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Memory Disturbances in Local Brain Lesions*

by

A.R. Luria (Moscow)

13 Frunze ul., Moscow G-19, USSR.

Received 16 June 1971

Memory disturbances associated with local brain lesions are often mentioned but seldom described carefully and never analysed in terms of the basic physiological mechanisms upon which they depend. Only during the last two decades have attempts been made to bring a more scientific approach to bear upon the problems of memory disorder (see e.g. [1,2]). We shall try in this paper to review some basic principles governing our clinical studies and to interpret the results where possible in terms of general psychological prinicples.

Factors in Memory Disorder

Two hypotheses have been proposed to explain defects of memory of the kind so common with local brain lesions. The first attributes forgetting to "trace decay" (see, e.g. [3]). Neurologists suppose that in pathological states of the brain the rate of this decay is increased, traces become unstable and their consolidation difficult. The second hypothesis attributes defect in memory not to impaired storage but to defective retrieval; it is supposed that memory difficulties are due to an increased blocking of traces by interfering impressions or actions [4,5]. This increased inhibition of traces by interference is regarded as the main cause of abnormal forgetting in pathological states [6,7].

We shall try to examine both hypotheses: let us start with two series of experiments. In the first we shall analyse how traces can be retrieved after an interval of 1-2 minutes free from interpolated impressions or actions ("free interval"). In the second, we shall repeat the experiment but with the interval filled by interpolated (i.e. "interfering") impressions or actions. These can be "heterogenous", as when the subject is asked to do mental arithmetic, or "homogenous", as when he is asked to remember a second series of words. If retrieval

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* This paper is based on work undertaken at the Bourdenko Institute of Neurosurgery, Moscow, with participation of L.T.Popova, M. Klimkovski, N. Kyascenko, N. Akbarova and Pham Ming Hac. A more detailed account will be given in our forthcoming book on The Neuropsychology of Memory.

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ABSTRACT

Neuropsychological study of memory disturbances associated with local brain lesions has shown that such disturbances seldom result from simple trace decay. In general, they are due to interference effects resulting from activity interpolated between presentation and retrieval. This is well shown by simple experiments in which retrieval of a short series of words, designs or actions is required with or without interpolated activity. Interference effects are found to be particularly marked if the interpolated material or activity closely resembles the original material or activity ("homogenous" interference). Memory disturbances of this kind, which may vary greatly in severity, are seen particularly in deep-seated lesions involving the medial portions of the hemispheres and the upper brain stem. They are quite general in nature and may or may not be associated with a confusional state, depending on whether or not the frontal lobes and upper brain stem are involved.

Partial (modality specific) disturbances of memory may be associated only with lesions of the convexity of the hemispheres (in particular the left) and are never associated with confusional states or confabulation.

In cases of lesions within the speech area, a specific type of verbal memory disorder (amnesic aphasia or misnaming) is not uncommonly observed. A neurodynamic explanation of this disorder is advanced.

In cases with massive frontal lobe involvement a defect of memory secondary to defect in programming actions and a pathological "inertia" of nervous processes (shown in difficulty in shifting attention and in perseveration) is described.

It is claimed that careful neuropsychological analysis of memory defects is essential if we are to understand the basic factors upon which they depend and the role of the brain in the memory process.

is found to be normal after the free period but is blocked by interfering stimuli, there are grounds for accepting the second hypothesis, viz. that pathological forgetting is due to the blocking of retrieval by interpolated activity.

Our results are clear: a large majority of patients with local brain lesions (tumours, haemorrhages, brain trauma) were found to be able to retain a short series of geometrical forms, pictures, movements, digits, words or sentences under the "free interval" condition but found marked difficulty in retrieving them under the "interference" condition. In such cases patients said that the traces of the earlier impressions "disappeared" and that they were unable to retrieve them, or they gave evidence of "contamination" of traces of the earlier series by those of the later series. These findings suggest that pathological states of the brain associated with focal lesions result in increased blocking of traces by activities interpolated between perception and recall and that the memory disturbances observed in these cases are due to an increased inhibition of traces by each interpolated activity.

Data have now been obtained from patients with deep tumours of the brain [8, 9, 10] with aneurysms of anterior communicating arthery [11], and with focal lesions of the convexity of the left hemisphere (9, 12) and also in patients with massive cerebral trauma [13]. Only lesions of the left temporal and temporo-parietal lobe has instability of immediate verbal-acoustic traces and defect in their consolidation been observed as well [9, 14]. We shall discuss these cases later.

The pathologically increased inhibition of traces by interference seems to be a basic factor underlying memory disturbances associated with all brain lesions. That is why we regard this mechanism as basic.

Memory disorders can be of a <u>general</u> (i.e. modality non-specific) type, as in the classical Korsakoff syndrome, or of a <u>partial</u> (modality-specific) type, e.g. acoustic, verbal, spatial. They can present at all levels of coding or can be limited to one level only, e.g. the lower (sensory) or the higher (intellectual). Furthermore, they can be present in patients without confusion or drowsiness, although their association with such states, or with pathological inertness of nervous activity, is not of course uncommon [15]. We shall see later how important these differences can be and consider their significance for the localization and physiological substrate of memory defect.

General Memory Disorder

General (non-specific) memory disorder can be observed in cases with deep lesions of the hemispheres involving the brain stem, thalamic nuclei and the pathways from the hypothalamus, mamillary bodies and hippocampus - the so-called Papez circuit. This kind of memory disorder is well described in classical studies as well as in recent papers by B. Milner and her collaborators [16]. The disturbances are observed equally in every modality and forgetting is brought about by the blocking influence of activities intervening between perception and retrieval and may be due to derangement of non-specific neurones of the hippocampus serving as comparators (Vinogradova, [17].

Although general amnesia is common with all deep brain lesions, the level of the disorder and its neurodynamic characteristics may vary with involvement of the different functional brain systems and with different aetiological factors, e.g. hypertension, toxic and deficiency states.

(a) Mild General Memory Disorder:

This is seen with mild cerebral dysfunction, e.g. pituitory tumours with involvement of the mesial parts of the hemispheres (Kyasconko, [10]. No disorder of higher cortical function or clouding of consciousness is present, and the patients have good awareness of the memory defect. No impairment of memory was in evidence under "free interval" testing, but marked disturbance was in evidence under "interference" conditions. This was on occasion shown even after 4 - 5 repetitions of the experiments.

The following is a typical observation:

A series of three words was given, and no difficulties of retrieval of this series were observed after a "free" pause of 2 minutes. When a second series of three words was given, immediately after the first, and the patient was asked to retrieve the latter, he was unable to do it; the same happened if he were then asked to retrieve the second series and his answers became confused.

	I. house-tree-cat	II. night-stove-cake	?/I	?/II
1.	+	+	no.. forgotten	I really cannot remember
	+	+		
2.	+	+	no.. cake?..	..no... I don't remember
3.	+	+	..cat..	.. tree...
			no..	cake
			I don't know	..no.. I can't

Here the blocking effect is very clearly shown. In such a patient, 100 percent correct recall is possible after "free intervals" but only 40 - 45 percent correct recall after intervals filled by "homogeneous" interfering material.

Similar data were obtained in experiments with retrieval of series of

are seen in this group of patients in experiments with retention of simple sensory traces (the Konorski-experiment), or sensori-motor traces (the Uznadze-set experiment) and that systems of well organized verbal structures (phrases) could be easily retrieved even after another phrase was presented as an interfering factor; the number of adequate retrievals of the first phrase remained at the level of 90 - 100 p.c.

(b) Severe General Memory Disorder: This is seen in cases with massive deep hemisphere tumours, especially those involving the medial surfaces, in deep midline tumours involving the thalamus, brainstem reticular formation and the limbic system, including the hippocampus. Cases reported by Milner et al. [16], Popova [8] and others fall into this group. A special sub-group comprises patients with anterior communicating artery aneurysms associated with haemorrhages or constriction of both anterior cerebral arteries (Luria et al, [11]; similar gross memory defect was also seen in cases of massive head injury with haemorrhages in the same general region of the brain (Akbarova, [13]).

These massive, non-specific derangements of memory were sometimes similar to the Korsakoff syndrome. They were often associated with drowsiness, confusion, pseudo-reminiscences and confabulation. In only a limited number of these patients was consciousness entirely clear. Further, the patients rarely complained about their memory defects and sometimes appeared unaware of them.

In such cases, traces of immediate impressions were very unstable and disappeared after every shift of attention and every interference. On the other hand, well-consolidated traces of earlier experiences might remain intact. Sometimes these patients would forget the physician's visit after only a minute or two and would greet him anew if he returned to the ward. Disorientation in place and time (especially the latter) was very prevalent and there was little or no memory of the immediate past. Uncontrolled confabulation was very prominent in patients with lesions involving both the frontal and diencephalic systems though never in patients with circumscribed hippocampal lesions.

At the same time, it would be wrong to suppose that the immediate impressions disappear entirely. Simple motor skills [16] or conditioned reflexes can often be established and preserved and isolated items of information are sometimes retained over long periods.

In all cases of this type, experimental evidence suggests that the memory defects are due not to spontaneous trace decay but to interference effects. This is well shown even in cases with a clear Korsakoff syndrome: such patients can easily retain a series of 5 - 6 elements and retrieve it after a "free" interval of 1.5 ~~to~~ 2 minutes, and can often retain the gist of a paragraph and reproduce it after a "free" interval of similar duration. But when the interval is filled by an interpolated activity, e.g. simple computation or presentation of 2 - 3 further items, retrieval of the first series decreases from 90 - 95 p.c. to 55 p.c., or in the case of sentences from 95 - 100 p.c. to

60 - 70 p.c., and in experiments with whole paragraphs from 90 - 95 p.c. to 45 - 50 p.c. (10). Similar results are obtained with experiments on geometrical figures, pictures, movements, etc.

In this group, memory defect is in evidence at all levels of memory organization and (unlike what is found in the milder cases) even the organization of words into phrases or paragraphs does not improve retention.

As has been said, clouding of consciousness, confusion and confabulation were found in association with memory defect only in cases with lesions involving the mesial portions of the frontal lobe and the brain stem. Important is the fact that contaminations of the kind that have been described in the retrieval experiment often presage the development of confabulation at a clinical level. It may also persist in some degree after otherwise good recovery from an amnesic-confabulatory state.

A representative case of severe general memory defect may be briefly communicated:

Case R. Female, aged 40. Tumour of the posterior parts of the corpus callosum involving the hippocampal areas. Severe amnesic syndrome with disorientation in space and time and severe disturbance of recent memory. Pathologically increased blocking of retrieval by interpolated stimuli.

1. Form Recognition (Konorski Technique). A blue triangle was exposed for 5 - 6 seconds. After a "free" interval of 30 seconds, one minute or one and a half minutes, a second figure was given. This might be the same blue triangle, a blue square or a green triangle. The patient had to say whether the second figure was the same or different. With "free" intervals she could easily master this task but the task was invariably failed if an activity (e.g. simple computations, observing a picture) was interpolated between presentation of the two figures. Indeed in such circumstances the patient might not recall that any figure had been previously shown.

2. Size Contrast Effects (Uznadze "Fixed Set" Technique). Two wooden balls differing in size were placed in the two hands, the smaller in the left. The patient was asked to say which ball was the larger. After 15 trials the patient was presented with two identical balls. As in the normal, the patient judged the ball in the left hand to be the larger and this illusion was preserved after a "free" interval of up to two minutes. If, however, the interval were filled by an interpolated activity, judgements of relative size became random.

3. Retrieval of Words: "Heterogenous" Interference. No difficulties were observed in repeating sets of 3 - 4 words after a "free" interval of up to two minutes. But when the interval was filled by an extraneous activity such as picture description or simple computation, only one or two elements could be retrieved.

4. Retrieval of Words: "Homogenous" Interference. Given a set of 2 - 3 isolated words and then a second set of similar length and asked to recall first the first set of words and then the second, the patient failed completely. She said the

first set of words completely eluded her and while trying to retrieve them she also lost the second. This was seen even after 5 - 6 repetitions of the experiment, though sometimes a contaminated set was reproduced.

5. Reproduction of Pictures or Actions: Similar results were obtained if sets of unrelated pictures of actions were substituted for words.

6. Retrieval of Sentences: Short meaningful sentences of 4 - 7 words presented orally could be reproduced without difficulty after "free" intervals of up to 2 minutes. When a second sentence of comparable length was given immediately after the first, however, and the patient asked to retrieve the first and then the second, she either failed completely or gave a version of the second sentence only incorporating elements of the first (contamination). There was little or no improvement with repetition.

7. Retrieval of Paragraphs: The gist of a very short story, presented orally, could be reproduced (with some omissions) immediately afterwards and performance improved with 2 - 3 repetitions. If, however, a second story was read and reproduced immediately after the first, and the patient then asked to repeat the first story, the patient either failed to recall the first altogether or gave a contaminated version. Performance was not improved after 5 - 6 repetitions.

In this patient, the memory defects were clearly of a modality unspecific type, and equally clearly owed their origin to a blocking of recent memory traces by interference. It will also be noted that memory defect was present at all levels of semantic organization.

Partial (Modality-Specific) Memory Disorder

This type of memory disturbance is seen in patients with local lesions of the posterior parts of the convexity of the left hemisphere. It is not associated with confusion or clouding of consciousness, disorientation or personality change. Insight into the gnostic and memory disorders is intact.

The basic nature of all "partial" memory disorders is that they are limited by sensory modality. Defects in retaining visuo-spatial relations are seen in left occipito-parietal lesions and in retaining verbal-acoustic information with lesions in the left temporal or temporo-parietal zone.

In earlier experiments ~~of Pham Ming Hac~~, [12], it was shown that patients with temporal lesions could easily retain a set of 3 - 4 visually presented figures and retrieve them after "free" interval of up to two minutes but failed to retain a set of 3 - 4 acoustically presented isolated words; retrieval after a "free" interval of 1.5 to 2 minutes reached only 30 - 40 p.c. On the other hand, patients with left occipito-parietal lesions easily retained sets of 3 - 4 words presented orally but failed to retain equivalent sets of geometrical figures after a "free" interval of 1.5 to 2 minutes, especially if such groups were organized in complex spatial relationships. Under such conditions, they retained no more than 50 p.c. of the elements presented, and this fell to 30 - 35 p.c. if a factor of interference

This evidence shows that, in contrast to patients with deep midline lesions, memory defects with cortical lesions are due not only to interference effects but also to a primary instability and easy decay of traces within a specific modality. For this reason, the "recency effect" was very striking in such cases, e.g. patients with left temporal lesions tended to retrieve the elements in a set of words in inverse order of recency (Luria, Sokolow and Klimkowski, [14]).

A marked feature of the behavior of these patients is that active search strategies remain preserved, and the patients who are unable to retain separate elements often can grasp the general meaning of a sentence or even of a paragraph. In repetition of a story they may say "A sparrow" instead of "a blackbird", "a fly" instead of "an ant", but can easily retain the general sense of the whole story. This preservation of the whole program (or general meaning) associated with instability of particular elements is a very essential feature of this form of memory disturbance.

A

System-Specific Memory Disorder: Defect in Word Choice

A special kind of memory disturbance is seen in patients with lesions in the cortical "speech area", in particular the tertiary (temporo-parieto-occipital) zones of the left hemisphere. This is concerned essentially with verbal retrieval in the ordinary course of speech.

> It is well-known that incoming input has to be coded, and that coding by means of language plays an essential role in memory. It is accepted, too, that a word is by no means a simple association of a sound complex with an image; rather is it a multidimensional matrix having its phonetic, morphological and semantic structure. In its acoustic organization the word "cat", is closely related to "hat"; in its morphological organization, the word "blackbird" is closely related to "whitehead"; in its semantic meaning, "cat" can be related to "dog", or "kitten"; "school" to "college", "library", as well as to "teacher", "student", etc. Only a correct word choice within such a matrix can provide a selectively organized language-system.

This selectivity presupposes some basic physiological conditions, of which one is Pavlov's "rule of force", according to which strong (or important) stimuli evoke strong cortical reactions whereas weak (or unimportant) stimuli evoke weak reactions. Only if this rule obtains can appropriately selective function of the speech area take place. In pathological states of the cortex, however, the "rule of force" may break down leading to a series of "inhibitory states", in which both strong and weak stimuli may evoke equal reaction (stage of equalization) or when weak stimuli evoke stronger reactions than strong ones (paradoxical stage). Such conditions (when they affect the cortical speech mechanisms) may grossly disturb the selective organization of speech and the selectivity of verbal traces. Thus

weak or non-specific traces may come to be evoked at the same time as strong or important ones and word choice in verbal memory becomes impaired. Such is the case in "amnesic aphasia" seen with tertiary (temporo-parietal-occipital) lesions of the left hemisphere.

The patient with amnesic aphasia can easily retain traces of non-verbal stimuli and retrieve them even after "interpolated" activities. Marked difficulties occur when he tries to remember a word and to retrieve it. In such cases a whole matrix of equally probable connections is evoked and correct word-choice becomes extremely difficult. Thus, seeking the word "hospital", the patient may say "school" or "hotel" or "Red Cross". Trying to retrieve the word "teacher", he may say "preacher" (acoustic or morphological similarity) or "pupil" (semantic connection); access to the required word is blocked. This seems to be the physiological basis of paraphasia, the neurodynamic basis of which has been so little explored.

Memory Disturbances with Frontal Lobe Lesions

A very special kind of memory disturbance is seen in some patients with massive frontal lobe lesions. In such cases, there are no primary defects of retention and the apparent memory loss is due to a pathological change in the whole structure of behaviour. This is due to high distractibility on the one hand and pathological inertia on the other, resulting in a loss of programmed forms of activity [15]. In such cases, there is no true amnesia, general or partial, and good retention of a series of items in any modality after "free" intervals of two minutes or more.

The defect of retrieval in these patients results from an inability to create a stable intention to remember together with failure to "shift" their recall from one group of traces to another. In learning experiments, these patients fail to produce a typical learning curve and remain indefinitely on a "plateau". No strategy is evolved and no new elements added on repetition of the task. On the "retrieval" tasks described above, moreover, they were unable to shift from the first series to the second (pathological inertia) and failed to correct their mistakes. This phenomenon is especially clear with lesions of the left (dominant) frontal lobe and, if it extends into the temporal region, may be associated with additional symptoms such as "alienation of word-meanings". If such a patient is asked to retrieve the names of three objects (e.g. apple/hen/bottle), he names these objects by a inert repetition of the same word or by words associated with it, e.g. "apple... and pear .. and an apple" etc. In general, these findings indicate that defects of memory in frontal patients are secondary to a more general disturbance of active, programmed behaviour with associated inertia of higher nervous processes.

One example may be given of a patient with a gross frontal syndrome.

Case K. A student, aged 26. Massive trauma involving both frontal lobes.

Unconscious for 4 - 5 days followed by prolonged confusional state. The patient was lacking in spontaneity, akinetic and echolalic, and with marked perseveration and mnestic defects. The findings reported below were observed between one and three months after the injury was sustained. At interview, the patient's speech was sparse and he was very perseverative.

1. Retrieval of Words: "Heterogenous" Interference: The patient could retain sets of 2 - 3 or even 4 - 5 words and retrieve them after "free" intervals of up to one and a half minutes. If a simple computation was interpolated between presentation and recall, the patient substituted digits for the required words and denied that he had been told them.

2. Retrieval of Words: "Homogenous" Interference: The patient easily repeated two sets of isolated words given in immediate succession. But if then asked to repeat the first set once more merely repeated the second, without correction.

3. Reproduction of Drawings and Actions: The patient could retain sequences of 2 - 3 drawings or actions and reproduce them after a "free" interval of one and a half to two minutes. But when given a second similar sequence and asked to reproduce the first after having reproduced the second he failed completely, merely repeating the latter without alteration or giving a "contaminated" response with marked perseveration.

4. Retrieval of Sentences: Sentences could easily be retrieved after "free" intervals of $1\frac{1}{2}$ to 2 minutes. But when asked to retrieve one sentence after having retrieved a second, he merely reproduced the latter. Although recognising that the two sentences were different in meaning and pronunciation, he appeared powerless to inhibit the perseveration.

5. Retrieval of Paragraphs: During the early period of observation the patient started to reproduce the paragraph given but was unable to stop and continued to give free, uncontrolled association which might continue for 15 - 20 minutes (confabulation). A few weeks later, he became able to reproduce a paragraph but when a second paragraph was then given, its reproduction was much contaminated by the first and stereotyped errors were in evidence. Mistakes were never corrected.

It is clear that the apparent memory defects in frontal lobe patients are mainly due to lack of capacity to switch from one topic to another with consequent perseveration and gross failure to correct stereotyped errors. Retrieval is consquently rendered difficult and inaccurate.

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A.R. LURIA : MEMORY DISTURBANCES IN LOCAL BRAIN LESIONS

Résumé - (h)

L'étude neuropsychologique des troubles de la mémoire à la suite de lésions cérébrales focales a montré que ces troubles sont rarement le résultat d'un simple affaiblissement des traces. En général, ils sont dûs à des effets d'interférences provenant d'activités intercalées entre la présentation et le rappel; ceci est bien montré par des expériences simples dans lesquelles le rappel de courtes séries de mots, de dessins ou d'actions est demandée avec ou sans activité intercalée. Les effets d'interférence sont particulièrement marqués si l'activité ou le matériel intercalé ressemble étroitement au matériel ou à l'activité originale (interférence "homogène"). Les troubles de la mémoire de ce type, dont la gravité est très variable, sont particulièrement rencontrés dans les lésions profondes atteignant les portions internes des hémisphères et de la partie supérieure du tronc cérébral. Leur nature est tout à fait générale et ils sont ou non associés avec un état confusionnel selon que les lobes frontaux et le tronc cérébral supérieur sont, ou non, atteints.

Les troubles partiels de la mémoire, c'est-à-dire spécifiques quant à la modalité, peuvent être seulement en rapport avec des lésions de la convexité des hémisphères, surtout le gauche; ils ne sont jamais associés avec des états confusionnels ou confabulatoires.

Lors des lésions de l'aire du langage, un type spécifique de troubles mnésiques verbal (aphasie amnésique ou "misnaming") n'est pas rare. On propose une explication neurodynamique de ce trouble.

Lors des lésions massives frontales, on décrit un déficit mnésique secondaire au déficit de l'activité de programmation et une inertie pathologique des processus nerveux qui se révèlent dans les difficultés de déplacer l'attention et dans la persévération.

L'analyse neuropsychologique soigneuse des déficits mnésiques apparait ainsi essentielle pour comprendre les facteurs fondamentaux dont ils dépendent ainsi que le rôle du cerveau dans le processus mnésique.

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