sup> century understanding of the aphasic condition in childhood, she systematically searched the GOSH archives, from the period 1852-1900, for cases of language loss or impairment in young children. (See Hellal 2005 for details.)

Careful examination of these case notes shows what appear to be significant discrepancies between Barlow's representation of the case as published in the BMJ and the record of the patient's symptoms, assessment, and progress, as detailed in the physician's casebook. This article reconsiders the Barlow case in the light of implications raised by the original medical notes.

## Barlow's BMJ report

Thomas Barlow's case concerned a 10-year-old, hemiplegic, aphasic boy admitted to Great Ormond Street Hospital in December 1875. The paper was published some 18 months after the patient's death. Barlow had received his M.D. the year before and had been recently appointed to the hospital staff as a junior assistant. This case was his first publication. The senior physician that Barlow assisted was Dr. William Howship Dickinson (1832-1913), who had been on the staff at GOSH for 14 years when this boy was admitted under his care.

William Howship Dickinson. (1832 – 1913) studied medicine from 1858 at Cambridge and in St. George's Hospital, London. He received his M.D. in 1862. He was curator of the pathological-anatomical museum in St. George's Hospital, where he became assistant physician, then physician in 1874. He was also assistant physician at GOSH from 1861-1869 and physician at the children's hospital from 1869-1874. An expert on diseases of the liver, he was also an authority on childhood diseases. He was examiner in medicine to the Royal College of Surgeons and to the universities of Cambridge, London, and Durham, and was made a fellow of the Royal College of Physicians in 1865. Although the boy that was the focus of Barlow's article had been admitted to GOSH under Dickinson's care, Barlow makes no mention of this fact in his report. (The most detailed childhood aphasic case in the GOSH archives was a 9 year-old patient of Dickinson's. Discussion of this 1871 case, and a full transcript, can be found in Hellal 2005.)

The details of the case as published by Barlow can be summarized as follows:

- Four months before admission to the hospital, the boy had suffered a right hemiplegia and aphasia.

- After a period of ten days, the child was said to have greatly improved in both motor control and expressive speech.
- A week before admission, the child had suffered a second attack with a left hemiplegia and aphasia.
- Less than two months later the boy died and the cause of death was given as heart disease.

This is the clinical picture presented by Barlow in his BMJ paper. After the boy died, an autopsy was carried out by Barlow. At death, the 10-year-old weighed only 47 lbs. The autopsy revealed two homologous lesions involving the cortex and some white matter (“less than a quarter of an inch deep”) located in “the ascending frontal and the hinder end of the middle and inferior frontal convolutions” of both hemispheres (Barlow, 1877, p.103).

The existence of an autopsy verification of localization of lesion added to the significance of the case. Post-mortem examinations were not regularly carried out in this period. Many published cases had only clinical descriptions of behaviour from which pathology was hypothesized. The clinical-pathological correlation method was the most sophisticated analysis available for theory in brain-behaviour relations at the time. Indeed the impact of Broca’s case of 1861, which argued for a frontal localization for the language faculty, was to a great extent due to the existence of the autopsy evidence.

#### Dickinson’s original case notes

In the clinical record book of Dr. Dickinson, observations on this patient were recorded regularly over a period of 56 days. We present below a summary of

Dickinson's original case notes, and highlight the discrepancies between them and the case as published by Barlow. The social background and family circumstances were noted first, followed by a description of the onset of illness and the history of its progression up until the time of admission to the hospital. The history of the boy's presenting condition and details of symptoms were recorded in the notes from an interview with the child's mother.

The child was a local London boy whose father worked as a stableman. As was standard, mention was made of the child's family history, past illnesses and presenting condition. The boy was one of 6 children, born full term. The other 5 were healthy, as were both parents. There was no history within the family of fits, paralysis, or rheumatism. Aside from an attack of measles when 4-years old, the boy seemingly had been well until 4 months prior to his admission to the hospital.

The mother's description of the history of illness was recorded in the notes:

Up to 4 months ago was able to run upstairs. No cough- pant. 4 months ago was holding the baby and was noticed to walk slowly suddenly burst out crying and then mother said that his face was drawn. Didn't lose his senses that day. Was going to fall but mother laid him down. Directly after when something was given him to eat he couldn't take a fork. Within quarter of an hour, he was able to walk upstairs. Walked right. Put to bed. Swallowed all right. Talked right. Slept. No twitchings. Next morning had lost his speech- only said hawhaw. Face drawn a little for 6 days. Unable to grasp with right hand. Could lift the arm. When mother came to move him, the boy dragged the right leg.

The onset of the second attack of hemiplegia was also described by the boy's mother. The child was reported to have been complaining about a pain in the back of his left leg for some twelve days, but no notice had been taken of it. A week before coming to the hospital, the boy had been working all night, cleaning the stables despite still

having difficulty using his right hand. His mother observed that he looked cold and was very irritable. The boy got in trouble with his mother for threatening the other children, had burst out crying, and was taken to bed. The next morning, he was unable to use his left hand. He also could not drink well. His face was not drawn but was puffy. He was speechless and only able to stand with support.

The case notes indicate that on admission to the hospital the boy had to be carried in and laid on a couch. It is noted that the child did not speak, but was able to use gestures in response to questioning. He was able to say “ah” when asked by the doctor, but was “unable to make any other articulate sound.” Dickinson notes that he had some difficulty swallowing, liquids tended to run out of his mouth, and he was unable to “purse his lips forward.” The child’s “perfect” receptive language skills were remarked upon.

#### Details of Clinical Practice and Assessment Procedures.

Archived medical case notes are important sources providing insights into medical treatment and practice. Hellal (2005) carried out a detailed analysis of the case notes of all children with acquired aphasia in the second half of the 19th century at GOSH. Examination of these case notes can illuminate the processes by which clinicians determined which symptoms or clinical signs are significant in making a diagnosis. The notes of this case are comprised in part of a written record of the mother’s verbal report of her son’s history of illness.

Both Hunter (1991) and Jacyna (2002) stress the typical obliteration of the patient’s voice within medical texts. In the case of child aphasia patients in the 19<sup>th</sup> century the



silence is absolute. No GOSH patients described the condition from their own perspective. The ambiguities and cross-textual incongruities of the Barlow case hinge on the interpretation of spoken discourse and written texts.

While physicians both at GOSH and elsewhere recognised the importance of questioning family members, they made frequent judgements as to the value of the information. Edwards (1899) assured his students that “knowledge of much importance may be gained from the parents.” He stressed, however, “a physician’s testimony is, of course, more valuable than a parent’s” (Edwards, 1899, p. 1172). The social status of the parents also, and openly, affected how seriously physicians treated the information.

Typical physical assessment procedures used at GOSH in the 19th century included observing the state of the tongue and checking for paralysis. Assessment of language function was made by observation of expressive speech. The clinical testing assessed hearing ability, muscle control, comprehension of spoken and occasionally written language. Comprehension of spoken language was tested by asking the child point, count on their fingers or carry out an action. Some attempts were made, particularly if the child had previously attended school, to assess knowledge of the alphabet. (See Hellal, 2005, for details.)

Very few of the GOSH Case Notes describe the loss and reacquisition of language in any detail. The physicians’ descriptions of linguistic impairments were idiosyncratic with very little, if any, phonetic or grammatical analysis. The term aphasia, suggested by Trousseau (1864), was used in the hospital notes in London from the mid-1860s. Its meaning was synonymous with the earlier historical term “loss of speech.” Indeed, until the turn of the

century, GOSH physicians favoured the term “speech affected,” to describe language disorders in their patients.

In this particular case, Dickinson recorded many significant aspects of the child’s behaviour and the methods used to assess them. Excerpts from the case notes which detail his language abilities are presented here:

[He] nods his head when questions asked and seems to understand. When asked his age he counts on questioner's fingers 10 which is correct. Shows his own fingers-4 when asked how many brothers and 1 when asked how many sisters....He has just written Walter on a piece of paper when asked to write his name.

[He] puts up his hand when he wants the utensil. Puts hand to mouth when he wants to drink. Makes no attempt to speak. Signs are always correct i.e. he never makes negative signs when he means affirmative or sign for drink when he wanted something else....[could not] be induced to protrude tongue....[could not] test masseters [because] he won't close his mouth when told. After much persuasion, he does give fingers a good bite when placed between his molars.

The boy’s temperature and pulse were taken daily and recorded on charts. There were 10 entries with detailed notes on behavioural assessments. Although regular examinations were made, there was little change in the boy’s motor or speech functions over the ensuing 56 days. Four days prior to his death, the boy was observed to vomit during the late evening, becoming rigid, cold, and clammy, but there was “no increase of any of the nervous signs-- except the greater difficulty of making him swallow.” He was “still obviously rational” the day he died. It was recorded that he was able to sit up, “in fact got out of bed once of his own accord. But he doesn’t really look any better.”

Discrepancies between the case notes and the BMJ article

The case notes, as would be expected, provide a number of additional details omitted from the published report. However, there are also a number of important discrepancies rather than simple omissions within the text. See Table 1 for a summary.

[Insert Table 1 about here]

In describing the boy's recovery after the first hemiplegic attack, Barlow stated, "the speech had returned on the 10th day" (103). In Dickinson's case notes, there is mention only that "speech began to return on the 10th day." The mother's account as recorded in the case notes is as follows:

...occasionally made a mistake, gave the wrong name for a boy [i.e. misnamed someone] - gave the wrong time. Also didn't seem always to understand correctly what was said to him. Dr asked him to put out right hand - he would put out left.

It is important to note that no reference is made to the paralysis of the right arm, already stated to be impaired, which might have affected on his ability to carry out the movement with the requested hand. In addition, it must be recalled that the doctor referred to here was the physician who had seen the boy prior to his admission, and not one of the hospital staff.

Although this description, as given by the mother, seems to indicate persisting language difficulties, in the BMJ article Barlow describes the boy as having made a full recovery from aphasia. Barlow made the important claim that "from the first he was able to write his name when asked and after a few weeks would answer in writing any question that was put to him" (Barlow, 1877, p. 103). This description appears to

be at odds with the documented writing ability found in the case notes. The boy's ability to write his own name is mentioned three times in the notes, but there is no evidence of any other written language production. If it were indeed the case, as stated by Barlow, that the boy could write answers to any question, it is surprising that there is no record of this in the case notes, despite detailed descriptions of attempts to elicit communication, i.e., his use of gestures, his impaired vocalisation, his reading skills, etc.

Barlow was equally emphatic about the extent and rapidity of the child's physical recovery: "In 10 days he was greatly improved. The leg improved before the arm" (Barlow, 1877, p. 103). The record in the case notes does not suggest that he had improved to the extent implied in Barlow's article. The case notes report that his mother said she sent him on errands one month after the first attack. However, it is also reported that he was only "using his right arm a little better when [the] present attack occurred... [but]... never got full use of it." The motor examination recorded in the case notes at the time of admission following the onset of the second attack with left hemiplegia stated: "Grasp both arms feeble. Grasp feeble right, very feeble left." Additionally, Barlow (1877) states that the boy was able to stand without support after the second attack. This statement is in direct contradiction to the case notes, which clearly state that on admission to the hospital he had to be carried in and laid on a couch and was only able to stand with support.

With respect to his general mental status, Barlow describes the boy as being intelligent throughout the article. The case notes suggest a different picture. His mental condition was described by the mother as "confused" after the second attack.

On admission, the boy is described as appearing “dull” [i.e. dull-witted as opposed to bright].

The autopsy report in the case notes is for the most part in agreement with the published article:

At junction of Fissure of Rolando with posterior limb of Sylvian fissure there is a slight depression holding tip of finger. Pia mater is complete.... This patch also extends to anterior limb of F of Sylvius. Altogether it might be covered by a shilling. On similar position right side- a patch as big as a filbert. With hard ridgy elevation and depression round it. Diseased part destroys lower part of ascending frontal and a small portion of middle frontal convolutions. In left middle cerebral 1 and quarter inch from its commencement there are some patches of calcify [sic; calcification] not actually occluding artery- nodules each as big as a millet seed. One branch can be traced into diseased portion. Another branch backwards sends branchlets into diseased portions. Small nodules in walls of these branches but not causing occlusion. Right cerebral artery- at same distance another calcified nodule as large as a millet seed. Vessels elsewhere at base natural. Ventricles natural. (archived case notes.)

One point which might alter the interpretation of the case is the size of the two lesions. In the title of his article Barlow called attention to the “symmetrical lesions” suffered by the child, emphasizing the homologous site of the lesions. The original autopsy report included in the clinical record states that the lesion on the left was the size of a shilling while that on the right was the size of a filbert (similar to a hazelnut). However, in Barlow’s article he describes “regions of softening, *each of which* might be covered with a shilling” (Barlow, 1877, p. 103), [emphasis added]. The size of English shillings changed several times during the latter portion of the 19th century, so it is difficult to state definitively its size. However, the wording of the autopsy report included in the clinical record clearly indicates a significant difference in the size of the two lesions, although the magnitude of this difference is not clear to modern readers. Foremost for Barlow was the homologous location of the lesion

sites. By concentrating on location, Barlow implied that the “symmetrical” lesions were the same size. However, issues regarding the amount of tissue damaged might also be relevant to the interpretation of the boy’s pattern of deficits. Perhaps subsequent readers would have interpreted the size difference as important, if it had been accurately recorded in the published account.

#### The 19th century medical context

When it opened in London in 1852, GOSH became the first hospital in the English speaking world to be devoted solely to young patients. The child had become an object of scientific medical interest. Prior to its founding, sick children were typically treated at home. For the first time, the large numbers of sick children examined at GOSH, both as outpatients and as inpatients, allowed for the quantitative analysis of diseases of childhood and comparison of various treatments.

During the second half of the 19th century, as a direct consequence of the establishment of GOSH and other specialist children’s hospitals (e.g., Norwich in 1853, Manchester in 1855, Edinburgh in 1860, Birmingham in 1861), a large number of paediatric textbooks began to appear in the English language. The first of these was written by GOSH’s founding physician Dr. Charles West in 1848. His book, Lectures on the Diseases of Infancy and Childhood, appeared in several revised editions, which included increasingly lengthy discussions of language difficulties in childhood; including language delay, stammering, stuttering, and aphasia (Hellal and Lorch, 2005). At the same time there was a growth of interest in the phenomena of language acquisition in the wider scientific community (e.g., Darwin, 1877; Sully, 1881).

In Britain at this time, a major focus with respect to clinical research on language impairment was concentrated in London at the National Hospital for Paralysis and Epilepsy in Queen Square. The National Hospital was a 5 minute walk from GOSH and shared many of its staff. Physicians such as John Hughlings Jackson (1835-1911), Henry Charlton Bastian (1837-1915), David Ferrier (1843-1928); William Gowers (1845-1915), and James Taylor (1859-1946) wrote extensively on brain-behaviour relations including aphasia (see Lorch, 2004). All of these authors were to cite and comment on the Barlow case in their subsequent publications.

The significance of Barlow's case as reported in the BMJ was seen to be the coincidence of having a right hemiplegia and aphasia, recovering, and then suffering a left hemiplegia and aphasia, with two symmetrical cerebral lesions at autopsy. In their review, Finger and colleagues point out that, although there have been a few cases showing certain similarities to the Barlow case, none have had lesions "as discrete or symmetrical as those described in Barlow's 1877 publication... from an anatomical-pathological standpoint, the Barlow case has remained in a class of its own" (Finger et al, 2003, p. 392).

The case, with its apparently identical, homologous lesions, and reportedly rapid and complete recovery from the original aphasia, lent itself to various interpretations. Barlow, who took a motor perspective, analysed it in the light of Broadbent's (1866) hypothesis that the bilateral muscles, which act together, are represented on the two sides of the brain. Hence, Barlow attributed the boy's condition to the loss of voluntary power over the mouth and tongue muscles. After the first attack of

hemiplegia suffered by the child, Barlow suggested that the region on the left hemisphere was probably permanently damaged, while the right side of the brain remained intact. The right motor cortex was, therefore, still able to control the bilateral mouth musculature. The second attack, though, damaged that region, leaving the child “irretrievably deficient” (Barlow, 1877, p. 104).

John Hughlings Jackson had been invited to observe the patient while he was in GOSH, and Gowers was asked to examine sections of the brain after autopsy. The significance of the case for them was also with regard to theories of motor control. Thirteen years later, Hughlings Jackson included a reference to the case in his Lumleian lectures (1890) given at the Royal College of Physicians. Like Barlow, Jackson cited Broadbent and described the case as showing that unilateral lesions of the motor area may have only a transient effect on the muscles of the tongue, whereas bilateral damage would have more permanent effects:

Hence, destruction of neither Broca’s region nor its fellow part of the right middle motor centres produces disability in the commonplace services of the muscles of the tongue etc., although beyond all doubt some movements of them are lost. But destruction of part of both halves does, as some cases of double hemiplegia show, notably one recorded by Dr. Thomas Barlow (Jackson, 1890, p .826).

#### Barlow’s 1877 Case as Theoretical Evidence.

The debate over the notion of localization of the language faculty that had been active at the beginning of the 19th century experienced a resurgence with the work of Broca (1861) and Jackson (1864) (Harrington, 1987; Young, 1990). However, many in the scientific and medical communities remained unconvinced that higher mental functions are localized in the cortex. One strong advocate for localization theory was



the Queen Square physician David Ferrier. Ferrier's own research interests involved the experimental mapping of cortical areas in animals. When he heard of Barlow's case from colleagues, Ferrier corresponded with Barlow on the importance of bringing it to the attention of the wider medical community.

In his discussion, Barlow drew a direct link between Ferrier's experiments with monkeys and the post mortem results of this boy. Barlow asserted that Ferrier's work "substantiated" the autopsy results: "Professor Ferrier has shown in the brains of monkeys that in this identical region or rather in the region homologous with it the centre for the movements of the mouth and tongue is situated" (Barlow, 1877, p. 104). Both men saw the case as an ideal example, provided by nature, of the relationship between clinical symptoms observed during life, lesion site discovered at autopsy, and experimental research carried out on living brains.

In The Localisation of Cerebral Disease, which was published the following year, Ferrier argued that it was not enough for the anti-localizationists to put forward cases where a lesion affecting the third frontal convolution (of either hemisphere) had not resulted in accompanying loss or deterioration of speech. It was "not contended that there is an absolute restriction of the speech centre to the left hemisphere. ...exceptions may be admitted without invalidating the localisation of the speech centre in one side or other" (Ferrier, 1878, p. 517). A case having a lesion to the left frontal hemisphere without accompanying speech disturbance could not count as evidence against the localization hypothesis. Indeed such cases were "admitted by all."

Instead, Ferrier argued that it was “incumbent upon the opponents of this localization to bring forward a case in which with bilateral lesions of this centre no aphasia occurred. But ...no such evidence exists” (Ferrier, 1878, p. 517). Ferrier suggested that the effect of such lesions would be “both aphasia and anarthria or paralysis of articulation” (Ferrier, 1878, p. 517). In support of his position he cites the “beautiful” and “unique” Barlow (1877) case.

As well as providing support for the language localization hypothesis, the case was also used to support hypotheses related to how language function might be reacquired after aphasia. Well before the Barlow case was published, physicians had speculated on how an adult aphasic might recover his language skills. The principal hypotheses put forward during the 19th century were that: an undamaged part of the same hemisphere might take over the function of the damaged region; the lower brain structures might be involved; or the homologous area of the opposite hemisphere might “take over” the function (Broca, 1865). This last theory gained the most support and it was embraced by Walter Moxon (1836-1886, physician at Guy’s Hospital London), who hypothesized that “in the right brain there will be all the organs, which if educated would become the seats of speech power; so that the ground-plan symmetry of the organs of speech is preserved” (Moxon, 1866, p. 485). This would suggest that, under certain circumstances, the right hemisphere, once it were educated to carry out its newly acquired role, could function as the dominant hemisphere for language (see Buckingham, 2003).

Samuel Wilks (1824-1911), senior physician at St. Bartholomew’s Hospital London, agreed with Moxon’s suggestion, believing that the localisation of language function

in one particular hemisphere is “entirely owing to education of two different sides of the brain” (Wilks, 1883, p. 89). Wilks defined his use of the term “education” as not simply referring to the individual but rather “to long usage of one hemisphere through many generations.” In support of his argument, he cited research by Broca and Bastian showing a difference in size between the two hemispheres. Wilks had observed several cases whereby, after right hemiplegia and aphasia, the patient would appear to relearn language skills. He argued that when language was relearned after aphasia “the other side is being educated for the purpose” in a process very similar to teaching the left hand to write after paralysis of the right (Wilks, 1883, p. 89). For language function to return “it must come by re-education and what more likely than that the part corresponding to the damaged one should be the seat of the training- that this should take up the lost function...if speech were originally learned in a special way, it must be regained by same method” (Wilks, 1883, p. 89).

The process that Wilks proposed would seem to imply a slow rate of recovery of language function. However in a number of cases, typically involving children, language was seen to recover swiftly, sometimes in a matter of days or weeks. By what mechanism was the opposite hemisphere “educated” so quickly? Gowers (1885) believed that in cases of all ages, when language function is recovered after organic lesion in Broca’s area, the recovery would take place fairly rapidly, “compensation by the right hemisphere occurring with great readiness” (Gowers, 1885, p. 111).

However, this “compensatory use of the right hemisphere,” he explained, occurs far more readily in children than in adults and more readily in some adults than in others, permanent aphasia in childhood being “almost unknown” (Gowers, 1885, p. 125).

Gowers (1885) proposed that the left hemisphere does not have a monopoly over language function. Like Broca and Moxon, he suggested that there are structures “of similar position and connections” in the right hemisphere. These structures are able to “supplement” those in the left hemisphere and involve sensory as well as motor functions. According to Gowers, the right hemisphere in both children and adults is involved in “speech processes... much emotional expression and automatic use of words is affected by it.” He cited as evidence cases where “automatic and emotional use of words remains although the voluntary use of words is lost by disease of left hemisphere” (Gowers, 1885, p. 125). Gowers followed Hughlings Jackson in concluding that, as these types of expression are not lost following disease of the left hemisphere, they must, unlike voluntary expression, be bilaterally controlled.

In his review of aphasia, Edmund Cautley (1864-1944), a physician at GOSH from 1888-1893, also argued that the left hemisphere is the “important one” for speech function, “while the corresponding one on the right has a supplemental function” (Cautley, 1889, p. 266). Bramwell (1897) further expressed the view that the two hemispheres are capable of carrying out language functions. Speech centres in the right hemisphere are, he suggested, “usually inactive” but capable of “taking up and carrying on the speech functions” when the language centres in the dominant left hemisphere are damaged. This “compensatory ability” varies from individual to individual” (Bramwell, 1897, p. 363).

Bramwell suggested that compensation after language loss or impairment could be effected “by the education of the inactive or apparently inactive speech centre” in the opposite hemisphere (Bramwell, 1897, p. 369). He made no claim that this will take

place rapidly. The amount of time needed, he thought, would depend upon a number of factors, including the amount of education the non-dominant hemisphere possessed at the time of lesion onset, which language centre was destroyed, the patient's age, previous state of health, etc. Furthermore, Bramwell used the Barlow case to argue for right hemisphere involvement in language function "as a normal physiological condition" (Bramwell, 1897, p. 460). He highlighted age at onset of illness as the significant factor. If complete compensation from motor aphasia could "be effected by the right motor speech centre taking up the function of the destroyed left" (as was argued to have occurred with Barlow's patient) it might be the case that, in the young at least, a "certain amount of functional activity" normally takes place in the non-dominant hemisphere, enabling the control of function after injury (Bramwell, 1897, p. 460).

Bramwell wrote that Barlow's "well know" case "conclusively proves" recovery is effected by the involvement of the opposite hemisphere:

The obstruction of the left Sylvian artery had produced the right sided hemiplegia and motor aphasia which was gradually recovered from, the recovery being without doubt due to the speech function being taken up and carried on by the right motor speech centre. The obstruction of the right Sylvian artery then occurred and both speech centres being destroyed the aphasia was absolute and permanent (Bramwell, 1897, p. 367).

Bramwell's depiction of Barlow's case as providing definitive proof of the involvement of the right hemisphere in language recovery had become the standard interpretation of the case by the 1890s. Edinburgh physician John Wyllie (1844-1916), in his major textbook on language disorders, described the boy's initial recovery in, by then, very familiar terms:

But he speedily regained the power of speech, by training the corresponding right third frontal convolution. At a subsequent period, a second embolism

plugged the artery of supply of this convolution also, so that the patient again became aphasic, and this time the aphasia was permanent (Wyllie, 1892, p. 1093).

Whereas Gowers used the Barlow case as evidence for the take over of language function by the undamaged hemisphere, Edmund Cautley went further in proposing that both hemispheres have equal potential for taking control of language function at birth, with lateralization only occurring at a later stage. Cautley used Barlow's case as evidence to argue that "some individuals, with a permanent lesion, recover speech much more quickly than others." He suggested it was very probable that, in childhood, the speech processes go on more equally in the two hemispheres. He could not accept that one hemisphere could have the monopoly on language function, and proposed that:

In childhood there is a double centre for speech, one on each side of the brain.... But in the course of growth the left hemisphere gradually assumes the monopoly of the speech function, except, indeed in left handed persons in whom the right centre assumes the monopoly (Cautley, 1889, p. 265).

By the turn of the century, the supposed plasticity of the infant brain was used to account for a number of anomalous cases.

One year after Cautley's 1889 aphasia article appeared, two American physicians published an article that served as the only English language citation generally given in reviews of 19th century literature on acquired child aphasia (e.g., Lees, 1993; Marien et al., 2002). Sachs and Peterson made no mention of Cautley but concluded, as had Cautley, that "as we grow older we appear to become more and more left brained. In the earlier years both hemispheres are equally entrusted, so it seems, with this highest faculty of speech" (Sachs and Peterson, 1890, p. 311). Their hypothesis was later designated as "the hemispheric equipotentiality and progressive

lateralization of language development” hypothesis by Basser (1962) and Lenneberg (1967).

James Taylor, a highly respected paediatrician during the early-20th century, also appeared to support a slow lateralisation of function when he stated that takeover of function after brain damage by either an undamaged region in the same hemisphere or the equivalent area in the opposite hemisphere is more likely to occur in children “before the various regions of the cortex are indelibly stamped with a special function, provided that there is a sufficient area of normal cortex remaining” (Taylor, 1905, p. 221). He noted that aphasia is almost never permanent in children less than 10 years of age. Barlow’s case is again given as evidence.

Interestingly Taylor adds that the child was right handed, yet how he came by this information is not known. Taylor was a physician at Queen Square. In 1905 Barlow would have been 60 years old (Dickinson was by then 73 years old and retired). Barlow (Sir Thomas Barlow by then) remained associated to GOSH practically until his death in 1945. It is possible that Taylor, who must have known his respected colleague working in a neighbouring institution, simply asked him to clarify this vexed question of the boy’s handedness.

Thus, it could be argued that the case presented by Barlow in the BMJ provided support for several of the major theories in neurolinguistics. But the disparities detailed above between Barlow’s published article and the clinical picture given by Dickinson’s clinical notes raises some doubts as to the interpretation of the case. This is of crucial importance, as it was Barlow’s 1877 publication that was used as

evidence for the major theoretical discussions of language and the brain at the end of the nineteenth century and into the twentieth century. These theories have been perpetuated to the present day and form the foundation of assumptions for cognitive neuropsychology.

### Discussion and Conclusions

In clinical case notes, the recording of a patient's symptoms and documentation of visits by examiners provides chronological updates on an unfolding picture. Hospital case notes are written for a different audience than published journal articles. Intended only to be read by the clinical staff, there is little or no theorising or generalisation. In contrast, cases selected for publication serve to illustrate the unusual; describing, for example, a rare pathology or unexpected course of an illness.

Case notes provide the histories of all individuals admitted to the hospital. They provide a rich source of data: including age, social background, history of illness, suspected etiology, treatment, and prognosis. These notes are a valuable source of information about clinical practice, assumptions, and expectations. Whereas the published records may provide a coherent presentation of a case, patient histories as reflected in clinical case notes can be used to gain insight into the process and production of clinical knowledge (Risse and Warner 1992; Hellal, 2005).

An examination of the original clinical notes found in the hospital archive which document the case presented in Barlow 1877 is illuminating. It reveals much about what kind of information was considered relevant at admission, the key points of



interest in the history of illness and the assessment techniques employed to reveal symptoms.

Barlow's case highlights cross-textual incongruities between the in-hospital case notes and the published report. (See Jacyna (2002) for an analysis of the importance of written and spoken texts in the history of aphasia.) Consideration of the clinical records of the case reported by Barlow in 1877 calls into question the validity of subsequent interpretations and theoretical argumentation. Placed side by side, the Barlow's 1877 BMJ publication and Dr. Dickinson's case obviously describe the medical circumstances of the same patient, albeit for different audiences and in different formats.

Does it really matter that a word has been omitted here, or a paragraph edited there? After all, every published case has been edited from earlier notes and drafts; the author sifting and refining the evidence. We would argue that in this case the implications of Barlow's editing are significant. It must be stressed that Barlow's own declared interest in the patient was from the perspective of a motor impairment rather than from the perspective of a language deficit that is followed by recovery. Subsequently, however, it was what the case seemed to reveal about language and the brain that caused the case to be cited as "perfect" evidence for a number of hypotheses about aphasia.

On reading the brief, two-page presentation in the published case, the picture appears clear-cut: left cerebrovascular accident, right hemiplegia and aphasia, complete recovery after 10 days; a second cerebrovascular accident 4 weeks after first, left

hemiplegia and aphasia, mirror lesions in Broca's area and in the homologous area in the right hemisphere. The clarity and simplicity of the published case, however, contrasts with the archival record that describes a more complex, less precise picture. If the child had not fully recovered his language abilities following his first cerebrovascular accident, as a reading of the archived notes would suggest, then it cannot be cited as strong supporting evidence for some of the neurolinguistic hypotheses described above. It must also be stressed that the evidence for the child's recovery of language and motor function after the first hemiplegic attack was taken from the history given by the mother at admission. Neither Barlow nor Dickinson directly observed the child until after the second hemiplegic attack.

As discussed above, there is general consistency between the BMJ article and case notes with respect to the autopsy findings, except regarding the crucial point on size of lesion. The original post-mortem report indicated that the two lesions differed in size, while Barlow reported them as being the same size. To compound matters, another aspect of the autopsy was subsequently misrepresented by a number of physicians in their discussions of the case in the following decades. Barlow described the middle cerebral arteries as having "no actual occlusion, but the calibre was obviously diminished" (Barlow, 1877, p. 103). In Ross's (1887) description of the case, it is erroneously stated that the arteries were blocked: "at the autopsy the anterior branch of each Sylvian artery was found blocked by an embolus" (Ross, 1887, p. 105). Wyllie (1892) and Bramwell (1897) perpetuated this error: "At a subsequent period, a second embolism plugged the artery" (Wyllie, 1892, p. 1093); "On post-mortem both of the Sylvian arteries were plugged with an infarction and there was a localised softening in each motor speech centre (Bramwell, 1897, p. 367).

The many citations of the Barlow case following its publication directly contributed to the perception of acquired childhood aphasia as being a transient condition.

Physicians who cited the case in the 19th and 20th centuries only had access to the published paper and subsequent interpretations by other authors. Finger and colleagues (2003) point out that they failed to “carefully consider the nuances or uncertainties of what they were claiming on the basis of incomplete clinical and limited anatomical material” (Finger et al, 2003, p. 391). They argue that the influence of both Gowers and Taylor, who “authored some of the leading English textbooks of neurology and child neurology respectively,” had been crucial to the prominence of this case’s theoretical significance. Finger and colleagues go on to state: “It is not difficult to imagine how others well into the twentieth century would follow their lead and think of the Barlow case as evidence for some sort of dynamic functional reorganization” (Finger et al, 2003, p. 391). This raises the intriguing question: if 19<sup>th</sup> century physicians had read the original case notes rather than the BMJ article, would they, like later authors who relied on their texts, have drawn the conclusions they did?

To provide anything other than a purely speculative answer we need to consider a) whether the three main hypotheses outlined above were, or could have been, proposed without the case as supporting evidence, and b) whether the case as presented in the archived notes could adequately support any or all of the hypotheses.

The localisation of language function in the human brain had exercised the minds of many, both in Britain and on the Continent, for decades before the case was

published. The Barlow case provided strong support. It was not the foundation upon which theories of language localisation were built, but its importance as supporting evidence is clear from the number of citations of the case in any discussion of language localisation. Could the archived case have provided similar support? If there is any doubt that the child made a full recovery from his initial aphasic state the case is simply one of many hundreds of right hemiplegic/aphasic cases presented in the medical literature by the 1870s. It supports the language localisation hypothesis, but none could claim for it the extraordinary perfection of the published case which, Ferrier claimed, was “an experiment of disease, *approaching to the conditions of an exact and precise experimental lesion*” (Ferrier, 1878, p. 518) [Italics added]. It was one of the most “satisfactory and conclusive demonstrations of the harmony between human pathology and experimental physiology” (Ferrier, 1878, p. 518). It is less certain that the case, as it appeared in the archives, could have been used to such effect.

The Barlow case provided support for a related hypothesis, much debated during the latter decades of the 19<sup>th</sup> century (as it is today). Almost all the late-19th century discussions of recovery of function involving the undamaged non-dominant hemisphere stress the significance of this case. As published, the evidence appears overwhelming. It is interesting to note that there was, though, a lone voice of caution raised in the late 19th century as to the validity of the standard interpretation of the Barlow case. Well respected early in his career, by the 1890s Henry Charleton Bastian had lost authority within the medical and scientific world due to his continued belief in the notion of spontaneous generation. Despite publishing on aphasia, he failed to mention the Barlow case until 1898, when he referred to it possibly in direct

response to Bramwell's 1897 article. He disputed the conclusions reached by other commentators:

Barlow's case is often referred to as proof that the right third frontal convolution may, in a young subject, completely and rapidly, take over the functions formerly carried on by the left third frontal convolution. This however, seems to be open to very grave doubt (Bastian, 1898, p. 322).

Unlike Bramwell, Bastian noted the time scale and remarked that it was doubtful that complete transfer could have taken place within only 10 days. He is also the first person to point out that Barlow failed to mention whether the child was left or right handed. This important information is also missing in the archive case notes.

For all we know the right hemisphere might have been the leading hemisphere for speech, and the first lesion on the left side may have merely occasioned some functional disability in the right centre, from which in a very short time he recovered. With the occurrence of the second lesion, however, both third frontal convolutions would have been damaged, and thus the "pseudo bulbar symptoms" would have been produced (Bastian, 1898, p. 322).

In an earlier article published in 1887, Bastian described cases of motor speech defect in which "there is loss of speech without any mental impairment, and with the power of writing preserved intact." The 10 year-old GOSH patient, according to Barlow 1877, had presented with similar clinical symptoms. In such cases, argued Bastian:

there may, after a longer or shorter period, be complete recovery, either owing to the disease being originally produced by some mere functional defect, or by the gradual establishment of a new "way out" for speech incitations, namely from the left to the right third frontal convolution (through fibres of corpus callosum) and thence downwards through the right hemisphere to the bulb, as originally suggested by Broadbent (Bastian, 1887, p. 987).

It is apparent, therefore, that Bastian was not opposed to the idea of right hemisphere takeover of language function after damage to the left hemisphere *per se*. Contrary to common medical opinion, he simply did not believe that the Barlow case could be interpreted as providing strong, direct support for that hypothesis.

Does the case, as presented in the archives, support the involvement of the right hemisphere in recovery of language function after aphasia? In what is described in the archived case notes, it is open to question that the child ever fully recovered from the first aphasic/ hemiplegic attack. Undeniably, though, his condition did improve. The right hemisphere may well have been involved in that improvement, as indeed might surrounding regions of the left hemisphere. What is clear is that the less than perfect recovery pattern, as detailed in the daily hospital notes, makes for a more imprecise picture than that outlined by Barlow. Indeed, discrepancies in the description of the clinical picture, as detailed in the archived notes, call into question the actual significance of the clinical pathological correlation.

Barlow's 1877 case study is considerably less important to the equipotentiality hypothesis than it is to theories of language localisation and right hemisphere involvement of language recovery. Equipotentiality, as discussed in this article, refers to the late 19<sup>th</sup> century hypothesis that both hemispheres, at birth, had the potential to control language function, with only later lateralisation of that function, typically in the left hemisphere. This hypothesis, focusing on the role of the two hemispheres in the development of language skills, while building on the earlier 19<sup>th</sup> century debate concerning brain structure and organisation of mental faculties, was not a return to the equipotentiality hypothesis proposed by Flourens.

How would a rereading of the case affect the equipotentiality hypothesis, which, unlike the language localisation hypothesis, was in its infancy when the case was published? Samuel Wilks, writing in 1883, makes no mention of the Barlow case. Edmund Cautley uses the case as supporting evidence six years later, but omits all

mention by 1910. Cautley may have re-evaluated the Barlow case following Bastian's (1898) article (see below). Cautley's (1910) Diseases of Childhood was one of the leading paediatric textbooks of the day. Two decades after his report to the Abernethian society and more than 30 years after the publication of the Barlow case, his views on the aphasic condition had altered somewhat. He acknowledged aphasia to be associated most frequently with cases of right hemiplegia. The condition was usually temporary, with recovery "taking place by the speech centre on the opposite side taking the functions of the one destroyed or damaged" (Cautley, 1910, p. 678). However, there were exceptions to this prognosis, i.e., cases of bilateral lesions or "one on the left side in later childhood." The patient's age at lesion onset is viewed as an important factor in recovery of language function after brain injury: "The speech centre is supposed to be in the posterior third of the left frontal convolution (Broca). It is bilateral at birth. The left one normally develops, but if it is damaged or destroyed in early life, the right takes on its functions. Aphasia in the first four years of life is soon recovered from" (Cautley, 1910, p. 790). The Barlow boy was ten years old at lesion onset and therefore presumably, given the above statement, less likely to recover rapidly.

Sachs and Peterson, as already mentioned, did not cite Barlow. Edmund Cautley's Abernethian article, in which the published case is mentioned as supporting evidence, is not cited in any of the main aphasic papers of the time (nor indeed, is it cited later). The sole English language paper on the subject of equipotentiality, widely cited well into the 20<sup>th</sup> century, is Sachs and Peterson, 1890. As described in the archives, the case would most likely not have been mentioned by Cautley either.

Interestingly, despite serving as physician at GOSH for three more decades, Barlow did not (at least to the knowledge of the authors) mention the case in print again. He makes no comment on its various interpretations. It is tempting to conclude that he might not have agreed wholeheartedly with them.

Although consideration of the original archived case notes now throws some doubt on crucial details of the Barlow case, it remains the most important and widely cited 19<sup>th</sup> century case of acquired childhood aphasia in English. The case, as reported in the BMJ by Barlow, directly influenced perceptions of the language disorders and brain function for generations. Due to the evidence presented by Barlow, influential British physicians found themselves in agreement with their continental colleagues who argued that aphasia in childhood was a transient condition. One important consequence was the marginalisation of acquired childhood aphasia research for many decades.



## References

- Barlow T (1877): On a Case of Double Hemiplegia, with Cerebral Symmetrical lesions. *The British Medical Journal* 2: 103-104.
- Basser L (1962): Hemiplegia of early onset and the faculty of speech with special reference to the effects of hemispherectomy. *Brain* 85: 427-460.
- Bastian HC (1887): On Different Kinds of Aphasia, with Special Reference to their Classification and Ultimate Pathology. *The British Medical Journal*, 2. 931-936, 985-990
- Bastian HC (1898): *A Treatise on Aphasia and Other Speech Defects* London:Lewis.
- Bramwell B (1897): Lectures on Aphasia. *Edinburgh Medical Journal* 11: 1-13, 117-128, 232-245, 356-370, 454-465, 527-551.
- Broadbent W (1866): An attempt to remove the difficulties attending the application of Dr Carpenter's theory of the function of the sensori-motor ganglia to the common form of hemiplegia. *British and Foreign Medico-Chirurgical Review* 37: 468-481.
- Broca P (1861): Remarques sur le siège de la faculté du langage articulé, suivies d'une observation d'aphémie. *Bulletin de la Société d'anthropologie* 6: 330-357.

Broca P (1865): Du siège de la faculté du langage articulé dans l'hémisphère gauche du cerveau. *Bulletin de la Société d'Anthropologie tome VI*: 377-393 translated by Berker EA, Berker AH and Smith A (1986): Localisation of speech in the third left frontal convolution. *Archives of Neurology* 43: 1065-1072.

Buckingham HW (2003): Walter Moxon and his thoughts about language and the brain. *Journal of the History of the Neurosciences* 12: 292-303.

Cautley E (1889): Aphasia and Allied Conditions. *St Bartholomew's Hospital Reports*: 263 –270.

Cautley E (1910): *The Diseases of Infants and Children*. London: Shaw & Sons.

Darwin C (1877): A Biographical Sketch of an Infant. *Mind* 2 (7): 285-294.

Edwards W (1899) (Ed.): *Cyclopaedia of the Diseases of Children*. London: Medical and Surgical Press.

Ferrier D (1878): The Localisation of Cerebral Disease. *British Medical Journal* 2: 399-402, 443-447, 471-476, 515-519, 555-559, 591-595.

Finger S, Buckner R and Buckingham H (2003): The Thomas Barlow case of 1877. Did the right hemisphere take over speech after damage to Broca's area? *Brain and Language* 85: 385-395.

Gowers WR (1885): *Lectures on the diagnosis of diseases of the brain*. London: J&A Churchill.

Harrington A (1987): *Medicine, Mind and the Double Brain*. Princeton: Princeton University Press.

Hellal P (2005). *Acquired Childhood Aphasia: Historical and Theoretical Perspectives*. Doctoral dissertation, Birkbeck College, University of London.

Hellal P and Lorch M (2005): Charles West: a 19th century perspective on acquired childhood aphasia. *Journal of Neurolinguistics* 18: 345-360.

Hunter KM (1991): *Doctors Stories. The Narrative Structure of Medical Knowledge*. Princeton University Press.

Jackson JH (1864): Loss of speech : its association with valvular disease of the heart and with hemiplegia on the right side.- Defects of speech in chorea. *Clinical Lectures and Reports, London Hospital 1*: Reprinted 1915: *Brain* 38: 28-42.

Jackson JH (1890): On convulsive seizures (Lumleian Lecture). *British Medical Journal* 1: 821-827.

Jacyna S (2000): *Lost Words. Narratives of Language and the Brain 1825-1926*. Princeton University Press.

Lees J (1993): *Children with Acquired Aphasias*. Whurr Publishers Ltd. London.

Lenneberg E (1967): *Biological Foundations of Language*. New York: John Wiley.

Lomax E (1982): A Mid-nineteenth Century British Pediatrician's Interpretations of the Mental Peculiarities and Disorders of Childhood. *Clio Medica* 17: 223-233.

Lomax E (1996): *Small and Special: The Development of Hospitals for Children in Victorian Britain*. *Medical History*. Supplement No. 16. London: Wellcome Institute for the History of Medicine.

Lorch M (2004): The unknown source of John Hughlings Jackson's early interest in aphasia. *Cognitive and Behavioral Neurology* 17: 124-132.

Marien P, Paquier P, Cassenaer S and De Deyn P (2002): Early development of concepts and hypotheses. *Journal of Neurolinguistics* 15: 129-142.

Moxon W (1866): On the connection between loss of speech and paralysis of the right side. *The British and Foreign Medico-Chirurgical Review* 37: 481-489.

Risse G and Warner J (1992): Reconstructing clinical activities: Patient records in medical history. *Social History of Medicine*: 182-203.

Ross J (1887): *On Aphasia: being a contribution to the subject of the dissolution of speech from cerebral disease*. London: J&A Churchill.

Sachs B and Peterson F (1890): A study of cerebral palsies of early life, based upon an analysis of one hundred and forty cases. *Journal of Nervous and Mental Diseases* 17: 295-332.

Sully J (1881): Babies and Science. *Cornhill Magazine* 43: 539-54.

Trousseau A (1864): De l'aphasie, maladie décrite récemment sous le non impropre d'aphémie. *Gazette des Hôpitaux*, 37 : 13-14.

West C (1848): *Lectures of the Diseases of Infancy and Childhood*. London. 2nd Ed 1852. 3rd Ed 1854. 4th Ed 1859. 5th Ed 1865. 6th Ed. 1874. 7th Ed 1884.

Wilks S (1883): *Lectures on Diseases of the Nervous System*. London. Churchill.

Wyllie J (1892): The Disorders of Speech. *Edinburgh Medical Journal* 1 & 2: 289-314, 401-421, 501-523, 585-604, 681-693, 777-793, 897-907, 977-992.

Young R (1990): *Mind, Brain and Adaptation in the Nineteenth Century*.

Oxford: Oxford University Press.

Table 1: Discrepancies between Dickinson's Case Notes and Barlow's BMJ Article

### Expressive Language Skills

#### After the First Attack--

Barlow: "The speech had returned on the 10<sup>th</sup> day"

Dickinson: "The speech began to return on the 10<sup>th</sup> day"

#### After the Second Attack--

Barlow: From the first he was able to write his name when asked and after a few weeks would answer in writing any question that was put to him."

Dickinson: No mention is made of the boy's ability to write anything other than his own name throughout. He "never cared to read after the attack".

### Motor Function

#### After the First Attack--

Barlow: "In 10 days he was greatly improved. The leg improved before the arm".

Dickinson: "In 10 days began to improve. First improved in leg. Improved a little in right hand but never got full use of it."

#### After the Second Attack--

Barlow: "He could stand ...without support."

Dickinson: "When carried in [on admission] he was laid on the couch."

