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APHASIA RECONSIDERED

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The history of Aphasia research can be divided in three stages. The first was a result of the 19 Century Clinical studies; the second developed during Last few decades; the Last has just begun. We shall try to describe these stages separately.

1. The classical approach

The Basic concepts of aphasia formulated by P.Broca in the 1861 and by C.Wernicke in 1873 have long ago became classical. They are well known, and we can dwell on their principles only briefly.

When P.Broca for the first time described the syndrome of motor aphasia resulting from lesions of the posterior parts of the third frontal convolution of the left hemisphere, he supposed this area to be "a centre for motor images of words". On a similar basis C.Wernicke formulated his concept of "sensory aphasia" supposing that the posterior parts of the upper convolution of the left temporal lobe are a center for "sensory images of words".

The idea that isolated parts or the "speech areas" of the left hemisphere can be "centres" for complicated speech functions was widely accepted and became one of the basic ideas of classical Neurology.

In a very short time a series of such "Centra" were discovered and new foci of aphasia - each having as a base lesion of certain circumscribed cortical areas - were

described. As a result-amnestic aphasia which resulted from lesions of temporal zone was added and then - "conduction aphasia" (inability to repeat words associated with preserved spontaneous speech) and "transcortical aphasia" (inability of spontaneous speech or understanding of speech associated with preserved repetition and recognition of words) were described. After 1870 the whole scheme of basic forms of aphasia was ready, each form having as a basic destruction of certain cortical "centre" of separate speech capacity of a disturbance of it's connection with other center. The remarkable fact is that these simple schemes, derived from concepts of immediate localization of complex functions in circumscribed cortical zones, have remained unchanged for a century and that neurologists still use these concepts although they long ago came into conflict with clinical reality and with basic ideas of contemporary science. There are two sources of this paradox. The first in the neglecting of negative information, the second - a certain carelesness of neurologists and psychiatrists to what concerns basic theoretical aspects of science.

The first can be described as follows: data which accordand with accepted notions remain in memory while contradictory facts are neglected. Only that can explain why neurologists continue to use the concept of "conduction aphasia" despite the fact that not one case has been carefully described when a patient who, whilst unable to repeat words yet retained spontaneous speech, or ~~the way it is maintained~~ The assumption that motor aphasia is always a result of the lesion of the Broca zone when in hundreds of cases the

lesion underlying motor aphasia left this zone undisturbed.

The second source of this paradox was the inertia of old concepts and a carelessness with regard to the theoretical aspect of science common to a majority part of neurologists and psychiatrists. Although the idea that psychological processes - including speech - are complicated self regulating systems is now widely accepted, old concepts of a strict localization of complex psychological functions in circumscribed areas of the cortex still persists, and attempts to find separate foci for verbal images or arithmetical operations, practical or gnostic schemes remains unchanged.

The two issues we mentioned explain the paradox of modern clinical thought, and the fact that even modern textbooks of neurology ~~which have defined psychological data~~ still use naive psychological concepts from the middle of the 19th century, remain one of the paradoxes of modern medical thought.

2. Neuropsychology

It was the goal of modern Neuropsychology to overcome this paradox and to make a farther step towards a scientifically based concept of Aphasia. That was made by a series of modern attempts to single out factors underlying speech disorders and to describe forms of aphasia resulting from disturbances of these factors (cf. Luria, 1965, 1970).

The first task of Neuropsychology was to describe carefully speech disorders associated with local brain lesions, to provide a qualification of symptoms and to single out factors underlying disturbances of speech in these cases.

Such approach of modern Neuropsychology is closely connected with the older attempts of French neurologist to understand speech disorders as a derivation of gnostic defects in one group of cases and as a result of praxic defects in another group of cases.

Following this approach we tried in a series of publications to find partial factors associated with different zones of the cortex and to understand several forms of aphasia as a result of such special factors. This approach brought us to a description of certain basic forms of aphasia. Classical sensory aphasia was described as a result of disintegration of phonematic hearing associated with lesions of the upper parts of the left temporal lobe or as a result of an instability of acoustic traces in cases of lesions of the middle parts of the left temporal lobe. Motor aphasia, as it was shown, could derive from a breakdown of afferent kinesthetic schemes associated with lesions of the lower parts of the left retrocentral cortex (afferent or kinesthetic motor aphasia) or from a breakdown of serial integration of kinetic melodies associated with lesions of the lower parts of the left premotor zone (efferent or kinetic motor aphasia). Semantic aphasia as it was seen, derived from breakdown of the simultaneous (quasi-spatial) syntheses associated with lesions of the tertiary temporo-parieto-occipital areas, and Dynamic aphasia was supposed to be a result of breakdown of speech programs associated with lesions of the lower parts of the left frontal cortex.

This neuropsychological approach to Aphasia we described in a series of publications (cf. A.R.Luria,1947, 1962, 1964, 1965, 1966, 1970) resulted in certain important

modifications in basic approaches to the problem of Aphasia. Instead of a phenomenological description of symptoms, we could now single out some basic units of speech which became disturbed in different forms of aphasia. We began to deal with defects of "phonemes" or "articulemas", "sequantial" or "spatial" schemes, and "programs" underlying speech processes. From a phenomehological description of complex syndromes we moved to an analysis of factors underlying these speech disorders. By an analysis of factors - we opened the way towards a better understanding of the role played by certain cortical zones in the organization of speech processes.

Needless to say such a Neuropsychological approach was an important step towards a scientific analysis of Aphasia and towards a breakdown of the naive ideas of an immediate localization of complex speech processes in circumscribed cortical areas which - as we have already said - remained unchanged for a whole century.

Neuropsychological analysis of Aphasia and its syndromes can be evaluated as one of the most important event in modern Neurology and we can hardly imagine a modern clinical approach to Aphasia which would neglect this decisive branch of science.

Neurophysiology was an important start to the modern scientific approach to Aphasia; but by no means is it the last step in the development of this chapter of neurological science.

3. Neurodynamic mechanisms

The analysis of basic factors underlying different forms of speech disorders was a significant step in the scientific development of aphasia study. But it was only the beginning of a new line of investigations which consisted in an analysis of neurodynamic mechanisms of these disorders. This step was not yet made, and we can hardly mention more than half a dozen publications of works done in this field,

Experiments of I.P.Pavlov showed clearly that a pathological cortex can change its normal states and can shift to one of the so called inhibitory phases typical for a deranged nervous apparatus. Whereas the normal cortex obeys the "rule of force" when strong or important stimuli evoke strong reactions and week or unimportant stimuli evoke week reactions, - during inhibitory phases these relation change. Strong or important stimuli begin to evoke reactions of the same strength as the week or unimportant ones (phase of equalization) or even week stimuli or their traces begin to evoke even stronger reactions than the strong or important ones. The same can be seen in focal brain lesions associated with widely observed changes in neurodynamics. It is obvious that these basic changes of neurodynamic rules in a pathological change of the basic behavioral processes.

As a result of such changes no selective organization of the mental processes is now possible; every stimulus begins to evoke a whole complex of reactions, and week and unimportant associations are evoked with the same probability as strong or important ones.

It is easy to see that such a change inevitably results in a most serious derangement in language processes, and if the focal lession is situated in the speech zones of the cortex, - marked changes of selectively organized speech activities take place.

It is well known that speech is a highly selective, multidimentional matrix. Every word evokes a complex of connections, partly phonetical ("mouse" - "house", "cat" - "hat"), partly-morphological ("black-bird" - "blue-tit"), partly-semantical ("mouse" - "cat" - "maze"; "dog" - "house" - "animal" etc.). In normal behavior the choice between all these connections and finding of the word needed does not ~~give~~ evoke any difficulty: the strongest (dominant) traces are found very easily, and all extra-connections are easily supressed. In pathological states of the cortex this process is by no means an easy one. All connections of this multi-dimensial matrix are evoked with equal probability and the choice of a proper connection becomes very difficult, sometimes even impossible.

That is why patients with pathological states of the speech areas of the cortex begin to experience difficulties in word finding, and a series of extra-words (or paraphasias) begin to appear, sometimes even blocking the necessary word. In these cases one can observe that the patient, trying to find the word "preacher" - can replace it by "feature" (phonetic similarity), by "teacher" (both phonetic and semantic similarity), by the words "school", "pupil", "blackboard" or "pencil" (matrix of contextual associations) etc.

Such loss of the selectivity of mental processes due to a pathological state of the cortex and a breakdown of the "rule of force" in the deranged cortical areas can be supposed as an important mechanism underlying speech disorders associated with local lesions of the speech areas.

Another important neurodyramic change often observed in a deranged cortex is loss of normal plasticity of the nervous processes and the appearance of a pathological inertness.

In normal cortex a fantastic plasticity of the nervous processes can be seen. The "dynamic mosaics" of excitations change with an enormous quickness, and a shift from one matrix of excitation to another one takes place without any difficulty. In pathological states of the cortex this plasticity becomes deeply disturbed. Traces of every excitation remain for an abnormally long period, and plastic changes of excitatory matrixes become impossible. The phenomenon we mentioned is well known as a pathological inertness or perseveration, and in different lesions of the brain it can receive new shapes from simple iteration in cases of lesions of the subcortical ganglia to inertness of complicated stereotypes in cases of lesions of higher cortical zones.

Pathological perseveration of speech structures is well known to every student in aphasia, and examples of perseveration of phonetic items of complex morphological structures or of whole linguistic sets are well described.

We could observe this phenomenon practically in every form of aphasia, but it is especially clear in lesions of the anterior parts of the speech areas. Sometimes - as is

in the case of ~~with~~ so called "transcortical motor aphasia", pathological inertia of nervous processes can become a mechanism underlying the defects of spontaneous speech; in these cases a patient who easily repeated the sentence "the girl was drinking milk" becomes unable to repeat the second phrase "the boy is riding a horse" and gives a perseverative (or contaminative) phrase - "the boy is ... drinking milk" or "the girl ... is riding a horse". In milder cases the same happens if the patient is asked to retrieve the first sentences after he has repeated the second. It can be supposed that such a pathological inertia of nervous processes can result in most severe troubles of spontaneous speech.

We gave only two examples of the role played by the deranged neurodynamics in speech pathology associated with local lesions of the brain.

It is very probable that intensive studies of the pathological changes in neurodynamic processes which are an inevitable result of local brain lesions will open new vistas in our studies of aphasia.

Summary

Three stages of the study of aphasia were mentioned. The first classical one - was a description of Basic forms of speech disorders associated with local lesions of the left hemisphere and an attempt to find a strict localization of complex speech processes in circumscribed cortical areas.

The second was a neuropsychological attempt to find factors which are primarily disturbed after local brain lesions and to understand the mechanism of the Basic forms of aphasia by the analysis of primary symptoms and these basic factors.

The third is an attempt to analyse neurodynamic changes associated with local lesions of the brain and to understand the basic symptoms of aphasia as a result of these neurodynamic changes.

These three steps of the study of aphasia are of Basic importance for the topic of the paper - "Aphasia reconsidered".

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