DYNAMIC FACTORS IN APHASIA.

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THE close relation of aphasia to lesions of particular brain areas may be responsible for the fact that factors other than lingual in their effects upon aphasic reactions have long been neglected. Pierre Marie (1906) came to the conclusion that in true aphasia the defect in language is regularly accompanied by a general intellectual defect. This opinion found little recognition; his statement was too general, and was not found to be in agreement with clinical experience. Head (1926) made a more elaborate attempt to break with the traditional ideas when he introduced the conception of symbolic expression and formulation, and viewed the clinical pictures of aphasia which came under his observation from a more general angle. Goldstein (1924, 1926), in applying the Gestalt conception to aphasic disorders, brought the brain function as a whole into the picture. He found the basis of aphasic disorders in the inability of figure-background formation and categorical behaviour. Such extra-lingual factors have consequently been considered in the analysis of particular cases by several authors (Woerkom, 1925, 1931), Bowman and Gruenbaum (1925), Klein (1929, 1931, 1932). Demonstrated on two aphasic patients, they are also the subject of this paper.

The first patient, an efficient district nurse, single, aged 52, had been on two previous occasions in this hospital with a depressive picture. A week before her present admission she was found holding her head and talking in a disconnected, incomprehensible way. She seemed to be unable to grasp what was said to her. As her condition did not improve she was taken to hospital. On admission her speech consisted mainly of severe, jargon-like paraphasias which made it mostly impossible to obtain an idea of what she wanted to express. This is an example of her spontaneous speech in conversation: " What my power . . . watch to stone . . . my drop, my pass . . . is te sooh gone . . . I am staff, chope, chook . . . my harage gone . . . my gone . . . younger beings gilt . . . my joid . . . I wish Gods fuddle . . . ." She made great efforts to express what was in her mind. There was a consistent attempt to put into words something definite and concrete; this was demonstrated by utterances which were partly correct and could be interpreted. She frequently supplemented her deficient speech by gestures and expressions. She often tried to correct her mistakes, and expressed her impatience and discontent by facial expressions when she did not succeed. Her speech was slow and it took some time before she started, although she had no difficulties in articulation. Occasionally when she did not succeed in expressing herself satisfactorily she tried to spell out the words, with an equal lack of success. The naming of objects was met by similar difficulties as her conversational speech. She could rarely give a correct answer. The word-confusion was mainly literal and there was little resemblance to the correct name. She was unable to repeat syllables or words, nor could she produce word series. During conversation there was never any tendency to take up in her speech words or sentences of the examiner. There

was a very severe word-deafness. She only occasionally grasped part of a simple question or order given to her, and was unable to pick out a particular object named from a number exposed. Her reading aloud was paraphasic similarly to her spontaneous speech, but she understood written language slightly better than the spoken word. Her writing was very poor. She produced incomplete words when asked to write spontaneously or on dictation. Copying was better; it started well, but became confused later. She could not be induced to draw or to copy drawings. She knew how to use objects, and carried out correctly simple actions such as combing, striking a match, etc. A more detailed testing of constructive abilities was not possible, partly because of her word-deafness. On neurological examination a dropping of the right angle of the mouth at rest and at innervation was found. There were no other pathological signs of the C.N.S. The blood pressure was 240/135 mm. Hg, and the retinal vessels showed arteriosclerotic changes. In the course of a three-month observation the speech improved slowly. She was then able to express herself better, but word paraphasias were still marked and constructional mistakes now became evident. She still showed considerable disturbance in comprehension. The character of the speech defect had remained the same.

The second patient, a labourer, 58 years old, was at work up to a week before admission. He was said to have been a sociable man and a reliable worker. He complained of heavy pain in the head. After a few days' rest in bed he became extremely voluble and started to talk nonsensically. Then he became irritable, excited and suspicious, and had to be taken to hospital. When admitted he had a tendency to logorrhoea. He kept on talking until interrupted. The following is an example of his conversational speech: "Well not too bad, I suppose, it is not too well, let me see, I should not be able to get a place anyway, well not too much I suppose, there is nothing wrong with me, when I went to the first place, just the same, yes doctor just the same. I came round with her, not too bad, she used to be with me all my life." Similar phrases made up most of his conversation; occasionally some word confusion slipped in, as: "I don't care to be forminded, I don't forget my gent to-day, it is all you have to is my idents, my ardents that you met me doctor." He often reacted before the examiner had finished his question. The sentences were usually in good grammatical order and spoken with the correct intonation. There was perseveration in his speech. Though the sentence formation was usually correct it conveyed little meaning and there was little interconnection between his utterances. Though he frequently repeated correctly some of the expressions of the questions put to him during a conversation, he did not do so when specially tested for repetition. Neither did he continue word series. In naming objects he rarely succeeded in finding the proper expression. He produced severe paraphasias at the beginning of the test; his stock phrases followed if they had not made their appearance from the start. His comprehension was very much impaired. He rarely understood the spoken word either in conversation, or on simple orders. Often he began with a relevant response, but soon lost the trend of his thought and carried on with his usual phrases. Only now and again did he read a word correctly; mostly his reading was severely paraphasic. He did not show any sign of understanding the written word. His writing, both spontaneous and on dictation, was strongly paragraphic and persevatory; copying was much better, but not without mistakes. He did not draw from memory, possibly because he was unable to understand what he was asked to do. Copying of drawing was accurate. He had difficulties in putting together pieces of a simple jigsaw puzzle; he did it in a haphazard way, without planning. He did not know what to do with a cigarette lighter, matches, keys, when given to him for handling. There was no pathological finding on neurological examination. W.R. was negative. Blood pressure was 160/100 mm. Hg. In the course of an observation over a period of 3½ months the flow of speech became slightly less, his comprehension became better. At times his answers in conversation were more to the point. His writing on dictation improved slightly. Otherwise the picture had not changed. Stock phrases predominated in his speech reaction and there was still considerable perseveration.

When we compare these two aphasic patients we find that they are unable to express themselves, to name an object by its correct name, to understand

what is said to them, to write or to read properly. Yet the total impression we obtain from their speech reactions is entirely different in the two cases. This is specially striking in the conversational speech. In the first case the speech is slow, the sentence formation is incomplete, word-confusion with literal paraphasias and perseveration is very marked. In the second patient the flow of speech is easy and, if anything, increased, the sentence formation is good and mistakes are not frequent. Word-confusion when combined as in these cases with disturbances in comprehension, reading and writing can be regarded as a disorder of the internal language. Since this disorder seems to be of about the same gravity in both cases, the entirely different character of their speech behaviour must be due to other factors. Though it is true to say that the conversational speech of the second patient is almost correct, this is so only from a purely linguistic point of view. Otherwise it is not a satisfactory speech performance; it consists of ready-made phrases of general character, with little or no inter-connection between them and little specific meaning. It is a kind of speech similar to that which Jackson called inferior speech, utterances which appear involuntarily as an emotional or automatic speech response. Such speech is, according to Jackson, organized at a lower level, and can often still be retained when functions of a higher order have broken down. This explanation may also apply to the retention of phrases of inferior quality in our case. As they are more automatized we might expect their greater resistance to destruction. But the problem remains that the automatic speech material appears so readily in one case and not in the other, although the disturbance in linguistic capacities is of the same severity in both cases. Explanation is therefore needed of the fact that the speech progresses in one case whenever possible in an automatic way while it is disautomatized in the other. As it is difficult to make the second patient produce speech other than that of a lower order, so it is impossible to induce automatic speech in the first patient. While articulatory difficulties are absent there are indications in the first case that the speech function is inhibited. Speech is slow, the sentences short and often incomplete with prolonged intervals between them. The reaction time is prolonged. There is lack of inhibition in the second case; the reaction time is shortened, there is a continuous flow of speech, with scarcely any intervals until the patient is stopped. This conforms with the disautomatization of speech and speech automatism respectively.

Inhibition and release must be due to some regulatory mechanism preceding speech formulation. It can be assumed that some scheme of thought is, as a rule, precedent to speech. This scheme may consist of a particular line of thought, a certain trend without a definite structure, or it may be structural thinking. This thought structure may be only loose and become more definite in the course of speech evolution, or it may be a more tight and well-formulated structure. The choice left to the subsequent speech formulation to follow its own pattern can therefore vary considerably. Under normal circumstances it is largely dependent upon the speech situation, the purpose of speech and probably the type of personality. There is, however, under ordinary conditions no difficulty in adjusting speech resources to the preceding scheme. Under pathological conditions, when general cortical functions

are involved or when a speech disorder is present, adjustment between thought and speech can be expected to become more difficult and the progression of speech may consequently be affected.

There is evidence of such difficulties in the thought-speech relationship in our two cases. The speech of the first case is controlled. The patient is trying to express something definite. She adheres strictly to a precise scheme of thought in her speech formulation. She endeavours to cover and express by her speech construction and choice of symbols a preconceived thought structure or conception, as exactly as possible whatever the situation is, whether conversational or test. As this effort at accuracy and completeness is too ambitious for the language resources at her disposal she is bound to fail, with a resulting constructional and word confusion. At the same time it prevents the release of more automatized speech, which potentially she is still able to perform. In contrast no precise thought structure precedes speech formulation in the other patient. When such structure emerges or is enforced upon him he takes it up in a casual way only to drop it, and his speech again follows a vague line of thought or, if at all, loosely structural thinking. This is particularly striking when he has to name an object. In his first attempt to do so he may produce a paraphasic word which is intended to express his conception, but he is immediately carried away by his usual phrases. Without the restriction of more concise thought such speech material is therefore set free which is in the range of his reduced language capacities. These cover only automatized speech, and this is, in fact, of what his conversational speech consists.

The two aphasic pictures here described, though common in their main structures, are difficult to put into any scheme. The first patient may give the impression more of a motor, the second of a sensory aphasia, but they have both the same heavy disturbances in comprehension. In analysing aphasic reactions we have to distinguish between actual loss of language abilities, which can be expressed as a disorder of internal language or the loss of symbols of language, and the way the afflicted person turns the remaining language capacities into speech. It is the distinction between language as a potential capacity and speech as a function: one is a static factor, the other involves a dynamic factor. The available language resources in aphasia can vary according to the severity of the lesion, but their difference in aphasic patients is only quantitative. The way, however, the remaining language capacity is utilized for speech varies in character. The factors involved are of peripheral (innervatory) and central nature. The peripheral factor interferes only in certain cases with the execution of language and is not under consideration here. The central mechanism is always involved as a factor which influences the speech progression in one way or another. In Goldstein's opinion the brain mechanism as a whole is altered whenever a functional disturbance occurs in brain lesions; there is a difficulty in integrating adequately the particular functional disorder into the brain function as a whole, consequently the figure-background formation becomes faulty and this gives rise to abnormal reactions. The figure-background formation is probably faulty in both our cases. But more specific factors are needed to explain the difference in their aphasic reactions. We found the nature of the preceding thought process,

the way in which it mobilizes the existing language resources, the determining factor. If the patient approaches his reduced language capacities by attempting to shape into speech a tight thought structure he is bound to fail altogether. If such a structure is more or less absent, speech automatism can freely develop. The first approach can be called global, the second focal.

The global approach is not always so marked as in our first case. There are speech situations, as for instance in word series and repetition, which specially facilitate an automatic approach. The patients may be able to give up their essentially global approach in these situations. Variations of this kind may be due to individual factors. We may expect tension to arise when, as in this group of aphasics, a set scheme has to be actualized by inferior means. The degree of tension, and with it probably the degree of speech inhibition and word confusion, may depend upon the make-up of the personality. There are further, the premorbid speech habits to consider. They may influence the style of the aphasic reactions, and favour either a global or focal approach to speech situations and so intensify or mitigate one or the other reaction type.

In the second group, in which the focal or automatic approach prevails, Marie's assumption of a special dementia seems to have some justification. There is in our second case, in contrast to the first case, an absence of ordered thinking, not only connected with speech, but with other functions as well. His thought process is erratic, his thinking is poorly formulated and incomplete, but this in itself does not produce the aphasia. We may, however, expect in this group of aphasics the specific language disorder to be frequently associated with a disturbance of general brain functions.

A process which involves both functions in the course of the illness is senile dementia. On a series of seniles and preseniles of the Alzheimer type which we observed, three stages of speech disintegration could be distinguished. In the first stage difficulties in naming appear, the speech is somewhat impoverished, but there are otherwise no definite signs of a disturbance of the internal language. In the second stage the flow of speech is increased, the speech becomes simpler, consisting mainly of phrases of little variety. Spelling and writing is impaired, word and literal paraphasias appear and in details understanding is faulty. In the third stage there are no spontaneous speech impulses; the patients respond with a few stereotyped phrases, often incomplete, or they mostly repeat part of what is said to them. Comprehension is greatly reduced or absent. In the first stage the speech resources are slightly involved whilst the intelligence has noticeably decreased. When therefore the speech evolution follows loosely structured thought no difficulties arise. In situations where a precise conception has to be formulated, as in naming, difficulties occur. In the second stage a marked loosening of thought structure and difficulties in internal language are present. There is little thought direction, no ordered scheme of thought which might tie up or restrict speech evolution. Accordingly, the flow of speech is increased and speech is of a lower order; word confusion appears mainly when a global approach is enforced upon the patients. In the last stage both intellectual faculties and internal language are of a very low level. In the absence of intellectual activity there is no spontaneous impulse to speech; reactively only the most automatized

and uncontrolled speech formation, such as repetitions and a very small set of the simplest phrases, is possible. These observations illustrate the automatic approach to speech situations throughout, when in the progression of a general brain process intellectual functions and internal language are involved at the same time. They show also that the automatic approach becomes more marked with the increasing disintegration of thought, and the speech automatisms more primitive with the progression of internal language difficulties.

Conclusion.

In the differentiation of aphasic reaction types two factors have been separated—the defect of internal language and the way in which the defective language resources are mobilized. This mobilization, as demonstrated on two cases, was found mainly dependent upon the approach to speech by the preparatory thought process. Two different ways of approach were distinguished:

(1) A global approach where ordered and structured thought precedes language formulation, enforcing itself on language construction. This rigid adherence to an elaborate scheme, which is too ambitious for the reduced language resources, leads to speech inhibition, constructive and word-confusion. (2) A focal or automatic approach in which structural thinking is very loose or altogether absent. There is therefore no restricting factor, no straight jacket for speech evolution, and speech can progress freely along lines of existing language capacities. Speech material of inferior order will therefore be released in an easy flow of speech. The interaction between progressive intellectual deterioration and progressive internal language disorder and its effect upon speech has been followed up in a series of seniles and preseniles.

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