A unilateral memory defect¹

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SYNOPSIS Several authors have suggested that arousal is an important component of learning. If the neglect syndrome is a unilateral defect of alerting (arousal) as recent studies have suggested, then patients with this syndrome should have a unilateral learning defect. To test these hypotheses, eight patients with unilateral neglect were given consonant trigrams randomly to either ear, and the patients were asked to identify the auditory stimuli either immediately or after an 18 second delay during which time the patients were asked to count. All eight demonstrated that the number of errors between the delay and non-delay conditions was greater on the neglected side (significance > 0.01), thereby supporting these hypotheses.

A recent study suggests that an attentional defect may be the basis of the neglect syndrome (inattention, hemispatial agnosia, extinction) (Heilman and Valenstein, 1972a; Watson *et al.*, 1973a, b). This attentional defect has been defined as a unilateral defect in the alerting (arousal) response to sensory stimuli (Watson *et al.*, 1973a).

William James (1890) noted 'that an object once attended to will remain in the memory whilst one inattentively allowed to pass will leave no traces behind'. More recently, Sokolov (1960, 1963) and Magoun (1969) have also suggested that alerting is an important component of learning. If neglect is a unilateral defect in alerting and alerting is important in learning, then one would expect a patient with unilateral neglect to have unilateral learning disorders. The purpose of the study was to test this hypothesis.

METHODS

Subjects for this study were eight patients from the Neurological Division of the University of Florida Shands Teaching Hospital and Veterans Administration Hospital, Gainesville, Florida. Only patients who were tested for and demonstrated auditory neglect were included. Auditory neglect in the study was assessed by several bedside methods of unilateral

and bilateral simultaneous stimulation (Bender and Diamond, 1961; Heilman and Valenstein, 1972b). Patients were given a unilateral stimulus to each ear. If they were able to hear the stimuli, they were given stronger stimuli in a random fashion to either the right, left, or both ears. If a patient were consistently able to identify unilateral stimuli but consistently missed one side with bilateral stimuli, he was considered to have auditory neglect. Three of these patients had infarction of their non-dominant cerebral hemisphere and one had cerebritis of her non-dominant parietal lobe. Two patients had tumours. One had metastatic disease to her non-dominant frontal lobe, and the other had an astrocytoma of his non-dominant parietal lobe. One patient had trauma to his non-dominant hemisphere, and one patient had non-dominant hemisphere dysfunction of unknown aetiology with seizures.

PROCEDURE Thirty-two consonant trigrams—that is, JQW—of less than 21% association value (Witmer, 1935) were given randomly to either right or left ear. These 96 letters were delivered from a Revox tape recorder deck via Sony DR-7A headphones. The subject's task was to identify the auditory stimuli immediately after presentation (non-delay) or after a delay of 18 seconds during which time the subjects were requested to count (Peterson and Peterson, 1959). These 32 consonant trigrams and procedures were repeated to each subject with the earphones reversed and the initial earphone position was alternated with each subject. The delay

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TABLE
DIFFERENCE BETWEEN NUMBER OF ERRORS IN DELAY AND NON-DELAY CONDITIONS

Patient	Neglected side	Errors non-delay		Errors delay		Difference in errors delay-non-delay		Difference of errors (delay-non-delay) between sides (neglected side-non-neglected side)
		Neglected side	Non-neglected side	Neglected side	Non-neglected side	Neglected side	Non-neglected side	
1	L	1	0	35	23	34	23	11
2	R	3	0	21	2	18	2	16
3	R	1	1	21	17	20	16	4
4	L	3	7	11	8	8	1	7
5	L	2	0	36	30	34	30	4
6	L	0	0	33	27	33	27	5
7	L	4	3	37	30	33	27	6
8	L	11	19	19	12	8	-7	15

and non-delay conditions were also selected randomly.

ANALYSIS OF DATA If a patient missed consonants in the non-delay condition, we considered that there was either a defect in hearing, attention, or perception. Since the stimuli were the same for delay and non-delay conditions, the difference between the number of errors in these conditions should reflect what is lost going to short-term memory. If this difference is higher in the ear on the side with neglect than it is in the side without neglect, it would suggest that there is a defect in memory associated with neglect.

RESULTS

In the non-delay condition there was no significant difference between the ears. All eight subjects showed that the difference in the number of errors (between the delay and non-delay conditions) was greater on the neglect side. This was significant at the 0.004 level (Table) using a binomial test.

DISCUSSION

Butters *et al.* (1970) assessed patients with right hemisphere damage to ascertain if there were a

FIGURE Cortic limbic-reticular formation loop.

visual or auditory memory defect associated with these lesions using the Peterson-Peterson (1959) procedure, which permitted separation of the perceptual and memory components. They found that there was a visual memory defect; however, they did not find a memory defect for auditory stimuli. This suggested to the authors 'that the deficit of the right parietal group is not a general memory impairment but rather one restricted to the storage of visually presented material'.

The mechanism for this memory disorder is not readily apparent. It is known, however, that lesions which occur in the right parietal lobe are frequently associated with the syndrome of neglect. It has been hypothesized that unilateral neglect is a unilateral defect of alerting caused by a corticolimbic reticular activating disconnection (Heilman and Valenstein, 1972a; Watson *et al.*, 1973a). Partial support of this hypothesis comes from anatomical and ablative studies. In monkeys it has been shown that primary sensory areas project to their association cortex, then to the arcuate gyrus in the frontal lobe and to the inferior parietal lobule and the posterior portions of the superior temporal gyrus (Pandya and Kuypers, 1969). These, in turn, project to the cingulate gyrus (and perhaps other portions of the limbic system) which has extensive connection with the mesencephalic reticular formation (Nauta, 1964) (Figure). Lesions in the frontal lobes in both man (Heilman and Valenstein, 1972a) and monkey (Welch and Stuteville, 1958), and parietal lesions in man (Critchley, 1953; Heilman and Valenstein, 1972b) and monkey (Heilman *et al.*, 1970; Eidelberg and Schwartz, 1971) have produced neglect. In addition, lesions of the cingulate gyrus (Watson *et al.*, 1973a) and mesencephalic reticular formation (Watson *et al.*, 1973b) also produce neglect. These lesions all have something in common: they all are capable of producing disconnection of the corticolimbic reticular activating system. Electrophysiological studies give further support to the arousal hypothesis. French *et al.* (1955) stimulated different parts of the cortex while recording from the reticular activating system. The areas which showed corticofugal projections to the reticular activating system were the same as those causing neglect when ablated. In addition, the discrete lesions in the mesencephalic reticular formation which produces profound

neglect causes diffuse electroencephalographic (EEG) slowing in both cats (Reeves and Hagamen, 1971) and monkeys (Watson *et al.*, 1973b). Cortical lesions which produce neglect are also with diffuse EEG changes both in man (Heilman *et al.*, 1973) and cats (Reeves and Hagamen, 1971).

If we accept the hypothesis that unilateral neglect is a unilateral defect in alerting, what then is the relationship between attention and learning? Support for the hypothesis that attention is not only important for perception but is also important for memory comes from the studies of Norman (1969) who asked patients to repeat what they heard as soon as they heard it from one ear (shadowing). For perfect shadowing, attention must be paid to one ear. In this task another message is simultaneously being given to the other ear. Usually little or nothing is remembered of the non-shadowed message. To determine if the non-shadowed message is perceived and remembered briefly before the 'trace disappears', Norman interrupted subjects while they were shadowing and quickly demanded of them what they heard in the non-shadowed ear and found that the trace was present.

Alerting appears to be mediated by the reticular activating system which has been thought by some to be an amplifying system (Sokolov, 1960). This amplifying system increases cortical excitability (Sokolov, 1960), increases discriminating power (Lynn, 1966), and therefore probably enhances learning. Further support that arousal is important for learning comes from studies which show that learning is facilitated by stimulatory drugs (Lashley, 1916; Irwin and Benuazizi, 1966) and by stimulation of the reticular activating system (Zuckermann, 1959; Irwin and Benuazizi, 1966).

A major discrepancy between Butters *et al.*'s study (1970) and this report is that they found no auditory memory defect in patients with right hemispheric lesions. There are two possible reasons. First, they presented their auditory stimuli in a free field and therefore could not compare one side with the other. Secondly, although neglect is frequently polymodal—that is, visual, auditory, somaesthetic—it is not necessarily so and, while our patients had

auditory neglect, we are not certain that their patients demonstrated auditory neglect.

Kimura (1963) and Milner (1962, 1967, 1968) have reported that memory storage in the temporal lobe is differentiated not by sensory modality but by the type of material being stored—that is, verbal on the left and spatial on the right. Samuels *et al.* (1971a) compared the patients with cortical brain damage studied by Butters *et al.* (1970) and compared these patients with patients with Korsakoff's syndrome. She found that the visual memory defect was similar in both groups and that the major difference was that the cortical group (non-limbic) had a defect which was modality specific (visual). Our study suggests that the defect is not modality specific and therefore the differentiation made by Samuels *et al.* (1971a) is not valid. However, we do feel that there is a difference between our patients and those with Korsakoff's syndrome on one hand and patients with temporal lobe ablations on the other. Unlike the Korsakoff and temporal lobectomy patients, our patients have a memory defect which is asymmetrical and affects mainly those stimuli coming in on the side with neglect. Support for this observation comes from an additional paper by Samuels *et al.* (1971b) in which they performed further analysis on their data and showed that the visual memory defect was worse in the contralateral field.

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