

## Case Study

### DICHOTIC LISTENING IN AN APHASIC MALE PATIENT AFTER A SUBCORTICAL HEMORRHAGE IN THE LEFT FRONTO-PARIETAL REGION

<sup>1</sup>KENNETH HUGDAHL <sup>2</sup>KNUT WESTER and <sup>1</sup>ARVE ASBJØRNSEN

<sup>1</sup>Department of Somatic Psychology, University of Bergen, Haukeland Hospital

<sup>2</sup>Department of Neurosurgery, University of Bergen

(Received January 18, 1990)

Dichotic listening (DL) performance to consonant-vowel (CV) syllables is reported in an aphasic right-handed male patient. The patient suffered initially from a complete expressive aphasia after an intracerebral hemorrhage caused by the rupture of an arteriovenous malformation (AVM) underlying Broca's area. The patient was tested three times with DL; one week after the hemorrhage, five weeks after, and after at about 6 months. At the first test he could not speak, but understood well. At the second test he uttered one-syllable words, and could answer "yes" and "no" to questions. At the third test he could speak whole sentences, although slow and "stutter-like." Dichotic listening performance showed an almost perfect match with speech recovery. At the first test he showed a left ear advantage which changed to no ear advantage at the second test, and to a right ear advantage at the third test. The overall performance was markedly reduced at the first test, but improved at the following tests. It is concluded that dichotic listening may be a valid complement to traditional language assessment procedures after unilateral brain lesions.

**Keywords:** dichotic listening; aphasia; subcortical hemorrhage

An often used technique for noninvasive assessment of language laterality is dichotic listening (DL) (see Hugdahl, 1988 for a review). DL was introduced into neuropsychology in the early sixties by Kimura (e.g., 1961) who reported that subjects with left hemisphere language were more accurate in reporting items presented to the right ear. This right-ear advantage (REA) is a surprisingly robust empirical phenomenon in most individuals (see Bryden, 1988; Geffen & Quinn, 1984). Conversely, individuals with right hemisphere dominance show a left ear advantage (LEA) on verbal tasks (e.g., Strauss, Gaddes and Wada, 1987).

The most favored explanation for the REA phenomenon in a free-recall DL task is the structural model suggested by Kimura (1967). According to Kimura (1967) (see also Bradshaw & Nettleton, 1988) the REA effect is the result of the interaction between four factors: 1) that one hemisphere (usually the left) is dominant, or specialized for speech and language; 2) that the contralateral auditory pathways are more preponderant than the ipsilateral ones (Rosenzweig, 1951; Maximilian, 1982); 3) that the ampler contralateral projection from the right ear to the left hemisphere will suppress or inhibit the ipsilateral left ear input to the left hemisphere; 4) that contralateral information from the left ear to the right hemisphere must be transfer-

---

Address all communications to: Kenneth Hugdahl, Department of Somatic Psychology, University of Bergen, Aarstadveien 21, N-5009 Bergen, Norway.

The present research was financially supported by a grant to Kenneth Hugdahl and Knut Wester from Nansen-fondet, and to Knut Wester and Kenneth Hugdahl from the Norwegian Council for Research in the Medical Science.

contralateral information from the left ear to the right hemisphere must be transferred across the corpus callosum in order to be processed.

As a consequence, ear differences in DL performance may reflect either the relative processing superiority of one hemisphere, or information loss due to interhemispheric transmission, or to an interaction between the two.

Support for the structural model has come from the classic studies by Sparks and Geschwind (1968), and Milner, Taylor and Sperry (1968) who found marked reduction in left ear performance in commissurotomy patients under dichotic presentations. This was explained by Sparks and Geschwind (1968) as due to the fact that auditory signals from the left ear, reaching the right auditory cortex, could not be transferred to the left hemisphere for oral report because of the disruption of the callosal commissures (see also Eslinger & Damasio, 1988).

If this explanation is correct, then also damage to areas in the left hemisphere responsible for language functions should produce an "ear extinction" phenomenon (cf. Damasio & Damasio, 1979; Eslinger & Damasio, 1988). However, in addition to producing left ear extinction, a lesion in the left hemisphere should also produce right ear extinction, i.e., both left and right ear correct recalls should be reduced compared

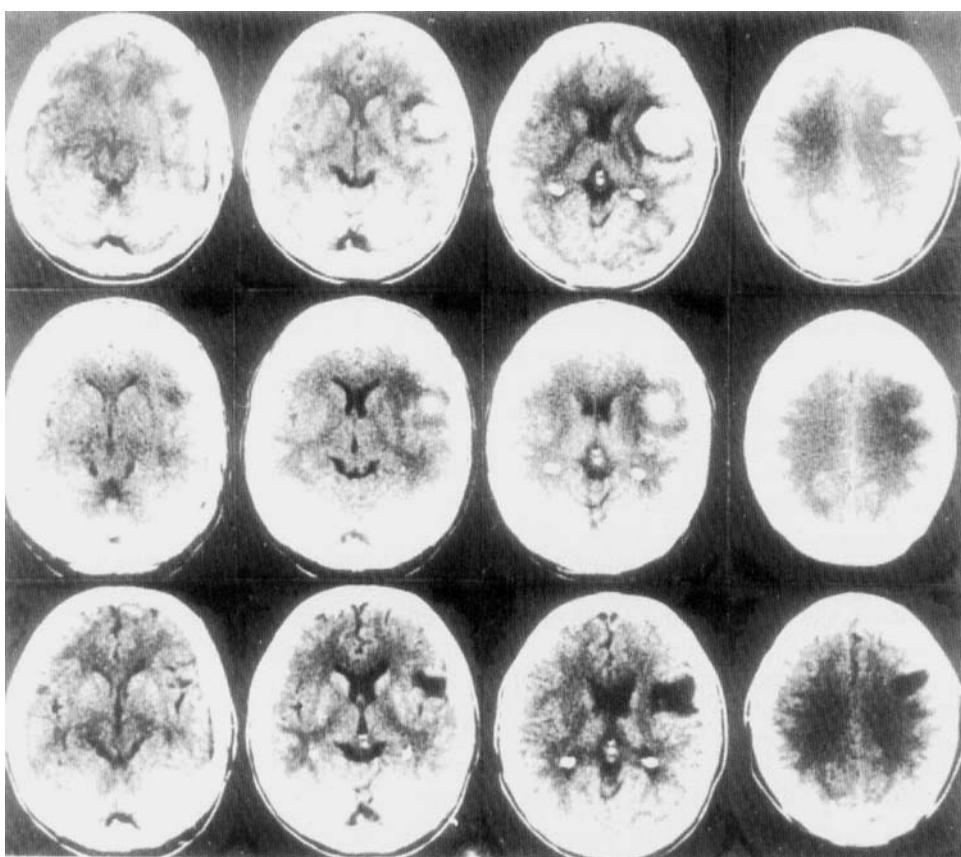


FIGURE 1 Serial, horizontal CT scans from a right-handed male with an intracerebral hemorrhage caused by an AVM in the left frontoparietal region. Note that the left hemisphere is shown to the right and vice versa according to radiological conventions. *Top row:* At the time of the first DL-test *Middle row:* At the time of the second DL-test *Bottom row:* At the time of the third DL-test

to the normal healthy individual. This effect should further be more dramatic the more damaged the frontoparietal areas are.

It could finally be argued that a reduced ear advantage should be most marked in the acute phase of an infarct or hemorrhage, with an increase in overall performance as well as in right ear advantage (REA) along with recovery of speech and language functions.

In the present study we present DL data from a patient who suffered from complete expressive aphasia acutely after an intracerebral hemorrhage caused by an arteriovenous malformation (AVM) covering the left parietal operculum and overlapping the frontal speech area (Broca).

## METHOD

### *The patient*

The patient was a 27 year old right-handed male. One week before the first test session, he woke up in the middle of the night, unable to speak. He was acutely admitted to the hospital, where a complete expressive aphasia and a slight right-sided hemiparesis was found. His consciousness was left undisturbed by the event. Although completely unable to speak, he was able to write simple messages, and seemed to understand all instructions. The deep reflexes were augmented on the right side, but the plantar reflexes were normal. The motor deficits improved considerably during the next 24 hours. A CT scan revealed an intracerebral, subcortical hemorrhage in the left frontoparietal region, corresponding to the area of Broca (Figure 1).

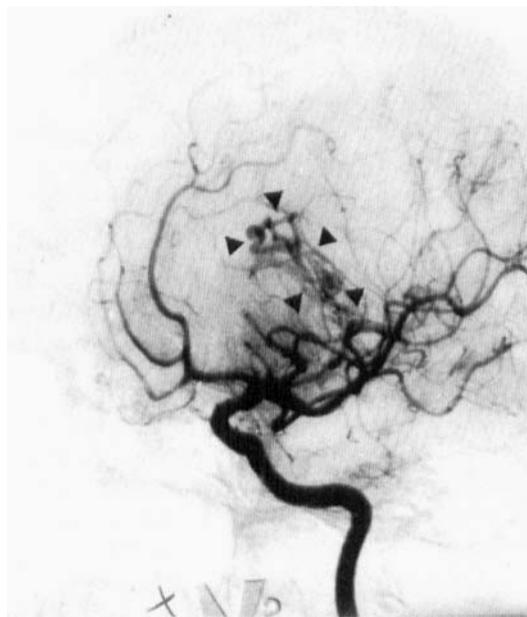


FIGURE 2 Left carotid angiogram showing a small arteriovenous malformation (AVM) situated medially to the hemorrhage in the left hemisphere. Arrows indicate the extent of the AVM. Oblique projection: anterior to the left.

An acute cerebral angiography revealed an AVM in the same region, with an arterial blood supply from the middle cerebral artery (Figure 2).

The patient's handedness was determined by using a modified version of the handedness-questionnaire developed by Raczkowski, Kalat and Nebes (1974). The patient was asked to indicate by nodding or shaking his head whether he used his right, left or both hands to a total of 15 questions related to manual items. The patient was 100% right-handed.

The patient was tested with free recall DL on three different occasions: 1) six days after the acute event, 2) after another four weeks, and finally, 3) six and a half months after the bleeding.

His neurological condition was as follows on the three DL-test occasions:

*First test* Complete expressive aphasia, with understanding well preserved. Able to follow simple instructions (like "raise your arms"), although with slight difficulties. Moderate right facial paresis, and slight hemiparesis. He could walk, but had some impairment of toe movements. His right arm/hand was weak.

*Second test* Speech improved, was now able to say brief words like "yes" and "no," but was completely unable to form sentences. Complete recovery of hemiparesis, but still a slight facial asymmetry.

*Third test* No signs of hemiparesis. Remarkable improvement of speech. He could now form sentences and interact in verbal communication. However, his speech was slow, hesitating and "stutter-like," and he had to search for certain words.

#### *Dichotic listening procedure*

The dichotic stimulus-materials consisted of the six stop-consonants *b, d, g, p, t, k* which were paired with the vowel *a* to form six basic consonant-vowel (CV) syllables (*ba, ga, pa, etc.*). The syllables were paired with each other for all possible combinations, thus yielding 36 dichotic pairs including the homonymic pairs (the homonyms were excluded from statistical analyses). Each pair was randomly recorded three times on the tape. Thus, the total number of trials on the tape was 108. The intertrial interval (ITI) between stimulus presentations varied between 4 and 5 s.

The dichotic tape was prepared on a PDP 11/45 computer with 12 bits A/D and D/A converters, and with D/A multiplexer. Each CV-syllable had a duration of 320 ms, and temporal alignment between channels was set at the first visually identifiable energy-release in the consonant-segment of each syllable. Maximum onset difference between the channels was 0.5 ms due to D/A multiplexer resolution and the sampling frequency (10 KHz).

Each syllable was originally read by a male voice and fed to the computer. After computer analyses, the syllables were recorded onto a NAGRA IV tape-recorder. In order to test the patient more easily in the hospital environment the NAGRA tape was copied onto a chrome dioxide cassette and played from a SONY WM DD minicassette player. The output of the minicassette player was calibrated and the mean intensity was 84 dB A SPL measured with a Brüel and Kjaer 2204 sound level meter.

#### *Test-procedure*

The patient was tested at the University Hospital the first time and at the dichotic listening laboratory in the Department of Somatic Psychology the second and third

listening laboratory in the Department of Somatic Psychology the second and third times. Because the patient could not speak at all during the first test, a modified "monitoring procedure" (Geffen & Sexton, 1978) was used. The patient was asked to point with his left index finger to the syllable he heard on each trial from a list containing all six CV-syllables used in the test. Since the patient was quite slow in monitoring trial-by-trial, a 10 s interrupt procedure was used; i.e., the experimenter waited 10 s after each syllable-pair presentation for a manual response. If no response had occurred during the 10 s interrupt interval, the trial was scored as an omission trial. The monitoring procedure was also used at the two other test occasions, in order to keep the test situations as identical as possible.

## RESULTS

Percentage correct recall from right and left ears, respectively, for the three tests are seen in Figure 3.

As can be seen in Figure 3, there are two main trends in the data. First of all, there is an overall increase in correctly reported items from both ears across time (and particularly from the right ear). This was statistically significant with the Chi-square test ( $\chi^2(2) = 29.04, p < .001$ ). The second trend in the data was a "flip-over" from a left ear advantage (LEA) at Test 1 to a right ear advantage (REA) at Test 3, with no ear advantage (NEA) at Test 2 (see Figure 3). This was statistically evaluated by calculating a laterality-index for each test-occasion and then applying the Chi-square test on the index scores. The laterality-index was calculated according to the formula:

$$\frac{R_c - L_c}{R_c + L_c} \times 100$$

with  $R_c$  = Correct right ear reports,  $L_c$  = Correct left ear reports. Thus, a negative

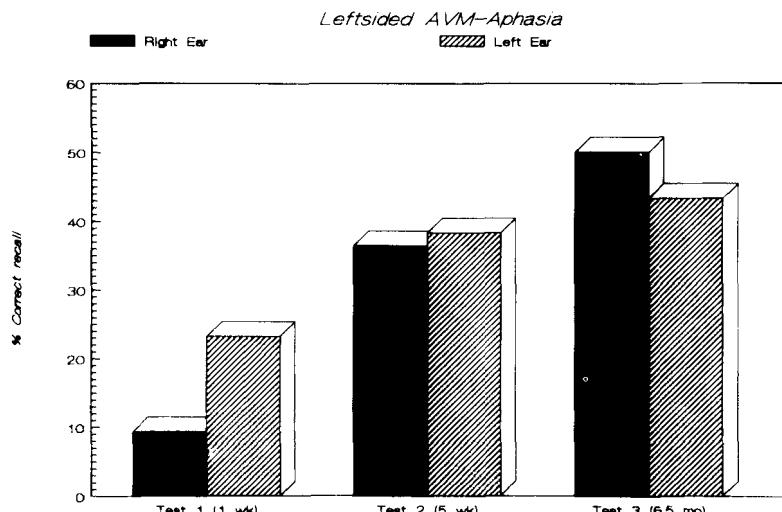


FIGURE 3 Percentage correct recall in dichotic listening for the three test-occasions. 1 wk = 1 week after the hemorrhage, 5 wk = after 5 weeks, 6.5 mo = after 6.5 months.

index indicates a LEA, a positive index a REA, and a zero index a NEA. Index scores for the three test-occasions were  $-42.8$  (Test 1);  $-2.54$  (Test 2) and  $+7.18$  (Test 3). A chi-square test showed a highly significant difference between the three index-scores, ( $\text{Chi}^2(2) = 110.41$ ,  $p < .001$ ).

Evaluating finally the difference between correct right and left ear scores for each test-occasion showed a significant LEA for Test 1 ( $\text{chi}^2(1) = 5.94$ ,  $p < .05$ ).

## DISCUSSION

To sum up the findings, Figure 3 reveals an almost perfectly linear increase in right ear correct reports across test occasions (together with an increase in overall correct performance). This development of dichotic listening performance across time was nicely corroborated by the development of speech functioning. At the first test the patient was completely mute, and did not utter a single word. At the second test he was able to utter single-syllable words like "yes" and "no." At the third test occasion (about 6 months after the hemorrhage) he did speak, although with reduced pace and with some errors.

Thus, there was an interesting parallel in speech-recovery, and the increase in overall DL-performance and particularly the switch from a LEA at test 1 to a REA at test 3. Note that the patient was tested with the same procedure (pointing to the list of CV-syllables) at all three test-occasions.

The present data thus support Kimura's (1967) structural model for explaining dichotic listening, with the REA-phenomenon conditional on the progress of left hemisphere functioning.

A comparison of the patient's DL performance with normative data for righthanded healthy males shows a striking difference at the first test with a clear tendency towards "normalization" across time. Normative data for righthanded adults are shown in Figure 4.

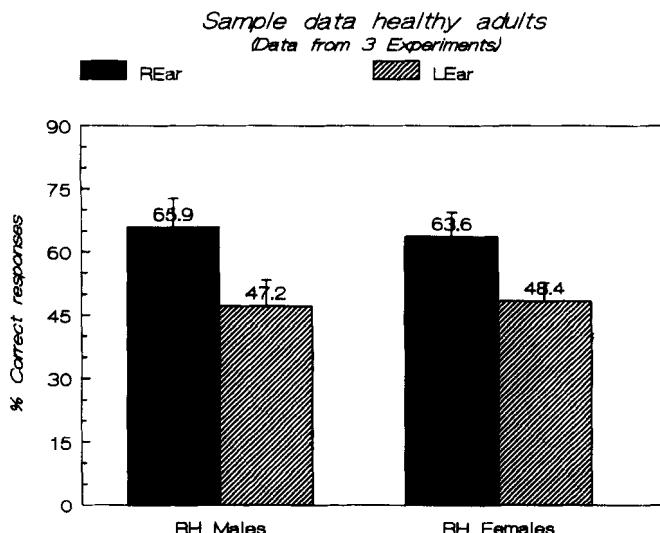


FIGURE 4 Normative data for dichotic listening performance in healthy righthanded adults. RH = righthanded.

As can be seen in Figure 4, percentage correct right ear reports normally varies between 55–75% with percentage correct left ear scores varying between 40 and 54%. Thus, the REA lies within a 10–20% difference for the right and left ear reports (see Hugdahl & Andersson, 1986; Bryden, Munhall, & Allard, 1983 for examples of normative data). The patient switched from a 15% LEA to a 8% REA across the three test occasions.

The LEA at Test 1 is probably best explained as an effect of failure of the left hemisphere to process the contralateral right ear signal at the acute stage of the hemorrhage (cf. Eslinger & Damasio, 1988).

Thus, the LEA effect is primarily not caused by an increase in left ear correct reports, but to a drastic *decrease* in correct right ear reports. This, however, recovers to a “normal” REA as a function of speech rehabilitation.

It is interesting to speculate as to the nature of the almost perfect match between the development in DL performance and speech recovery. It may be argued that DL basically is a test of verbal recognition, or perception (Bradshaw & Nettleton, 1983) with no obvious connection to speech production. More than 95% of all right-handers have speech localized to the left hemisphere (Wada & Rasmussen, 1960). However, DL usually correctly identifies language dominance in only about 85–90% of individuals tested (Strauss, Gaddes, & Wada, 1987). Thus, there is a discrepancy of about 10% in reliability between invasive techniques like the sodium-amytal test, and a noninvasive technique like DL. This has been explained that the invasive technique basically monitors expressive language function while DL mainly monitors impressive function.

However, the patient in the present study had an intracerebral hemorrhage mainly overlapping frontoparietal speech areas, seemingly leaving temporal areas intact. Since the perisylvian fissure and surrounding temporal areas are considered important in language perception (Brodal, 1981), one could argue that dichotic listening performance should not have been affected.

As is obvious from Figure 3 this is, however, not the case. On the contrary, DL performance closely follows speech performance over time. This leaves the conclusion that dichotic listening is not uniquely tied to language perception and speech reception, but that in addition it also reflects changes in the functional status of frontoparietal areas, and consequently in speech production per se.

The impairment of speech and DL performance was probably caused by a *permanent* destruction of some neural elements as well as a *temporary* dysfunction of other neural elements due to local pressure from the hematoma and the oedema surrounding it. Consequently, the parallel improvement in both speech and DL performance was caused by the gradual resolution of the hematoma and normalization of tissue pressure in the affected area.

If the present findings are valid and replicable, then dichotic listening opens up an objective way of complementing traditional speech-tests in assessing overall language functioning after unilateral cortical lesions.

## REFERENCES

Bradshaw, J. L., & Nettleton, N. C. (1983). *Human cerebral asymmetry*. Englewood Cliffs, N. J.: Prentice-Hall.

Brodal, A. (1981). *Neurological anatomy* (3rd Edition). New York: Oxford University Press.

Bryden, M. P. (1988). An overview of the dichotic listening procedure and its relation to cerebral organization. In K. Hugdahl (Ed.), *Handbook of dichotic listening: Theory, methods and research*. Chichester, UK.: Wiley.

Bryden, M. P., Munhall, K., & Allard, F. (1983). Attentional biases and the right-ear effect in dichotic listening. *Brain and Language*, 18, 236-248.

Damasio, H., & Damasio, A. R. (1979). "Paradoxical" ear extinction in dichotic listening: Possible anatomic significance. *Neurology*, 29, 644-659.

Eslinger, P. J., & Damasio, H. (1988). Anatomical correlates of paradoxical ear extinction. In K. Hugdahl (Ed.), *Handbook of dichotic listening: Theory, methods and research*. Chichester, U.K.: Wiley.

Geffen, G., & Sexton, M. A. (1978). The development of auditory strategies of attention. *Developmental Psychology*, 14, 11-17.

Geffen, G., & Quinn, K. (1984). Hemispheric specialization and ear advantages in processing speech. *Psychological Bulletin*, 96, 273-291.

Hugdahl, K., & Andersson, L. (1986). The "forced-attention" paradigm in dichotic listening to CV-syllables: A comparison between adults and children. *Cortex*, 22, 417-434.

Kimura, D. (1961). Cerebral dominance and the perception of verbal stimuli. *Canadian Journal of Psychology*, 15, 166-171.

Kimura, D. (1967). Functional asymmetry of the brain in dichotic listening. *Cortex*, 3, 163-178.

Maximilian, V. A. (1982). Cortical blood flow asymmetry during monaural verbal stimulation. *Brain and Language*, 15, 1-11.

Milner, B., Taylor, L., & Sperry, R. W. (1968). Lateralized suppression of dichotically presented digits after commissural section in man. *Science*, 161, 184-186.

Raczkowski, D., Kalat, J. W., Nebes, R. D. (1974). Reliability and validity of some handedness questionnaire items. *Neuropsychologia*, 12, 43-47.

Rosenzweig, M. R. (1951). Representations of the two ears at the auditory cortex. *American Journal of Physiology*, 167, 147-214.

Sparks, R., & Geschwind, N. (1968). Dichotic listening in man after section of neocortical commissures. *Cortex*, 4, 3-16.

Strauss, E., Gaddes, W. H., & Wada, J. (1987). Performance on a free-recall verbal dichotic listening task and cerebral dominance determined by the carotid amygdal test. *Neuropsychologia*, 25, 747-753.

Wada, J., & Rasmussen, T. (1960). Intracarotid injection of Sodium amygdal for the lateralization of cerebral speech dominance. *Journal of Neurosurgery*, 17, 266-282.