

DYSLEXIA

NEURONAL, COGNITIVE & LINGUISTIC ASPECTS

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INTRODUCTORY REMARKS

RAGNAR GRANIT

We take pleasure in hearing from our Cabinet Minister, Mrs Britt Mogård, that the Ministry of Education is concerned about the serious problem whose understanding we are trying to promote by inviting a number of distinguished experts to discuss it. Developmental dyslexia appears in otherwise intelligent school children as a shortcoming in processing spoken and written language so well, as one would be entitled to expect from them. Some improvement, often quite considerable, takes place as they grow up, indicating the operation of mechanisms of compensation.

We shall hear at this Conference that dyslexia in some cases is likely to be the sign of structural deficiencies in the language centres. If in such cases some improvement takes place the compensatory processes are likely to operate outside those centres. We know from much physiological and clinical work that the plasticity of the young brain is of a remarkable order. But wheresoever the compensatory events take place, their progress and nature deserve to be followed with particular attention. To the science of pedagogy, keen on mitigating the tribulations of dyslectic children, study of nature's ways in compensating for their deficiencies offers means of devising curative measures.

I cannot speak about the field of dyslexia with the authority of our distinguished guests. It is not my own. Faced with the enormous number of papers in this field, an outsider like myself must thread warily through the brushwood of tests designed to extricate factors such as phonemes, words, sequences, intervals, iconic memory etc. The physiologist likes to stand closer to his material than is possible in the study of dyslexia, simply because the only speaking animal cannot be investigated with the microelectrodes that have meant so much for understanding the way in which the brain handles information. Does this dearly acquired knowledge mean anything also for the understanding of dyslexia?

Assuming validity of the principles discovered with single cortical cells in, for instance, vision, the neurons in speech centres would also exhibit re-representation and recombination of elements of information, whose nature so far is undetermined. There is nothing inherently unlikely in postulating that any single phoneme is represented with an acoustic and a visual element in single cells, assuming a phoneme being the real unit like, say, spatial frequency, orientation or direction in vision. If so, what might be called a phonemic cell would recombine with equivalent partners to form groups representing words. The cascading recombinations contain so many transformations, any one or all of which can go wrong, that it seems impossible to conclude that dyslexia is just one single defect. Perhaps it is easier to understand a comparison with the precision grip for which the cortical thumb area alone is enormous relative to, for instance, the area of the great toe. There is a correspondingly large variety of ways in which the precision grip can go wrong considering the muscles involved.

These comments on cortical physiology may seem discouraging but they at least serve to underline that advance in understanding the nature of dyslectic deficiencies in verbal processing in the end will depend on progress in the understanding of everything connected with the language centres. One ray of hope in this endeavour is provided by the research on the lateralization of speech and its development in the young. Knowledge in this domain has ancient roots in the clinic but it has received a fresh impetus from the ideas and experiments of Roger Sperry on split brain patients.

One decisive observation from this many-faceted research has stirred up the imagination of people in different fields: I mean the fact that the right hemisphere actually has a substantial vocabulary and responds to a limited number of verbal instructions though in these commissurotomed patients it is lacking access to verbal communication in the centre that we associate with the name of Broca. The silent hemisphere is restricted to pointing and drawing when compelled to identify objects. From the point of view of dyslexia it would seem to be of great interest to study the development of language in early commissurotomed or left-side hemispherectomized patients. Pre-existent cognitive properties of the silent hemisphere must then adjust to verbal communication. Will it then become normal at a lower level or will it become dyslectic? Perhaps some of the experts at this conference know the answer. I have not been able to find it in the studies available to me of speech development in such patients.

We look forward to having from the experts at this conference the latest experimental results and conclusions concerning dyslexia in relation to the lateralization of speech. I wish we knew why evolution has put a premium on making one side only of the brain handle verbal processing. As such functional localization

is something to expect. The more we have learned about the physiology of the brain, the greater has become our conviction that every important function has been allotted its own site and cells in the cortex. We are less well informed about factors of interaction, suppression and rivalries between such sites. What I mean is well exemplified by the fact that when tying up the eye lids on one eye of a kitten, the normally binocular cortical cells become monocular. Or one might mention the suppression of the ipsilateral acoustic input in dichotic listening.

We cannot neglect the possibility that for unknown reasons blocks of inhibition, ultimately stemming from interhemispheric interaction, might be responsible for dyslectic disturbances. In short, we shall have to take seriously the proposals of authors in this field ascribing dyslexia to a variety of causes connected with lateralization, familiar to the participants of this conference. No one will deny that there are major dyslectic types such as the auditive and the visual form. There may well be a large number of less well definable dyslectic deficiencies, as already pointed out in discussing elementary principles of cortical physiology.

I have said nothing about cures for dyslexia but obviously this is a major interest of this conference. I have followed the well trodden path of believing that theoretical knowledge of the mechanisms engaged in verbal processing in the end must lead to improvements in the treatment of dyslexia. I am convinced that this attitude is shared by the participants of this meeting.

I will finally use this opportunity to thank them all and, in the first instance, our invited guests, who have taken the trouble to join up and to prepare papers for the conference.