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Albert Einstein's Dyslexia and the Significance of Brodmann Area 39 of His Left Cerebral Cortex

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Abstract — By his own admission, Albert Einstein, '*started to talk comparatively late...certainly not younger than three*', and also had '*poor memory of words*', during his childhood years. If lesions in Brodmann Area 39 of the cerebral hemisphere results in dyslexia, the 1985 report on the study of Einstein's brain that the neuron:glial ratio of Area 39 in the left cerebral hemisphere of the physicist was significantly smaller than that of the control values, provides a neuroanatomical clue to Einstein's childhood dyslexia. Though not discrediting this finding, some questions are raised in this paper regarding the controls employed in this 1985 report (1).

Introduction

It is well known that the physicist Albert Einstein (1879–1955) had difficulty in speech development during his infancy and childhood (2, 3). As he recalled in a letter he wrote in 1954,

> *'My parents were worried because I started to talk comparatively late, and they consulted the doctor because of it. I cannot tell how old I was at that time, but certainly not younger than three' (4).*

As his biographer Philipp Frank noted, while the subject was alive, '*Even when Albert was nine years old and in the highest grade of the elementary school, he still lacked fluency of speech*' (2).

One of Einstein's associates, Straus, who heard the physicist's explanation of his slow development of speech, reminisced in 1979,

> *'He (Einstein) said that when he was between two and three years old he formed the ambition to talk in whole sentences. If somebody asked him a question and he had to answer, he would form a sentence in his mind and then try it out on himself, thinking that he was whispering it to himself. But, as you know, a child is not very good at whispering so he said it softly. Then, if it sounded all right, he would say it again to the person who had questioned him. Therefore, he sounded, at least to his nursemaid, as if he said everything twice, once softly and once loudly, and she called him, "der Depperte", which is Bavarian for*

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"the dopey one". The nick name stuck, and that, at least in Einstein's mind, was the cause of all the stories about his slow development. As to the story about his dyslexia, I had not heard it, and he never commented on it' (5).

As revealing as this reminiscence may be, Straus did not mention when Einstein told him this story. Circumstantial evidence shows (that Straus was 3 years old when Einstein first visited their house in Munich in 1925) that Einstein would have reached at least 50 years by the time he told the above cited version of his childhood days to Straus. In his autobiographical notes, written at the age of 67, Einstein also has cautioned about over-reliance of old-age reminiscence; '*Every reminiscence is colored by today's being what it is, and therefore by a deceptive point of view*' (6).

Noted psychoanalyst Erik Erikson also had analyzed Einstein's late development of speech in terms of his speciality, while observing, '*I shall leave to specialists any attempt to diagnose little Albert's symptoms in the light of today's knowledge (on dyslexia, say or on the two sides of the brain) and discuss them rather in the context of his overall development*' (7). What Erikson had acknowledged is the fact that speech development involves a tripodal interplay of psychological, physiological and anatomical factors (8).

Since the observations of Straus (5) and Erikson (7) were made in the Jerusalem Einstein Centennial Symposium held in 1979, long before the publication of the histological analysis of the neuron:glial ratios of Einstein's cerebral cortex (1), one can reappraise the rationale for Einstein's late development of speech and his self-acknowledged, '*poor memory of words*' (4).

Data on the histological analysis of Einstein's cerebral cortex

In 1985 Diamond et al. (1) reported their findings on the neuron:glial ratios of Brodmann Areas 9 and 39 of the left and right cerebral hemispheres of Einstein in comparison to those of the brains of 11 males, '*47 to 80 years of age who had died from nonneurologically related diseases*'. The average age of the control subjects was 64 years, while Einstein was 76 when he died. The brains of the control subjects were obtained from the Veterans' Administration Hospital in Martinez, California. The interesting findings of this histological analysis were:

1. The neuron:glial ratios of all 4 cerebral cortex areas of Einstein studied were smaller in comparison to the control subjects. However, in 3 areas (Area 9 of left hemisphere, Area 9 of right hemisphere and Area 39 of right hemisphere) the difference was not statistically significant.

2. The neuron:glial ratio of Area 39 of Einstein's left cerebral hemisphere was significantly smaller than that of the control values of Area 39 of left hemisphere.

Since Brodmann Areas 39 and 40 are the speech-related areas which show extreme enlargement in the hominid cerebral evolution and that Areas 39 and 40 are, '*the latest to myelinate of all areas on the convexity of the human cortex*' and that this myelination process is '*delayed until after birth and dendritic development and cellular maturation, may not be completed until late childhood*' (9), it can be inferred that the neuroanatomical evidence (which was lacking till 1985) for Einstein's late development of speech, is thus established. Computerized tomographic studies in aphasic patients which became available in the mid 1970s also revealed that lesions in Brodmann's Areas 22 and 39 led to aphasic speech, poor repetition and significant auditory comprehension deficit, characterized as Wernicke's aphasia (10). It seems plausible that Einstein's late development of speech can be attributed to a delay in myelination, dendritic development and cellular maturation in Brodmann Area 39 of his left cerebral hemisphere.

Einstein's cephalic features

According to his sister Maja's biographical sketch written in 1924, after Einstein's birth, their

'mother was shocked at the sight of the back of his (Einstein's) head, which was extremely large and angular, and she feared she had given birth to a deformed child. But the doctor reassured her, and after a few weeks the shape of the skull was normal. The child, very heavy from the outset, was always quiet and required little care ...' (11).

However, the angular cephalic features of

Einstein were prominent even in his adult years. When he made his visit to Paris at the age of 43, the astronomer of Paris Observatory, Charles Nordmann provided a sketch of these prominent feature as follows:

> *'His (Einstein's) skull is clearly, and to an extraordinary degree, brachycephalic, great in breadth and receding towards the nape of the neck without exceeding the vertical. The skull of Einstein reminds me, about all else, of that of Renan, who was also a brachycephale. As with Renan the forehead is huge; its breadth exceptional, its spherical form striking one more than its height'* (12).

It is also interesting to note that, Charles Darwin in his book, *The Descent of Man* (published 8 years before the birth of Einstein in 1871), mentions the 1868 observations of T.H. Huxley in a foot-note that, *'the skulls of many South Germans and Swiss are as short and as broad as those of the Tartars'* (13). Einstein was a native of southern Germany, and his ancestors had lived in Buchau, a small town between Lake Constance and Ulm (where Einstein was born), since the 1750s (12).

Some questions about the 1985 study

It is pertinent to raise some questions about Diamond et al.'s study (1) regarding the controls employed for comparison with Einstein's brain. First, this study was published almost 30 years after the removal of Einstein's brain following his death in 1955. The reasons for this undue delay in the experimental analysis has not been satisfactorily explained by the physician Thomas Harvey who removed Einstein's brain for study (14, 15). Secondly, the details provided by Diamond et al. (1) about their control subjects are somewhat incomplete. Did all the control subjects belong to Caucasoid heritage as Einstein was? Thirdly, Diamond et al. (1) had obtained the brains of control subjects from the Veterans' Administration Hospital in Martinez, California. Is it not more convincing if the brains of control subjects were collected from the region in which Einstein was born, i.e. southern Germany? Fourthly, Diamond et al. (1) did state in their description that

> *'chronological age is not necessarily a useful indicator in measuring biological systems. Environmental factors also play a strong role in modifying the condition of the organism. One major problem in dealing with human specimens is that they do not come from controlled environments'.*

If this is so, an optimal control sample for comparing with Einstein's brain should come from the males of Ulm, Germany, who were born within a decade or two around 1879 (Einstein's year of birth). Fifthly, the manner in which the data obtained were presented in the publication leaves much to be desired. One figure was provided to show the locations (Areas 9 and 39) from which the samples were removed for cell counts. Collected data had been summarized in only one table. Even in this table, absolute numbers of the cell counts determined for Areas 9 and 39 were not provided for both Einstein's brain and the 11 control brains. Only the neuron:glial ratios were indicated.

Conclusion

According to Eccles (9), lesions in Brodmann Area 39 lead to dyslexia. The 1985 finding of Diamond et al. (1) that the neuron;glial ratio of Area 39 in Einstein's left cerebral hemisphere was significantly smaller than that of the control values of Area 39 of the left cerebral hemisphere suggests a strong possibility of some kind of lesion of this specific speech-related area in Einstein's brain which could have resulted in childhood dyslexia. However, some questions relating to the controls employed in the 1985 paper by Diamond et al. (1) need critical evaluation. In the absence of additional neuroanatomical reports exploring other areas of Einstein's brain, one cannot conclude about whether Einstein suffered from any other types of aphasia (10, 16) during childhood and how he recovered later.

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