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MEMORY DISORDERS IN APHASIA— I. AUDITORY IMMEDIATE RECALL*

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Abstract—Vascular lesions within the territory of the left middle cerebral artery were identified by CT scans for 28 aphasic adults. When damage involved parts of Heschl's, middle temporal and superior temporal gyri or the inferior parietal lobule, immediate recall of binary sequences of either digits or tones was only 3 ± 1 bits. Lesions to various loci were identified with primacy, recency, transposition, and isolated errors. The discussion considers various neural mechanisms subserving auditory immediate recall.

RECALL of numbers or other items immediately after presentation was first studied in ... by JACOBS [23] and he called this memory, "span of apprehension". Thereafter, JAMES ... in 1890, termed it "primary memory". Twentieth century labels for this same ...mory have included "short-term", "buffer", or "recent", but the term now frequently ...d in neuropsychological research is "immediate memory". Immediate recall for a ...uence of items endures only seconds after presentation unless the material is rehearsed ...].

...One of the substrates of verbal language is the ability to recall sequences of auditory ...rmation. MILLER [28] demonstrated that the amount of information that can be held in ...mediate memory is limited; in the neurologically intact adult, only 7 ± 2 digits can be ...rrectly reproduced. The neuropsychological consequences of a profoundly diminished ...an of auditory immediate recall have not been systematically explored. In the majority of ...asic patients, immediate memory is probably more impaired than occurs as the result of ...y other neurological syndrome in adults with circumscribed cortical lesions. Patients with ...petition defects and usually labeled as conduction [42, 44, 45], WERNICKE's [13], BROCA's ...], mixed and global aphasics correctly repeat in order only about 3 digits. The defect is ...so manifested by a deficient pointing span as measured by the number of common objects ...ese patients can point to when imitating the examiner. The pointing span is limited to only ...out 2–3 items in the correct order [1, 2, 17, 13].

The lesions producing deficits of auditory immediate recall have reportedly involved the ...rritory surrounding parts of the Sylvian fissure of the left hemisphere including the inferior ...rietal lobule, the posterior aspect of the superior temporal gyrus (Wernicke's area), the ...ddle temporal gyrus, and anterior perisylvian structures including Broca's area [5] (see also ...HR [30, 31] for negative evidence regarding the enduring effects of damaging Broca's area).

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Whenever these perisylvian structures are spared, as in transcortical motor [6], transcortical sensory [25] or mixed transcortical aphasias [16], auditory immediate recall is essentially intact. In addition, non-aphasic adults with damage to the left hemisphere or right hemisphere [13] did not differ significantly from normal controls in their digit or pointing span.

Immediate memory also remains intact in amnesic disorders such as following bilateral mesiotemporal resection [38] and following degeneration of diencephalic structures (dorsomedial thalamus and mammillary bodies) in alcoholic Korsakoff syndrome [8, 43]. Thus, at least some aphasic patients are apparently more likely to be able to learn new information than Korsakoff syndrome patients [10]. Further, left temporal lobectomy produces deficient learning of verbal materials without a deficit of immediate recall or aphasia [29]. However, when aphasia occurs as the result of infarction of the middle cerebral artery, some degree of amnesia may presumably coexist if the temporal polar region is damaged. By contrast, infarction of the middle cerebral artery spares structures of the mesiotemporal lobe and diencephalalon identified with some amnestic disorders.

Although various amnestic patients are reportedly more likely than controls to forget the initial item in the free recall of serially presented verbal lists (a reduction of the primacy effect) [4], aphasic patients with a selective disturbance of immediate memory were more likely to forget the final item of a list (a reduction of the recency effect) [37, 44]. Consequently, it is informative to examine in aphasic patients the lesions associated with primacy and recency errors.

ALBERT [11] proposed that the left hemisphere is dominant in its capacity to sequence information, irrespective of sensory modality. By contrast, DE RENZI and NICHELLI [13] and KIM *et al.* [26] have reported that various patients with right hemisphere damage are more deficient than aphasic patients in their ability to sequence visual and tactile stimuli. It has not yet been determined, however, whether aphasic patients are as impaired in their immediate auditory recall of non-verbal (sung) sequences as they are in their recall of verbal (spoken) sequences. This question may be germane to the study of how Melodic Intonation Therapy facilitates recovery of propositional language. This therapy utilizes sung verbal sequences in the rehabilitation of various nonfluent aphasias [2, 40]. It has been hypothesized that the effectiveness of Melodic Intonation Therapy is related to the excellent sparing of singing among most aphasic patients, an ability which may utilize predominantly the right hemisphere [47]. If immediate recall of verbal and tonal sequences are equally impaired in aphasic patients, this result would support the concept that the left hemisphere is dominant in its capacity to sequence all novel auditory stimuli. Thus, it is the aim of the present investigation to establish the range over which deficits of auditory immediate memory hold for sequences of digits and tones among patients with distinct aphasia syndromes and lesions. With the advent of CT scans, this objective can be achieved with greater precision than previously possible in helping to establish how damage to various neuroanatomical loci contributes to the breakdown of different aspects of auditory immediate memory in aphasic disorders.

METHOD

Subjects

Aphasia in the 28 patients of this report resulted from a single cerebral infarction within the territory of the middle cerebral artery of the left hemisphere as determined by clinical neurological and CT follow-up. The patients, 26 males and 2 females, ranged in age from 34 to 78 yr, with a mean of 58 yr. All subjects had been right-handed, native speakers of English and had received at least a high school education.

Aphasia testing was initiated at least 2 months post-onset of symptomatology at the Palo Alto Veterans Medical Center. Aphasia classification was based on tests including the Boston Diagnostic Aphasia Examination by GOODGLASS and KAPLAN [19], the Token Test by SPREEN and BENTON [41] and the FAS Word Fluency Test by BENTON [7, see 33]. At the time of testing immediate memory, seven patients were designated as transcortical motor, three as Broca's, three as Wernicke's, five as conduction, seven as mixed and three as global aphasics.

Radiological studies

CT scans were obtained at the Palo Alto Veterans Medical Center with a Syntex Systems 60 CT Scanner. Scans were taken 20 degrees to the orbito-meatal line with each horizontal slice representing a 1 cm thick section of brain. Ten consecutive slices were constructed from the level of the base of the brain to the top of the skull. The scans used to localize the lesions were taken at least 2 months post-onset of symptoms with a mean of 30 months and were obtained within 4 weeks of testing for immediate memory and aphasia.

The presence of a lesion in each of 21 structures within the territory of the left middle cerebral artery was indicated for each patient by a staff radiologist (R.W.H.). The sites were labeled as: (1) frontal zone (anterior to the precentral gyrus and to Broca's area); (2) Broca's area (posterior portion of the third frontal convolution); (3) head of the caudate nucleus; (4) lenticular nucleus; (5) anterior limb of the internal capsule; (6) genu of the internal capsule and (7) posterior limb of the internal capsule; (8) zone of the external capsule (external capsule, claustrum and extreme capsule); (9) insula; (10) temporal pole; (11) middle temporal gyrus; (12) Heschl's gyrus; (13) cortical portion of Wernicke's area (i.e. the posterior two-thirds of the superior temporal gyrus); (14) white matter underlying Wernicke's area; (15) cortical portion of the supramarginal gyrus; (16) white matter underlying the cortex of the supramarginal gyrus; (17) cortical portion of the angular gyrus; (18) white matter underlying the angular gyrus; (19) precentral gyrus; (20) postcentral gyrus; and (21) corona radiata. The localization of lesions involving language-related structures was performed according to cranial and ventricular landmarks [see 19].

Table 1 depicts the configuration of lesions detected with CT scan film for each of the 21 loci among the 28 patients. A + designates the presence of a lesion. The structures presented in Table 1 are usually listed from anterior to posterior loci at horizontal planes from ventral to dorsal. Following the terminology of NAESER *et al.* [33], structures 1–11 were identified as occurring as ventral as slice B (including portions of Broca's area), 12–14 originated as slice BW (including aspects of Broca's and Wernicke's area) and 15–18 originated at slice SM (at the level of the supramarginal gyrus).

Analysis of data

A variation of the method of "multiple dissociation" based on an "intersect of sums" technique [36] was used to analyze the results. The scores of all patients with a lesion of a particular structure were compared by a one-tailed *t*-test with the scores of the remaining patients. Following this procedure, loci were identified with defects of immediate recall. The analysis of 21 sites in each patient as performed by CT scan studies provides excellent detail regarding the loci of vascular lesions. The lesions share a common vascular anatomy and the 21 sites and thus are not entirely statistically independent. "Multiple dissociation" mitigates against this lack of independence. Although one may use a conservative statistic such as Bonferroni's criterion for multiple *t*-tests, the result of this procedure may markedly increase the likelihood of a Type II error [32]. The present study, thus, was intended to achieve maximum sensitivity in order to statistically identify loci potentially related to the impairment of immediate recall.

Immediate memory testing

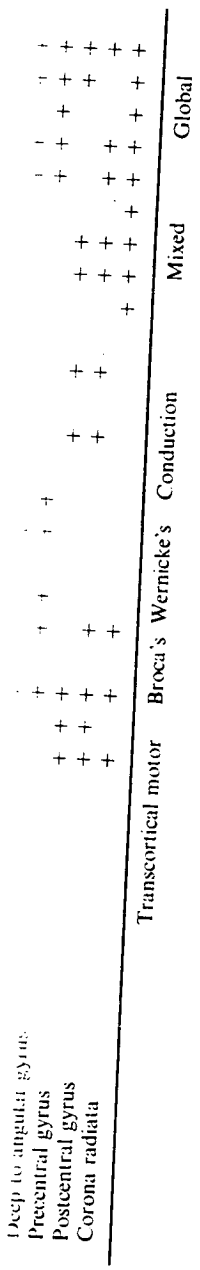
Table 2 shows the sequences of binary digits and tones utilized in the determination of the span of auditory immediate memory. The testing of forward digit span utilized binary sequences; since normal adults have a longer forward span of binary than decimal digits [28] it was anticipated that binary sequences would be a highly sensitive index of recall in aphasic adults. In order to minimize articulatory difficulties, only numbers 1 and 2 were used. A pilot study demonstrated that all the patients upon request could correctly reproduce both the numbers 1 or 2, in either order.

Digits were presented at the timed rate of one per sec. The examiner first read the single digit 1 from column I. Following correct repetition, the subject was presented with two digits from the same column, 1 and 2, and if correctly recalled, three digits, 212. The number of digits was increased until an error was recorded. The second column was then utilized and the subject read the sequence of digits one item shorter than had been presented on the previous trial. If this response was in error, the next shorter sequence was read, and if correct, the next longer was read, following the same procedure, until again an error was recorded. This response-adjusted method, known as a 50% titration schedule, rapidly estimates the span the patient can reliably reproduce 50% of the time. By averaging the highest correct response from the four columns, a single mean was obtained which represented the patient's digit span. Odd–even reliability coefficients were computed by combining the scores with the columns I and III and scores of columns II and IV. The odd–even reliability coefficient for digits was $r = 0.964$.

In administering the binary tonal sequences, two tones, either high (H) or low (L), were sung by examiner at the rate of one tone per sec. The difference between the high and low tones was the musical interval of the fourth as has been utilized in Melodic Intonation Therapy. All patients were tested following the method described for binary

Table 1. CT scan localization in aphasia: results from 28 patients

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28
Frontal zone		+		+	+	+	+																					
Broca's area		+				+		+		+											+	+						+
Head of caudate n.	+							+		+											+	+	+			+		+
Lenticular n.	+		+					+	+	+										+	+	+	+			+		+
Anterior limb of i.c.	+				+			+		+										+	+	+	+			+		+
Genu i.c.	+							+												+	+	+	+	+		+		+
Posterior limb of i.c.	+							+							+						+	+	+					+
External capsule		+				+	+	+	+												+	+	+	+	+	+	+	+
Insula		+		+		+	+	+	+	+	+					+	+	+			+	+				+		+
Temporal pole		+								+	+	+	+	+							+	+	+	+		+	+	+
Mid. temp. gyrus														+				+			+	+		+	+	+	+	+
Heschl's gyrus										+	+	+	+								+	+			+	+	+	+
Wernicke's area										+	+	+	+								+	+			+	+	+	+
Deep to Wernicke's										+	+										+	+			+	+	+	+
Supramarginal gyrus		+				+	+		+	+			+								+	+			+	+	+	+
Deep to supramarginal gyrus		+						+	+	+			+								+	+			+	+	+	+
Angular gyrus								+	+	+			+								+	+			+	+	+	+
Deep to angular gyrus								+	+	+															+	+	+	+
Precentral gyrus					+	+	+								+		+				+	+			+	+	+	+
Postcentral gyrus					+	+	+		+								+				+	+						+
Corona radiata					+		+		+																			
	Transcortical motor			Broca's		Wernicke's			Conduction							Mixed									Global			



...s. Note that the digital and tonal sequences have equivalent patterns. All subjects could sing both the low and ... tones in either order when imitating the examiner. The odd–even reliability coefficient for tonal sequences was ...90.

Table 2. Binary digit span (DS) and binary tone span (TS) sequences used in testing immediate memory

DS	TS
Column I	Column I
1	H
12	HL
212	LHL
1121	HHLH
21221	LHLLH
221212	LLHLHL
1121212	HHLHLHL
Column II	Column II
2	L
21	LH
121	HLH
2212	LLHL
12112	HLHHL
112121	HHLHLH
2212121	LLHLHLH
Column III	Column III
1	H
12	HL
212	LHL
1121	HHLH
21221	LHLLH
221212	LLHLHL
1121212	HHLHLHL
Column IV	Column IV
2	L
21	LH
121	HLH
2212	LLHL
12112	HLHHL
112121	HHLHLH
2212121	LLHLHLH

Statistical analyses were performed for errors made by the patients in their recall of digital and tonal sequences as ...llows:

(1) Primacy errors were defined as failures to correctly recall the first item of any sequence.

(2) Recency errors were defined as failures to correctly recall the last item in a sequence.

(3) Transposition errors were defined as occasions when the patient incorrectly recalled a sequence of two or more ...tems (e.g. 121 as 212 or HLH as LHL).

(4) Isolated errors were defined as those occurring as a single failure in a sequence of three or more items when the ...patient had correctly recalled the first and last items.

Errors in which the patient mispronounced digits (as long as they were intelligible) were scored as correct as were ...errors in which the patient sang out of key (provided that the high and low tones could be distinguished).

RESULTS

Span of immediate recall

The overall mean digit span for the entire sample of aphasic patients was 3.72 ($s = 1.55$) and the range was 1.25–6.75 bits. The transcortical motor patients attained a mean digit span

of 5.43 approximating the norm for controls without neurological disease [9]. The other aphasic groups showed varying degrees of impairment: conduction aphasics (4.40), Broca's (3.83), Wernicke's (3.0), global (2.5) and mixed (2.39). When transcortical motor subjects were excluded, the overall mean digit was 3.10 ($s=0.96$). Thus, the majority of these patients had a span of about 3 ± 1 bits.

Mean tone span for the entire sample was 3.50 ($s=1.25$) with a range of 1.5–6.25 bits. Transcortical motor patients obtained the highest mean tone span (4.55) followed by Wernicke's (3.75), conduction (3.55), global (3.25), Broca's (3.16) and mixed (2.39). Excluding the transcortical motor patients, the mean tone span was 3.14 ($s=0.95$).

Statistical evaluation of differences between digit span and tone span was performed by a two-tailed t-test and failed to achieve statistical significance ($t=0.945$, $P>0.10$). Thus, aphasia-producing lesions of the left hemisphere produce a comparable impairment for the span of binary digital and tonal sequences. These data are depicted in Fig. 1.

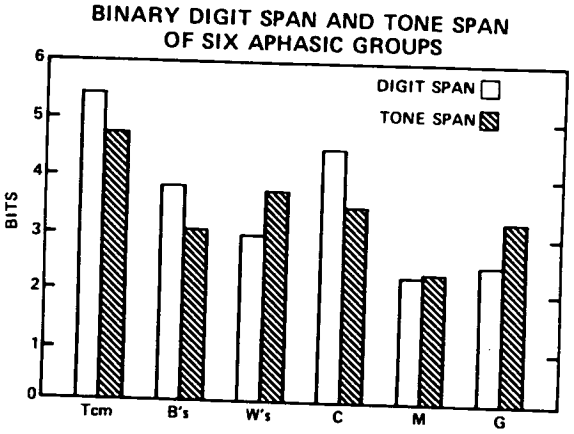


FIG. 1. The span of auditory immediate recall for digits and tones for the six aphasic groups. Note that except for the transcortical motor patients, the others had a span which approximated 3 ± 1 bits of information, whether digits or tones.

A two-factor ANOVA mixed design with repeated measures was used to evaluate the span for digits and tones. The harmonic mean correction was applied for unequal numbers of subjects in each group [46]. This analysis yields three F-ratios, (1) between conditions for digits and tones combined, (2) digits vs tones (trials) and (3) trials $\times$ conditions.

A significant F-ratio was obtained for between groups ($F=12.868$, 27/5, $P<0.004$). The Tukey test was used to evaluate mean comparisons. Transcortical motor patients had significantly longer spans (digits and tones combined) than the Wernicke's ($P<0.05$), mixed ($P<0.01$) and global ($P<0.01$) aphasics. The F for trials was not significant ($F=0.765$, 1/26, $P>0.10$). Thus, the overall comparison of digit span vs tone span for the groups was not significantly different. The trials $\times$ conditions interaction was significant ($F=5.60$, 1/26, $P<0.025$). When only digit span was evaluated, transcortical patients were superior to the Wernicke's ($P<0.05$), mixed ($P<0.01$) and global ($P<0.05$) aphasics. By contrast, for tone span, the transcortical patients were superior only to the mixed ($P<0.05$) aphasics. Thus, more significant comparisons were obtained for digit span.

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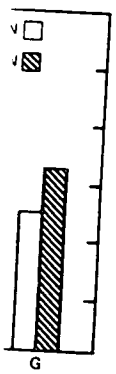

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CT scan localization and immediate recall

Lesion sites related to significantly inferior performance on the digit span test were evaluated as follows: patients with damage to a particular structure, e.g. frontal zone, were sorted into a single group and compared with a second group, the subjects without this lesion. In the same manner, analyses were performed for each site by means of between-group one-tailed t-tests. When homogeneity of variance was not achieved, the d.f. was corrected by the Walsh test [46]. The means and t-values for digit span are presented in Table 3.

Table 3. Lesions identified with impairment of digit span and tone span

	$\bar{X}$ Digits	t	P	$\bar{X}$ Tones	t	P
Frontal zone	4.87	−2.18		4.25	−1.70	
Broca's area	3.22	1.09		3.34	0.43	
Head of caudate n.	3.17	1.31		3.19	0.91	
Lenticular n.	3.43	0.79		3.39	0.41	
Anterior limb of i.c.	3.27	1.35		3.31	0.71	
Genu of internal cap.	3.58	0.25		3.58	−0.16	
Posterior limb of i.c.	3.05	1.94	<0.05	3.23	0.95	
External capsule zone	3.61	0.25		3.28	0.66	
Insula	3.67	0.25		3.47	0.20	
Temporal pole	3.00	1.75	<0.05	3.44	0.18	
Middle temporal gyrus	2.56	3.14	<0.01	2.69	2.62	<0.01
Heschl's gyrus	2.54	4.02	<0.001	2.91	2.18	<0.05
Wernicke's area	2.50	3.78	<0.001	3.05	1.48	
Deep to Wernicke's area	2.55	4.02	<0.001	2.89	2.48	<0.01
Supramarginal gyrus	3.27	1.59		3.14	1.59	
Deep to supramarginal gyrus	2.96	2.91	<0.01	3.07	1.95	<0.05
Angular gyrus	2.72	2.57	<0.01	2.92	1.79	<0.05
Deep to angular gyrus	2.70	2.93	<0.01	3.00	1.66	
Precentral gyrus	4.11	−0.90		3.64	−0.37	
Postcentral gyrus	3.50	−0.69		3.25	1.023	
Corona radiata	2.90	2.93	<0.01	3.13	1.51	

The structures identified with significantly diminished digit span included the posterior limb of the internal capsule, and perisylvian structures including the temporal pole, middle temporal gyrus, Heschl's gyrus, cortex of Wernicke's area, white matter deep to Wernicke's area, white matter deep to the supramarginal gyrus (but not the cortex of the supramarginal gyrus), and the cortex and underlying white matter of the angular gyrus. Additionally, the corona radiata was identified with diminished digit span. The lowest digit span mean for any site was for patients with damage to Wernicke's area (2.50); the highest was for patients with damage to the frontal zone (4.80). Figure 2 displays a lateral view of the left hemisphere drawn with two horizontal cross-sections each 4 mm thick and 8 mm apart, [after 12]; the cross-sections are shown below in the plane of CT for slices BW (left) and SM (right). The loci identified with reduced digit span are shaded according to their probability values (i.e. $P<0.05$, 0.01 and 0.001).

The lesions identified with significantly lower tone spans are presented in Fig. 3 and include only perisylvian sites: the middle temporal gyrus, Heschl's gyrus, white matter deep to Wernicke's area and white matter deep to the supramarginal gyrus. Note that these structures had also been identified with poor digit span but that fewer sites overall were identified for the tonal sequences. The lowest mean tone span was for patients with damage

of the frontal zone (4.25). Thus, frontal damage was identified with both the highest digit and tone spans.

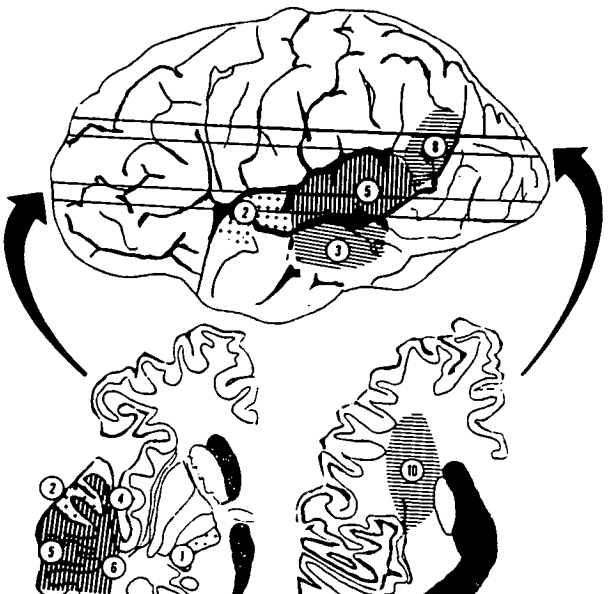

FIG. 2. The lateral aspect of the brain and two horizontal cross-sections below at the levels indicated by the parallel lines. Lesions significantly related to digit span are represented graphically by their associated probability values (plus signs, $P<0.05$; horizontal lines, $P<0.01$; and vertical lines, $P<0.001$). The sites related to inferior digit span include: (1) the posterior limb of the internal capsule, (2) the temporal pole, (3) the middle temporal gyrus, (4) Heschl's gyrus, (5) the cortex of Wernicke's area, (6) white matter deep to the supramarginal gyrus, (8) the cortex of the angular gyrus, (9) white matter deep to the angular gyrus and (10) the corona radiata.

Error analysis by aphasia classification

Table 4 summarizes the percentage of errors made by the six aphasia groups for digit and tone spans. The cumulative percentages exceed 100% because more than one error classification could be recorded on a single trial.

The transcortical motor aphasics (TCM) made few transposition errors (4% for both digits and tones) but the most isolated errors (21% for digits and 29% for tones) of any other group. Both Broca's (B) and Wernicke's (W) aphasics made a higher number of recency errors than did transcortical motor and conduction patients. The number of transposition errors made by various patients with a mean of about three elements far exceeded the a-priori probability ($P=0.125$). For example, 58% of all incorrect sequences of the Wernicke's aphasics included transposition errors, overall more such errors than any other group. Moreover, Wernicke's made substantially more transposition errors than did

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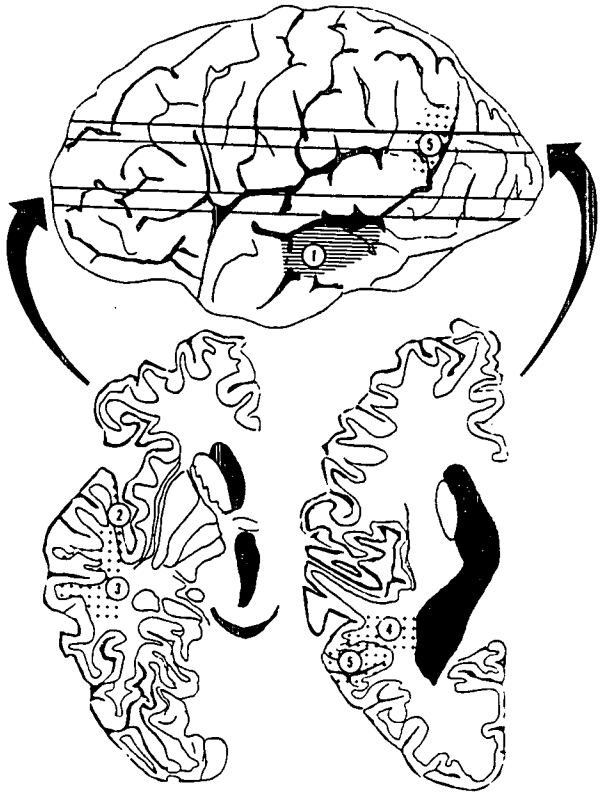

FIG. 3. Lesions significantly related to impaired tone span. The loci include: (1) middle temporal gyrus, (2) Heschl's gyrus, (3) white matter deep to Wernicke's area, (4) white matter deep to the supramarginal gyrus and (5) the cortex of the angular gyrus.

Table 4. Percentage of errors made by the six aphasia groups

Aphasia syndrome	Primacy		Recency		Transposition		Isolated	
	DS	TS	DS	TS	DS	TS	DS	TS
T	29	32	50	50	4	4	21	29
B	58	42	92	67	25	8	0	17
W	83	91	91	67	58	58	0	8
C	20	30	40	60	0	20	10	10
M	57	71	86	79	25	61	0	0
G	91	75	92	92	75	33	0	0

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Error analysis by lesion sites

Table 5 presents structures significantly identified with the highest likelihood of primacy, recency, transposition and isolated errors for digits and tones. In order to save space, the reader is referred to this table for indication of the probability values identified with impairment following damage to each locus.

Table 5. Lesions identified with errors of auditory immediate recall

	Primacy DS		Primacy TS		Recency DS		Recency TS	
Frontal zone	−0.36		−0.25		−1.56		−0.49	
Broca's area	2.14	<0.05	1.68		0.49		1.09	
Head of caudate n.	1.67		1.28		1.61		1.20	
Lenticular n.	1.59		0.36		1.30		0.20	
Anterior limb of i.c.	1.57		1.08		2.09	<0.05	1.11	
Genu of internal cap.	0.73		−0.25		0.79		0.03	
Posterior limb of i.c.	0.92		0.36		2.47	<0.05	0.99	
External capsule zone	2.47	<0.01	0.45		1.24		0.36	
Insula	1.62		0.11		1.07		0.31	
Temporal pole	2.47	<0.01	1.88	<0.05	1.61		0.36	
Middle temporal gyrus	1.67		1.28		2.45	<0.01	2.14	<0.05
Heschl's gyrus	2.77	<0.01	2.01	<0.05	2.93	<0.01	2.35	<0.05
Wernicke's area	3.34	<0.01	1.78	<0.01	2.92	<0.01	1.75	<0.05
Deep to Wernicke's area	1.59		1.41		2.93	<0.01	2.35	<0.05
Supramarginal gyrus	1.90	<0.05	0.49		1.46		1.36	
Deep to supramarginal gyrus	1.55		0.49		1.82	<0.05	1.36	
Angular gyrus	1.67		1.28		2.45	<0.01	2.14	<0.05
Deep to angular gyrus	2.40	<0.05	1.78	<0.05	2.92	<0.01	2.24	<0.05
Precentral gyrus	1.31		−0.33		−0.81		−0.44	
Postcentral gyrus	1.55		0.28		0.41		0.83	
Corona radiata	2.69	<0.01	1.58		2.56	<0.01	2.12	<0.05

	Transposition DS		Transposition TS		Isolated DS		Isolated TS	
Frontal zone	−1.01		−1.53		2.08	<0.05	1.84	<0.05
Broca's area	1.09		0.08		0.55		0.14	
Head of caudate n.	1.06		1.26		−1.19		−0.56	
Lenticular n.	1.04		0.47		0.82		−0.50	
Anterior limb of i.c.	1.70		0.69		−1.02		−1.18	
Genu of internal cap.	0.07		0.59		−0.60		0.11	
Posterior limb of i.c.	1.36		0.77		−1.59		−0.97	
External capsule zone	1.77	<0.05	−0.58		−1.19		0.08	
Insula	2.22	<0.05	0.33		−1.62		−1.11	
Temporal pole	3.58	<0.001	0.63		−1.19		−1.05	
Middle temporal gyrus	1.06		1.96	<0.05	−2.06		−2.14	
Heschl's gyrus	2.47	<0.01	2.07	<0.05	−2.48		−1.99	
Wernicke's area	2.96	<0.01	1.16		−2.26		−1.78	
Deep to Wernicke's area	1.71	<0.05	2.07	<0.05	−2.48		−2.59	
Supramarginal gyrus	1.70		−0.85		−1.44		0.22	
Deep to supramarginal gyrus	2.06	<0.05	−0.28		−1.44		−0.67	
Angular gyrus	1.77	<0.05	−0.58		−2.06		−0.56	
Deep to angular gyrus	2.96	<0.01	−0.33		−2.26		−0.76	
Precentral gyrus	0.10		−0.58		1.08		0.39	
Postcentral gyrus	0.73		−1.09		0.19		−0.46	
Corona radiata	2.05	<0.05	0.63		−1.22		−1.91	

highest likelihood of primacy errors. In order to save space, the probability values identified with

mediate recall

Recency DS		Recency TS	
-1.56		- 0.49	
0.49		1.09	
1.61		1.20	
1.30		0.20	
2.09	<0.05	1.11	
0.79		0.03	
2.47	<0.05	0.99	
1.24		0.36	
1.07		0.31	
1.61		0.36	
2.45	<0.01	2.14	<0.05
2.93	<0.01	2.35	<0.05
2.92	<0.01	1.75	<0.05
2.93	<0.01	2.35	<0.05
1.46		1.36	
1.82	<0.05	1.36	
2.45	<0.01	2.14	<0.05
2.92	<0.01	2.24	<0.05
0.81		-0.44	
0.41		0.83	
2.56	<0.01	2.12	<0.05

Isolated DS		Isolated TS	
2.08	<0.05	1.84	<0.05
0.55		0.14	
1.19		-0.56	
0.82		-0.50	
1.02		-1.18	
0.60		0.11	
1.59		-0.97	
1.19		0.08	
1.62		-1.11	
1.19		-1.05	
2.06		-2.14	
2.48		-1.99	
2.26		-1.78	
2.48		-2.59	
1.44		0.22	
1.44		-0.67	
2.06		-0.56	
2.26		-0.76	
1.08		0.39	
0.19		-0.46	
1.22		-1.91	

Primacy errors for digits had the highest probability of occurrence among patients with damage to Broca's area, zone of the external capsule, temporal pole, Heschl's gyrus, Wernicke's area, the cortex of the supramarginal gyrus, white matter deep to the angular gyrus and the corona radiata.

Primacy errors for tonal sequences were statistically identified with damage to the temporal pole, Heschl's gyrus, Wernicke's area and white matter deep to the angular gyrus. Note that primacy errors for both digital and tonal sequences involved anterior perisylvian structures such as the temporal pole, middle perisylvian structures including the region of the superior temporal gyrus and to a lesser extent posterior perisylvian loci such as the region of the inferior parietal lobe.

Recency errors for digits were identified with infarction of the anterior and posterior limbs of the internal capsule, the middle temporal and Heschl's gyri, Wernicke's area and white matter underlying both Wernicke's area and the supramarginal gyri and the gray and white matter of the angular gyrus; the corona radiata was also identified.

Unlike primacy errors, recency errors for both digits and tones failed to show a significant relationship to lesions of anterior perisylvian structures including Broca's area, zone of the external capsule or the temporal pole. By contrast, recency errors were more closely associated with damage to the inferior parietal lobule including the cortex of the angular gyrus.

Transposition errors for digits were identified with lesions of the zone of the external capsule, temporal pole, Heschl's gyrus, Wernicke's area, deep to Wernicke's area, the supramarginal and angular gyri, and the corona radiata. Transposition errors for tones were identified with damage to the middle temporal and Heschl's gyri and deep to Wernicke's area.

Note that transposition errors for digits and tones were identified with the region of the superior temporal gyrus. This finding is consistent with the observation that the Wernicke's aphasics made more transposition errors than any other group.

Isolated errors were related only to lesions of the frontal zone for both digits and tones. Although the span was the longest for the transcortical motor patients, they tended to forget single elements in the sequence more frequently than the other groups. By contrast, most patients who could recall only about three elements tended to have a greater fragmentation of recall including errors in which every item was erroneously recalled (i.e., transposition errors).

DISCUSSION

Vascular lesions of the left hemisphere involving the territory of the middle cerebral artery and particularly the region around Wernicke's area limit the span of auditory recall to about 3 ± 1 bits. Digit span was most deficient among patients who had lesions of Wernicke's area and tone span was most impaired in patients with lesions of the middle temporal gyrus. Frontal zone lesions were identified with the highest spans.

The frequency of errors for each classification were similar for both digits and tones, i.e. patients were as likely to make transposition errors for tones as they were for digits. Primacy errors were apparently more likely with anterior perisylvian damage such as of the temporal pole than were recency errors. By contrast, recency errors were more often identified with damage to parts of the inferior parietal lobule, such as the cortex of the angular gyrus. Transposition errors for digits were related to damage of the majority of perisylvian sites;

transposition errors for tones were most consistently identified with the region of the superior temporal gyrus including white matter deep to Wernicke's area, Heschl's and the middle temporal gyri. Thus, transposition errors included overlapping loci for both digits and tones in the region of the superior temporal gyrus. Isolated errors occurred most frequently among patients with lesions of the frontal lobe who had transcortical motor aphasia and intact repetition of auditory sequences.

Although most patients with aphasia can sing familiar songs more easily than they are able to utilize verbal language, the results of this study demonstrate that for novel auditory sequences, immediate recall is equally impaired for both digits and tones. These findings suggest that the ability of aphasic adults to sing familiar melodies may engage different structures (i.e. including the right hemisphere) than required to sing novel tonal sequences.

The following discussion is organized according to the listing of anatomical sites in Tables 1, 3 and 5.

(a) The results of the study are consistent with findings that lesions producing transcortical motor aphasia, i.e. anterior to the precentral gyrus and superior to Broca's area, do not significantly impair auditory immediate recall [6]. Although patients with Broca's aphasia have a deficit of repetition, the impairment was not statistically identified with area 44 Broca's area. Moreover, a failure to produce an enduring Broca's aphasia following damage to Broca's area has been reviewed [30, 31, 35].

(b) DAMASIO and DAMASIO [11] proposed that lesions of the insula and particularly of the fibers of the extreme capsule produced conduction aphasia. Various connections from the auditory cortex to the pre-motor frontal lobe reportedly travel in the extreme capsule underneath the insular cortex. In the present study, the insula was not statistically related to a defect in repetition.

(c) Temporal polar lesions produced a high probability of primacy errors for both digits and tones. If primacy errors are produced by a consolidation deficit [45], these findings would be consistent with amnestic impairments resulting from anterior temporal lobectomy [29] and electrical stimulation of the temporal pole [14]. The primacy errors may be potentially attributed to interruption of fibers from the anterior temporal pole and via the entorhinal cortex, to the hippocampus and amygdala [39].

(d) A severe repetition deficit occurred for digits and tones among patients with damage to Heschl's and the middle temporal gyri and white matter deep to Wernicke's area. Further, the lowest mean digit span (2.5 bits) was encountered for lesions of Wernicke's area. Although Wernicke's aphasia is regarded as an impairment which includes impoverished comprehension of speech, it is hypothesized that a failure to comprehend could arise solely on the basis of deficient immediate recall. HEILMAN and SCHOLES [22] reported a greater frequency of syntactical errors among Wernicke's aphasics than for Broca's and conduction aphasics. These syntactical errors resemble transposition errors and thus support the view that auditory comprehension is diminished by a distortion of immediate recall. Further, it is possible that the mechanisms by which some paraphasic errors are made could be related to errors of transposition.

(e) All the loci of the inferior parietal lobule were identified with significantly diminished immediate auditory recall except for the cortex of the supramarginal gyrus. Damage to the cortex of the left supramarginal gyrus in man is usually identified with somesthetic rather than auditory deficits [20]. By contrast, GESCHWIND [15] reviewed findings supporting the view that lesions involving white matter underlying the supramarginal gyrus, and interrupting the arcuate fasciculus, produce conduction aphasia. Both digit and tone spans were significantly

duced by lesions deep to the cortex of the supramarginal and angular gyri. Lesions of this general region produce profound repetition deficits [27, 45] and a high frequency of recency errors in the recall of serially presented lists [37, 44].

In summary, the findings of this study suggest that various neural mechanisms subserve auditory immediate memory as follows: (1) the determination of *order* is dependent, at least in part, upon Wernicke's area and surrounding tissue, as evidenced by the preponderance of transposition errors encountered for this region for both digits and tones. (2) The immediate *storage* of information in the sequence appears particularly related to the integrity of white matter deep to the supramarginal gyrus and to the gray and white matter of angular gyrus. This view is supported by the high frequency of recency errors made by patients with lesions of the inferior parietal lobule. (3) The translation of the sequential information into the limbic system and diencephalon for *consolidation* is presumably related to the integrity of the temporal pole and perhaps adjacent prerolandic structures; lesions of the temporal pole were identified with a high probability of primacy errors.

Isolated errors, in contrast to the other three error categories, are conceived as brief lapses of attention rather than the more drastic degradation of immediate recall resulting from perisylvian damage.

In conclusion, the present report is consistent with previously published findings that damage to posterior perisylvian structures of the left hemisphere which produce aphasia result in a profound reduction in the span of auditory immediate recall for verbal or tonal sequences. Further, this study provides additional evidence to detail patterns of lesions producing primacy and recency errors and documents the preponderance of transposition errors. Although no unique sites were identified for tones that had not been associated with digits, it is not yet possible to assert that the digital and tonal sequences utilize a common mechanism. Further, caution should be exercised in attempting to generalize statistical findings for the entire group to individual patients with specified perisylvian lesions or to patients without aphasic disorders.

The impairment of recall limited to 3 ± 1 auditory sequences cannot be directly attributed to a generalized disturbance of attention, a failure to comprehend the test instructions or to deficient articulation; note that all patients could reliably reproduce one or two digits or tones upon request. The degree to which the impairment of auditory immediate recall may underlie various facets of auditory–verbal comprehension is under investigation.

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Résumé

Des lésions vasculaires dans le territoire de l'artère cérébrale moyenne gauche ont été identifiées chez vingt huit aphasiques adultes au moyen de la tomographie axiale transverse. Lorsque la lésion impliquait une partie du gyrus de Heschl, du gyrus temporal moyen ou supérieur, ou du lobule pariétal inférieur, la mémoire immédiate pour des séquences binaires de chiffres ou de sons était réduite à 3 ± bits. Selon son emplacement, la lésion pouvait donner lieu à des erreurs sur le premier ou le dernier item de la séquence, à des erreurs isolées. Dans la discussion sont passés en revue les différents mécanismes nerveux responsables de la mémoire auditive immédiate.

Zusammenfassung

Vaskuläre Läsionen im Versorgungsgebiet der linken A. cerebri media wurden für 28 erwachsene Aphasiker durch zerebrale Computertomographie identifiziert. Wenn die Schädigung Teile von Heschls Gyrus, den Gyrus temporalis medius und superior oder den Lobulus parietalis inferior einschloß, war die unmittelbare Merkspanne für binäre Sequenzen von Zahlen oder Tönen 3 ± 1 bit. Läsionen verschiedener Lokalisation wurden durch Fehler in bezug auf dichten oder längeren zeitlichen Abstand, Verwechslungen oder isolierte Fehler identifiziert. In der Diskussion werden verschiedene neuronale Mechanismen diskutiert, die dem unmittelbaren akustischen Erinnern zugrunde liegen können.