

APHASIA RECONSIDERED

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

1. The classical approach

The Basic concepts of aphasia P.Broca formulated in the sixties and C.Wernicke in the Seventieth of 19th Century have long ago become classical. They are well known, and we can dwell on their principles only shortly.

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 center 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 circumscribed parts of the "speech areas" of the left hemisphere can be "centers" 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 focus of aphasia - each having as a base lesion of certain circumscribed cortical areas - were described. As a result-amnesic aphasia which resulted from

lesions of temporo parietal 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) was described. During the 80^{ies} of the 19 century the whole scheme of basic forms of aphasia was ready, each of them having as a basis a destruction of certain cortical "center" of separate speech capacity or a disturbance of its connection with other center. The remarkable fact was that these simple schemes derived from concepts of immediate localization of complex functions in circumscribed cortical zones remained unchanged during a century and that up to now neurologists use these concepts although they long ago came in conflict with clinical reality and with basic ideas of contemporary science. Two issues are the cause of this paradox. The first is the neglecting of negative information, the second - a certain carelessness of neurologists and psychiatrists to what concerns basic theoretical aspects of science.

The first issue can be described as follows: data which are in accordance with presumptions accepted remain in memory while contradicting facts are neglected. Only that rule can explain why up to now neurologists use the concept of "conduction aphasia" in spite of the fact that not one case was carefully described when a patient was unable to repeat words but preserved spontaneous speech, or the assumption that motor aphasia is always a result of the lesion of the

Broca zone although in hundreds of cases the lesion in motor aphasia left this zone undisturbed.

The second issue of this paradox was the inertia of old concepts and a carelessness to the theoretical aspect of science common for a major part of neurologists and psychiatrists. Although the idea that psychological processes - including speech - are complicated self regulating 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 the modern clinical thought, and the fact that even modern textbooks of neurology which have been defined psychological data still use naive psychological concepts from the middle 19th century, remain as one of the paradoxes of the modern medical thought.

2. Neuropsychology

It was the goal of modern Neuropsychology to overcome this paradox and to make a further 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 way 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 cases 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 left temporal lobe or as a result of an unstability 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 retro-central 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 pre-motor 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 tortiary 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 steps. 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 begun to deal with defects of "phonemes" or "articulemas", "sequential" or "spatial" schemes, and "programs" underlying speech processes. From a phenomenological 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 that such 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 already told - remained unchanged during 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 of the modern scientific approach to Aphasia; but in 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 a 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 hardly can mention to more than half a dozen publications of researches made in this direction.

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 weak or the unimportant stimuli weak reactions, - at these inhibitory stages, these relations change. Strong or important stimuli begin to evoke reactions of the same strength as the weak or unimportant ones (phase of equalization) or even weak 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 result in a pathological change of the basic behavioral processes.

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

It is easy to see that such a change inevitable results in a most serious derangement in language processes, and if the focal lesion 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, multi-dimensional matrix. Every word evokes a complex connections, partly phonetical ("mouse" - "house", "cat" - "hat"), partly-morphological ("black-bird" - "white-had"), partly-semantic ("mouse" - "cat" - "maze"); "dog" - "house" - animal" etc.) In normal behavior the choice between all these connections and finding of the word needed does not involve any difficulty: the strongest (dominant) traces are found very easily, and all extraconnections are easily suppressed. In pathological states of the cortex this process is by no means an easy one. All connections of this multi-dimensional 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 feel 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 "teaches" (both phonetic and semantic similarity), by the words "school", "pupil", "blackboard" or "pencil" (matrix of situative 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 lesion of the speech areas.

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

In normal cortex a phantastic 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 sub-cortical 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 it is in the cases of so called "transcortical motor aphasia" pathological inertia of nervous processes can become a mechanism underlying the troubles 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 patient is asked to retrieve the first sentence after he 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 mechanisms 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 of the topic of the paper - "Aphasia reconsidered".

References

- A.R.Luria. Factors and forms of Aphasia. "Disorders of Language". CIBA Foundation Symposium. London, 1969.
- A.R.Luria. Higher cortical functions in Man. N.Y. Basic Books. 1966.
- A.R.Luria. Traumat. Aphasia. The Hague Mouton & Cic., 1970.