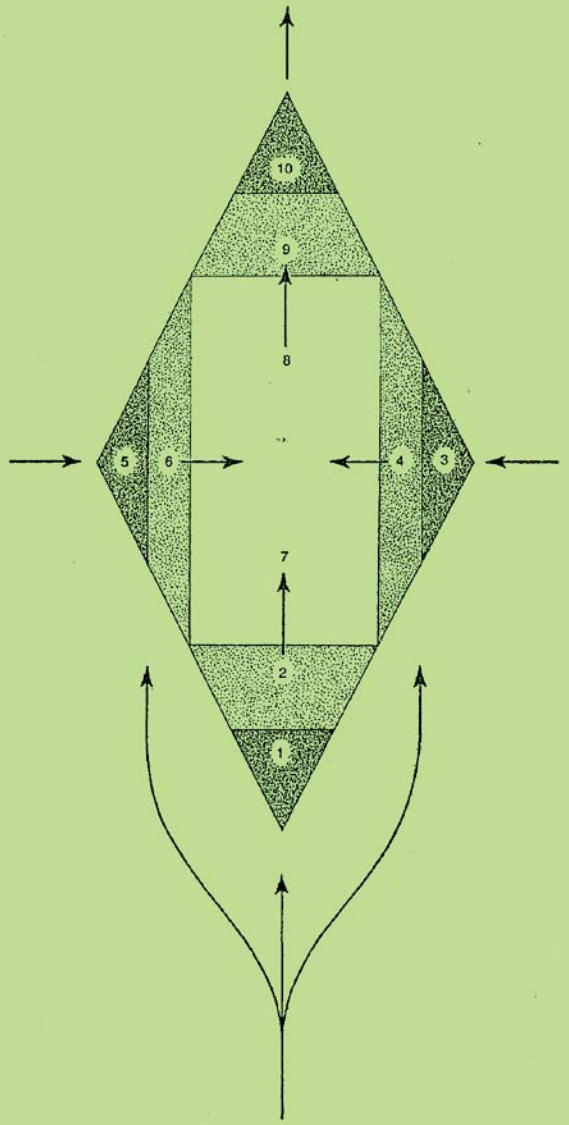
Instituut voor
toegepaste
Neurowetenschappen

VRIJE UNIVERSITEIT AMSTERDAM
Faculteit der Bewegingswetenschappen

Leliestraat 7c
2011 BL Haarlem

Opleiding Neurorevalidatie: deel 1

Probleemanalyse



INHOUD

The Decline and Fall of Hemispheric Specialization

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LEA LAWRENCE ERLBAUM ASSOCIATES, PUBLISHERS
1990 Hillsdale, New Jersey Hove and London

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The Decline of Hemispheric Specialization

INTRODUCTION

Every reader of this book—indeed everyone who reads a daily newspaper—has heard that the left hemisphere is specialized for language, mathematics, detailed analysis, logical thought, temporal and sequential analysis, and serial processing of sensory information. You also have heard that the right hemisphere is specialized for emotional expression, intuition, the recognition of faces and the emotions expressed in faces, artistic achievement, attention, recognition of musical passages and other musical aptitudes, visual-spatial analysis, and parallel processing of sensory information.

Over the past 30 years a number of investigators have periodically attempted to reduce the multiple specializations of each hemisphere to a single, more encompassing, function. Of course, since there are only two hemispheres, the two global functions must necessarily be expressed in a dichotomous fashion. Thus you have also heard that the left hemisphere is specialized for "verbal" functions whereas the right hemisphere is specialized for "non-verbal" ones; or that the left hemisphere is specialized for "linguistic" functions and the right for "visual-spatial" functions; or that the left hemisphere is specialized for detailed, "analytic" functions, whereas the right hemisphere provides us with a big picture of the world, as it is specialized for "holistic" functions; or that the left hemisphere is specialized for "propositional" functions, whereas the right hemisphere is specialized for "appositional" functions. Although none of these dichotomies has successfully integrated the wide variety of diverse functions attributed to each hemisphere, and almost no one today accepts them as valid

generalizations, they nevertheless paved the way for the concept that there are left- and right-hemisphere "cognitive styles" or personality types—terms that, as you might expect, are commonly used either humorously or pejoratively, as illustrated in Fig. 1.

These global abstractions have been extended far beyond the realm of neuropsychology itself, and have spawned imaginative new disciplines called "neuropolitics," "neuroanthropology," and "neurosociology" (Ten-Houten, 1985). These disciplines purport to account for the differences between Western and Oriental philosophies, our political ideologies, as well as the communication gap between generations, by the degrees to which our two hemispheres come to differ in their panoply of talents, styles or modes of cognition as a consequence of our education, social class (in the sense used by Karl Marx), urbanization, culture, and native language. The *political* implications of the concept of hemispheric specialization are now considered to be so self-evident that it has been seriously argued in some quarters that our European-based educational system, with its heavy emphasis on teaching the young to read, write, and do arithmetic, is in blatant violation of the constitutional rights of the right hemisphere to an equal education!

Some of these claims involving the differential specialization of the two hemispheres have been dismissed by serious scholars as unwarranted overgeneralizations from the known facts by "academic hucksters" or "non-professionals." These same scholars also have acknowledged that the field of what has now come to be called "laterality research" has been characterized by a disproportionately large number of contradictions and failures

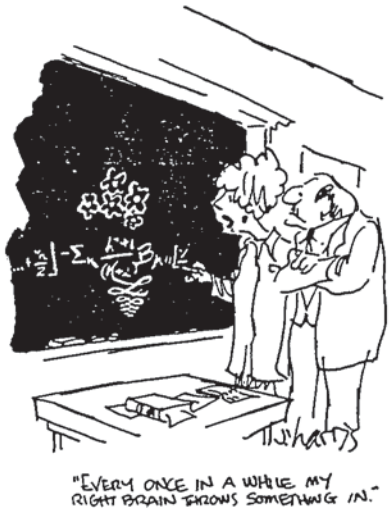

FIGURE 1. Copyright 1984 by Sidney Harris, originally published in *American Scientist* magazine. Reproduced by permission of Sidney Harris.

to replicate the experimental results of others. At least one of them has voiced his suspicion that there has been a strong tendency for selective publication of results that are consistent with the prevailing theoretical Zeitgeist and thus "interesting," with a parallel tendency to ignore work that does not fit or support the prevailing views (Bertelson, 1982).

These problems of laterality research have been so thoroughly discussed by others, most notably Bryden (1982), Bertelson (1982), Beaumont (1982), Corballis (1983), and the open peer commentaries on the paper by Bradshaw and Nettleton (1981), that I will not attempt to document them once again. Instead, my purpose in this chapter is to discuss some of the deeper theoretical and epistemological problems entailed by the concept of hemispheric specialization for cognitive functions that some of these books, review articles, and book chapters have alluded to but not addressed adequately. I provide concrete examples to illustrate how these issues affect the way research findings are interpreted and why the failure to understand the epistemological problems of the field has led to the misguided views about hemispheric specialization that I have just described. In Chapter 2 I go one step further and show how these unresolved problems have led the field of laterality research into a state of total scientific collapse. Chapter 3 offers a viable solution.

THE MEANING OF "SPECIALIZATION"

Before we can even begin to consider the issues in this complex area, it is first necessary to distinguish between the uses of the term "specialization" by neurophysiologists and by neuropsychologists. There is absolutely no doubt that various cells in the nervous system have markedly different properties: The rods and cones of the retina are exquisitely sensitive to light, the hair cells in the cochlea to mechanical displacement, cells in the medulla to minute changes in carbon dioxide concentration, and cells in the striate cortex that respond with high rates of discharge to one orientation of a pattern but not to another. Even at subcellular levels, the membrane responses to specific neurotransmitters, for example, are highly selective. Although such uniquely specific responses of single cells and their components are usually referred to as "selectivity" by neurophysiologists, it is neither an abuse of language nor does it create any ambiguity of meaning to say that they are "specialized" in their *response characteristics* in precisely definable ways.

It is also the case that cells with certain types of selective responses are not distributed randomly within the brain, but highly concentrated in certain areas. Thus there are reasonably well-defined areas containing cells that have specific responses to visual, auditory, olfactory, or tactile stimuli, and

these areas are appropriately referred to as visual, auditory, olfactory, or tactile "centers." There are also motor centers that, if stimulated electrically, give rise to specific, definable muscular contractions, for example, the frontal eye fields, which play an important role in eye movement control. However, the cells within such a center, say, the visual center in the lateral geniculate nucleus of the thalamus or the one in the striate cortex, are aggregates of interconnected cells having *different* types of selective responses. Although it is not difficult to define in what way a *single neurone* is "specialized," it is not a trivial task to define the specialized function of such a "center," whether it be a sensory or motor one. It is when we attempt to deal with very large numbers of interconnected neurones, each having different selectivities, that the concept of "specialization" of function becomes exceedingly difficult to apply. For example, after more than 30 years of intensive research on single neurones in the striate cortex, it is clear that its individual cells have many different types of selective responses, but it still is not possible to formulate a single abstraction that adequately defines the principal function(s) of this critically important and anatomically well-demarcated region of the cerebral cortex—despite the fact that monkeys and men are essentially blind if this area is destroyed bilaterally. Indeed, it would be fair to say that most visual neurophysiologists would consider it somewhat silly, given our present knowledge, even to attempt such an abstraction. If pushed, they might say that it is at the striate cortex that the radial symmetry of the center- surround discharge pattern of single neurones of the lateral geniculate is changed to a bilaterally symmetrical discharge pattern, and that this may indicate the *first* step in the Fourier analysis by which a two-dimensional spatial frequency map is created.

In contrast, when neuropsychologists use the term "specialization" they mean something quite different from the neurophysiological concept of "selective response." They mean that a particular region of the brain is critically responsible for the performance of a specific cognitive *function* or a small group of closely related cognitive sub-functions. It should be emphasized that the root concepts and vocabularies of neurophysiology and neuropsychology do not overlap, except with respect to anatomical issues. This is not surprising since one discipline deals with *brain* functions whereas the other deals with *mental* functions. Indeed, until such time as the mind body dichotomy is resolved, these vocabularies will continue to differ.

The field of neuropsychology, however, has a unique problem that is not shared by the two disciplines to which it is conceptually related. Cognitive psychologists do not have to relate their findings to any neuroanatomical or neurophysiological data. For them the brain can be considered merely a "black box." Neuropsychologists, however, are not so fortunate: The implicit mandate for their field is to relate mental phenomena to the internal structure and function of that black box. Given this mandate, their

conceptualizations, vocabulary, and methodology will inevitably be less "pure" epistemologically, and thus more subject to criticism, than those of neurophysiology or cognitive psychology. This burden should make them *more* careful when they define their terms, state their inferences, and evaluate their evidence. Unfortunately, such epistemological fastidiousness is rarely exhibited.

INFERRING FUNCTION FROM BEHAVIORAL DEFICITS

Having said that neuropsychology uses the term "specialization" to mean that a particular region of the brain is critically responsible for the performance of a specific cognitive function, I now want to discuss the inferential procedures that are used to make such a claim. Historically, the primary method used to determine the function of some region of the nervous system has been to see what deficit results if that area is damaged by disease (or by an overly zealous neurosurgeon). Although this approach has generated a vast and scientifically exciting body of information, it is not as easy as you might imagine to infer the *function* of the tissue that has been physically or biochemically damaged from the observable *deficit*.

As this difficulty is central to any evaluation of the claims for hemispheric specializations with which I began this chapter, I will illustrate the problem with two examples. The first relates to a characteristic behavioral deficit that is seen when the cerebellum is damaged. In such cases, when the patient is asked to touch an object with his finger tip he will reach out in the appropriate direction but will alternately over- and under-shoot the target. This readily observable behavioral deficit has been called "dysmetria." Many neurologists had concluded that one of the *functions* of the cerebellum is to *inhibit* this over- and under-shooting. Over 40 years ago, in a discussion of dysmetria, F.M.R. Walshe rejected the inference that the cerebellum has an inhibitory "braking" function, and as part of a longer argument used the following analogy (Walshe, 1947): Imagine, he said, an automobile transmission system, crudely illustrated in Fig. 2, in which one tooth of a gear has been knocked off, by the kind of stroke that affects all of our cars from time to time. Because the loss of this one tooth does not render the gear-train totally inoperative, the only striking behavioral deficit of which we are aware is a "thunk" that is heard on every revolution of the drive shaft when it is turned slowly, and a vibration at higher speeds. Now what is the function of this missing tooth? What did it do *before* it was knocked off? To someone who knows as little about mechanical systems as we know about cerebral function, an apparently plausible answer is that the *function* of the missing gear tooth is to prevent or inhibit the "thunk" and

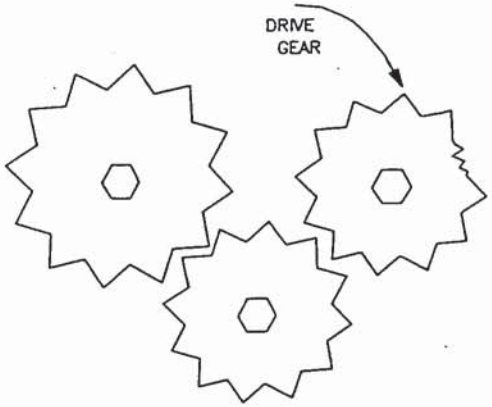

FIGURE 2. Inferring the function of the missing gear tooth on the drive shaft from the "thunk" heard on each revolution. Example of F.M.R. Walshe (1947). See text for discussion.

vibration. Further, if the tooth is welded back on, and the symptoms disappear, the hypothesis regarding its inhibitory function appears to be supported. The actual function of the gear teeth—to transmit power from the drive to the driven shaft—has been missed entirely!

The point of Walshe's clever example is to warn us that if we define the *function* of a region of the brain in terms of the *symptom* that results if it is damaged, then it is almost inevitable that we will *misidentify* its actual function. Thus, if a stroke in some area of the brain results in the inability to tie one's shoe laces, while the performance of all other bimanual tasks, such as typing and cutting steak at the dinner table, are intact, we inevitably would be led to the almost certainly erroneous conclusion that the *function* of the tissue prior to the damage was to tie shoe laces, that it is "specialized" for this function.

Let us now consider the behavioral deficit in another machine, one appreciably more complicated than Walshe's gear-train, but vastly less complicated even than the brain of a bird. I refer now to my desk top computer. Imagine that, while I am working happily on my computer adding a set of numbers from a file and displaying the sum on the screen, a cosmic ray particle strikes one particular chip in the computer and damages it. I suddenly notice that some, but not all, of my additions are producing absolutely bizarre results. As a neurologist, I suspect that my computer has developed what is known in the trade as an acalculia, a specific deficit in performing simple arithmetic functions seen in some patients with strokes. Further diagnostic tests reveal that major errors occur not only in addition, but subtraction, multiplication, and *every* other numeric operation, providing additional evidence that the computer has developed an acalculia. When the repairman replaces the defective chip, moreover, the acalculia disappears, "converging evidence" supporting my inference that this particular chip is "specialized" for mathematical functions.

Now what actually has happened is that one of the functions of the chip is to convert a few bit patterns corresponding to decimal digits into the proper numerical characters, and that when damaged this conversion fails—every time a bit pattern representing a 1 is sent to the chip, the defective chip converts it to a 9 which is then displayed on the screen. My computer's behavioral deficit was only that the numerical result *as displayed on the screen* was incorrect. Had I known more about computers, I could have tested this hypothesis directly by sending the numbers to be printed on a piece of paper, rather than on the screen. Since computer printers contain a chip having a similar function, and the one in the printer was *not* hit by the cosmic particle, all digits would have been printed correctly on the paper, thus demonstrating that the computer had never suffered from an acalculia at all. Finally, without having performed a very large number of diagnostic tests on the computer, I would never know what other functions of the chip had been damaged.

I would maintain, and I'm sure you will agree, that by disregarding Walshe's warning not to confuse the symptom with the function I had made an egregious error in concluding that the chip was "specialized" to perform calculations, or that its function was to calculate. I also would maintain that it is our exceedingly primitive understanding of the unimaginably complex neural mechanisms underlying language, calculation, and other cognitive functions that predisposes us to confuse the observed behavioral deficit, the symptom, with the actual function or functions of that tissue *prior* to the damage.

LOCALIZATION VERSUS SPECIALIZATION

My purpose in using the gear-train and computer analogies is to focus attention on two closely related concepts that, more than any others, have been the source of epistemological chaos in the field of neuropsychology—the concepts of *localization and specialization* of function. I have already pointed out that visual neurophysiologists still are unable to define the function of the striate cortex, despite the fact that primates become essentially blind if this cortical area is damaged bilaterally. Similarly, we cannot yet define the functions of the left fronto-temporal lobe despite the fact that in more that 95% of human beings profound disturbances of language occur if this area is damaged. But what is added to our understanding of the actual functions of these two areas if this knowledge is restated by saying that one is "specialized for vision," and that the other is "specialized for language"? Absolutely nothing.

Most neuropsychologists insist that they use this word *only* as a *shorthand expression* to refer to these correlations between lesion site

and symptom. Although this may have been the original intention, even a cursory review of the literature supports my contention that this is no longer the case. Over time they have come to believe that a function called vision is "located" in the striate cortex, when it is no more "located" there than in the retina, optic nerve, or parastriate cortex, and that the function called language is "located" in the left fronto-temporal area. Expressed in another way, this scientifically empty restatement of the facts has predisposed them to forget that the striate cortex and left fronto-temporal cortex are only *components* of anatomically extensive visual and linguistic *systems*.

Recent research using PET and SPECT brain imaging provides compelling evidence that many different areas of the brain in *both* hemispheres become metabolically active when we engage in a specific cognitive activity. These maps, now in pseudo-Technicolor, reveal that neural circuits supporting a cognitive function are located in anatomically disparate regions and that the *same* areas may become active in two distinctly different cognitive activities, for example, actually moving your hand and *imagining* that you are moving your hand. This *physiological* evidence underscores my point: that use of the term "specialization of function" has predisposed neuropsychologists to believe that a specific cognitive function is performed "in" a specific cerebral area, which is "specialized" for that cognitive activity, rather than by an anatomically extensive system involving both hemispheres.

At a deeper level, however, when the term specialization of function is used as a short-hand way of referring to a *correlation* between the site of a lesion and some cognitive symptom, the short-hand user is predisposed to confuse a correlation with an explanation or hypothesis. A correlation is testable by repeating, with more patients, the incidence of various cognitive deficits with lesions at different brain sites. Regardless of the strength, and the reproducibility of the correlation, it does not explain *why* this relationship is observed, or permit any *prediction* outside of itself.

A correlation is *not* a hypothesis: In the development of a science, a correlation is a *first* step, albeit a necessary one, which must, if it is to go anywhere, be followed up by a tentative explanation—the hypothesis itself—whose predictions are then tested by well-designed experiments. If this procedure is not followed, as is often the case among neuropsychologists, it leads to circular and misleading statements such as, "The patient cannot speak *because* the center for speech production has been damaged, but he can understand speech *because* the center for speech comprehension is intact." It is *circular* since it refers to no further information than that contained in the initial clinical-pathological correlation, and it is *misleading* since it predisposes the speaker, as well as the unwary listener, to believe that an explanation has been offered when it hasn't.

Another statement, found in many articles and books, is even more

misleading: "It is unarguable that the left hemisphere is specialized for language." This covertly implies that some scholars dispute the validity of the clinical-pathological correlation, or are unaware of it—when neither is the case—and leaves the unwary reader with the distinct impression that any objections to this proposition could only come from an ignoramus who still believes that the earth is flat! When I object to such misleading formulations, most neuropsychologists look at me incredulously and ask, "Are you saying that the left hemisphere is *not* specialized for language?" or "On what grounds do you reject this hypothesis?", failing to remember that their concept of specialization has the status of an observed correlation between the symptom and a lesion location but is not a *refutable* scientific hypothesis concerning the actual function of the damaged tissue. It is not refutable, of course, since the function has been *defined* in terms of the symptom.

What *is* indisputable is that the *strongest* correlation between the localization of the brain lesion and a cognitive dysfunction is for language functions: More than 95% of the population will develop one or another type of language disturbance, an aphasia, following a left fronto-temporal stroke. The existence of this correlation undoubtedly indicates that this brain region contains neural circuits that support language functions, but it does not necessarily mean that the function called "language" is performed by these circuits. It is more likely that these circuits perform a variety of functions, some of which are *required* for normal language function. This is precisely the point that Walshe made with his gear-train analogy: Don't confuse the function with the symptom.

Despite the existence of this strong correlation between the symptoms of aphasia and lesions in the left fronto-temporal region, it has been known for over 100 years that even with large lesions extending well beyond the area of the so-called language centers in the left fronto-temporal area, some speech remains. A patient with such a large lesion may be incapable of verbally communicating any simple wish or idea but, perhaps in frustration or despair, will sometimes curse or utter other expletives with perfect articulation and may even sing common songs, *using words*, with some degree of proficiency.

Such observations led neurologists in the late 19th century to suspect that this "residual" language is accomplished by speech centers in mirror-image locations within the *right* hemisphere and that recovery from aphasia might be due to the right-sided areas "taking over" language functions that could no longer be performed by the damaged tissue on the left side. This suspicion was supported by the fact that *recovered* aphasics who subsequently had a stroke in their *right* hemisphere developed a profound and *lasting* aphasia. More recent evidence indicates that with careful testing of right- handed patients with right hemisphere lesions subtle language distur-

bances can be detected, and that in split-brain subjects whose fronto-temporal areas in both hemispheres are undamaged, the right hemisphere, like a 2-year old, appears to "understand" vastly more speech than it can express (Ardila, 1984; Zaidel, 1985).

The right hemisphere's apparent capacity to understand spoken language will be discussed again in Chapter 2 in the context of dichotic listening experiments. For the moment, however, I call your attention to the curious fact that those who assert that it is indisputable that the left hemisphere is "specialized for language" usually are aware that damage to the mirror-image regions of the right hemisphere often result in disturbances of the prosodic and intonational aspects of spoken language (Ross, 1981; Ross & Mesulam, 1979). However, they rarely say that the right hemisphere is "also specialized for language, but less so or differently": They seem more comfortable in using the phrase "right-hemisphere speech," and dropping the term specialization in this case. These same neuropsychologists also know that aphasia occurs following lesions in the thalamus, a subcortical area far removed from the so-called speech centers in *either* hemisphere (Damasio & Damasio, 1989; Ojemann, Fedio, & Van Buren, 1968; Ojemann & Ward, 1971; and *BRAIN AND LANGUAGE*, 1975, *2*, 1–120, for a series of review articles.). To be consistent, they should describe the thalamus as "specialized for language," but I have *never* heard this formulation used, and when I use it to point out their logical inconsistency, a bewildered expression crosses their faces!

Finally, there are interesting patients who have lost their hearing before speech developed and who learned to communicate by signing. A recent report of a left-hemisphere stroke in one such patient (Chiarello, Knight, & Mandel, 1982) describes the typical manifestations of aphasia but in sign language. However, the cerebral damage was *not* in the area usually considered to be a language area, the region near the auditory cortex, but rather, in a region where sensory information from the hands reaches the cortex. Thus, for this individual, who had learned to use a manual means of communication, neural circuits supporting linguistic functions developed in somesthetic cortex. To be consistent, one should then say that the somesthetic cortex is "specialized for language."

Such logical consistency has not been followed, I suspect, because it would make it even more evident that the many different, and at present unidentified, sub-functions required for efficient language comprehension or production are not "located" in one or two cortical areas but are supported by an anatomically extensive *system*, a point of view that runs counter to the neuropsychologist's agenda but which is nevertheless *visible* in PET and SPECT images. In using the word "agenda," I do not mean to imply any conspiracy, but rather to emphasize that the original role of the neuropsychologist, when arteriograms or CAT and MRI scans did not exist,

was to assist the neurologist in localizing the patient's *lesion* using psychological measures. It is easy to understand that this historical role led to the misconception, unfortunately still prevalent, that they were localizing the defective cognitive *function*.

In sum, although there is overwhelming evidence that many important neural circuits supporting language functions are indeed located in the left fronto-temporal area, it is evident that neural circuits are present and/or can be developed in a number of *other* cerebral areas that also support these functions. By now you must have noticed that I have repeatedly used the phrase "neural circuits that *support*" a function and have abjured the phrase "neural circuits that are *specialized*" for a function. I will continue to use this neurophysiologist's vocabulary for three reasons: (1) It does not confuse the symptom with the yet-to-be-discovered function; (2) It does not convey any hidden and misleading implications with respect to the *location* of a cognitive function; and (3) It does not confuse a correlation with an explanation.

REDUCTION OF MULTIPLE BEHAVIORAL DEFICITS TO ONE COGNITIVE DEFICIT

My discussion so far has been focused exclusively on the problems entailed in inferring the function of a cerebral area from the behavioral deficit observed when that region is damaged. I turn now to a related, and even more difficult, problem of inference. In virtually all cases of brain damage, even from a relatively circumscribed lesion, there are a number of different behavioral deficits that become evident as the investigator tests the patient with various tasks. The issue is how to decide whether there are multiple, unrelated, and essentially *different* cognitive functions that have been impaired simultaneously, or a *single* impaired cognitive function that underlies or accounts for all the behavioral deficits. I will illustrate this issue in a patient with a rather dramatic set of behavioral deficits that resulted from diffuse, bilateral brain damage (Efron, 1969). The nature of this inferential process, however, is identical whether the patient's lesion is in one hemisphere only or in both, or whether it is a focal or diffuse lesion.

A young soldier slept overnight in a room with a defective space heater and suffered a massive overdose of carbon monoxide. When found in the morning, he was comatose and remained in a deep coma for some days because of the widespread neuronal damage throughout his brain. As he slowly regained his ability to speak and understand, he appeared to be blind. Except for a normal reflex contraction of his pupils to light, he seemed to have no useful vision at all. He lay in bed with his eyes roving the visual scene in an apparently random fashion. On more careful examination

several days later, however, it was evident that he was not blind in the usual sense of the word, because he responded normally to small light flashes in various parts of his visual field. Further, when asked, he could track, by pointing with his index finger, a small object that was moved in front of him, but he could never *name* the object that he obviously was seeing and tracking. Now, was this inability to name objects due to a disturbance of linguistic functions? Was he simply aphasic? Although he had an obvious aphasia as well as a number of other cognitive and physical disabilities, none of these deficits seemed to be *sufficient* to explain why he could not name any object placed before him, since he *could* name any object placed in his right or left hand, and say how it was used, about as well as any blindfolded normal subject. Thus, his naming problem was restricted to the visual modality. Was his visual acuity so poor that he couldn't see the object clearly enough to name it? This was easily ruled out by tests of visual acuity. At this point some of you will know that his behavioral deficit is characteristic of a syndrome called visual agnosia, a name coined by Sigmund Freud when he was a young neurologist to indicate merely the loss of visual knowledge.

Members of the medical profession, as you are no doubt aware, are trained to use Latin names to hide their ignorance of what is really wrong with their patients. Although this practice may reassure the patient, Freud was not so naive that he confused a name with an explanation, and he came up with the hypothesis that visual agnosia results from a loss of *visual memories*. Note that this reduction of a number of behavioral deficits to a single impaired cognitive function seems quite plausible and, indeed, it is still considered to be a correct interpretation, at least for some cases of visual agnosia. In the present case, however, such an explanation seems unlikely since the patient could describe many objects from memory—a shoebox, a telephone and its usual color (at that time almost always black), a hammer, a basketball, a hockey stick, a key ring, and so on. But immediately after describing such an object from memory he could not name it when it was shown to him. Further, if he was shown a *moving* key ring and asked to describe it, he only could say he saw something "shiny," but could not name it until the keys were jingled. As soon as he heard the sound he instantly shot out the name "keys." He could not recognize any of the ward staff or his physicians until they spoke, and, on hearing them speak, he addressed them by their correct names. He also could not identify his wife, a photograph of himself or even himself in a mirror, a syndrome called prosopagnosia, another non-explanatory Latin term to say that a patient cannot recognize even familiar faces.

Despite the extensive brain damage he suffered, the patient was aware that something was wrong with his vision, and this awareness was reflected not only in his verbal communications, but also in his *dreams*: He had a

recurrent nightmare in which he was in a sealed dark barrel from which he couldn't escape: The barrel was tumbling over Niagara Falls and he could hear the noise and people yelling for him to get out. This dream gives some insight into the horrifying emotional impact that cognitive deficits can have on a patient.

Quite determined to discover what was wrong with this patient, I spent many months devising various tests of his visual behavior. It soon became obvious that he could not find or point to any object that was *not* in motion with respect to a complex visual background. Here are two examples: When brought to my office in a wheel chair, he was totally unaware that I was sitting in my chair if I didn't move. If I did move and was wearing a white coat, he'd say, "Hi, doc," but he still did not recognize me until I spoke. The second example involved placing various objects on a colored advertisement torn from a Sears, Roebuck catalogue in such a way that he could not see me doing it. I then would ask him to point to the "thing" on the piece of paper. He *never succeeded*. However, if I jiggled the advertisement so that the object moved with respect to the background he *never failed*: His hand shot out instantly and he touched it accurately. Although I would not maintain that he had a normal level of intelligence, it was clear that he exhibited some ability to make deductions on the basis of visual input: For example, I taught him, one at a time, the names of some 20 common objects (scissors, pencil, paper clip, etc.) each time keeping the object in motion against the advertisement. After several days of practice he could unfailingly apply the correct name to each of these moving objects by making use of one or more distinctive features such as its reflectance, size, or color.

One of these objects was a playing card that had a complex pattern in red and white. He had been taught to call this a playing card, and he had always seen only its back, the red and white pattern, while I kept it in motion on the colored advertisement. One day I substituted a small red and white postage stamp. A wry smile crossed his face, indicating that he recognized the trick, and he said, "Oh, that's a piece of a playing card." Since he clearly could name colors, and had a good sense of the size of an object, his response, a *piece* of a playing card, indicated that he could utilize the information available to him and come up with an intelligent, albeit incorrect, inference.

At this point, one might reasonably conclude that his fundamental cognitive deficit was an inability to distinguish figure from background. Indeed, as long ago as World War I, Poppelreuter (1917) showed soldiers, with gunshot wounds of the occipital cortex, overlapping line drawings of common objects. Figure 3 is one of Poppelreuter's drawings. These patients had great difficulty in finding a named object in such a jumble and for many years neurologists carried one or more such figures in their medical bags since they were reasonably reliable in diagnosing occipital lesions in the

FIGURE 3. Overlapping line drawing by Poppelreuter (1917). From Walsh, K.W. (1978), *Neuropsychology: A Clinical Approach.* Reproduced by permission of Churchill Livingstone, Edinburgh.

days before CAT and MRI scans. Although my patient undoubtedly had a problem with figure- ground discrimination, would it have been correct to say that *all* his visual disturbances could be explained by (i.e., reduced to) this deficit of figure- ground discrimination? I think this would have been an error. Even when the figure-ground problem was *overcome* by moving the object against the background, so that he could see and point to it, he still did not know what it was—except for the 20 objects he had been taught to name by virtue of the distinctive features of color, size, or reflectance.

In thinking more about this issue, it seemed possible to me that the patient could not name the object even when he saw it moving because he could not determine one other key attribute of an object, its *shape*. This hypothesis was tested in a number of ways. The patient was shown the series of Pseudoisochromatic test plates that are widely used to evaluate color vision. Each of these plates contains a large number of circles of varying sizes, and shades of gray. In addition, each card contains two simple geometrical shapes (crosses, circles, triangles), created by replacing some of these circular gray patches with colored ones, also of varying saturation. The subject is usually required to name the two shapes on each plate, shapes which perceptually pop out of the background by virtue of their color, if the the subject has normal color vision. When my patient was asked what he saw on each plate, he invariably named the color correctly (indicating that his color vision was normal), but he was never able to name or even trace the outlines of the two shapes. Furthermore, when very simple geometrical shapes, such as squares, circles, or triangles were slowly drawn in front of him so that he could see the *movement* of the tip of the pen, he frequently named the shape, but when he was shown the same drawing a few moments later, he could no longer identify it. In another test, the patient was asked to report whether two objects of identical area on a blank background, as

seen in Fig. 4, were the same or different. Only when presented with a square and a very long, thin, rectangle was he able to report reliably that they were different.

These, as well as other studies, showed that he had virtually no capacity to discriminate objects on the basis of their *shape* in the visual modality. A good argument can be made, I think, for the hypothesis that a single deficit in visual shape perception accounted for *all* his other visual behavioral deficits: Without the ability to perceive shape, you cannot distinguish figure from background without relative motion, and when it can be distinguished from the background by introducing relative motion you still do not know what it is, and therefore cannot name it. This hypothesis of a single disorder of shape perception might account for the prosopagnosia too, since faces, perhaps more so than any other objects, are recognized by virtue of subtle differences in shape. However, because there are *other* patients with prosopagnosia who do not have a deficit in shape perception, it is not possible at the present time to reject the alternate hypothesis that my patient had *two* independent and unrelated cognitive deficits—one for shape perception and the other for face recognition. One could even argue that he had *three* simultaneously occurring but independent cognitive deficits: shape perception, face recognition, and figure-ground discrimination.

It is obvious that I raise these alternatives to make a point: Here we have a demonstrable visual *perceptual* problem, and even at this low cognitive level it is extremely difficult to apply Occam's Razor, that is, to account for the patient's problem with the *fewest* possible number of hypothesized cognitive functions.

Now why does the clinical neuropsychologist have to use Occam's Razor at all? The answer is obvious: If we don't we are left only with a very large

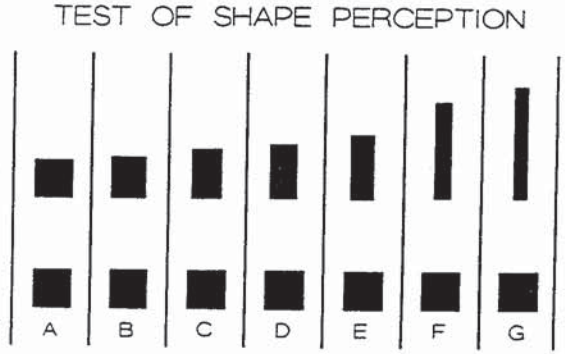

FIGURE 4. Panels A through G each contain two shapes of equal area. The shapes were identical only in Panel A. The subject was required to report if the two shapes presented on a trial were the same or different. The performance of the patient with visual agnosia was above chance levels only for the shapes in Panels F and G.

list of different *tasks* that the patient fails to perform or performs poorly, and we can never conceptually isolate an impaired *cognitive function*. Although it is extremely difficult and risky to identify an impaired cognitive function, the alternative—listing all the behavioral deficits on different tasks—is obviously heuristically sterile.

TIME AND LANGUAGE

I have just indicated how difficult it is to reduce the *perceptual* deficits of visual agnosia and prosopagnosia to a single disturbed function. I now want to illustrate the nature of the problems one faces when the deficits are not perceptual but involve still "higher" levels of cognitive function. In the late 1950s I was studying the inhibitory neurophysiological mechanisms that bring epileptic seizures to an end. In the course of taking many careful neurological histories on patients with epilepsy, I was struck by the frequency with which seizures beginning in the left temporal lobe, that is, in the area of the language centers, gave rise to various subjective disturbances of *time sense* during the aura preceding the epileptic seizure. Time seems to be passing either in slow motion or exceedingly rapidly, events seem to be out of order, or the patient may have particularly intense experiences of *déjà vu* or clairvoyance. In addition, seizures originating in the same anatomical region often are accompanied by a transient aphasia.

These observations led me to speculate on the possible reasons why functional disorders of the time sense and language might be associated. It seemed to me that language is inherently a *temporal* activity: Words in every language are composed of a small sub-set of sounds (the entire set is usually less than 40 in any language) uttered in a particular temporal order; sentences convey meaning because of the particular temporal order of the words which they contain; and a set of sentences also conveys the intended meaning only by virtue of temporal order. This is true, of course, for both written and spoken language. Wielding Occam's Razor deftly, I asked, why should there be *two* independent sets of neural circuits, one used to make judgments about the sequence of a series of events in the environment, and another just used to keep track of the sequence of sounds in a word and the sequence of words in a sentence? Wouldn't it be more parsimonious to assume that the *same* neural circuits supported both functions? And wouldn't it be interesting if these timing circuits were *anatomically co-extensive*? If the left-hemisphere circuits that support language function were the *same* ones as those that keep track of the temporal order of events and the sequence of muscular contractions needed to enunciate words, then damage to these circuits or an epileptic seizure involving them would be

expected to result in some significant disturbance of *both* cognitive activities.

Obviously the first step in testing this hypothesis was to devise a way to discover the location of the circuits supporting the capacity to tell the temporal order of two events (Efron, 1963a). The simple principle underlying this method is illustrated in Fig. 5, using an example from astronomy, where two supernovae have exploded simultaneously. Since light waves travel with the same velocity in all directions, an observer located to the left of the plane half-way between the two explosions will see the light from supernova #1 before that of supernova #2 and will report that supernova #1 exploded first. Obviously, an observer located on the other side of the plane will report the reverse sequence of events. Only observers located on the plane will report that the two explosions were simultaneous. However, since *we* know that the explosions were simultaneous, the observer's location in space can be deduced from his report of the temporal sequence of events. I thought that this principle could be used to determine the location of the "observer" in the brain.

I was aware of an experiment by Klemm (1925), illustrated in Fig. 6, that

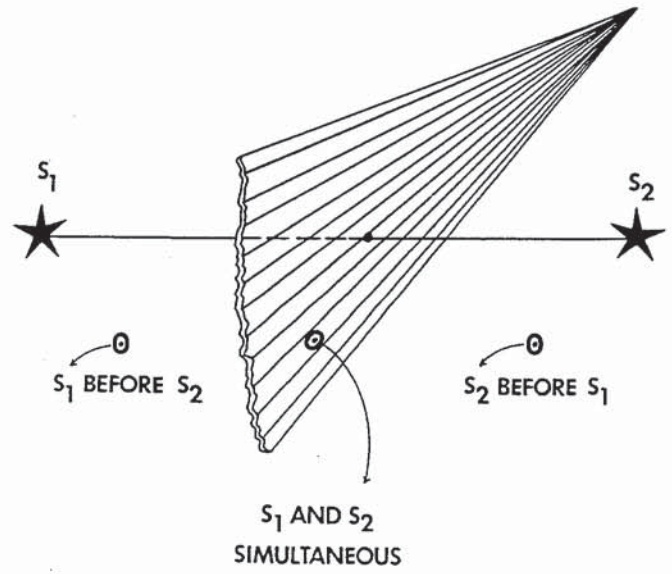

FIGURE 5. Inferring the location of an observer from his report of the temporal sequence of two events. S1 and S2 represent supernovae that have exploded simultaneously. An observer located to the left of the plane equidistant from the two explosions will report that S1 exploded before S2. An observer located to the right of the plane will give the opposite report. From the report given, one can locate the position of the observer.

could be adapted for my purpose. Klemm gave weak electrical shocks to the forehead and toe of subjects and found that when the two shocks were simultaneous, the subjects reported that the forehead shock preceded the toe shock. It was only when the toe shock, which set up impulses that had further to travel to the brain, was given *before* the forehead shock that his subjects reported the two shocks to be simultaneous. Although Klemm's experiment only tells us that the "observer" is located closer to the forehead than to the toe, hardly a surprise, his method of using judgments of simultaneity was just what I needed.

The two experiments I performed are illustrated in Fig. 7. In the first, equally intense shocks were given to the right and left index finger tips of neurologically normal subjects. In the second, equally bright lights were flashed in the right and left half-fields. Thus the shocks and light flashes were presented in such a way that one was projected to the right hemisphere and the other to the left. In each experiment I varied the order and interval between the two stimuli and merely required the subjects to report if they were simultaneous or not. Assuming that I was very careful to use equally intense stimuli and positioned them carefully, so that the conduction time to the brain would be the same (the biological equivalent of the constant speed of light), I should be able to tell from the subjects' reports whether the "observer" in their brain was closer (in terms of neural conduction time) to the right- or left-sided stimuli, or was equidistant from the right- and left-sided events, as implied by Descartes' theory in which the Soul was located in a midline structure—the pineal gland.

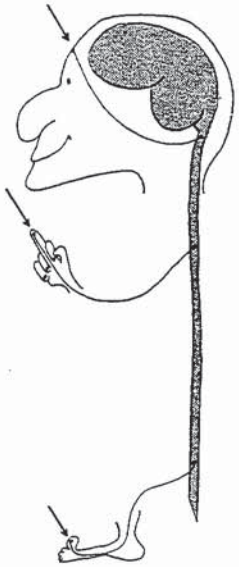

FIGURE 6. An experimental paradigm used by Klemm (1925). With simultaneous shocks delivered to toe and forehead the latter is perceived as occurring first. Subjective simultaneity is achieved only when the toe shock precedes the forehead shock.

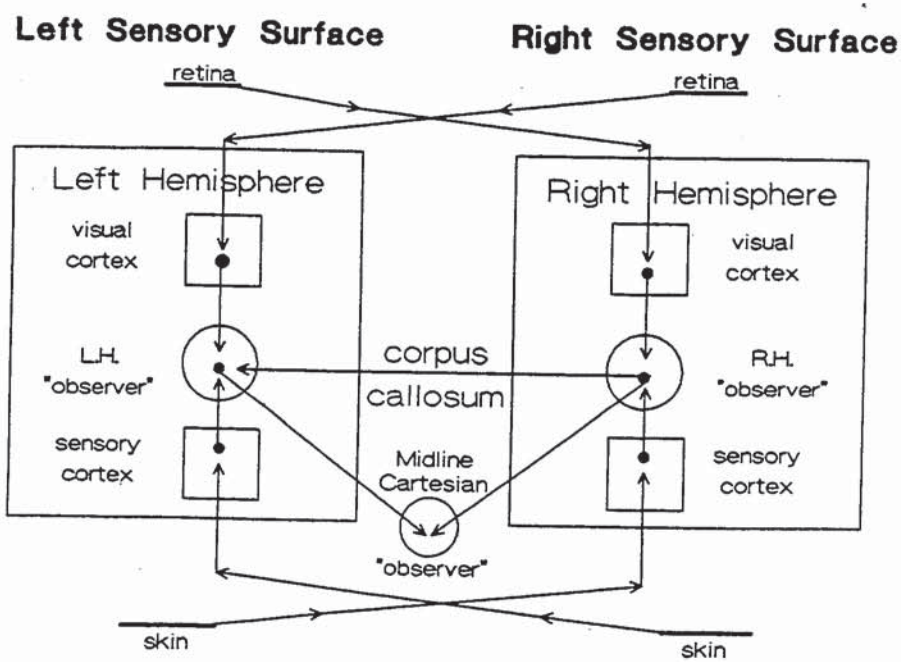

FIGURE 7. Schematic diagram of neural connections from retina (top) and from skin surface (bottom) to the hypothetical observer located somewhere in the brain. The observer might be located in a midline structure, *equidistant* in terms of transmission time from the right and left sensory surfaces, e.g., in the pineal gland—referred to as a Cartesian observer in his honor because he located the Soul there. The diagram also illustrates an observer located in the right or left hemisphere in which case the transmission time to it from the right- and left-sided stimuli would not be equal. The experiment described in text was aimed at localizing the observer.

The results for both sensory modalities showed that Descartes was wrong! In right-handed subjects the stimuli were reported as being simultaneous when the one projected to the right hemisphere *preceded* the one projected to the left hemisphere by about 4-6 milliseconds. This finding suggested that the observer is located somewhere to the *left* of the midline, because it took the neural message delivered to the right hemisphere 4-6 milliseconds longer to reach it. Electrophysiological evidence available at the time these experiments were performed indicated that a weak electrical shock delivered to the cerebral cortex in one hemisphere produces an electrical response in the mirror image region of the other hemisphere in about 5-6 milliseconds. Putting two and two together, and desperately hoping that I would not get five, I concluded that the observer was located

somewhere in the left hemisphere and that the information received by the *right* hemisphere was *delayed* by just about the right amount of time necessary for it to be relayed via the corpus callosum to "the observer" in the left hemisphere.

Obviously, this experiment in right-handed subjects provided no information concerning the location of the observer *within* the left hemisphere. Was it anywhere near the left hemisphere language centers? In this respect, the data from the left-handed subjects were interestingly ambiguous: In brief, some of them seemed to have an observer located in the left hemisphere whereas others seemed to have one in the right hemisphere. Since neurological studies of left-sided temporal lobe strokes in left-handed subjects have shown that only about half of them exhibit any signs of aphasia, the results on the left-handed subjects were at least *consistent* with the possibility that the observer might be located right smack in the middle of a subject's speech centers—whether these centers were in the right or left hemisphere. A more direct test of this hypothesis was required.

If the observer and the speech centers are anatomically co-extensive, then an individual with damage to his speech centers should also have a damaged observer, and this in turn might be reflected as a disturbance in the ability to report the correct temporal order of two stimuli, even non-linguistic ones. To test this I performed the following experiments in patients with cerebral lesions (Efron, 1963b): In the first, they were presented with brief red and green light flashes located at the fovea; in the second they were presented with brief high and low pitch sounds from a loud- speaker directly in front of them. For each modality the subjects were required to report the temporal order of the two stimuli. The interval between the two stimuli was varied between 0 and 600 milliseconds. Two groups of subjects were used: patients with left-hemisphere strokes with a relatively moderate aphasia (those with a severe aphasia could not be made to understand the instructions) and patients with strokes but without aphasia, of which only one had had a left-sided stroke. The results were dramatic: The group of patients *with* aphasia had a marked disturbance in performing this task, requiring much longer intervals between the two stimuli to reliably report the correct temporal order than the patients without aphasia. However, the patients with lesions in the roughly comparable region of the right hemisphere were also impaired on this task, as compared to normal subjects, although appreciably less so than the aphasic subjects. It seemed that my initial suspicion might be correct: The neural circuits in the left fronto-temporal area that support language functions appeared to be anatomically co-extensive with those that support temporal order judgments.

The feeling of elation that I experienced, particularly when a number of investigators confirmed and extended my findings of a deficit in temporal order judgments in aphasic subjects, is most accurately conveyed in Fig. 8.

Figure 8 also communicates another feeling, that of despair, which followed when I realized that I had not established any *causal relationship* between the deficit in temporal order judgments and the language deficit but merely an *association* between them. But, while *I* was frustrated by my failure to explain the aphasic language disturbance as damage to the physiological mechanisms that support the ability to keep track of the sequence of events, neuropsychologists were enthusiastic because another task had been discovered that was more impaired following a left- than a right-hemisphere lesion. Ignoring the fact that right-hemisphere damaged patients were also impaired, *they* concluded that my experiments had demonstrated the existence of another cognitive *function*, since referred to as temporal or sequential analysis, for which the left hemisphere is "specialized." And this is how "temporal analysis" ended up on the list of "specialized" left-hemispheric functions with which I began this chapter!

INFERRING HEMISPHERIC "SPECIALIZATIONS" IN NORMAL SUBJECTS

The unseemly haste with which the results of my experiments were added to the rapidly growing list of left-hemispheric specializations alerted me that

"By God, for a minute there it suddenly all made sense!"

FIGURE 8. Drawing by Gahan Wilson; copyright 1986. Reproduced by permission The *New Yorker* Magazine, Inc.

any report of a right/left performance asymmetry would inevitably suffer the same fate. The highly favorable response to my papers was an early warning sign, I thought, that the burden of proof was rapidly shifting to one in which the investigator would have to demonstrate that any right/left performance asymmetry he had found in a cognitive task was *not* a manifestation of a hemispheric "specialization": He would face the impossible task of having to prove the negative. This suspicion proved to be prophetic!

The most dramatic evidence confirming this prophesy can be seen in the huge body of research whose explicitly stated goal is to infer hemispheric specialization for various cognitive functions in *neurologically intact subjects*. Although cognitive psychologists had used normal subjects in their studies for many years, the first *concerted* attempt to infer hemispheric specialization of cognitive functions in normal subjects began with a series of papers by Kimura in the 1960s (1961a; 1961b; 1964; 1967).

Kimura's conclusions had such a major effect on *all* subsequent research on hemispheric specializations that a brief summary of her work is essential. Kimura used a technique, originally invented by Broadbent (1954), now universally known as "dichotic listening," in which two *different* acoustic signals are presented *simultaneously* to the right and left ears using earphones. As can be seen in Fig. 9, one series of spoken digits, for example 1-8-9 is delivered to one ear at the same time that another, say 6-3-4 is presented to the other ear. The subjects' task is to report all the digits heard in any order they wish. When this task was given to patients with lesions in the temporal lobe, before and after unilateral lobectomies that spared the primary auditory cortex, they exhibited a modest post-operative decrease in the number of digits reported from the ear *contralateral* to the lobectomy.

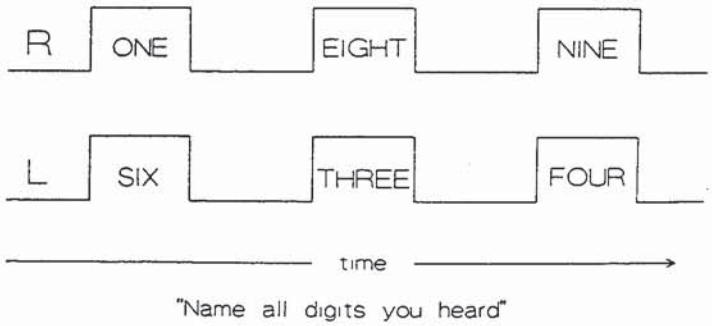

FIGURE 9. Diagrammatic representation of the dichotic listening procedure of Kimura. Three spoken digits (one, eight, and nine) presented to the right ear via earphones simultaneously with three digits (six, three, and four) presented to the left ear. The abscissa represents time.

The decreased contralateral performance was more marked, however, in those patients who had a *left* rather than a right temporal lobectomy.

Kimura also made use of another technique, which had just been introduced in Montreal by Wada and Rasmussen (1960), in which a rapid injection of a barbiturate into one or another carotid artery goes initially to one hemisphere and, so to speak, "puts it to sleep" briefly. When injected into the carotid on the side of the individual's so-called "dominant" hemisphere he develops a transient aphasia. Correlating the subject's performance on the dichotic listening task with the side of injection that resulted in transient aphasia, Kimura reported that patients with left-sided speech centers, the usual location in right-handed subjects, tended to have higher recognition scores for the digits presented to the right ear, whereas patients with right-sided speech centers tended to have better performance for the digits played to their left ear. Finally, a group of normal right-handed subjects, whose speech centers would be expected to be located in the left hemisphere, exhibited a superior performance in recognizing the words delivered to the right ear than those to the left ear. In sum, Kimura found that the ear superiority with dichotic presentation of words was *correlated* with the hemisphere which, when anesthetized, produced an aphasia. Clearly, the next step was to explain this correlation.

Unlike the visual system, where information presented to one visual *half-field* is sent to the visual cortex in the opposite hemisphere, in the human auditory system information presented to *one ear* is sent to the auditory cortex in *both* hemispheres. Because each hemisphere receives input from both ears, the destruction of one auditory cortex does not result in deafness of the opposite ear. Why then should there be a right-ear superiority for dichotically presented speech sounds? Kimura (1967) based her explanation on physiological evidence (Rosenzweig, 1951; Tunturi, 1946) in cats and dogs that the electrophysiological response in each auditory cortex is slightly larger for a *click* delivered to the opposite ear than for one delivered to the ear on the same side. She said, "The explanation for the right-ear superiority on the digits test, then, was that the right ear had better connections with the left hemisphere than did the left ear, and since the left hemisphere was the one in which speech sounds were presumably analyzed, the right-ear sounds had the advantage of having better access to these speech centers. In the case where speech is represented in the right hemisphere, however, the opposite pattern of ear-superiority should occur, since here it is the left ear which has the favoured connections" (p. 164). This has come to be known as the direct/indirect access theory.

Kimura was also aware, however, that the difference between the ipsilateral and contralateral physiological response to clicks was small, and she adopted Rosenzweig's suggestion that the stronger response in the primary auditory cortex produced by the contralateral signal might partially

"occlude" or "suppress" the information from the weaker ipsilateral stimulus—in effect making it still weaker functionally. Although this occlusion or suppression effect would be present in *both* auditory cortices, the *unsuppressed* information from the right ear would have *direct access* to the centers in the left hemisphere that are specialized for language, whereas the unsuppressed left-ear information in the right hemisphere would have only *indirect access* to the left hemisphere language centers via the corpus callosum. Because the unsuppressed auditory information in the right auditory cortex had a longer path to reach the language center in the left hemisphere, Kimura assumed that it would be somewhat degraded. Thus in the dichotic listening paradigm it was assumed that the language centers in the left hemisphere directly receive non-degraded, unsuppressed information from the *right* ear and indirectly receive degraded, unsuppressed information from the *left* ear.

In 1964 Kimura provided what then appeared to be the clinching piece of evidence that her assumptions were valid: Neurologists previously had noted that impaired performance in some musical tasks was more common with right- than with left-hemisphere damage. By the 1960s this was expressed in the prevailing jargon as a right hemispheric "specialization" for music. If her assumptions were correct, and two different *musical passages* were presented dichotically, a *left*-ear superiority would be expected. Using dichotically presented Baroque melodies, Kimura reported a *left*-ear recognition superiority in normal subjects. This later report appears to have convinced many investigators that by using the dichotic listening technique with the appropriate material, one could infer the hemisphere which was specialized for performing any auditory task. This conviction was reinforced by two reports in 1968 (Milner, Taylor, & Sperry, 1968, and Sparks & Geschwind, 1968) that split-brain subjects reported very few of the spoken digits presented to the left ear, and some of them claimed that they heard *nothing* in the left earphone at all. These experiments seemed to confirm the validity of Kimura's second assumption of a suppression of the weaker ipsilateral auditory signal by the stronger contralateral signal in each hemisphere: The split-brain subjects appeared to be deaf for digits presented to their left ear when other digits were presented simultaneously to their right ear, but heard the digits *normally* in their left ear when there were *no* digits presented in the right ear.

It seemed obvious that Kimura's reasoning should be as applicable to the visual as to the auditory system. Indeed, because of the anatomy of the visual system the suppression assumption was not even required. All one needed was the first assumption—of differential access to the hemisphere that was "specialized" to perform the task. The direct/indirect access assumption appeared to explain the much earlier observations of others that words and letters presented to the right visual half-field, and thus directly

projected to the left hemisphere, were recognized with higher accuracy or with a shorter reaction time than those projected to the left visual half-field and then presumably transmitted with degradation to the left-hemisphere language center.

IMPACT OF THE DIRECT/INDIRECT ACCESS THEORY

Kimura's work had a major impact in four respects. *First*, it convinced many investigators that the simultaneous presentation of different categories of information to the two ears, the two visual half-fields, or the two sides of the skin surface offered a powerful method by which they could identify many different types of hemispheric specializations in a readily available *normal* population whose performance would not be contaminated or obscured by the effects of brain damage.

Second, it created a thriving "low-tech" cottage industry in which all one needed was a cheap two-channel tape recorder and/or slide projector with a shutter and a little bit of electricity. What previously had been a well-guarded academic preserve of a few neurologists and neuropsychologists having access to patients with rare neurological lesions had now opened up, more democratically, to anyone with an interest in the subject of hemispheric specialization.

The *third* major impact of Kimura's work was that she redefined the concept of "specialization." Prior to her work, the cognitive function of a brain region, its specialization, had been defined in terms of the symptoms produced when it was damaged. But even this *flawed* definition of "specialization" is *inapplicable* to the normal subject who has no cerebral damage. Instead of using *symptoms* to define the specialized cognitive function of a damaged cerebral area, Kimura used the *right/left performance asymmetry* to define the specialized cognitive function of an undamaged hemisphere: In essence, *the performance asymmetry now became the symptom*. If a dichotic listening or a tachistoscopic experiment in normal subjects revealed a left-ear or a left visual half-field superiority, then this operationally defined the existence of a right-*hemispheric* specialization for the task. Conversely, a right-ear or a right visual half-field performance superiority constituted *prima facie* evidence for a left-*hemispheric* specialization. The word "hemispheric" has been emphasized in the two preceding sentences to call attention to the fact that with these methods, the localization of the purported cognitive function could be assigned to no anatomical region smaller than an entire hemisphere.

The *last* major reason for the enthusiasm was the guaranteed success of *any* experiment with these methods. This assurance of success is a direct logical consequence of the new way of defining cognitive specializations.

Let me amplify: In such experiments there are only three possible results, *and every one is publishable*! Using some type of visual, tactile, or auditory material, one *must* find a right, left or no performance superiority—there is no other possible outcome. Whatever the result, an important contribution to the field of laterality research had been made, which, of course, necessarily leads to further experiments, each with a similarly guaranteed outcome. And, performing experiments with such a guaranteed outcome also improves one's chances of obtaining academic tenure!

A simple example should suffice: If an experiment demonstrated a *left* visual field performance superiority in recognizing, detecting or reporting some *novel* type of visual shape or pattern, this provided critically important "converging evidence" further defining the precise characteristics of the already-claimed right-hemispheric superiority for visual-spatial functions. However, if the experiment gave rise to the opposite result, this would be particularly worth publishing because it would mean that the *left* hemisphere is specialized for the processing of the *particular* visual-spatial stimuli that were used in that experiment. Finally, if no asymmetry was observed, this too would be important evidence that for the *particular* visual-spatial stimuli used, the two hemispheres are equally "specialized."

This is known on the street as a "win-win-win situation" and had the prophesied result that hundreds of different "hemispheric specializations" were claimed within a relatively short period of time. Thus an investigator might ask, and inevitably one did (Gordon, 1970), whether the left-ear performance superiority in Kimura's experiment that used melodies was truly a reflection of right-hemispheric specialization for all musical tasks or an indication of a right-hemispheric specialization for melodies only. Although he found *no* performance asymmetry when he used dichotically presented melodies and digits, thus *failing to confirm* Kimura's original claims for these two types of stimuli, he did find a weak, but statistically significant, *left*-ear superiority in the recognition of dichotically presented musical chords created with an electric organ. In the field of laterality research even the *failure* to replicate the reports of another investigator has a special status: Unlike other scientific disciplines, where a failure to replicate may be published because it is a possible adverse reflection on the validity of the *initial* claim, in the field of laterality research the failure is usually dismissed by the *later* investigator on the grounds of minor methodological differences! Indeed, since it is almost impossible to duplicate another's experiment exactly, the chronic failure to get the same results in closely similar experiments has been taken as evidence of the exquisite subtlety of hemispheric specializations!

I hate to spoil the party, but it is time to call your attention to the fact that we are dealing with a closed conceptual system resembling the Ptolemaic

theory of planetary motions, where any new observation was accounted for by postulating yet another planetary epicycle. In this case, any new experimental observation is accounted for by postulating another, heretofore unrecognized, "hemispheric specialization." In common with other closed conceptual systems, this new way of defining hemispheric specializations *cannot be falsified* by any new right/left performance asymmetry that is discovered. But the most devastating characteristic of such closed theoretical systems is that they inhibit research on alternative explanations of the phenomena or direct tests of the assumptions that are the basis of the system, at least among the "true believers."

The views I expressed in this chapter are not likely to be received enthusiastically by these true believers. Since the *facts* I have presented are indisputable, the only charge that could be leveled against me is that I have made too much of the way words are used in the field of laterality research, and that the arguments I have advanced represent nothing more than a semantic dispute. I have already dealt with the claim that the concept of "specialization" is used merely as a short-hand way of restating the existence of a correlation between the site of a brain lesion and some type of cognitive deficit. Even a cursory reading of the literature reveals that the true believers actually mean that the purported cognitive function had been *performed by* and *in* the piece of brain tissue prior to its damage. When they refer to studies of normal subjects, they actually mean that the purported cognitive function is *performed by* and *in* the hemisphere presumed to be responsible for the superior performance. This is *not* a matter of semantic dispute: Kimura's direct/indirect access theory is *explicitly* and *unambiguously* based on the idea that the purported cognitive function is performed within the hemisphere that has direct access to the relevant information.

The real problem, however, is that we do not at present understand the cognitive function of *any* brain area, let alone an entire hemisphere! For example, what is the cognitive function of the left fronto-temporal cortex, which, when damaged, results in aphasia? And what is the cognitive function of the striate cortex, which, when damaged bilaterally, results in blindness? To declare that these areas are specialized for language and vision respectively is logically equivalent to, *and no less banal* than, the declaration that the legs are specialized for walking since walking is seriously impaired without legs! Unfortunately, when the speaker is referring to that most fascinating of all organs, the brain, the unsophisticated listener, and many neuropsychologists as well, are led to believe that the field of cognitive neuroscience has achieved, to use Chairman Mao's phrase, "A great leap forward."

In the following chapter I will show how this logical error is actually a great lurch *sideways* into a scientific dead end.

Neuropraxis (januari 2001) 5:52–56
DOI 10.1007/BF03070983

ARTIKELEN

Persoonlijkheidsverandering door hersenletsel: een veelkoppig monster

J. Zegerius

Abstract Stoornissen van de frontale en temporale kwabben leiden vaak tot beperkingen en handicaps op het gebied van de cognitie, de emoties en het gedrag. Er treden meestal veranderingen op in het sociaal functioneren. Bovendien kunnen secundaire gedragsveranderingen een gevolg zijn van negatieve interacties met de omgeving. Dit alles heeft tot gevolg dat een persoonlijkheidsverandering na een hersenletsel vaak het beeld bepaalt en dat de patiënt en zijn verwanten hiervoor hulp zullen zoeken. Hulpverleners zullen de verschillende dimensies van de veranderde persoonlijkheid moeten analyseren om rationeel te kunnen gaan behandelen.

Hersenletsel, door wat voor oorzaak ook, heeft vaak gevolgen voor het cognitief functioneren, de emoties en het gedrag.Veranderingen van sociale en empathische processen, executieve functies, motivationele en attentiemechanismen leiden tot een complexe combinatie van beperkingen en handicaps, die zich in eerste instantie vaak uiten als een persoonlijkheidsverandering. Op de voorgrond staat immers de verandering van het gebruikelijke gedragspatroon en de karakteristieke persoonlijkheid van de patiënt. Naast de directe gevolgen van het hersenletsel kan een secundaire gedragsverandering door interacties met de omgeving het beeld in een latere fase nog verder kleuren. Het is in het algemeen niet gemakkelijk om in de veelheid van symptomen onderscheid te maken tussen primair cognitieve, emotionele of gedragsdefecten en om te bepalen wat het aandeel is van interacties met de omgeving. Een persoonlijkheidsverandering door hersenletsel is dan ook een veelkoppig monster.

Na een hersenletsel kan een verandering van de persoonlijkheid veel meer op de voorgrond staan dan neurologische uitvalsverschijnselen, zoals motorische en sensibiliteitsstoornissen, die zelfs geheel afwezig kunnen zijn. De gevolgen van functiestoornissen van de temporaalkwabben en de prefrontale gebieden zijn hier een goed voorbeeld van. In dit artikel zal vooral worden ingegaan op de klinische betekenis van deze syndromen.

De frontale syndromen

De frontale kwabben worden gevormd door de hersenstructuren die vóór de sulcus centralis gelegen zijn. Zij bestaan uit zeven gespecialiseerde gebieden, die taken hebben op het gebied van motoriek, taal, planning en organisatie, initiatief en motivatie, geheugen, emotionele en sociale processen, en attentiemechanismen. De functies zijn zo divers dat laesies van deze gebieden tot een complexe combinatie van verschijnselen kunnen leiden.

Het veelgebruikte begrip prefrontale cortex duidde aanvankelijk die delen van de frontale kwabben aan waar de thalamus op projecteert, maar tegenwoordig wordt het meestal gebruikt als aanduiding van de frontale cortex minus, de motorische gebieden (area's 4, 6 en 8 van Brodmann, zie fig, 1a en 1b). Hiermee is het begrip prefrontale cortex synoniem geworden met onderdelen van de frontale kwabben die overwegend een rol spelen bij de cognitieve functies, taal, emoties, gedrag en sociaal functioneren.

Functiestoornissen van de prefrontale cortex veroorzaken daarom een breed spectrum aan afwijkingen die zich vooral voordoen op cognitief, gedragsmatig en emotioneel gebied. Oorzaken van deze functiestoornissen

hoeven zich beslist niet in de frontale gebieden te bevinden, maar kunnen ook gelokaliseerd zijn in de vele verbindingen die zich op afstand bevinden, bijvoorbeeld in de hersenstam, het cerebellum, de thalamus of witte stof. De prefrontale cortex is namelijk via meerdere neuronale netwerken verbonden met hersenstructuren die een essentiële rol spelen bij cognitieve en emotionele functies, zoals de nucleus caudatus, de thalamus en limbische gebieden (McPherson en Cummings, 1998). Er is ongeveer bekend welke gebieden hierbij betrokken zijn en op welke wijze deze gebieden in verbinding staan met andere onderdelen van de hersenen. De klinische praktijk gebruikt een gesimplificeerde onderverdeling in dorsolaterale, orbitofrontale en mesofrontale gebieden en daarbij behorende defecten en syndromen. Het dorsolaterale prefrontale gebied omvat ruwweg de Brodmann area's 9, 10, 44, 45 en 46, het orbitofrontale gebied de area's 11, 12 en 47 en het mesofrontale gebied de area's 24, 25 en 32. (zie fig. 1a en 1b).

Lateraal aanzicht van de cytoarchitectonische map van Brodmann (frontale en temporale gebieden)

Mediaal aanzicht van de cytoarchitectonische map van Brodmann (frontale en temporale gebieden)

Harlow beschreef in 1868 als eerste een frontaal syndroom in de geschiedenis van Phineas Gage. Door een ongeval met springstof was een ijzeren staaf Gage's schedel binnengedrongen, waarbij de orbitofrontale delen van de frontale kwabben ernstig beschadigd waren.Gage was daarna veranderd in een geagiteerde, ontremde, empathie-arme man met asociale trekken en executieve stoornissen. Het effect op zijn persoonlijkheid en zijn gedrag is onderwerp geweest van vele uiteenzettingen over de gevolgen van frontale letsels.

Dankzij uitgebreid onderzoek bij patiënten met frontale functiestoornissen is het inmiddels goed mogelijk om een differentiatie aan te brengen in de symptomatologie. Tegenwoordig maakt men klinisch onderscheid tussen een drietal frontale syndromen: een apathisch/amotivationeel, een gedesorganiseerd en een ontremd type waarbij de laesie of functiestoornis respectievelijk mediofrontaal, dorsolateraal en orbitofrontaal wordt gelokaliseerd (zie tabel 1). De verschillende onderdelen van deze frontale syndromen kunnen overigens gemengd voorkomen. Bij nadere beschouwing kunnen zowel een afname van vaardigheden, leidend tot bijvoorbeeld apathie en initiatiefarmoede, als het ontstaan van nieuwe verschijnselen, zoals ontremming en agressie, het gevolg zijn van frontale laesies. Men kan ook spreken over inhibitie en excitatie, of over negatieve en positieve symptomen zoals in de psychiatrie gebruikelijk is.

Tabel 1 Symptomatologie van de frontale syndromen (naar Cummings en Trimble, 1995)

Dorsolateraal prefrontaal syndroom
• verminderde verbale fluency
• verminderd abstractievermogen
• oordeel- en kritiekstoornissen
• geheugenstoornissen, vooral van het werkgeheugen
• stoornissen van de planning en organisatie
• 'motor impersistence' (onvermogen om een motorische handeling vol te houden, bijvoorbeeld de ogen gesloten houden. Vooral bij rechtszijdige laesies)
• utilisatiegedrag (het automatisch gaan gebruiken van voorwerpen in de omgeving, zoals: ongevraagd gaan vegen als er een bezem staat, gaan schrijven met een pen, et cetera) en
• 'environmental dependency' (het afhankelijk-zijn van de omgeving voor het uitvoeren van handelingen, zoals: ongevraagd in een tuin bloemen gaan plukken, in een keuken gaan afwassen, et cetera)
•attentiestoornissen
Orbitofrontaal syndroom
• ontremming
• impulsiviteit
• tactloosheid
• stemmingswisselingen
• ongepaste grapjes
• decorumafname
• empathie-afname
• oordeels- en kritiekstoornissen
Mediofrontaal syndroom
• apathie
• abulie
• mutisme
• initiatiefarmoede
• psychomotore traagheid

Symptomatologie van de syndromen (naar Cummings en Trimble, 1995)

De stoornissen van attentiefuncties, de executieve functies en het sociaal functioneren kunnen zich in eerste instantie presenteren als een verandering van de persoonlijkheid. Vooral attentiestoornissen spelen hierbij steeds weer een belangrijke rol. De prefrontale cortex is immers bij nagenoeg alle attentietaken actief, zodat prefrontale laesies kunnen leiden tot afwijkingen van de vele facetten van deze zo essentiële functie. Er kunnen stoornissen ontstaan van de volgehouden attentie (de vigilantie), van de verdeelde attentie, van de mogelijkheid om de attentie te verplaatsen (het 'shiften') en van het vermogen om een ongewenste respons te inhiberen. In de klinische praktijk kan de patiënt of zijn omgeving over een slechte concentratie klagen, waarvan snel duidelijk zal zijn dat hieraan een gestoord attentiemechanisme ten grondslag kan liggen. Ook vluchtigheid, snel afgeleid zijn, kortaangebondenheid, dwangmatigheid en van-de-hak-op-de-tak springen kunnen uitingen zijn van gestoorde attentiefuncties, maar deze verschijnselen worden niet altijd direct hieraan toegeschreven. Problemen met het onderdrukken van een ongewenste respons uiten zich in de praktijk onder meer als het zich ongewenst met van alles bemoeien en het reageren op allerlei omgevingsprikkels. Een voorbeeld hiervan is het utilisatiegedrag, waarbij de patiënt voorwerpen uit de omgeving direct gaat gebruiken (zie in tabel 1). De responsinhibitie kan onderzocht worden met het 'go-no go'-model: de patiënt steekt zijn duim op als de onderzoeker tweemaal onder het tafelblad klopt en doet dit niet als hij éénmaal klopt.

Stoornissen van de executieve functies, zoals planning en organisatie, en van motivationele mechanismen kunnen leiden tot chaos en zelfverwaarlozing. Ook hieraan liggen niet zelden attentiestoornissen mede ten grondslag.

Veranderingen van het sociaal functioneren uiten zich als empathie-afname, onverschilligheid, egocentriciteit, cognitieve rigiditeit, decorumafname en agressiviteit. Vooral Damasio heeft gewezen op het feit dat cognitieve functies bij frontale functiestoornissen geheel intact kunnen blijven, waardoor zich na een hersenletsel alleen stoornissen van het sociaal functioneren kunnen manifesteren (Damasio, 1995).

Het zal duidelijk zijn dat al de genoemde verschijnselen zich primair kunnen uiten als een verandering van de persoonlijkheid, zodat voor de omgeving op de voorgrond staat dat de patiënt zo veranderd is.

Het is de ervaring dat de persoonlijkheidsverandering bij mensen met een al dan niet bekend hersenletsel in eerste instantie soms te gemakkelijk als een primair psychische disfunctie wordt beschouwd, zeker wanneer cognitieve stoornissen afwezig zijn. Vaak wordt automatisch naar externe oorzaken gezocht, die niet zelden worden gevonden, zoals relatieproblemen, onmin op het werk en seksuele functiestoornissen. Zij zijn meestal niet de oorzaak van de veranderde persoonlijkheid maar een gevolg. Ook komt het vaak voor dat motivationele problemen, zoals apathie en initiatief armoede ten onrechte worden aangezien voor een depressie.

Uit het voorgaande blijkt duidelijk dat hét frontaal syndroom niet bestaat. Er is een aantal verschillende frontale syndromen met ieder een min of meer karakteristieke symptomatologie met aspecten van inhibitie en excitatie. Verder is het duidelijk dat de frontale syndromen veroorzaakt kunnen worden door laesies in alle gebieden die verbonden zijn met de frontale kwabben, en dat zijn er vele. Met andere woorden: het begrip 'frontaal' duidt zeker niet altijd op de plaats van de laesie.

De temporale syndromen

De temporaalkwab is gelegen onder de fissura Sylvii en omvat aan de laterale zijde ongeveer de area's 20, 21, 22, 37, 38, 41 en 42 van Brodmann (zie fig. 1a en 1b). Aan de mediale zijde omvat hij een aantal oudere lagen cortex. Het is meer dan honderd jaar bekend dat laesies van temporale gebieden aanleiding kunnen geven tot geheugenstoornissen, taalstoornissen en veranderd gedrag. Dit volgt uit het feit dat mediotemporale structuren ' met name de hippocampus ' essentieel zijn voor het geheugen, dat het gebied van Wernicke voornamelijk de temporale area 22 omvat en dat temporale structuren, zoals de amygdala, onderdeel van het temporolimbische systeem zijn. Na dubbelzijdige mediotemporale laesies, bijvoorbeeld door een schedeltrauma of een herpes simplexencefalitis, kunnen ernstige geheugenstoornissen ontstaan, in het bijzonder een anterograde amnesie. Taalstoornissen zijn een gevolg van laesies van het gebied van Wernicke of van verbindingen met dat gebied.

De invloed van temporolimbische laesies op psychische functies en gedrag is vooral bekend geworden uit onderzoek naar patiënten met temporale epilepsie. Veranderingen van emoties en affect, gevoelens van derealisatie en depersonalisatie, déjà-vu sensaties, psychoseverschijnselen, agressie, verwardheid en dissociatie, angst- en paniekgevoelens en bizar gedrag zijn bekende manifestaties van temporale epileptische activiteit. Er kan vóór, tijdens of tussen epileptische aanvallen sprake zijn van een op schizofrenie lijkende psychose. Het aandeel van de temporale kwabben bij het ontstaan van schizofrenie heeft ertoe geleid dat een belangrijk deel van het moderne schizofrenie-onderzoek zich richt zich op veranderingen in temporale gebieden, zoals een aberrante cytoarchitectuur in de hippocampus en mediotemporaal weefselverlies.

Tabel 2 Een aantal kenmerken van het 'temporale epilepsie karakter'

• emotionaliteit
• euforie
• agressiviteit
• veranderde seksualiteit (libido-afname, hypo- of hyperseksualiteit, parafilieën)
• obsessieve trekken
• neiging om uit te weiden en te herhalen
• plakkerigheid in het contact ('stickyness')
• hypergrafie (schrijven van uitgebreide dagboeken, ingezonden brieven, lijvige romans)
• hypermoralisme
• hyperreligiositeit
• humorloosheid
• achterdocht
• toegenomen belangstelling voor filosofie, paranormale fenomenen, etc.

Van belang, maar toch nog steeds speculatief is het zogenaamde 'temporale epilepsie karakter', zoals dat vooral door Geschwind is beschreven (zie tabel 2). Het gaat om een combinatie van persoonlijkheidskenmerken die bij patiënten met temporale epilepsie vaak gezien zouden worden, met als meest prominente kenmerken hypergrafie, hyperreligiositeit, plakkerigheid in het contact en een veranderd seksueel functioneren (Bear en Fedio, 1997).

Een specifiek temporaal syndroom is het Klüver-Bucy-syndroom. Het treedt op bij dubbelzijdige laesies van de voorste temporale gebieden, die onder meer kunnen ontstaan door een schedeltrauma, een herpes simplexencefalitis of een fronto-temporale degeneratie, zoals bij de ziekte van Pick. Klüver en Bucy beschreven in 1939 een reproduceerbare gedragsstoornis bij resusapen na een bilaterale temporale lobectomie. De apen ontwikkelden een visuele agnosie ('psychic blindness'), een dwang om alle objecten oraal te onderzoeken, een eetontremming, een onweerstaanbare neiging om aan te raken, een verlies van normale angst- en woedereacties en een toegenomen seksuele activiteit. Deze verschijnselen ontstonden niet na een unilaterale laesie.

Het eerste Klüver-Bucy-syndroom bij de mens is beschreven in 1955. Het ging om een negentienjarige man die een dubbelzijdige temporale lobectomie onderging vanwege onbehandelbare epilepsie. Een compleet beeld is in 1975 beschreven bij een twintigjarige man met een herpes simplexencefalitis. De hyperoraliteit uitte zich bij deze patiënt als het ongeremd tot zich nemen van voedsel. Er was een dwangmatige neiging om alles in de omgeving aan te raken of vast te pakken en vervolgens in de mond te stoppen. Sindsdien is het beeld geregeld beschreven bij de mens, meestal incompleet, waarbij dan gesproken wordt over een partieel Klüver-Bucy-syndroom (Lilly e.a. 1983). Het syndroom komt in zijn partiële vorm waarschijnlijk vaker voor dan wel eens gedacht wordt. Een opsomming van de symptomen staat in tabel 3.

Tabel 3 Symptomen van het Klüver-Bucy-syndroom

• apathie
• extreme meegaandheid ('pet-like compliance')
• verlies van angst en agressie
• ook woede- en agressieve aanvallen (explosies) kunnen optreden
• visuele agnosie (ook auditieve en tactiele agnosie zijn bekend)
• hypermetamorfosis: manuele en vervolgens orale exploratie van de omgeving
• eetontremming: boulimie/hyperfagie
• veranderd seksueel gedrag
• geheugenstoornissen
• fatische stoornissen, meestal van het type Wernicke
• overige gedragsveranderingen, zoals dwangmatigheid, zucht naar orde en nauwkeurigheid, 'plakkerigheid', kleptomanie

Het ongeremd eten kan zich in de kliniek uiten als het gulzig eten van grote hoeveelheden voedsel, meestal met een vergroving van de tafelmanieren. Vaak wordt het voedsel van medepatiënten gepakt en worden hun kopjes leeggedronken. De ijskast is niet veilig en wordt soms geplunderd. Het ongeremd aanraken richt zich op medepatiënten, begeleiders en hulpverleners. Het wordt vaak gezien als ongewenste intimiteit, zeker als het gepaard gaat met seksuele ontremming in woord en gebaar. Ook is het mogelijk dat de patiënt dwangmatig eigendommen van anderen wil vastpakken en soms verzamelen, hetgeen kan leiden tot een vorm van kleptomanie. Ook bij ernstig geïnvalideerde patiënten kunnen vormen van gedrag voorkomen waarbij verzorgers ' al dan niet stevig ' vastgepakt worden en bijvoorbeeld hun handen in de mond worden gestopt. Al deze verschijnselen komen voor in klinieken waar patiënten met hersenletsel verblijven, maar zij worden vaak niet als partieel Klüver-Bucy-syndroom herkend. Hierdoor blijven zinvolle medicamenteuze interventies tenonrechte achterwege.

Conclusie

Zowel bij de frontale als bij de tempo rale syndromen is de vaak prominent aanwezige persoonlijkheidsverandering meestal het gevolg van een complexe combinatie van verschillende cerebrale functiestoornissen en van een gestoorde interactie met de omgeving. Het is aldus beschouwd een veelkoppig monster. Bij een

verandering van de karakteristieke persoonlijkheid en het gedragspatroon na een hersenletsel behoort onderzocht te worden wat het aandeel is van cognitieve, emotionele, sociale en gedragsdisfuncties en wat de bijdrage is van de interactie met de omgeving. Het is voor de hulpverleners en vooral ook voor de familie en verwanten van belang dat de persoonlijkheidsverandering nauwkeurig geanalyseerd wordt om de behandeling en begeleiding te optimaliseren. Zo kunnen medicamenteuze behandelingen, psychotherapeutische interventies en vormen van neurorehabilitatie rationeler worden toegepast.

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De artikelen op de volgende pagina's geven een indruk van de ontwikkelingen in de neurowetenschappen. Enkele artikelen zijn volledig weergegeven, sommige alleen met de eerste pagina (of iets meer). De nadruk ligt bij deze artikelen op de probleem-analyse en de koppeling tussen brein en gedrag.
Van elk artikel is de samenvatting (abstract) op z'n minst interessant en voor de geïnteresseerde lezer zijn uiteraard de volledige artikelen verkrijgbaar.

NEUROPSYCHOLOGICAL REHABILITATION, 2001, *11* (3/4), 197–200

Crossing the i's and dotting the t's

Una Holden

Years ago I published an article in an American journal complaining that the use of test batteries was more appropriate for hens than people. Although such routine testing is no longer the norm there does appear to be a continued problem with providing appropriate assessment procedures for older people suffering from some form of cognitive or behavioural change.

The obvious first question here is why are tests required at all? There are various reasons which have been listed and recognised for many years, for instance, to establish a baseline or to identify changes in research programmes. However, if rehabilitation programmes are to be effective, it is absolutely essential to ascertain which abilities are retained in good working order, which are partially damaged, and which are severely impaired.

Over and over again errors in perception of a person's problems in functioning are demonstrated both in hospital and residential home as well as in a person's own home. Setting unrealistic goals only adds to the individual's stress and confusion. To treat someone with dysphasia as though he or she is a parrot and will eventually learn to copy through constant repetition, or to shout continually at a person with receptive dysphasia expecting that it will make him or her comprehend, will hardly prove to be successful as retraining programmes. Furthermore, misunderstandings can add emphasis to the assumption that a dementia is present. Not only those observing, but also the unfortunate patient him or herself, can be led to believe that serious degeneration is occurring. If a person presents with behavioural and cognitive changes it is no longer appropriate to conclude that "dementia" is the cause.

Unfortunately, despite tremendous progress in understanding the enormous number of conditions affecting brain function, the term "dementia" continues to be misused. Far too many staff and members of the public believe it to be a condition in its own right, see it as causing total damage to the brain, as

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http://www.tandf.co.uk/journals/pp/09602011.html DOI:10.1080/09602010042000006

irreversible, and as the explanation of every observed change in cognition or behaviour in people over the age of 65 years. Unfortunately, this persistent fallacy is held by older people themselves and frequently they react badly to self-perceptions of changes in ability, jumping to incorrect conclusions which can lead to depression and a pseudo-dementia state. In order to avoid errors, thorough investigations should be carried out, but this does not necessarily mean putting an individual in an examination situation. Although relevant testing is required, it should not be the initial step.

Good history taking can establish a person's previous social, educational, and occupational background. Careful observation of the person's behaviour and responses can prove invaluable. Challenging behaviour may be explained by watching, considering the possibilities of apraxia, agnosia, or neglect of one side. Does the person fight staff trying to feed, wash or dress him or her, does the person get lost, has he or she shown difficulty in recognising relatives, friends or even objects? What happens when the person is left alone to do things, is dressing suddenly done with ease? It is possible that a person may have, for example, an apraxia yet is capable of functioning normally apart from not being able to cope with being told what to do? Most human beings of any age are not too happy about being "told what to do"!

Have staff been trained to recognise or look for specific changes in ability or behaviour? Often there can be confusion over observed behaviour, and the person's difficulties may be seen as a conscious attempt to annoy or to seek attention instead of as clues to a more accurate interpretation. Staff and relatives may not appreciate that because a person sings but refuses to talk to them, he or she is not being deliberately rude—the fact that music and rhythm are located in another part of the brain is the real explanation.

Another situation when behaviour can be perceived as deliberate rudeness or offensive in some way can occur with frontal damage. Perseveration is a common indication of such damage and although it is usually demonstrated in the constant repetition of words or phrases and sometimes gestures, it can also appear in an unusual form. A person suddenly seems to ignore what is being said and continues to talk about a particular subject, so staff and relatives think that deafness, rudeness or boredom with the conversation are possible explanations. The person's attention has become focused on a subject and thought processes have got caught in a groove—the "stuck-needle syndrome" of frontal lobe damage. The only rehabilitation programme to minimise perseverations of this sort is one of sharp distraction—clapping hands, ringing bells or dropping something may provide the mental jerk that breaks the connection! Other forms of frontal damage such as poor sequencing and difficulties with using abstract thought need to be investigated with specific tests and simple retraining methods initiated. Day-to-day material such as shopping lists, order in dressing, in washing and making sense of mixed up picture stories are all useful tools.

Memory problems are cited as the start of a degenerative condition, but what form do these so-called memory lapses take? Does the teenager who forgets to pass on a message suffer from dementia? Does the person who climbs to the top of the stairs and wonders why he or she went up them, but recalls why after returning to the bottom, suffer from dementia? Memory lapses are common to everyone; those with heavy demands on recall are liable to forget some detail but are well within normal limits. The belief that memory error is a sign of deterioration is just an added stress which can precipitate misunderstandings of observed and perceived functions.

History taking and observation need also to be amplified by an awareness of the psychological and social situation of an individual. How do other people treat the person, what attitudes do they have, what social outlets are there, what are family relationships like? If the person is a patient on a ward, or a resident in a home, what knowledge do the staff have, what procedures are used, what attitudes and beliefs are current?

The first step in "diagnosis" is to ascertain if the problem is simply a matter of misconception, either by the patient, or the staff, or relatives. Why has a person's social behaviour deteriorated—is it because there has been no opportunity to employ social skills? Why does everyone think that a person is deteriorating—is it because the person has a high level of intelligence and has panicked because some errors in performance have arisen? Has the person been left alone for so long that depression has been the result and skills have not been employed and so have become rusty? Have the relatives or staff taken over and robbed the person of independence with the result that the unfortunate being has given up? All these are questions to be considered, along with a personal history. Rehabilitation takes on a different form when social and psychological influences are the root of the problem. Perhaps it is staff or relatives that require retraining! If the person has suffered a pseudo-dementia as a result of the social situation then therapy directed at restoring confidence and demonstrating retained abilities is required.

When a full history is available and no satisfactory explanation of changes has been found, more formal, relevant testing is appropriate. The emphasis must be placed on *relevant*. The person may be suffering from one of the many different degenerative conditions which damage part of the brain—from mere bruising of a small area to severe widespread damage. It is important, if at all possible, to identify the causal condition as well as identifying the functions and abilities affected, including the degree of impairment involved. The tests should cover not only basic intelligence, but should include useful neuro-psychological screening at levels commensurate with the person's basic level of ability. For instance if there is a problem with speech or language then tests will need to be modified or selected in order to obtain meaningful results. The rehabilitation programme and its goals should be planned to meet the needs of

the individual as well as seeking ways to strengthen, retrain or compensate for the specific impairments identified.

Without taking all aspects and influences into consideration the result will be too many crossed i's and dotted t's.

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Hemispatieel neglect: Meer dan slechts een aandachtsprobleem?

Samenvatting

Hemispatieel neglect wordt vaak geassocieerd met gelateraliseerde stoornissen in de visuele aandacht. Volgens veel van de huidige theorieën wordt neglect veroorzaakt door een gebrek aan aandacht voor een deel van de ruimtelijk informatie die patiënten moeten verwerken. Als gevolg daarvan negeren ze deze informatie. Er zijn echter problemen met deze standaarduitleg van neglect als een aandachtsprobleem. Hoewel een goede verklaring voor een gedeelte van de problematiek van neglectpatiënten, kunnen veel van hun problemen niet worden verklaard door een aandachtsprobleem. In dit artikel zullen we resultaten van studies bespreken die suggereren dat een gedeelte van de problemen die eerst werden geschaard onder een aandachtsstoornis, beter verklaard kunnen worden door een probleem in het bijwerken ('updaten') van locatie-informatie. In combinatie met een aandachtsprobleem, zou een probleem in het updaten van informatie kunnen leiden tot het verdwijnen van relevante informatie uit het bewustzijn. Mede doordat neglect een heterogene stoornis is, kunnen verfijningen in de beschrijving van de onderliggende stoornis tot verbetering leiden van diagnostiek en revalidatie.

Introductie

Na hersenbeschadiging kan hemispatieel neglect ontstaan, waarbij patiënten informatie in het contralesionale veld verwaarlozen. Dit fenomeen wordt niet veroorzaakt door een sensorisch of motorisch defect: patiënten zijn niet blind en zijn in staat een beweging naar objecten in het contralesionale veld te maken, maar de informatie wordt simpel-

weg genegeerd: ze gedragen zich alsof de helft van de wereld niet meer bestaat. Neglect wordt meestal gezien na schade aan de inferieure pariëtale cortex (Driver e.a., 1998) of de superieure temporale cortex (Karnath, 2001) en is in de chronische fase doorgaans het gevolg van beschadiging aan de rechterhemisfeer. In de acute fase wordt neglect ook na linkszijdige beschadigingen waargenomen (Driver e.a., 1998).

Neglect is een heterogene aandoening en heeft verschillende verschijningsvormen: het kan specifiek zijn voor een sensorische modaliteit, voor een motorisch domein, voor een regio in de ruimte of voor een egocentrisch of allocentrisch referentiekader (Mesulam, 1999). Het lijkt dan ook alsof neglect een verzamelnaam is voor een verscheidenheid aan problemen/stoornissen. Het is daarom misschien ook niet verrassend dat de theorievorming uitgaande van een unitair onderliggend mechanisme tot nu toe nog weinig succesvol is gebleken. In deze theorieën wordt neglect geassocieerd met gelateraliseerde verstoringen in de aandacht: ofwel een verstoring om aandacht naar het contralesionale visuele veld te verplaatsen (Kinsbourne, 1987; Mesulam, 1999) ofwel een verstoring om aandacht los te maken van het ipsilesionale visuele veld (Marshall & Halligan, 1989; Posner e.a., 1984). Het idee achter deze theorieën is dat neglect wordt veroorzaakt doordat patiënten geen aandacht besteden aan een deel van de ruimtelijk informatie die zij moeten verwerken met als gevolg dat zij deze informatie negeren.

Er zijn echter problemen met de standaarduitleg van neglect als een aandachtsprobleem. Veel van het gedrag van neglectpatiënten kan namelijk niet worden verklaard door een probleem met de aandacht, zoals het veelvuldig opnieuw bezoeken van elementen in het ipsilesionale veld. Daarnaast is aandacht een slecht gedefinieerd concept: wat betekent het precies dat een patiënt ergens geen aandacht voor heeft? En waarom zou een object dat geen aandacht krijgt niet tot het bewustzijn doordringen? Een gezond individu richt de aandacht ook niet op alle objecten in de ruimte, maar zal deze informatie niet negeren. In dit artikel zullen we bespreken welke facetten van neglect niet verklaard kunnen worden door een aandachtsprobleem en ingaan op recente aanwijzingen dat neglect mede veroorzaakt lijkt te worden door een probleem met het updaten van visuele informatie.

Problemen met uitleg van neglect als aandachtsprobleem

In de kliniek wordt neglect vastgesteld door een neuropsychologische testbatterij, waarin pen-en-papiertesten worden gebruikt om het bewustzijn voor bepaalde gedeeltes van het visuele veld in kaart te brengen. Een voorbeeld hiervan is de 'star cancellation'-taak, waarin de patiënt wordt gevraagd de kleine sterren door te strepen en de overige elementen te negeren. Van de afwijkende prestaties (het missen van kleine sterren aan één zijde) op deze test kan echter een aantal factoren niet verklaard worden door een aandachtsprobleem. Wanneer een patiënt van tevoren de vier hoeken van een pagina moet markeren, leidt dit niet verbetering van de prestatie op deze taak (Halligan & Marshall, 1993). Met andere woorden, terwijl de patiënt bewust wordt gemaakt van het volledige zoekveld en dus de aandacht verplaatst naar het contralesionale visuele veld, vertoont de patiënt nog steeds neglect. De resultaten van een andere studie hebben daarentegen duidelijk gemaakt dat er geen neglect is voor sterren wanneer deze sterren in isolatie worden gepresenteerd in het contralesionale veld (Eglin e.a., 1989). Dit betekent dat de aandacht wel gericht kan worden wanneer er geen competitie is van visuele informatie in het ipsilesionale visuele veld, zoals

ook geobserveerd wordt bij visuele extinctie. Ook wordt vaak gezien dat sterren in het ipsilesionale visuele veld veelvuldig aangekruist worden, ondanks dat het eerder gezette streepje goed zichtbaar is (Nys e.a., 2010; Rusconi e.a., 2002). Wanneer oogbewegingen gemeten worden tijdens visuele zoektaken wordt een vergelijkbaar fenomeen waargenomen: patiënten met neglect maken veel oogbewegingen naar eerder bezochte locaties (Behrmann e.a., 1997). Als er alleen een aandachtsprobleem voor het contralesionale veld zou zijn, zou er geen reden zijn om veel locaties opnieuw te bezoeken in het ipsilesionale veld.

Een andere veelvuldig gebruikte taak om neglect vast te stellen is een kopieertaak. Bij deze taak wordt de patiënt gevraagd om bijvoorbeeld een huis of een klok na te tekenen. Neglectpatiënten kopiëren bij deze taak vaak maar een half object. Interessant hieraan is dat deze patiënten wel correct kunnen aangeven of ze een heel of een half object zien; ze hebben dus kennis over alle componenten van een plaatje, maar kopiëren maar een half object als hun gevraagd wordt om een heel object na te tekenen (Ishiati e.a., 1996). Patiënten zien dit zelf niet direct na het natekenen, maar geven na een pauze wel aan dat ze maar een half object hebben getekend. Tevens wordt tijdens deze taak veelvuldig waargenomen dat patiënten details van de contralesionale kant van de tekening verplaatsen naar de ipsilesionale kant (Halligan e.a., 1992). Dit fenomeen wordt 'allochiria' genoemd en wordt ook gezien tijdens het natekenen van een klok: details van de linkerkant worden verplaatst naar de rechterkant (Di Pellegrino, 1995). Het tekenen van een klok wordt wel correct uitgevoerd wanneer de ogen van de patiënten gesloten zijn en gaat ook correct wanneer de te kopiëren klok geen cijfers aan de ipsilesionale kant heeft. Er is dan namelijk geen informatie die van de ipsilesionale kant verplaatst kan worden. Het is onduidelijk waarom patiënten alleen bij het kopiëren een half object zien en waarom informatie wordt verplaatst in de ruimte als er alleen een aandachtsprobleem zou zijn (zie ook, Nys e.a., 2008).

Een laatste probleem met de standaarduitleg van neglect komt van studies die hebben laten zien dat locatie-informatie in het contralesionale visuele veld verstoord is. Wanneer een object kort wordt aangeboden in het genegeerde visuele veld, kunnen neglectpatiënten vervolgens niet correct aangeven waar het object precies werd aangeboden: er is een mislokalisatie waarbij de informatie over de locatie van het object verkeerd gerepresenteerd wordt (Di Pellegrino & De Renzi, 1994). Dit soort resultaten wordt vaak gekoppeld aan de term 'representationeel neglect', waarbij er een verstoring is van de waargenomen ruimte (Bisiach e.a., 1994; Kerkhoff, 2000; Lenggenhager e.a., 2012). Deze representatie is dan asymmetrisch, waarbij de contralesionale ruimte vergroot is en de ipsilesionale ruimte verkleind.

Een nieuwe kijk op neglect

Uit het voorgaande zal duidelijk zijn dat er behoefte is aan een betere beschrijving van de onderliggende problematiek bij neglect. In de afgelopen jaren is er steeds meer bewijs verzameld voor het idee dat een groot gedeelte van de problemen bij neglect niet alleen verklaard kan worden door een aandachtsprobleem, maar ook door een probleem in het updaten van informatie. Wanneer wij de wereld zien, komt deze op ons over als stabiel. Visuele informatie wordt echter niet stabiel geregistreerd: elke keer als wij een oogbeweging maken, verschuift het gehele visuele veld en staan alle objecten weer op een andere plek. Toch zult u geen problemen hebben met het lokaliseren van uw kop koffie na het maken

van een oogbeweging: de representatie (bijvoorbeeld locatie, grootte, oriëntatie) van uw kop koffie is bijgewerkt na de oogbeweging. Hoewel het object op het ene moment op een andere plek van uw retina valt dan op een ander moment, heeft u de indruk van een stabiele wereld en niet een wereld van schokkerige beelden elke keer als u met uw ogen beweegt. Het opslaan, updaten en de relokalisatie van de verschillende onderdelen van een visuele scene zorgt voor de constantheid van visuele perceptie.

Een simpel experiment om dit updating concept duidelijk te maken is de 'double step saccade'-taak (Becker & Jürgens, 1979). In deze taak zien proefpersonen twee stippen die kort achter elkaar op verschillende plekken op een computerscherm worden getoond. Proefpersonen wordt vervolgens gevraagd om twee oogbewegingen te maken: een saccade naar de eerste stip en vervolgens een saccade naar de tweede stip. Echter, op het moment dat de twee oogbewegingen moeten worden gemaakt, is de visuele informatie reeds van het scherm verdwenen. De sequentie van oogbewegingen moet dus helemaal uit het geheugen worden gemaakt. Nu zal de eerste oogbeweging in het algemeen geen probleem zijn, maar voor de tweede oogbeweging is een update nodig: de locatie van de tweede stip is een andere locatie ten opzichte van de huidige locatie van het oog dan op het moment waarop deze stip werd aangeboden. Er moet dus een update plaatsvinden van de locatie van de tweede stip om de tweede oogbeweging correct uit te voeren. Als deze update niet succesvol is, zal de tweede oogbeweging naar een verkeerde plek worden gemaakt.

Neurofysiologische metingen bij apen hebben laten zien dat gebieden in de posterieure pariëtale cortex een belangrijke rol spelen bij het updaten van visuele informatie (Duhamel e.a., 1992). De rol van dit gebied werd bevestigd in studies bij patiënten met een beschadiging aan de pariëtale cortex (Heide e.a., 1995). Deze patiënten hadden een probleem met het updaten van visuele informatie waardoor zij een incorrecte tweede oogbeweging uitvoerden (zie ook Sapir e.a., 2004). Dezelfde problemen werden ook gezien in een vervolgstudie waarin ook enkele patiënten met neglect werden getest (Heide & Kompf, 1998). Door deze resultaten en de sterke overlap in breingebieden die coderen voor spatieel geheugen en aandacht (Corbetta e.a., 2002; Husain & Rorden, 2003) hebben een aantal onderzoekers voorgesteld dat een probleem in het updaten van visuele informatie een cruciaal onderdeel van neglect zou kunnen zijn (Pisella & Mattingley, 2004; Vuilleumier e.a., 2007). Hieronder zullen enkele studies worden besproken die bewijs hebben geleverd voor dit idee.

Een eerste indicatie voor een probleem met updaten van visuele informatie komt van een studie waarbij patiënten werd gevraagd om de verandering van de locatie, vorm en kleur van stimuli te detecteren (Pisella e.a., 2004). Patiënten kregen hierbij een matrix te zien met vier objecten in verschillende posities. Na een korte pauze verscheen een tweede matrix en moesten patiënten veranderingen in locatie, kleur en vorm detecteren. Neglectpatiënten hadden moeite met veranderingen in objectlocatie in beide visuele velden, terwijl het geheugen voor de andere eigenschappen in orde was. Interessant hierbij was dat dit probleem alleen optrad bij een korte pauze tussen de aanbieding van de twee matrices. Wanneer de twee matrices direct na elkaar werden getoond, was dit probleem niet aanwezig. Hoewel studies elkaar tegenspreken of dit fenomeen nu optreedt in beide visuele velden, zoals in deze studie, of alleen in het contralesionale veld (Denis e.a., 2002), was de verklaring voor dit effect dat er oogbewegingen gemaakt worden in de pauze tijdens de

twee aanbiedingen. Door deze oogbewegingen gaat er informatie over de locatie verloren door een probleem in de spatiële updating van informatie. Gezien het feit dat updating plaatsvindt in de pariëtale kwab, was een interessante bevinding dat het probleem met het coderen van objectlocatie alleen zichtbaar was bij patiënten met pariëtale schade en niet bij neglectpatiënten zonder pariëtale schade.

De belangrijke rol van oogbewegingen bij het wel of niet onthouden van locatie-informatie werd in een latere studie direct gemanipuleerd (Vuilleumier e.a., 2007). In deze studie werd aangetoond dat het geheugen voor de locatie van een stimulus inderdaad alleen verstoord is als er een oogbeweging gemaakt wordt. In deze studie werd geconcludeerd dat locatie-informatie verminderd is indien deze bijgewerkt moet worden. Dit was niet het geval voor een groep gezonde controles. De mate waarin deze informatie verminderd was, was gecorreleerd met de ernst van neglect.

Ook het feit dat neglectpatiënten tijdens experimenten veelvuldig plekken bezoeken die ze reeds bezocht hebben (zie ook Malhotra e.a., 2004), blijkt te verklaren door een probleem met het updaten van locatie-informatie. Wanneer patiënten tijdens een zoektaak bij elk element moesten aangeven of dit een nieuw object is of dat ze hier al eerder naar hebben gekeken, schatten ze dit slechter in dan controles (Husain e.a., 2001). Dit probleem gold vooral voor het ipsilesionale veld, simpelweg omdat in dit visuele veld vooral gezocht werd. In een verdere casusbeschrijving werd een inventieve manipulatie gebruikt, waarbij de patiënt een onzichtbare streep zette op het aan te kruisen object (de dop bleef op de pen, maar carbon papier registreerde wel de pennenstreek, Wojciulik e.a., 2001): het gevolg was dat de neglectverschijnselen in dit geval ernstiger waren dan wanneer de streep zichtbaar was. De patiënt was dus niet in staat om de locaties te onthouden die reeds weggestreept waren en dit probleem verergerde zonder het 'geheugensteuntje' dat de patiënt normaal zou hebben door het zien van het eerder geplaatste streepje. Maar als u zich realiseert dat u in het dagelijks leven in het algemeen geen streepje plaatst op de plek waar u reeds gezocht hebt, kunt u zich voorstellen dat dit een groot probleem is bij patiënten met neglect. Indien de patiënt dan bijvoorbeeld een boek zoekt in een boekenkast zal er op een inefficiënte manier gezocht worden.

Het opnieuw aankruisen van stimuli lijkt op de motorperseveraties die vaak gezien worden bij frontale beschadigingen (Na e.a., 1999). Dit lijkt echter een ander fenomeen te zijn, aangezien deze patiënten niet worden tegengehouden door het herkennen van een eerder geplaatste streepje. Daarbij neemt voor patiënten met pariëtale schade de kans op het opnieuw bezoeken van een stimulus tijdens een zoektaak toe met het verstrijken van de tijd (doordat het geheugen voor de locaties steeds slechter wordt), terwijl dit niet het geval is voor patiënten met frontale schade (Mannan e.a., 2005).

Deze nieuwe kijk op neglect verklaart ook veel van de observaties gedaan naar aanleiding van natekentaken. Voor het kopiëren van een tekening moeten er namelijk frequent oogbewegingen worden gemaakt, omdat de originele afbeelding veelvuldig bekeken moet worden om een exacte kopie te kunnen maken. Echter, na het maken van een oogbeweging is de locatie-informatie verstoord. Hierdoor kan informatie op de verkeerde plek worden neergezet en dat kan leiden tot fenomenen die eerder beschreven zijn als 'allochiria' (Halligan e.a., 1992). Dit verklaart ook waarom allochiria niet zichtbaar is als mensen tekenen met de ogen dicht (Di Pellegrino, 1995), aangezien hierbij geen oogbewegingen gemaakt

hoeven te worden. Patiënten met linkszijdig neglect zullen daarom ook nog steeds sterren in het linker visuele veld missen nadat ze kort daarvoor de linkerkant van het zoekveld hebben gemarkeerd (Halligan & Marshall, 1993): de informatie over de linkerkant van het zoekveld wordt niet onthouden en is dus geen marker om het verdere zoeken te bevorderen.

Het lijkt dus alsof veel van de problemen die eerst werden geschaard onder een aandachtsstoornis, ook deels verklaard kunnen worden door een probleem in het updaten van locatie-informatie. Wanneer de aandacht gericht is op het ipsilesionale veld, zullen patiënten moeite hebben met het verplaatsen van de aandacht naar het contralesionale veld (het zogenaamde 'disengagement'), simpelweg omdat de locatie-informatie van het contralesionale veld niet meer beschikbaar is; deze informatie is niet geüpdatet na de verplaatsing van de aandacht naar het ipsilesionale veld. Ook al maken neglectpatiënten tijdens een zoektaak net zoveel oogbewegingen naar links als naar rechts (Niemeier & Karnath, 2000), zullen ze niet in het contralesionale veld zoeken, aangezien veel van de locatie-informatie over deze ruimte niet meer aanwezig is. In combinatie met een aandachtsprobleem, zou een probleem in het updaten van informatie dus uiteindelijk kunnen leiden tot het verdwijnen van relevante informatie uit het bewustzijn.

Conclusie

Er wordt niet beweerd dat deze componenten *alle* problematiek bij neglect verklaren; er blijft een initiële bias om sterker op informatie in het ipsilesionale veld te reageren. Gezien het feit dat de gebieden in het brein die verantwoordelijk zijn voor het updaten van informatie, zich in de posterieure pariëtale cortex bevinden, is de verwachting dat dergelijke problematiek ook alleen aanwezig zal zijn bij patiënten met een beschadiging aan dit specifieke gebied. Wel kan het zo zijn dat deze patiënten een veel ernstigere vorm van neglect hebben, omdat zij uiteindelijk nooit in het contralesionale veld gaan zoeken doordat de informatie over het contralesionale veld grotendeels verloren is gegaan door een probleem in het updaten.

In tegenstelling tot aandacht is updating een goed gedefinieerd concept. Toekomstige revalidatietechnieken zouden gebruik kunnen maken van deze nieuwe kijk op neglect. Een goede optie zou prisma-adaptatie kunnen zijn: hierbij wordt de visuele wereld verplaatst door een primabril, waarna een patiënt zich aanpast aan deze nieuwe situatie. Het is bekend dat prisma-adaptatie een positief effect kan hebben bij neglectpatiënten (Nijboer e.a., 2008; Nijboer e.a., 2011; Rossetti e.a., 1998). Recentelijk hebben we aangetoond dat het updatingproces bij gezonde controles beïnvloedt wordt door prisma-adaptatie: na prisma-adaptatie werd de cruciale tweede oogbeweging op de 'double step saccade'-taak minder accuraat uitgevoerd dan voor de adaptatie (Bultitude e.a., 2013). Het zou dus zo kunnen zijn dat vooral patiënten met posterieure pariëtale schade baat hebben bij deze adaptatie, omdat dit hersengebied verantwoordelijk is voor het updaten van informatie. Mede doordat neglect zo'n heterogene stoornis is, kunnen verfijningen in de beschrijving van de onderliggende stoornis tot verbetering leiden van diagnostiek en revalidatie.

DOI: 10.1093/brain/awh085 *Brain* (2004), **127,** 239–242

Editorial

The pathology of experience

The fundamental assumption of cognitive neuroscience is that the way we behave and the way we experience the world is determined by the way our brains work. Pathological cases provide the most stringent tests for this assumption. From our knowledge of the way our brains work it should be possible to predict what kind of behaviour or experience will occur as a result of damage to a specific brain region or system. Lichtheim (1885) was the first to describe this approach. In his simple model of how the brain processes speech Lichtheim pointed out that there were seven possible 'interruption points' in this system. He then specified the different kinds of aphasia that should result from damage at each of these points. A striking and erroneous early example of the approach concerns cerebral achromatopsia. For many years neurologists refused to accept the existence of this disorder on the basis of the mistaken belief that it was incompatible with the way the brain worked (Zeki, 1990). Today the demonstration of segregation in the visual system predicts the existence of many specific disorders including achromatopsia (impaired colour perception), akinetopsia (impaired visual movement perception) and prosopagnosia (impaired face recognition).

These disorders are all negative in the sense that the patients lack a particular aspect of normal experience. It is not too difficult to understand how damage to the brain could prevent some behaviour or experience. It is much more difficult to understand how damage to the brain can create a positive symptom in which the patient experiences something, such as an hallucination, which most of us don't experience. A second problem is that there is a long tradition of treating hallucinations not as false, but as veridical perceptions from a spiritual world; telepathy or a voice from beyond the grave. This tradition gave rise to what is probably the most thorough investigation of hallucinations to date, "The Report on the Census of Hallucinations" (Sidgwick et al., 1894). With a sample of 17,000 informants this contrasts very favourably with modern surveys. The motives of the distinguished Cambridge academics who compiled the report were mixed. Some hoped that the data would prove the existence of the spirit world. Others wanted to uncover the psychological and physiological mechanisms underlying hallucinations. Care was taken to ascertain that, at the time of the experience, the informant was sane and was not suffering from any physical disorder such as a fever. About 10% of the sample had experienced hallucinations and the majority of these (8.4%) were visual. The compilers noted that this differs from hallucination in the insane of which the majority are auditory.

There are many examples of apparent spiritual communication. From Mr Eggie: "On October 5th, 1863, I awoke at 5 a.m. ….. I heard distinctly the well-known and characteristic voice of a dear friend, repeating the words of a well-known hymn. I have always thought it remarkable that at the very same time, almost to the minute, my friend was seized suddenly with a mortal illness." Other informants, however, did not trust their experiences. From Mr. W.S.: "I became painfully anxious, and sat down for a minute …. and then I saw, floating, as it were, between me and the mantel-piece, the upper half of my father's body. …. I looked steadily at my half-ghost, and saw how a spot in the mantel-piece, a knot in the wainscot, &c., &c., had combined to produce the spectral appearance." The report also includes a discussion of possible physiological mechanisms. "The view that … hallucinations are originated centrally, in the brain and not in the sense organs, is that now generally held by physiologists, as well as by psychologists." A mechanism for this central origin is attributed to Professor Sully: "The cerebral activity that produces a hallucination may probably diffuse itself downwards to the periferal regions of the nerves so that the sense-organs may thus become involved secondarily". But the report concludes that "this hypothesis of the secondary participation of the sense-organs in hallucinations through a downward sensory impulse from the brain …. is inconsistent with generally accepted physiological theories of the actions of the nervous system."[There is an interesting discussion in the report about whether hallucinations or dreams or mental images can cause visual after-images, and, whether this implies top-down influences on the retina. There is evidence such top-down effects can occur in some people (Weiskrantz, 1950)]. Today we believe that 'downward sensory impulses' (called variously top-down, feedback or re-entrant signals) have a major role in brain function (e.g. Lamme *et al*., 1998). What we perceive depends upon the interaction between these top-down signals and the information coming from the sense organs. Abnormal dominance of the top-down signals would lead to false perceptions including illusions and hallucinations.

Visual Hallucinations

Unlike Sidgwick and his collaborators, we know now that activity in the sense organs is not necessary for sensory experience. Direct electrical stimulation of visual cortex produces visual hallucinations (Lee *et al*., 2000) and the nature of the visual experience, whether it be of colour, form or motion, depends on where exactly the cortex is stimulated. The hallucinations experienced by patients with Charles Bonnet syndrome are associated with activity in extra-striate cortex and here too the location of the activity determines the form of the experience (ffytche *et al*., 1998). There is a striking similarity between the visual hallucinations associated with a variety of different causes including peripheral damage (Charles Bonnet syndrome), occipital lobe epilepsy, and mescalin. Patients report flashing and spinning colours, complex repetitive patterns, organic forms and grotesque faces and scenes (ffytche and Howard, 1999). These visual hallucinations come from a restricted set of categories determined by the anatomy of the visual brain (Santhouse *et al*., 2000).

False beliefs

There has been even greater resistance to fitting psychotic disorders into our neuro-cognitive framework. The hallucinations and delusions associated with disorders like schizophrenia were for a long time considered to be 'not understandable'. Karl Jaspers (1963) wrote that 'the profoundest difference … seems to exist between that type of psychic life which we can intuit and understand, and that type which, in its own way, is not understandable and which is truly distorted and schizophrenic …'. He implies that an understanding of normal cognitive processes will not help us to understand these experiences. In the case of psychosis, there is no clear distinction between hallucinations (false perceptions) and delusions (false beliefs). I shall consider two examples: Capgras syndrome and delusions of control. The patient with Capgras syndrome falsely believes that a close relative or friend has been replaced by a double. The patient with a delusion of control falsely believes that his actions are being directly controlled by alien forces. These two possibilities lie so far from our normal experience that these disorders were typically classified as examples of false beliefs rather than abnormal experiences. Recent formulations suggest that these disorders are better characterised as abnormal experiences that derive their particular form from basic neural functions.

Capgras syndrome

We know that information is processed in many parallel streams in the brain and that each stream is optimised for different functions (e.g. the 'what' and 'where' streams; Ungerleider and Mishkin, 1982). Another example is the rapid and unconscious processing stream that evaluates objects in terms of whether they should be approached or avoided (the '****!' stream). This stream allows us to respond unconsciously to fearful objects and involves the amygdala (Morris *et al*., 1999). Explicit identification of objects is achieved much more slowly and via a different neural pathway involving inferior temporal cortex (the 'what' stream). As a result we can find ourselves running away from something before we are aware of precisely what it is (LeDoux, 1996). These two streams can be damaged independently. Damage to the stream that explicitly identifies faces leads to prosopagnosia. Patients with this disorder can no longer identify individual faces (including friends and relatives). However, rapid, implicit evaluation of faces remains intact and such patients still show emotional responses to familiar faces (e.g. Blount, 1994). Damage to the evaluation stream leads to a situation in which the patient can recognise a face, but no longer experiences the emotional response associated with evaluation of the face. The face may look like that of the patient's wife, but the emotional colouring normally associated with that face is no longer present (Ellis and Lewis, 2001). The conclusion that this person, who claims to be his wife, must be an impostor is almost inevitable. [Perhaps this conclusion is not quite inevitable. It is possible that additional frontal damage is needed in order to entertain this rather outlandish hypothesis (Burgess *et al*., 1996)].

Delusions of control

Our understanding of delusions of control also results from the discovery of multiple representations, but in this case in the system underlying the control and awareness of action rather than the visual system (see Blakemore & Frith, 2003 for a review). In particular there are separate representations of the intended consequences of our movements (based on prediction) and the actual consequences of our movements (based on sensory feedback). Whenever we make a movement this will cause changes in tactile and kinaesthetic sensation, but we are largely unaware of these changes. Most of the time it is representations of the intended consequences of our movements that determine our awareness rather than representations of the actual consequences of our movements. Only if the discrepancy between intended and actual consequences of movement is quite large do representations of the actual state of our motor system enter awareness. Furthermore the physiological activity associated with tactile sensations caused by our own movements is attenuated. This, of course, is why we cannot tickle ourselves. In patients with delusions of control something as yet to be determined goes wrong with the system whereby sensations caused by the patient's own actions are attenuated. They are abnormally aware of these sensations (Blakemore *et al*., 2000) and manifest over-activity in parietal cortex (Spence *et al*., 1997). For them, active movements *feel* like passive movements. On this basis it is perfectly understandable that it is indeed as if their movements were being caused by external forces.

Multiple representations of the body

Our understanding of delusions of control was informed by the observation that there are separate representations in the brain for the intended and the actual state of the motor system. However, there are many other representations differentiated in terms of temporal and spatial coordinates. There are representations of past, present and future states of the body. There are also representations of the body in terms of different spatial coordinate systems: my arm in relation to the rest of my body, my arm in relation to the object I am reaching for, my arm in relation to the room in which I am standing. In the normal case we are not aware of all these different representations, but only of an integrated sense of our body in space. However, damage to the motor system can lead to abnormal awareness of un-integrated representations. For example, McGonigle *et al*. (2002) describe a patient with a phantom supernumerary left arm. This phantom arm occupies the position previously occupied by the patient's real left arm about a minute previously and disappears whenever the left arm is moved. In this case the lesion was in the left pre-Supplementary Motor Area (SMA) and cingulate motor area and the activity associated with presence of the phantom was in SMA proper.

Out-of-the-body experiences

In the case of out-of-the-body experiences it is the whole body rather than just one arm that is replicated. In one form (autoscopy) we see our double. In true out-of-the-body experiences we look down at our own body from above. Such experiences are associated with psychiatric disorders, but are also estimated to have a prevalence of 10% in the general population, a figure close to that reported for hallucinations. Like hallucination these experiences have exerted a particular fascination since they seem to provide access to a spirit world. For example, it was believed that we each have a dopplelgänger who normally remains unseen. If we see our doppelgänger then our death is imminent. Meetings with doppelgangers were a poplar theme in romantic literature of the 19th century (Webber, 1996). Even today many people believe that during an out-of-the-body experience (astral projection) the mind literally leaves the body and thereby can view things that would be invisible from the vantage point of the body. Experimental tests of this possibility continue to be conducted (e.g. Tart, 1968).

Against this background of mysticism the study of Blanke et al. in this issue of *Brain* stands out with admirable clarity. Blanke and his colleagues provide a very thorough review of earlier literature on out-of-the-body experiences and autoscopy and then report a detailed examination of 6 neurological patients who report such experiences. They find that patients with such experiences also report pathological sensations such as floating and rotating (associated with the vestibular system) and visual body-part illusions such as shortening or movement of limbs. Furthermore they have been able to localise an area of common brain dysfunction in all these cases to the temporo-parietal junction. In one patient (case 3) direct electrical stimulation of this region reliably generated out-of-the-body experiences and other abnormal experiences of the body.

We know that the brain contains multiple representations of the body for proprioceptive, tactile and visual information and that some of these representations are in body centred coordinates while others are coded in terms of external space. Normally all these representations are integrated to provide a unitary sense of the body in space. However, as a result of damage (or perhaps during low arousal when on the verge of sleep) this unity can be lost leading to the experience of a divergence between the felt and seen position of our body. I find it particularly interesting that the temporo-parietal junction has a special role in the experience. Recent brain imaging experiments with normal volunteers have shown that activity in this region is elicited by observing biological motion, actions and perhaps simply by the apparent presence of other people (Saxe *et al*. 2003). This raises the possibility that an out-of-the-body experience occurs when a strong sense of the presence of another person is coupled with a discrepancy between the felt and the seen position of our own body. In these circumstances a representation of our body can get falsely bound with the representation of another person in external space.

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doi:10.1093/brain/awh560 *Brain* (2005), **128**, 1480–1497

REVIEW ARTICLE

Apraxia in movement disorders

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The definition of apraxia specifies that the disturbance of performed skilled movements cannot be explained by the more elemental motor disorders typical of patients with movement disorders. Generally this does not present a significant diagnostic problem when dealing with 'higher-level' praxic disturbances (e.g. ideational apraxia), but it can be a major confound in establishing the presence of limb-kinetic apraxia. Most motor disturbances characteristic of extrapyramidal disorders, particularly bradykinesia and dystonia, will compromise the ability to establish the presence of loss of dexterity and deftness that constitutes this subtype. The term 'apraxia' has also been applied to other motor disturbances, such as 'gait apraxia' and 'apraxia of eyelid opening', that perhaps are misnomers, demonstrating the lack of a coherent nomenclature in this field. Apraxia is a hallmark of corticobasal degeneration (CBD) and historically this has received the most attention among the movement disorders. Corticobasal degeneration is characterized by various forms of apraxia affecting limb function, particularly ideomotor apraxia and limb-kinetic apraxia, although buccofacial and oculomotor apraxia can be present as well. The syndrome of parkinsonism and prominent apraxia, designated the 'corticobasal syndrome' (CBS), may be caused by a variety of other central nervous system pathologies including progressive supranuclear palsy (PSP), Alzheimer's disease, dementia with Lewy bodies and frontotemporal dementias. Distinct from the CBS, PSP and Parkinson's disease can demonstrate varying degrees of apraxia on selected tests, especially in those patients with more severe cognitive dysfunction. Diseases that cause the combination of apraxia and a primary movement disorder most often involve a variety of cerebral cortical sites as well as basal ganglia structures. Clinical-pathological correlates and functional imaging studies are compromised by both this diffuse involvement and the confusion experienced in the clinical evaluation of apraxia in the face of the additional elemental movement disorders. Finally, although apraxia results in clear disability in patients with the CBS, it is not clear how milder ideomotor apraxia found on specific testing contributes to patients' overall day-to-day motor disability.

Keywords: apraxia; corticobasal degeneration; Huntington's disease; movement disorders; Parkinson's disease; progressive supranuclear palsy

Abbreviations: ALO = apraxia of eyelid opening; CBD = corticobasal degeneration; CBS = corticobasal syndrome; DLB = dementia with Lewy bodies; IMA = ideomotor apraxia; LKA = limb-kinetic apraxia; MSA = multiple system atrophy; OFA = orofacial apraxia; PSP = progressive supranuclear palsy

Received November 1, 2004. Revised March 10, 2005. Accepted May 12, 2005. Advance Access publication June 1, 2005

Introduction

Apraxia covers a wide spectrum of disorders that have in common the inability to perform a skilled or learned act that cannot be explained by an elementary motor or sensory deficit or language comprehension disorder. Praxis errors have been well defined clinically and kinematically and can be superimposed on elementary motor disorders such as weakness, bradykinesia, rigidity, tremor, dystonia and ataxia (Heilman, 1985; Roy and Square, 1985; Poizner *et al.*, 1990, 1995). In many higher order apraxic disorders, such as ideational apraxia, this does not usually pose a diagnostic

dilemma. Although bradykinesia and other extrapyramidal signs (e.g. rigidity or dystonia) might cause a delay in initiation or performance of a gesture, the quality of the gesture should not be affected by these and so gestural impairment could not be explained by their presence alone. However, in some cases, such as those with limb-kinetic apraxia (LKA), bradykinesia and rigidity could readily compromise the ability to distinguish the loss of dexterity and coarseness of movement that characterize this subtype. In this review we will begin with a summary of the basic types of apraxia and the underlying neuroanatomical and physiological mechanisms thought to play a role, particularly as they relate to diseases of the basal ganglia. We will then discuss the different movement disorder syndromes commonly, and not so commonly, associated with apraxia. The neurological diseases chosen are based largely on descriptions of apraxia in those conditions in the literature as well as clinical experience. Certain disorders that are called 'apraxia', such as eyelid opening apraxia (ALO), but are probably better classified separately from apraxia, are also addressed.

Overview of apraxia

The first contemporary ideas of apraxia stem from the work of Liepmann, who proposed that in order to perform an action, the motor engram (or 'space–time plan') has to be conveyed from the left parietal lobe via association fibres to the 'Central region', in which Liepmann included the precentral and postcentral gyri, the middle and superior frontal gyri and their underlying white matter tracts. The Central region effected the action through the primary motor cortex, i.e. through the final common pathway of the pyramidal tract. If the left limb is to perform a task, then the information needed to be transmitted through the corpus callosum to the Central region on the right in order to activate the right pyramidal tract to carry out the action (Liepmann, 1908, 1920). Since that time, many other studies have confirmed the dominance of the left hemisphere in praxis (Basso *et al.*, 1980; De Renzi *et al.*, 1980, 1982). Apraxia, as tested by imitation and object use pantomime, has been found in ~50% of patients with left hemisphere damage and in <10% with right hemisphere damage. This suggests that many patients have bilateral representation of praxis functions (De Renzi, 1989). Even Liepmann pointed out that the right hemisphere probably has some praxis skills, and this has been used to explain why there is sparing of certain left-hand praxis functions after callosal or left hemisphere lesions (Geschwind and Kaplan, 1962; Graff-Radford *et al.*, 1987).

Damasio and Geschwind (1985) defined apraxia as demonstrating varying combinations of the following disturbances in order of progressive dysfunction: the failure to produce the correct movement in response to a verbal command, the failure to correctly imitate a movement performed by the examiner, the failure to perform a movement correctly in response to a seen object and the failure to handle an object correctly. It is classified by both the nature of the errors made and the means by which they are elicited. For instance, abnormal performance can be due to 'temporal errors' (such as impaired timing and poor sequencing of a movement that requires multiple positionings, as long as the overall content of the movement remains recognizable), 'spatial errors' (such as abnormal amplitude, internal or external configuration orientation and body-part-as-object substitution), 'content errors' (such as perseveration) or 'other errors' (such as lack of response or an entirely unrecognizable response) (Rothi *et al.*, 1988). Since Liepmann's original description, others have tried to advance his model in order to account for different types of praxis errors. Roy and Square (1985) proposed a two-part model in which a conceptual component encodes an abstract knowledge base for actions, including information about tool use and sequencing a series of single actions, and a production component provides sensorimotor information on how to perform an action 'programme' and then translates these programmes into actions. Much of our understanding of apraxia is based on lesioning studies and yet no single area alone has consistently been involved in the production of apraxia. This suggests that praxis functions are distributed through different neural networks working together. Depending on the neural network involved, the types of errors will differ. For example, as pointed out by Leiguarda and Marsden (2000), there is a parietofrontal system that encodes reaching and grasping mechanisms, and a frontostriatal system that encodes sequential motor events. Moreover, the extent to which these systems are affected depends on the context of the movement and the cognitive demand of the action (Leiguarda and Marsden, 2000). Table 1, which summarizes the major classification of motor limb apraxias (Rothi and Ochipa, 1991), will serve as a background to the types of apraxia found in movement disorders discussed below.

Role of the basal ganglia and apraxia

The motor and premotor areas of the cortex send projections to the basal ganglia (Alexander *et al.*, 1986), as do areas of the parietal cortex that are interconnected with those areas of the motor cortex, making up the parietofrontal circuits. These circuits act in parallel. Each one is involved in sensorimotor integration or in the translation of specific sensory data into information for movement production (e.g. visual and somatosensory transformation for reaching and body part location data for control of body part movements). There are also distinct frontostriatal circuits that play a role in action sequencing. The circuit activated depends on whether the action to be performed is prelearned or new, and on the complexity of the cognitive demands of the task (Grafton *et al.*, 1995; Catalan *et al.*, 1998). Apraxia can be found in diseases of the basal ganglia, including Parkinson's disease, progressive supranuclear palsy (PSP) and Huntington's disease, and reportedly in isolated lesions of the basal ganglia.

Table 1 Summary of apraxia types

Apraxia type	Definition	Clinical testing	Localization
Limb-kinetic	Loss of hand and finger dexterity resulting from inability to connect or isolate individual movements (Kleist, 1907)	All movements are affected—symbolic, non-symbolic, transitive (i.e. using tools and instruments, e.g. a hammer or a hairbrush) and intransitive (i.e. communicative gestures, e.g. representational tasks such as waving goodbye and non-represenational tasks such as touch your nose and wiggle your fingers) Mainly distal in finger and hand Movements are coarse and mutilated No voluntary automatic dissociation:	All pathologically confirmed cases have shown a degenerative process involving frontal and parietal cortices (Fukui *et al*., 1996) or primary motor cortex (Tsuchiya *et al*., 1997)
Ideomotor	Disorder of goal-directed movement. Patient knows what to do but not how to do it. Disturbance of timing, sequencing and spatial organization of gestural movement (Rothi and Ochipa, 1991)	Impairment of pantomiming ability to use tool. Movement is incorrectly produced but the goal of the action can usually be recognized. Abnormal errors include spatial errors [i.e. (i) abnormal amplitude; (ii) body-part-as-object substitution, e.g. the patient uses his own finger to represent a toothbrush when asked to brush his teeth; (iii) abnormal orientation of body part performing the action, e.g. when the patient is asked to pantomime brushing his teeth he closes his fist tightly with no space for the imagined toothbrush handle or he may hold his hand right next to his mouth without demonstrating the distance necessary to accommodate the imagined toothbrush] and temporal errors (i.e. irregular timing, which can be either an increased or decreased rate of production of a pantomine and sequencing abnormalities, e.g. an addition, deletion, or transposition of movement parts as long as the overall movement structure remains recognizable) (Rothi *et al*., 1988). Improves on imitation and with use of actual tool. Transitive more affected than intransitive. Voluntary automatic dissociation is present, so that deficit is more apparent in clinical setting than in everyday life.	Anatomically diverse lesions mainly in left hemisphere; typically involve parietal association areas and white matter bundles connecting frontal and parietal association areas. Less commonly premotor and supplementary motor cortex are involved as well as basal ganglia and thalamus. Unilateral lesions of the left hemisphere in right-handed patients produce bilateral deficits, usually less severe in the left than in the right limb
Ideational/ conceptual	Patient does not know what to do. Content errors. This terminology can be confusing not only because definitions of ideational and conceptual apraxia vary among authors (Ochipa *et al*., 1992; DeRenzi and Lucchelli, 1988) but also because a distinction between the two is debated by some. Error types include, impairment in carrying out sequences of actions requiring the use of various objects in the correct order so as to achieve an intended purpose (Liepmann, 1920), and loss of tool action knowledge	Inability to perform a multiple-step task (e.g. prepare a letter to mail) owing to errors such as perseveration. Disturbance of single tool use—cannot associate tool and object with the corresponding action (e.g. unable to choose a hammer to drive a nail or correctly pantomimes an action when requested to perform a very different one) (Roth *et al*., 1988; Leiguarda *et al*., 2000*a*; Ochipa *et al*., 1992)	No one anatomical area has been identified, although in focal hemispheric lesions, most have damage to left hemisphere. Damage typically thought to involve left parieto-occipital and parietotemporal regions (Liepmann, 1920) but can also involve left frontal, frontotemporal and temporal regions with or without subcortical involvement (Heilman *et al*., 1997)

JOURNAL OF MEDICAL CASE REPORTS

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Case report

Schizophrenia-like psychosis following left putamen infarct: a case report

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Received: 25 February 2008 Accepted: 12 March 2009 Published: 14 July 2009

Journal of Medical Case Reports 2009, **3**:7337 doi: 10.4076/1752-1947-3-7337

This article is available from: http://jmedicalcasereports.com/jmedicalcasereports/article/view/7337

Abstract

Introduction: Subcortical structures of the brain have been studied extensively to establish their implication in the development of psychotic symptoms in schizophrenia. Any pathology in these structures of the brain identified on neuroimaging techniques can give us helpful information in learning the neuropsychiatric background of psychotic symptoms in schizophrenia.

Case presentation: We report an interesting case of a 38-year-old man with new onset psychosis who was found to have a lacunar infarct of the putamen region in the left basal ganglia on a computed tomography scan of his brain.

Conclusion: It is possible to hypothesize that the psychotic symptoms in our patient may be the direct result of the putamen infarct, as pathology in the basal ganglia at the level of the striatum can result in complex cognitive and behavioural symptoms. Understanding organic causes of psychosis, including cerebrovascular compromises leading to damage of structures in the basal ganglia, can provide important information about the causality of psychosis and ways to treat it.

Introduction

The basal ganglia and cerebellum have been extensively studied in schizophrenia as these areas of the brain are involved in the control of movement. Disease in these areas has been implicated in the pathophysiology of schizophrenia as movement disorders have been observed in patients with schizophrenia even in the absence of medications that induce movement disorders [1]. Basal ganglia diseases have been associated with a wide range of neuropsychiatric symptoms including depression, anxiety, delusions, apathy, irritability and disinhibition [2].

Neuroimaging studies conducted in the presence of focal cerebrovascular disorders, particularly involving the basal ganglia, can provide useful information about the neuropsychiatric background of schizophrenia symptoms. We report on an interesting case of schizophrenia-like psychosis in a patient with a putamen infarct.

Case presentation

A 38-year-old Caucasian man was seen in the emergency department following a referral by his general practitioner, as there were concerns from his family that he was voicing bizarre ideas. He complained of feeling increasingly

stressed, anxious and paranoid and he was having flashbacks of fictional events from his past. He described visualizing these events as if they were being played in a movie. He had beliefs that many people from his local community were involved in a conspiracy against him and he was being provoked by other people to get into fights. He also had beliefs that he was being followed and watched, and he elaborated that he could feel the presence of evil and evil things being passed onto him by other people.

He had no past psychiatric history and there was no family history of any mental health related problems. He had experimented with recreational drugs such as cocaine and ecstasy in the past but he denied any use of recreational drugs in the last 2 years. He worked as a successful self-employed businessman. His symptoms, particularly the persecutory delusions and delusional memory, worsened and he was admitted to the local psychiatric hospital.

His physical and neurological examination was unremarkable, and blood tests including urea and electrolytes, liver function tests and thyroid function tests were normal. An electroencephalogram (EEG) was inconclusive of any seizure activity and a computed tomography (CT) scan of his brain revealed a lacunar infarct of the putamen region in the left basal ganglia. A detailed history of any cardiovascular incidents was explored and he gave a history of severe crushing chest pain 6 months before presentation. He did not seek medical attention, the pain did not recur, and there were no cardiological or neurological sequels. An electrocardiogram and echocardiogram showed no compromise of cardiac functions.

The lacunar infarct was treated conservatively and he was commenced on an atypical anti-psychotic medication, aripiprazole, and his psychotic symptoms gradually improved over the following weeks. He tolerated aripiprazole quite well and did not experience any extrapyramidal or significant side effects to the medication. His insight into his delusional beliefs improved and he was able to rationalise them. Following 3 weeks of hospitalization, his symptoms had improved and he was successfully discharged with community follow-up.

Discussion

Due to multifactor causality, the aetiology of psychotic illness remains a matter of much debate, and organic pathology including acute compromise of brain function has been investigated in many studies. Interesting observations have been noted when people with Parkinson's disease are treated with dopaminergic agents. They experience a variety of psychiatric symptoms such as hallucinations, delusions, mood elevation, nightmares and occasionally sexual behaviour disturbances. This marked overlap of movement disorders, neuropsychiatric symptoms and cognitive changes in patients with basal ganglia disorders is found to be related to disruption in the complex set of frontal subcortical circuits. These neuronal circuits link the cortex (predominantly frontal areas) to regions of the striatum, globus pallidus and/or substantia nigra and thalamus [3]. The striatum composed of putamen and caudate nucleus is the subcortical area that contains rich dopamine projections. Research thus far postulates that this neuromodulator contributes significantly to the pathophysiology of psychosis and its treatment with neuroleptic drugs through an impact on the corticostriato-thalamo-cortical loop [4].

Of the five basic frontal-subcortical circuits, the dorsolateral prefrontal circuit, orbitofrontal-subcortical circuit and anterior cingulate-subcortical circuit mediate important aspects of human behaviour [5]. These circuits mediate important aspects of cognition, emotion, civil behaviours and impulse control. Dysfunction of this region results in disinhibition, tactlessness, impulsivity and disrupted social interaction [6].

Our patient had an acute presentation with psychotic symptoms without any substance- induced, past or family psychiatric history. The lacunar infarct in the putamen area, a part of the striatum, on the CT brain scan indicates a possible role of organic cause in the onset of the acute psychosis.

Conclusion

It is possible to hypothesize the psychotic symptoms in our patient may be the direct result of the putamen infarct, as pathology in the basal ganglia at the level of the striatum can result in complex cognitive and behaviour symptoms depending on which of the above mentioned brain circuits are affected. A previous case report highlights the emergence of psychotic symptoms in a patient with a right putamen infarct [7]. Interestingly, another case report identifies the resolution of psychotic symptoms in a patient with schizophrenia following haemorrhage in the left putamen [8].

Understanding of organic causes of psychosis including cerebrovascular compromises, infarction or ischaemia of basal ganglia can provide important information about the causality of psychosis and ways to treat it. Further research in this area will possibly lead to promising results.

Consent

Written informed consent was obtained from the patient for publication of this case report. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Neuroanatomy of Pathological Laughing and Crying: A Report of the American Neuropsychiatric Association Committee on Research

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Pathological laughing and crying (PLC) is a clinical condition that occurs in patients with various neurological disorders. It is characterized by the presence of episodic and contextually inappropriate or merely exaggerated outbursts of laughter and/or crying without commensurate feelings. This review provides an in depth analysis of the neuroanatomy of lesions seen in patients with this clinical condition, discusses the relevant functional neuroimaging and electrophysiological stimulation studies in human subjects, and summarizes the current treatment options. It concludes with a presentation of the remaining questions and directions for future research.

(The Journal of Neuropsychiatry and Clinical Neurosciences 2009; 21:75–87)

The Clinical Phenotype

Emotional *experience* is the subjective feeling during an emotional event whereas emotional *expression* is the objective behavior that is expressed during such event, including changes in autonomic functions such as heart rate[1,2] and skeletal movements such as facial expression. Both the experience and the expression of an emotion are dependent in part upon the cognitive appraisal of the emotional stimuli which are triggering it, as reviewed extensively by others.[2] While many psychiatric conditions lead to a problem with emotional *experience* (e.g., mood disorders), many patients with neurological disorders suffer from problems with dysregulation of emotional *expression*. In these patients the problem involves mostly the expressions of laughter or crying. While some patients exhibit problems with only laughter, others have problems with only crying, and some exhibit problems with both. Although these patients do not laugh or cry at all times, when they do, the actual behavior of laughing or crying is often indistinguishable from normal acts of laughter and crying. Because the outbursts may occur in socially inappropriate times, this problem causes social handicap and suffering. In these patients, the actual motor behavior resembles normal laughing or crying behavior, so the problem of these patients is not simply in generating the motor act, as seen in patients with motor paresis or paralysis, or the flat affect encountered in patients with Parkinson's disease. Insight is spared unless the affected patient's awareness or consciousness is impaired. Most patients have accurate knowledge of cognitive, affective, and social norms of the moment and of the context in which their inappropriate emotional expression takes place.[3] Many of them are embarrassed by their inappropriate emotional display, which they cannot voluntarily prevent from happening or stop once it has occurred. The problem in these patients is clinically different than a mood disorder in which a pervasive and sustained change in emotional experience causes excessive but congruent emotional expression. Unlike mood disorders, inappropriate emotional expression in these patients is not a sustained phenomenon but a paroxysmal and episodic one. Moreover, the clinical phenotype, instead of being secondary to the problem of manic or depressive mood, is due to a dysregulated generation of an expression in the absence of a congruent or commensurate feeling. The clinical condition in our discussion is also a different entity than labile emotions seen in patients with personality

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disorders or in patients with emotional lability due to a general medical condition who change their emotional *experience* from extreme happiness to extreme sadness or vice versa. Again, the major distinguishing factor for the clinical phenotype in our discussion is the disassociation between emotional experience and expression. In some patients the expression of laughter or crying is incongruent with, or even contradictory to, the mood of the patient and to the emotional valence of the provoking stimulus, or is elicited chaotically without any trigger or by a stimulus without a clear emotional valence. Some may even switch from one expression to another in the setting of the same triggering stimulus. In other patients, emotional expression may not be absolutely incongruent with the mood of the patient or the valence of the triggering stimulus. However, the emotional expression of laughing or crying is "pathologically" exaggerated in intensity and frequency compared with the patient's premorbid baseline. For example, a patient who never used to cry develops frequent weeping episodes that are excessive and exaggerated to the context in which they occur.

In summary, the clinical condition is about generating an emotional expression that is contextually inappropriate either because it is without any congruent or commensurate feeling, or because it is excessively exaggerated compared to the patient's premorbid baseline. In either case, the condition is pathological because outbursts of contextually inappropriate laughing or crying cause social handicap and suffering for patients or their caretakers.

Terminology

The clinical condition has been known by different names, but the most widely used terms are "pseudobulbar affect," "emotional lability," "emotional incontinence," and "pathological laughter and crying" or "pathological laughing and crying."[4] Recently, a group proposed the further term of "involuntary emotional expression disorder" or IEED.[5] The terminology of this clinical condition has been confusing.[6] Different terms have been used to describe the same clinical presentation or similar terms are used for clinically different conditions.[6] In addition, some of these terms have their own inherent problems,[7] a discussion of which is beyond the scope of this report.

The aim of this report is not to focus on the problem of nosology. Instead, the primary aim of this review is to provide an in-depth analysis of the neuroanatomy of lesions leading to disassociation of emotional expression from emotional experience. Toward this aim, we will focus on clinical expressions where every clinician can argue beyond a reasonable doubt that there is a pathological problem with emotional expression that is not due to an underlying mood or personality disorder. Therefore, we will focus on cases in which there is a clear incongruence between the experienced and expressed emotion. In these cases, the term pathological laughing (or laughter) and crying (PLC) has been favored historically in the literature.[4,8,9]

Neurological Disorders Causing PLC

The precise prevalence of PLC in various neurological disorders is difficult to determine given the nosological problem discussed above. In addition, many of the studies of PLC did not use any specific diagnostic criteria and, in each study, the scope of the problem has been largely estimated in a small sample of cases. Thus the prevalence of PLC in various neurological disorders is largely based on limited and sketchy data (Table 1). These limitations aside, the problem of PLC has been encountered in varying frequency and severity in patients suffering from stroke,[3,9–15] traumatic brain injury,[16,17] multiple sclerosis,[18–20] amyotrophic lateral sclerosis,[21–23] brain tumor,[10,24–27] central pontine myelinolysis,[28] Parkinson's disease,[29] and the cerebellar type of multiple system atrophy.[30] The scope of the problem in dementia is obscured by assessment differences among studies. In one report, the authors defined the problem as simply "observable sudden changes in emotional expressions"[31] and observed it in about 74% of mildly to moderately impaired patients with Alzheimer's disease. In contrast, Starkstein and colleagues[32]

TABLE 1. Causes of Pathological Laughing and Crying*

Disease	Prevalence
Amyotropic lateral sclerosis	49%
Multiple system atrophy – cerebellar type	36%
Cerebrovascular disease	11–34%
Multiple sclerosis	10%
Parkinson's disease	4–6%
Traumatic brain injury	5–11%
Dementia	Unknown
Migraine	Unknown
Progressive supranuclear palsy	Unknown
Mass lesions	More often seen with mass lesions in the cerebellopontine junction

*References in (4).

NeuroImage 44 (2009) 489–501

Contents lists available at ScienceDirect

NeuroImage

journal homepage: www.elsevier.com/locate/ynimg

Functional topography in the human cerebellum: A meta-analysis of neuroimaging studies

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ARTICLE INFO

Article history:
Received 4 June 2008
Revised 3 August 2008
Accepted 28 August 2008
Available online 16 September 2008

Keywords:
Cerebellum
Cognition
Motor
Emotion
Topography
Functional imaging

ABSTRACT

Clinical, experimental and neuroimaging studies indicate that the cerebellum is involved in neural processes beyond the motor domain. Cerebellar somatotopy has been shown for motor control, but topographic organization of higher-order functions has not yet been established. To determine whether existing literature supports the hypothesis of functional topography in the human cerebellum, we conducted an activation likelihood estimate (ALE) meta-analysis of neuroimaging studies reporting cerebellar activation in selected task categories: motor (n=7 studies), somatosensory (n=2), language (n=11), verbal working memory (n=8), spatial (n=8), executive function (n=8) and emotional processing (n=9). In agreement with previous investigations, sensorimotor tasks activated anterior lobe (lobule V) and adjacent lobule VI, with additional foci in lobule VIII. Motor activation was in VIIIA/B; somatosensory activation was confined to VIIIB. The posterior lobe was involved in higher-level tasks. ALE peaks were identified in lobule VI and Crus I for language and verbal working memory; lobule VI for spatial tasks; lobules VI, Crus I and VIIB for executive functions; and lobules VI, Crus I and medial VII for emotional processing. Language was heavily right-lateralized and spatial peaks left-lateralized, reflecting crossed cerebro-cerebellar projections. Language and executive tasks activated regions of Crus I and lobule VII proposed to be involved in prefrontal-cerebellar loops. Emotional processing involved vermal lobule VII, implicated in cerebellar-limbic circuitry. These data provide support for an anterior sensorimotor vs. posterior cognitive/emotional dichotomy in the human cerebellum. Prospective studies of multiple domains within single individuals are necessary to better elucidate neurobehavioral structure–function correlations in the cerebellar posterior lobe.

Introduction

The cerebellum is involved in a wide range of tasks, including sensorimotor control, language, spatial and executive functions. Deficits resulting from cerebellar lesions include motor dysmetria, ataxia, and intention tremor (Holmes, 1939), but also the cerebellar cognitive affective syndrome (Schmahmann and Sherman, 1998) including executive, visual–spatial, linguistic and emotional deficits, and even mutism and psychosis (Botez-Marquard et al., 1994; Grafman et al., 1992; Heath et al., 1979; Levisohn et al., 2000; Molinari et al., 2004; Rapoport et al., 2000; Riva and Giorgi, 2000; Schmahmann et al., 2007; Steinlin et al., 2003).

The anatomical basis of this proposed cerebellar role in non-motor function is the existence of cerebro-cerebellar channels (cortico-ponto-cerebellar and cerebello-thalamo-cortical loops) that link the cerebellum with motor cortices as well as with association cortices and paralimbic regions of the cerebral hemisphere (Botez et al., 1985; Kelly and Strick, 2003; Leiner et al., 1986; Middleton and Strick, 1994; Schmahmann, 1991, 1996; Schmahmann and Pandya, 1989, 1997; Voogd and Glickstein, 1998). Experimental investigations in animals (Chambers and Sprague, 1955a,b; Snider and Eldred, 1951), imaging studies in humans (Bushara et al., 2001; Grodd et al., 2001, 2005) and clinical reports (e.g., Victor et al., 1959; Schoch et al., 2006) have supported the original hypothesis of Bolk (1906) that there is topography of motor function within the cerebellum (see Manni and Petrosini (2004) for a review). Contemporary clinical studies suggest that whereas the cerebellar anterior lobe is principally engaged in motor control, the cerebellar vermis is involved in affective processing, and the posterior cerebellum contributes to complex cognitive operations (Exner et al., 2004; Levisohn et al., 2000; Schmahmann, 2004, 2007; Schmahmann and Sherman, 1998; Schoch et al., 2006; Tavano et al., 2007). Furthermore, consistent with the crossed cerebro-cerebellar fiber pathways, linguistic impairments can arise following right cerebellar hemisphere lesions, whereas visual–spatial difficulties may follow left cerebellar hemisphere damage (Fiez et al., 1992; Gottwald et al., 2004; Gross-Tsur et al., 2006; Hokkanen et al., 2006; Riva and Giorgi, 2000; Scott et al., 2001). Yet some studies fail to detect non-motor problems after cerebellar tumor or stroke, and others find no reliable structure–function relationships (see Frank et al., 2007). Better understanding of the functional topography of the cerebellum

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doi:10.1016/j.neuroimage.2008.08.039

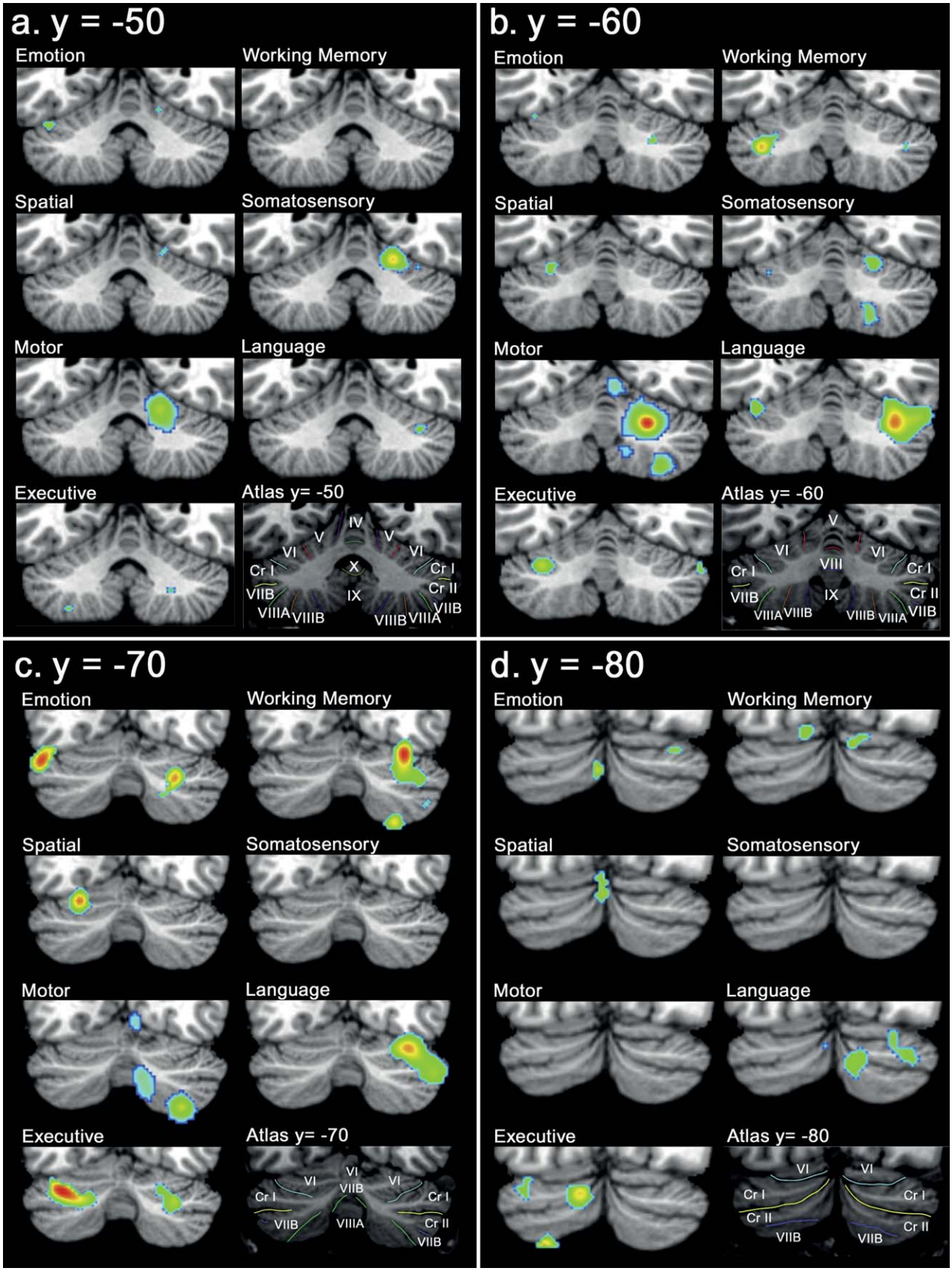

Fig. 1. ALE activation maps for each domain at (a) $y=-50$, (b) $y=-60$, (c) $y=-70$ and (d) $y=-80$ mapped onto representative coronal sections of the Colin27 brain (left cerebellar hemisphere is shown on the left). For each coronal level, at the bottom right are corresponding coronal sections from the MRI Atlas (Schmahmann et al., 2000) with the cerebellar fissures and lobules demarcated and labeled.

Dysphagia (2008) 23:244–250
DOI 10.1007/s00455-007-9127-y

ORIGINAL ARTICLE

Clinical Utility of the 3-ounce Water Swallow Test

Debra M. Suiter · Steven B. Leder

Published online: 4 December 2007

Abstract The 3-ounce water swallow test is frequently used to screen individuals for aspiration risk. Prior research concerning its clinical usefulness, however, is confounded by inadequate statistical power due to small sample sizes and varying methodologies. Importantly, research has been limited to a few select patient populations, thereby limiting the widespread generalizability and applicability of the 3-ounce test. The purpose of this study was to investigate the clinical utility of the 3-ounce water swallow test for determining aspiration status and oral feeding recommendations in a large and heterogeneous patient population. Fiberoptic endoscopic evaluation of swallowing (FEES) was performed in conjunction with the 3-ounce water swallow test on 3000 participants with a wide range of ages and diagnoses. A total of 1151 (38.4%) passed and 1849 (61.6%) failed the 3-ounce water swallow test. Sensitivity of the 3-ounce water swallow test for predicting aspiration status during FEES = 96.5%, specificity = 48.7%, and false positive rate = 51.3%. Sensitivity for identifying individuals who were deemed safe for oral intake based on FEES results = 96.4%, specificity = 46.4%, and false positive rate = 53.6%. Passing the 3-ounce water swallow test appears to be a good predictor of ability to tolerate thin liquids. However, failure often does not indicate *inability* to tolerate thin liquids, i.e., low specificity and high false-positive rate. Use of the 3-ounce water swallow test alone to make decisions regarding safety of liquid intake results in over-referral and unnecessary restriction of liquid intake for nearly 50% of patients tested. In addition, because 71% of participants who failed the 3-ounce water swallow test were deemed safe for an oral diet, nonsuccess on the 3-ounce water swallow test is not indicative of swallowing failure. The clinical utility of the 3-ounce water swallow test has been extended to include a wide range of medical and surgical diagnostic categories. Importantly, for the first time it has been shown that if the 3-ounce water swallow test is passed, diet recommendations can be made without further objective dysphagia testing.

Keywords Deglutition · Deglutition disorders · Aspiration · Dysphagia screening

Accurate identification of individuals who are at risk for oropharyngeal dysphagia is critically important because of the high incidence of pneumonia associated with unrecognized prandial aspiration [1–4]. A clinically useful screening test for dysphagia should provide both high sensitivity and specificity, i.e., accurate identification of individuals who aspirate and require further testing while ruling out nonaspirators who do not require intervention [5, 6]. In clinical practice, a screening test for oropharyngeal dysphagia has three goals: (1) to determine the likelihood that aspiration is present, (2) the need for formal swallow evaluation, and (3) when it is safe to recommend resumption of oral alimentation. One of the Joint

Work was performed at Yale University of Medicine and The University of Memphis.

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doi:10.1093/brain/awq297 Brain 2010: 133; 3578–3597 | 3578

BRAIN
A JOURNAL OF NEUROLOGY

Anosognosia for hemiplegia: a clinical-anatomical prospective study

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Anosognosia for hemiplegia is a common and striking disorder following stroke. Because it is typically transient and variable, it remains poorly understood and has rarely been investigated at different times in a systematic manner. Our study evaluated a prospective cohort of 58 patients with right-hemisphere stroke and significant motor deficit of the left hemibody, who were examined using a comprehensive neuropsychological battery at 3 days (hyperacute), 1 week (subacute) and 6 months (chronic) after stroke onset. Anosognosia for hemiplegia was frequent in the hyperacute phase (32%), but reduced by almost half 1 week later (18%) and only rarely seen at 6 months (5%). Anosognosia for hemiplegia was correlated with the severity of several other deficits, most notably losses in proprioception, extrapersonal spatial neglect and disorientation. While multiple regression analyses highlighted proprioceptive loss as the most determinant factor for the hyperacute period, and visuospatial neglect and disorientation as more determinant for the subacute phase, patients with both proprioceptive loss and neglect had significantly higher incidence of anosognosia for hemiplegia than those with only one deficit or no deficits (although a few double dissociations were observed). Personal neglect and frontal lobe tests showed no significant relation with anosognosia for hemiplegia, nor did psychological traits such as optimism and mood. Moreover, anosognosia for neglect and prediction of performance in non-motor tasks were unrelated to anosognosia for hemiplegia, suggesting distinct monitoring mechanisms for each of these domains. Finally, by using a voxel-based statistical mapping method to identify lesions associated with a greater severity of anosognosia, we found that damage to the insula (particularly its anterior part) and adjacent subcortical structures was determinant for anosognosia for hemiplegia in the hyperacute period, while additional lesions in the premotor cortex, cingulate gyrus, parietotemporal junction and medial temporal structures (hippocampus and amygdala) were associated with the persistence of anosognosia for hemiplegia in the subacute phase. Taken together, these results suggest that anosognosia for hemiplegia is likely to reflect a multi-component disorder due to lesions affecting a distributed set of brain regions, which can lead to several co-existing deficits in sensation, attention, interoceptive bodily representations, motor programming, error monitoring, memory and even affective processing, possibly with different combinations in different patients.

Keywords: anosognosia; consciousness; hemiparesis

Abbreviation: VLSM = voxel-based lesion-symptom mapping

Received March 15, 2010. Revised August 19, 2010. Accepted August 23, 2010

doi:10.1093/brain/awm145 *Brain* (2007), **130**, 2146–2158

Ipsilesional motor deficits following stroke reflect hemispheric specializations for movement control

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Recent reports of functional impairment in the 'unaffected' limb of stroke patients have suggested that these deficits vary with the side of lesion. This not only supports the idea that the ipsilateral hemisphere contributes to arm movements, but also implies that such contributions are lateralized. We have previously suggested that the left and right hemispheres are specialized for controlling different features of movement. In reaching movements, the non-dominant arm appears better adapted for achieving accurate final positions and the dominant arm for specifying initial trajectory features, such as movement direction and peak acceleration. The purpose of this study was to determine whether different features of control could characterize ipsilesional motor deficits following stroke. Healthy control subjects and patients with either left- or right-hemisphere damage performed targeted single-joint elbow movements of different amplitudes in their ipsilateral hemispace. We predicted that left-hemisphere damage would produce deficits in specification of initial trajectory features, while right-hemisphere damage would produce deficits in final position accuracy. Consistent with our predictions, patients with left, but not right, hemisphere damage showed reduced modulation of acceleration amplitude. However, patients with right, but not left, hemisphere damage showed significantly larger errors in final position, which corresponded to reduced modulation of acceleration duration. Neither patient group differed from controls in terms of movement speed. Instead, the mechanisms by which speed was specified, through modulation of acceleration amplitude and modulation of acceleration duration, appeared to be differentially affected by left- and right-hemisphere damage. These findings support the idea that each hemisphere contributes differentially to the control of initial trajectory and final position, and that ipsilesional deficits following stroke reflect this lateralization in control.

Keywords: lateralization; stroke; control; arm movements

Abbreviations: EMG = electromyography

Received March 6, 2007. Revised May 16, 2007. Accepted May 29, 2007. Advance Access publication July 11, 2007

Introduction

While contralateral motor deficits are common following stroke, a number of studies have also revealed more subtle ipsilateral motor deficits that emerge acutely (Jones *et al.*, 1989; Yelnik *et al.*, 1996; Sunderland *et al.*, 1999) and persist chronically (Winstein and Pohl, 1995; Carey *et al.*, 1998; Sunderland, 2000; Haaland *et al.*, 2004; Yarosh *et al.*, 2004; Wetter *et al.*, 2005). These deficits likely reflect the fact that both hemispheres contribute to unilateral limb movements, an interpretation supported by neural activation studies in humans (Kawashima *et al.*, 1994; Dassonville *et al.*, 1997; Kawashima *et al.*, 1998) and by electrophysiology in other animals (Tanji *et al.*, 1988; Donchin *et al.*, 2002; Cisek *et al.*, 2003). It should be noted that the contribution of ipsilateral cortex to unilateral movement does not appear to be symmetric (Kawashima *et al.*, 1993; Kim *et al.*, 1993; Verstynen *et al.*, 2005), which suggests that ipsilesional deficits following unilateral brain damage might also vary with lesion side.

Some previous studies have shown that damage to the left hemisphere selectively impairs the acceleration phase of motion, while right hemisphere damage might selectively impair the deceleration phase (Fisk and Goodale, 1988; Haaland and Harrington, 1989a; Winstein and Pohl, 1995). This has led to the idea that the left and right hemispheres may be differentially specialized for 'open- and closed-loop

JOURNAL OF CLINICAL AND EXPERIMENTAL NEUROPSYCHOLOGY
2012, 34 (10), 1089–1100

Psychology Press
Taylor & Francis Group

Musical memory in a patient with severe anterograde amnesia

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The ability to play a musical instrument represents a unique procedural skill that can be remarkably resilient to disruptions in declarative memory. For example, musicians with severe anterograde amnesia have demonstrated preserved ability to play musical instruments. However, the question of whether amnesic musicians can learn how to play new musical material despite severe memory impairment has not been thoroughly investigated. We capitalized on a rare opportunity to address this question. Patient S.Z., an amateur musician (tenor saxophone), has extensive bilateral damage to his medial temporal lobes following herpes simplex encephalitis, resulting in a severe anterograde amnesia. We tested S.Z.'s capacity to learn new unfamiliar songs by sight-reading following three months of biweekly practices. Performances were recorded and were then evaluated by a professional saxophonist. S.Z. demonstrated significant improvement in his ability to read and play new music, despite his inability to recognize any of the songs at a declarative level. The results suggest that it is possible to learn certain aspects of new music without the assistance of declarative memory.

Keywords: Amnesia; Memory; Music; Skill learning; Emotion.

INTRODUCTION

Patients with dense amnesia due to bilateral medial temporal lobe damage (Anderson et al., 2007; Wilson, Baddeley, & Kapur, 1995) or due to dementia of the Alzheimer's type (Beatty et al., 1999; Beatty et al., 1994; Beatty et al., 1988; Cowles et al., 2003; Crystal, Grober, & Masur, 1989; Fornazzari et al., 2006; Schacter, 1983; for a review see Baird & Samson, 2009) have demonstrated a remarkable ability to continue to perform certain types of activities that they learned prior to brain injury (e.g., driving, playing a musical instrument, and playing golf). The ability to learn and retain new perceptual or motor skills (e.g., rotary pursuit, mirror tracing, and mirror reading) and the ability to learn new habits (e.g., probabilistic learning) are also known to be intact in amnesic patients (e.g., Cavaco, Anderson, Allen, Castro-Caldas, & Damasio, 2004; Cohen & Squire, 1980; Gabrieli, Corkin, Mickel, & Growdon, 1993; Hay, Moscovitch, & Levine, 2002; Milner, 1962; Tranel, Damasio, Damasio, & Brandt, 1994). Cohen and Squire (1980) found that amnesic patients were able to acquire the mirror-reading skill at a normal rate despite poor memory for the words that they had read. This dissociation

Sara Cavaco and Justin S. Feinstein contributed equally. We are greatly indebted to S.Z. and his family for their unwavering support and continued commitment to brain research. We also would like to thank S.Z.'s caregivers and his orchestra for allowing us to observe. Nicolau Pinto Coelho, Mikiko Kanemitsu, Gilberto Bernardes, and Fernando Ramos provided important musical expertise, Kenneth Manzel contributed with the neuropsychological evaluation, and Steven W. Anderson provided invaluable comments on earlier versions of this manuscript. This research was supported by NIH P50 NS19632 and the Kiwanis Foundation.

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http://www.psypress.com/jcen http://dx.doi.org/10.1080/13803395.2012.728568

led these authors to distinguish between declarative forms of memory (dependent on the medial temporal lobe system) and procedural, nondeclarative forms of knowledge, which are often spared in amnesic patients. Declarative memory refers to the capacity for conscious recollection about facts and events, whereas nondeclarative memory is expressed through performance rather than recollection (Squire, 2004). Nondeclarative memory includes different forms of learning and memory abilities, including the perceptual and motor skills involved in musical performance. Even though some aspects of the musical performance can be declared, the actual skills are often carried into action without conscious retrieval of information regarding the procedural aspects of music.

Understanding how the brain processes music and how music can help neurological patients heal and overcome adversity is a rapidly growing field of study (Levitin, 2007; Sacks, 2008). A series of case reports have described patients with significant declarative memory impairments who can still play musical instruments somewhat skillfully (Baur, Uttner, Ilmberger, Fesl, & Mai, 2000; Beatty, Brumback, & Vonsattel, 1997; Beatty et al., 1999; Beatty et al., 1994; Beatty et al., 1988; Cowles et al., 2003; Crystal et al., 1989; Fornazzari et al., 2006; Wilson et al., 1995). All of these reports describe instances of amnesic musicians who are able to perform songs that they had learned how to play prior to the onset of their amnesia. Perhaps the most well known of these cases is Clive Wearing, a renowned musicologist with severe amnesia after sustaining bilateral medial temporal lobe damage due to herpes simplex encephalitis (Wilson et al., 1995, p. 679–680). According to the authors, Clive demonstrated an intact ability to "sight-read, obey repeat marks within a short page, and understand the significance of a metronome mark . . . ornament, play from a figured bass, transpose, and extemporize." This description of Clive's musical skills was the first non-neurodegenerative evidence of relatively preserved ability to perform a musical instrument despite severe multimodal declarative memory impairment. It is currently unknown, however, whether or not Clive is able to learn how to play new songs.

Two early case reports described attempts to teach unfamiliar songs to piano players with dementia of the Alzheimer's type: one by sight-reading (Beatty et al., 1988) and the other by ear (Beatty et al., 1999). Even though both patients were able to play familiar songs that had been learned premorbidly, their ability to learn a new composition was rather limited. However, the patients' significant nonamnestic cognitive impairments may have hampered their ability to engage with the training process. Cowles and colleagues (2003) later described the case of a moderately demented patient with probable Alzheimer's disease who was able to play a new song on the violin and demonstrated some limited capacity to play parts of the song by request (i.e., playing without sheet music) at delays of 0 and 10 min. The attempts to cue the patient's performance by providing the first measures of the new song were unsuccessful. Fornazzari and colleagues (2006, p. 611) assessed the ability of a professional pianist with probable Alzheimer's disease to learn unfamiliar musical pieces and observed "gradual improvements in overall performance and in rhythm, field elements, harmony, melodic accuracy, and sophistication in the accompaniment of the left hand" over a seven-day period. However, the authors did not provide any quantification of the improvements. Baur and colleagues (2000) described a herpes simplex encephalitis patient (C.H.) who learned how to play the accordion, autodidactically, after the onset of her amnesia. Patient C.H. did not have any premorbid sight-reading training nor did she have any experience of playing a musical instrument. Yet, remarkably, she was able to learn how to play 90 pieces of Austrian and German folk music after listening to the songs on the radio or on tape. Moreover, she was able to play a song when cued with the song title, and she was also able to provide the song title when cued with a recording of the music. This suggests that C.H. had preserved declarative memory for the music, despite her overall poor performance on a battery of standardized memory tests. Thus, at least some of C.H.'s intact ability to learn new music could be explained by her reservoir of preserved declarative memory for music. Taken together, the results of the five aforementioned case studies are mixed. Two of the Alzheimer's patients were unable to learn new music, whereas two other Alzheimer's patients showed some residual learning. In addition, the findings in encephalitic patient C.H. are confounded by the patient's ability to learn new declarative information about music.

To date, then, available research does not provide a definitive conclusion about whether the ability to learn and play unfamiliar music can be preserved in the context of a severe impairment in declarative memory. Here, we explored the capacity of an amateur musician, who had dense multimodal anterograde amnesia, to perform and learn a series of new songs after three months of intense practice.

NEUROPSYCHOLOGICAL REHABILITATION
2007, 17 (3), 374–396

Psychology Press
Taylor & Francis Group

Postural abnormalities and contraversive pushing following right hemisphere brain damage

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We investigated the presence of postural abnormalities in a consecutive sample of stroke patients, with either left or right brain damage, in relation to their perceived body position in space. The presence or absence of posture-related symptoms was judged by two trained therapists and subsequently analysed by hierarchical classes analysis (HICLAS). The subject classes resulting from the HICLAS model were further validated with respect to posture-related measurements, such as centre of gravity position and head position, as well as measurements related to the postural body scheme, such as the perception of postural and visual verticality. The results of the classification analysis clearly demonstrated a relation between the presence of right brain damage and abnormalities in body geometry. The HICLAS model revealed three classes of subjects: The first class contained almost all the patients without neglect and without any signs of contraversive pushing. They were mainly characterised by a normal body axis in any position. The second class were all neglect patients but predominantly without any contraversive pushing. The third class contained right brain damaged patients, all showing neglect and mostly exhibiting contraversive pushing. The patients in the third class

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This project has been funded by a Special Doctoral Grant of the Fund for Scientific Research (F.W.O.) - Flanders, Belgium.

http://www.psypress.com/neurorehab DOI:10.1080/09602010601058854

showed a clear resistance to bringing the weight over to the ipsilesional side when the therapist attempted to make the subject achieve a vertical posture across the midline. The clear correspondence between abnormalities of the observed body geometry and the tilt of the subjective postural and visual vertical suggests that a patient's postural body geometry is characterised by leaning towards the side of space where he/she feels aligned with an altered postural body scheme. The presence of contraversive pushing after right brain damage points in to a spatial higher-order processing deficit underlying the higher frequency and severity of the axial postural abnormalities found after right brain lesions.

INTRODUCTION

It has become increasingly clear that the maintenance of body position in space is guided by a complex interplay of many sensory inputs integrated into a postural body scheme or an internal representation of our body in alignment with the gravitational vertical (Bartolomeo & Chokron, 1999; Farné, Ponti, & Ladavas, 1998; Grüsser & Guldin, 1995; Karnath, 1994; Mergner & Hlavacka, 1995; Pérennou, Amblard, Leblond, & Pelissier, 1998; Vallar, Antonucci, Guariglia, & Pizzamiglio, 1993). In man, as well as in most primates, the posterior parietal cortex is the most specialised area of the brain involved in this multisensory integration (Andersen, 1995; Andersen, Snyder, Li, & Stricanne, 1993; Battaglini, Galetti, & Fattori, 1997; Sakata & Kusunoki, 1992; Thier & Andersen, 1997).

Right brain damage is often associated with a disturbed body-centred representation of space, resulting in an ipsilesional mislocalisation of the origin of the spatial axis relative to the body (Andersen et al., 1993; Karnath, 1994, 1997; Ventre, Flandrin, & Jeannerod, 1984). Based on those findings, Taylor, Ashburn, and Ward (1994) mentioned that it is not inconceivable that postural abnormalities, like an asymmetrical trunk orientation, are more likely to be observed in right brain damaged patients. The presence of an abnormal body geometry is also noticed in brain damaged patients, often with hemispatial neglect (Lafosse et al., 2005), showing a very typical group of symptoms, characterised by a postural imbalance caused by a lateropulsion and "pushing away" reaction of the body towards the contralesional side of space (Karnath, Ferber, & Dichgans, 2000), and an active resistance against any attempt at correction of the postural imbalance across or over the midline of the body towards the ipsilesional side. These characteristics are also known as the pusher syndrome (Davies, 1985). Recently, Lafosse et al. (2005) have demonstrated that the increased incidence of contraversive pushing after right brain damage points to a spatial higher-order processing deficit underlying the higher frequency and severity of the axial postural abnormalities after right brain lesions.

Orbitofrontal Cortex and Social Behavior: Integrating Self-monitoring and Emotion–Cognition Interactions

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Abstract

■ The role of the orbitofrontal cortex in social behavior remains a puzzle. Various theories of the social functions of the orbitofrontal cortex focus on the role of this area in either emotional processing or its involvement in online monitoring of behavior (i.e., self-monitoring). The present research attempts to integrate these two theories by examining whether improving the self-monitoring of patients with orbitofrontal damage is associated with the generation of emotions needed to guide interpersonal behavior. Patients with orbitofrontal damage, patients with lateral prefrontal damage, and healthy controls took part in an interpersonal task. After completing the task, participants' self-monitoring was increased by showing them a videotape of their task performance. In comparison to healthy controls and patients with lateral prefrontal damage, orbitofrontal damage was associated with objectively inappropriate social behavior. Although patients with orbitofrontal damage were aware of social norms of intimacy, they were unaware that their task performance violated these norms. The embarrassment typically associated with inappropriate social behavior was elicited in these patients only after their self-monitoring increased from viewing their videotaped performance. These findings suggest that damage to the orbitofrontal cortex impairs self-insight that may preclude the generation of helpful emotional information. The results highlight the role of the orbitofrontal cortex in the interplay of self-monitoring and emotional processing and suggest avenues for neurorehabilitation of patients with social deficits subsequent to orbitofrontal damage. ■

INTRODUCTION

A host of clinical observations, case studies, and two empirical studies show that orbitofrontal damage is associated with impaired interpersonal behavior. Descriptions of orbitofrontal patients have associated damage to this area with the impaired ability to prioritize solutions to interpersonal problems (Saver & Damasio, 1991), a tendency to greet strangers in an overly familiar manner (Rolls, Hornak, Wade, & McGrath, 1994), and disruptive behavior in a hospital setting (Blair & Cipolotti, 2000). Aside from the descriptive evidence, two empirical studies show that orbitofrontal damage impairs interpersonal behavior (Beer, Heerey, Keltner, Scabini, & Knight, 2003; Kaczmarek, 1984). These studies suggest that patients with orbitofrontal damage behave with strangers in ways that are more appropriate for interactions with close others. Patients with orbitofrontal damage tease strangers in inappropriate ways and are more likely to include unnecessary personal information or tangential information when answering questions. Although it is clear that the orbitofrontal region is critically involved in adaptive interpersonal behavior, there has been less agreement on the psychological mechanism responsible for such adaptive behavior.

Social Function of the Orbitofrontal Cortex: Emotion–Cognition Synthesis and Self-monitoring

Current theories suggest that two types of variables contribute to the social deficits associated with orbitofrontal damage: deficient emotional systems or a lack of online behavioral monitoring. Several theories propose that emotional deficits, in one form or another, account for the impaired interpersonal behavior associated with orbitofrontal damage (e.g., Kringelbach & Rolls, 2004; Bechara, Damasio, & Damasio, 2000; Elliott, Dolan, & Frith, 2000). For example, the *somatic marker hypothesis* proposes that the orbitofrontal cortex is critical for interpreting somatic sensations (equated with emotion in this framework) that are needed to make decisions (e.g., Bechara et al., 2000; Bechara, Damasio, Tranel, & Damasio, 1997). From this perspective, people avoid making social blunders because particular physiological sensations guide them toward adaptive behavior and away from maladaptive behavior. Empirical support for the somatic marker hypothesis comes from a series of gambling studies that have found that patients with

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Preserved Self-Awareness following Extensive Bilateral Brain Damage to the Insula, Anterior Cingulate, and Medial Prefrontal Cortices

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Abstract

It has been proposed that self-awareness (SA), a multifaceted phenomenon central to human consciousness, depends critically on specific brain regions, namely the insular cortex, the anterior cingulate cortex (ACC), and the medial prefrontal cortex (mPFC). Such a proposal predicts that damage to these regions should disrupt or even abolish SA. We tested this prediction in a rare neurological patient with extensive bilateral brain damage encompassing the insula, ACC, mPFC, and the medial temporal lobes. In spite of severe amnesia, which partially affected his ''autobiographical self'', the patient's SA remained fundamentally intact. His Core SA, including basic self-recognition and sense of self-agency, was preserved. His Extended SA and Introspective SA were also largely intact, as he has a stable self-concept and intact higher-order metacognitive abilities. The results suggest that the insular cortex, ACC and mPFC are not required for most aspects of SA. Our findings are compatible with the hypothesis that SA is likely to emerge from more distributed interactions among brain networks including those in the brainstem, thalamus, and posteromedial cortices.

Citation: Philippi CL, Feinstein JS, Khalsa SS, Damasio A, Tranel D, et al. (2012) Preserved Self-Awareness following Extensive Bilateral Brain Damage to the Insula, Anterior Cingulate, and Medial Prefrontal Cortices. PLoS ONE 7(8): e38413. doi:10.1371/journal.pone.0038413

Editor: Yoko Hoshi, Tokyo Metropolitan Institute of Medical Science, Japan

Received June 11, 2011; **Accepted** May 9, 2012; **Published** August 22, 2012

Funding: This study was supported by grants from the National Institute of Neurological Disorders and Stroke (NINDS, P01 NS19632), National Institute on Drug Abuse (NIDA, R01 DA022549), and the Mathers Foundation, and by funds from the University of Iowa, College of Medicine. The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

Competing Interests: The authors have declared that no competing interests exist.

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Introduction

Self-awareness (SA) is a complex, rich and integrated phenomenon of self-knowledge, which is central to consciousness and incorporates multiple components [1,2,3,4,5,6,7]. As a first approximation, one can distinguish several putative components of SA, which are hierarchically organized, and which might be partially dissociated, both functionally and neuroanatomically. Each component is in turn made up of multiple elements. The following description is meant to serve as a heuristic tool.

a) *Core SA* is the most basic and fundamental component of SA and forms the foundation for all other components. It is grounded on the protoself, which includes ''primordial feelings'' of the living body [3,8] and a preattentive, elementary form of self-consciousness [9]. On a moment to moment basis, Core SA generates a sense of personal agency and ownership over behavioral actions and sensory representations. Processes such as self-recognition and sentience require Core SA.

b) *Extended SA* broadens Core SA to include an autobiographical self [3,8], which involves an elaborate self-concept built upon a repository of autobiographical memories and representations of physical, affective and personality traits.

c) *Introspective SA* relies on higher-order executive, attentional and metacognitive functions, which enable *introspection*, the ability to perform a more or less controlled *reflection* on one's own mental states, behaviors, and their consequences. Supported by memory and learning, introspective SA allows for the development of accurate knowledge about one's self, a capacity critical for efficient navigation of the social world [10,11].

The study of the neural basis of SA has grown significantly over the past two decades [3,4,8,12,13,14,15,16,17]. Several recent theoretical frameworks produced partially overlapping hypotheses regarding the specific neural substrates of SA or various components of SA. One class of hypotheses emphasizes specific brain regions, which would play a central role in essential components of SA [18,19,20,21,22]. Another class of hypotheses focuses on more distributed cortico-cortical and/or subcortical-cortical networks [4,8,16,23,24,25,26,27].

Here we concentrate on the first class of hypotheses. Four main anatomical targets have been proposed based on a variety of

OEFENINGEN EN OPDRACHTEN

ITON-Observatielijst bij video “Ik zie niet waar ik voel”

Noteer welke symptomen / stoornissen u meent te zien.

A ZIEKTE-INZICHT (noso-agnosie) 1. verbaal 2. gedrag	
B GEHEUGEN 3. oriëntatie tijd, plaats, persoon 4. onthouden informatie - korte termijn - lange termijn	
C COMMUNICATIE 5. motorische afasie 6. sensorische afasie 7. gemengde afasie 8. amnestische afasie 9. agrafie 10. alexie 11. aprosodie 12. acalculie	
D RUIMTELIJKE FUNCTIES 13. visuospatiële agnosie 14. ruimtelijke apraxie 15. links-rechts onderscheid	
E WAARNEMEN 16. visuele agnosie 17. akoestische agnosie 18. tactiele agnosie / stereo-agnosie 19. somato-agnosie 20. prosopagnosie	
F HANDELEN 21. motorische apraxie 22. ideatorische apraxie 23. kleding-apraxie 24. diversen: faciaal, verbaal etc. 25. perseveratie	
G AANDACHT 26. bewustzijn / alertheid 27. neglect - visueel - akoestisch - tactiel - motorisch 28. extinctie	
H ‘FRONTALE FUNCTIES’ 29. anticiperen 30. sociaal adequaat gedrag 31. generaliseren	
I PRIMAIRE NEUROLOGISCHE FUNCTIES 1. parese / paralyse 2. sensibiliteitsstoornis 3. hemi-anopsie 4. diversen	
ANDERE OBSERVATIES	

Oefening 1.1: Neuroanatomie “in vogelvlucht”

Voor het goed kunnen volgen van de cursus is een zekere minimumkennis van de neuroanatomische termen noodzakelijk. Deze kennis is te vinden in hoofdstuk 3.5 van het boek “Neurowetenschappen”. Ook kan een neuroanatomie-atlas gebruikt worden. Tijdens de cursus zijn meerdere hersenmodellen (uitneembaar) aanwezig.

Zoek op resp. wijs aan op het hersenmodel de volgende structuren:

- linker en rechter hemisfeer
- corpus callosum
- cerebellum
- lobus frontalis, parietalis, temporalis en occipitalis
- sulcus lateralis (Sylvii)
- sulcus centralis (Rolandi)
- gyrus pre- en postcentralis
- prefrontale en orbitale cortex
- insula

- limbisch system, hypothalamus, hypofyse, amygdala
- basale kernen: nucleus caudatus + putamen = striatum; globus pallidus
- thalamus
- capsula interna
- hippocampus, gyrus cinguli

- diencephalon, mesencephalon, pons, medulla oblongata
- formatio reticularis
- hersenzenuwen: N. Olfactorius, N. Opticus
- chiasma opticum en tractus opticus

- ventrikelsysteem: laterale, 3^{e} en 4^{e} ventrikel, aqueductus cerebri

- hersenarteriën : cirkel van Willis, A. Cerebri anterior, media, posterior, A. Basilaris en vertebralis

Wat wordt bedoeld met:
- dorsaal, ventraal, rostraal, caudaal, lateraal, mediaal en mediaan?

Oefening 1.2: “Brein en dagelijks leven”

Bij het tot stand komen van allerlei taken en functies spelen verschillende hersengebieden een specifieke rol. bepaalde gebieden zijn tijdens één taak tegelijkertijd actief. Deze gebieden weerspiegelen de deelfuncties die bij zo’n taak betrokken zijn.
Andere gebieden worden geactiveerd in een bepaalde volgorde, corresponderend, met bepaalde fasen van de taak of handeling (bijvoorbeeld reiken, grijpen, buigen).

Probeer bij de onderstaande opdrachten twee vragen te beantwoorden:
1. Welke deelfuncties zijn betrokken bij de genoemde taak (bijv. armbeweging, taalbegrip, visuele herkenning)?
2. Waar bevinden zich de betrokken gebieden in het hersenmodel?

Hierbij kan gebruik gemaakt worden van het boek “Neurowetenschappen” hoofdstuk 17, met name figuur 17.17.

1. Iemand komt in een kamer, zoekt een stopcontact, vindt dit, steekt de stekker van zijn laptop erin en begint te typen.

2. Een vader fietst met zijn kind naar school, ziet van links een auto aankomen en zegt: “Kijk uit, daar links komt een auto!”

3. Een violist pakt zijn studieboek en begint een etude te oefenen. Daarna zet hij een CD op, probeert de melodie te onthouden en tracht dit op viool na te spelen.

4. Een tennisser rent naar voren en geeft een smash.

5. Een voetballer schiet op doel vanuit een voorzet.

6. Iemand zit te suffen na een vermoeiende werkdag, schrikt op door de telefoon, vloekt en neemt op.

7. Een moeder leest haar kind voor uit een spannend sprookjesboek (N.B. beschrijf wat er gebeurt in moeder en kind).

8. Een kruidenier inventariseert zijn voorraad. Hij loopt door de winkel en schrijft ieder artikel dat hij ziet op een lijst.

9. Een lijnrechter bij een tenniswedstrijd fixeert zijn blik op de tennisbal en volgt deze voortdurend.

10. Een fysiotherapeut zegt tegen een patiënt/cliënt: ”Hef uw arm horizontaal naar voren.” Dit lukt niet. Vervolgens zegt de fysiotherapeut: “Geef mij eens een stomp.” Dit lukt wel (de arm wordt naar voren gestrekt).

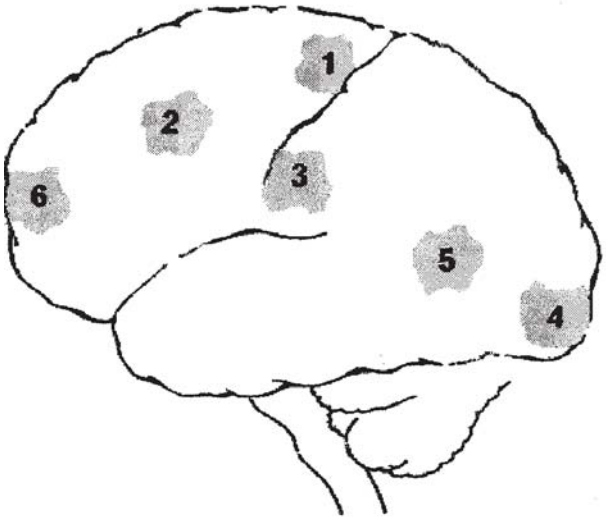
Oefening 2: “van laesie naar symptoom”, “van stoornis naar consequenties”

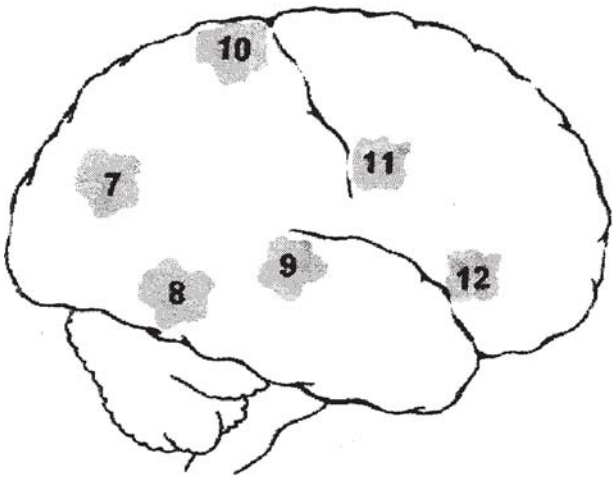
A. Bij lokale laesies van de hersenschors ontstaan soms epileptische aanvallen. Deze zijn gekenmerkt door prikkelingsverschijnselen (soms ook uitvalsverschijnselen) die samenhangen met de functie van het geprikkelde gebied.
   Beschrijf nu het mogelijke verloop van een focale epileptische aanval bij lokalisatie van het focus:
   - links, gyrus precentralis lateraal
   - links occipitaal
   - links fronto-lateraal
   - rechts, gyrus postcentralis mediaal
   - rechts temporaal mediaal
   - rechts premotorische schors lateraal

B. Vul de matrix in die hoort bij de twaalf leasies van Figuren 1 en 2 op de volgende pagina (zie ook Figuren 5.4 en 6.5 in het boek “Neuropsychologie”). Kies per lokalisatie steeds één functie en één stoornis. Besteedt vooral aandacht aan de 4e kolom: geeft steeds een mogelijke consequentie op activiteiten- en participatieniveau.
   Vergelijk je antwoorden met die van Tabellen 5.1 en 6.1 uit het boek “Neuropsychologie”. Omdat deze tabellen slechts één of enkele voorbeelden geven, zullen er zeker verschillen zijn met je eigen antwoorden. Ga na of je antwoord desalniettemin juist is.

C. Met het oog op therapie/training is het goed te weten wat de sterke en zwakke kanten van de patiënt/cliënt zijn. Geef op de volgende pagina in beide kolommen aan wat bij ieder van de laesies de “zwakke/gestoorde en dus riskante kanalen” zijn en wat de “intacte, relatief sterke kanalen” zijn.

D. Na opdrachten B en C is het zinvol alvast te gaan werken aan het prakticum van de laatste dag (zie achterin). Beschrijf zo compleet mogelijk het klinische beeld van de patiënten/cliënten met laesies op de aangegeven plaatsen (hanteer hierbij de systematiek die in de cursus besproken is).





	Intact / relatief sterk kanaal	Gestoord / relatief zwak kanaal
1		
2		
3		
4		
5		
6		
7		
8		
9		
10		
11		
12		

	Lokalisatie in anatomische termen	Belangrijkste functie	Functiestoornis bij laesie	Consequentie op activiteiten- en participatieniveau in gewoon Nederlands
1				
2				
3				
4				
5				
6				
7				
8				
9				
10				
11				
12				

Oefening 3: “van symptomen naar laesie”

A. Waar zou de laesie gelokaliseerd kunnen zijn bij de volgende symptomen?
   - o spastische parese van beiden benen
   - o hemi-anesthesie van linker arm en gelaat
   - o dwangstand van de ogen naar rechts. De ogen kunnen de vinger van de onderzoeker niet naar links volgen
   - o prosopagnosie
   - o linkszijdige facialisparese. De oogsluiting is symmetrisch
   - o tactiele agnosie van de rechter hand

B. Welke combinaties van symptomen zijn waarschijnlijk en waarom?
   - spastische parese van beide armen
   - linkszijsige hemi-anopsie met expressieve afasie
   - parese van het rechter been met hypesthesie van het rechter been
   - tactiele extinctie rechts met afasie van Wernicke
   - neglect met afasie
   - spastische parese en apraxie

C. Welk ander symptoom kan men verwachten wanneer het volgende symptoom is vastgesteld?
   (Ga na: welke hemisfeer, welk hersengebied, wat is de functie van de aangrenzende gebieden?)
   - linkszijdige centrale facialisparese
   - parese van de rechter hand en het gelaat
   - noso-agnosie
   - rechtszijdige hemi-anopsie
   - tactiele agnosie van de rechter hand

D. Figuur 9.7 in het boek “Neuropsychologie” geeft een aantal storingen waarmee apraxie verward kan worden.
   Geef in ieder van de genoemde gevallen criterial op grond waarvan men de aangegeven stoornissen kan onderscheiden (N.B. op pagina 205 in het boek “Neuropsychologie” zijn reeds 4 voorbeelden besproken).

Oefening 4: Huisjes

Voer een discussie naar aanleiding van Figuur 5.6 in het boek “Neuropsychologie”. Welke aspecten zijn kenmerkend voor (laesies van) linker en rechter hemisfeer? Geef aan welke problemen in het dagelijks leven hiermee verband zouden kunnen houden.

Oefening 5: Informatie geven aan patiënt/cliënt/partner/familie

A. Leg in eenvoudige bewoordingen, maar wel correct, uit wat de onderstaande stoornissen inhouden:
   - neglect in het algemeen, akoestisch neglect in het bijzonder
   - agnosie in het algemeen, visuele agnosie in het bijzonder
   - apraxie in het algemeen, ideatorsiche apraxie in het bijzonder

   Geef in ieder van deze gevallen een duidelijk dagelijks leven voorbeeld

B. Bovenstaande stoornissen kunnen voorkomen in het kader van een dementie, maar ook **zonder** dementie. Het is belangrijk dit te onderscheiden. Hoe zou je dit uitleggen?

C. Welk advies zou je geven aan het bezoek in het geval van een patiënt/cliënt met gezichtherkenningsstoornissen (prosopagnosie)?

D. De echtgenoot van een vrouwelijke CVA-patiënt zegt tegen je: “als ze maar weer kan lopen en praten, dan kan ze naar huis en dan komt alles wel weer goed.” Hoe zou je reageren?

Oefeningen 6: Casus gedragsverandering na hersenbeschadiging

Vorm je een oordeel over onderstaande casus. Pas hierbij toe wat je in hoofdstuk 12 gelezen hebt.

1. Een vrouw van 72 jaar is na een CVA links en een langdurige revalidatie weer thuis. Ze is redelijk ADL-zelfstandig, haar parese is nog wel aanwezig, maar vormt nauwelijks meer een handicap. De aanvankelijk ernstige afasie is goed hersteld: er zijn alleen af en toe nog wat woordvindingsproblemen.
   Het gaat thuis echter niet goed. De relatie met haar man wordt steeds moeilijker. Haar man zegt dat ze sinds die beroerte zo egocentrisch is geworden: ze praat alleen maar over zichzelf en bekommert zich niet om het wel en wee van haar partner. Haar man denkt dat zij nu wraak neemt op hem omdat hij 10 jaar geleden iets met een secretaresse had.

2. Een man verblijft na een CVA links in het verpleeghuis. Opvallend is dat hij de laatste tijd zijn kamer niet meer uit wil (hoewel hij goed ter been is). De fysiotherapeut en de logopedist moeten maar op zijn kamer komen. Hij komt z'n bed niet meer uit en vereenzaamt. De verpleeghuisarts suggereerde dat er sprake was van een depressie.

3. Een man met een CVA rechts is motorisch goed hersteld. Toch is hij lang niet ADL-zelfstandig. Er zijn o.a. grote problemen met aankleden. Zijn vrouw heeft hem een paar keer langdurig met een overhemd zien worstelen. Ze zegt dat ze bang is dat hij dement wordt, want hij doet zo dom tijdens dat aankleden. Bovendien is er, vertelt zij, een broer van hem zeer dement geworden; bij hem was het ook met aankleed-problemen begonnen.

4. Een vrouw van 67 jaar kreeg een infarct in het stroomgebied van de A. Cerebri posterior rechts. Haar dochter denkt dat zij dement wordt. Zij zegt/vraagt namelijk heel vaak hetzelfde, soms wel 5 maal binnen een half uur. Als haar dochter (vrijwel dagelijks) op bezoek komt wordt ze vaak niet herkend, hoewel haar moeder pal voor het raam zit en haar op 50 m afstand (de bushalte) al kan zien aankomen. Pas als ze eenmaal bij haar op de kamer is lijkt het door te dringen wie er is.

5. Een man van 78 jaar kreeg een zgn. apoplexie. Hij heeft een zeer sterke linkszijdige parese van arm en been, kan niet los zitten en staan, is incontinent en sterk afhankelijk van hulp. Opvallend is dat hij zoveel eet. Hij eet alles op wat de andere patiënten op zijn kamer (4) laten staan. De zusters zijn hier langzamerhand erg verbaasd over. Zijn vrouw beweert dat hij zoveel eet uit frustratie: "wat moet je anders als je niks meer kan?"

6. Een vrouw is na een CVA rechts in het verpleeghuis. Vrijwel dagelijks komt er familie op bezoek. Haar man klaagt bij de verpleeghuisarts dat ze zo vaak "ongeïnteresseerd" is, ze let niet goed op het verloop van het gesprek en lijkt vaak dingen niet op te nemen.
   Haar man vraagt of dit aan de pillen ligt, of aan het CVA.

7. Een man van 81 jaar krijgt een CVA rechts en wordt na een verblijf van 3 dagen in een ziekenhuis, opgenomen in het verpleeghuis. Hij is er ernstig aan toe: kan niet lopen, kan zichzelf niet wassen en aankleden, kan niet zelf naar het toilet. In de gesprekken die met hem gevoerd worden vertelt hij steeds een ander verhaal waarom hij hier is: voor de behandeling van z'n rechter knie (die gebroken is geweest en indertijd niet goed behandeld is), voor bloedonderzoek, voor controle van z'n trombosebeen etc.

Oefeningen 7: Casuïstiek

Bespreek welke stoornissen ten grondslag kunnen liggen aan de geschetste problemen. Geef een nadere precisering van de stoornis (“apraxie” of “neglect” is dus te vaag!) en geef aan waarom dit noodzakelijk is.

1. Een 63-jarige man met een CVA in de rechter hemisfeer heeft een matige parese van linkerarm en been. Er zijn geen duidelijke sensibiliteitsstoornissen, en er is geen hemianopsie. In de revalidatiekliniek is de patient al meerdere malen met de rolstoel tegen de deurpost gebotst. Hoewel het toilet zich onmiddellijk links van zijn kamer bevindt, is hij daar al een paar maal voorbijgereden en raakte verdwaald op ver verwijderde afdelingen. Bij de verpleging klaagde hij regelmatig over het feit dat de foto van z’n vrouw was weggehaald (dit was niet waar: de foto stond links op het tafeltje naast zijn bed). Onlangs liep deze man een lelijke verwonding op aan zijn linkerhand die tussen de spaken van het wiel van de rolstoel bekneld raakte.

2. Een vrouw van 70 jaar met een CVA in de linker hemisfeer heeft een ernstige receptieve afasie (Wernicke); zij begrijpt niet wat er tegen haar gezegd wordt. Er is een lichte parese van rechter hand en gelaat, de sensibiliteit van de rechter hand is wat verminderd en er is uitval in de rechter gezichtsvelden (vooral de onderquadranten). Tijdens het orienterend neuropsychologisch onderzoek is de patiente er op geen enkele manier toe te bewegen een duidelijke grote klok met wijzerplaat te tekenen: tot viermaal toe produceert zij een onooglijk, klein en nauwelijks herkenbaar klokje. Ook bij vele andere testjes volhardt zij in een hardnekkig “niet-begrijpen”, ondanks al onze pogingen om de bedoeling van de test op een niet-talige manier duidelijk te maken (o.a. aanwijzen, voordoen). Misschein zijn de begripsstoornissen zeer fundamenteel en betreffen deze niet alleen maar de taal.

3. Een vrouw van 71 jaar lijkt goed hersteld van een hemiparese na een CVA in de linker hemisfeer. Ze heeft echter grote moeite met aankleden en wassen. Wanneer zij haar rechter hand gebruikt valt de zeep herhaaldelijk uit haar hand; bij het haar kammen met de rechter hand maakt zij slechts doelloze bewegingen in de lucht; ze heeft veel moeite met het dichtmaken van knoopjes. Ze is het gebruik van de rechter arm (die qua spierkracht goed hersteld is) gaan vermijden. Als ze aan tafel het zoutvat moet pakken (dat geheel rechts van haar staat) doet ze dat heel omslachtig met haar linker arm. De sensibiliteit van rechter arm en hand (tast en kinesthesie) is opgeheven.
   NB: welke compensatiestrategie heeft deze vrouw “gekozen”? Welke andere compensatie- strategie is ook mogelijk?

4. Een jongen van 19 jaar liep door een verkeersongeluk linkszijdig hersenletsel op. Hij had een hemiparese rechts en een motorische afasie. Een jaar na het ongeval is het met veel moeite gelukt hem, met rolstoel, weer bij zijn ouders thuis te krijgen. De taal is redelijk hersteld. Op zijn verzoek werd veel aandacht besteed aan het autorijden. Met een aangepaste auto (gedeeltelijke handbediening) is dit nu goed gelukt: sturen, remmen etc. gaat uitstekend. Er zijn echter wat problemen in het verkeer: hij beging meerdere overtredingen. Laatst werd hij op een drukke weg aangetroffen in de verboden rijrichting. Hij vraagt advies in de revalidatiekliniek omdat hij zich een testje met kruispunten herinnerde.

5. Een 12-jarig meisje kwam met haar fiets onder een auto en liep ernstig hersenletsel op. Ze lag 5 weken in coma. Ze heeft een spastische parese van alle ledematen. De linker arm kan nog enigszins gebruikt worden. Ze zit in een rolstoel. Tijdens de therapie en recreatie heeft zij vaak ineens heftige uitbarstingen van “woede”: ze gooit dan alles om wat in de buurt is, doet vaak andere kinderen pijn, en schreeuwt als een bezetene. Volgens de verpleging en de activiteitenbegeleiders is dit gedrag een reactie op een gevoel van machteloosheid. De fysio- en ergotherapeuten hebben een andere mening: volgens hen wordt het gedrag direct veroorzaakt door het hersenletsel.

NB in hoofdstuk 14 van het boek “Neuropsychologie” staan nog 11 andere casussen waarover op dezelfde wijze gediscussieerd kan worden.

Oefeningen 8: Testresultaten

1. Hoofdstuk 14.2 van boek “Neuropsychologie”. Probeer in een onderlinge discussie tot een interpretatie te komen. Hierbij is vooral van belang af te wegen of een resultaat als een kenmerkende “stoornis” gezien kan worden, of ook als een gewone “vergissing”.

2. Een CVA-patiënt heeft een uitgebreide neuropsychologische test ondergaan. De conclusie luidt: “ernstige apraxie”. Op de afdeling heeft de verpleging echter niets bijzonders gemerkt, d.w.z. wassen, aankleden, eten gaat allemaal uitstekend. Wie heeft er gelijk?

3. Maak 2 lijstjes van redenen:
   A. Waarom de patiënt bij de test beter presteert dan thuis of in het gewone dagelijks leven.
   B. Waarom de patiënt thuis of in het dagelijks leven juist beter presteert dan bij de test.

4. In verband met neglect heeft een patient intensief geoefend met “H-tjes doorstrepen”. Dit gaat nu perfect en ook het lezen van de krant geeft geen problemen meer. Buiten gedraagt de patiënt zich echter gevaarlijk: al drie keer is bijna een ongeluk gebeurd omdat hij verkeer van links te laat opmerkte. NB: dit waren steeds fietsers. Kan je dit verklaren?

Opdracht 1: Korte casus neuropsychologie

Neuropsychologische functiestoornissen komen vaak voor bij CVA-patiënten. Dit zijn bijv. stoornissen in waarnemen of handelen die niet terug te voeren zijn op de eventuele verlamming. Men spreekt van agnosie, apraxie, amnesie, afasie etc.

Een lijst van definities is te vinden op pagina 367 e.v. in het boek "Neuropsychologie". Tijdens de werkzaamheden in verpleeghuis/kliniek heb je ongetwijfeld te maken gehad met CVA-patiënten die dergelijke stoornissen vertoonden, bijv. een patiënt die zich niet kan aankleden door een apraxie, of een patiënt die zijn eigen arm met de uwe verwart.

Opdracht:

Beschrijf in maximaal 10 regels (getypt) een CVA-patiënt uit uw eigen ervaring, waarbij je vermoedt dat zo'n neuropsychologische stoornis een rol speelde. Lever dit in, en maak voor jezelf een kopie.
Geef deze korte casus een duidelijke of sprekende titel die het vermoede symptoom aangeeft. Voeg eventueel een vraagstelling toe.

Voorbeeld

Linkszijdig neglect?

Een 71-jarige vrouw met een CVA in de rechter hemisfeer had geen verlamming van ledematen. Wel gaf zij aan met de linker arm niets te voelen. Ze had zich al tweemaal verwond aan haar linker arm: eenmaal tussen de spaken van het wiel van de rolstoel, een andere keer liep zij "zomaar" met de linker arm hard tegen de deurpost.
Ze had vaak problemen met het vinden van ruimten binnen het verpleeghuis (toilet, therapie, recreatieruimte).
Zou hier een linkszijdig neglect in het spel kunnen zijn?

Opdracht 2: Korte casus gedragsstoornis

Bij CVA-patiënten komen gedragsstoornissen regelmatig voor. Sommige bemoeilijken de behandeling van de patiënt, sommige bemoeilijken de sociale contacten van de patiënt. Veel gedragsstoornissen blijven hardnekkig bestaan, ondanks de moeite die er al is gedaan om ze te verhelpen. In een aantal gevallen zijn ze zo ernstig dat de patiënt niet meer te handhaven is in verpleeghuis of kliniek.

Voorbeelden van gedragsstoornissen: patiënt weigert mee te werken; huilt voortdurend; ontkent dat er iets met hem/haar aan de hand is; trekt zich geheel terug in zichzelf; is erg “depressief” of initiatiefloos; heeft woede aanvallen enz.

Gedragsverandering na hersenbeschadiging kan het directe gevolg zijn van de laesie, maar ook een meer indirecte reactie op de handicap. Soms reageert de omgeving extreem, met alle gevolgen van dien. Misschien was het gedrag al aanwezig vóór het CVA.

Het lezen van hoofdstuk 12 uit “Neuropsychologie” roept wellicht een herinnering op aan een patiënt die extreem of merkwaardig gedrag vertoonde.

Opdracht

Beschrijf in maximaal 10 regels (getypt) een CVA-patiënt uit uw eigen ervaring die een gedragsstoornis vertoont/vertoond heeft.
Lever deze casus in, en maak voor Uzelf een kopie. Geef deze casus een duidelijke titel en voeg eventueel een vraagstelling toe.

Voorbeeld

Ontremd, sociaal onaangepast gedrag

Een 68-jarige manlijke CVA-patiënt heeft vele keren per dag ruzie met zijn medepatiënten. De man zoekt voortdurend gezelschap van anderen en vraagt dan niet aflatend de aandacht: hij maakt - vaak seksueel gekleurde - grappen, klaagt over het personeel of over zijn kinderen. Hij lacht vaak zonder aanwijsbare reden heel hard. Laat na veel geruk aan zijn kleren enkele ernstige littekens op zijn buik zien, laat harde boeren en lacht er zelf langdurig om. Hij gedraagt zich daarbij zeer indringend: vaak moet hij door het personeel bij zijn “slachtoffer” weggehaald worden, omdat deze zelf niet in staat is hem kwijt te raken.

Practicum neuropsychologisch onderzoek (In het kader van de ITON cursus)

Doelen van het practicum zijn o.a.:
- een idee krijgen hoe een oriënterend neuropsychologisch onderzoek eruit kan zien
- inleven in een patiënt: voorbereiden aan de hand van lokalisatie van de Iaesie
- interpreteren van de onderzoeksgegevens

Werkwijze:
Er zullen tijdens het practicum groepjes geformeerd worden van elk drie personen. De één "speelt" patiënt, de ander is onderzoeker, de derde persoon observeert en scoort (d.m.v. loting wordt bepaald wie welke rol heeft).
Voorafgaande aan het practicum zal in groepjes de rol van patiënt, onderzoeker en observator besproken worden (dit is een ander groepje dan bij het practicum). Voor het onderzoek heb je één uur de tijd. Na het onderzoek is er tijd om het geheel in je groepje te bespreken. Verder zal er een plenaire nabespreking zijn.

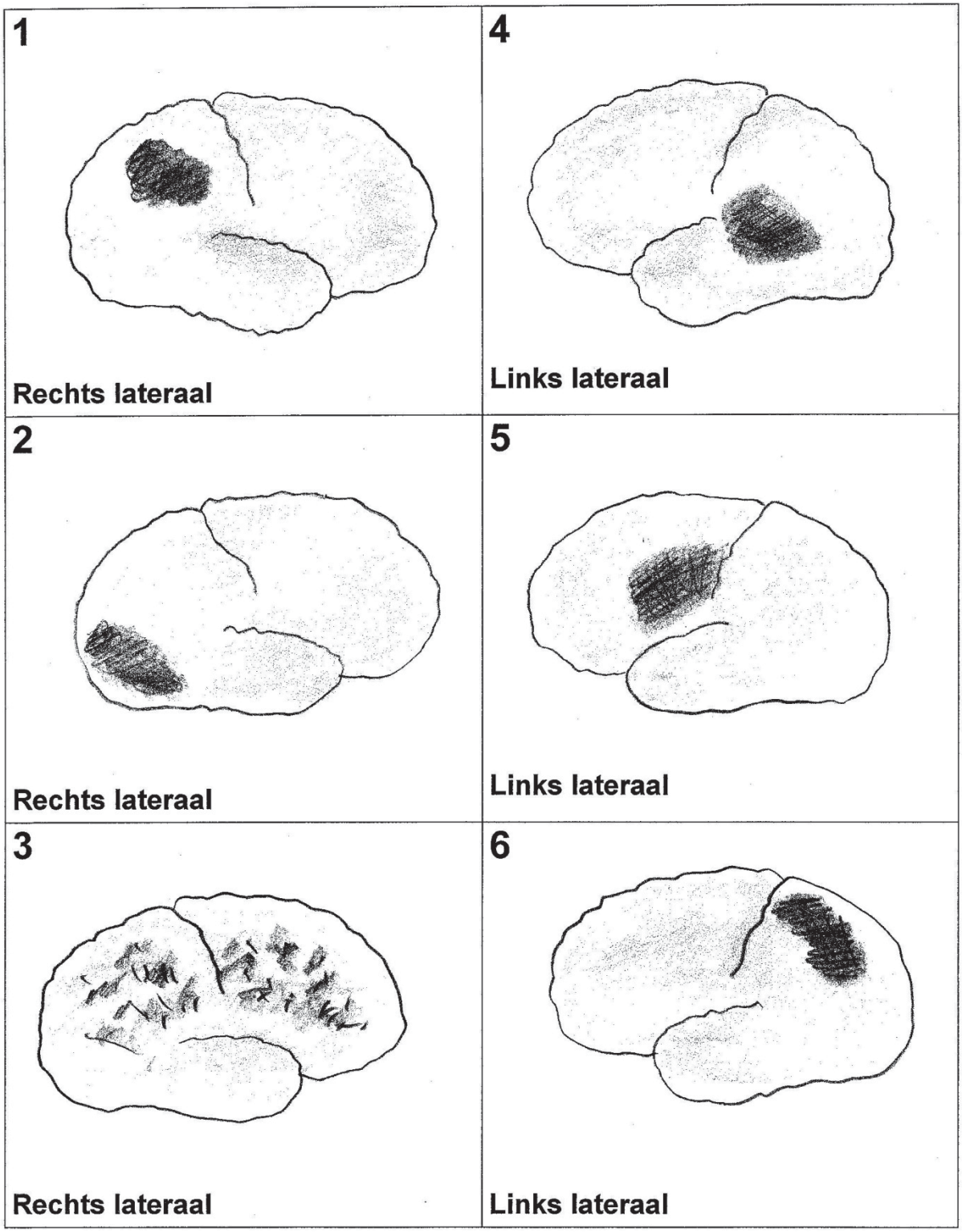
Voorbereiding:
- Patiënt: Op de volgende pagina is een schema van zes patiënten met hersenletsel. Het laatste cijfer van je telefoonnummer dat valt onder de cijfers 1 t/m 6 is het nummer van je "letsel". Dus 3838748 levert op: letsel 4. Het is de bedoeling dat iedereen het klinische beeld voorbereidt dat kan passen bij deze lokalisatie. Ga na welke onderdelen van het neuropsychologisch onderzoek vooral bemoeilijkt zullen zijn.
- Onderzoeker: bedenk aan de hand van de bijgevoegde tekst hoe je het onderzoek vorm gaat geven, en welke materialen nodig zijn: zie hiervoor de blokken bij de verschillende onderdelen.
- Observator: bekijk de wijze waarop gescoord wordt.

Voor alle duidelijkheid: iedereen bereidt zich zowel voor als patiënt, onderzoeker en als observator; zo is het leereffect het grootst. Het kan dus zijn dat je het materiaal dat je verzameld hebt, niet gebruikt omdat je als patiënt of observator speelt!

Het onderzoek:
Ga aan een tafel zitten, stal het materiaal zo uit dat je er makkelijk bij kunt. Standaardiseer ook de wijze waarop het materiaal wordt aangeboden, leg het materiaal bijvoorbeeld steeds midden voor de patiënt. Dit is met name van belang in verband met de symptomen neglect en/of hemianopsie. Gebruik de bijgevoegde tekst als handleiding. Een voorbeeld scoreformulier is tevens bijgevoegd.

Let op: Het hieronder beschreven onderzoek is niet hetzelfde als het Oriënterend Neuropsychologisch Onderzoek (ONO), er zijn overeenkomsten, maar in verband met de doelstelling en praktische haalbaarheid van dit practicum zijn enkele onderdelen weggelaten en is de scoring versimpeld. Er zal één ONO-koffer aanwezig zijn, indien daar belangstelling voor bestaat zal het ONO door een ITON medewerker bij één van de cursisten afgenomen kunnen worden, een andere cursist kan tijdens dit practicum observeren en scoren.

Schema zes patiënten met hersenletsel



Scoring

Alle onderdelen worden gescoord op kwaliteit van het resultaat middels een 3 - puntsschaal. In het kader van dit practicum is dit vereenvoudigd in “normaal”, “enigszins afwijkend” en “zeer afwijkend”. Geheel rechts op het formulier is ruimte voor opmerkingen. Hier kunnen opvallende bevindingen genoteerd worden. In sommige gevallen is een onderdeel niet afneembaar, bijv. wanneer een verbale respons wordt verwacht van een patiënt met een motorische afasie. Ook kunnen er andere redenen zijn waarom een onderdeel wordt overgeslagen; dit wordt weergegeven in de kolom ‘niet onderzocht’. Noteer in de kolom “opmerkingen” waarom. Voor sommige onderdelen zijn bij het ONO alternatieven aangegeven. Deze zgn. ‘schaduwvragen’ vormen een variant van het ONO voor patiënten bij wie het standaard ONO niet mogelijk is, en dienen op het scoreformulier apart gescoord te worden. In je voorbereiding op dit practicum kun je vast alternatieven bedenken voor het geval dat er sprake is van bijv. motorische afasie.

Naast de scoring op kwaliteit, is het ook van belang om te scoren op tempo. Het scoren van kwaliteit en tempo is relevant gezien de relatie die tussen beide kan bestaan. Een taak kan slecht verricht worden door een te hoog tempo, of ondanks een traag tempo; een prestatie kan goed zijn dankzij een traag tempo (of ook ondanks een hoog tempo). Geef onderaan het scoreformulier aan of je de patiënt snel, normaal of traag vindt werken tijdens het onderzoek.

Onder aan het scoreformulier is ruimte voor een conclusie. Formuleer, in voor de patiënt begrijpelijke termen, wat je hebt gezien.

Uitleg van het onderzoek aan de patiënt

De patiënt krijgt, in aansluiting aan de reeds gegeven informatie bij het maken van de afspraak voor het ONO, informatie over het onderzoek.
Denk daarbij aan bijvoorbeeld:
- reden / doel van het onderzoek
- tijdsduur
- wat zal er zo ongeveer aan de orde komen
- wat wordt er van de patiënt verwacht

Bedenk alvast een lopende tekst.

Ziekte-inzicht

1 Algemeen
Stel enkele vragen om een idee te krijgen of de patiënt weet wat er met hem / haar aan de hand is. Weet de patiënt bijvoorbeeld of hij / zij kan lopen, lezen, ed.?
Waarom is de patiënt in het ziekenhuis of verpleeghuis?

2 Oordeel over de eigen prestaties
Het is van belang te weten of de patiënt kan inschatten of iets zal lukken en of de patiënt vervolgens ook kan beoordelen of iets gelukt is. Bedenk een aantal onderdelen binnen dit onderzoek waarbij je vooraf en / of achteraf een eigen oordeel kunt vragen aan de patiënt. Denk er bij deze onderdelen aan dat je niet al zelf zegt of iets goed of fout is gegaan!

Geheugen

3 Oriëntatie
Weet de patiënt wie hij/zij is, welke dag/maand het is, en waar we nu zijn?
Hoe kun je het oplossen als de patiënt afatisch is en de antwoorden niet kan zeggen?

4 Afspraak onthouden
Maak een afspraak met de patiënt: met wie, hoe laat en waar. Vraag de patiënt dit goed te onthouden. Aan het eind van het onderzoek vraag je of de patiënt het nog weet.

5 Persoonsnaam onthouden

MATERIAAL: foto met een afbeelding van een persoon (portret).

De patiënt dient de naam en de foto te onthouden. Aan het eind van het onderzoek vraag je of de patiënt het nog weet.

6 Opbergplaats onthouden

MATERIAAL:	een voorwerp dat voor de patiënt belangrijk is, bijvoorbeeld een portemonnee.

Laat aan de patiënt zien dat je het voorwerp ergens wegstopt, op een zodanige plaats dat de patiënt deze niet meer kan zien (bijvoorbeeld in je tas). Vraag de patiënt of hij/zij wil onthouden wat er is opgeborgen en waar. Aan het eind van het onderzoek vraag je of de patiënt nog weet wat er is opgeborgen en waar het ligt.

7 Leestekst onthouden

MATERIAAL:	krantenbericht

Knip een kort bericht uit de krant of tijdschrift (evt. vergroten m.b.v. kopieerapparaat), noteer voor jezelf de belangrijkste onderdelen (i.v.m. scoring). Laat de patiënt het lezen en vraag in eigen woorden samen te vatten. Het reproduceren na ongeveer één uur wordt bij 7 gescoord, het kunnen samenvallen en in eigen woorden weergeven bij 8.

Communicatie

8 Leestekst

MATERIAAL:	krantenbericht (zie 7)

Zie 7. Hier gaat het nu om het kunnen samenvatten in eigen woorden.

9 Noteren

MATERIAAL:	een bericht van TV of radio (bijvoorbeeld weerbericht of journaal) een laptop, CD-speler of cassettespeler pen en papier

Lees een kort bericht voor (weerbericht, journaalbericht), of speel het af (via laptop, CD- of cassettespeler); de patiënt moet de hoofdzaken van het bericht opschrijven.

10 Rekenen

MATERIAAL: afhankelijk van de opdracht die je verzint

Bedenk een dagelijkse situatie waarbij iets uitgerekend moet worden, bijvoorbeeld afrekenen bij de kassa. Mag de patiënt het op papier uitrekenen? Waarom wel / niet?

Ruimtelijke functies

11 Ruimtelijke handeling

MATERIAAL: afhankelijk van de opdracht die je verzint

Bedenk opdrachten waarbij de patiënt grootte, afstand en positie moet kunnen inschatten (puzzel, groot / klein, ver weg / dichtbij inschatten, positioneren: bijv. glaasje inschenken)

12 Links-rechts oriëntatie

Vraag de patiënt bij zichzelf, bij anderen (bijvoorbeeld de onderzoeker) en bij voorwerpen in de ruimte aan te geven wat links en rechts is. Laat de patiënt op door jou getekende kruispunten met twee auto's aangeven welke auto van rechts komt en dus voorrang heeft. Hoe verwerk je de gokkans in de scoring?

13 Tekenen

MATERIAAL: blanco A4-papier en potlood.

Vraag de patiënt een aantal dingen uit het hoofd te tekenen, bijvoorbeeld een mens, een klok.

14 Natekenen

MATERIAAL: een tekening van bijvoorbeeld een huis
blanco A4-papier en potlood.

Vraag de patiënt het voorbeeld na te tekenen. Bedenk vast wat voorbeelden van wat er allemaal mis kan gaan, en waardoor dit veroorzaakt kan worden.

Waarnemen

15 Visueel-verbaal

MATERIAAL:	Foto's van simpele voorwerpen (hamer, kam, o.i.d., kijk bijvoorbeeld eens in het krantje van de bouwmarkt) en complexe foto's, bijvoorbeeld uit een tijdschrift geknipt of vraag de logopedist eens!

Vraag de patiënt te benoemen wat op de foto's is afgebeeld. Waarom zou je simpele en complexe foto's moeten nemen? Wat weet je bijvoorbeeld als de simpele wel goed gaan, maar de complexe foto's niet? Of andersom?

16 Tactiel-visueel

MATERIAAL:	een aantal kleine voorwerpen (schaar, potlood e.d. foto's waarop deze voorwerpen staan afgebeeld (bijvoorbeeld uit tijdschrift; vraag de logopedist eens!)

Leg de foto's neer voor de patiënt en legt de goede of minst aangedane hand van de patiënt met de handpalm naar boven. De voorwerpen zijn niet zichtbaar voor de patiënt. Laat de patiënt voelen en laat daarna aanwijzen. Houdt indien nodig de hand voor de ogen van de patiënt. Vervolgens hetzelfde voor de meest aangedane hand van de patiënt (wel andere voorwerpen gebruiken).

Handelen

17 Imitatie

Doe een aantal bewegingen voor met gezicht en lichaam, vraag de patiënt dit na te doen. Wat doe je als de patiënt bijvoorbeeld een paretische arm heeft?

18 Gebruik van voorwerpen

MATERIAAL:	een aantal dagelijkse voorwerpen (bijv. kam, sleutel e.d.)

Vraag de patiënt de voorwerpen te benoemen, te vertellen wat je er mee kunt doen, en vervolgens voor te doen wat je ermee kunt doen. Herken je verschillende onderdelen van het handelen in deze opdracht? Wat kan er mis gaan en waardoor?

19 Complexe handeling

MATERIAAL: afhankelijk van de opdracht

Verzin een (dagelijkse) complexe handeling waarbij twee handen moeten worden gebruikt en één of meer voorwerpen (bijvoorbeeld boterham smeren, overhemd aandoen, telefoneren)

Aandacht

20 Advertentie zoeken

MATERIAAL: krantenpagina met advertentierubrieken

Noem een aantal advertentierubrieken op die de patiënt vervolgens op moet zoeken. Wat doe je als de letters te klein zijn, of als de patiënt niet kan lezen? Verzin een oplossing.

21 Halveertaak

MATERIAAL: papier met langwerpige voorwerpen (stokbroden, zuurstokken o.i.d.) van minimaal 10 cm lengte (horizontaal op papier getekend)
pen

Leg het papier midden voor de patiënt neer en vraag of hij/zij de voorwerpen precies doormidden wil delen.

22 Extinctie

Kijk of bij simultane prikkeling één zijde uitgedoofd wordt. Wanneer echter één kant consequent niet gevoeld, gezien of gehoord wordt kun je geen extinctie fenomeen onderzoeken!

Extinctie tactiel
Raak de patiënt op verschillende delen van het lichaam (hoofd, handen, romp) afgewisseld links, rechts of aan beide kanten aan (ogen dicht!). Doe dit verschillende keren.

Extinctie visueel
Ga recht tegenover de patiënt zitten (± 50 cm afstand) en spreid je armen. Vraag de patiënt naar je neus te kijken. Beweeg vervolgens afgewisseld links, rechts of aan beide kanten je vingers. Doe dit verschillende keren.

Extinctie akoestisch

MATERIAAL: twee voorwerpen die geluid maken, bijvoorbeeld samba-ballen

Ga achter de patiënt staan en laat het geluid horen: links, rechts of aan beide kanten. Doe dit verschillende keren.

Organisatie

23 Sorteren

MATERIAAL: dagelijkse voorwerpen die gesorteerd kunnen worden (zie voorbeeld) twee lege bakjes waar de voorwerpen in passen

Bedenk voorwerpen die je kunt sorteren op grootte vorm en kleur. De originele taak wordt gedaan met rondjes en vierkantjes, geel en rood en groot en klein.

2 gele rondjes, 1 groot en 1 klein	2 rode rondjes, 1 groot en 1 klein
2 gele vierkanten, 1 groot en 1 klein	2 rode vierkanten, 1 groot en 1 klein

Verzin voorwerpen die je op de volgende wijze kunt sorteren, maar die wel met het dagelijks leven te maken hebben (bijvoorbeeld schroefjes, knopen)

Zet de twee doosjes leeg recht voor de patiënt neer.

Sorteren op kleur:
De onderzoeker legt in de ene doos:
Het grote gele vierkant en het kleine gele rondje.
En in de andere:
Het grote rode rondje en het kleine rode vierkantje.
De onderzoeker geeft de rondjes en vierkanten stuk voor stuk aan de patiënt en vraagt waar ze bij horen.
Let op: Wanneer de patiënt foutief sorteert, zegt de onderzoeker ‘nee, deze hoort hier’, en legt
het in het juiste bakje zonder aan te geven waarom.
Als alles gesorteerd is vraagt de onderzoeker wat het verschil is tussen wat er in de bakjes ligt.

Sorteren op vorm:
De onderzoeker legt in de ene doos:
Het grote rode rondje en het kleine gele rondje.
En in de andere:
het grote gele vierkant en het kleine rode vierkant.
De onderzoeker geeft de overige rondjes en vierkanten stuk voor stuk aan de patiënt en vraagt waar ze bij horen.
Let op: indien fout: corrigeren als voorheen
Als alles gesorteerd is vraagt de onderzoeker wat nu het verschil is tussen de bakjes.

Sorteren op grootte:
De onderzoeker legt in de ene doos:
Het grote gele rondje en het grote rode vierkant.
En in de andere:
Het kleine rode vierkantje en het kleine gele rondje.
De onderzoeker geeft de overige rondjes en vierkanten stuk voor stuk aan de patiënt en vraagt waar ze bij horen.
Let op: Indien fout, corrigeren als voorheen.
Als alles gesorteerd is vraagt de onderzoeker wat nu het verschil is tussen de bakjes.

24 Planningstaak met onderbreking
Bedenk een taak waarbij de patiënt een handelingsreeks moet plannen, bijvoorbeeld vraag aan de patiënt stap voor stap te beschrijven wat hij/zij doet als hij/zij wakker wordt. Onderbreek de patiënt een paar keer. Kijk of de patiënt van de wijs raakt.

4 Afspraak onthouden
Vraag of de patiënt nog weet met wie, op welke dag, hoe laat en waar de afspraak is die aan het begin is gemaakt.

5 Persoonsnaam onthouden

MATERIAAL:	foto's van personen, waaronder die van aan het begin van het onderzoek

Leg de foto's voor de patiënt neer en vraag of de patiënt nog weet wie aan het begin is aangewezen en hoe hij/zij heet.

6 Opbergplaats onthouden
Vraag aan de patiënt of hij/zij nog weet wat er is opgeborgen en waar.

7 Leestekst onthouden
Wat weet de patiënt nog van het gelezen krantenbericht? Stel eventueel hulpvragen.

EINDE ONDERZOEK

Formulier practicum neuropsychologisch onderzoek
(behorende bij de ITON-cursus)

	Kwaliteit				
Onderdeel	normaal	enigszins afwijkend	afwijkend	niet onderzocht	**Opmerkingen**
Ziekte-inzicht					
1 Algemeen					
2 Oordeel over eigen prestaties					
Geheugen					
3 Oriëntatie					
4 Afspraak onthouden					
5 Persoonsnaam onthouden					
6 Opbergplaats onthouden					
7 Leestekst onthouden					
Communicatie					
8 Leestekst samenvatten					
9 Noteren bericht					
10 Rekenen					
Ruimtelijke functies					
11 Ruimtelijke handeling					
12 Links-rechts oriëntatie					
13 Tekenen					
14 Natekenen					
Waarnemen					
15 Visueel – verbaal					
16 Tactiel – visueel					
Handelen					
17 Imitatie					
18 Gebruik van voorwerpen					
19 Complexe handeling					
Aandacht					
20 Advertenties zoeken					
21 Hallveertaak					
22 Extinctie tactiel/visueel/akoestisch					
Organisatie					
23 Sorteren					
24 Planningstaak met onderbreking					

Video's: vragen ter verdieping

Het gedachte lichaam
De hersenen bevatten een mentale representatie van het lichaam ("lichaamsschema"). Leg uit wat er verandert bij:
- een onderarm-amputatie
- een plexus brachialis pares
- een spastische hemiparese van een arm.

Afasie
1. Bij welk type afasie heeft de patient vaak geen inzicht in de eigen stoornis? Wat zijn de consequenties hiervan?
2. Geef een voorbeeld waaruit blijkt dat er bij afasie geen "spraakstoornis" maar een "taalstoornis" is.

Broken Images
1. Welke functionele hersenschors is bij John beschadigd, de primaire, secundaire of tertiaire schors?
2. Op welke twee manieren compenseert John zijn gezichtsherkenningsstoomis (prosopagnosie)?
3. Hoe wordt in de film getoond dat Lany onbewust kan waarnemen? Hoe komt het dat dit bij John niet gebeurt?
4. Wat zegt Larry over de ,,aantrekkelijkheid van vrouwen"? Wat is je conclusie?

Stranger in the family
1. Neal heeft woede-aanvallen. Probeer hiervoor een verklaring te formuleren (gedragsanalyse), gebruikmakend van de modellen uit hoofdstuk 12 (4 determinanten en gedragscirkel).
2. Geef een suggestie voor een gedragstherapeutische aanpak.
3. Geef aan in hoeverre het ICF-model en het somato-psycho-socio-model bruikbaar zijn om de gezinsproblemen die hier getoond worden te benaderen.

Prisoner of consiousness
1. Geef aan welke vormen resp. aspecten van geheugen bij Clive nog goed werken. Ga hierbij na: declaratief, episodisch, semantisch, procedureel; ultrakort, kort en langetermijngeheugen; inprenting, opslag, oproepen, en geef dan steeds de scene aan waar dat uit blijkt.
2. Kan je verklaren dat Clive steeds maar benadrukt dat hij "net wakker is" en "niets voelde, zag en hoorde"?
3. Wat is je conclusie met betrekking tot de rol van de hippocampus bij geheugen?

Ik zie niet waar ik voel
1. Mevrouw Steensma gebruikt haar rechter arm weinig of niet. In hoeverre is dit logisch resp. pathologisch?
2. Hoe zou je kunnen vaststellen of haar responsen "borstel" (bij plaatje van een veer) en "kanarievogel" (bij plaatje van een eekhoorn) berusten op een herkenningsstoornis (agnosie) of op een benoemingsstoornis (anomie of amnestische afasie)?

Moeite met aankleden
1. Noem - in het algemeen - 10 oorzaken van problemen met aankleden.
2. Noem mogelijke oorzaken waarom het aankleden bij de heer Van 0. niet lukt, en geef aan hoe je dit kunt verifiëren.
3. Wat zou je nog meer over deze man willen weten? Welke vragen zou je hem nog willen stellen?
4. Bespreek een mogelijke aanpak van een behandeling. Denk hierbij aan het ICF-model.

Notities