



Differential impairments in recalling people's names: A case study in search of neuroanatomical correlates

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(Received 25 May 1996; accepted 26 October 1996)

Abstract—The case of a patient with selective left hemispheric medial and lateral temporal lobe damage is described. The patient was of slightly supra-average intelligence and had no problems in normal memory functions, but was severely anomia with respect to people's names. One month post-onset, this deficit held for names of colleagues and friends she had gotten to know during the last 10 years prior to the infarct and for all names confronted with post-infarct. On the other hand, learning of face-name associations was preserved and was independent of the ability to generate context-specific information for the subjects whose names were requested. The results support the existence of category-specific naming impairments, and, moreover, indicate a deficit that has to be differentiated with regard to memory systems. A time-limited, but prolonged engagement of interconnected left medial and adjacent lateral temporal lobe structures in echophory is stressed for context-restricted information such as proper names. © 1997 Elsevier Science Ltd.

Key Words: category-specific anomia; medial temporal lobe; memory systems; lateral temporal lobe; positron-emission-tomography.

Introduction

Evidence from a number of sources including positron-emission-tomographic studies [18, 26, 35, 36, 42, 58] and single case reports [12-14, 50] makes it likely that circumscribed cortical regions represent specific semantic attributes. After certain kinds of brain damage patients may no longer be able to name and describe living objects such as a snake or a crocodile, while they are still able to name and describe objects like a tent or a compass [11, 61]. A special class of semantic termini is defined by proper names and—as a specific subset—by people's names, and may be selectively impaired by cortical damage [5, 25, 30, 37, 46-49].

Possibly dependent on specific features of the impairment in name reproduction, left thalamic [6, 30, 39], left temporal [22, 37, 49], left parieto-occipital [47] and left-hemispheric fronto-temporal damage [25, 48] have been

found to result in impairment for proper names. While lesion data are divergent, Damasio and colleagues [9] conducted word-retrieval experiments with both brain damaged patients and normal subjects undergoing positron-emission-tomography (PET). For both groups of subjects, retrieval of words signifying concrete entities (names for persons, animals, tools) depended on different regions of the temporal lobe, whereas naming actions activated the left frontal cortex. In a study likewise based on PET data, Martin *et al.* [36] found that naming pictures of animals was associated with bilateral activations of the ventral temporal lobe, Broca's area and the medial occipital lobe.

With regard to the nature of the deficit, anomia may or may not be embedded in the context of more widespread amnesia. Based on different assumptions, numerous authors have suggested that anomia for proper names constitutes a retrieval deficit [15, 22], or, more specifically, an inability to form—for retrieval—new associations between the meanings of words and their phonological forms [49].

Several further findings have helped to clarify the cognitive aspects involved in naming people. Flude and col-

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leagues [17] assumed that names are stored separately from semantic information about people, and Damasio *et al.* [9] even hypothesized segregated neural systems subserving conceptual and word-form knowledge for the same stimulus. From experimental studies with normal subjects, Valentine *et al.* [57] concluded that name recognition units mediating between word recognition and the access to identity-specific semantics exist. Similarly, Fery and colleagues [15] presumed that anomia is caused by an impaired access to intact phonological representations from semantic memory. On the other hand, as there is evidence that names (e.g. 'Baker') with name-occupation homophones are more difficult to recall than occupations (e.g. baker) [38], this disadvantage for proper names cannot be attributed to differences in the phonological word-form [7], but has to be discussed in the context of word meaning. Indeed, proper names seem to be unique because of their low meaningfulness; they therefore work as 'pure referring expressions' [48] without describing any properties or attributes of the corresponding persons [25]. Moreover, Burton and Bruce [4] emphasized the uniqueness of a name to a given person. A survey of current models and a discussion of recent cases can be found in Cohen and Burke [8].

We here report a new case of a selective anomia for people's names focusing on its neuronal correlates and on the nature of the distinct impairments of different memory systems.

Methods

Case report

CU is a 61-year-old, right-handed, female music teacher who came to our attention with conspicuous problems in remembering names. Following 10 days of indisposition, symptom-onset on 10 December 1995 presented with a bad left-sided headache, nominal aphasia and transient confusion.

She suffered an ischaemic/embolic (?) insult in the left medial temporal lobe probably caused by an atrial septum aneurysm. Acute computer tomographic and magnetic resonance imaging (MRI) scans showed an oedema zone near the left hippocampus with a projection to the territory of the left posterior cerebral artery. Further neurological examinations revealed no changes in sensory or motor functions.

We first saw the patient 10 days post-onset and examined her in a number of general neuropsychological tests (including the Famous Faces Test). A few indications of a naming problem led to a second time point of testing one month later (39 days post-onset). At this date she was also further studied neuroradiologically by MRI and PET and neuropsychologically with several tests specifically designed for investigating anomia.

Neuroradiological examinations

To characterize the chronic state of the patient's brain, magnetic resonance images were performed about one month after the infarct using T1-weighted native and Magnevist-based coronal and T2-weighted axial slices.

Positron emission tomography was performed on a high resolution scanner (Siemens ECAT EXACT HR) [64] with septa retracted in 3D acquisition mode. Acquisition over 90 sec started with i.v. injection of 370 Mbq (10 mCi) ^{18}F FDG. After reconstruction with a Hanning filter (0.4 cycles/pixel) and correction for random coincidences, attenuation and scatter, 47 transaxial slices of 3.125 mm thickness were obtained.

The second MRI confirmed the previous results and, furthermore, specified damage to the left parahippocampal gyrus and to portions of the left thalamus confined to the pulvinar and the retrosplenial cortex (Fig. 1). PET revealed marked metabolism decrease in the left temporo-medial and temporo-occipital cortex as well as in the left posterior thalamus and, thus, supported the MRI-data.

Subjects used for comparison

For tests without normative data (categorical word fluency task, picture-naming task, face-naming task, multiple choice task for assigning names to faces) the patient's performance was compared to that of female control subjects further matched in age ($M=59.5$, $S.D.=2.43$), education and intellectual ability (Verbal IQ: $M=114.3$, $S.D.=8.69$).

General neuropsychological investigation

Ten days post-onset, the patient was tested with a German intelligence test consisting of three verbal and three nonverbal subtests. She showed normal intellectual functions ($IQ=115$) [52] with no difference between verbal and nonverbal performance. A special test for verbal intellectual functions [28] which confirmed this result was added because of the noticeable verbal deficit of the patient. CU's attention and concentration were in the normal range (Concentration-Endurance Test d2 [3]; Trail Making Test [10]), and her cognitive flexibility was unimpaired (Wisconsin Card Sorting Test [40]). Memory assessment by the Wechsler Memory Scale—Revised (WMS-R [62]) and the Auditory Verbal Learning Test (AVLT [24, 29]) revealed no difficulties in learning and recall of verbal and nonverbal material (WMS-R: General Memory Index = 113, Verbal Memory Index = 107, Visual Memory Index = 120, Delayed Recall Index = 95; AVLT: A1 = 6/15, A5 = 15/15, A7 = 8/15 and Recognition List A = 12/15) (for control data in AVLT see Abu-Salih [1] and Geffen *et al.* [19]).

At the time of testing, nominal aphasia emerged only occasionally. The Aachener Aphasia Test (AAT [27]) was managed without problems. Despite subjective complaints in remembering names and digits she had no problems with the subtest Naming from the AAT (t -score = 72) or with the subtest Arithmetic from the Hamburg-Wechsler Intelligence Test (HAWIE [63]; German

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