PERGAMON PRESS

Journal ..... Neuropsychologia .....

INSTRUCTIONS FOR AUTHORS ON CORRECTION AND RETURN OF PROOFS AND ORDERING REPRINTS

Author ..... Luria et al ..... Ms. No. 156

Title ..... Towards a Neurodynamic Analysis .....

PROOFS

1. Check and correct the marked set of proofs and answer any questions on either the marked set or the manuscript. If at all possible, please avoid making any alterations apart from correcting printer's errors. **Additional alterations will inevitably introduce delay.** The second set of proofs is provided for your retention and it is suggested that the corrections be copied on to this set before returning the marked set. This may be of considerable help in answering queries which might be raised on receipt of your corrected proofs here.

2. Please check the engraver's proofs of your illustrations which accompany your page proofs. Some of your diagrams may have been redrawn in our Drawing Office; these have been checked, but should you find any errors you should indicate the corrections required on the engraver's proof and make a note at the side of the figure in the page proofs. Please confine your corrections to errors in redrawing. If alterations to the figure as originally submitted are absolutely essential a charge may be made for the correction and for making a new printing block.

3. Please return your marked set of proofs, engraver's pulls (and the manuscript if so requested on the front page of the manuscript), as quickly as possible **(by airmail if overseas)** to the address given on the gummed label which is enclosed.

PROOF CORRECTION SYMBOLS

Margin	*Text*	*Meaning*	*Margin*	*Text*	*Meaning*	*Margin*	*Text*	*Meaning*
⌀	/	Take out	*italic*	—	Change to italics	#	⋏	Insert space
⌀	⌇	Delete and close up	*rom.*	○ encircle	Change to roman	*tr.*	⌇	Transpose
⌢	⌣	Close up				*n.p.*	[before first word	Begin new paragraph
stet	...	Leave as printed	*w.f.*	○ encircle	Replace by letter of correct fount	*run on*	⊃ between paras.	No new paragraph
caps	≡	Change to capital letters	9	○ encircle	Invert type	(/)	⋏⋏	Insert parenthesis
s.c.	=	Change to small capitals				[/]	⋏⋏	Insert brackets
l.c.	○ encircle	Change to lower case	X	○ encircle	Damaged character, replace	/-/	⋏	Insert hyphen
bold	ww	Change to bold type						

PLEASE READ THESE NOTES BEFORE COMPLETING THE ORDER FOR REPRINTS

1. This form is sent in duplicate. Please complete and return the top copy with your proofs and pass the duplicate to your purchasing office if an official purchase order is required. The duplicate must be returned with your official order.
2. Unless instructions to the contrary have been received only the first-named author will receive proofs and this order form. **All orders for reprints of your article must be included on this form.**
3. 50 free reprints of the paper are supplied. Enter only **additional** orders overleaf.
4. Reprints are normally supplied with standard covers having a window through which the title, author(s) and other bibliographic details appear. Please indicate clearly if reprints are required without covers or with special covers.
5. Indicate here if an official purchase order is to be raised and if possible give the order number to be quoted on invoices.

Official Purchase Order to Follow/Attached ☐ **Purchase Order Number if known**.....

Proofs & MS to

A.R.Luria, ~~E.N.Sokolov, M.Klimkowski~~

Department of Neuropsychology
Moscow University,
Moscow, USSR

12pt Roman Caps

TOWARDS A NEURODYNAMIC ANALYSIS OF MEMORY DISTURBANCES WITH LESIONS OF THE LEFT TEMPORAL LOBE

A.R. LURIA, E.N. SOKOLOV and M. KLIMKOWSKI

Department of Neuropsychology, Moscow University, Moscow, USSR

(Received 25 June 1966)

MS & Author Proofs to A.R. Luria at the address given here

Recto running headline "TOWARDS ..... DISTURBANCES"

21 folios MS

4 line blocks to follow

22 folios inclusive

Moscow
1966

Abstract

In a Neuropsychological analysis of lesions of the left temporo-parietal region of the brain we tried to describe disturbances in the retention and reproduction of acoustico-verbal traces.

These disturbances were modality-specific: no such disturbances were observed in lesions with different localization. The main symptom observed was an inability to reproduce a series of phonemes or words presented orally.

This symptom could not be explained as a simple weakening of acoustico-verbal traces; it could be explained as a result of pathological changes in the neurodynamic processes.

Two kinds of neurodynamic factors underlying the disturbance of retention and reproduction of traces were described.

The first factor is a pathological increase of external (pro- and retroactive) inhibition; in these cases summation of inhibitory influences results in a blocking of the connections between elements, and the regular serial reproduction of traces becomes impossible.

The second factor is that of "equalization of excitation": the dynamic range of the excitation becomes restricted, and a selective reproduction of dominant traces is no more possible; the "paraphasic" reproduction of equivalent traces is typical of this kind of disturbance.

A neurodynamic analysis of memory disturbances in cases of local brain lesions is an important step in the further development of Neuropsychology from a descriptive to an explanatory science.

TOWARDS SOME NEURODYNAMIC MECHANISMS OF MEMORY DISTURBANCES

A.R.Luria, E.N.Sokolov, M.Klimkovski

Received 25 6 66

The problem of memory has become, during the last decades, one of the most active fields of research. Biologists from all over the world have turned their interests towards the analysis of the basic mechanisms of retention and reproduction of memory traces. The growing interest in the biological bases of memory became even more active after new discoveries of the molecular and biochemical mechanisms of memory (Hyden, 1964) [1] and the sophisticated investigations of the post-tetanic potentiation and inactivating of synapses with their disuse (Eccles, 1962) [2]. In several works a tendency can be seen toward an identification of memory with the establishing and reproduction of conditioned reflexes. Such an approach hardly can be granted. Even I.P.Pavlov (1947) [3] mentioned the importance of distinguishing the external rules of conditioning and the inner mechanisms of retention of traces in the nervous system. So, the facts of external inhibition of the conditioned reflexes and that of a "spontaneous" recovery of the reflex after its actual inhibition show that inhibition is not at all identical with a disappearence of the trace in the nervous system. In his "Wednesdays" Pavlov [3] often mentioned that children's impressions never disappear totally, citing examples when early childhood memories returned after trauma. The same facts were indicated by W.Penfield [4] in his experiments with electrical stimulation of the cortex during neurosurgical operations.

That is why a detailed study of the mechanisms neurodynamic bases of memory with a distinct delimination of the mechanisms

of the fixation of traces in the nervous system and that of their reproduction, is of first importance. Well-known data on the retro- and pro-active inhibition of memory traces as well of the "reminiscence" (increased reproduction of memory traces after a certain pause) can be used as examples of such a neurodynamic approach.

We have every ground to believe that basic neurodynamic mechanisms of memory can become more clearly seen in pathological states of the cortex, and that neuropsychological analysis of memory disturbances in cases of the local brain lesions can offer invaluable opportunities for their study.

It is well-known that in one group of lesions ~~(with~~ lesions of the higher brain stem and reticular formation~~)~~ marked general disturbances of memory can be observed; they are modality-unspecific, and give us an opportunity to describe a whole series of deficits, from slight disfunctions to a total loss of encoding and decoding of memory traces, typical of Korsakoff's syndrome. In a second group of lesions (circumscribed cortical lesions) a different picture of memory disturbances can be observed. Here we can observe forms of limited disturbances of memory which can be highly modality-specific, and the whole history of neuropsychological analysis of agnosias and apraxias show very distinctive pictures of such deficiencies.

The most important point, which was mentioned very rarely, is that even in lesions of the same (or nearly the same) parts of the brain the neurodynamic mechanisms of disturbances of the memory traces can be highly different. The reason for this fact is hardly known. One can suppose that different kinds of

pathological agents can result in different physiological states of the neurons, and different kinds of changes of the neurodynamic processes can be seen.

We shall try to show this in an analysis of different forms of disturbances of the acoustic verbal traces in two cases of lesions of the left temporo-parietal zone.

In both cases injuries were limited by the left temporal (and temporo-parietal) part of the brain. In both cases disturbances of acoustic traces of verbal stimuli could be observed although visual and kinesthetic traces were preserved. But in both cases the pathological process resulted in quite different types of neurodynamic disturbances. In the first case pathological increase in retroactive inhibition, which resulted in a temporary arrest of the traces for reproduction, could be seen; in the second case the pathological process evoked a kind of "equalization" of the excitation process which resulted in a different syndrome of memory disturbances. We shall try to make a comparative analysis of both forms of memory disturbances.

Two case histories (A)

Two patients with injuries of the left temporal lobe were the subjects of our observations. Both were a long time in the wards of the Burdenko Neurosurgical Institute in Moscow.

Both patients had the syndrome of acoustico-mnestic aphasia [5] (A.R.Luria, 1947, 1966); in both cases we had good opportunities to study disturbances of memory.

Both patients underwent rehabilitative training, and the observations we made were of a long lasting type.

1. Patient B, 35 (case history 37637), an officer, had a car accident with an injury of the left temporal lobe. ~~During~~ For 15 minutes he was unconscious; for a short time slight paresis of the right hand was observed, then the weakness of the hand increased and he lost ~~his~~ consciousness for a second time. In the next period hemiparesis of the right hand, and aphasic disorders of speech were observed. A pneumogram showed that ~~Pneumogram~~ - the ventricles were displaced towards the right side. During the subsequent period partial restoration of the ~~right~~ movements of the right hand; the patient recovered understanding of speech, he was able to repeat words but could not actively find necessary words and remained practically speechless. A slight central paresis of the right facial nerve, of the right hand and a very slight paresis of the right leg remained. Symptoms of right-sided exaggeration of reflexes were seen as well as a disturbance of ~~epicrisis~~ epicritic sensibility and astereognosis on the right side. There was pathological hyperreflexy of the right side. ~~indicatio - indicatione~~. The visual field remained normal. ~~EEG x x data~~ EEG - delta waves of high amplitude were demonstrated in the left temporal lobe; these symptoms increased after functional tests and were partially observed in the left temporal occipital parts as well.

Neuropsychological analysis showed some slight symptoms of oral apraxia; spatial orientation remained normal, no defects of visual gnosis were observed. He could repeat rhythmical structures acoustically presented as well as separated words. He showed marked disturbances in repetition of series of words (this symptom will be specially analysed) and in naming

objects; his expressive speech was severely disturbed because of failing to find the appropriate words. Reading was possible; though difficulties were seen in the writing of complicated words and phrases: the patient was unable to find necessary groups of sounds (letters) or words in a phrase.

All this shows that the injury was located primarily in the left temporal and temporo-parietal part of the brain.

2. Patient K., 21 (case history 37690), worker. In March 1963 he received a blow in the left side of the head. A fracture of the cranium in the left temporo-parietal part was found with fragments of the bone imbedded in the brain tissue. He lost consciousness, was operated upon and skull fragments were removed. Complications (ventriculitis) followed the operation and a severe aphasia was observed with a paresis of the right hand and the right facial nerve. After six months additional skull fragments and a part of the prolaps were removed. By December 1963 the patient was much better. Right sided paresis (primary in the right hand), right-sided hyperreflexy and aphasia were noticeable.

He entered the Neuro Surgical Institute in march 1964 with severe troubles of speech. He still had paresis of the right hand, right sided defects of sensibility and right sided pathological reflexes; EEG showed slight general changes of the electrical activity with a marked focus of pathological activity in the left temporal lobe.

Neurosurgical analysis demonstrated neither defects in visual or spatial orientation; nor symptoms of postural or spatial apraxia of the left hand. He understood speech but showed marked symptoms of a loss of understanding of separate words. The patient could

repeat separate phonemes and words without any difficulty; but the repetition of series of words was highly disturbed. He could name separate objects but had troubles in naming series of objects. In both processes paraphasias were observed. He had slight trouble in reading words aloud; writing was severely disturbed; and the patient failed in finding proper letters and their sequences.

We had here a case of a complicated fracture of the left temporal part of the skull with an injury to the left temporal and sensory-motor parts of the brain as a result of an inflammatory process. An acoustico-mnestic aphasia with symptoms of afferent motor aphasia was seen.

A Methods of investigation

A series of special methods of neurodynamic analysis of fixation and reproduction of verbal traces was used.

1. A series of different stimuli (acoustic and non-acoustic; verbal and non-verbal) was presented and the patients had to reproduce them.

Acoustic stimuli used were: tones - phonemes - words - series of words, all presented orally.

Visual - kinesthetic stimuli were evoked by positions of the hand, presented visually or kinesthetically, which the patient had to reproduce.

The analysis of the reproduction of the information given made it possible to observe whether the patients had any troubles in retention and reproduction of traces, and whether there were defects in the detection of the quantity or the sequence of the signals.

A comparative analysis could show whether the defect was general or sensory specific.

2. The number of signals presented gradually increased, and that made it possible to observe changes in retention and reproduction as a result of the scope of the information given.

a result of the scope of the information given.

3. The conditions of the reproduction of the stimuli varied: patients had to repeat the information given (a) immediately, (b) after a pause of 10-20 seconds and (c) after a pause with some distracting activities (conversation).

The change of conditions of the reproduction enabled us to observe some changes of the traces, their stability, their temporary inhibition, their recovery after a pause, etc.

4. Retention of the given information during several repetitions was studied. That made it possible to study the gradual changes in fixation or inhibition of traces as a function of repetition.

These series were repeated many times, and it was the large number of experiments and the variation of conditions made the information obtained reliable.

Results

The data obtained in our observations provided us with significant information on the neurodynamics of memory disturbances in our cases.

(a) Modality-specific disturbances in the reproduction of traces: quantitative data

The data obtained showed that disturbances of reproduction of the traces was limited to the acoustic sphere and was specific to acoustico-verbal stimuli: defects observed were very severe in the reproduction of series of phonemes and words (presented orally); they were much less pronounced in the reproduction of series of tones; and no disturbances in the reproduction of visually presented figures and in the recognition of drawings was observed. Both patients could repeat separate sounds, phonemes and words, but they failed in the reproduction of series of 2-3-5 phonemes and

words. They had slight troubles in the repetion of a series of 3 - 5 musical tones, of a sequence of 3 - 4 movements; they had no difficulties in reproduction of a series of 3 - 4 visually presented figures.

Table 1 shows some data obtained in a series of 10 applications of the tests. The difference in the scope of information retained and reproduced in different tests is clearly seen.

Table 1 here

The same data were obtained in the analysis of learning curves of both patients. The patients retained the visual or kinestatic series of 3 - 4 elements after 2 - 3 repetion, each repetition brining a certain facilitation to the links already retained. That was not the case in the learning of acoustically presented elements (phonemes or words): it can be easily seen that both patients were unable to retain a series of 3-5 acoustico-verbal elements even after 10 repetitions; sometimes no gain in subsequent repetitions was seen, and sometimes further repetitions of the series even resulted in a decrease of the number of reproduced elements.

Table 2 shows the results in the forme of a cumulative curve of retention.

Table 2 here

It is clear that in increasing the number of elements the patient had to retain we observe a dynamic blocking of the reproduction in the verbal-acoustic sphere, whereas no such blocking in the visual or kinesthetic sphere is seen.

(B)2

(b) Sequential disturbances: retro- and pro-active inhibition

The limitation of the scope of acoustico-verbal information

retained and reproduced by our patients was the first, but not the most important, of our findings. Much more important were some data on sequential characteristics of the traces which both patients could retain and reproduce.

Here features of similarity and of marked differences between both patients could be seen.

Both patients were able - without any difficulty - to retain and reproduce a series of visually presented stimuli, and the failures in reproduction of elements observed in these cases followed the rule well formulated in normal psychology: they tried to reproduce the series presented in the sequence of the elements given, and they reproduced, as a rule, the first and the last elements, sometimes failing in the reproduction of the middle elements of the series. This means that a normal kind of pro- and retroactive inhibition of the traces could be observed.

In reproduction of verbal ~~information~~ series acoustically presented another picture was seen, and both patients differed markedly As a rule - patient B. - never retained the sequence of elements ~~That could be seen in cases when he had~~ in series given; he started to reproduce the last element of the series and failed in reproduction of the first elements. ~~That could be seen~~ in cases when he had to retain and reproduce a series of 3 or 5 phonemes or words, he reproduced the most recent elements and was unable to reproduce the former.

Here are some examples of this data.

house-night	table-clock	leg-window
night.. Oh, no..	clock... no	window... and leg

above - the series given; below - the series reproduced.

table -knife-eye	glass-hand-spoon	eye-key-step
eye...leg...no...	spoon...hand...	step...oh, no

bird-apple-knife-fork-light	smoke-ball-shade-goose-brides
light..fork...	bridge..goose...oh, no....

This tendency remained stable and did not change even when the patient was instructed to repeat the series in the ~~pro~~ proposed order. He complained that it is only the last element he can retain, and that the former elements disappeared from his memory. ~~(see tabl. 3)~~

Figs 1, 2

We can conclude that pathological increase of retro-active inhibition was observed in this case.

It has to be noted that such a pathological increase of retro-active inhibition was seen only if the patient had to retain acoustically presented elements, and to reproduce them orally. If the series of elements was presented visually, the situation changed, and no predominance of the reproduction of the last (most recent) elements was seen. The same happened even if a series of phonemes or words was presented acoustically, but the patient was asked to reproduce them by writing letters. A change of the modality of the decoding or encoding of traces resulted in a change in the neuro-dynamics of the retention and reproduction of the traces, and the predominance of the pathological retro-active inhibition of traces was observed only when the whole process of decoding and encoding remained in the verbo-acoustic sphere.

Fig 3 here

~~Table~~ Figure 3 gives some graphic summaries of the data.

Different data were obtained in our experiments with the Patient K.

No pathological increase in retro-active inhibition of acoustico-verbal traces was seen. The patient tried to reproduce the series of phonemes or words ~~given accordingly~~ the given order. He reproduced as a rule the first elements of the series but failed to reproduce the last ones. In that case we could see the predominance of the primary factor and the inability to retain the whole scope of the elements presented. The data in Table 1 can be used to illustrate this point.

It can be supposed that in this case other physiological factors were underlying the memory disturbances, and that the whole phenomenon observed is due either to the increased proactive inhibition of acoustico-verbal traces, or to some different factors, which up to now remained unknown.

(c) Qualitative disturbances: loss of selectivity

The qualitative analysis of the kind of word-series reproduction deficit in both patients can possibly throw new light on some of the mechanisms underlying these disturbances.

The first of our patients did not make mistakes in reproducing the words of a given series. As a rule, he reproduced the last words or sometimes last two words of the series, and said be had forgotten other words and was unable to reproduce them.

The second patient's behavior was different. Reproducing only a limited part of the series, he gave, as a rule, a large quantity of mistakes, reproducing words close to the given ones, but failing to give correct and selective reproduction only of words included in the series presented. Trying to find the word "cart" given in the series presented, he would say "carriage"; trying to reproduce the word "hair" he would say "coiffure", trying to find "window"

he would say "glass" etc. A given phrase "On the ~~board~~ edge of a forest the hunter killed a wolf" was reproduced as "On the ~~board~~ edge of a forest.. the wolves... were fighting.." or "On the ~~board~~ edge of a forest the hunter killed a bear... no that is not right..", etc.

In many cases the patient's selective reproduction of words was disturbed by perseverations and alliterations

It has to be mentioned that the number of such paraphasic responses increased markedly in experiments with reproduction of long (5-word) series. (~~Table~~ Fig. 4).

We had an impression that such a loss of selectivity in reproduction of given words is typical for the second patient, and that we had to suppose here some pathological state of the cortex, which resulted not only in a restriction of the reproductive capacity, but in a loss of selectivity of traces as well.

(d) Disturbances in reminiscence

We have to mention the last distinction observed in our two patients: it deals with quite different kinds of the phenomenon of so-called reminiscence.

This phenomenon is well known in normal psychology. It is known that a subject who can give only a limited reproduction of a series of elements presented, can increase the number of elements reproduced after a pause of 0.5 - 1 min~~utes~~, and sometimes even after a longer period. This phenomenon, known as "reminiscence" was supposed to show that memory traces, blocked by a series of presented impressions, could be revived after a certain ~~rest~~ period of rest of the nervous elements. In both of our patients we could see marked differences in that phenomenon.

In the first patient, with a pathologically increased retro-

active inhibition, a good reminiscence of the blocked traces after a short period of rest could be observed. There were cases when this patient, who was unable to reproduce more then one element from the given series, could after a pause of 1-2 minutes reproduce two elements. We had the impression that memory traces were blocked, and that they revived after a period of rest; in other words, that not a weakness of traces, but a temporary inhibition of these traces took place.

In the second patient, with no reminiscense of traces, the temporary inhibited was seen, and after a pause of 1-2 minutes he could reproduce even less elements than before.

~~Some fragments of the protocols can be used as illustrate of the both types of disturbances.~~

Discussion

Data we observed in our experiments with two patients who showed marked symptoms of "acoustico-mnestic aphasia" after injury to the temporo-parietal region of the left hemisphere proved the important role of neurodynamic mechanisms in disturbances of memory processes.

Not only modality-specific disturbances of acoustic (or more precise, of auditory-verbal) memory traces were observed. Important neurodynamic factors of memory disfunctions could be observed as well.

Two basic nervous mechanisms could be supposed as taking part in the genesis of both syndromes: the effect of pathological accumulation of inhibitory influences which blocked the traces and which masked traces already fixed, - and the effect of pathological "equalization of excitations", which made dominant and subdominant,

present and former traces equal in strength.

Although both factors were limited in our patients to the auditory-verbal sphere, they were not equally present in both cases.

The first of these factors - that of the pathologically increased external inhibition of traces - could be seen in two forms: the effects of proactive and of retroactive inhibition. Both effects resulted in a blocking of the connection between elements and a temporary blocking of the memory traces. It is only natural that the most recent traces remained more powerful, and that the effect of retroactive inhibition could predominate. That is why in the first patient there was a clear tendency to memorize only the last elements of the series, the former elements being blocked.

We can see that in such cases it is not the weakness of memory traces, but the increase of their retroactive inhibition which can be supposed to be the physiological mechanism in these disturbances.

It is well known that in normal subjects the connection between separate elements are the leading mechanism of the reproduction of the whole series; in pathological cases these connections can become inhibited, and the serial ~~representation~~ retention of the given information becomes impossible.

In pathological states of the cortex and in cases of local brain injuries, the effect of an increased accumulation of inhibitory influences is seen only in modality-specific zones. The role of this dynamic factors in the genesis of the symptoms of "acoustico-mnestic apasia" seems to be of principal importance and should be especially studied.

A very different neurodynamic mechanism of memory disturbances

can be seen in our second patient. A local injury to the left temporo parietal lobe complicated by a severe inflammatory process, resulted here in a permanent pathological state of the cortex. ~~We have all grounds to suppose:~~ A restriction of the dynamic range of excitation can be supposed, and it can be thought that the level of excitation when new connections are established rapidly approaches a threshold, equal ~~with~~ to the thresholds of different, previously established connections. This "equalisation of excitation" is supposed to be the source of the phenomenon described, where the reproduction of traces loses its selectivity and where interconnected traces can easily be reproduced.

The hypothesis mentioned can explain why no selective reproduction of the last elements of the series is seen in the second patient, why a series of paraphasic reproductions of some close associations are so often observed, and why no clear phenomenon of reminiscence after a pause takes place.

The analysis of neurodynamic mechanisms of the memory disturbances in the cases of focal brain injuries was up to now a neglected field of neuropsychology ~~it~~ which remained a descriptive rather then an explanatory science.

That is why we have ~~all~~ every grounds to suppose that further investigations in the neurodynamic analysis of disturbances of higher cortical functions in local brain lesions, and the descriptions of different types of neurodynamic disfunctions will open new vistas in this important field of our science.

Legenda

Fig.1. Reproduction of a series of traces of different modalities (acoustico-verbal and visual).

Abscisse - sequential number of elements included in the series

Ordinate - number of elements, reproduced in 10 separate experiments.

It is easily seen that retention of visual information (recognition of drawings and their selection from a group of 10-12 drawings) remains normal. Retention and reproduction of orally presented phonemes or words is disturbed. It is clear that deficits of retention and reproduction are increased with the length of the series presented.

It is seen that in patient B, there is a clear tendency to repeat the last elements of the series; no such tendency is seen in patient K.

Fig.2. Efficiency of learning of series of acoustically presented verbal elements (phonemes and words) during 10 presentations of the same series.

Abscisse - presentations of the same series.

Ordinate - Cumulative number of reproduced elements (a result of summation of elements subsequently reproduced in all series).

The ideal curve of accumulation has an angle of 45°. The weaker the process of accumulation, the lower is the curve.

When a series of two elements is given - the patients become very soon able to reproduce the total series.

When the series is longer - no effective learning is possible.

Fig.3. Reproduction of series of acoustically presented verbal elements by patient B. in different conditions:

(A) Oral presentation – oral reproduction

(B) Oral presentation – visual reproduction (by writing)

(C) Visual presentation – visual reproduction (by writing)

It can be seen that the effect of retro-active inhibition is typical only few series (A), but not of series (B) and (C).

Fig.4. Number of "paraphasic" reproductions of acoustical presentation of words, and its dependence ~~from~~ on the length of the series presented.

Abscisse – number of the words in the series presented

Ordinate – mean of paraphasic reproduction, relative to the total number of reproductions. (coefficient of paraphasic reproductions).

It is seen that patient B. doesn't give any paraphasic reproductions; in patient K. The ~~coefficient~~ coefficient of paraphasic reproductions increases with the length of the series.

LITERATURE

A REFERENCES

1 HYDEN, H. (1965) - Activation fo the Nuclear RNA of Neurons and glia in learning, in J.P.P. Kimble (ed.) - The Anatomy of Memory (Edited by) Science and Behaviour books, Palo Alto, 1965.

2 ECCLES, J.C. (1961) - The effects of use and disuse of synaptic function. in : J.K. Delafresnaye (ed.) . Brain mechanisms and Learning. Oxford, p. 335-352, 1961.

3 PAVLOV, I.P. (1947) - Complete writings, vol. 3, Moscow-Leningrad. Acad. Sci. USSR Publishing House, p. 351, 1947.

4 PENFIELD, W. and RASMUSSEN, Th. (1952). - The cerebral cortex in Man. N.Y. Mac Millan, p. 248, 1952.

5 LURIA A.R. (1966) - Higher Cortical functions in Man. N.Y. Basic Books, New York, 1966.

Towards a neurodynamic analysis of Memory Disturbances with Lesions of the left temporal lobe

by A.R. Luria, E.N. Sokolov and M. Klinkowski

RÉSUMÉ

Dans une analyse neuropsychologique des lésions de la région temporo-pariétal gauche, nous avons tenté de décrire les troubles de la rétention et de la reconstruction des traces acoustico-verbales.

Ces troubles étaient spécifiques quant à la modalité et l'on observait pas de troubles de ce type dans les lésions de sièges différents.

Le principal symptôme observé consistait en une incapacité de reproduire une série de phonèmes ou de mots présentés oralement. Ce symptôme ne pouvait pas être expliqué par un simple affaiblissement des traces acoustico-verbales mais comme le résultat des modifications pathologiques dans les processus neuro-dynamiques.

On décrit deux types de facteurs neuro-dynamiques à la base de ces troubles de la rétention et de la reproduction des traces.

Le premier facteur est une augmentation pathologique de l'inhibition externe (pro et rétroactive) ; dans ce cas, la sommation des influences inhibitrices a pour résultat le blocage des connexions entre les éléments, ainsi la reproduction sérielle régulière des traces devient impossible.

Le second facteur est celui de "l'égalisation de l'excitation" : il y a restriction de l'étendue dynamique de l'excitation de telle sorte qu'une reproduction sélective des traces dominantes n'est plus possible : la reproduction paraphasique des traces équivalentes est typique de ces troubles.

22

2

Une analyse neuro-dynamique des troubles de la mémoire dans les cas de lésions cérébrales représente une étape importante du développement de la neuropsychologie qui doit devenir une science non seulement descriptive mais aussi explicative.

German Abstract
here
- copy to follow