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Memory Disorders Following Focal Neocortical Damage

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Memory disorders following focal neocortical damage

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Two aspects of memory defects following circumscribed neocortical lesion are considered. First, the selective impairment involving one category of stimuli (e.g. faces, colours) or a specific mnestic ability (spatial orientation). The deficit affects 'new' as well as 'old' memories and suggests that engrams are located in discrete cortical areas. The second issue concerns the relation of the hemispheric side of lesion to memory of non-verbal visual material, as a function of the differential utilization of the visual and verbal code in carrying out the task. Short-term memory tests are performed poorly by aphasics. In long-term memory tests, the performance depends on the nature of the task: in the early stages of paired-associate learning aphasics are impaired, on recurring figure recognition no hemispheric difference emerges, on sequential memory right brain-damaged patients have the poorest scores.

1. Introduction

It is the purpose of this paper to evaluate the contribution that the neocortex makes to memory functions, reviewing the data provided by clinical studies and experimental investigations carried out in patients with focalized brain lesions. Traditionally, the neocortex is not thought to be implicated in the circuitry of memory, since it is not damaged in patients presenting with the classical syndrome of global amnesia, nor is the lesion of any cortical area invariably associated with a definite amnestic picture. This is not to deny that memory deficits sometimes follow focalized neocortical diseases, e.g. a frontal lobe tumour (Hécaen 1964), but most students would agree that they are related more to the rise of intracranial pressure, namely a factor globally affecting the brain, than to the injury of a region playing a critical role in memory functions.

Yet it would be wrong to gather from the lack of a full-blown amnesic syndrome following neocortical damage that this part of the brain has nothing to do with memory, for it remains the most likely site where the neuronal changes underlying engram storage take place. The function of the limbic structures, though of paramount importance, is envisaged as critically related to consolidation (Milner 1968), retrieval-interference processes (Warrington & Weiskrantz 1970), or to the discrimination of the spatial and temporal contextual cues associated with an event (Huppert & Piercy 1976). Nobody, however, would identify them as the repository of engrams, which are conceivably stored near to the areas where stimuli have been processed at the time of perception. Since events are usually known through more than one modality, it is reasonable to assume that the objects of perception undergo a multiple coding and their traces are represented in discrete, interconnected cortical loci, making long-term memory resilient to circumscribed brain lesion. Moreover, man takes record of events by means of an additional code, language, which permits things to be remembered on the basis not only of their sensory features but also of their names, thus remarkably reinforcing memory power.

From the above assumptions one is led to make two predictions: (1) memory of those aspects

of reality that are processed through a single sensory channel, and are therefore coded in just one modality, will be vulnerable to focal damage so located as to destroy the corresponding associative cortex; (2) the disruption of the linguistic code, as seen in aphasia, will be reflected in a decrement of non-verbal memory performances, because of the failure of the aid provided to memory by verbal mediation. Some of the evidence relevant to these issues will be reviewed in the following pages.

2. Examples of selective memory defects following circumscribed neocortical lesion

(*a*) *Face amnesia*

Faces have a special status in the history of neurology in that they provide an example of a category of percepts that can be selectively disrupted after a focalized brain lesion. Prosopagnosia is the term used for defining the patient's inability to recognize people, including those who are long familiar to him, by their facial appearance, in spite of their preserved identification by other visual or auditory clues, e.g. clothing, a facial mark, voice. The disorder is relatively rare and available pathological evidence would appear to indicate that both occipital lobes must be injured for the deficit to become evident (Meadows 1974; Cohn *et al.* 1977), although clinical signs pointing to right-sided disease are usually more marked, and difficulty in recognizing unfamiliar faces in photographs has been repeatedly shown to be associated with posterior right hemisphere damage. The superiority of this side of the brain in processing faces has received support from the finding that right-handed normal subjects are more rapid and precise in identifying face photographs tachistoscopically projected to the left visual field than to the right visual field; as a consequence, faces have become a popular material among psychologists for investigating right hemisphere specialization.

The nature of the deficit underlying prosopagnosia is still a matter of debate but, from case reports that have been adequately investigated, it would appear that there are two different, though not incompatible, explanations: a few patients fail to identify familiar faces because of a perceptual impairment, and others because of a memory impairment. The basis for inferring whether the inability to recognize people by sight is due to faulty processing of the relevant visual features or to poor storage or recall of facial images is mainly afforded by the patient's performance on tasks requiring him to match snapshots of the same person taken from different perspectives and conditions of illumination, or variously disguised, i.e. tasks taxing perceptual skills, but not implying memory. If a prosopagnosic patient performs well on these tests, it can be legitimately argued that he is able to discriminate faces and that his inability to identify people by sight must be attributed to a faulty retrieval of facial images from his long-term memory. The assumption is not reversible, i.e., whereas a poor performance in matching unknown faces constitutes evidence of impaired perception, it does not exclude a memory defect but simply points to the need to corroborate its presence with non-perceptual performances. Theoretically, verbal description of the individual features of a face might appear to be the most suited method to assess facial memory; however apart from people's great variability in this skill, the examiner must be careful to distinguish the report of facial details testifying a mental representation from that of details simply manifesting a verbal knowledge of the marking features of a face. The patient of Lhermitte & Pillon (1975) knew that De Gaulle had a 'great nose', but remained doubtful as to whether he wore spectacles.

Case reports where faulty face perception was substantiated by impaired performance on

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unknown face matching tasks, are those of De Renzi *et al.* (1968) and Whiteley & Warrington (1977). On the other hand, Assal (1969), Tzavaras *et al.* (1970), Benton & Van Allen (1972) have described patients who performed the matching test normally and were nevertheless unable to recognize familiar faces. The two deficits can coexist. The patient of Lhermitte & Pillon (1975) failed to recognize different photos of well known politicians as belonging to the same person and, at the same time, could not evoke his own face as well as those of his close relation. There are hints that in some of these cases the inability to conjure up the mental images may extend to other categories of objects, e.g. buildings or animals. It is worth emphasizing, however, that prosopagnosia is not a necessary accompaniment of the symptom of loss of mental imagery, a condition where the patient complains of being unable to revisualize familiar objects, scenes and faces, in spite of his preserved ability to recognize them (Basso *et al.* 1980). These patients are unable to describe the distinctive features of the face of a close relative, or friend, but can identify him by sight without hesitation.

In conclusion, clinical evidence, in conjunction with experimental investigation, is supportive of the contention that a focal cortical lesion can result in the loss of face engrams, leaving the patient unable to recognize people on a pure physiognomic basis, if he cannot avail himself of the aid provided by other perceptual clues.

(*b*) *Colour amnesia*

Since colours represent a stimulus category that is unimodally perceived, they afford a good opportunity to test the hypothesis that engrams are stored in the areas neighbouring the cortex involved in perceptual analysis. If so, bilateral destruction of the visual cortex should result in colour amnesia, and patients with cortical blindness should present this symptom, although loss of vision *per se* suggests bilateral damage of the calcarine cortex and not of the visual association areas. Unfortunately, this aspect of the symptomatology is rarely mentioned by the clinical literature on cortical blindness, where a cursory reference to loss of visual imagery is sometimes made, but colour memory has never been specifically investigated (cf. Maginot & Hartmann (1927) and Symonds & Mackenzie (1957)). It must be recognized that its assessment is beset with difficulties, some patients being confused, others aphasic and blindness preventing their investigation with object–colour matching tasks not involving language. Waiting for future studies clarifying this issue, we can take advantage of the results of some recent researches, where the performance of patients with unilateral brain damage has been investigated on a variety of perceptual and verbal tasks dealing with colours.

In one study (De Renzi & Spinnler 1967) two colour-memory tests were administered, which required the association of colours with objects characterized by a definite hue. In one test the patient was presented with an object name and had to provide its colour name; in the other test he was presented with an object line drawing and had to colour it, choosing the appropriate pencil from a set of ten. The verbal memory test involves language and thus the finding that it was sensitive to the presence of aphasia is trivial: 79% of aphasics performed it poorly as against only 5% of non-aphasic left brain-damaged patients and 9% of right brain-damaged patients. Not easily predictable and much more intriguing is the finding that a moderate, but highly significant, impairment was also found when right brain-damaged patients who scored poorly in discriminating hues on Ishihara plates and a colour matching task, i.e. who showed central dyschromatopsia, were compared with the patients of the same hemispheric group who scored in the normal range on the same tests.

Since dyschromatopsic patients did not show any language impairment in other areas, their

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failure can be hardly explained in verbal terms and points to a specific revisualization inability, possibly contingent upon the proximity of the areas where colour are processed to those where their engrams are stored. Though mild, this memory deficit is remarkable, considering the unilaterality of lesion and the fact that the test involved many colour–object associations, which are deeply rooted in language, either because they designate a specific hue (yellow lemon) or because they are endowed with a metaphoric meaning (red-blooded, white as a sheet). I suspect that the impairment would stand out even more clearly if it were possible to test patients with questions related to unconventional associations, such as the colours of personal garments and belongings, which cannot be described simply by relying on verbal knowledge.

Another type of colour amnesia, implying quite different mechanisms, was the impairment shown by nearly 50% of aphasic patients in appropriately colouring drawings of objects that are naturally or by social convention associated with a particular colour. For instance, a patient chose the red pencil to colour a banana and the green pencil for cherries. The aphasics' deficit on this entirely non-verbal task has been repeatedly borne out by subsequent research (Tzavaras *et al.* 1971; De Renzi *et al.* 1972; Basso *et al.* 1976; Cohen & Kelter 1979; Varney 1982) and is conspicuous not only in relation to the controls' performance, but also to that of non-aphasic left hemisphere-damaged patients, right hemisphere-damaged patients and schizophrenics. Though less striking, because the task is easier, a comparable selective impairment was also found when the task was turned from a recall into a recognition performance, requiring the patient to distinguish absurdly coloured from correctly coloured drawings (Basso *et al.* 1976).

Does the defective activation of the bond linking a drawing with its colour depend on the unavailability of their names, as the selective impairment of aphasics would suggest? There are many reasons to question the validity of this interpretation. First, the size of the correlation between memory deficit and aphasia severity reported by De Renzi & Spinnler (1967) is not impressive (0.48) and there are a number of severe Wernicke patients who perform in the normal range (De Renzi *et al.* 1972). Second, the performance of aphasics remained significantly poorer, even when the influence on the colouring scores of the scores obtained in naming and understanding the names of the colours and drawings used in the matching task was partialled out with covariance analysis (Basso *et al.* 1976). Third, since both the pencils and the drawings were under the subject's eyes, it is intuitively unconvincing that their matching could only be made by resorting to verbal mediation. I am therefore inclined to view this amnesic deficit as an aspect of a more general cognitive disorder of aphasics in constructing and maintaining firm concepts, so that it becomes hard to amalgamate the defining features of an object and to recover from memory one of them when the other is present. The relation of this deficit to the language disorder is open to question.

(*c*) *Topographical amnesia*

The information specifying the location of an object in the environment and the spatial relation that it bears to other objects is conveyed to the cortex through different sensory channels, but a substantial body of clinical and experimental evidence suggests that spatial data are ultimately processed in the posterior parietal lobes, with an ascendant participation of the right one.

One of the earliest manifestations of spatial disability reported by the neurological literature, and probably the best documented example of a memory deficit confined to one category of

experiences, is represented by the inability to learn a new route and to take one's bearings in familiar surroundings, in the absence of any sign of global amnesia. Although the deficit is at times merged into the complex picture of spatial disorientation, and thus may be difficult to analyse adequately, there are cases where it appears in isolation, independently of scanning and perceptual impairment. In the wards the patient gets lost when he must find his way back to the room, hesitating at any turning point whether to go left or right, and recognizes his own bed only by relying on some distinguishing feature, e.g. a particular picture or furniture, and not on the basis of its spatial relations. The same obtains when he must take his bearings in surroundings well known to him before illness: street names, sign-boards and bus numbers are recognized as familiar, but fail to provide clues for choosing the correct direction, because they have lost their topographical value. Patients are also impaired in giving a verbal description of an itinerary and in locating cities and states on a blank map. The selectivity of the deficit is shown not only by the patient's preserved ability to report everyday events and by his intact performance on tests requiring to learn verbal and visual material, but also by clinical observations pointing to an opposite pattern of amnesia, namely patients who forget everything and yet have no trouble in taking their bearings in old as well as in new surroundings (De Renzi *et al.* 1977*c*).

Paterson & Zangwill (1945) pointed out that two discrete deficits may underlie the disorder, agnosia for buildings, which are landmarks for taking one's bearings, and topographical amnesia, i.e. the inability to recall the spatial layout of a route. In the majority of cases, the latter rather than former symptom appears to play a crucial role.

A growing body of evidence has accumulated relating spatial amnesia to the dysfunctioning of right posterior areas, confirming early conjectures made at the beginning of the century by neurologists and based on the prevalent association of topographical amnesia with left hemianopia. In the majority of patients for whom reliable localizing data were available (Pomme & Janny 1954; Hécaen *et al.* 1956; Vighetto *et al.* 1980; Aimard *et al.* 1981), the brunt of injury was borne by the temporo-parieto-occipital junction, but there are at least two case reports (Gloning *et al.* 1955; Hécaen *et al.* 1980) where damage was confined to the territory of distribution of the posterior cerebral artery, namely the medial occipital lobe.

The asymmetric participation of the hemispheres in spatial memory has been substantiated by studies that investigated unselected series of patients with unilateral brain lesions for their ability to learn a path on a maze or to tap a given sequence of blocks that are identifiable only by their spatial position. Right posterior-damaged patients were consistently found to be impaired relative not only to normal controls, but also to other groups of patients with damage confined to one hemisphere. I shall cite as an example the outcome of two personal investigations (De Renzi *et al.* 1977*a*, *c*). One was carried out with a stepping stone maze similar to that first introduced in neuropsychology by Milner (1965), where patients have to memorize a path discovered by trial and error, and the other with the tapping cube test devised by Corsi (Milner 1971), where patients have to learn a sequence, two cubes in excess of their span. Learning was carried on until the criterion of three successful runs was attained or 50 trials were given. As shown in table 1, on both tests patients with right posterior damage required a significantly higher number of trials to criterion than any other group and a greater percentage of them were unable to reach criterion in 50 trials.

A comparable deficit was found by Milner (1965, 1971) in epileptic subjects who had undergone the excision of the right hippocampus and who too showed a specific impairment

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in learning a path on the stepping stone maze or a block sequence in Corsi's test. The main difference between neocortical and limbic patients is to be sought at the clinical level, because no patient with hippocampal removal has ever been reported to lose his bearings, unless the ablation was carried out on both sides. Even in this case the topographical deficit involved anterograde memory and not retrograde memory, i.e. it concerned the ability to find a route in surroundings that had been practiced after the disease and did not extend to those that were familiar before it. The same holds true with Korsakoff patients, whose relatively spared topographical memory even in the hospital setting was underlined by Talland (1965) and contrasted with their profound amnesia for whatever had occurred to them after onset of the disease.

Table 1. Mean trials to criterion of control and brain-damaged patients on two spatial memory tests

	controls	LH−	LH+	RH−	RH+
visual maze	13.41	15.98	21.31	20.47	31.76
cube tapping	12.80	20.25	27.50	18.75	41.35

Thus topographical amnesia represents the most impressive example of a selective memory deficit closely linked to neocortical damage. Whether it is due to a permanent loss of engrams or to a failure to retrieve them remains an open question.

3. The relation of language impairment to non-verbal memory

The study of patients with focal injury affords the opportunity to address the question of whether language facilitates memory of visual material by adding a verbal code to the imaginal code. The rationale for such an inquiry stems from the observation that man tends to remember concrete experiences not only as images, but also as names, and from the hypothesis that memorial representation and subsequent retrieval of stimuli derive benefit from this dual coding. There has been much debate among psychologists concerning the differential effect of verbal labelling in relation to the nature of the visual material and the demands of the task, and an answer has generally been sought by manipulating the label features so as to interfere with verbal mediation. Aphasia offers an alternative to these procedures. If the ability to provide a verbal tag to a visual stimulus facilitates its encoding and retrieval, aphasic patients who have lost this ability should be impaired in comparison with patients having comparable brain lesions but no difficulty in using the linguistic code.

We have carried out a number of studies, with the aim of relating the patients' performance to the nature of the memory task and the degree of language deficit. The general methodology followed in these investigations was to compare unselected samples of right-handed control, right brain-damaged and left brain-damaged patients on visual recognition of meaningful figures, the expectation being that, if verbal coding is spontaneously used by the subject to memorize easily labelled stimuli, left hemisphere damage should result in a detectable impairment, because of the frequent occurrence of aphasia in patients with a lesion on this side.

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(*a*) *Short-term memory*

Different patterns of results have emerged, depending on whether a short-term or a long-term memory paradigm was used. In the former condition the interval between stimulus presentation and recognition is kept brief and free from interference, and the number of presented stimuli is small. At its most elementary level, short-term memory can be tested as memory span, i.e. the number of items that the subject is able to reproduce, or recognize immediately after their presentation, maintaining the same order. When this procedure was used with presentation of picture strings of increasing length, followed by their recognition on a display (Cremonini *et al.* 1980), left brain-damaged patients scored significantly worse than normal controls, while right brain-damaged patients did not. The result was similar to that obtained with a verbal span test, digit forwards, although only on the latter test did left hemisphere-damaged patients also do worse than right hemisphere-damaged patients. That in both tests the memory deficit was contingent on the language deficit was substantiated by the finding that group differences disappeared when Token Test scores were introduced as covariates in the analysis of the memory span scores. An analogous reduction of aphasics' visual span was reported by Goodglass *et al.* (1974), who demonstrated in addition that aphasics are not affected, as patients without language deficit are, by the homophony of picture names, which suggests that they do not avail themselves of covert verbal mediation.

Consistent with the idea that the availability of the linguistic code helps in the retrieval of visual stimuli from the short-term storage are the results of an investigation (De Renzi & Spinnler 1966) where a card with six animal figures was presented for 10 s, immediately followed by a card bearing twelve animal figures, one of which alone was common to the former card and had to be recognized. Aphasics were significantly less accurate than left non-aphasic and right brain-damaged patients. Also Ammon (1973) found aphasics impaired in a figure-matching test, while the negative results reported by Kelter *et al.* (1977) might be due to the insufficiently demanding characteristics of the task.

There is, however, a problem with a straightforward interpretation of these findings in terms of defective verbal coding of stimuli. Neither De Renzi & Spinnler nor Ammon found the memory scores of their tests to be significantly correlated with oral naming and comprehension scores, i.e. with measures of aphasia severity. It is not easy to reconcile this negative finding with the selective failure of aphasics, and with the evidence provided by the above mentioned investigations on visual span, which did show language impairment to be correlated with memory deficit. The question deserves further investigation with more sensitive measures of aphasia severity.

(*b*) *Long-term memory*

Long-term mechanisms are thought to be implicated when the number of items presented exceeds the capacity of the short-term storage and their retention is tested after a certain delay filled with interference. Again, its evaluation in brain-damaged patients is achieved better through recognition than through recall in order to avoid confounding aphasics' speech disruption with impaired verbal recall.

Data so far collected on this subject suggest a differential participation of the hemispheres in memory performance, as a function of the characteristics of the material, the nature of the task and the stage at which learning is assessed. We have investigated the issue by using three

different procedures: recurring figure recognition, paired-associate recognition and reconstruction of the serial order of item appearance.

In the first task, stimuli that have been previously presented in a study trial must be recognized as 'old' when they reappear in successive blocks intermingled with 'new' stimuli, never seen before. The study carried out in brain-damaged patients by De Renzi & Spinnler (1966), in which 'old' and 'new' stimuli belonged to the animal category, showed no hemispheric difference, even when left brain-damaged patients were divided into aphasic and non-aphasic patients and the comparison was confined to the difference between the former group and right brain-damaged patients. Since the patients in this investigation were the same as in the short-term memory study carried out by the same authors (see above), where an inferior performance of aphasics was shown, it would appear that language impairment plays a different role according to whether information must be retrieved from the short-term or the long-term store. The cogency of this conclusion must, however, be diminished in view of the results obtained when paired-associate recognition was used instead of recurring figure recognition to assess long-term memory.

With paired-associate recognition, pairs of unrelated figures are given to examine one after the other and at the end of the study trial the first member of each pair is presented again together with a multiple choice display for the subject to recognize the associate. We employed this technique in two studies, and results were consistent in showing the bearing of language impairment on the performance. In the first investigation (Boller & De Renzi 1967), there were 10 pairs of meaningful figures and learning was carried out for three successive trials, so that the maximum score a patient could achieve was 30. Right brain-damaged patients (mean 19.65) were only mildly inferior to normal controls (mean 21.24), while left brain-damaged patients (mean 14.87) were significantly impaired in comparison not only with controls, but also with right brain-damaged patients. That the poor performance of this group was due to aphasia was confirmed by the finding that the difference was markedly reduced and fell far short of significance when the influence of language disruption on the memory performance was partialled out by covarying for visual naming and sentence comprehension scores. Essentially the same result was obtained in the second study (De Renzi *et al.* 1981), which too showed that after three trials the impairment was confined to left brain-damaged patients and that the correction of the memory scores, achieved by introducing Token Test scores as a covariate in the analysis, was significant. In this study, however, the test was not stopped after the third trial, but continued until the criterion of two errorless runs was reached or 25 trials were given, therefore making it possible to assess learning at a much more advanced stage. When trials to criterion were compared across the groups, it was found that the significant factor in disrupting the memory performance was no longer the hemispheric side of lesion but the presence of visual field defects. Moreover, the Token Test correction of memory scores fell far short of significance. There are indications, therefore, that with repeated stimulus presentation the aid provided by the verbal code in identifying the member of the pair progressively fades and the patient relies more and more on the visual code.

A different aspect of memory that the studies so far reviewed have not taken into account is the retention of the order of appearance of stimuli, i.e. the ability to assign to each item of a series the position that it occupied when first presented. In the procedure that we used in two successive investigations (De Renzi *et al.* 1977*b*; Cremonini *et al.* 1980), patients were first presented with a number of pictures in succession and then with the complete array of pictures

laid down on the table in a different order with the request to rearrange them in the original sequence. Study trials, always with the same order of presentation, and test trials, always with a different order of presentation, followed one another until the criterion of three correct reconstructions was reached or 50 trials were given. On this task, in which all the stimuli of test trials are signals, right and not left brain-damaged patients were found to be impaired. The mean number of trials to criterion was increased with respect to normal controls in right posterior-damaged patients (table 2), and the proportion of patients failing to reach criterion in 50 trials was significantly greater in the right-sided than in the left-sided group. Covarying for Token Test scores did not change this pattern of results.

TABLE 2. MEAN TRIALS TO CRITERION OF CONTROL AND BRAIN-DAMAGED PATIENTS IN TWO SEQUENTIAL MEMORY STUDIES

	controls	LH−	LH+	RH−	RH+
De Renzi *et al.* (1977*b*)	9.89	10.94	13.59	9.75	16.15
Cremonini *et al.* (1980)	8.58	12.27	14.30	18.33	22.90

In view of the failure of the verbal code to activate visual memory in the paired associate paradigm when trials were continued until the attainment of criterion, the question may be raised of whether the absence of a left hemisphere damage effect on sequential memory is contingent upon the use of trials to criterion as measure of impairment. The analysis was therefore repeated on the data of Cremonini *et al.* (1980) with a different statistic, number of errors in the first ten trials, which permitted the studying of learning at an earlier stage. No remarkable difference with respect to previous results came out: right brain-damaged patients performed significantly worse than normal controls and patients with right posterior lesions were also impaired relative to patients with left anterior damage, of whom a number were aphasic.

What inferences can be drawn from this varied pattern of results with respect to the differential contribution made by the two sides of the brain to non-verbal memory? The effectiveness of the verbal code in reinforcing stimulus retention and retrieval seems to be maximal in the early stages of the memory process, when visual engrams are still labile and exposed to the disrupting influence of interference, so that a remarkable advantage accrues from the availability of an additional code. When learning is assessed at a more advanced stage, which is entirely dependent on a long-term memory mechanism, the use of the verbal code depends on the depth of processing required by the test. Recurring figure performance rests upon quick discrimination between signals and noises, and a figure is identified as a signal when the feeling of familiarity that it produces exceeds a certain critical value. This sensitivity is not sufficient with the paired-associate paradigm that we used, because only half the foils shown on the multiple choice displays were 'new', while the remainder consisted of the second members of other pairs and thus were as 'old' as the signal. In order to make a correct choice, the patient has to focus on the bond linking the arbitrarily paired figures, a difficult task that favours the development of auxiliary strategies, of which verbal mediation is one. Consequently, aphasics, who cannot avail themselves of the verbal code, are at a disadvantage. Verbal coding seems, however, to be an emergency device, spontaneously relinquished by the subject when, with trial repetition, visual engrams acquire a certain degree of stabilization.

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In the sequential memory test the problem is not the identification of the correct stimulus from foils, since all the pictures presented in test trials are signals. What the patient has to do is to build up in memory a chain of images corresponding to the order with which items were seen in study trials. While it is in principle conceivable that the performance may be facilitated by memorizing a parallel series of names, in fact this possibility was not exploited and the task was carried out by relying on the visual code. The deficit shown by patients with injury of the right side suggests that the right hemisphere plays a pre-eminent role when visual imagery is deeply engaged.

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