

Paper II

LEFT HEMISPHERE DYSFUNCTION AFFECTS DICHOTIC LISTENING IN PATIENTS WITH TEMPORAL LOBE EPILEPSY

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This study evaluated the relative effect of left hemisphere dysfunction and side of seizure onset on dichotic listening performance in patients with temporal lobe epilepsy and left hemisphere speech dominance. Seventeen patients were divided into groups based on side of seizure onset and based on scores on a composite measure revealing left hemisphere

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dysfunction. The group with left hemisphere dysfunction had more correct responses from the left ear, and a left ear advantage, on dichotic listening. The group with normal left hemisphere function showed the expected right ear advantage. Side of seizure onset did not affect dichotic listening performance significantly.

Keywords dichotic listening, lateralization, neuropsychology, temporal lobe epilepsy

In the present study, the main hypothesis was that dichotic listening (DL) performance with verbal stimuli in patients with temporal lobe epilepsy (TLE) may be principally dependent upon the integrity of the left cerebral hemisphere, rather than the side of the epileptic focus. This would be in keeping with the classical "lesion hypothesis," that a left-sided lesion causes disruption of the normal right ear advantage (REA) in DL (Kimura, 1967).

Diagnosis of lesional versus nonlesional epilepsies are often based on radiological evidence of structural lesions. However, lack of normal REA has been found in functional language deficits such as dyslexia (Bakker & Kappers, 1988; Cohen, Hynd, & Hugdahl, 1992; Hugdahl, Helland, Faerevaag, Lyssand, & Asbjørnsen, 1996), that not necessarily imply corresponding structural changes. Therefore, neuropsychological status may be an independent and possibly more relevant criterion of hemispheric integrity than radiologic findings.

DL consists of simultaneous bilateral presentation of auditory stimuli. The procedure is widely used in studies of functional and structural brain laterality (Bryden, 1988; Hugdahl, 1995). Patients with left hemisphere speech dominance demonstrated by the Intra-carotid Amobarbital Test (IAT; Wada & Rasmussen, 1960) in general perceive verbal stimuli to the right ear more accurately than those to the left ear. When forced to choose, they report verbal right-ear stimuli more often. This phenomenon is called the REA, and is frequently found in groups of normal right-handers (Hugdahl, 1995). Similarly, patients with right hemisphere speech dominance in general show a left ear advantage (LEA) (Kimura, 1967; Hugdahl, Carlsson, Uvebrant, & Lundervold, 1997). With bilateral language representation, no ear preference or a less pronounced REA than normal is

usually found (Studdert-Kennedy & Shankweiler, 1970; Strauss, Gaddes, & Wada, 1987; Strauss, 1988; Zatorre, 1989). A unilateral epileptogenic lesion may impair DL performance for stimuli to the ear contralateral to the lesion (Oxbury & Oxbury, 1969; Berlin, Lowe-Bell, Jannetta, & Kline, 1972; McIntyre, Pritchard, & Lombroso, 1976; Mazzucchi & Parma, 1978; Efron & Crandall, 1983; Mazzucchi, Visintini, Magnani, Cattelani, & Parma, 1985; Lee et al., 1994; Grote, Pierre-Louis, Smith, Roberts, & Varney, 1995). However, it is unclear how the epileptic focus in itself affects DL performance. Two studies (Mazzucchi & Parma, 1978; Mazzucchi et al., 1985) showed an increase in REA in patients with left-sided epileptogenic foci without corresponding morphological lesions. This was interpreted as a possible facilitatory effect of the epileptogenic focus. Speech dominance was not controlled for in those studies. Another study (Lee et al., 1994) controlled for speech dominance with IAT, found a significant decrease in REA in patients with left hemisphere speech dominance, left seizure focus, and no evidence of structural lesions. However, the results were not considered robust enough to permit prediction of seizure focus in the individual case. None of these studies (Mazzucchi & Parma, 1978; Mazzucchi et al., 1985; Lee et al., 1994) explicitly controlled for general hemisphere function.

Most studies on DL performance in patients with epilepsy have used dichotic words or digits presented in sequence or with fused words, whereas studies in normal groups most often have used consonant-vowel (CV; Bryden, 1988; Hugdahl, 1995) or consonant-vowel-consonant (Studdert-Kennedy & Shankweiler, 1970) stimuli. Different results between studies may thus also be due to the use of different stimuli. The temporal lobes are particularly involved in the processing of phonetic stimuli, like CV-syllables (Binder, Frost, Hammeke, Rao, & Cox, 1996; Jäncke, Wüstenberg, Scheih, & Heinze, 2002). Increasing the semantic complexity of the stimuli may involve other brain regions to a larger degree, which may confound the findings. One study, using CV-syllables in children and adolescents with epilepsy tested with IAT (Hugdahl et al., 1997), clearly showed REA with left hemisphere speech and LEA with right hemisphere speech. No performance differences relative to side of epileptic focus, nor

any significant effect of surgery, was found. However, the patients had various intrahemispheric foci, with only 3 of 13 patients showing exclusively temporal focus. Moreover, children and adults may differ in DL performance (Hugdahl, 1992). To evaluate hemispheric functional integrity, it is probably important to use neuropsychological measures with documented sensitivity to effects of structural brain lesions. The Halstead-Reitan Battery (HRB) has repeatedly been shown to be sensitive to such lesions (Kløve, 1974; Reitan & Wolfson, 1993).

Based on tests from this battery, the Left Neuropsychological Deficit Scale (LNDS; Reitan & Wolfson, 1993) is a composite measure incorporating motor and sensory-perceptual dysfunction in addition to language-related deficits. This may emphasize the impact of structural brain lesions in contrast to other sources of language difficulties. The original study (Reitan & Wolfson, 1993) compared groups with left, right, and generalized brain damage of various etiology in 169 patients. A double dissociation was found between LNDS and a similar measure of right hemisphere integrity (Right Neuropsychological Deficit Scale/RNDS; Reitan & Wolfson, 1993). Patients with focal left hemisphere brain damage showed elevated LNDS and normal RNDS, and patients with focal right hemisphere brain damage showed elevated RNDS and normal LNDS. Patients with generalized brain damage showed elevations on both scales. A control group of 41 subjects without brain damage showed normal results on both scales. To our knowledge, no independent cross-validation of the LNDS has been performed. However, the original validation data quite convincingly demonstrated sensitivity of this scale to left hemisphere brain damage. In the present study, LNDS was used as criterion for evaluating effect of "lesion," or disrupted left hemisphere cerebral integrity, in contrast to effect of epileptic focus per se, in patients with focal TLE and left hemisphere speech dominance. Our main hypothesis was that, regardless of lateralization of epileptic focus, TLE patients with left hemisphere dysfunction would lack normal REA on DL, whereas patients with normal left hemisphere function would show normal REA on DL. Thus, in contrast to previous studies, we hypothesized that lateralization of epileptic focus would be relatively less critical for the DL results than lateralization of general hemisphere function.

METHODS

Study Sample

Seventeen patients (10 males and 7 females) with a definite diagnosis of TLE were included. Diagnoses were based on clinical evaluation, video-EEG-monitoring, and neuroradiological investigations, including MRI scans according to a specific protocol focusing study of temporal lobe structure. Seven patients had left-sided temporal focus of seizure onset, 10 had right-sided temporal focus. All subjects were patients in the Department of Neurology, Haukeland University Hospital, in the period 1995–2001. Mean age at testing was 33.7 years (range 18–48, SD = 9.0). All patients were evaluated for surgery because of focal TLE, and had left hemisphere speech dominance established by IAT. The sample represents all patients with TLE and left-sided speech dominance being evaluated with IAT during that time period. Three patients who underwent the IAT were excluded because they showed right hemisphere speech dominance. Two patients were excluded because speech dominance could not be reliably determined, in one case because of a lack of drug effect, in the other case because an emotional reaction led to inadequate cooperation. Five patients (no. 3, 7, 8, 12, and 13 of Table 1) showed some right hemisphere language representation, mainly by correct execution of movement on command, or as dysphasic speech. This group of patients did not show any significant deviations from the rest of the patients in any of the neuropsychological or DL data. However, one of the patients (no. 13) did show a more significant right hemisphere speech representation, combined with some naming difficulties in the left hemisphere on the IAT. This patient also showed clinical naming difficulties. Left hemisphere speech dominance was, however, indicated by ability to finish counting immediately after injection, which was not accomplished on testing of the right hemisphere. This patient had a LNDS < 6 and a left ear preference on DL. Details of diagnosis, medication, and seizure variables for each patient are given in Table 1.

Four patients (no. 3, 11, 14, and 17) had a generalized tonic clonic seizure (GTC) the last month before testing. Patient 7 had three episodes of convulsive status epilepticus, and patients 2 and 3

TABLE 1. Demographic and seizure variables of patients in the study

No.	Sex	Age at onset	Age at test	Focus R/L	Etiology	AED N(drugs)	Seizures	CPS/ month last year
1	F	23	44	R	Venous cavernoma	3 (CBZ, LTG, VGB)	CPS>GTC	3
2*	F	23	47	R	Early febrile seizure?	1 (CLB)	CPS>GTC	20
3	F	11	18	R	Early febrile seizure?	2 (CBZ,LTG)	CPS>GTC	4-6
4*	F	20	21	R	Developmental disorder	1 (CBZ)	CPS>GTC	<1
5*	M	17	37	R	Cerebral trauma	3 (CBZ, LTG, VPA)	CPS>GTC	2
6*	M	4	48	R		1 (CBZ)	CPS>GTC	15
7	M	1.5	36	R		3 (CBZ, PB, VGB)	CPS>GTC	>2
8*	M	33	34	R	Benign tumor? Gliosis?	2 (CBZ, TPM)	CPS	7-8
9*	M	0.5	33	R	Early febrile seizure?	1 (CBZ)	CPS>GTC	>5
10	M	12	29	R	Cerebral trauma	2 (CBZ, LTG)	CPS	6
11*	F	29	36	L	Dermoid cyst in temporal pole	1 (CBZ)	CPS>GTC	<1
12	F	10	24	L		2 (CBZ,TPM)	CPS>GTC	4-10
13	F	5	20	L		3 (CBZ, LTG, TPM)	CPS>GTC	2-5
14	M	15	32	L	Benign tumor; DNET in temporal pole	1 (OXC)	CPS>GTC	1
15	M	16.5	41	L		3 (CBZ, LTG, VGB)	CPS>GTC	>20
16*	M	35.5	38	L		2 (PHT, LTG)	CPS	4-6
17	M	0.5	35	L		2 (CBZ,TPM)	CPS>GTC	10-15

*Left neuropsychological deficit scale ≥ 6 .

Only strongly suspected or confirmed etiologies are listed.

Abbreviations: F = female, M = male, R = right temporal, L = left temporal, CPS = complex partial seizures, GTC = secondary generalized tonic clonic seizures, AED = antiepileptic drugs, CBZ = carbamazepine, LTG = lamotrigine, VGB = vigabatrin, VPA = sodium valproate, PB = phenobarbital, TPM = topiramate, OXC = oxcarbazepine, CLB = clobazam, PHT = phenytoin, DNET = dyssembryoplastic neuroepithelial tumor.

had likely single convulsive status epilepticus as febrile seizures in early childhood. Focus lateralization have been verified by later operation, with successful outcome in terms of seizure control, in 12 patients. For various reasons, operations have not been performed on patients no. 4, 8, 9, 15, and 17. All patients were right-handed, judged from the lateral dominance examination of the HRB (Reitan & Wolfson, 1993). No subjects had clinically evident hearing disorders, nor seizures with auditory hallucinations. Perception of finger rubbing (Auditory Imperception Test; Reitan & Wolfson, 1993) was normal for all patients. Mean full scale IQ (Wechsler Adult Intelligence Scale/WAIS; Wechsler, 1955) was 97.1 (range 75–120, SD = 12.8). Mean General Memory Index (GMI, Wechsler Memory Scale–Revised/WMS-R; Wechsler, 1987) was 87.1 (range 58–111, SD = 14.4). On the HRB, mean Impairment Index (Matthews, Shaw, & Kløve, 1966) was 0.45 (range 0.1–1.0, SD = 0.29), and mean score on the General Neuropsychological Deficit Scale (GNDS; Reitan & Wolfson, 1993) was 28.9 (range 13–47, SD = 10.6).

Intracarotid Amobarbital Test (IAT)

The procedure used for this test was based on the procedure used at the Montreal Neurological Institute (Jones-Gotman, 1987). All tests were performed at the Department of Radiology, Haukeland University Hospital, by the first author in collaboration with one of two experienced neuroradiologists. An EEG was always recorded. A standard dose of 125 mg sodium amobarbital was injected via a catheter in the internal carotid artery. In the first patient tested the dose was 87.5 mg. All tests were videotaped for documentation. Ability to count, name objects and repeat sentences and digit sequences were used as criteria for defining speech dominance. In addition, ability to follow verbal instructions and incidences of spontaneous speech were evaluated.

Neuropsychological Testing

All patients were administered a standardized test battery by Dr. Gramstad or by trained test personnel under his supervision. It included DL, WAIS, WMS-R, HRB, subtests of the Kløve-Matthews

Motor Steadiness Battery (Reitan & Davison, 1974), Wisconsin Card Sorting Test (Heaton, Chelune, Talley, Kay, & Curtiss, 1993), Stroop Test (Golden, 1978), and a facial recognition task (Hugdahl, Iversen, Ness, & Flaten, 1989). All patients answered several questionnaires, including Minnesota Multiphasic Personality Inventory-2 (Butcher, Dahlstrom, Graham, Tellegen, & Kaemmer, 1989; Ellertsen, Havik, & Skavhellen, 1996) and Washington Psychosocial Seizure Inventory (Dodrill, Batzel, Queisser, & Temkin, 1980), for differential diagnostic, clinical, and research purposes (Engelsen, Karlsen, Gramstad, Lillebø, & Aarli, 2000; Gramstad, Iversen, & Engelsen, 2001). Only results of WAIS, WMS-R, and HRB will be analyzed here, in relation to DL performance.

The HRB administered deviated somewhat from the standard instructions by Reitan and Wolfson (1993). A Norwegian version of Halstead-Wepman Aphasia Screening Test (Halstead & Wepman, 1949) was used instead of Reitan-Indiana Aphasia Screening Test (Reitan, 1984; Reitan & Wolfson, 1993). The two tests are reasonably similar in structure and purpose, and scoring rules for the Reitan-Indiana test were closely followed in scoring the performance and calculating the Neuropsychological Deficit scales. Because of translation difficulties, the Speech Perception Test was omitted. GNDS, LNDS, and RNDS were calculated. GNDS is originally based on 42 different HRB test items. Because Speech Perception Test was omitted, GNDS was based on 41 items in this study. Each item was rated on a four-point scale or given a designated score indicating degree of deviation from perfectly normal. More deviant performances gave higher scores. LNDS (21 items) and RNDS (13 items) evaluate hemisphere-specific deficits. Signs of verbal dysfunction, defined as a relative deficit on verbal compared to nonverbal WAIS IQ or signs of dysphasia on the aphasia screening test, were scored on LNDS. In addition, relative deficit of the right compared to the left body half on six sensory-perceptual measures (tactile, auditory and visual imperception, tactile finger localization, fingertip number writing, and tactile forms test), and three motor measures (tactual performance test, finger tapping, and grip strength) was scored on LNDS. Similarly, a result was scored on RNDS if it revealed nonverbal dysfunction or a relative deficit of the left body half. Scoring details and validation data are given in Reitan and Wolfson (1993).

DL Test and Procedure

The subjects were seated in a quiet room. DL stimuli were presented via headphones. The experimenter had an extra set of headphones in order to overhear the tape output. Oral answers were continuously marked on a special scoring sheet. Stimulus materials and test and scoring procedures were taken from the guidelines of Hugdahl (1995). Dichotic stimuli consisted of the six stop consonants paired with the vowel /a/ to form six consonant-vowel syllables (ba, da, ga, pa, ta, ka). The syllables were paired with each other in all possible combinations, thus giving 36 different syllable pairs (including the 6 homonymic pairs). The dichotic tape was prepared on a computer (Digital Corporation PDP 11/45), and digital-analog converters and with a digital-analog multiplexer. Each CV-syllable was approximately 450 ms in duration, and intertrial interval was approximately 4 s. The temporal alignment between channels was set at the energy-release in both the consonant and the vowel segments of the CV-syllable. Maximum-onset difference between the channels was 0.5 ms due to the digital-analog multiplexer resolution and the sampling frequency (minimum, 10 kHz). The syllables were originally recorded from the computer onto a reel-to-reel tape recorder (NAGRA IV), and copied onto a chrome dioxide cassette and played to subjects from a standard cassette player at about 80 dB. The 36 dichotic pairs were recorded three times on the tape with three different randomizations, for each attentional instruction.

There were three different attentional conditions, with different instructions on how to focus attention. In the first condition, the patients were simply told they would be presented with a list of CV-syllables. Thus no specific instruction regarding attention was presented. This condition was called the non-forced (NF) attention condition. The subject's task was to answer with the syllable he or she heard on each trial. Thus, one response for each trial was emphasized, even though subjects might have perceived both syllables on some trials. This was done to eliminate the risk of artificial change in ear-advantages due to comparison of double-responses against single response trials. During the forced-right (FR) attentional instruction, the patients were told to pay close attention to only the right ear syllables, and only to report what they heard in the right

ear. During the forced-left (FL) attentional instruction, the patients were told to pay close attention to the left ear syllables, and to report only what they heard in the left ear. The order of presentation was with the NF condition first, and the FR and FL conditions incompletely counterbalanced. Only the results from the NF condition are analyzed in this study.

Data were scored for each subject as the frequency of correctly recalled syllables for the right and left ear input. To facilitate comparisons with other DL studies, the raw scores were converted to percentage scores. A laterality index score was calculated according to the formula: $[(\text{right ear} - \text{left ear}) / (\text{right ear} + \text{left ear})] \times 100$, where right ear and left ear represent the number of correct right ear and left ear scores, respectively. The data analysis included group comparisons, with *t*-tests for comparison of group means, and a regression analysis to sort out relative sources of explained variance on DL data. Results of subgroups without further statistical analyses are also presented. All the statistics were calculated using SPSS 10.0 for Windows NT 4.

RESULTS

As shown in Table 2, there were no significant differences between the left and right focus groups on IQ, memory indexes, or neuropsychological summary measures. The group with right epileptic focus showed a higher mean score on the LNDS, although this difference did not reach statistical significance. No significant differences in DL performance between groups with left or right focus were found. Mean RE% in the left focus group was 43.4 (SD = 8.9), in the right focus group it was 46.0 (SD = 9.5). The *t*-value of the differences between group means was 0.56 (*p* = .58). Mean LE% in the left focus group was 37.1 (SD = 11.2), in the right focus group it was 39.7 (SD = 9.0). The *t*-value of the difference between group means was 0.52 (*p* = .61). Mean laterality index score in the left focus group was 7.8 (SD = 20.1), in the right focus group it was 7.4 (SD = 17.7). The *t*-value of the difference between group means was 0.04 (*p* = .97).

The scoring manual (Reitan & Wolfson, 1993) gives no standard

TABLE 2. Means, standard deviations, and *t*-test results on age, IQ, memory indexes, and neuropsychological summary measures in groups with left or right seizure focus

<i>Variable</i>	<i>Left focus Mean (SD)</i>	<i>Right focus Mean (SD)</i>	<i>t Value</i>	<i>Significance (2-tailed)</i>
N (males/females)	7 (4/3)	10 (6/4)		
Age	32.3 (7.6)	34.7 (10.1)	0.53	.603
WAIS				
VIQ	97.0 (14.7)	96.6 (11.8)	0.06	.951
PIQ	99.6 (19.0)	96.7 (8.1)	0.43	.674
FSIQ	97.9 (16.9)	96.5 (10.0)	0.21	.837
WMS-R				
VeMI	85.4 (17.2)	93.2 (14.3)	1.02	.326
ViMI	88.7 (12.5)	87.1 (8.4)	0.32	.753
GMI	84.0 (16.4)	89.3 (13.3)	0.74	.473
HRB				
LNDS	4.9 (2.1)	7.0 (3.3)	1.52	.149
RNDS	4.9 (2.3)	4.4 (2.6)	0.37	.715
GNDS	26.4 (11.0)	30.7 (10.6)	0.81	.473

Abbreviations: WAIS = Wechsler Adult Intelligence Scale, VIQ = Verbal Intelligence Quotient, PIQ = Performance Intelligence Quotient, FSIQ = Full Scale Intelligence Quotient, WMS-R = Wechsler Memory Scale—Revised, VeMI = Verbal Memory Index, ViMI = Visual Memory Index, GMI = General Memory Index, HRB = Halstead-Reitan Battery, LNDS = Left Neuropsychological Deficit Scale, RNDS = Right Neuropsychological Deficit Scale, GNDS = General Neuropsychological Deficit Scale.

cut-off score between normal and brain-damaged patients on LNDS. We arbitrarily defined a cut-off score between 5 and 6, which were the values that best suited the data in terms of defining comparable groups and yielding maximum contrasts on DL results. A score of 5 or less is within one standard deviation of the mean result in a control group without brain damage (Reitan & Wolfson, 1993). Thus, it might be argued that the group with LNDS<6 showed normal left hemisphere function, whereas the group with LNDS≥6 showed some degree of left hemisphere dysfunction. As can be seen from Table 3, the groups were comparable on IQ values and memory indexes. Because LNDS was the criterion for group division, there obviously was a significant difference between the groups on this variable. In addition, there was a significant difference between the groups on GNDS, with a higher mean value in the group with LNDS≥6. Significant group differences emerged on two DL measures. In the LNDS<6 group, mean LE% was 32.7 (SD = 6.2), in the LNDS≥6 group it was 45.4 (SD = 8.6). The *t*-value of the difference between group means was 3.53 (*p* = .003). In the LNDS<6 group, mean

TABLE 3. Means, standard deviations, and *t*-test results on age, IQ, memory indexes, and neuropsychological summary measures in groups with Left Neuropsychological Deficit Scale scores under or at and above 6

<i>Variable</i>	<i>LNDS</i> ≥6 <i>Mean (SD)</i>	<i>LNDS</i> <6 <i>Mean (SD)</i>	<i>t Value</i>	<i>Significance</i> <i>(2-tailed)</i>
N (males/females)	8 (5/3)	9 (5/4)		
Age	36.8 (8.5)	31.0 (9.0)	1.35	.198
WAIS				
VIQ	97.3 (15.5)	96.3 (10.3)	0.15	.887
PIQ	97.1 (15.3)	98.6 (12.1)	0.22	.832
FSIQ	97.0 (15.9)	97.1 (10.2)	0.02	.986
WMS-R				
VeMI	92.0 (15.7)	88.2 (16.1)	0.49	.632
ViMI	87.4 (13.0)	88.1 (7.1)	0.15	.885
GMI	88.4 (15.6)	86.0 (14.1)	0.33	.746
HRB				
LNDS	8.6 (2.1)	3.9 (1.4)	5.52	.000
RNDS	5.1 (2.8)	4.1 (2.2)	0.85	.407
GNDS	34.3 (10.9)	24.2 (8.3)	2.15	.049

Abbreviations: See Table 2.

laterality index was 18.2 (SD = 15.3). In the LNDS≥6 group, it was -4.5 (SD = 13.0). The *t*-value of the difference between group means was 3.27 (*p* = .005). The difference in RE% was not significant, but in the direction of more correct responses in the group with LNDS<6. In this group, mean RE% was 48.2 (SD = 10.1). In the LNDS≥6 group, it was 41.3 (SD = 6.5). The *t*-value of the difference between group means was 1.67 (*p* = .116). Because there was a significant difference between the two groups on the GNDS scale, regression analyses with RE%, LE%, and laterality index as dependent variables were performed to sort out the relative contribution of the LNDS and GNDS on these measures. The results of these analyses are given in Table 4.

As can be seen, the combined prediction of GNDS and LNDS on RE% was relatively low, explaining only 9% of the variance on this variable. The correlation and beta values of LNDS were slightly higher than those of GNDS. However, both on LE% and Laterality Index, substantially higher percentages of the variance (respectively, 30% and 27%) were explained by the regression model. Moreover, the relative contribution of GNDS in the regression equation was quite marginal on both these variables, as reflected by its low beta

TABLE 4. Results of regression analyses

<i>Dependent variable</i>	<i>Independent variable</i>	<i>Pearson correlation</i>	<i>Beta</i>	<i>Partial correlation</i>	<i>R²</i>
Right ear %	GNDS	-.22	-.10	-.09	.09
	LNDS	-.29	-.22	-.19	
Left ear %	GNDS	.37	.04	.04	.30
	LNDS	.55	.53	.46	
Laterality Index	GNDS	-.31	-.03	-.03	.27
	LNDS	-.52	-.50	-.44	

and partial correlation values. Thus, the regression analyses confirmed that LNDS was a powerful predictor of results on LE% and laterality index, and that the predictive value of GNDS on these variables was marginal in comparison.

The LNDS \geq 6 group showed larger LE% than RE%, giving a LEA. The LNDS $<$ 6 group showed larger RE% than LE%, giving a REA. To illustrate this difference when seizure focus lateralization is taken into consideration, the DL results are shown in Figure 1, split into four groups according to focus lateralization and LNDS scores. As can be seen, both groups with a LNDS \geq 6 showed LEA, whereas both groups with LNDS $<$ 6 showed REA.

DISCUSSION

The major finding of this study was that left hemisphere dysfunction predicted DL performance in patients with TLE and left hemisphere speech dominance, whereas side of seizure focus in itself did not influence DL performance significantly. As a group, patients with left hemisphere dysfunction did not show normal asymmetry with REA. In fact, they showed a slight LEA. Patients with normal left hemisphere function showed a normal REA on DL. This was the case irrespective of side of epileptic focus. No other demographic or seizure variable could explain the DL deficit in the LNDS \geq 6 group.

This finding is probably best explained by the classic hypothesis

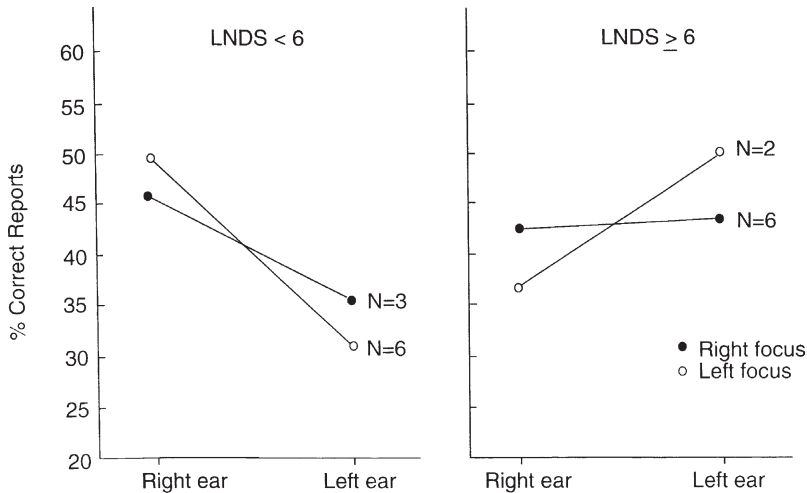


FIGURE 1. % correct right and left ear reports in groups with Left Neuropsychological Deficit Scale (LNDS) scores under or at and above 6, split according to side of seizure focus.

that a left hemisphere lesion interferes with normal asymmetry on DL (Kimura, 1967). The results suggest that this effect can be demonstrated using a neuropsychological criterion for presence of left hemisphere dysfunction (or “lesion”). A similar measure revealing right hemisphere dysfunction (RNDS) did not seem to influence DL performance. This may also be explained by a structural model of DL, in which the right ear item has direct access to the left hemisphere, while the left ear item is transferred from the right hemisphere across the corpus callosum for processing (Sparks & Geschwind, 1968; Pollmann, Maertens, von Cramon, Lepsien, & Hugdahl, 2002). According to this model, the right hemisphere does not actively process the stimuli, and right hemisphere dysfunction should have minimal impact on the processing aspect of DL.

Another important finding was that the shift in ear preference occurred mainly as a result of an increase in reported left ear items. Reduction in reported right ear items was present, but less pronounced, and did not reach statistical significance. Accepting the idea that no stimulus processing is done in the right hemisphere, the best explanation seems to be a deficit in left hemisphere suppres-

sion of left ear input, which could be regarded as a release from inhibition phenomenon. Anatomically, there are both contralateral and ipsilateral projections from the primary sensory cells in the cochlear nucleus to the auditory cortex, but the contralateral projections are stronger (Brodal, 1981). According to the structural model of DL, dominance of the contralateral signals and suppression of the ipsilateral signals normally occur (Kimura, 1967; Hugdahl, 1995). There is neurophysiologic evidence of descending pathways from the auditory cortex to the medial geniculate body possibly sustaining such suppressive cortical-thalamic activity (Rouiller & de Ribau-pierre, 1985). Thus, facilitated perception of left ear stimuli in the group with left hemisphere dysfunction may be explained mainly by less effective suppression of ipsilateral auditory information.

The lack of significant suppression of right ear input may be explained by the nature of the brain dysfunction seen in these patients. There is evidence of normalization of dichotic listening results in the course of recovery from stroke (Hugdahl, Wester, & Asbjørnsen, 1990), and with improvement of seizure status after anticonvulsant medication (Roberts, Varney, Paulsen, & Richardson, 1990). In the present study, no patients had acute or destructive lesions, and mean values on both LNDS, RNDS, and GNDS were below those of 169 patients with heterogenous brain damage in the original validation group (Reitan & Wolfson, 1993), even in the group selected because LNDS was elevated. Thus, suppression of right ear items might be more prominent in patients with large, acute, or destructive left hemisphere lesions; patients with aphasia; or patients with lesions specifically involving primary or secondary auditory cortices (Niccum & Speaks, 1991). Deficient suppression of left ear items may be affected also by mild left hemisphere dysfunction, and thus may be more sensitive to this condition than suppression of right ear items.

No significant association between DL results and side of epileptic focus was found. In several studies this has been the main target of investigation, but results have been contradictory and difficult to interpret. Lack of adequate control for left hemisphere integrity may in part explain these contradictory results. This study suggests that side of epileptic focus does not independently affect DL performance.

Significance of structural lesions on neuropsychological performance in patients with epilepsy has been frequently demonstrated. One study (Matthews & Kløve, 1967) showed increased dysfunction with a known etiology of epilepsy and normal performance in patients with only psychomotor seizures of unknown etiology. In mesial TLE, deficits in verbal memory with left sided hippocampal dysfunction, and deficits in visual memory with right sided hippocampal dysfunction, have been shown (Miller & Munoz, 1993; Chelune, 1995; Hermann, Seidenberg, Schoenfeld, & Davies, 1997). However, this is most reliably shown when there are radiological or pathological signs of hippocampal damage (Hermann et al., 1997). There is little evidence for significant neuropsychological deficits exclusively related to location of an epileptic focus in the temporal lobe, without a known etiology or signs of structural pathology. Other epilepsy-related factors, such as lifetime number of tonic-clonic seizures and status epilepticus (Dodrill, 1986), age of onset, and duration of the disorder (Dikmen, Matthews, & Harley, 1975, 1977; Strauss et al., 1995) and effects of antiepileptic medication (Vermeulen & Aldenkamp, 1995; Meador, Gilliam, Kanner, & Pellock, 2001) may be of potential significance for cognitive function. Such variables may be more important for cognitive function than side of epileptic focus in itself.

No significant effect of right hemisphere language representation on DL performance was found. There is no general agreement on how bilateral language based on the IAT should be defined (Snyder, Novelly, & Harris, 1990; Rausch et al., 1993). This study indicates that minor signs of language representation, such as isolated ability to follow commands or to make dysphasic utterances during the IAT, do not significantly affect DL performance. However, one of the patients did show alterations in DL performance, with a slight LEA, as a probable result of more substantial bilateral language representation.

The LNDS scale has not been independently cross-validated, and there is no independent validation of the cut-off value that was applied on the scale. This study supports the idea that LNDS is a useful measure of left-hemisphere function, and that a cut-off between 5 and 6 may be meaningful in separating normal from deficient left hemisphere function. However, there is a need of further independent validation studies to establish these findings.

The group with right-sided focus showed a higher mean score on the LNDS than the group with left-sided focus, which was unexpected. A trend in the opposite direction would normally be expected in an unselected group of epilepsy surgery patients. The group with pathological scores on the LNDS showed somewhat better verbal memory than the group with normal LNDS score, which also was unexpected. Such unexpected findings may raise concern about the representativeness of the sample studied. However, patient selection was done by including consecutive patients that fulfilled the inclusion criteria. This should indicate that the sample was reasonably representative of epilepsy patients typically being evaluated for surgery. The patient sample was relatively small, and as a consequence, chance findings may have relative large effects. Because of this, nonsignificant findings should be interpreted with caution. However, when statistical significance is reached in such a small sample, it may indicate relatively robust findings. Replication in a larger patient sample would, however, strengthen the conclusions.

In conclusion, for patients with TLE and left hemisphere speech dominance, intact integrity of the left hemisphere seems to be of crucial importance for normal processing of phonological stimuli in a standard DL situation. Lateralization of the temporal epileptic foci does not seem to influence this processing to the same degree.

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