Higher Nervous Functions

Preface

Foreword

This publication entitled "Higher Nervous Functions" is not a book on a specific subject compiled by several authors; it contains a collection of papers on various subject fields, which were presented at the Symposium on Higher Nervous Functions on the occasion of the 7th Asian Oceanian Congress of Neurology on September 22, 1987, in Bali, Indonesia.

Introduction

The term "Higher Nervous Functions" is here understood in contrast to reflexes and functions as processes taking place mainly on the subcortical level. Speaking, writing, concentration, memory functions and critical thinking are some of these higher functions. The concept of central nervous function, or the function of the nerves, has been complemented within the course of development by the noetic function, which is understood as an expression of a more complex activity on the cortical level.

In the search for effective methods of treating impaired central nervous function, which may have a variety of causes, e. g. hypoxia, metabolic disorders or lesions as sequelae of degenerative or vascular processes, we encounter a number of unsolved problems:

It is extremely difficult to diagnose impaired central nervous functions intra vitam, or to classify these disorders etiopathogenetically. As a resort, gerontopsychiatrists usually turn to symptoms (impaired cognitive function or mood disturbances), or to syndromes on which such symptom complexes are based, e. g. the organic brain syndrome or senile dementia. The anglo-american literature often uses the term dementia of the "Alzheimer-Type" without having positive evidence of such a diagnosis.

Where impairment of specific central nervous systems are concerned, the diagnostic instruments, on the neurophysiological as well as the neuropsychological level, have greatly improved for aphasia and agraphia. Several of the papers bear witness to this view.

The scientific discipline of neurology has tried to solve the problem by differentiating peripheral disorders and, based on the resulting diagnosis, deducing the localization of the impairment in the brain. The disciplines of gerontopsychiatry and gerontopsychology, on the other hand, have developed self-ratings and observer ratings as well as performance tests by means of which impaired brain function can be determined globally and quantified in such a way that they can be used for diagnosis and disease monitoring as well as for assessment of drug effects. This subject is treated in some of the papers.

Another aspect covers the neurobiochemical changes, mainly in the central cholinergic system, which may cause impairments of higher nervous functions. This so-called cholinergic hypothesis of senile dementia of the Alzheimer type is at present under intensive study, especially in view of possible substitution therapy with substances specifically activating the central cholinergic system.

May this compilation of papers help one discipline to learn from the other; may it also induce neuropsychiatrists and neuropsychologists – after they have succeeded in proving clear drug effects in clinical models (which are also plausible indicators of therapeutic efficacy) – to develop more differential instruments for diagnosis and disease monitoring, so that differential disorders may be assessed beyond an overall impression of improved cerebral performance. Impulses in this direction also became evident during the course of the symposium.

It would be desirable, for example, to be able to distinguish whether a drug acts via the affective or via the cognitive level. On the cognitive level, memory research is gaining increasing importance. It would also be necessary to clarify whether memory functions may be affected directly or whether they are influenced via a change in vigilance or motivation.

Neurologists, on the other hand, should in future try to carry out prospective clinical trials with a confirmatory objective and to demonstrate effects with the help of differential diagnostic instruments as have been described at this symposium. If the effect can be proven, the interpretative step of drawing conclusions on therapeutic efficacy and therapeutic relevance would not be as difficult in neurology as in gerontopsychiatry and gerontopsychology.

These papers should moreover help to convey to the clinical therapist as well as the researching pharmaceutical industry that improved therapeutic intervention making use of drugs is only possible if, in cooperation between pharmacologists, biochemists, clinical pharmacologists and physicians, higher effective substances are searched for, which are then tested with the help of new or further developed instrumentaria and new clinico-pharmacological and clinical models.

The development of drugs for the treatment of impaired brain function in the elderly – which today we call nootropics – is only sensible if the development of methods runs parallel from early pharmacology to clinical research. Highly developed pharmacological models will not lead to newer and more effective substances if the clinic is not prepared to cooperate and does not have the methods required to demonstrate differential effects in therapeutic reality.

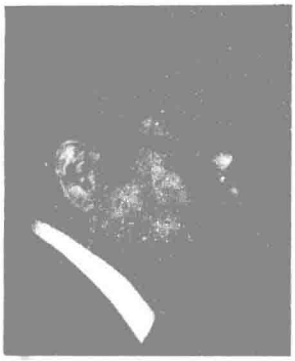
The prejudice that vascular and degenerative processes cannot be diagnosed safely intra vitam, or that a more differential diagnosis of dementia is bound to fail anyway, will not help progress. A negative attitude towards the pharmacotherapy of impaired brain function in general or the efficacy of nootropics in particular does not help either to clarify the benefits and risks of such drugs. Open-mindedness towards new methodological and therapeutic approaches, while maintaining a critical distance, is what is needed for this aim.

The course that this symposium has taken leads us to hope that an epoch of open-mindedness and critical argumentation may have begun, and that in the next decade we will be able to better judge the role of drugs in the therapeutic concept of impaired brain

function and dementia. However, this requires very detailed knowledge of special mechanisms of action of the various substances, as well as better possibilities of finding the etiopathogenetic cause of the impairment in each individual patient with the help of simple diagnostic methods. In the ideal case, this would permit the respective responder to be defined for each individual drug.

Problems can only be solved to the extent to which they can be defined. There is no doubt, however, that today we are able to define the problems associated with impaired brain function very much better than we were a decade ago, so that there is justified hope that more impulses of this kind are in store in the future.

W. M. Herrmann, M. D.
Professor of Psychophysiology



Opening Remarks

S. Sastrodiwirjo

Neurological Department, University of Indonesia, Jakarta, Indonesia.

Prof. Soemargo Sastrodiwirjo held the opening speech at the Symposium on Higher Nervous Functions. He pointed out that the knowledge about higher nervous functions (HNF) is still young, still developing, and has not yet received full attention. This is reflected by the number of papers on this subject presented at this congress. Great progress in the research of HNF was achieved with the discovery of the split brain which then became the basis for many research activities. In medical practice, Prof. Soemargo continued, HNF occupies an important place, and since disturbances of the higher nervous functions may cause complications in social and occupational life, early detection is necessary, not only of obvious symptoms such as dementia or aphasia, but also of less obvious symptoms, like mild aphasia, memory and concentration disturbances, learning disabilities and dyslexia. According to Prof. Soemargo it is evident that in daily practice all activities in the field of higher nervous functions should be done with a well coordinated team of specialists. This was taken into consideration in the arrangement of the Bali Symposium, where experts from other disciplines, such as neurologists, psychologists, psychiatrists, and speech pathologists, whose work is closely connected with higher nervous functions, were invited to take part as speakers.

Contents

Current Concepts of Aphasia

K. Poeck

Department of Neurology,
RWTH Aachen, F. R. Germany

Language is a system of signs or symbols and of rules that govern the concatenation of these symbols. The symbols, of course, are the words, i. e., content words and function words. The rules involve what we call syntax. If a patient does not produce well formed sentences, does not select his words appropriately and does not seem to understand his partner very well, it is suspected that this patient might have aphasia (Rose 1984).

However, not every patient who does not speak standard language or who speaks somewhat awkwardly has aphasia. The patient might have one of the many nonaphasic language disorders. For the clinician it is most important to distinguish aphasia from the various types of dysarthria, a group of motor disturbances of speech, as opposed to supramodal disturbances of language. Dysarthria is associated with functional impairment at any level where motor functions are organized in the brain. Table 1 lists nonaphasic language disorders and the various types of dysarthria (Huber et al. 1983).

Psychosis	
Dementia	
Tiredness	
Educational Deficit	
Cortical	Dysarthria
Basal Ganglia	Dysarthria
Cerebellar	Dysarthria
Bulbar	Dysarthria

Tab. 1
Non-Aphasic Language Disorders

Aphasia is brought about by dysfunction of the language area of the brain. Aphasia is a central disorder of language where all linguistic components are com-

prised. The deficit embraces all expressive and receptive modalities, that is, expressive speech and comprehension, reading as well as writing.

In a patient who might have an aphasic language disorder, an intuitive diagnosis can be tried. It is, however, much more reliable to assess his language performance by giving him a semistandardized interview and to apply a standardized test. The Boston Diagnostic Examination (Goodglass and Kaplan 1983) and the Western Aphasia Battery (Kertesz 1982) are very popular in English speaking countries. For the German speaking countries there exists a standardized aphasia test that meets modern psychometric requirements, the Aachen Aphasia Test (AAT, Huber et al. 1983, Huber et al. 1984). This test has been translated into English, Dutch and Italian and is therefore very well suited for comparative, cross-cultural studies. Table 2 gives the levels of speech that are assessed with this test.

Part of Test	Constituents	Scoring Per Scale / Item
1. Spontaneous Language	6 scales	0 – 5
2. Token Test	5 parts 10 items each	0 – 1
3. Subtest Repetition	5 parts 10 items each	0 – 3
4. Subtest Written Language	3 parts 10 items each	0 – 3
5. Subtest Confrontation Naming	4 parts 10 items each	0 – 3
6. Comprehension	4 parts 10 items each	0 – 3

Tab. 2
Aachen Aphasia Test

It will be easily recognized that these levels cannot be assessed by a necessarily superficial bedside examination. We have also developed a computer program for the diagnosis and classification of aphasia that complements the personal diagnosis by the examiner. We have fed the test results taken from 376

patients into a computer as a learning sample. The computer is now able to compare the actual test results of any given patient with the data of the learning sample. The computer program (ALLOC) confirms or rejects the presence of aphasia in the patient under study and furthermore allocates him/her to one of the different subtypes of aphasia (Huber et al. 1984, Willnes and Ratajczak 1987). Figure 1 shows a typical example. This patient was diagnosed by the computer as being 100 % aphasic with an 85,3 % probability of having Wernicke's aphasia.

Aachen aphasia test / Alloc-diagnosis
(Learning sample: 314 Aphasia, 100 Contr. / 90 GL, 74 WE, 79 BR, 71 AM)

Family name, first name:	B., G.	
Date of birth:	15/03/29	
Date of examination:	29/06/82	
Patient-identification:	881	2-nd Examination

AAT-Profile

Spontaneous speech (1-6)						TT	REP	WRIT	NAME	COMP
3	5	4	4	4	3	13	100	51	97	102

Classification

Diagnosis	Posterior-probabilities (in %)	
1. Aphasia	Aphasia: 100 %	no Aph.: 0.0 %
2. Syndrome Wernicke	GL: 0.0 %	WE: 85.8 %
	BR: 13.3 %	AM: 0.8 %

Fig. 1
ALLOC diagnosis of patient B. G., Wernicke's Aphasia

Any classification of aphasic patients must reveal doubtful cases which cannot be attributed to one of the well-known subtypes of aphasia. One should be very skeptical about an aphasia test that does not result in a certain amount of doubtful cases.

These considerations do not imply that we propose to rely on a computer diagnosis. We establish our diagnosis on the basis of an individual assessment made by the examiner which is then verified by the computer program. If there are discrepancies, the test results of this particular patient are discussed in the group of examiners, and experienced aphasiologists arrive at a concensus.

This consensus leads to the diagnosis of one of the standard aphasias, i. e., global, Broca's, Wernicke's or amnesic aphasia, or one of the nonstandard aphasias. These are conduction aphasia, transcortical, fluent and nonfluent aphasia. The latter two subgroups are supercategories that are not very useful because each of them includes several types of standard or nonstandard aphasia (Huber, Poeck and Weniger 1982). This classification is not very important for the localization of the underlying brain lesion which today is done by CT scan. It is important for the allocation of a given patient to a treatment group. Aphasia is not only a field of Brain and Behaviour Research. Aphasia is also a treatable condition and it will be shown below what the effects of aphasia treatment can be.

There is a strong tendency among certain psycholinguists in the United States and in Great Britain to globally condemn group studies. Group studies are said to be useless in view of the fact that each patient is different. These researchers do not recognize any scientific interest in regarding the features that groups of patients have in common. Instead, they propose to look exclusively at the traits that characterize the individual patient. Accordingly, in this line of research, single cases are described meticulously, certain phenomena under study are highlighted and the entirety of the aphasic syndrome is ignored.

One of the issues where single case studies have opened new avenues is deep dyslexia (Coltheart, Patterson and Marshall 1980) and other variants of acquired dyslexia. Researchers who study acquired dyslexia tend to completely ignore the brain lesion of their patients, and they also ignore to what extent the patients are aphasic, let alone what subtype of aphasia they present with. It is hard to see how the brain mechanisms underlying these syndromes can be elucidated by this approach. Also, the methods of examining these patients are in general no reliable, standardized tests. Rather, they are collections of tasks that are developed ad hoc for an individual patient and, consequently, differ from one patient to the other making comparisons across the published single cases very difficult.

On the basis of methods derived from group studies we are, at present, developing standardized tests to

assess the performance of single patients in a psychometrically reliable way (Poeck and Göddenhenrich 1988, see Table 3).

Multimodal Matching and Naming
Lexical Discrimination
Dyslexia
Understanding of Syntactic Constructions

Tab. 3
Supplements to the AAT

The supplements to the AAT include multimodal matching and naming, lexical discrimination, understanding of syntactic structures and a dyslexia test. We believe that standardized tests can – and, in fact have to – be used also for the examination of single patients. The procedure of psychometric single-case analysis (Huber 1973, Willmes 1985) permits comparison of the performance of one single patient with that of another single patient. Unless researchers apply standardized tests and appropriate statistical methods, it is difficult to believe in the heuristic value of single case studies.

Aphasia is a treatable condition. If aphasic patients are given language therapy, spontaneous recovery must, of course, be taken into account. It is a very important task to collect reliable spontaneous recovery data. There are not very many studies on spontaneous recovery on record, and many of them are in some way biassed.

We have run a Multicenter Aphasia Spontaneous Recovery Study on 96 patients who were comparable for all practical purposes (Willmes and Poeck 1984). These patients did not receive speech therapy during the period of the study, nor had they received speech therapy earlier. The main finding was a large average improvement within the first 3 to 4 months, i. e. for the first part of the study, after which time the recovery curve became flatter, almost horizontal. After 6 or 7 months post onset, there was no appreciable improvement anymore. It may be safely concluded from these data that the bulk of spontaneous recovery in aphasia occurs in the first 3 or 4 months, and that after 6, or at the most after 8 months, spontaneous recovery comes to an end.

We have used the detailed data of this study as a baseline for a treatment study. In our department, we have an aphasia ward where at any given time 12 aphasic patients receive inpatient aphasia therapy twice a day. One hour is devoted to individual therapy, one hour to group therapy. We have compared the results of this intensive therapy with the baseline data from the Multicenter Spontaneous Recovery Study. We were able to show that even taking into account spontaneous recovery, more than 60 % of the patients profited from intensive aphasia therapy (Poeck et al. 1988). It is understood that speech therapy twice a day for periods of 6 – 7 weeks is a very favourable condition, even more so in an inpatient setting. It could be demonstrated unequivocally, however, that aphasia therapy is effective.

Another line of research in aphasic patients has a long tradition in neurology, namely localization studies. The concept of distinct centers (Broca vs Wernicke) and the well-known diagrams produced e. g. by Lichtheim are not present day issues anymore. The introduction of CT scan seemed to facilitate modern localization studies and has, in fact, been the incentive for a wealth of anatomo-clinical studies. There are, however, some important drawbacks that do not permit easy conclusions on localization. CT scans as such do not permit scientific evaluation of the localization of the lesion because the conditions of imaging are too different. Brains are different, skulls are different and the starting level in CT scanning is different. Also one cannot feed the observed data into a computer, which is mandatory for group studies (de Bleser and Poeck 1984).

We have tried to overcome these handicaps by drawing the lesion of a given patient onto a standardized template where each point showing a lesion is given an entry of 1 and each point not showing a lesion is given an entry of 0. This yielded a binary matrix which could be worked up in a computer (De Bleser and Poeck 1984, Willmes and Ratajczak 1987).

On the basis of these data simple problems, such as, for instance, the localization of the lesion common to all Broca's aphasics, can be assessed (Figures 2a, 2b).

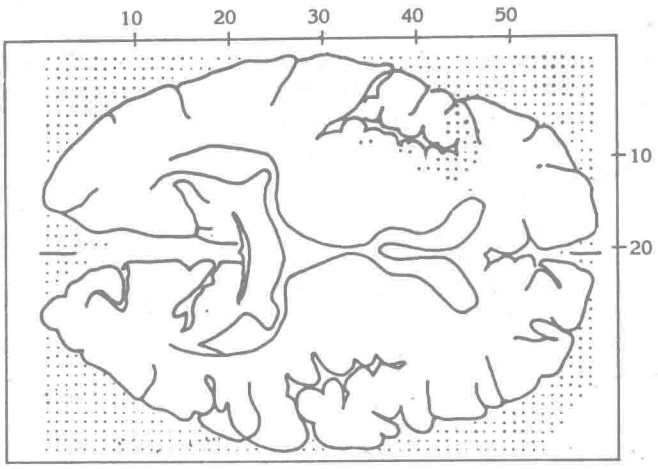


Fig. 2 a
Cumulative lesion in the subgroup of patients with Broca's Aphasia, slice 3

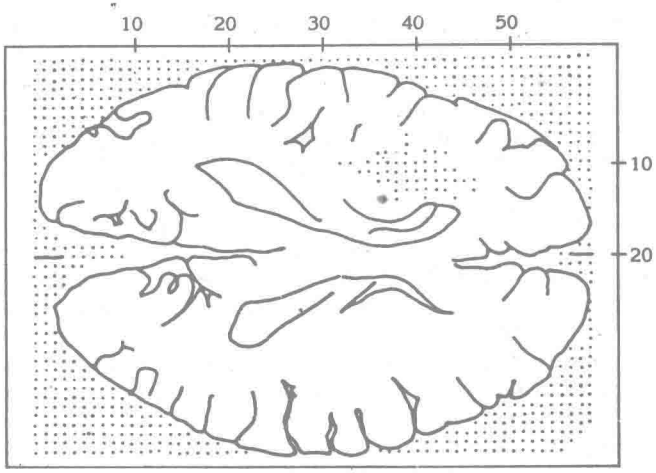


Fig. 2 b
Cumulative lesion (white matter damage) of patients with Broca's Aphasia, slice 4

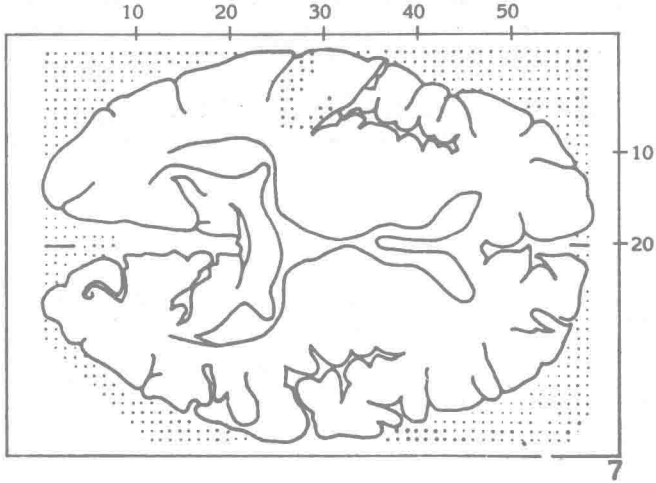


Fig. 3
Cumulative lesion of subgroup of patients with Wernicke's Aphasia, slice 3

It can be seen that the lesion is not exactly in Broca's area but is rather an undercutting or a disconnecting type of lesion. Figure 3 shows the lesion found in patients with Wernicke's aphasia. It is exactly where Wernicke predicted that it would be.

One can, of course, also ask more detailed questions, e. g. for the lesion common to all patients who are poor in repetition, or for the lesion of all patients with agrammatism. This method opens a promising avenue for studies on localization in aphasia (Poeck, de Bleser and von Keyserlingk 1984).

In the last three years we have developed a method for the three-dimensional reconstruction of lesions in the brain. On the basis of 30 brains, a standard brain model was constructed in a computer. This was done by following each of the sulci on the surface of the brain with a joy stick. The contact with the brain was fed via an XY plotter into the computer, together with the anatomical name of the sulcus involved. Then the superficial layer was removed and the sulci and other underlying structures were recorded by the same procedure. In this way, layer after layer was removed like in an onion, and the complete anatomical data of the 30 normal brains were stored into the computer. This permits the presentation of a three-dimensional stereoscopic image of the brain. It is possible to compare the lesion in the brain of a stroke patient with the stored anatomical data of the standard brain. The great advantage of this method is that the signal/noise ratio in the images can be extremely reduced. By this method it is possible to state with some confidence where the lesion on the surface or in deeper areas of the patient's brain is localized (von Keyserlingk et al. 1985).

Time and space available only permit highlighting some of the aphasia research programs done on an international scale.

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