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SECCIÓ FILOLÒGICA

INTERNATIONAL WORKSHOP ON LANGUAGE, BRAIN AND VERBAL BEHAVIOR: NEUROBIOLOGICAL ASPECTS OF LINGUISTIC CAPACITIES AND LANGUAGE PROCESSING

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SECCIÓ FILOLÒGICA

International Workshop on language, brain and verbal behavior: Neurobiological aspects of linguistic capacities and language processing

Aproaches from (neuro)linguistics, (neuro)biology, and (neuro)psychology

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Joan Albert Argente i Giralt
Institut d'Estudis Catalans

Coordinator

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Presentation

Two years ago, in the presentation of the Workshop on Language, Cognition and Computation, I wrote: "although mental processes or cerebral functions such as perception, reasoning or language may be studied and described in terms of structural regularities and formal patterns independently of their notional content, it is also true that these functions may be studied independently of their physical and biological basis; the metaphor of the brain as hardware and the mind as software has been proved extremely fruitful at tightening the relationship between the humanities and the computational sciences. The main goal of this workshop is to explore the results, the validity and the limitations of such a metaphor."

The Workshop was oriented, then, towards those perspectives that tackle the study of cognitive processes with a methodology that could be described as "top-down": from software to hardware. There remained open the possibility of organizing a second workshop where the same topics were present but as understood from the other methodological perspective, "bottom-up", from hardware to software.

The great impact of new technologies on medical sciences and the development of molecular biology have contributed in arousing the interest on the study of the physical and biological underpinnings of mental processes, both from the point of view of their implementation in the cerebral hardware and from the point of view of their origins within the general framework of biological evolution. And such renewed interest on the brain is not only circumscribed to neurosciences and biology, but to a wider range of disciplines within the humanities, from anthropology and paleontology to psychology and philosophy.

This is a world full of questions, but new and original questions that open new ways for research: what is the relationship, if any, between the origins of language and the origins of other human capacities such as tool-making? Must we interpret the existing correlation of certain aphasias with variable degrees of apraxia as evidence in favor of such a relationship, given the neuroanatomical proximity of certain centers of motor control to the areas where linguistic capacities appear to reside? Does that mean that the origins of language and culture are, at least in part, amenable to an explanation in biological terms, or they are independent phenomena? Is there any parallelism between the process of language acquisition and embryological development; that is, is child language a sort of primitive stage of language just like the different stages of embryological growth have been interpreted by some biologists as a "short history of animal evolution" from the simplest forms to the more complex ones? What can be said about the inverse process: the loss or the decay of linguistic capacities either as a consequence of external or pathological agents or as a consequence of the natural decay of cerebral tissues imposed by age?

It is one of our main goals in this second workshop to provide with the framework for a reflection on the results, validity and possible limitations of the lines of research defined by the questions above. In addition, it will also be interesting to think about the relationships between the "top-down" and "bottom-up" methodologies and about the future of such relationship: will there be convergence or just substitution of the neurosciences for the cognitive sciences? Such a reflection has both a scientific and a practical interest. It must be observed, for instance, that most practical applications in the language industries have been following the cognitivist paradigm with only a minority of applications following the ideas of neural modeling, which has nevertheless had the consequence of transferring the methodological debate to this area as well.

Another practical consequence is the one derived from a better understanding of the processes of acquisition and decay of linguistic capacities to the extent that such understanding may have repercussions on two situations of a great social importance nowadays: the need for a general knowledge of foreign languages and the decay of cognitive capacities due to greater life expectations.

Joan A. Argente

Joan Albert Argente
Secció Filològica
Institut d'Estudis Catalans
Carrer del Carme, 47
E-08001 Barcelona
Tel. (34 3) 318 55 16
Fax (34 3) 412 29 94
e-mail: lcastellon@iec.es

Sergio Balari
Dept. de Filologia Catalana
Universitat Autònoma de Barcelona
E-08193 Bellaterra
Tel.: 34 3 581 23 50
Fax: 34 3 581 27 82
e-mail: ilftg@cc.uab.es

Dominique Cardebat
Institut National de la Santé et de la Recherche Médicale
INSERM Unité 455
Service de Neurologie
C.H.U. Purpan
F-31059 Toulouse Cedex
Tel. 33 61 49 89 26
Fax 33 61 49 95 24
e-mail: u455@purpan.inserm.fr

Marie-Louise Kean
School of Social Sciences
University of California
Irvine CA 92717
Tel.: 1 714 824 40 29
Fax: 1 714 824 23 07
e-mail: mlkean@uci.edu

Loraine Obler
The Graduate School and University Center
Ph. D. Program in Speech and Hearing Sciences
33 West 42 Street
New York, NY 10036-8099
Tel.: 1 212 642 23 52
Fax: 1 212 642 23 79

Michel Paradis
Department of Linguistics
McGill University
1001 Sherbrooke Sr. West
Montréal
Québec H3A 1G5
Tel.: 1 514 398 42 22
Fax: 1 514 398 7088
e-mail: paradis@langs.lan.McGill.ca

Jordi Peña i Casanova
Hospital del Mar
Dept. de Neuropsicologia
Passeig Marítim 25-29
E-08003 Barcelona
Tel.: 34 3 221 10 10
Fax: 34 3 221 05 41

Friedemann Pulvermüller
Institut für Medizinische Psychologie und Verhaltensneurobiologie
Universität Tübingen
Gartenstraße 29
D-72074 Tübingen

Tel. 49 (0) 7071 29 42 20
Fax 49 (0) 7071 29 59 56
e-mail: pumue@uni-tuebingen.de

Núria Sebastian
Dept. de Psicologia Bàsica
Universitat de Barcelona
P. de la Vall d'Hebron, 171
08035 Barcelona
Tel. 34 3 402 10 63 ext. 0-3169
Fax: 34 3 402 13 63
e-mail: nsebastian@psi.ub.es

Institut d'Estudis Catalans
Carrer del Carme, 47
08001 Barcelona
Tel.: 34 3 318 55 16
Fax: 34 3 412 29 94
e-mail: nportet@iec.es

Fundació Catalana per a la Recerca
Passeig Lluís Companys, 23
08010 Barcelona
Tel.: 34 3 315 23 23
Fax: 34 3 268 01 01

Comissió Interdepartamental de Recerca i Innovació Tecnològica (CIRIT)
Via Laietana, 33 6a planta
08003 Barcelona
Tel.: 34 3 310 22 63
Fax: 34 3 310 73 22

Joan A. Argente is Professor of General Linguistics and Chair at the Universitat Autònoma de Barcelona, and President of the Philology Section of the Institut d'Estudis Catalans. He has served on several editorial boards and has been the editor of a Catalan Journal on Language Sciences. Joan A. Argente has published many papers on Catalan, Galician and General Linguistics and Sociolinguistics. His research concerns in the past range across the fields of syntax and grammatical theory, poetics, and language contact in its socio-cultural context, and he has dealt, among others, with topics in Catalan and Romance syntax and the syntax/semantics relationship, the linguistic basis of poetic structures, and the re-structuring of verbal repertoire in language maintenance/shift processes. As a member of the Institut d'Estudis Catalans he has been involved in work on corpus planning for Catalan.
Joan A. Argente is an associated member of the International Center for the Study of Multilingualism (Brussels) and a member of the New York Academy of Sciences.

Sergio Balari holds a Ph.D. degree in Philosophy (Hispanic Philology) from the Universitat Autònoma de Barcelona. Since September 1993 he is a Professor of General Linguistics in the same university. From 1987 until 1991 he was a member of the Spanish team in the EUROTRA (machine translation project).
His main research topics move between Computational Linguistics and Grammar Theory. As a member of the EUROTRA Project, Sergio Balari worked in several areas both theoretical and applied, of natural language processing in the framework of unification-based grammar formalisms. We can emphasize his participation in the design of the Constraint Logic Grammar formalism, whose latter versions have been adopted as a standard in some European Union Projects.
During 1991 and 1992 he was a researcher at the University of the Saarland in Saarbruecken (Germany). His stay was financed by a CIRIT scholarship. During this period he wrote his Ph.D. thesis about the agreement relations in unification-based formalisms, supervised by Prof. Hans Uszkoreit and Prof. Josep Maria Brucart.

Sergio Balari has continued his research both on the theoretical and applied aspects of language processing and grammatical theory. We can point out his participation as a coordinator (jointly with Josep Maria Brucart) of the Universitat Autònoma de Barcelona team in the European Project LATESLAV for the development of advanced grammar checkers and his participation as editor and author in the book *Romance in HPSG.*

Dominique Cardebat is a linguist; she obtained her Ph.D. at the University of Toulouse Le Mirail and has been working, for 10 years, as a Senior Scientist in an INSERM (Institut National de la Santé et de la Recherche Médicale) unit that is localized in the department of Neurology of Toulouse University Hospital (Purpan Hospital). Her main topic of interest is psycholinguistics processes in brain-damaged patients, especially at the lexical semantic level. Her works consist in a two-fold approach combining cognitive neuropsychology and functional neuroimaging.

Mary Louise Kean is Professor in the Department of Cognitive Sciences at the University of California at Irvine. She received a Ph.D. in Linguistics from the Massachusetts Institute of Technology (MIT) in 1975, and has been a Research Fellow at the Max Planck Institute for Psycholinguistics (Nijmegen, 1980-1987). She has been elected a member of the Academia Rodinensis pro Remediatione (1988) and of the Academy of Aphasia (1994). Her research work is focused on the linguistic theory —especially the theory of markedness—, and on the linguistic interpretation of aphasic syndromes— especially agrammatism. She is the author of the *Theory of Markedness in Generative Grammar* (1980), and many professional articles; has been the editor of *Agrammatism* (1985), and co-editor of *Amsterdam-Nijmegen Test voor Alledagse Taalvaardigheid.*

Loraine K. Obler is a Distinguished Professor in the Program in Speech and Hearing Sciences at the Graduate School of the City University of New York. She received her Doctorate in Linguistics from the University of Michigan and an Honorary Doctorate from Stockholm University. Her areas of research interest have been: language in healthy aging and dementia, neurolinguistics of bilingualism, cross-language studies of morphological impairment in agrammatism, and dyslexia. These are reflected in a number of the books she has written or edited which include:

Albert, M. L. and Obler, L.K., *The Bilingual Brain: Neuropsychological and Neurolinguistic Aspects of Bilingualism. New York*: Academic Press, 1978.

Obler, L. K. and Albert, M. L., Eds. *Language and Communication in the Elderly: Clinical, Therapeutic, and Experimental Aspects.* Lexington, Massachusetts: D. C. Health and Co., 1980.

Obler, L. and Fein, D., Eds. *The Exceptional Brain: Neuropsychology of Talent and Special Abilities.* New York: Guilford, 1988.

Hyltenstam, K. and Obler, L., Eds., *Bilingualism Across the Lifespan: Acquisition, Maturing, and Loss.* Cambridge University Press, 1989.

Menn, L. and Obler, L., Eds., *Agrammatic Aphasia: A Cross-Language Narrative Sourcebook.* Amsterdam: Benjamin, 3 Vols., 1990.

Michel Paradis is Professor of Neurolinguistics and Chair, Department of Linguistics, McGill University, Montreal. He received a Ph.D. in Philosophy (McGill U.) and a Ph.D. in Neurolinguistics (U. de Montreal), and is Fellow of the Royal Society of Canada. He is the author of *The Assessment of Bilingual Aphasia*, available in Spanish as *Evaluación de la afasia en los bilingues* (Barcelona: Masson, 1993), and of The Bilingual Aphasia Test which is currently available in over 60 languages (Lawrence Erlbaum Associates). He has been engaged in research on neurolinguistic aspects of bilingualism for the past 20 years and is currently investigating the neural bases of simultaneous translation.

Jordi Peña i Casanova holds a degree in Medicine and Surgery from the Universitat Autònoma de Barcelona and a MD from the Universidad de Navarra. He studied Neurology with Prof. L. Barraquer-Bordas and at the Hospital de Bellvitge. As a medical student he was already very interested in Neuropsycology and the Behavioral Neurology. He also studied the Russian language and translated "Neurolinguistics" by A.R. Luria into Spanish. He got a scholarship from the French government to work at the Hôpital de la Salpêtrière (Prof. Lhermitte and Signoret) (1975). From 1980 he works as attending physician at the Institut Municipal d'Assistència Sanitària (Hospital del Mar and Centre Geriàtric). He is also an associated professor at the Universitat Autònoma de Barcelona. He has developed and validated a methodology of Neuropsychological Assessment, the Barcelona Test (1980-1996). He has published eight books. Among them: A textbook of Neuropsycology, a handbook of Logopedia and a book about the rehabilitation of aphasia. He has published more than forty articles in journals like: Revista de Neurologia, Revue Neurologique, Brain and language, Aphasiology, The Clinical Neuropsychologist, The Journal of Neurolinguistics. Currently he works on the reading and writing disorders (in an international project) and Alzheimer's disease (methods of diagnosis and pharmacological treatment). He is a member of several professional societies like the Societat Catalana de Neurologia, Sociedad Española de Neurologia, Société de Neuropsychologie de Langue Française, the International Neuropsychological Society, the European Society of Neurology and the Societat Catalana de Neuropsicologia.

Friedemann Pulvermüller is Heisenberg-fellow at the Institute of Medical Psychology and Behavioral Neurobiology of the Universität Tübingen. His work focuses on the neurobiology of language, in particular the questions of how phonemes, syllables, words, syntactic categories, and sentence structures are represented and processed in the human brain. In addition, he has done work on related topics in cognitive neuroscience, in particular on visual form perception, and on therapy of neuropsychological syndromes. His books *Aphasische Kommunikation* and *Neurobiologie der Sprache* appeared in 1990 and 1996, respectively.

Núria Sebastian is Professor of Basic Psychology at the Universitat de Barcelona.
Her research focuses on the mechanisms of perception, production and acquisition of speech and in the mechanisms from which the phonological characteristics of each language depends on. She is in charge of a quality research group in the Universitat de Barcelona, and collaborates with numerous research groups, among them we can emphasize the one with the Laboratoire de Sciences Cognitives et Psycholinguistique (LSCP) of the Centre National de la Recherche Scientifique (CNRS, Paris) and with the Institute Max-Planck of Psycholinguistics in Nijmegen (The Netherlands). The scientific methods she uses include the behavioral types as well as mental imagery (PET scan and Evoked Related Potencials (ERPs)). Recently her research group has begun the first laboratory on acquisition of language in babies in the country.

Cognitive neuroanatomy of language

Dominique Cardebat
Institut National de la Santé et de la Recherche Medical
Toulouse, France

What you're looking at, represents one of the main starting points of the study of aphasia. They are very old data on aphasia explored with a modern imaging technique.

These high-resolution MR images were obtained, in 1994, by Cabanis and co-workers in Paris, from the still preserved brain of the first patient described hundred thirty years before by Paul Broca, in 1865. The patient, called Leborgne, was also known by his nick-name "Tan-Tan" which was the only stereotypic oral production he could produce after his stroke. You can see how this very large lesion destroys a fair amount of frontal cortex and the underlying white matter, and spreads towards the head of the caudate, far beyond the limits of what we call Broca's area.

Brain/language relationships : the "aphasia model"

Over the past century, many attempts have been made to find clear relationships between aphasia and brain lesions, that is to use aphasia as a pathophysiological model to study brain/language relationships. As we all know, some aspects of this model are quite well-established; the exact site of the lesion, its size and its etiology strongly influence the observed aphasia and its prognosis. There are few critical regions such as the posterior part of the left superior temporal region - also called Wernicke's area - whose lesions usually cause massive deficits on several language dimensions. However, most of aphasic symptoms, such as anomia, are related to various lesion sites and this is what I'm referring to on this slide as a distributed anatomy of lesions, suggesting that a given symptom may arise from lesions localized at different points of a network distributed over the left hemisphere if not the entire brain. Besides, the relationships between lesion anatomy and language disorders appear more stable when symptoms, rather than syndromes, such as Broca or Wernicke's aphasia, are considered In fact, the classical dogma on brain & language is frequently challenged by clinical observations and there are what Anna Basso and co-workers (1985) called Exceptions, such as fluent aphasias with pre-rolandic lesions and vice-versa or even more paradoxical cases showing no aphasia at all despite destruction of the whole left peri-sylvian area, or crossed-aphasia resulting from right-sided lesions in right-handed subjects. Many "additional" factors have been invoked to explain such exceptional cases, not really exceptional as they were about 12 % in the Basso's series.

Apart from handiness, other subject-specific factors such as age and gender may influence aphasia type and severity; cultural factors starting from literacy to bi- or multi-lingualism and familiarity with language material or language exercises are obviously major factors, although only poorly understood.

Most importantly, the dynamics of post-lesional phenomena should be considered in this model, both in their neural and linguistic dimensions.

And finally, the effects of patient motivation and the influence of aphasia therapy should certainly not be overlooked.

Brain/language relationships : functional imaging data

Functional imaging techniques, and in particular PET, are potentially of great heuristic value to try and disentangle all these complex influences on the brain/language model. In particular, these techniques should sooner or later help us to understand more about recovery of language functions; indeed, aphasia should be considered not only, from a negative viewpoint, as a set of deficits caused by brain lesions but also, from a positive viewpoint, as the behavioral consequences of both reorganization of neural systems and cognitive strategies of compensation. In fact, there are different ways to use functional brain imaging to expand our knowledge beyond the limits of the classical lesion-based model.

Activation in normal subjects

The first one is to address whether the regions related to impairments of specific language functions when damaged, might be activated in normal subjects relying on the same functions during a language task. For instance, several authors such as Geschwind or Cappa et al. (1981) claimed that phonological disorders are associated with lesions close to the left sylvian fissure whereas lexical semantic disorders are linked to lesions of regions that are more distant to the fissure, such as the inferior parts of the parietal or the temporal lobes. Using PET and a language activation experiment, we addressed whether such a topographical segregation between phonological and lexical semantic processes might be observed in normal subjects when performing two language tasks, respectively related to each of these processes. We used monitoring tasks with 30% targets among distractors in series of stimuli presented binaurally at a constant rate (1 per 3s); subjects responded by clicking on a button with right fingers. In the Phonemes task, stimuli were 3- or 4-syllable non-words and the target was the phoneme /b/ if, and only if, the phoneme /d/ was detected in a preceding syllable. In the Words task, targets were nouns of small animals (smaller than a chicken or a cat) preceded by a "positive" adjective in adjective-noun pairs. This slide represent statistical maps displaying voxels in the brain in which significant changes were observed as blood flow increases in the semantic task compared to the phonological one (this is the superior part of the slide) or blood flow increases in the phonological task compared to the semantic task in the lower part of the slide. These results are in very good accord with our hypotheses based on findings in aphasic patients, as the topography of blood flow increases matched well the distribution of lesions generating either phonemic disorders, namely regions close to the sylvian fissure or lexical semantic disorders, namely inferior temporal and inferior parietal localizations.

Resting state in patients

The second way to use functional imaging and explore brain correlates of aphasia is to investigate the metabolic abnormalities that are induced by the lesions and are seen in functional images during a resting state. A fair amount of studies have been done especially in the States during the eighties with a series of glucose steady-state PET studies in relatively small groups of patients. To my view, one of the major contributions of these studies was to demonstrate the existence of massive remote effects of lesions with metabolic depression spreading far away from the anatomical site of the actual lesion. The most striking example of these remote effects relates to so-called subcortical aphasia in which hypometabolism in the ipsilateral cortex is very frequently observed. This image is from one of our previous studies and represents an example of such remote effects with a sub-cortical ischemic lesion restricted to the white matter of the left frontal lobe, and, in this SPECT slice, a profound hypoperfusion in the corresponding frontal cortex. Some of these studies also reinforced previous finding that direct or indirect damage to specific lesion such as the left posterior temporal region has a critical role in both aphasia type and prognosis. Finally, some follow up studies have been done and some others are currently reported or going on. However, these longitudinal data are still unclear, if not contradictory. In general, the functional significance of the abnormalities or longitudinal changes in brain metabolism observed at rest, remains to be clarified. For instance, remote hypometabolic effects may represent, at least, two different phenomena. On the one hand, the affected regions may be only de-afferented but still can participate in functional activation via other connections or networks.

On the other hand, these hypometabolic regions, particularly when they lie not too far away from the actual lesion or within the same vascular territory, may be actually affected by a neuronal loss, meaning an irreversible lack of function.

Activation in patients

The shortcomings of resting state PET studies obviously incline to explore the functionality of the undamaged brain by using activation tasks to test which spared territories in damaged brains could be involved when patients performed these tasks. This will constitute the third, and last, part of my talk. In fact very little has been done so far using up-to-date methodological standards of PET activation that is high-resolution rCBF recordings using the O15 technique.

Here are the results of one of such rare studies which was published last year in Annals of Neurology, by Cornelius Weiller and Walter Huber and their co-workers from Essen and Aachen. They've studied 6 Wernicke type aphasic patients with retro-rolandic lesions and a good recovery. By comparison to the activations observed in a non-word repetition task and in a verb generation task in normal subjects, aphasic demonstrated of course no activation in the damaged region and increased supra-normal activations in the right hemisphere, both in the superior temporal and the inferior frontal regions and in both tasks. Although appealing at first glance, this type of studies soon appear particularly complex because they combine two main sources of variance:

— one is related to brain lesions and aphasia and we've already seen some of these factors in the first part of this presentation

— the other source of variance comes from the many factors that may distort the results of cognitive activation even in normal subjects. In general such a complexity suggests that activation can only be explored on the basis of single-subject studies. Among factors related to the paradigm, I'd like to show only two brief examples.

The first one is related to the influence of stimulating conditions such as rate of presentation and exposure duration of stimuli. These factors have been recently investigated in great details by Cathy Price from London. As you can see here, there is a linear relationships between the amount of activation in the primary auditory cortex and the increasing number of words that subjects were listening to during conditions b to f, whereas a is a silent condition. Such linear relationship is not observed however in Wernicke's area which tends to respond equally to words presentation whatever the rate of presentation.

Another very important factor is related to the degree of familiarity with the task; these results were published in 1994 by Marcus Raichle and his co-workers in St Louis. On the left, are the activation observed in subjects who performed a verb generation task for the first time. As you can see in the middle part of the image, the activations seen in the naive stage, in particular that in the left frontal cortex, almost completely vanished after subjects had over-practiced the task and the same word list stimuli.

These factors should certainly be controlled in any activation studies and particularly in aphasic patients. However, the major factor related to aphasia is obviously the lesion. This is a massive lesion inducing, among other language disorders, a deep dyslexia. During a reading task in which patient performance were impaired, an activation of the right hemisphere was observed. But in fact, what else could be predicted? , as only very few regions were spared in the left hemisphere.

The question of the specificity of such activations in the right hemisphere can be illustrated by data recently obtained in another patient by Liz Walburton, Cathy Price and Richard Wise from London. These are PET activation results co-registered with the actual MRI of this particular patient who presented a left posterior lesion. The experimental task was verb generation in which this patient performed well. Although right hemispheric activations were seen in the verb generation minus rest comparison, these were no longer apparent in the verb generation minus listening comparison, suggesting that right sided signals do not correspond to some sort of vicarious processes that can be involved in the verb generation task but are rather related to listening to words.

Thus, as the key issue of such studies is the mechanism of recovery and compensation of aphasia disorders, we are facing an even more complex problem. This is to specify activations in terms of signal localization, task-specificity and time curse after lesion onset and to establish causal relationships between such functional data and recovered performance. Many aspects of this problem remain to be addressed in the future. I'd like to close my talk by giving you an example of activation of a particular case of aphasia in which activation data provided some hints on the mechanisms of functional compensation in aphasia.

This young man suffered from an ischemic stoke which destroyed the left posterior sylvian region. He presented a severe Wernicke's aphasia in the first stage of evolution; after few months, he evolved towards a rare syndrome called deep dysphasia in which the main symptom is a deficit of repetition: repetition of concrete nouns is possible but with semantic paraphasias such as fork instead of plate, whereas repetition of abstract nouns, grammatical words, and non-words is just impossible. Auditory comprehension is quite good but again far better for concrete nouns than for abstract ones. In general performances on semantic tasks are fairly good but on phonological tasks, he is really poor. The patient condition can be summarized as understanding the meaning of words (at least concrete words) without accurate processing of their phonological forms.

We activated this patient, unfortunately only using SPECT but still, with, I think, interesting results. By comparison to a reference condition (listening to connected speech spoken in a foreign language), we observed activations in two tasks in which performances were very different. During a phonological task, which was phoneme monitoring in connected French speech, patient performances were at chance level, despite increases of CBF that were seen in almost all the undamaged cerebral territories. During a semantic task, which was monitoring for animal names in connected speech, his performances were fairly good and this was specifically associated with an activation in the right posterior temporal region just as if the activation of this right-sided region could compensate for the lesion effects in a semantic task but not in a phonological task. Whatever the technical limitations of this work, I think it shows how the combination of brain imaging methods with single-case studies of clear-cut psycholinguistic dissociations may have a major impact on our understanding of the brain correlates of language functions and dysfunctions.

In any case, this would give us the opportunity to reconcile two sometimes antagonist approaches to cognitive neuroscience.

Recent Advances in Neurolinguistics: Agrammatism, A Case Study

Mary Louise Kean
University of California, Irvine

Introduction

Modern interest in the study of language and the brain emerged out of the confluence of several independent research initiatives. In linguistics, research has been dominated since the 1960's by the theory of generative grammar, originally developed by Noam Chomsky. A central tenet of Chomsky's theory is that linguistics is, in fact, a branch of theoretical biology; under this view, a theory of grammar is a theory of how the brain organizes and represents knowledge of language (e.g., Chomsky, 1965). Simultaneous with the emergence of generative grammar and quite independent of that, the American neurologist Norman Geschwind had become interested in the writings of the late 19th and early 20th century European neurologists who had studied language and the brain. Geschwind reintroduced this work and, synthesizing it, laid out a model of language organization in the brain (Geschwind, 1965). In 1967, in *Biological Foundations of Language*, Eric Lenneberg attempted to bring together what was known from neurology (including neuroanatomy) and linguistics (including psycholinguistics) to provide the basis for explorations in the biology of language. Despite the significance of the work of each of these men, through the early 1970's little systematic attention was given to neurolinguistics and only rarely were there attempts to build on this initial background (e.g., Whitaker, 1971). Suddenly, in the mid-1970's there was a burst of active research, and since then there has been a rapid proliferation of interest in studying various aspects of language and the brain.

Research on aphasia has held a central place in the development of neurolinguistics over the past two decades. From linguistics, we have the assumption that all human beings are biologically endowed with a specific capacity to acquire, know, and use a language given normal experience in a speech community; that is, there must be some biologically dedicated neural system(s) for linguistic capacity. This assumption, coupled with the consistent observation from neurology that the breakdown of language is not random, but rather has a systematic pattern, makes the study of aphasia an obvious context for investigating human linguistic capacity, in general, and language and the brain, in particular. From the onset of linguistically and psycholinguistically based aphasia research in the mid-1970's, Broca's aphasia has been the dominant area of inquiry. This has occurred, I believe, for three pragmatic reasons: First, Broca's aphasics have, in normal discourse, relatively spared comprehension and thus, in contrast to some other populations, are fairly reliable as subjects - one need not worry inordinately whether a patient's performance on a task reflects a failure to understand the task demands themselves. Second, Broca's aphasics typically present a striking and intriguing deficit in language production, agrammatism - the systematic tendency to omit function words and omit or misuse various inflections. Finally, Broca's aphasia is a relatively common form of aphasia, so there are subjects available for research.

In this paper I will focus on studies of sentence comprehension in Broca's aphasia. In the first section, my emphasis will be on work which was carried out between the mid-1970's and mid-1980's. This work did much to establish the questions which have been of primary research interest since and, as importantly, to determine what areas would be ignored. In the second section of the paper, my emphasis will be on work done largely in the last decade. This work is striking for a number of reasons. First, unlike the majority of experimental research carried out with neurologically intact populations, a great deal of this research has been explicitly guided by linguistic theory. Second, a significant proportion of this work has been carried out with the goal of relating data on pathological processing of language to theories of the representation of linguistic capacity.

1. What is the domain of study?

In my view, two distinct avenues of investigation can be said to provide the starting point for the modern spate of activity in neurolinguistic studies of Broca's aphasia, the first being the development of awareness of a comprehension deficit and the second being the attempt to develop formal analyses of the disorder. Wernicke (1874) had observed that there were some comprehension problems associated with Broca's aphasia, but these were not considered a core or significant component of the disorder; the central deficit of Broca's aphasia was seen as lying in the domain of language production. This view was consistently maintained in neurology, psychology, and neuropsychology texts for more than a century, and, indeed, it is still found in many standard texts (e.g., Gleitman, 1995). However, in the 1970's, papers began appearing which reported a systematic comprehension deficit in Broca's aphasia (Parisi and Pizzamiglio, 1970; Lesser, 1974; Caramazza and Zurif, 1976; Heilman and Scholes, 1976; Zurif and Caramazza, 1976). The most influential of these reports (in terms of citations) were Caramazza and Zurif's (1976) and Zurif and Caramazza's (1976) papers in which it was reported that subjects with Broca's aphasia performed poorly on sentence comprehension and metalinguistic tasks with a variety of sentence types, notably reversible passive sentences. Because correct interpretation of passive sentences involves tacit cognizance of the grammatical role of function words and inflection, findings such as these were taken as evidence that Broca's aphasia involves a parallel deficit in production and comprehension.

Caramazza and Zurif hypothesized that the disruption underlying agrammatism of speech and comprehension involved an inability "to compute full syntactic representations."This view was supported by subsequent studies of sentence understanding. While work such as that of Caramazza and Zurif was informed by psycholinguistic and linguistic concerns, that work made little attempt to provide any *formal* characterization of the impairment of Broca's aphasia; their proposal, for instance, was that patients encode semantic relations based largely on lexical content and plausibility rather than computing syntactic structure.

Kean (1977) presented the first detailed attempt at providing a formal analysis of the deficit(s) associated with Broca's aphasia. In that analysis, based on the assumption of parallel deficits in production and comprehension, the goal was to see if it was possible to account for the general range of deficit data typically ascribed to agrammatic Broca's aphasics under a single representational hypothesis. The previous research on agrammatism had provided evidence of compromises in both the syntactic and semantic analysis of sentences in the manifest performance of patients. The loci of overt deficits are not, however, necessarily the locus/loci of the underlying deficit(s) which give rise to observed behavioral limitations. The full computation of a linguistic representation involves a partially ordered set of stages. An impairment at any single stage can, in principle, lead to overt limitations in the products of other stages because well-formed inputs to the impaired level(s) of representation/ processing will be distorted by the impairment(s) and the ill-formed outputs of the impaired level(s) will inevitably lead to a lack of well-formedness in the outputs of succeeding unimpaired levels. In Kean (1977) it was argued that the then known features of agrammatism could be accounted for in terms of phonological representations rather than syntactic and/or semantic representations. Specifically, it was proposed that agrammatism of speech and comprehension involve a tendency to reduce a string to the minimal sequence of well-formed phonological words. As items such as articles, non-lexical prepositions, and auxiliary verbs are not, from a grammatical perspective, independent words but rather affixes, this hypothesis predicts a tendency toward their omission. With regard to inflections, the hypothesis makes different predictions for relatively uninflected languages like English than it does for more richly inflected languages like Spanish. In the case of English, a noun stem and its minimal well-formed word are typically one and the same, e.g., *dog, house, woman*; this is also the case with verbs, e.g., *walk, eat, sleep*. In Spanish, by way of contrast, noun stems are not typically well-formed words, e.g., *perr-, cas-*, but *mujer*; verb stems, likewise, are not minimal well-formed words, e.g., *and-, com-, dorm-*. It was argued that in a language such as Spanish, the minimal phonological word was typically the standard unmarked (citation) form of the item; thus, it was predicted that in agrammatism there would be a tendency toward the production of singular nouns and infinitives, e.g., *perro, casa, mujer, andar, comer, dormir*. In languages which mark case on nouns, this hypothesis predicts a tendency toward the use of the nominative singular. It was argued that if agrammatism involves the tendency to reduce strings to sequences of minimal phonological words then it would follow that agrammatic aphasics would be unable to fully compute syntactic representations, as Zurif and Caramazza (1976) had hypothesized. While Kean (1977) was this first attempt at a formal analysis, it held a key property in common with its predecessors: Specifically, this is a descriptive analysis of the impairment and does not provide an account of the underlying source of agrammatism.

In 1983, the thesis that agrammatism might involve a parallel deficit in all facets of language use was, apparently, dealt a fatal blow by Linebarger, Schwartz, and Saffran. In their research, three agrammatic aphasic patients were asked to make grammaticality judgments. The data analysis suggested that these patients had a relatively preserved ability to make grammaticality judgments. If this were so, then it would have to follow that these patients were capable of computing syntactic structures. This is, in fact, the conclusion Linebarger, Schwartz, and Saffran draw. To account for agrammatism, they propose the "mapping hypothesis". Under this hypothesis, the deficit of Broca's aphasics involves a compromise in the mapping from well-formed and complete syntactic representations onto semantic representations - in particular, a compromise in the ability to map grammatical functions to semantic roles. Their notion of what the syntax-to-semantics mapping function is and the nature of the resulting semantic representation is, however, left undefined.

There is, however, a devastating conceptual problem with the grammaticality judgment research of Linebarger and her collaborators. It is based on the assumption that when an aphasic patient says that a grammatical sentence is indeed grammatical they are computing the same syntactic representation of the sentence as would a normal neurologically intact individual. However, there is no basis for making such a radical assumption. Consider, for example, the sentence in (1):

1. Sally promised Mary to wash the dishes, and she did.

Two individuals could agree that this was a grammatical sentence, but from that it would not necessarily follow that they were computing the same syntactic and formal semantic representations; one of the subjects could interpret the sentence as meaning that Sally washed the dishes while the other could incorrectly interpret the sentence as meaning that Mary washed the dishes. It is only in the former case that the subject can be argued to have provided the 'correct' grammaticality judgment; in the latter case, while the judgment is apparently correct, probing demonstrates that the judgment is, in fact, not correct in the sense that the appropriate structure had been computed. Thus, to assess an individual's grammaticality judgment, one needs not only a yes/no response but also some independent data on the basis of that judgment in order to interpret the yes/no response. This is the critical issue which Linebarger and her colleagues failed to take into account.

My colleague Charlotte Koster and I carried out a judgment study in which we not only asked subjects to make judgments but also probed those judgements in order to determine their basis, thereby overcoming the crucial problem with the Linebarger, Schwartz, and Saffran (1983) study. Our subjects included 36 healthy adults and 18 Broca's aphasics; all the subjects were native speakers of Dutch. The test consisted of 54 sentences; for each sentence, the subject had to make a judgment and then that judgment was probed. Example sentences and the probes are given in (2).

2. a. *Hans beloofde Thomas niet over zichzelf te praten*
   Hans promised Thomas not to talk about himself
   Probe: I'll let you hear it again. Pay attention ... Who will not be talked about?

   b. *Hetty overtuigde Inge een nieuwe jurk voor zichzelf te kopen*
   Hetty convinced Inge to buy a new dress for herself
   Probe: I'll let you hear it again. Pay attention ... Who bought a dress?

While all 36 of the control subjects provided 'correct' judgments, i.e., said that the sentences were grammatical, and 'correct' probe responses for such sentences, only 4 of the Broca's aphasics consistently provided correct judgments and correct probe responses for sentences like those in (2); the remaining 14 Broca's subjects typically judged the sentences to be grammatical but were individually inconsistent in their probe responses, sometimes identifying the correct actor and sometimes identifying the incorrect actor. Among our other findings was that all the Broca's patients were inconsistent in their judgments of the grammaticality of ungrammatical sentences both within and across types of sentence structures and of the grammaticality of certain classes of well-formed sentences (e.g., those involving nesting). Other researchers have also noted that Broca's aphasics are compromised in their abilities to make correct grammaticality judgments to some noticeable degree on some sentence types (e.g., Hagiwara, 1995; Grodzinsky, 1996). Thus, the claim that Broca's aphasics have relatively intact judgment capacities, as Linebarger, Schwartz, and Saffran (1983) argued, is not supported either conceptually or empirically. That notwithstanding, the impact of the original judgment work has been to significantly undermine the hypothesis of parallelism.

The thesis of parallelism was also dealt a blow by work which purported to show that agrammatism of speech could occur in patients with intact comprehension (Goodglass and Menn, 1985; Kegl, 1996; Kolk. van Grunsven, and Keyser 1982; Miceli, Mazzuchini, Mann and Goodglass, 1983; Friedmann and Grodzinsky, 1994, Nespoulous et al., 1984). Where there are anatomical data reported, the patients do not have a left frontal lesion as is neuroanatomically definitional of Broca's aphasia. For example, the patients reported by Kolk et al. (1982) and Kegl (1995) have parietal lobe lesions, while the CT of the patient described by Friedmann and Grodzinsky (1994) is described as "showing no signs of stroke" but "reveal[ing] an asymmetry in the size of the lateral ventricles, the left being substantially enlarged. It also shows an enlarged Sylvian fissure."Largely unnoted in the discussions of the data of Linebarger and her colleagues is the fact that the three patients they studied do not have 'classic' Broca lesions consequent to stroke. It is also significant, that a number of the patients in this group of good comprehending 'agrammatics' do not seemingly show typical agrammatic production profiles. For example, the patient discussed by Friedmann and Grodzinsky (1994), who has been extensively studied, has a highly selective deficit; her impairment is restricted to production involving (a) tense but not agreement, (b) copular constructions, and (c) realization of sentential subjects; the patient reported by Nespoulous et al. (1984) also seemingly has the same highly restricted deficit. One of the patients studied by Miceli et al. (1983) was not agrammatic in reading and was able to repeat. Thus, it is clear that some variant(s) of agrammatism of speech, but *not* comprehension or judgment, can be found in patients without Broca's lesions. What has not been shown, however, is a patient with a classical Broca's lesion (modulo considerations of depth of lesion and disconnection) who demonstrates agrammatism of speech in the absence of an impairment in comprehension and production. The data available would seem to argue that locus of lesion is a critical variable which must be taken into account if generalizations about the representation of language and the brain are to be drawn from behavioral data from aphasic patients. One cannot assume that some essentially intuitively defined phenomenon such as agrammatism is a uniform deficit across patient populations independent of locus of lesion; rather both lesion site and a constellation of symptoms seem to be critical for developing coherent and general analyses. This observation should in no way be considered surprising since it is well-attested in other areas of neuropsychology (e.g., the differences between patients with amnesia consequent to hippocampal (limbic) lesions vs. those with diencephalic lesions). If anything is surprising, it is that in the domain of aphasia research this has not been acknowledged in practice to any notable degree.

Both the grammaticality judgment research, which includes numerous papers in addition to the original work of Linebarger et al. (1983) (e.g., Wulfeck, Bates and Capasso, 1991; Shankweiler, Crain, Gorrell, and Tuller, 1989) and the reports of so-called anomalous cases of agrammatism without a comprehension deficit have had the seeming consequence freeing investigators to focus on particular facets of disorders. In the domain of agrammatism and Broca's aphasia, the facet which has received the greatest attention is comprehension.

2. Two approaches to agrammatism in comprehension

Given the scope of the literature now available, it is impossible to review all the proposals which have been made and received serious attention in recent years. Therefore, I will restrict my discussion here to two avenues of inquiry: studies of category processing, which initially arose out of the parallelism hypothesis, and studies of sentence comprehension. In the former case, an explicit effort has been made to account for impairments in the context of specifically proposed normal sentence processing mechanisms, while in the latter case the emphasis has been to account for comprehension impairments in the context of recent theories of grammatical representation. While the work on category processing, which has investigated the so-called open-class/closed-class distinction, might be considered a failure since it has not yielded a viable hypothesis to account for the comprehension deficit of agrammatic Broca's aphasics, it is a success story in that it illustrates how there can be a productive interaction between neurolinguistics and psycholinguistics. The work on sentence comprehension using the government-binding theory of grammar has not, on the surface, been a success either if success is solely measured on the basis of propounding a truly viable hypothesis. However, I would argue that this work has been highly successful since it represents an ongoing effort to understand the representation of language in the brain in precise representational terms.

2.1 The Open-Class/Closed-Class Distinction

That the closed-class vocabulary is somehow compromised is definitional of agrammatism; the question has always been the scope and source/cause of the compromise.

One significant line of research has focused directly on the possible processing distinction between the open- and closed-classes as superordinate syntactic categories. In 1980, Bradley, Garrett, and Zurif argued that in normal language processing two lexica are used, one restricted to closed-class items and the other encompassing all closed- and open-class items. An intuitive argument was made for postulating two lexica: The closed-class, as its name suggests, contains quite a small number of items and, therefore, can be rapidly and exhaustively searched easily; since closed-class items provide potent cues to syntactic structure, it would be an asset to an on-line processor to be able to rapidly and selectively access this part of the vocabulary. Data from normal subjects were presented to show a dissociation of vocabulary types. Based on experimental findings from aphasic subjects, Bradley and her colleagues argued that the closed-class lexical access system is compromised in agrammatism. In a wide variety of studies using both visual and auditory tasks (primarily lexical decision) there was a relatively consistent failure to replicate some or all of the findings of Bradley et al. with neurologically intact subjects and/or with agrammatic subjects (Gordon and Caramazza, 1982, 1983, 1985; Friederici and Heeschen, 1983; Matthei and Kean, 1989; Segui, Mehler, Frauenfelder, and Morton, 1982). However, there were some partial replications (e.g., Friederici, 1985; Matthei and Kean, 1989; Shapiro and Jensen, 1986). Such work led to the proposal that the compromise of agrammatism in comprehension was not with the ability to access closed-class items, but rather with post-access processes associated with closed-class items. Most recently, this view has evolved into the notion that the underlying cause of agrammatism involves the *use* of closed class items in real time (Friederici, 1988; Garrett, 1992; Zurif, Swinney, Prather, Solomon, and Bushell, 1993; Pulvermuller, 1995; Blackwell and Bates, 1995). What is striking about all of this work is that in no case have syntactic categories been systematically contrasted in an on-line sentence processing study in which both normal and aphasic subjects participated, thus the hypotheses put forward are empirically quite tenuous.

In order to study the processing of syntactic categories, we carried out a study in which we contrasted both specific syntactic categories and the general open-/closed-class distinction. The task selected was 'identical word monitoring', a task in which a subject hears a target word followed by a sentence in which the target word appears; the subject presses a response key as soon as he recognizes the target word in a sentence. The materials consisted of 'minimal pairs' of sentences such as those in (3), where the word in italics is the target.

3. a. Some animals EAT *ANTS* and other kinds of insects
   Some animals EAT *IN* their dens instead of in the open

   b. Modern artists paint ON *THIN* paper and fabrics
   Modern artists paint ON *THE* sides of buildings

The stimuli were constructed so that each sentence pair consisted of an item where the target was an open-class word and an item where the target was a closed-class word. The open-class target categories used were Noun, Verb, and Adjective, and the closed-class target categories were Preposition, Quantifier, and Determiner. Because many verbs in Dutch do not have overt inflection in sentences, the distinction between verbs with and without overt inflection (V+ and V-, respectively) was also systematically manipulated. Each target category contrast occurred in a sentence pair in word order positions 4, 5, and 6 to control for word position effects. Furthermore, the contexts of the targets was systematically manipulated so that for half the pairs the word preceding the target was an open class item and for the other half the preceding word was a closed-class item (e.g., EAT vs. ON in (3)). This manipulation was essential since it is well-established that in the identical word monitoring task responses to targets can be influenced by the immediately preceding word. Two such pairs were constructed for each category contrast allowed; an example from the Dutch materials used in this study is provided in (4) and (5).

4. a. Kinderen kunnen MEER *ZIEN* door voor in
   Children can more see by in front of
   da groep te gaan staan
   the group standing
   b. Kinderen kunnen MEER *DOOR* hun vrienden worden beinvloed
   Children can more by their friends be influenced
   dan je denkt
   than you think
5. a. Leraren kunnen VEEL *ZIEN* in sommige oudere leerlingen
   Teachers can much see in some older students
   b. Leraren kunnen VEEL *DOOR* hun leerlingen worden gepest
   Teachers can much by their students be pestered

Two tapes were constructed; on one tape sentences (4a) and (5b) occurred, and on the other sentences (4b) and (5a) occurred. Subjects were tested on two occasions, hearing one tape in the first test session and the other in the second test session. Thus, all subjects heard all 240 stimulus items as well as 60 filler items, 30 with targets in word order position 3 and 30 in word order position 7 and the targets for each position equally divided between open and closed class items. The subjects, 36 normals, 8 Broca's aphasics, and 7 Wernicke's aphasics were all native speakers of Dutch.

In order to demonstrate that there is such a thing as an open-class/closed-class distinction which is systematically honored in language processing, it would be necessary to show that *each* closed-class item varied from *each* open-class item as well as showing that the two superordinate classes differed significantly from each other. While the latter finding was obtained for all three subject groups, there was no systematic distinction between the specific categories of the open-class and the specific categories of the closed-class for any subject population (Tables 1, 2, and 3).

	V- (263)	P (299)	Q (318)	V+ (326)	A (336)	N (337)	D (342)
V-	—	ns	ns	***	***	***	***
P		—	ns	ns	ns	***	***
Q			—	ns	ns	ns	***
V+				—	ns	ns	ns
A					—	ns	ns
N						—	ns
D							—

Table 1

Results of post hoc Newman-Kuels comparison of normal subjects' mean reaction times (in parentheses) to target categories (*** = p < 0.01)

	V- (408)	P (499)	Q (458)	V+ (459)	A (461)	N (444)	D (529)
V-	—	***	***	***	***	***	***
P		—	ns	ns	ns	*	***
Q			—	ns	ns	ns	***
V+				—	ns	ns	***
A					—	*	***
N							***
D							—

Table 2

Results of post-hoc Newman Kuelss comparison of Broca's aphasic subjects' mean reaction times (in parentheses) to target categories (* = p < 0.05; *** = p < 0.01)

	V- (377)	P (441)	Q (434)	V+ (420)	A (417)	N (411)	D (481)
V-	—	***	***	***	***	***	***
P		—	ns	ns	ns	ns	***
Q			—	ns	ns	ns	***
V+				—	ns	ns	***
A					—	ns	***
N						—	***
D							—

Table 3

Results of post hoc Newman Kuels comparison of Wernicke's aphasic subjects' mean reaction times (in parentheses) to target categories (*** = p < 0.01)

With respect to specific categories, for all populations verbs without overt inflection showed a significantly different mean response latency from inflected verbs, adjectives, nouns, and determiners, and determiners were significantly different from prepositions. That is, categories which would be expected to differ from each other under any of the versions of the open-class/closed-class processing hypotheses did not, e.g., in no population did prepositions or quantifiers differ from inflected verbs or adjectives. At the same time, categories which would *not* be anticipated to differ from each other did, e.g., in all populations verbs without overt inflection differed from all the other open-class categories, and, also for all populations, determiners differed from at least one of the closed-class categories. These findings support the notion that the so-called open-class/closed-class distinction is an artifact of summing across categories. While the patients had slower reaction times than the normal subjects, globally their performance showed the same pattern as that encountered with normals.

In order to further investigate the data for evidence of the open-class/closed-class distinction, the patients' error data were considered. There was no difference between the two aphasic populations in terms of error rate, and the pattern of errors was the same for both groups, e.g., among the Broca's there were 11 failures to respond to Adjectives, 24 failures to respond to uninflected verbs, and 25 failures to respond to Quantifiers, while the Wernicke's had 15, 23, and 24 failures to respond on these categories, respectively. Both patient groups showed significantly more errors with the closed-class categories than with the open-class categories, but this can be attributed to the comparably high rate of failure to respond to determiners by both groups of aphasics. Thus, when the open-class/closed-class distinction is investigated in detail one finds that not only is there an absence of evidence supporting its role in normal processing but there is also an absence of evidence supporting its role in distinguishing Broca's aphasics from Wernicke's aphasics in sentence comprehension.

In recent work a new approach to the open-class/closed-class distinction can be found in work which distinguishes functional categories from lexical categories and their syntactic projections. Both Hagiwara (1995) and Friedmann and Grodzinsky (1994) have taken this approach. While the cases they discuss are restricted, the general idea bears consideration. Put generally, the idea would be that agrammatic aphasics have a deficit with respect to functor or specifier categories; in any structure where one of these categories appears, all nodes above it are defective. One consequence of such an approach is that it predicts that there will be impairments in sentence processing for sentences in which anomalous performance is not overtly the result of problems with some specific closed-class item(s). This is a line of conjecture which is potentially promising for the analysis of both normal and impaired sentence processing.

2.2 Government-Binding approaches to agrammatism

Since the mid-1980's, a major avenue of research into the study of agrammatism has been syntactic analyses of so-called 'agrammatic comprehension' carried out within the government-binding grammatical framework. In this work 'agrammatic comprehension' refers not just to comprehension problems which are directly attributable to the closed-class, but rather to the comprehension deficits of patients whose speech is agrammatic and who exhibit the following performance constellation on comprehension tasks.

(6) Chance Level Performance

a. Center-Embedded Object Relatives (Caramazza and Zurif, 1976): *The dog that the horse is kicking is brown*

b. Reversible Syntactic Passives (Schwartz, Saffran, and Marin, 1980): *The boy is chased by the girl*

c. Right-Branching Object Relatives (Grodzinsky, 1984): *Show me the boy that the girl pushed*

d. Object Clefts (Caplan and Futter, 1986): *It was the horse that the dog chased*

(7) Above Chance Performance

a. Center-Embedded Subject Relatives (Grodzinsky, 1984): *The horse that is kicking the dog is brown*

b. Reversible Active Sentences ((Schwartz, Saffran, and Marin, 1980): *The boy chased the girl*

c. Right-Branching Subject Relatives (Grodzinsky, 1984): *Show me the boy that the girl is pushing*

d. Subject Clefts (caplan and Futter, 1986): *It was the horse that chased the dog*

A variety of proposals have been put forward to account for this range of data, just a few of which that are closely related will be considered here to illustrate how vibrant this line of research has become.

Grodzinsky's (1986a,b) Trace Deletion Hypothesis (TDH) provided one of the first attempts to account for the pattern of agrammatic comprehension, (6) and (7), within the framework of Chomsky (1981). His basic observation was that comprehension is seemingly impaired where there is movement from object position but not when there is movement from subject position. In the normal case, where there is movement, a trace of the moved element remains at its original locus, and this trace and the moved element are co-indexed. Theta-roles (e.g., AGENT, THEME) assignment is mediated by a chain between the trace and the moved element. In agrammatic comprehension, Grodzinsky argued, the trace is deleted or invisible, and it is, therefore, impossible for the moved element to be assigned a theta-role via the chain. Chance performance on sentences such as those in (6) arises because, when an item is not assigned a theta-role, the Default Principle (8), takes over.

8) The Default Principle

An NP which is not assigned a thematic role...should be assigned a theta-role according to a *list* which universally associates default values with positions.
[Grodzinsky, 1986a, p. 145]

This principle, which is not developed on the basis of linguistic considerations but rather through experience, will assign an agent role to clause initial nouns in English. Thus, in a sentence such as *The dog that the horse is kicking is brown* both *dog* and *horse* will be assigned AGENT, which is the source of the chance performance on such sentences. As has frequently been observed, a central problem with this proposal is that the Default Principle is ad hoc - not based on any established psychological or psycholinguistic principles of strategies - and consequently difficult to evaluate.

Hickok (1992) observes that there are aspects of agrammatic comprehension which Grodzinsky's TDH cannot account for: (a) Hickok and his colleagues found that for sentences such as *The tiger that chased the lion is big* comprehension performance of agrammatics was below chance even though there is mediating between the subject and matrix predicate. (b) Caplan and Futter (1986) and Caplan and Hildebrandt (1988) observed chance level performance with two verb subject-relative constructions, e.g., *The horse that chased the cow kicked the pig.* And, (c) Caplan and Hildebrandt (1988) and Grodzinsky and his colleagues (reported in Grodzinsky, 1990) reported chance level performance on simple sentences with pronouns like *The girl pushed her.* To account for these data as well as those in (6) and (7), Hickok proposes the Revised Trace Deletion Hypothesis (RTDH) in which it is also claimed that traces are deleted or inaccessible.

The RTDH is based on the syntactic assumption of the VP-Internal Subject Hypothesis under which subjects are base generated in the Spec of VP, where they receive there theta-role, and then move to Spec of IP to receive Case at S-Structure. Hickok's analysis also, crucially, makes use of the thematic assignment representation of a verb; this representation is of the form "Verb (x (y))", where x denotes the external argument of the verb and y the internal one (Williams, 1981; Grimshaw, 1990), and unspecified arguments are denoted *. For example, the thematic representation assignment for *The girl chased the boy,* [$_{IP}$ The girl [$_{VP}$ * chased the boy] would be *chase (* (boy)); girl.* Hickok proposes that it is just such representations which are available to the general cognitive system. In sentences such as those in (7), where performance is above chance, an internal argument is specified and only one NP is left to be interpreted as the agent. In contrast, for sentences such as those in (6 a, b, and d), there is more than one NP available for interpretation as the unspecified arguments leading to indeterminacy, hence chance performance. Having thus accounted for the basic cases, the RTDH also provides a fairly straightforward analysis for the other cases of chance performance at issue. There are two features of note in the RTDH: First, it provides an analysis of a wider range of data than does the original TDH. Second, it does not require resort to an ad hoc strategy such as the Default Principle. A serious weakness of the RTDH, as well as the TDH, is that neither can account for cases of performance which is below chance as has been reported by Grodzinsky et al. (1988) with passives of psych verbs, e.g., *The man is adored by the woman.*

The only data considered in both Grodzinsky's original TDH and Hickok's RTDH involve NP movement. If some variant of either general theory were correct, then it would be expected that agrammatic aphasics would have difficulties in comprehension with sentences which involve verb movement. However, Lonzi and Luzzatti (1993) have suggested that agrammatics are not impaired in processing sentences with verb movement. To address this finding, Grodzinsky (1995) proposes that only traces in theta-positions are deleted (or invisible) in agrammatic sentence representations. At the same time, he restricts the Default Principle, proposing a variant, the R(eferential) Strategy, which assigns a referential NP a theta-role "by its linear position" just in case it has no theta-role. The R Strategy is claimed to be a non-linguistic strategy which does not apply to non-referential NP's. However, if the R Strategy is a non-linguistic strategy, how can it critically be sensitive to a specific linguistic distinction, that between referential and non-referential elements?

The issue of referentiality has emerged in recent years as a key topic in the analysis of agrammatic comprehension. To take one example, Avrutin and Hickok (1992) engage this topic through consideration of *Which*-N questions, involving subject and object extraction (9), *Who* questions, which involve a bare wh-operator (10).

9. Which horse chased the giraffe? (subject extraction)
   Which horse did the giraffe chase? (object extraction)
10. Who chased the giraffe? (subject extraction)
    Who did the giraffe chase? (object extraction)

The account they propose is based on the linguistic distinction between *binding* and *government*. Binding relations are generally unbounded and formed by the movement of a referential element, while government relations are bounded by locality principles and arise from movement of non-referential elements (Rizzi, 1990). Building on this, Cinque (1990) proposes that *which*-NP head binding chains while bare wh-operators head government chains. Avrutin and Hickok (1992) presented actions scenarios to agrammatic patients and then asked either a *which*-NP or *who* question. Performance on subject extracted NP's for *which questions* was above chance, while performance on object extraction *which* questions was at chance. For both subject and object extraction *who* questions, performance was above chance. To account for these data, they propose that the deficit of agrammatism involves binding chains but not government chains; the asymmetry with *which* questions is explained by the preservation of government chains. [See also, Hickok and Avrutin, 1995.] As Grodzinsky (1995) has observed, this account seems to fail to account for the passive data since passives do not involve binding chains in Cinque's theory. Another problem with this analysis is that it seemingly predicts above chance performance with object clefts and object relatives, which is contrary to the observations of agrammatic comprehension.

What is striking about this line of research is its overwhelming success in invigorating research on agrammatism and bringing detailed and current linguistic theory to bear on the analysis o agrammatism. Such research illustrates how far we have come since the work of Caramazza and Zurif in 1976; at that point it was a break through to observe simply that agrammatics had a problem in computing syntactic representations. Because of the detail of the hypotheses being put forward, they are easily falsified, but, more importantly, they suggest new areas of investigation. Beyond that, this research raises significant questions about the mechanisms of normal processing. For example, one of the conjectures of Avruten and Hickok (1992) is that there are differential processing mechanisms for binding and government chains with binding chains demanding more processing resources because they involve potentially unbounded relations. In this, neurolinguistic research is posing a significant question for the understanding of normal sentence processing.

3. What's next?

It is clear that the burst of research activity which was set off following the work of Caramazza and Zurif has been highly productive. Not only do we now know that there is a comprehension deficit associated with agrammatism, but the details of that deficit are only beginning to be understood. It is clear that this approach will continue to be fruitful. However, there are two serious weaknesses with the work that is being done that need to be addressed in the future. First, while there have been great advances in the study of comprehension, there has been relatively little research on production. Agrammatic Broca's aphasics have both production and comprehension deficits and both facets of the disorder demand exploration.The hypothesis of parallelism has been abandoned for no empirical reason, rather it has simply become irrelevant to most investigators. Whether or not there is parallelism - or even partial parallelism - has major implications for our understanding of the structure of normal linguistic capacity. Aphasia research offers a unique window on both representation and computation in production and comprehension which it is a mistake to ignore. Second, despite the wealth of available data and the implications of the analyses of those data for theories of normal processing, there has as yet been relatively little attempt to connect hypotheses related to agrammatic deficit to explicit theories of computational processes for normal representation. It will only be when approaches to normal processing in adults show the same vigor and attention to linguistic detail as aphasiological research as work such as that described here that neurolinguistic research will make the contribution, which is its potential, to our understanding of the organization of linguistic capacity. Thus, there are important areas which we have yet to provide sufficient attention to.

The advent of imaging technologies and their increasing availability for research will provide us with a new means of assessing the organization of language in the brain. As viable techniques are developed for exploiting imaging technologies to investigate questions of detail and subtlety in syntax, studies with both normal and aphasic subjects will give us a new window on the representation of language in the brain. Where those investigations will lead us is unknown, but it is certain that the excitement of the past two decades will come to pale by comparison.

References

Avrutin, S. and Hickok, G. (1992) Operator/variable relations, referentiality, and agrammatic comprehension. Ms. MIT.

Blackwell A., and Bates, E. (1995) Inducing agrammatic profiles in normals: Evidence for selective vulnerability of morphology under cognitive resource limitations. *Journal of Cognitive Neuroscience, 7,* 228-257.

Bradley, D., Garrett, M., and Zurif, E. (1980) Syntactic deficits in Broca's aphasia. In D. Caplan (Ed.), *Biological studies of mental processes.* Cambridge, MA: MIT Press.

Caplan, D. and Futter, C. (1986) Assignment of thematic roles to nouns in sentence comprehension by an agrammatic patient. *Brain and Language, 27,* 117-134.

Caplan, D. and Hildebrandt, N. (1988) *Disorders of syntactic comprehension.* Cambridge, MA: MIT Press.

Caramazza, A. and Zurif, E. (1976) Dissociation of algorithmic and heuristic processes in language comprehension: Evidence from aphasia. *Brain and Language, 3,* 572-582.

Chomsky, N. (1965) *Aspects of the theory of syntax.* Cambridge, MA: MIT Press.

Chomsky, N. (1981) *Lectures on government and binding.* Dordrecht: Foris.

Cinque, G. (1990) *Types of A' dependencies.* Cambridge, MA: MIT Press.

Friederici, A. (1985) Levels of processing and vocabulary type: Evidence from on-line comprehension in normals and agrammatics. *Cognition, 19,* 133-166.

Friederici, A. (1988) Agrammatic comprehension: Picture of a computational mismatch. *Aphasiology, 2,* 279-284.

Friederici, A. and Heeschen, C. (1983) Lexical decision of inflected open class items and inflected closed class items. Academy of Aphasia. Minneapolis, MN.

Friedmann, N. and Grodzinsky, Y. (1994) Verb inflection in agrammatism: A dissociation between tense and agreement. Academy of Aphasia, Boston, MA.

Garrett, M. (1992) Disorders of lexical selection. *Cognition, 42,* 143-180.

Geschwind, N. (1965) Disconnection syndromes in animals and man. *Brain 88,* 237-294 and 585-644.

Gleitman, H. (1995) *Psychology.* New York: Norton.

Gordon, B. and Caramazza, A. (1982) Lexical decision for open- and closed-class words: Failure to replicate differential frequency sensitivity. *Brain and Language, 15,* 143-160.

Gordon, B. and Caramazza, A. (1983) Closed- and open-class lexical access in agrammatic and fluent aphasics. *Brain and Language, 19,* 335-345.

Gordon, B. and Caramazza, A. (1985) Lexical access and frequency sensitivity: Frequency saturation and open/closed class equivalence. *Brain and Language, 21,* 95-115.

Grimshaw, J. (1990) *Argument structure.* Cambridge, MA: MIT Press

Grodzinsky, Y. (1984) *Language deficits and linguistic theory.* Unpublished dissertation, Brandeis University.

Grodzinsky, Y. (1986a) Language deficits and the theory of syntax. *Brain and Language, 27*, 135-159.

Grodzinsky, Y. (1986b) Neurological constraints on linguistic theories. In N. Fukui, T.R. Rapaport, and E. Sagey (Eds.), *MIT Working Papers in Linguistics, 8*, 173-190.

Grodzinsky, Y. (1995) A restrictive theory of agrammatic comprehension. *Brain and Language, 50*, 27-51.

Grodzinsky, Y., Finkelstein D., Nicol, J., and Zurif, E.B.(1988) Agrammatic comprehension and the thematic structure of verbs. Academy of Aphasia, Montreal.

Grodzinsky, Y. and Finkel, L. (1996) Severe grammaticality judgment deficits in agrammatism and Wernicke's aphasia. Academy of Aphasia, London.

Hagiwara, H. (1995) The breakdown of functional categories and the economy of derivation. *Brain and Language, 50*, 92-116.

Heilman, K.M., and Scholes, R.J. (1976) The nature of comprehension errors in Broca's, conduction and Wernicke's aphasics. *Cortex, 12*, 258-265.

Hickok, G. (1992) Aggrammatic comprehension and the trace-deletion hypothesis. MIT Department of Brain and Cognitive Sciences Occasional Papers, 45.

Hickok, G. and Avruten, S. (1995) Representation, referentiality, and processing in agrammatic comprehension: Two case studies. *Brain and Language, 50*, 10-26.

Kean, M-L (1977) The linguistic interpretation of aphasic syndromes: Agrammatism in Broca's aphasia. *Cognition, 5*, 9-46.

Kegl, J. (1995) Levels of representation and units of access relevant to agrammatism. *Brain and Language, 50*, 151-200.

Kolk, H.H.J., van Grunsven, M.J.F., and Keyser, A. (1982) On parallelism in agrammatism: a case study.

Lenneberg, E. (1967) *Biological foundations of language*. New York: Wiley.

Lesser, R. (1974) Verbal comprehension in aphasia: An English version of three Italian tests. *Cortex, 10*, 247-263..

Linebarger, M., Schwartz, M.F., and Saffran, E. (1983) Sensitivity to grammatical structure in so-called agrammatic aphasics. *Cognition, 13*, 361-392.

Lonzi, L. and Luzzatti, C. (1993) Relevance of adverb distribution for the analysis of sentence representation in agrammatic patients. *Brain and Language, 45*, 306-317.

Matthei, E. and Kean, M-L (1989) Post-access processes in the open vs. closed class distinction. *Brain and Language, 36*, 163-180.

Miceli, G., Mazzuchini, A., Menn, L., and Goodglass, H. (1983) Contrasting cases of Italian agrammatic aphasia without comprehension disorder. *Brain and Language, 19*, 65-97.

Parisi, D., and Pizzamiglio, L. (1970) Syntactic comprehension in aphasia. *Cortex, 6*, 204-215.

Pulvermüller, F. (1995) Agrammatism: Behavioral description and neurological explanation. *Journal of Cognitive Neuroscience, 7*, 271-281.

Rizzi, L. (1990) *Relativized minimality.* Cambridge, MA: MIT Press.

Schwartz, M., Saffran, E., and Marin, O. (1980) The word order problem in agrammatism: I. Comprehension. *Brain and Language, 10,* 249-262.

Segui, J., Mehler, J., Frauenfelder, U., and Morton, J. (1982) The word frequency effect in lexical access. *Neuropsychologia, 20,* 615-627.

Shankweiler, D., Crain, S., Gorrell, P., and Tuller, B. (1989) Reception of Language in Broca's aphasia. *Language and Cognitive Processes, 4,* 1-33.

Shapiro, L. and Jensen, L. (1986) Processing open and closed class-headed words: Left hemisphere support for separate vocabularies. *Brain and Language, 28,* 303-317.

Wernicke, C. (1874) *Der aphasiche Symptomcomplex: Eine psychologische Studie auf anatomische Basis.* Breslau: Cohen & Weigert.

Williams, E.S. (1981) Argument structure and morphology. *The Linguistic Review, 1,* 81-114.

Wulfeck, B., Bates, E., and Capasso, R. (1991) A cross-linguistic study of grammaticality judgments in Broca's aphasia. *Brain and Language, 41,* 311-336.

Zurif, E.B., and Caramazza, A. (1976) Psycholinguistic structures in aphasia: Studies in syntax and semantics. In H. Whitaker and H. Whitaker (Eds.), *Studies in neurolinguistics, Vol. 1.* New York: Academic Press.

Zurif, E.B., Swinney, D., Prather, P. Solomon, J., and Bushell, C. (1993) An on-line analysis of syntactic processing in Broca's and Wernicke's aphasia. *Brain and Language, 45,* 448-465.

Language in dementia: implications for neurolinguistic theory

Loraine K. Obler
City University of New York Graduate School

Introduction

For a generalist audience, it is worthwhile to define dementia and discuss the phenomenology of language changes associated with it before turning to the theoretical issues of interest. Thus I will start this presentation with a discussion of dementia broadly, then narrow in on the type of dementia of that is of greatest interest for those interested in language, Alzheimer's dementia. Then I will describe the language changes associated with its various stages. In the second part of the paper, I will focus on four areas of theoretical interest: (Transparency 1)* **first** the interaction between language and cognition that is revealed by the language changes of dementia, particularly as these can be compared to the language changes associated with frank brain damage such as in aphasia resulting from stroke or brain trauma; **secondly,** the related question of whether, underlying the language changes of Alzheimer's dementia is an actual dissolution of the semantic store, or rather problems with access to it; and **thirdly,** what we learn from bilinguals who are demented about the pragmatic and underlying cognitive abilities associated with bilingualism.

Definition of dementia

Neurologists define dementia as being a disease state resulting from cellular changes in the brain whereby cognitive abilities are progressively impaired. Three out of the following four characteristics must occur in order for dementia to be recognized, as you see in the next transparency: **one,** changes in language; **two,** changes in memory; **three,** behavioral changes, such as markedly increased irritability or belligerence or, in one case I know of, a disconcerting increase in "niceness" in someone who had previously always been quite critical; and **four,** impairment of manipulation of acquired knowledge; the standard test for this is the ability to, say, recite the alphabet backwards, or spell a word such as "world" backwards.

* The transparencies mentioned throughout this text are included as an annex at the end of the paper. (E.N.)

There are a number of different sorts of dementia and they occur with relatively different frequency in the general population. Some of them are considered to be the result of primarily subcortical damage, that is, damage to the interior areas of the brain, while others are considered to be the result of damage primarily to the cortex, that is damage to the external surface, its convolutions and gyri, of the surface of the brain. I'm not going to talk about the primarily subcortical dementias, but for your information they include diseases like Parkinson's Disease and Progressive Supranuclear Palsy. The primarily cortical dementias include Pick's Disease which is associated with cellular damage primarily in the frontal lobes of the brain, and, my focus for today because the language changes are so interesting, Alzheimer's Disease which is associated with cellular changes both in frontal lobes and in temple lobes of the brain as you see in the next transparency (Transparency 3).

For our purposes here, we don't need to discuss the specific cellular and, presumably neurochemical changes that are associated with the dementias; it is the behavioral phenomena that are of interest to us.

One of the forefathers of modern neurolinguistics, Carl Wernicke, in his famous 1874 article in which he described the fluent aphasia of Wernicke's aphasics, was the first to point us to a case of Alzheimer's Disease. It is worth reminding ourselves how Wernicke's Aphasia manifests because in certain stages towards the middle of the decline of Alzheimer's Disease, the language is quite similar to it. In the Wernicke's aphasic, whose lesion is associated with brain damage to the posterior part of the language area of the brain (see Transparency 4) the production of language is quite fluent — unlike that of the non-fluent Broca's aphasics who had been described a decade before by Paul Broca — but, again unlike the Broca's aphasics, comprehension is quite poor. When Wernicke talked about these cases, he gave two examples. The first is clearly the case of a patient who had suffered the sudden onset that is typical of the aphasias and Wernicke dealt with this patient at length; the second case he brings in to bolster his argument that the phenomenon is a more general one; this case is actually, upon careful reading (as in Mathews, Obler and Albert, 1994) more likely a case of Alzheimer's dementia, as the decline was progressive.

Alois Alzheimer himself published two important papers on the dementing disease that was to be named after him, one in 1907 and one in 1911. In addition to careful analysis of the cellular changes associated with the atrophy in the brains of patients he had seen before they died, Alzheimer includes superb descriptions of the language changes and other behavioral changes associated with what we have come to know as Alzheimer's dementia. It was his clinical observation that, in the demented patient one sees - as in the next transparency (5) - impairment in the ability to name things, the ability to comprehend what is said to the patient, the ability to read and write. In the modern period we have understood that the ability to read aloud is markedly better spared than other language abilities, that repetition that may be relatively spared, and that automatic speech shows some decline as well. Problems with discourse are seen in that while a substantial amount of language may be produced, it is quite empty and often impossible for the listener to make sense of.

In the modern period, also, we have come to have an understanding of the stages of dissolution. For the purposes of this presentation, I will speak about three stages: early, middle, and late, although it is often of interest to focus on the stages between early stages and mid-stage and between mid-stage and late stage.

In the **early** stages naming is most likely to be impaired, comprehension in normal conversation appears to be relatively spared, in discourse the patient may wander from what he's said, or not respond fully to what he's been asked, but conversation can still go on. Reading aloud is quite spared, though reading for comprehension of any complex materials poses some problems.

The patient can still write and his speech will be as meaningful as his oral presentation. Automatic speech is relatively spared, although the patient may skip a month of the year in reciting the months of the year, for example, or need to be given the first month in order to start reciting the months of the year.

In addition to the language changes, clinically the patients' family will complain about their behavior in the real world: perhaps they are no longer able to appropriately use a checkbook, or will leave food cooking on the stove or leave crucial ingredients out of a recipe. Patients' memory problems will also become quite severe; they may become lost a few blocks from their house, forgetting where they standardly keep keys, etc.

By the peak of the **middle** stages of the disease (next Transparency, 7), the patient looks like a classic Wernicke's aphasic. On naming tasks, the patient can name only the most common items, but he or she will come up with interesting substitutions for names, these may include examples of the visual misperception e.g. **cucumber** for **escalator** (Transparency 8) or semantically related items (e.g. **elevator** for **escalator).** The patient's comprehension is as poor as that of a Wernicke's aphasic, so if patients respond to some association to a single word in a question, that is not surprising. While most reading aloud may be spared, for languages like English that have many irregularly spelled words, the patient may regularize these, for example pronouncing the word "yacht" as /yatcht/. In writing there are numbers of misspellings and omitted words, as well as nonsense words. Such nonsense occurs in discourse as well; indeed discourse is quite empty as you see in the next transparency (Transparency 9). The patient is unlikely to complete items of automatic speech as well and cannot perform on metalinguistic tasks.

Pragmatic abilities are often remarkably spared, however. The patients can be interrupted in their logorrheic outpouring and will respond to questions even if the response does not make sense in light of the question (or independently!). By this stage the patient requires substantial home care - a classic book on how the caregivers' feel is entitled "The 36-hour day" - and they are no longer able to undertake all but the simplest activities from their pre-morbid life.

In the late stages of the Alzheimer's dementia (next Transparency, 10), language is virtually nonexistent. The patient initiates little speech, may respond pragmatically with a formula, or keep eye contact, but has no sophisticated pragmatic abilities left, and really cannot be tested by any standard language tests. Such patients are frequently institutionalized in the United States.

Now that you have a picture of how the language of a patient with Alzheimer's Disease is likely to look across the progression of the disease, let me turn to the theoretical questions that are raised by patients with the disease. Let us consider first the relation between language and cognition. Carl Wernicke himself had already called attention to the potential link between language and cognition in his classic 1874 article but his position was that the two can be separable. Indeed, he was right in that in aphasia, on which he intended to focus his discussion, they are. Alois Alzheimer, by contrast, saw "aphasia" — that is language problems associated with brain damage — as being one of the behavioral components of Alzheimer's disease.

Consider the problems posed by the phenomena of Alzheimer's Dementia for modern linguists or neurolinguists: We are interested in studying the brain bases of language, and many of us are committed to a belief that a certain subsection of the brain is associated primarily with language, although many other parts may be involved as well. We are committed, too, to a belief that language and cognition are independent. While in aphasia it is often possible to demonstrate that patients have understood, or can problem-solve, even if they cannot articulate an answer, in dementia the language and cognitive problems are clearly confounded with each other. Consider the next transparency (11). Most overtly, if a patient has difficulty with remembering the name of an item, or calls it "thing" or substitutes another word for it altogether, can we say that the problem is strictly a language problem, or is it conceivable that it is another aspect of the many memory problems the patient has? When the patient presents empty discourse, is it because the patient isn't thinking of content to express, or is the thought in fact there, but the ability to articulate it is impaired?

When the patient has difficulty comprehending us, how can we know if this is strictly a linguistic problem, or a problem of memory, or manipulating acquired knowledge, or attending to the materials (one often finds attention problems in patients with Alzheimer's dementia or other dementias) or problems with working memory or short-term memory? In sum, it is when the language problems co-occur with the dementia that we cannot be sure if they are primary phenomena of language impairment or linguistic epiphenomena.

To resolve these questions from the data from Alzheimer's dementia, it is important to recall the aspects of language that are relatively spared (Transparency 12). Thus it is clear that **syntax** remains quite unimpaired in production for patients with Alzheimer's disease as those with Wernicke's aphasia. **Phonology,** too, and **morphophonological** rules are quite spared. Thus the patient will not show the problems that a non-fluent aphasic may show of distortion of phonemes. Although the patients do produce nonsense words - we call these **neologisms** - would they never create words that are **structurally** impermissible in a language, either in terms of the phonemes that are permitted to follow each other, or in terms of the morphemes that are permitted to follow each other. These sparings hold, I must point out, even in that florid middle stage when the patient may produce empty speech, even virtual nonsense. To the extent that these items are spared, of course, we must consider that the other language problems reflect a cognitive decline independent of linguistic abilities.

The second question I promised to treat, is whether the language problems are primarily problems of access to the linguistic store or dissolution of it. This question has been a focus of many researchers over the past decade. It is closely linked to the first question, as you will see. I mentioned earlier that one question we ask when patients are unable to name an item that they see or see a picture of, is whether this is a language or a memory problem. Many psychologists do not see a frank difference between them. They talk about the "semantics store" which includes, as far as I can tell, all language components, including words, as well as all the rest of the knowledge we have learned in our life - e.g. the fact that New Year's Day is January 1st or how to get to this conference room. In Alzheimer's Disease, when patients are unable to read irregularly spelled words that we know they must have read relatively automatically before the Alzheimer's disease, or when they are no longer able to produce automatic speech in its entirety, we begin to suspect that the problem is frank dissolution of the semantic store. One important cue lies in the consistency of the response. If the patient is consistently unable to name a certain item, we may suspect that that item itself is impaired. Myrna Schwartz and her colleagues report a patient named WLP who was no longer able to name reliably common items, and was not even able to reliably sort pictures of dogs, cats, and birds into three piles. However, it is important to note that while the patient often sorted dogs and cats into the same piles, she never confused them with birds. Thus we may say there was **some** dissolution of her semantic categorization abilities, but not complete dissolution, obviously. The more superordinate category, bird vs. four-legged pet, was retained.

We recently conducted a study because patients with Alzheimer's dementia are observed to produce some neologisms - that is, nonexistent words, in their discourse, and also are observed to make semantically-related errors on naming tasks as well as in discourse (Nicholas et al., 1996). It was our hypothesis that patients with Alzheimer's disease, when they made semantically-related errors, would manifest errors that were semantically more distant from the target items than were such semantically-related errors that can also be made, albeit to a lesser extent, by normal elderly. First we had raters exclude patients' responses that represented visual misperceptions. In fact it is the case that patients with Alzheimer's dementia make markedly more of these than normal elderly individuals. Then when we had the remaining responses that could be deemed to be semantically-related responses, we had another set of raters rate the semantic distance of each error item from the target. We expected that, as I said, the semantic distance of the semantically-related errors of the Alzheimer's patients would be greater than that of the normals, and thus reflect the dissolution of the actual semantic store. For example, we expected more close errors like **elevator** for the target **escalator** from the normal elderly and more distant errors, such as **seal** for **beaver** or **hot dogs** for **pretzel** from the patients with mild-to-moderate Alzheimer's Dementia. To our surprise, there was no difference, nor even a tendency in the direction we anticipated. This then suggests that the semantic networks of the patients with Alzheimer's disease are as spared as those of normal elderly, and the problem on the naming task, markedly more severe for even these moderately demented patients, of course, is a problem of lexical access.

Another study that suggested that semantic organization is intact in Alzheimer's Dementia, while conscious access to it is impaired, by contrast, is a series of studies by Nebes and his colleagues (Nebes, Martin, and Horn, 1984; Nebes 1989; Nebes and Brady, 1988; 1990). Moreover the tasks they used were on-line tasks of semantic processing. Because these tests of semantic processing are on-line tasks, they eliminate the memory and other language production problems in the studies that are consistent with decline in the semantic organization itself. In those studies, then, problems with the additional memory load may be falsely suggesting problems in semantic store.

However there is also data in the literature that suggests impairment in the actual semantic store. Studies of consistency in naming also speak to the question of whether the lexical store is impaired or simply hard to access in Alzheimer's Disease. Of course, virtually all neurobehavioral performance in patients with Alzheimer's disease is markedly more variable from day to day, even from minute to minute than in aphasics, who themselves perform somewhat more variably than normal elderly (Transparency 13). Normal elderly themselves perform more variably than normal young subjects on many neurobehavioral measures. When consistency scores on naming batteries are studied, as in Henderson, et al., 1990, we see a certain consistency of naming errors that suggests that actual items have been erased or conflated with others in the lexicon, and thus that semantic memory itself is impaired.

In a recent study by Hodges and colleagues (1996), the authors ask patients with dementia and normal age-matched controls to give oral definitions of words that the patient has been able to name on a picture-naming task and also words that the patient was not able to name. Even for items they were able to name, the patients with Alzheimer's Disease gave worse definitions, and for the items they were not able to name, patients with Alzheimer's Dementia were unlikely to be able to provide even core concepts, although they were able to describe physical aspects of the object. This led the researchers to conclude that the problem in Alzheimer's Dementia is one of dissolution of the semantic store, although of course the problem is that they evaluated their subjects' oral definitions and thus compounded whatever definition problems the subjects might have with the problems of lexical access in discourse!

The question then, of whether it is access or dissolution that accounts for the language problems we see in patients with Alzheimer's dementia, remains unresolved. It certainly may be that there are both aspects of dissolution and of problematic access, but further work in many linguistic arenas remains to be done to determine the complex answer to this question.

The final point I want to cover here is the question of what happens in the bilingual demented patient and what it means. If language stores and the boundaries among items in them dissolve, one might expect bilingual patients with Alzheimer's disease to mix languages at every level, from phonological through morphological, syntactic, and at the discourse level. In fact, to one's surprise, there is relatively little intermixing. Some patients do show the sort of mixing that we can sometimes see in aphasic bilinguals, although there too, the normal bilingual's ability to keep two languages quite separate for production is remarkably spared. Instead of seeing such mixing on a large scale, except perhaps to borrow words in when the appropriate word cannot be found in discourse in the relatively early stages of dementia, the interesting phenomenon one sees in bilingual dementia is an inability to appropriately choose the language according to the interlocutor. A number of instances have been reported in the laboratory of Kenneth Hyltenstam and Christopher Stroud, my laboratory, and those of others, where bilingual demented patients will speak a language that the interlocutor does not understand. Usually this is an immigrant grandparent, say, speaking the first language to a grandchild or to a health care practitioner who simply does not understand it.

Now this phenomenon is an interesting one because we do not see such "regression" to the first language in aphasic patients. The aphasic bilingual patient, while often able to speak - albeit aphasically - in impaired fashion in both languages proportional to how they were known before the aphasia-producing accident, will sometimes recover one or the other language disproportionally to how it was known before, as Michel Paradis has perhaps discussed or will discuss in this conference. In the latter cases, even when one language recovers disproportionally well, it is most frequently the language the patient has been using around the time of the accident, and thus, usually, the appropriate one in the environment the patients find themselves in. With the demented patient, as you will have understood, the opposite is the case. Here we often see the "regression" to the first language that Ribot posited would obtain (although it only obtains with chance frequency - Obler and Albert, 1977) in the bilingual aphasic, where the first-learned language should be better spared.

What is of theoretical import from this phenomenon is the bolstering of the notion with respect to bilingualism in normal individuals that from a very early age - I've seen it at two myself and it's usually reported from three - the child has developed a system for keeping the two languages separate for production and, moreover, the children are quite sensitive about figuring out with whom they speak. With bilinguals, the healthy bilingual child or adult may, in culturally appropriate instances, code-switch between the two languages; with monolingual speakers of either or any of their languages they will appropriately restrict themselves to that language. It is **this** ability that appears to break down in dementia, and as is so often the case in neurolinguistics, it is precisely from the systematic breakdown that we learn about the modularity of abilities in normals. Clearly there is a component of normal bilingualism that consists in assessing the interlocutor's language abilities (and tolerance for code-switching) and determining which language to speak. It is this particular ability, then, that breaks down in discourse of bilingual patients with Alzheimer's Disease, on top of the other problems we have discussed earlier.

To conclude, then, we have evidence that certain aspects of language are remarkably spared in Alzheimer's Dementia as long as speech is produced. These are phonology, morphology, morphophonology, and even syntax. Thus language itself can be seen, even in dementia, to be relatively independent of other forms of cognition. The most problematic intersection of the two is in the lexicon or semantic memory. Here the question is unresolved as to the independence of the linguistic elements from the other data on both sides of the question at this point. Pragmatics too is a point of interesting interface. In the monolingual, as in the bilingual, we see certain aspects of pragmatics relatively spared into the late stages of the disease (Causino, et al., 1994). In the bilingual, one crucial aspect of pragmatic competence is impaired, namely the ability to appropriately assess what language or combination of languages the interlocutor expects to hear.

References

Causino, M., L.K. Obler, J. Knoefel, and M.L. Albert, Pragmatics in Late-Stage Dementia. In R. Bloom, L.K. Obler, S. De Santi, and J. Ehrlich, Editors, Discourse Analysis and Applications: Studies in Clinical Populations, Hillsdale, New Jersey: Erlbaum, 1994.

De Santi, S., L.K. Obler, H. Sabo-Abramson, and J. Goldberger, Discourse Abilities and Deficits in Multilingual Dementia, in Y. Joanette and H. Brownell, Editors, Discourse Abilities in Brain Damage: Theoretical and Empirical Perspectives, New York: Springer, 1989.

Henderson, V.W., W. Mack, D.M. Freed, D. Kempler, and E.S. Andersen, Naming Consistency in Alzheimer's Disease, *Brain and Language*, 1996, 39:530-538.

Hodges, J.R., K. Patterson, N. Graham, and K. Dawson, Naming and Knowing in Dementia of Alzheimer's Type, *Brain and Language*, 1996, 54:302-325.

Hyltenstam, K., and C. Stroud, Bilingualism in Alzheimer's

Dementia:Two Case Studies, in K. Hyltenstam and L.K. Obler, Bilingualism Across the Lifespan: Aspects of Acquisition, Maturity, and Loss, Cambridge: Cambridge University Press, 1989, 202-226.

Margolin, D.I., D.S. Pate, and F.J. Friedrich, Lexical Priming by Pictures and Words in Normal Aging and in Dementia of the Alzheimer's Type, Brain and Language, 1996, 54:275-301.

Mathews, P.J., L.K. Obler, and M.L. Albert, Wernicke and Alzheimer on the Language Disturbances of Dementia and Aphasia, *Brain and Language*, 1994, 46:439-462.

Nebes, R.D., Semantic Memory in Alzheimer's Disease, *Psychological Bulletin*, 1989, 106:377-394.

Nebes, R.D. and C.B. Brady, Integrity of Semantic Fields in Alzheimer's Disease, *Cortex*, 1988, 24:291-299.

Nebes, R.D. and C.B. Brady, Preserved Organization of Semantic Attributes in Alzheimer's Disease, *Psychology and Aging*, 1990, 5:574-579.

Nebes, R.D., Martin, D.C., and L.C. Horn, Sparing of Semantic Memory in Alzheimer's Disease, *Journal of Abnormal Psychology*, 1984, 93:321-330.

Nicholas, M., L.K. Obler, R. Au, and M.L. Albert, On The Nature of Naming Errors in Aging and Dementia: A Study of Semantic Relatedness, *Brain and Language*, 1996, 54:184-195.

Obler, L. and M. Albert, Influence of Aging on Recovery from Aphasia in Polyglots. *Brain and Language*, 1977, 4, 460-463.

Schwartz, M.F., O.S.M. Marin, and E.M. Saffran, Dissociations of Language Function in Dementia: A Case Study, *Brain and Language*, 1979, 7:277-306.

Wernicke, C., 1874. Der aphasische Symptomencomplex. Breslau: Cohn und Weigert. Reprinted 1893 in *Gesammelte Aufsaetze und kritische Referate zur Pathologie des Nervensystems*. Pp. 1-70. Berlin: Fischer.

Wernicke, C. 1908. The symptom-complex of aphasia. In A. Church (Ed.), *Diseases of the nervous system*. New York: Appleton.

Annex

Transparency 1:
The Structure of this Paper

1. Define the dementias
2. The stages of language dissolution in Alzheimer's dementia
3. The interaction between language and cognition revealed in dementia
4. Is the semantic store lost or is access to it impaired?
5. What do the phenomena of bilingual dementia tell us about pragmatics in bilingualism?

Transparency 2:
Alzheimer's Dementia Consists of Three Out of Four:

1. Language changes
2. Memory decline
3. Behavioral changes
4. Difficulties with manipulation of acquired knowledge

Transparency 3:
(brain areas that change in AD)
Transparency 4:
(Wernicke's area)

Transparency 5:
Language Problems in Alzheimer's Dementia

1. Lexical access for naming or in discourse
2. Comprehension problems

3. Reading problems - oral reading better than reading for comprehension
4. Writing problems
5. Impairment with automatic speech
6. Discourse problems

Transparency 6:

Alzheimer's Dementia

Language Changes in the Early Stage

— Naming decline

— At lower end of normal

— Comprehension and conversation good but some problems in testing sophisticated materials

— Some tangentiality in discourse

— Automatic speech relatively spared

— Pragmatics relatively spared

— Reading aloud fine

— Few errors in writing

Transparency 7:

Alzheimer's Dementia

Middle Stage

— Naming markedly impaired

— Comprehension severely impaired

— Discourse empty

— Reading aloud may be relatively spared

— Reading for comprehension severely impaired

— Writing impaired

— Some pragmatics spared

— Automatic speech impaired

— No ability to perform metalinguistic tasks

— Some spared pragmatics

Transparency 8:

(Picture of BNT **escalator)**

Transparency 9:

(Discourse sample from DAT)

Transparency 10:

Late Stage Alzheimer's Dementia

— No speech produced

— Little evidence of comprehension beyond the single word level

— No testable reading, writing, or automatic speech

Transparency 11:

Evidence for Language and Cognition Independence/Interdependence

1. Naming impairment
2. Empty discourse
3. Comprehension problems

Transparency 12:

Spared language abilities in Alzheimer's Dementia

— Syntax

— Phonology

— Morphophonology

Transparency 13:

Variability Hierarchy

1. Patients with Alzheimer's disease
2. Aphasics with frank brain damage
3. Normal elderly
4. Normal young adults

Transparency 14:

Conclusions

1. Language and cognition are dissociable in Alzheimer's dementia.
2. Lexical access is certainly impaired; semantic stores may also be.
3. The bilingual patient with Alzheimer's dementia demonstrates breakdown in normal bilinguals' pragmatic ability to assess interlocutors' bilingual status and language-choice preferences.

What bilingual aphasia tells us about language processing in the unilingual brain

Michel Paradis
McGill University, Montreal, Québec

The study of bilingual aphasia has made us focus on a number of issues that have proven useful for the understanding of aphasia in unilinguals and of the way the brain processes language in general: (1) An attempt to account for the various recovery patterns has led to the notion of inhibition/disinhibition in the use of languages, and of activation threshold of the various language subsystems in unilinguals as well; (2) sociolinguistic registers in unilinguals have come to be viewed as neurofunctionally fractionable in the same way as two languages in the brain of bilinguals; (3) the dissociation between linguistic competence and metalinguistic knowledge in second language learners has led to a better grasp of the roles of procedural and declarative memory in language acquisition and use; and (4) the study of the use of pragmatic features in order to compensate for the lack of linguistic competence in second language speakers has shed light on the role of pragmatics in normal language processing as well as in 2-year-olds' incipient first language acquisition and in unilingual aphasic patients. Each of the above considerations has implications not only for our understanding of the way languages are represented and processed in the brain, but also for a better diagnosis and rehabilitation of neurogenic communication disorders. We shall briefly consider each of these four issues in turn.

Patterns of recovery and explanations: activation threshold

Because bilingual aphasic patients do not always recover both languages to the same extent or at the same time, and in fact one of the languages may never be recovered, some authors had speculated that perhaps each language was located in a different part of the cortex. Pitres (1895) proposed instead that each language independently could be temporarily or permanently inhibited. This suggestion prefigures two present-day notions: that of modularity of language systems and that of differential inhibition which in turn led to the activation threshold hypothesis (Paradis, 1993).

Certain recovery patterns, reported long after Pitres's monograph, confirmed his insight. Antagonistic recovery, and in particular alternating antagonism could definitely not be accounted for by differential localization. For, if the reason why a patient could not speak Arabic on Monday was that its neural substrate had been destroyed, in contrast with French, located in an area that had been spared by the lesion, and hence that she could speak, how could we explain that, on Tuesday, she was able to speak Arabic again, but not French? Or in the case of successive recovery, that a language spontaneously recovered several months later? Thus, the temporary or permanent inaccessibility of languages must be accounted for by something other than location at different cortical sites.

Pitres proposed that the neural substratum of languages that are not accessible is not physically destroyed but functionally impaired. This inhibition, however, is not an all-or-nothing phenomenon: it admits of degree of severity, as evidenced by languages that are more impaired than others in cases of differential recovery. It was quickly assumed that, in the normal use of language, in order to avoid interference, one language was being inhibited while the other was activated (on the model of any function and its antagonist).

However, experimental evidence showed that the language not currently in use was nevertheless never totally deactivated (Green, 1986; Grosjean & Soares, 1986). It had also been observed that in non-brain-damaged individuals, language items were sensitive to frequency and recency of use, in that they were more easily available when they had been frequently or recently used. Elements that have been activated show a priming effect: they are easier to activate again. From these various observations emerged the Activation Threshold Hypothesis.

The Activation Threshold Hypothesis proposes that an item is activated when a sufficient amount of positive impulses have reached it. The amount necessary for the item to be activated is its activation threshold. Every time the item is activated, its threshold is lowered, and fewer impulses are then required to reactivate it. After each activation, the threshold is lowered-but it gradually raises again and if it is not stimulated, becomes more and more difficult to activate. Attrition is the result of long term non-stimulation. Comprehension of a given item does not require a threshold as low as for production of that item. It other words, comprehension requires fewer impulses than production, and is thus easier. This is probably due in part to the fact that the item is activated by the impulses generated by the stimulus as it impinges on the senses. No such external support exists for self-activation of an item, hence the total sum of the impulses required to reach the activation threshold have to be internally generated. Entire systems or subsystems may be inhibited in this way (their threshold raised beyond possible activation). Thus, after a long period of disuse, one of the languages might still be understood but no longer spoken spontaneously. This is of course true of any item within each language.

Pathology (or normal aging) may disrupt the normal activation levels, causing word finding difficulty, for example. Aphasia would correspond to the blanket raising of the threshold of a system, or subsystem, or module, thus selectively or differentially affecting the entire language system, or one of the languages, or phonology, syntax, or lexical access in only one of the languages. The hypothesis can be extended without modification to unilingual systems.

Neurofunctional Modularity: sociolinguistic registers

After considering a number of possible ways in which the two languages of a bilingual speaker might be represented in the brain, and having rejected the extended system, the dual system, and the tripartite system (Paradis, 1987a) as not being compatible with all of the reported data, the Subsystems Hypothesis was adopted as the most adequate working hypothesis. It proposes that each of the languages forms a subsystem within the larger system of language. The various dissociations between languages in bilingual aphasia has shown that each language is capable of selective impairment, and hence must at some level constitute a coherent neurofunctional system. But the ability to mix languages without apparent loss of fluency and cross-linguistic priming point to both languages being part of a larger system, the language faculty, which can selectively be inhibited as a whole, with other cognitive functions remaining relatively intact.

A comparison between unilinguals and bilinguals revealed that there are no functional differences between them: Borrowing, mixing, switching, and translating have their unilingual counterpart in using words from different registers, switching registers in response to changes in the social contexts, and paraphrasing (that is, relating the same message in different words, sometimes with a different pronunciation, syntax and morphology, as well as different lexical items). This led to the assumption that there were probably no neurofunctional differences either, and that therefore, the brain of a unilingual should be organized in the same way as that of a bilingual, with its registers organized in the same way as the languages of multilinguals. The study of Japanese dyslexia, with its double dissociations between the various writing systems (kana and kanji, roman and kanji numerals), as well as dissociations between musical notation, Morse code, or shorthand and cursive writing, and between languages in bilingual aphasic patients led to the hypothesis of neurofunctional modularity (Paradis, 1987b). The Subsystems Hypothesis was then extended to cover the different registers of unilingual speakers as well. One could thus expect the same kind of dissociation between registers in unilingual aphasia as have been observed in bilingual aphasia. Indeed some cases of such dissociations have been described, between formal and familiar registers (Riese, 1949) and between Cockney and Oxford dialects but such reports remain few because the phenomenon has not been systematically investigated so far. Once one starts looking for them, more cases are likely to be found.

The role of the right hemisphere in language processing

A major question about the bilingual brain has been the extent to which it might differ from the unilingual brain. On the basis of results from a few dichotic listening and visual half-field tachistoscopic studies, it was first speculated that language organization in the brain of the average bilingual may be more bilateral than in that of a unilingual and that patterns of cerebral dominance may be different for each language in the brain of a bilingual (Albert & Obler, 1978). Even at that time it was apparent that the few studies on which the differential lateralization hypothesis was based presented several contradictions. As additional studies failed to support predictions, the hypothesis was gradually narrowed to apply to more and more restricted subgroups of bilinguals: Late acquirers (the age hypothesis), late acquirers at the beginning stages of acquisition (the stage hypothesis), late acquirers at the beginning stages of acquisition in an informal environment (the stage + manner of acquisition hypothesis). While many studies continued to find no difference, those studies that did report a difference were making contradictory predictions. For example, in direct contradiction with the stage and manner hypothesis, students in a formal learning environment were reported to become less lateralized as they became more proficient (Bergh, 1986). Finally, a meta-analysis of all the available experimental data could not find evidence of lesser asymmetry of language representation in the brains of bilinguals of any type (Vaid & Hall, 1991). Clinical studies have consistently reported the same incidence of crossed aphasia in bilingual as in unilingual subjects (Chary, 1986; Karanth and Rangamani, 1988; Rangamani, 1989), suggesting that the contradictory results of experimental studies might be due to the lack of validity of the laterality paradigms used in these experiments, given a 90% chance of misclassification of subjects into a right-brain language group, as was argued 20 years ago by Satz (1977). Colbourn (1978) also pointed out that there was no foundation for the assumption that the degree of a performance asymmetry reflects the degree of lateralization for the task or stimulus material used.

An inquiry into what the alleged increased participation of the right hemisphere might consist of (Paradis, 1987a) has led to the realization, on the basis of language-related deficits reported in right-brain damaged patients, that non-balanced bilinguals might well rely to a greater extent on pragmatic aspects of language in order to compensate for the gaps in linguistic competence in their weaker language. It became clear that in order to derive the meaning of any utterance in context-that is, in the normal use of language-both linguistic competence and pragmatic competence are needed. Both are necessary, but neither is sufficient, and each is subserved by a different hemisphere (Paradis, 1994a). A left hemisphere lesion will result in dysphasia (the disruption of phonology, morphology, syntax and/or the lexicon); a right hemisphere lesion will result in dyshyponoia[1] (an impairment in making appropriate inferences from the context or from general knowledge).

The use of pragmatic features to compensate for lack of linguistic competence is also a fact of incipient first language acquisition (Bloom, 1974). (Fig. 3, Hi & Lois) It then becomes apparent that there is no clinical evidence of right hemisphere involvement in the processing of linguistic competence in unilingual two- to five-year old children. The original rationale for suspecting the involvement of the right hemisphere in the beginning stages of second language acquisition was that it recapitulated first language acquisition. But there is no evidence that children process grammar (i.e., phonology, morphology, syntax, and the lexicon) in the right hemisphere even at the earliest stages of language development. There is therefore no foundation to the assumption that the acquisition of linguistic competence in a second language, like that in the first, would involve the right hemisphere in the beginning stages. Second language acquisition may indeed recapitulate the sequence of processes engaged in first language acquisition, including right hemisphere participation-but by implicating pragmatic, not linguistic competence.

(I assume researchers that claimed right hemisphere participation for language referred to grammar since they used methodologies that purport to measure language as it is represented in the left hemisphere of unilinguals, and could not be influenced by right hemisphere-based pragmatic features without admitting that their procedures were invalid (i.e., not measuring what it is purported to measure). In fact it is difficult to see how results could be contaminated by pragmatics when the stimuli consisted in digits, syllables or, at best, isolated words).

The use of metalinguistic knowledge and implicit linguistic competence

Another means by which second language speakers are able to compensate for their lack of linguistic competence is metalinguistic knowledge. While competence in a native language is acquired incidentally, i.e., by focusing attention on some aspect of utterances other than that which is internalized (e.g., on meaning while acquiring a grammar; on acoustic properties of sounds while acquiring motor programs for the production of those sounds); is stored implicitly (i.e., outside the scope of awareness) and remains for ever opaque to introspection, and is used automatically (i.e., without conscious control), metalinguistic knowledge, on the other hand, typically encountered in school, is learned consciously, that is, by paying attention to what is memorized, can be recalled and recounted, and is produced in a controlled manner. The observation that some language students who obtain good marks in school do poorly in conversational settings, while some students who obtain poor marks communicate quite fluently (albeit not necessarily very accurately) drew attention to the implicit linguistic competence/explicit metalinguistic knowledge distinction.

Clinically a double dissociation is observed between amnesic and aphasic patients. Anterograde amnesic patients are unable to acquire new knowledge, in fact to remember anything of which they have been conscious since the onset of their condition. They cannot remember where they parked their car or what their new address is if they have moved. They cannot learn new words or remember the names of new acquaintances (or remember having seen them before) or new place names (like the name of their new hospital or town). Yet, they are able to acquire new motor or cognitive skills and show the same improvement with practice as normals, without ever being aware of having encountered the task before. In other words, their declarative memory (knowing that) is impaired but their procedural memory (knowing how) is intact. Aphasic patients, on the other hand, have deficits in the procedural memory system that subserves their language competence (a cognitive skill) but have no problem with declarative memory. Implicit competence is represented in those cortical areas that were active in their acquisition. Thus implicit linguistic competence is represented in the perisylvian area long identified as the "language area" including Broca's area in the frontal lobe, and Wernicke's area in the temporal and parietal lobes of the left hemisphere. Declarative knowledge, of which explicit metalinguistic knowledge is a part, is bilaterally represented in large areas of associative cortex. The metalinguistic rules of pedagogical grammar, like most overlearned material, may in fact be represented preponderantly in the right hemisphere.

The amnesic patient's difficulty with learning new words has highlighted a distinction between morphosyntax and vocabulary. While morphosyntax (as well as phonology) is implicit, vocabulary is to a large extent explicit: speakers consciously know the sound and the meaning of words and can produce either on demand-something they cannot do about the algorithms that underlay morphosyntax or phonology (professional linguists themselves continue to disagree on the form of linguistic representations, a testimony to the opacity of implicit linguistic competence). Lexical access and the automatic insertion of lexical items in the course of the microgenesis of an utterance produced under normal circumstances is equally unavailable to awareness. In addition, there is a clear dissociation between phonology and morphosyntax on the one hand and vocabulary on the other in individuals with genetic dysphasia (Paradis & Gopnik, in press), in children who are not exposed to language until seven (Lebrun, 1978) or thirteen (Curtis, 1977) years of age, for whom the acquisition of implicit grammar is arduous, whereas vocabulary expansion is relatively easy.

The declarative and procedural memory systems are not only neurofunctionally distinct, but involve different subcortical neural structures. The acquisition of declarative memory relies crucially on the integrity of the hippocampal system whereas procedural memory engages other subcortical structures, such as the basal ganglia (Butters, Salmon & Heindel, 1994; Dubois, Malapani, Verin, Rogelet, Deweer & Pillon, 1994), the striatum, as well as the cerebellum (Leiner, Leiner & Dow, 1991; Ito, 1993). Both memory systems depend upon cortical and subcortical structures, but different ones.

These observations led to a reconsideration of the selective or differential paradoxical recovery of some bilingual aphasic patients who had recovered their least known language over their previously fluent native language. It may well be the case that patients who have no longer access to the procedural memory system underlying linguistic competence for both their languages have nevertheless retained access to their declarative metalinguistic knowledge which may be more extensive in their formally learned second language. This may also explain the observed better prognosis that is generally correlated with a higher level of education in unilingual speakers. One may speculate that metalinguistic knowledge should still be available (or taught) to aphasic patients and thus compensate for their lack of access to their implicit linguistic competence, in the way that individuals with genetic dysphasia and some second language learners do.

Conclusions

What applies to the bilingual brain also applies to the unilingual brain: there are no qualitative differences. Individuals find themselves on a continuum from several registers in a unidialectal speaker to bidialectal speakers, to speakers of closely related languages, to speakers of unrelated languages. All use the same cerebral mechanisms, albeit to differing extents.

When the procedural memory system for language is genetically impaired or when the system has not been engaged during the time of its normal development (i.e., between the ages of 2 and 5), speakers compensate (for their lack of competence in their first or second language) from two sources: right-hemisphere based pragmatic competence and metalinguistic knowledge. Unilingual individuals with acquired aphasia should have the same options.

The evidence points to a neurofunctional modular system for language representation, with specific neuroanatomical substrates, irrespective of the number of languages stored in the brain. Differences between cerebral processes involved in language representation and use in unilinguals and different types of bilinguals appear to be only quantitative, as speakers of a second, weaker language may rely to a greater extent on explicit metalinguistic knowledge and pragmatic features to compensate for lacunae in their implicit linguistic competence. What is represented may differ, how it is processed does not. However, if the results reported by Weber-Fox and Neville (1996) are confirmed, namely, that bilingual individuals after the age of six or seven process functional items as lexical items, then it may be that later acquirers of a second language, like individuals with genetic dysphasia (Paradis & Gopnik, in press), do in part process language in a qualitatively different manner. A distinction will then be necessary between bilinguals -that is, early bilinguals- and fluent speakers of a second language.

Note

1 Dyshyponoia: From the Greek hypohoō (to grasp what is "understood" in an utterance, albeit unsaid, in the sense of the French "sous-entendu", Spanish "sobrentendido", Catalan "sobreentès").

Impairment of the use of linguistic pragmatics (e.g., the inability to draw correct inferences from the context or from general knowledge, leading to problems in the interpretation of indirect speech acts, metaphors, and in general of the unsaid component of an utterance).

References

Albert, M.L. & Obler, L.K. 1978. The bilingual brain. New York: Academic Press.

Bergh, G. 1986. The neuropsychological status of Swedish-English subsidiary bilinguals. Goteborg: Acta Universitatis Gothoburgensis

Bloom, L. 1974. Talking, understanding, and thinking. In R. L. Schieffelbusch and L. L. Lloyd (pp. 285-311). Language perspectives-acquisition, retardation, and intervention. Baltimore: University Park Press.

Butters, N., Salmon, D., & Heindel, W.C. 1994. Specificity of the memory deficits associated with basal ganglia dysfunction. Revue neurologique, 150, 580-587.

Colbourn, 1978. Can laterality be measured? Neuropsychologia, 16,283-289.

Dubois, B., Malapani, C., Verin, M., Rogelet, P., Deweer, B., & Pillon, B. 1994. Fonctions cognitives et noyaux gris centraux: Le modele de la maladie de Parkinson, Revue neurologique, 150, 763-770.

Green, D.W. 1986. Control, activation and resource: a framework and a model for the control of speech in bilinguals. Brain and Language, 27, 210-223.

Grosjean, F. & Soares, C. 1986. Processing mixed language: some preliminary findings. In J. Vaid (ed.), Language processing in bilinguals.(pp. 145-179).

Hillsdale, NJ: LEA.

Ito, M. 1993. New concepts in cerebellar function. Revue neurologique, 149, 596-599.

Leiner, H., Leiner, A.L., & Dow, R.S. 1991. The human cerebro-cerebellar system: its computing, cognitive, and language skills. Behavioral Brain Research, 44, 113-128.

Paradis, M. 1987a. The assessment of bilingual aphasia. Hillsdale, NJ: Lawrence Erlbaum Associates.

Paradis, M. 1987b. The neurofunctional modularity of cognitive skills: Evidence from Japanese alexia and bilingual aphasia. In E. Keller & M. Gopnik (eds.), Motor and sensory processes of language (pp. 277-289) . Hillsdale, NJ.: Lawrence Erlbaum Associates.

Paradis, M. 1994a. Toward a neurolinguistic theory of simultaneous translation: The framework. International Journal of Psycholinguistics, 10, 319-335.

Paradis, M. 1994b. Neurolinguistic aspects of implicit and explicit memory: Implications for bilingualism and LSA. In N. Ellis (ed.), Implicit and explicit learning of languages (pp. 393-419). London: Academic Press.

Paradis, M. & Gopnik, M. (in press). Compensatory strategies in genetic dysphasia Journal of Neurolinguistics, Pitres, A. 1895. Etude sur l'aphasie des polyglottes. Revue de medecine, 15, 873-899.

Satz, P. 1977. Laterality tests: an inferential problem. Cortex, 13, 208-212.

Vaid, J. & Hall, D.G. 1991. Neuropsychological perspectives on bilingualism: Right, left, and center. In A. Reynolds (ed.), Bilingualism, multiculturalism and second language learning (81-112). Hillsdale, NJ.: Lawrence Erlbaum Associates.

Weber-Fox, C. & Neville, H. 1996. Maturational constraints on functional specializations for language processing: ERP and behavioral evidence in bilingual speakers. Journal of Cognitive Neuroscience, 8, 231-256.

How and where are words represented and processed in the brain?

Friedemann Pulvermüller
Universität Tübingen, Germany

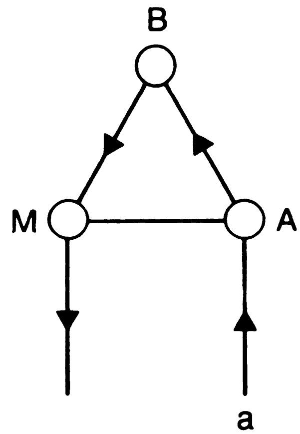

Figure 1: Lichtheim's model of word representation. Separate and autonomous representations of the articulatory form (in motor center M), sound pattern (acoustic center A) and meaning (concept center B - German "Begriff" means "concept") are postulated. Adopted from Ref. 2.

The problem of language and the brain catches the attention of neurologists, psychologists and linguists since the second half of the 19th century, when Broca [1] published his seminal description of language loss due to brain lesion (aphasia). It was in these early years of the language-and-brain sciences when a simple model of cortical language mechanisms was proposed. This model posits that two small centers in the left hemisphere of typical right-handed individuals are the "seat" of word representations (Figure 1) [2,3]. More precisely, a motor language center housed in the left inferior frontal lobe (areas 44 and 45, see Figure 2) was believed to store articulatory plans of words, and a separate acoustic language center in the left superior temporal lobe (area 22) was believed to house the sound patterns of words. Although the exact definition of these "language centers" somewhat varies between different authors[4] they are usually localized close to the sylvian fissure and are, therefore, part of the "perisylvian" areas.

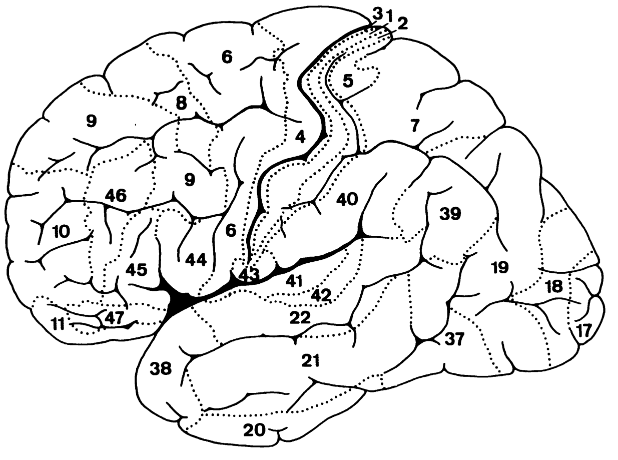

Figure 2: Lateral view of the left cortical hemisphere. Brodmann's areas are indicated. Adopted from Ref. 52.

This narrow localizationist view was subject to some criticism already before the turn of the century, which was formulated, for example, by a famous psychoanalyst who did some brain science in his early career. Based on theoretical considerations, this researcher claimed that processing of individual words should involve not only the two small perisylvian centers in the left hemisphere, but additional widespread cortical areas that are, for example, essential for visual perception. According to this author, there are not two separate brain-internal representations of articulatory plans and sound patterns of words, but, instead, a widely distributed neuron network would represent the articulatory and acoustic word form together with its meaning. Figure 3 presents a sketch of such a network.

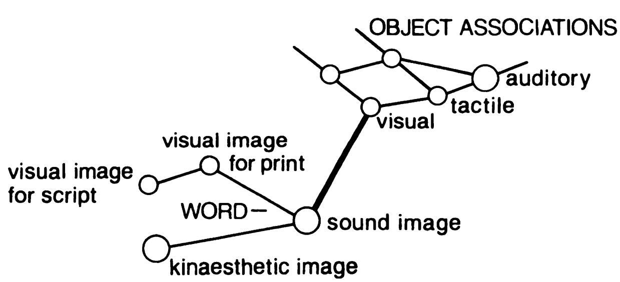

Figure 3: Freud's model of word representation. A widely distributed network is assumed to represent the various aspects of a word (articulatory and acoustic pattern, semantic properties). Lesion anywhere in the network may impair its function. Adopted from Ref. 15.

Are there arguments that would support one or the other view- either the narrow localizationist view of Wernicke and Lichtheim, or the holistic view put forward by Freud? According to what is known from aphasia research, lesions of Broca's area and adjacent areas in the inferior frontal lobe of the language-dominant hemisphere lead to motor aphasia characterized by severe deficits in producing speech, and lesions in Wernicke's area in the superior temporal gyrus causes sensory aphasia, for which a deficit in understanding language is most characteristic. These facts appear to speak in favor of the localizationist model.

However, after lesion in Wernicke's area additional deficits in speech production can be observed, and after lesion in Broca's area the patient usually exhibits additional problems in comprehending sentences. Although there are a few cases in the literature for whom it has been claimed that there is a language production deficit without any deficit in language comprehension[5], a test of language comprehension (and short-term verbal memory), the so-called Token Test[6], is usually clinically used for aphasia diagnosis. Thus, it appears that the large majority of aphasics, even those who have one intact language area, exhibit deficits in both language production and comprehension, although one of these deficits may be more pronounced than the other. This fact can only be explained if both language areas are assumed to contribute to both language production and comprehension, a fact which obviously speaks against the narrow localizationist approach and supports the holistic view[7]. However, one may nevertheless object against the holistic approach that probably not all cortical areas are equally involved in word processing, and that the areas involved may not be the same for different parts of speech. In summary, the truth appears to lie in-between the classical localizationistic and holistic views. A brain-theoretical framework is necessary in order to allow for more specific postulates.

One of the most important neuropsychologists of this century, Donald Hebb[8], proposed a brain-theoretical framework that may be of particular relevance for language representation and processing. Hebb assumes that the cortex is an associative memory machine and the strength of connections between cortical neurons depends on how frequently these neurons have been co-activated in the past. Meanwhile, there is strong evidence for this view from both neuroanatomy[9,10] and neurophysiology, although the original Hebbian ideas about learning principles had to be modified based on physiological data[11] and computational considerations[12]. If several neurons are frequently active at the same time, they will acquire strong connections to each other and, therefore, this "cell assembly" will act as a functional unit: If only some of its neurons are being activated by external input, activity will automatically spread throughout the assembly so that all of its members will be active. This explosion-like process has been called ignition of the assembly[9]. Furthermore, if an assembly has ignited, neuronal activity will probably not cease at once, but will reverberate for some time in the various neuronal loops within the assembly[13,14]. Thus, ignition and reverberation appear to be important processes occurring in strongly connected cell assemblies.

Words may be organized in the cortex as strongly connected cell assemblies

If Hebb is correct, simultaneous neuronal activity should be the basic brain principle underlying the formation of cortical representations (cell assemblies). What would this mean for language representation and processing?

If a word is articulated by the infant, neuronal activity controlling articulatory movements is present in the inferior frontal lobe. In addition, neurons in auditory cortices in the superior temporal lobe will be stimulated by the self-produced acoustic signal. If I talk, I also hear myself talking and this necessarily leads to simultaneous neuronal activity in inferior frontal and superior temporal cortices (Broca's and Wernicke's areas). Therefore, in this case Hebb would advocate the Freudian opinion rejecting separate cortical representations of articulatory programs and sound patterns, and emphasizing that cell assemblies distributed over motor and sensory regions should form the neuronal counterpart of word forms[15-17]. Figure 4 presents a sketch of such a perisylvian assembly. Although the existence of such assemblies cannot be proven in humans for ethical reasons, the recent discovery of "mirror neurons" in monkeys' frontal lobes that fire in relation to both hand movements and perceptions of such movements appear to support the view that motor and sensory patterns are not separately stored in cortex but are, instead, bound together in sensory-motor cell assemblies[18].

word form representation

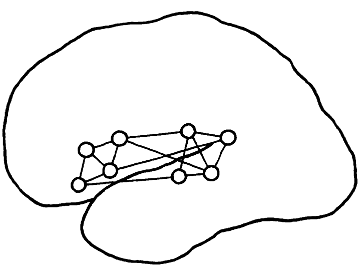

Figure 4: The cell assembly representing a phonological word form may be distributed over perisylvian areas. Circles represent local neuron clusters and lines represent bidirectional fiber bundles between such clusters. The connections are assumed to have strengthened because of correlated activity of neurons during articulation of the word form.

How would the meaning of words be stored in cortex? Associative learning is probably one of the important processes that may occur during acquisition of word meanings. A certain word may frequently be heard when a certain object is being visually perceived, or when the language-learning infant performs certain actions, or when it smells a certain smell, hears certain sounds or has some other perceptions. Therefore, when word forms become meaningful neurons in the perisylvian language areas and neurons located outside these areas, probably in various sensory and motor cortices and also in higher association cortices, are activated at the same time. According to Hebb, these neurons will strengthen their mutual connections and will develop into a cell assembly that comprises neurons in the language areas and outside.

So far, it appears that, from a modern perspective, Freud's approach to language representation was correct. However, not all words are the same, and for certain word classes the Freudian assumptions are most likely incorrect[7]. There are, for example, words with highly abstract meaning that primarily serve a grammatical function. For these grammatical function words (including pronouns, articles, auxiliary verbs, conjunctions etc.) a representation in widely distributed cell assemblies appears unlikely, because for these words there is no strong correlation between the occurrences of the word form and non-linguistic stimuli or actions. Therefore, grammatical function words should be cortically represented by cell assemblies restricted to the perisylvian areas.

It is well-known that language is localized to the left hemisphere. However, the Hebbian approach suggests that laterality of language is not complete but gradual. If I hear myself say a word, neurons in both hemispheres are necessarily activated at the same time and, according to the associative learning principle, the co-activated neurons in both hemispheres should become part of the assembly representing the word form[19]. Laterality of language may therefore mean that more neurons in the left hemisphere are included in the assemblies than neurons in the right hemisphere. If word meanings are being associated with word forms, the lateralized assembly representing the word form is probably activated together with neurons in both hemispheres, because the perception of a visual stimulus (or the execution of motor programs) will most likely lead to activation of similar numbers of neurons in both hemispheres. Therefore, meaning association should reduce the laterality of word representations. Cell assemblies representing nouns or verbs and other so-called "content words" should be less strongly lateralized to the left than assemblies representing grammatical function words [7,20].

More fine-grained word class-distinctions are desirable based on the Hebbian approach. Some words refer to objects that can be visually perceived, others refer to actions that are usually performed by the own body, and even other words refer to sounds, tastes, somatosensory perceptions etc. According to the modality through which meaning-related information is being transmitted, these word categories can be called "visual words", "motor words", and so on. If Hebb is correct, the cortical distribution of the assembly is a consequence of simultaneous activity occurring in different areas. This implies that a word frequently perceived together with certain visual stimuli (a likely event during learning of words referring to objects) has a cortical assembly quite different from the assembly representing a motor word (which may frequently co-occur with certain movements of the own body). Most nouns with well-imaginable meaning probably are visual words whose assemblies include additional neurons in visual cortices, whereas many action verbs are motor words whose assemblies may include additional neurons in motor cortices, and some nouns, such as tool names, may be considered a mixed category (motor and visual) from this point of view [21]. These modality-distinctions are, however, not the only ones suggested by the Hebbian approach. Because of the somatotopic organization of the motor cortex, words referring to foot movements (to kick) should include neurons in more dorsal motor cortices than words referring to hand movements (to write), and "semantic neurons" of words related to movements involving only a few muscles (to knock) may have a more narrow localization compared to those of words related to complex body movements (to caress). Similar more fine-grained distinctions are, of course, possible for visual words [21,22] and for words whose semantics are anchored in other modalities.

To make these ideas more plastic, Figure 5 presents sketches of possible cortical counterpart of function words, motor words and visual words, respectively. In addition to differences in the language-dominant left hemisphere, a strong degree of laterality can be assumed for function word assemblies and a reduced laterality degree for the other assembly types.

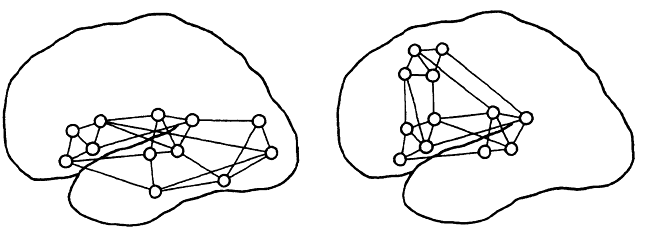

Figure 5: Grammatical function words (pronouns, articles etc.), and words referring to objects and actions may have different neuronal counterparts. A function word may be cortically represented by a perisylvian assembly (see Fig. 4). Words referring to objects usually perceived visually ("visual words") may be organized in assemblies distributed over perisylvian and additional visual cortices (A), and words that usually refer to movements of the own body ("motor words") may be organized in assemblies distributed over perisylvian and additional motor cortices (B). Many (but not all) concrete nouns are visual words and many action verbs are motor words.

Processing of different word categories involves different cortical areas

Starting with the considerations offered by Freud [15], there were numerous studies investigating language deficits arising from lesions outside the perisylvian language areas, some of which proved that word categories were selectively affected by lesions in areas outside the perisylvian regions [21-26]. This lesion evidence can, in part, be interpreted as empirical support for the Hebbian perspective outlined above [7,27]. However, the Hebbian ideas can also be tested in psychophysiological investigations of word processing in the intact human brain. Physiological studies can use various imaging techniques based either on direct measures of activity signs caused by electrophysiological activity in neurons (electroencephalography (EEG), magnetoencephalography (MEG), event-related potentials (ERP)), or on indirect measures of metabolic changes probably related to neuronal activity (positron emission tomography (PET), functional magnetic resonance imaging (fMRI)).

Electrocortical differences between content and function words have been found by several ERP studies [28-31]. A finding which was present in all studies - or at least in those using large electrode arrays (> 20, sometimes 64 and more electrodes) - was the following: Function words led to lateralized event-related potentials, whereas the potentials caused by presentation of content words were more symmetrical over the hemispheres. This is consistent with the idea of lateralized assemblies representing function words and less lateralized assemblies underlying processing of content words [27].

Numerous metabolic imaging studies have looked at processing of nouns and verbs. In most of these studies, the so-called "verb generation task" was used, that is, subjects were requirec to say aloud (or think of silently) a verb semantically related to a noun presented acoustica' or visually. The brain activity pattern obtained during verb generation was usually compare the activity pattern while reading or repeating (silently or aloud) the same nouns. Resu these experiments are highly heterogeneous. However, taken together, enhanced meta during verb generation was found not only in Broca's and Wernicke's areas, but, in ad adjacent prefrontal and temporal areas and sometimes in both hemispheres [32-35]. Th taken as evidence that verb processing involves perisylvian language areas and cort However, it has been argued that verb generation and repeating nouns are tasks only with regard to the words being relevant. Therefore, other psychological pro attention, search and judgement processes etc.) may be related to the ob differences.

In studies of electrocortical noun/verb differences in the intact brain, b presented in the same tasks, for example lexical decision, where sub whether letter strings are meaningful words or meaningless pseudo revealed word category differences in event-related potentials (ERPs) data to current source density analysis, a method for enhancing the cortical generators to the electrocortical signal, event-related potentials of cortical activity at central recording sites - over motor and premoto words (action verbs) were being processes, whereas activity signs wer recording sites - over visual cortices - when visual words (nouns with were processed. This pattern of results provides support for the Heb motor words are represented and processed differently in the corte

One may argue, however, that nouns and verbs do not only semantic properties, they also belong to different lexical cate differences observed may, therefore, be related to lexical rather th is certainly an important point, however, the assumption of th relevant can explain why differences in electrocortical activity imaginable nouns were present over visual and motor cortices, a present interpretation. Furthermore, more recent imaging wo brain metabolism between animal names and tool names w results. Most animal names belong to the category of visual learned (by most individuals in the western culture) based on ir whereas tool names probably elicit not only visual associ

e ca aro neu bein show althou respon explain for this nhancem nulated and n tem co subjects of the body movements involved when using the tools. Processing of tool names in a naming task led to activation of premotor cortices in frontal lobe, whereas processing of animal names in the same task enhanced metabolism in visual cortices in the occipital lobe [38]. (In the case of tool naming, an additional focus of activity enhancement was present more posteriorly in the middle temporal gyrus which may be related to associations of visually perceived movements related to tool usage or to imagination of their shapes [24,38].) These data provide additional evidence for the view that words with motor and visual associations are represented differently in the intact brain, and that they involve areas outside the classical language areas that reflect semantic word properties.

Reverberation of neuronal activity in cell assemblies representing words may be reflected in high-frequency cortical responses

The cell assembly concept is a tool for theorizing about cortical representations. It is difficult to actually prove that cell assemblies exist in cortex, and it is even more difficult to provide a roof that they are the basis of cognitive processing, as suggested by Hebb. However, recent idence from neurophysiology demonstrates that multiple neurons in various cortical areas n exhibit synchronous rhythmic activity patterns in a rather high frequency range, that is und 30 Hz and above [39]. High-frequency activity is stimulus-specific, that is, particular on sets may synchronize their rhythmic responses when a particular visual stimulus is presented, whereas other neurons become synchronized when a different stimulus is n. This kind of synchrony in cortex is only possible if cortico-cortical fibers are intact, gh subcortical connections may play an additional role in synchronizing cortical ses [40]. Stimulus-specific synchronous high-frequency activity in cortex is difficult to without using the cell assembly concept, and may, therefore, be considered as evidence notion. If reveberation of neuronal activity in cortical cell assemblies causes nent of well-timed high-frequency responses in these neurons, some of the ideas d above can be experimentally tested. For non-invasive recordings of such responses, 1EG-mapping are necessary, because only these recording techniques have the fine-poral resolution in the millisecond range necessary for recording high-frequency tex.

that reverberation of neuronal activity in cell assemblies is visible in high-ponses one would predict that these responses are stronger when a cognitive is being activated compared to a state in which no such representation is being ding to the Hebbian view, words are represented in cortical assemblies while udowords, such as "noom", lack a cortical representation because they have ned. This predicts stronger high-frequency cortical responses to words udowords ("moon" vs. "noom"). In a series of experiments, we obtained empirical support for this prediction [41-44]. EEG and MEG responses to words and pseudowords presented acoustically or visually consistently revealed differences in spectral responses in the 30 Hz-range. Importantly, no similar differences were present in lower parts of the spectrum (alpha-band around 10 Hz) or in the higher spectrum, where muscle activity would be most strongly visible. Differences in high-frequency responses were most pronounced and significant at recording sites above the language cortices in the left hemisphere of right-handed experiment participants. These results are consistent with the view that cell assemblies exhibiting well-timed reverberation of neuronal activity with a predominant frequency around 30 Hz become active when words are being processed but fail to ignite after presentation of meaningless pseudowords. Similar dynamics of 30 Hz-responses have recently been reported from a comparison of meaningful visual gestalts vs. physically similar but meaningless visual stimuli that are not perceived as a coherent gestalt [45]. All of these findings support the view that meaningful elements (words, gestalts) - but not similar meaningless stimuli - activate cell assemblies generating 30 Hz-activity.

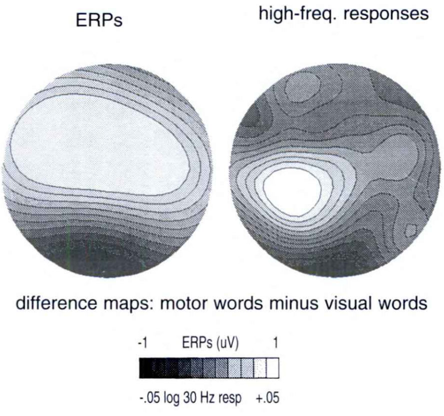

Figure 6: Processing of motor words (verbs) and visual words (nouns) is accompanied by significantly different electrocortical responses. Difference maps (nouns minus verbs) are shown. Large circles represent the head seen from above (anterior is up). Verbs elicit stronger signs of activity over motor cortices of both hemispheres, whereas nouns elicit stronger signs of activity over visual cortices. Differential topographies of event-related potencials are compared to evoked spectral responses in the frequency range 25-35 Hz. Adopted from Ref. 27.

The Hebbian cell assembly perspective would, however, allow for even more specific predictions. For example, processing of words with different meanings, such as motor and visual words, should not only induce different global activity in motor and visual cortices, but, in additon, there should be a specific change of high-frequency activity in the same cortical areas. This prediction was tested in a recent experiment again using action verbs and nouns with well-imaginable visual meaning [44]. Significant differences in 30 Hz EEG responses were present over motor cortices, and additional differences were seen over occipital visual areas (recording sites C3/C4 vs. O1/O2 of the international 10/20-system). High-frequency responses to motor words were stronger over motor cortices, whereas they were stronger over visual cortices for visual words. This provides another piece of evidence for the Hebbian perspective on language representation in the brain.

It may, however, be claimed that differential high-frequency responses are not necessarily a sign of cell assembly ignition and reverberation of neuronal activity therein. More global neuron loops may also generate high-frequency activity, as has been made evident by recordings in arthropods [46] and in the retina of vertebrates [47]. One may, therefore, claim that differential high-frequency cortical responses can be a consequence of various cortical activation processes. However, it is important to note that there is at all any difference between high-frequency responses to physically similar meaningful and meaningless elements, to words and pseudowords, to gestalts and pseudogestalts, to nouns and verbs. This can only be explained if high-frequency responses are interpreted as a consequence of the activation of cortical representations that depend on the meaning (or gestalt properties) of stimuli. Furthermore, if dynamics in 30 Hz responses were a sign of global changes of cortical activity in cortical areas, their spatio-temporal properties should be the same as for other global activity indicators such as event-related potentials. This, however, is clearly not the case [27]. At this point, more experimental work is necessary in order to decide whether 30 Hz-responses actually reflect fast reverberation of neuronal activity within cell assemblies or reverberation processes caused by activation of cognitive cortical representations (ignition of cell assemblies) but involving additional neurons outside the representation (assembly).

The Hebbian approach to language representation in the brain may provide biological answers to additional questions from language science

These results provide support for the claim that words of different semantic classes are represented in cell assemblies with different cortical distributions. All of these assemblies appear to have some of their neurons located in the perisylvian language areas of Broca and Wernicke, and some words may be represented by assemblies including additional neurons outside the language areas, and possibly in both hemispheres. Semantic word properties appear to be reflected in the additional areas becoming relevant. Evidence for different distributions of cell assemblies can be obtained from global activity measures such as provided by metabolic or neurophysiological imaging techniques, and, in addition, important clues about reverberating neuronal activity in cell assemblies (or related to cell assembly activation) may come from investigations of high-frequency cortical responses recorded in the EEG and MEG.

It should be emphasized that the Hebbian model put forward here is related to large-scale neuronal theories of language that are based on Hebbian associationist learning principles [16,22,48,49]. All these models have in common that 1) widely distributed neuron sets in cortex (and additional subcortical structures) are assumed to be the substrate of language processing, and that 2) such assemblies are assumed to form as a consequence of associative learning. Distinctive features of the approach discussed above include the assumptions (i) that processing of an individual word (and of any meaningful stimulus) does not only lead to the activation of cortical areas, but rather to the activation of a distinct neuron set, a cell assembly representing the individual meaningful element, (ii) that phonological, semantic and other features of a word are bound together in its neuronal representation so that stimulation of the assembly leads to almost simultaneous activation of the word representation implying simultaneous access to all of its features on the cognitive level, (iii) that two distinct processes, ignition and reverberation of neuronal activity, follow stimulation of an assembly, and (iv) that right-hemispheric processes are involved in word processing and that right-hemispheric processes are different for words of different classes. Claim (i) is primarily motivated by theoretical considerations, but the finding that there are cortical neurons specifically activated by low-frequency words provides support for this assumption [50]. Claim (ii) is supported by early electrocortical differences between vocabulary types which were present as early as around 200 ms after stimulus onset not only over perisylvian areas, but, in addition, over motor and visual areas probably involved in processing of word meanings. Assumption (iii) is consistent with the finding that word-class differences in event-related potentials (possibly indicating differences in ignition) occurred shortly after stimulus onset (around 200 ms) whereas dynamics in high-frequency responses (possibly related to reverberation) usually occurred only later. Finally, assumption (iv) is supported by studies evidencing a) different degrees of laterality of electrocortical activity elicited by words of different classes and b) word class-specific activity differences in the right hemisphere.

From a linguistic point of view, however, the question addressed above - the question of the cortical organization of words of different classes - is only a very basic one, and it is absolutely clear that neurobiological models cannot, at this point, answer complex questions about the brain mechanisms that govern the sequencing of words in sentences and the sequencing of speech acts in complex dialogues. Whereas some sequencing rules may be biologically realized as connections between cell assemblies that form based on associative learning principles, genetically programmed information may be necessary for other syntactic mechanisms [51]. Specification of these mechanisms in terms of neurons and cell assemblies appears to be one of the most exciting goals in cognitive neuroscience.

References

1. Broca, P. (1861). Remarques sur la sige de la faculté de la parole articulée, suivies d'une observation d'aphémie (perte de parole). Bulletin de la Société d'Anatomie 36, 330-357.
2. Lichtheim, L. (1885). Ueber Aphasie. Deutsches Archiv für Klinische Medicin 36, 204-268.

3. Wernicke, C. (1874). Der aphasische Symptomencomplex. Eine psychologische Studie auf anatomischer Basis. Breslau: Kohn und Weigert.
4. Bogen, J.E. & Bogen, G.M. (1976). Wernicke's region - where is it? Annals of the New York Academy of Sciences 280, 834-843.
5. Kolk, H.H.J., Grunsven, J.F.van & Keyser, A. (1985). On parallelism between production and comprehension in agrammatism. In Agrammatism, ed. Kean, M.-L., pp. 165-206. New York: Academic Press.
6. De Renzi, E. & Vignolo, L. (1962). The Token Test: a sensitive test to detect receptive disturbances in aphasics. Brain 85, 665-678.
7. Pulvermueller, F. (1992). Constituents of a neurological theory of language. Concepts in Neuroscience 3, 157-200.
8. Hebb, D.O. (1949). The organization of behavior. A neuropsychological theory. New York: John Wiley.
9. Braitenberg, V. (1978). Cell assemblies in the cerebral cortex. In Theoretical approaches to complex systems. (Lecture notes in biomathematics, vol. 21), eds. Heim, R. & Palm, G., pp. 171-188. Berlin: Springer.
10. Braitenberg, V. & Schuez, A. (1991). Anatomy of the cortex. Statistics and geometry. Berlin: Springer.
11. Singer, W. (1995). Development and plasticity of cortical processing architectures. Science 270, 758-764.
12. Palm, G. & Sommer, F.T. (1995). Associative data storage and retrieval in neural networks. In Models of neural networks III, eds. Domany, E., van Hemmen, J.L. & Schulten, K., pp. 79-118. New York: Springer Verlag.
13. Abeles, M., Bergman, H., Margalit, E. & Vaadia, E. (1993). Spatiotemporal firing patterns in the frontal cortex of behaving monkeys. Journal of Neurophysiology 70, 1629-1638.
14. Fuster, J.M. (1994). Memory in the cerebral cortex. An empirical approach to neural networks in the human and nonhuman primate. Cambridge, MA: MIT Press.
15. Freud, S. (1891). Zur Auffassung der Aphasien. Leipzig, Wien: Franz Deuticke.
16. Braitenberg, V. (1980). Alcune considerazione sui meccanismi cerebrali del linguaggio. In L'accostamento interdisciplinare allo studio del linguaggio, eds. Braga, G., Braitenberg, V., Cipolli, C., Coseriu, E., Crespi-Reghizzi, S., Mehler, J. & Titone, R., pp. 96-108. Milano: Franco Angeli Editore.
17. Braitenberg, V. & Pulvermueller, F. (1992). Entwurf einer neurologischen Theorie der Sprache. Naturwissenschaften 79, 103-117.
18. Rizzolatti, G., Fadiga, L., Gallese, V. & Fogassi, L. (1996). Premotor cortex and the recognition of motor actions. Cognitive Brain Research 3, 131-141.
19. Mohr, B., Pulvermueller, F. & Zaidel, E. (1994). Lexical decision after left, right and bilateral presentation of content words, function words and non-words: evidence for interhemispheric interaction. Neuropsychologia 32, 105-124.

20. Pulvermueller, F. & Mohr, B. (1996). The concept oftranscortical cell assemblies: a key to the understanding of cortical lateralization and interhemispheric interaction. Neuroscience and Biobehavioral Reviews 20, (in press).
21. Warrington, E.K. & McCarthy, R.A. (1987). Categories of knowledge: further fractionations and an attempted integration. Brain 110, 1273-1296.
22. Damasio, A.R., Damasio, H., Tranel, D. & Brandt, J.P. (1990). Neural regionalization of knowledge access: preliminary evidence. In Cold Spring Harbour Symposia on Quantittative Biology. Vol. LV: the brain, Cold Spring Harbour: Cold Spring Harbour Laboratory Press.
23. Caramazza, A. & Hillis, A.E. (1991). Lexical organization of nouns and verbs in the brain. Nature 349, 788-790.
24. Damasio, H., Grabowski, T.J., Tranel, D., Hichwa, R.D. & Damasio, A.R. (1996). A neural basis for lexical retrieval. Nature 380, 499-505.
25. Daniele, A., Giustolisi, L., Silveri, M.C., Colosimo, C. & Gainotti, G. (1994). Evidence for a possible neuroanatomical basis for lexical processing of nouns and verbs. Neuropsychologia 32, 1325-1341.
26. Warrington, E.K. & Shallice, T. (1984). Category specific semantic impairments. Brain 107, 829-854.
27. Pulvermueller, F. (1996). Hebb's concept of cell assemblies and the psychophysiology of word processing. Psychophysiology 33, 317-333.
28. Garnsey, S.M. (1985). Function words and content words: reaction time and evoked potential measures of word recognition. Rochester, NY: University of Rochester.
29. Neville, H.J., Mills, D.L. & Lawson, D.S. (1992). Fractionating language: different neural subsystems with different sensitive periods. Cerebral Cortex 2, 244-258.
30. Nobre, A.C. & McCarthy, G. (1994). Language-related EPRs: scalp distributions and modulation by word type and semantic priming. Journal of Cognitive Neuroscience 6, 233-255.
31. Kreiter, A.K. & Singer, W. (1992). Oscillatory neuronal responses in the visual cortex of the awake macaque monkey. European Journal of Neuroscience 4, 369-375.
32. McCarthy, G., Blamire, A.M., Rothman, D.L., Gruetter, R. & Shulman, R.G. (1993). Echo-planar magnetic resonance imaging studies of frontal cortex activation during word generation in humans. Proceedings of the National Academy of Sciences,USA 90, 4952-4956.
33. Petersen, S., Fox, P., Posner, M., Mintun, M. & Raichle, M. (1989). Positron emission tomography studies of the processing of single words. Journal of Cognitive Neuroscience 1, 153-170.
34. Wise, R., Chollet, F., Hadar, U., Fiston, K., Hoffner, E. & Frackowiak, R. (1991). Distribution of cortical neural networks involved in word comprehension and word retrieval. Brain 114, 1803-1817.

35. Fiez, J.A., Raichle, M.E., Balota, D.A., Tallal, P. & Petersen, S.E. (1996). PET activation of posterior temporal regions during auditory word presentation and verb generation. Cerebral Cortex 6, 1-10.
36. Dehaene, S. (1995). Electrophysiological evidence for category-specific word processing in the normal human brain. NeuroReport 6, 2153-2157.
37. Preissl, H., Pulvermueller, F., Lutzenberger, W. &Birbaumer, N. (1995). Evoked potentials distinguish nouns from verbs. Neuroscience Letters 197, 81-83.
38. Martin, A., Wiggs, C.L., Ungerleider, L.G. & Haxby, J.V. (1996). Neural correlates of category-specific knowledge. Nature 379, 649-652.
39. Singer, W. & Gray, C.M. (1995). Visual feature integration and the temporal correlation hypothesis. Annual Review in Neuroscience 18, 555-586.
40. Steriade, M., Amzica, F. & Contreras, D. (1996). Synchronization of fast (30-40 Hz) spontaneous cortical rhythms during brain activation. Journal of Neuroscience 16, 392-417.
41. Lutzenberger, W., Pulvermueller, F. & Birbaumer, N. (1994). Words and pseudowords elicit distinct patterns of 30-Hz activity in humans. Neuroscience Letters 176, 115-118.
42. Pulvermueller, F., Eulitz, C., Pantev, C., Mohr, B., Feige, B., Lutzenberger, W., Elbert, T. & Birbaumer, N. (1996). High-frequency cortical responses reflect lexical processing: an MEG study. Electroencephalography and Clinical Neurophysiology 98, 76-85.
43. Pulvermueller, F., Preissl, H., Lutzenberger, W. & Birbaumer, N. (1995). Spectral responses in the gamma-band: physiological signs of higher cognitive processes? NeuroReport 6, 2057-2064.
44. Pulvermueller, F., Preissl, H., Lutzenberger, W. & Birbaumer, N. (1996). Brain rhythms of language: nouns versus verbs. European Journal of Neuroscience 8, 937-941.
45. Tallon, C., Bertrand, O., Bouchet, P. & Pernier, J. (1995). Gamma-range activity evoked by coherent visual stimuli in humans. European Journal of Neuroscience 7, 1285-1291.
46. Kirschfeld, K. (1992). Oscillations in the insect brain: do they correspond to the cortical gamma-waves of vertebrates? Proceedings of the National Academy of Sciences,USA 89, 4764-4768.
47. Neuenschwander, S. & Singer, W. (1996). Long-range synchronization of oscillatory light responses in the cat retina and lateral geniculate nucleus. Nature 379, 728-732.
48. Edelman, G.M. (1992). Bright air, brilliant fire: on the matter of the mind. New York: Basic Books.
49. Mesulam, M.M. (1990). Large-scale neurocognitive networks and distributed processing for attention, language, and memory. Annals of Neurology 28, 597-613.
50. Creutzfeldt, O., Ojemann, G. & Lettich, E. (1989). Neuronal activity in the human lateral temporal lobe. I. Responses to speech. Experimental Brain Research 77, 451-475.
51. Pulvermueller, F. (1994). Syntax und Hirnmechanismen. Perspektiven einer multidisziplinaeren Sprachwissenschaft. Kognitionswissenschaft 4, 17-31.
52. Pulvermueller, F. & Preissl, H. (1991). A cell assembly model of language. Network 2, 455-468.

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