

Spontaneous recovery from acalculia

ANNA BASSO,¹ ALESSANDRA CAPORALI,¹ AND PIETRO FAGLIONI²

¹Department of Neurological Sciences (Neurology Unit), Milan University, Milan, Italy

²Neurological Clinic, Modena University, Modena, Italy

(RECEIVED January 21, 2004; REVISED October 18, 2004; ACCEPTED October 20, 2004)

Abstract

A topic much considered in research on acalculia was its relationship with aphasia. Far less attention has been given to the natural course of acalculia. In this retrospective study, we examined the relationship between aphasia and acalculia in an unselected series of 98 left-brain-damaged patients and the spontaneous recovery from acalculia in 92 acalculic patients with follow-up. There was a significant association between aphasia and acalculia although 19 participants exhibited aphasia with no acalculia and six acalculia with no aphasia. We observed significant improvement between a first examination carried out between 1 and 5 months post-onset and a second examination carried out between 3 and 11 months later (mean: 5 months). The mechanisms of spontaneous recovery are discussed. (*JINS*, 2005, *11*, 99–107.)

Keywords: Acalculia, Aphasia, Frequency of co-occurrence, Spontaneous recovery from acalculia

INTRODUCTION

In our daily lives, the ability to perform simple calculations is a prerequisite for carrying out a hoard of such everyday activities as using money or calculating time. Cerebral damage, particularly in the left hemisphere, can result in an impaired ability to calculate. Acaculcia, the impairment of processing numbers and calculating following brain damage, is a complex neuropsychological syndrome that has attracted much attention since it was first described by Henschen in 1919 (Henschen, 1919).

The early contributions, despite acknowledging the existence of isolated calculation disorders following brain damage, argued that calculation functions were seldom autonomous and impairments were generally caused by damage to other cognitive functions, mainly spatial disorders and language deficits. The role of spatial abilities in number processing and calculation, however, was intuitively defined and never made explicit. Instead of starting from clear predictions of what a spatial disorder would entail, when errors were observed that could apparently be classified as spatial, a spatial disorder was inferred. Incorrect

alignment of intermediate products, for instance, was generally classified as a spatial error but in many cases the underlying cause of the error is unclear. Shifting, for instance, can result from spatial disorders or even a breakdown of knowledge of the significance of the decimal system.

Several clinical studies have focused on the relationship between language and calculation disorders. Rosselli and Ardila (1989) studied 41 left-brain-damaged and 21 right-brain-damaged participants who were subdivided according to type of aphasia and pre- or post-rolandic site of lesion. All groups were found to perform less well than a group of normal controls matched for age, sex, and educational level, with the left post-rolandic group faring worst of all. Each group, however, presented some specific peculiarities. Accordingly, the authors argued that different cognitive impairments could explain the performance of the different groups; spatial disorders, for instance, could explain the performance of Wernicke aphasic participants and of participants with right retro-rolandic lesions. They concluded that calculation skills require the integration of multiple cognitive processes and can be disrupted following damage to any of the necessary cognitive function.

More recently, Delazer et al. (1999) studied the relationship between language impairment and numerical skill deficits in 15 control participants and 50 left-hemisphere-damaged participants subdivided according to the presence

Reprint requests to: Anna Basso, Department of Neurological Sciences (Neurology Unit), Milan University, Via F. Sforza 35, 20122 Milan, Italy. E-mail: abasso@fastwebnet.it

and type of aphasia. In a written calculation task only global and Wernicke aphasic participants significantly differed from controls but the number of mathematical problems was very low—two addition, two subtraction, and two multiplication problems—and insufficient to identify also mildly acalculic participants. In reading Arabic numerals and number words all groups performed worse than control participants, with the exception of anomic participants. The number of errors was related to the severity of the aphasia but the types of errors in Broca and Wernicke aphasic participants differed. Broca aphasic participants made frequent syntactic errors in reading Arabic numerals, reading for example 406 as “forty-six”, and Wernicke aphasics made more lexical errors in reading Arabic numerals and number words, reading for example 24 as “thirty-four”. The authors argued that these errors partially reflect the nature of the specific language problem—syntactic in Broca aphasia and lexical in Wernicke aphasia—and suggested an association between impairment in language and number processing.

One aspect of acalculia that has received scant attention is its evolution. The process of recovery from disruption of cognitive functions due to an acute pathological involvement of the central nervous system is variable in the first months after injury. Systematic attempts to understand the recovery process have mainly involved participants with aphasia (Basso, 1992; Cappa, 1998; Laska et al., 2001; Pedersen et al., 2004).

Recovery of other cognitive functions has received sporadic attention. Kertesz et al. (1984) first sought the presence of ideomotor apraxia in a group of 118 left-hemisphere stroke participants within the first month of illness and then at 3 or more months post-onset. In the acute period, 54.6% of the participants were found to be apraxic contrasting with only 39.9% in the chronic group (note that the chronic group was made up of the 118 acute participants and 25 new participants so that the number of participants who were apraxic at first examination but not at second cannot be computed with precision). Foundas et al. (1993) evaluated a group of left-hemisphere stroke participants at 6 weeks, and 3 and 6 months post-onset. They found that participants improved over time and that their improvement was greater in the first 3 months. In a more recent study on long-term follow-up of ideomotor apraxia, Basso et al. (2000) found that participants' ability to imitate meaningful and meaningless gestures improved in all but one of 44 participants between a first and a second evaluation carried out between 5 and 27 months post-onset (mean post-onset time 11 months). However, at a third evaluation many months later very few participants showed further recovery and six participants had worsened.

Data on recovery from acalculia are even scantier. Caporali et al. (2000) reported recovery from acalculia in 51 vascular acalculic participants. Recovery was significant in the subgroup of acute subjects first evaluated less than 3 months post-onset, but it did not reach significance in participants first seen between 3 and 5 months post-onset. The difference in recovery in the two groups was significant.

The aims of the present retrospective investigation were to assess the frequency of co-occurrence of language and calculation disorders and to study the natural course of acalculia in left-brain-damaged acalculic participants.

METHODS

Research Participants

This research is part of a large ongoing study of recovery from aphasia and associated disorders. Since 1984, all participants seen at the Aphasia Unit of Milan University have been offered a follow-up evaluation either in the clinic or at their home whenever they met the following criteria: (1) aphasia had been present at first evaluation, (2) there was a left-hemisphere vascular lesion with no evidence of right-hemisphere damage; (3) right-handedness according to the Edinburgh Inventory (minimum score: 9/12; Oldfield, 1971), (4) age at onset between 20 and 75 years old, and (5) at least 5 years of formal education.

Of the 1896 participants seen at the Aphasia unit between 1984 and 2000, 414 were eligible for recruitment to the follow-up investigation; 169 participants declined and 245 gave their consent. To be included in the present research, participants had to (1) have undergone a first complete neuropsychological evaluation between 30 and 150 days post-onset, (2) be acalculic with a score equal or below 60 (see below), and (3) have undergone a follow-up acalculia evaluation at least 3 months after the first. Ninety-two participants entered the study. Twenty-four were female and 68 were male; mean age was 58.7 years ($SD = 12.7$; range 20–75 years), mean educational level was 10.3 years ($SD = 4.5$; range 5–17 years), and mean length of illness at first examination was 64 days ($SD = 42$ days; range 30–150 days). Improvement in the acalculia test was measured in this group of participants. As stated, however, the group did not form a random sample of left-brain-damaged participants because only aphasic participants were offered a follow-up evaluation and only acalculic participants were included in the study. We corrected the sampling error by applying Naranjo and McKean's (2001) method: the mean and the standard deviation on the acalculia test were calculated on a larger sample including the 92 acalculic participants and 98 participants who conformed to the same criteria—except for the presence of aphasia—but did not enter the recalling list, mainly for logistic reasons. There were 65 males and 33 females, mean age was 60.6 ($SD = 11.7$, range 24–75 years), mean educational level was 11.4 ($SD = 4.6$, range 5–17 years), and mean length of illness was 57 days ($SD = 34$, range 30–150 days). The frequency of co-occurrence of aphasia and acalculia was studied in this second group of 98 participants.

There were no significant differences between the two groups in age ($t = 1.029$, $df = 188$, ns), educational level ($t = 1.610$, $df = 188$, ns), or length of illness ($t = -1.235$, $df = 188$, ns).

Testing

Acalculia

The presence of acalculia was assessed using an acalculia test consisting of a pretest and four calculation tasks (Basso & Capitani, 1979). The pretest verifies whether or not participants are able to understand the value of numbers, which is a prerequisite for the correct execution of any calculation. It consists of 12 rows of three written numbers from each of which participants have to point to the highest one. Only participants who commit no more than two errors in the pretest continue the test. The four calculation tasks consist of written calculations of increasing difficulty: seven addition, seven subtraction, seven multiplication, and six division problems. Each task is preceded by an example. In each task a score of one is given to each correct digit. In multiplications that include partial results, digits of the final results are not taken into consideration because they result from addition of partial results and not from multiplication. In the division tasks, digits of the final results and of partial remainders are computed. The maximum possible scores are 20 for addition, 18 for subtraction, 34 for multiplication, and 29 for division problems. The total score is obtained by adding up the four partial scores and correcting the raw result for the effect of age and educational level (see below).

The test was also administered to 302 normal controls, 178 females, and 124 males, with at least 5 years of formal education and aged between 15 and 70 years (Basso & Capitani, 1979). The statistical work-up revealed that age and educational level had a significant effect. A correction table was drawn up in order to convert the total raw score into a corrected score and to set a cut-off score, which was found to be 74/101. Participants scoring below 74 (corrected score) are considered acalculic. Appendix 1 reports the operations included in the test and the correction table.

For the present research, we included only patients with a score of 60 or less to guarantee a reasonable amount of possible amelioration.

Token test

A shortened version of the token test was administered with 36 items and a cut-off score, adjusted for the influ-

ence of age and education, of 29/36 (De Renzi & Faglioni, 1978).

Language evaluation

All participants were given a Standard Language Examination currently in use in our aphasia unit (Basso et al., 1979) that includes, among other tasks, oral and written naming of 20 common pictures of objects; oral and written comprehension of 20 unrelated, 20 semantically related words, and 10 sentences; and repetition, reading aloud and writing to dictation of words, legal nonwords, and sentences. Tasks of spontaneous oral and written production are also included.

Participants were classified as non-aphasic if their scores in the token test and all the language tasks were in the normal range and there were no signs of aphasia in their spontaneous production. Participants were classified as borderline if their scores were in the normal range but there were some typical aphasic errors in their spontaneous speech.

Characteristics and test results of the group of 92 participants who underwent two examinations are reported in Table 1 and characteristics and test results of the 98 participants who were evaluated just once are reported in Table 2.

RESULTS

Aphasia and Acalculia

To investigate the relationship between aphasia and acalculia, only the 98 participants with a single evaluation have been considered because the 92 participants with follow-up were all aphasic and acalculic and did not form a random sample of left-brain-damaged participants. We also excluded 15 cases classified as borderline at the aphasia evaluation. No patient was classified as borderline in the acalculia test because the cut-off score reliably differentiated participants with calculation ability in the normal range from participants with a calculation score that fell below the normal range. The frequencies of participants with both aphasia and acalculia, one or neither of the two impairments are

Table 1. Characteristics and test results at first and second evaluation of the 92 acalculic participants

	1st Acalculia Test									2nd Acalculia Test						DIFF	
	Age (years)	Education (years)	Days post-onset	1st TT (0–36)	Total score (0–101)	A (0–20)	S (0–18)	M (0–34)	D (0–29)	1st–2nd Exam (days)	2nd TT (0–36)	Total score (0–101)	A (0–20)	S (0–18)	M (0–34)		D (0–29)
<i>Mean</i>	58.7	10.3	64	16.0	34.3	13.0	9.2	6.1	3.2	156	21.0	59.1	16.9	12.8	17.0	9.6	24.8
<i>SD</i>	12.7	4.5	42	7.8	15.2	5.8	5.2	5.4	2.4	64	7.7	23.9	4.5	4.8	11.6	9.0	18.4
<i>Range</i>	20–75	5–17	30–150	0–34	3–60	0–20	0–18	0–24	0–9	90–330	3–35	10–101	1–20	1–18	0–34	0–29	–12–66

A = addition, S = subtraction, M = multiplication, D = division; DIFF = difference in total score between 1st and 2nd evaluation.

Table 2. Characteristics and test results of the 98 participants evaluated once

	Age (years)	Education (years)	O-e (days) (30–150)	Token Test (0–36)	Acalculia Test				
					Total score (0–101)	A (0–20)	S (0–18)	M (0–34)	D (0–29)
<i>Mean</i>	60.6	11.4	57	23.8	65.0	16.4	13.0	20.6	13.3
<i>SD</i>	11.7	4.6	34	7.9	27.9	5.5	5.3	12.0	10.7
<i>Range</i>	24–75	5–17	30–150	2–34	5–101	0–20	0–18	0–34	0–29

O-e = onset-exam; A = addition; S = subtraction; M = multiplication; D = division.

reported in Table 3. Frequencies have been compared by means of Cohen's (1960) kappa. The analysis indicates that there is a significant tendency either to have both aphasia and acalculia or neither. In other words, the number of participants with either aphasia or acalculia alone was not significantly higher than would be expected by chance.

However, six acalculic participants were not aphasic, and in 19 aphasic participants there was no evidence of acalculia as evaluated by our test of written calculation. Below is a brief description of an acalculic non-aphasic individual and of a severely aphasic participants without acalculia.

SK was a 48-year-old University professor of mathematics who suffered a left CVA (cerebral vascular accident) in June 1993. Neurological examination disclosed a mild right hemiparesis and mild language disorders. A computed tomography (CT) scan performed on the same day was normal; a few days later an MRI (Magnetic Resonance Imaging) disclosed areas of altered signal in the left internal capsule, the frontal-temporal region and in the subcortical structures. His motor and language disorders recovered rapidly. SK was first seen at the Aphasia Unit 50 days after onset.

No apraxia was present; his non-verbal reasoning was in the normal range (29/36 on the Raven's Coloured Progressive Matrices; Raven, 1965). In a thorough language examination (Miceli et al., 1994), he only made some rare misspellings in writing and a few errors in a sentence comprehension task. His score on the token test was 29. His naming to confrontation was good, as shown by his score on the Boston Naming Test (56/60; Kaplan et al., 1983) and his spontaneous speech was apparently perfect,

with a good choice of lexical items and grammatical structures.

He had a marked deficit in the acalculia test (27/101) and the decision was made to further investigate his number and calculation processing disorders using a more detailed and model-driven test (Deloche et al., 1993). He performed normally in counting, dot enumeration, transcoding from Arabic to orthographic or phonological numbers, and *vice versa*, naming and writing arithmetical signs, number comparisons, simple mental calculation, placing numbers on an analog number line, perceptual quantity estimation, and numerical knowledge (e.g., how many days are there in a week?). He was however impaired in written calculation, even with rather simple addition ($55 + 89$), subtraction ($72 - 35$), multiplication (142×5), and division ($456 \div 6$) problems that could easily be solved mentally.

In July 1994, a year later, he was reexamined in another hospital using a very similar test (Miceli & Capasso, 1991). He is reported to have performed well in tasks evaluating the number system except for two errors in transcoding. Arithmetical facts were well preserved but he made frequent errors in written calculation tasks that require the activation of calculation procedures. In other words, no qualitative difference was apparent although it cannot be excluded that he had partially recovered.

To sum up, SK, a university professor of mathematics, had an isolated calculation disorder with very mild and transient aphasia. His calculation disorder was selective for calculation procedures and was still evident a year post-onset.

GG was a 52-year-old employee with 13 years of formal education who became aphasic following the rupture of a temporal-parietal haematoma in May 1993. A CT scan in August showed a left parietal lesion. Neurological examination revealed right hemianopia and aphasia. He underwent a language examination 4 months post-onset, in September 1993.

His speech was fluent but scarcely informative because of frequent semantic paraphasias and no responses. He could repeat, read, and write most of two- and three-syllable non-words and words. Oral and written confrontation naming were severely impaired for nouns and verbs; errors were semantic paraphasias and no responses. He was, however, sometimes able to produce a short description of the to-be-

Table 3. Frequency distribution of participants with both aphasia and acalculia, one, or neither of the two impairments

		Acalculia		Total
		+	-	
Aphasia	+	36	19	55
	-	6	22	28
	Total	42	41	83

Cohen's kappa = 0.395; $p \leq .0001$.

named picture. Comprehension of single words was mildly impaired. He scored 9/36 on the token test. He had no oral, ideomotor, or ideational apraxia and his score on Raven Coloured Progressive Matrices was 32/36 (Raven, 1965). On the acalculia test he scored 20/20 in addition, 17/18 in subtraction, 29/34 in multiplication, and 27/29 in division problems, performing all calculations quickly and with assurance. All partial results were well within the normal range; the total raw score was 93 and the corrected total score was 92.5 (cut-off score: 74).

In conclusion, GG had a severe fluent Wernicke-type aphasia with a significant degree of impairment of the lexicon; praxis, written calculation, and reasoning were preserved.

Recovery

Statisticians have long debated how to evaluate change between two successive measures when the sample is chosen on the basis of their score at first evaluation (Bonate, 2000; Campbell & Kenny, 1999), and there still is no agreed upon solution to the problem. One much pondered question concerns the possible effect of the initial level of impairment on recovery. It is still unclear whether severely damaged patients have the same chances of recovery as less damaged patients and, if not, whether severity exerts a positive or a negative effect.

In an attempt to circumvent these problems, Naranjo and McKean (2001) came up with a new and seemingly convincing proposal. The difference between the first and the second evaluation is broken down into three parts. The first part evaluates the regression toward the mean (Galton, 1885), the second corresponds to the real difference between the first and the second evaluation, and the third part measures the effect of the initial level of performance. True recovery is estimated by the second and third part; the value of the regression toward the mean must be calculated and subtracted from the apparent improvement.

We measured improvement in the acalculia test in the sample of 92 left-brain-damaged aphasic participants with follow-up. The group, however, was not representative of the left-brain-damaged population because it included only acalculic participants with a score of 60 or less. We overcame the sampling error by applying Naranjo and McKean's (2001) method. The mean (50.1) and the standard deviation (27.3) on the acalculia test were calculated on the first examination of the 190 participants: 92 acalculic participants and 98 participants representing a continuous series of left-brain-damaged individuals with the same general characteristics (except for the requirement of presence of aphasia) evaluated in the same years as the experimental sample.

The difference between the means of the first acalculia score and the second acalculia score was measured in the 92 acalculic participants and was broken down into the three subcomponents—regression toward the mean, amount of recovery independent of initial score, amount of recovery

proportional to the initial severity—as suggested by Naranjo and McKean (2001). Appendix 2 reports the statistical work-up.

Results were the following:

$$T2-T1 = 59.12 - 34.34 = 24.78 = 4.06 + 24.82 - 4.10$$

24.78 is the apparent change that must be broken down into (1) the value of the regression toward the mean (4.06), (2) the amount of recovery shown in the acalculia test independent of the initial severity (24.82; $p < .0000001$), and (3) the value depending on the initial score (-4.10 ; $p < .01$).

Interestingly, the effect of initial severity is significant but negative, indicating that more severely damaged participants have less chance of recovery than less severely damaged patients. This negative effect is almost perfectly counterbalanced by the effect of the regression toward the mean.

DISCUSSION

The aims of the investigation were to analyze the frequency of the presence of either aphasia or acalculia alone and to study spontaneous recovery from acalculia. Our data indicate that, notwithstanding their functional autonomy, co-occurrence of language and calculation disorders is not fortuitous, which, however, does not mean that they are theoretically related. Functional independence implies neural separability but the actual separability of two cognitive functions or two components of the same function (e.g., number processing and knowledge of arithmetic facts) depends on how easily their neural substrates can be independently damaged. In vascular participants, the frequent co-occurrence of language and calculation disorders may have an anatomical basis as is the case for the clinical syndromes of aphasia—Broca, Wernicke, transcortical, and so on—that have formed the basis of classical teaching for many decades and that are no longer thought to be “real” because a defining and necessary symptom has never been found. They are “to a large extent, artifacts produced by the vascularisation of the language area” (Poeck, 1983, p. 64).

A number of participants in our series, however, showed either aphasia ($n = 19$) or acalculia ($n = 6$) alone and altogether represented a quarter of the population studied. Interestingly, contrary to what may appear from the literature where many case reports of non-aphasic participants with acalculia (Basso & Beschin, 2000; Cipolotti et al., 1991; Corbett et al., 1986; Lampl et al., 1994; Lucchelli & De Renzi, 1993; Martory et al., 2003; Takayama et al., 1994; Warrington, 1982) can be found while only one participant with aphasia without acalculia is reported (Rossor et al., 1995; see also Diesfeldt, 1993 and Thioux et al., 1998 for a less clear-cut dissociation), we found many more non-acalculic aphasic participants than participants with the reverse pattern.

To conclude, dissociations between language and calculation disorders—as evaluated by the written calculation task and the language tests used in the study—may not be the rule but are not rare; in our sample a quarter of the population showed either one or the other impairment.

The second aim of the research was to study the spontaneous evolution of the calculation disorder. So-called *spontaneous* recovery has been demonstrated in left-brain-damaged participants with aphasia and, less frequently, in participants with ideomotor apraxia (Basso et al., 2000; Foundas et al., 1993; Kertesz et al., 1984), and in right-brain-damaged participants *spontaneous* recovery from neglect is relatively common (e.g., Stone et al., 1992). It is however not at all clear what *spontaneous* means.

According to Hebb (1949), networks of cells constitute functional units that underlie cognitive functions, and learning—or re-learning in the case of recovery from cognitive damage—occurs because neurons that fire together increase their synaptic strength. Neurons that have been disconnected by a lesion may reconnect if they are simultaneously activated. Individuals do not live in a vacuum and simultaneous activation of previously disconnected neurons can happen by chance in everyday life.

Individuals with aphasia, for example, are variably motivated to listen to what other people say to them and to express themselves and this generic but constant stimulation can be the cause of the improvement many aphasic participants undergo without being rehabilitated. Visual neglect may recover because of the constant presence of stimuli in the contralateral space that can occasionally be processed by neglect participants, maybe by casually turning their head and eyes toward the neglected space. It is more difficult to explain recovery from apraxia by fortuitous stimulation because apraxia is characterized by marked dissociation between purposeful movements (generally preserved) and impaired movements when carried out in artificial situations. It is not clear how the carrying out of movements in ecological situations can ameliorate their performance in a testing situation.

In everyday life people use as much language as they can, are surrounded by stimuli in the neglected space, and use gestures—at least meaningful gestures—in ecological situations. Most people, however, at least Italians with very few exceptions, do not calculate mentally if they have any alternative such as using a calculator or waiting for somebody else to do it for them. Notwithstanding apparent lack of stimulation, partial recovery from acalculia occurs in most participants, and a question that remains open is how simultaneous activation of previously disconnected neurons occurs if they are not simultaneously stimulated, be it by chance or intentionally. However, rehabilitation affects recovery—we argue—through intensity of stimulation and feedback. When the effect of intense and prolonged aphasia therapy was compared to more traditional regimens, participants who were more intensively rehabilitated for a longer time were found to recover more (e.g., Basso & Caporali, 2001; Denes et al., 1996). Feedback, which is

generally offered in aphasia therapy, is another mechanism that can affect recovery. McCandliss et al. (2002), for instance, studied /l/ and /r/ discrimination in Japanese adults. They contrasted errorless and errorful learning and presence/absence of feedback. Errorless learning was more effective than errorful learning and feedback was a critical factor.

Another important question about reorganization of the nervous system in aphasia, apraxia, and acalculia regards whether functional changes are sustained by the left hemisphere zones spared by the lesion or by recruitment of homologous right hemisphere (RH) regions.

Indirect evidence that supports transfer of language dominance comes from studies using tachistoscopic or dichotic presentation of linguistic stimuli (Moore & Weidner, 1974, 1975). A contribution of the RH to recovery of language in aphasic participants has also been suggested by results of studies using an evoked potential paradigm (Papanicolaou et al., 1984, 1987).

However, take-over of language functions by the RH differs remarkably from one participant to another and can hardly be considered the rule. With rare exceptions (e.g., Cummings et al., 1979), in fact, global aphasic individuals, who presumably have large lesions destroying all the classic language areas, do not improve substantially. That compensation by the RH is rare is also suggested by Rasmussen and Milner (1977). They found that only 12% of adult participants with *early* left hemisphere (LH) damage had RH speech representation, as determined by carotid barbiturate injection. It is conceivable that the percentage would be even less in participants who have incurred LH lesions at a more advanced age.

Recent neuroimaging investigations have reported complex and sometimes contradicting patterns of activation in recovered aphasic individuals. A right-sided functional shift in two participants with aphasia studied longitudinally has been reported by Thulborn et al. (1999) who documented a shift to the right Wernicke's area after the stroke. In contrast, Warburton et al. (1999), in a positron emission tomography (PET) study of verbal fluency, argue that perilesional areas of the LH have a crucial role in recovery. Heiss et al. (1999) in another PET study on recovery of repetition concluded that good recovery is associated with reactivation of left temporal areas. Finally, Belin et al. (1996) reported extensive right-sided activation during single-word repetition in a group of chronic participants; after successful Melodic Intonation Therapy (MIT) training, however, a significant increase of activation in the left frontal areas when repeating words with MIT intonation was noted.

The question of whether the LH undamaged areas are more or less efficient for the recovery of language function (as well as other cognitive functions) than homologous structures of the RH remains unanswered. The answer is probably not a yes or no. Many other factors, such as size and site of lesion, may affect the response (Vitali et al., 2003).

To conclude, we suggest that spontaneous recovery does not heavily depend on stimulation, intentional or otherwise, but that it can be expanded by intensive stimulation and feedback. Furthermore, either left or right hemisphere areas, probably depending on site and size of lesions, can sustain recovery.

REFERENCES

- Basso, A. (1992). Prognostic factors in aphasia. *Aphasiology*, *6*, 337–348.
- Basso, A. & Beschin, N. (2000). Number transcoding and number word spelling in a left-brain-damaged non-aphasic patient. *Neurocase*, *6*, 129–139.
- Basso, A., Burgio, F., Paulin, M., & Prandoni, P. (2000). Long-term follow-up of ideomotor apraxia. *Neuropsychological Rehabilitation*, *10*, 1–13.
- Basso, A. & Capitani, E. (1979). Un test standardizzato per la diagnosi di acalculia. Descrizione e valori normativi. *AP Rivista di Applicazioni Psicologiche*, *1*, 551–564.
- Basso, A., Capitani, E., & Vignolo, L.A. (1979). Influence of rehabilitation on language skills in aphasic patients: A controlled study. *Archives of Neurology*, *36*, 190–196.
- Basso, A. & Caporali, A. (2001). Aphasia therapy or the importance of being earnest. *Aphasiology*, *15*, 307–332.
- Belin, P., Van Eeckhout, P., Zilbovicious, M., Remy, P., François, C., Guillaume, S., Chain, F., Rancurel, G., & Samson, Y. (1996). Recovery from nonfluent aphasia after melodic intonation therapy: A PET study. *Neurology*, *47*, 1504–1511.
- Bonate, P.L. (2000). *Analysis of pretest–posttest designs*. London, England: Chapman & Hall.
- Campbell, D.T. & Kenny, D.A. (1999). *A primer on regression artifacts*. New York: The Guilford Press.
- Caporali, A., Burgio, F., & Basso, A. (2000). The natural course of acalculia in left-brain-damaged patients. *Neurological Sciences*, *21*, 143–149.
- Cappa, S. (1998). Spontaneous recovery from aphasia. In B. Stemmer & H.A. Whitaker (Eds.), *Handbook of neurolinguistics* (pp. 535–545). San Diego, California: Academic Press.
- Cipolotti, L., Butterworth, B., & Denes, G. (1991). A specific deficit for numbers in a case of dense acalculia. *Brain*, *114*, 2619–2637.
- Cohen, J. (1960). A coefficient of agreement for nominal scales. *Educational and Psychological Measurement*, *20*, 37–46.
- Corbett, A.J., McCusker, E.A., & Davidson, O.R. (1986). Acalculia following a dominant-hemisphere subcortical infarct. *Archives of Neurology*, *43*, 964–966.
- Cummings, J.L., Benson, D.F., Walsh, M.J., & Levine, H. (1979). Left to right transfer of language dominance: A case study. *Neurology*, *29*, 1547–1550.
- Delazer, M., Girelli, L., Semenza, C., & Denes, G. (1999). Numerical skills and aphasia. *Journal of the International Neuropsychological Society*, *5*, 213–221.
- Deloche, G., Seron, X., Baeta, E., Basso, A., Claros Salinas, D., Gaillard, F., Goldenberg, G., Stachowiak, F.J., Temple, C., Tzavaras, A., & Vendrell, J. (1993). Calculation and number processing: The EC301 assessment battery for brain-damaged adults. In F.J. Stachowiak (Ed.), *Developments in the assessment and rehabilitation of brain-damaged patients* (pp. 401–406). Tübingen, Germany: Guntter Narr Verlag.
- Denes, G., Perazzolo, C., Piani, A., & Piccione, F. (1996). Intensive versus regular speech therapy in global aphasia: A controlled study. *Aphasiology*, *10*, 385–394.
- De Renzi, E. & Faglioni, P. (1978). Normative data and screening power of a shortened version of the token test. *Cortex*, *14*, 41–48.
- Diesfeldt, H.F.A. (1993). Progressive decline of semantic memory with preservation of number processing and calculation. *Behavioural Neurology*, *6*, 239–242.
- Foundas, A.L., Raymer, A., Maher, L.M., Gonzales-Rothi, L.J., & Heilman, K.M. (1993). Recovery in ideomotor apraxia. *Journal of Clinical and Experimental Neuropsychology*, *15*, 44.
- Galton, F. (1885). Regression towards mediocrity in hereditary stature. *Journal of the Anthropological Institute of Great Britain and Ireland*, *15*, 246–263.
- Hebb, D.O. (1949). *The organization of behavior. A neuropsychological theory*. New York: Wiley.
- Heiss, W.D., Kessler, J., Thiel, A., Ghaemi, M., & Karbe, H. (1999). Differential capacity of left and right hemispheric areas for compensation of poststroke aphasia. *Annals of Neurology*, *45*, 430–438.
- Henschen, S.E. (1919). Ueber Sprach- Musik- und Rechenmechanismen und ihre Lokalisationen im Grosshirn. *Zeitschrift für die gesamte Neurologie und Psychiatrie*, *52*, 273–298.
- Kaplan, E., Goodglass, A., & Weintraub, S. (1983). *Boston naming test*. Philadelphia, Pennsylvania: Lea & Febiger.
- Kertesz, A., Ferro, J.M., & Shewan, C.M. (1984). Apraxia and aphasia. The functional-anatomical basis for their dissociation. *Neurology*, *30*, 40–47.
- Lampl, Y., Eshel, Y., Gilad, R., & Sarova-Pinhas, I. (1994). Selective acalculia with sparing of the subtraction process in a patient with left parieto-temporal haemorrhage. *Neurology*, *44*, 1759–1761.
- Laska, A.C., Hellblom, A., Murray, V., Kahan, T., & Von Arbin, M. (2001). Aphasia in acute stroke and relation to outcome. *Journal of Internal Medicine*, *249*, 423–422.
- Lucchelli, F. & De Renzi, E. (1993). Primary dyscalculia after a medial frontal lesion of the left hemisphere. *Journal of Neurology, Neurosurgery, and Psychiatry*, *56*, 304–307.
- Martory, M.D., Mayer, E., Pegna, A.J., Annoni, J.M., Landis, T., & Katheb, A. (2003). Pure global acalculia following a left subangular lesion. *Neurocase*, *9*, 319–328.
- McCandliss, B., Fiez, G., Protopoulos, A., Conway, M., & McClelland, J. (2002). Success and failure in teaching the /r/-/l/ contrast to Japanese adults: Tests of a Hebbian model of plasticity and stabilization in spoken language perception. *Cognitive, Affective, & Behavioural Neuroscience*, *2*, 89–108.
- Miceli, G. & Capasso, R. (1991). *I disturbi del calcolo*. Milan: Masson.
- Miceli, G., Laudanna, A., Burani, C., & Capasso, R. (1994). *Batteria per l'Analisi dei Deficit Afasici*. Rome: CEPSAG.
- Moore, H.W. & Weidner, W. (1974). Bilateral tachistoscopic word perception in aphasic and normal subjects. *Perceptual and Motor Skills*, *39*, 1001–1011.
- Moore, H.W. & Weidner, W. (1975). Dichotic word-perception of aphasic and normal subjects. *Perceptual and Motor Skills*, *40*, 379–386.
- Naranjo, J.D. & McKean, J.W. (2001). Adjusting for regression effect in uncontrolled studies. *Biometrics*, *57*, 178–181.
- Oldfield, R.C. (1971). The assessment and analysis of handedness: The Edinburgh inventory. *Neuropsychologia*, *9*, 97–113.

- Papanicolaou, A.C., Levin, H.S., & Eisenberg, H.M. (1984). Evoked potentials correlates of right hemisphere involvement in language recovery in adults. *Neurosurgery*, *14*, 412–415.
- Papanicolaou, A.C., Moore, B.D., Levin, H.S., & Eisenberg, H.M. (1987). Evoked potentials correlates of right hemisphere involvement in language recovery following stroke. *Archives of Neurology*, *44*, 521–524.
- Pedersen, P.M., Vinter, K., & Olsen, T.S. (2004). Aphasia after stroke: Type, severity and prognosis. *Cerebrovascular Diseases*, *17*, 35–43.
- Poeck, K. (1983). What do we mean by “Aphasic Syndromes?” A neurologist’s view. *Brain and Language*, *20*, 79–89.
- Rasmussen, T. & Milner, B. (1977). The role of early left-brain injury in determining lateralization of cerebral speech functions. *Annals of the New York Academy of Sciences*, *299*, 355–369.
- Raven, J.C. (1965). *Guide to using the coloured progressive matrices*. London, England: HK. Lewis.
- Rosselli, M. & Ardila, A. (1989). Calculation deficits in patients with right and left hemisphere damage. *Neuropsychologia*, *27*, 607–617.
- Rossor, M.N., Warrington, E., & Cipolotti, L. (1995). The isolation of calculation skills. *Journal of Neurology*, *242*, 78–81.
- Stone, S.P., Patel, P., Greenwood, R.J., & Halligan, P.W. (1992). Measuring visual neglect in acute stroke and predicting its recovery: The visual neglect recovery index. *Journal of Neurology, Neurosurgery, and Psychiatry*, *55*, 431–436.
- Takayama, Y., Sugichita, M., Akiguchi, I., & Kimura, J. (1994). Isolated acalculia due to left parietal lesion. *Archives of Neurology*, *51*, 286–291.
- Thioux, M., Pillon, A., Samson, D., de Partz, M.P., Noel, M.P., & Seron, X. (1998). The isolation of numerals at the semantic level. *Neurocase*, *4*, 371–389.
- Thulborn, K.R., Carpenter, P.A., & Just, M.A. (1999). Plasticity of language-related brain function during recovery from stroke. *Stroke*, *30*, 749–754.
- Vitali, P., Tettamanti, M., Abutalebi, J., Danna, M., Ansaldi, A.I., Perani, D., Cappa, S., & Joanette, Y. (2003). Recovery from anomia: Effects of specific rehabilitation on brain reorganization: An er-gMRI study in 2 anomic patients. *Brain and Language*, *87*, 126–127.
- Warburton, E., Price, C.J., Swinburn, K., & Wise, R.J. (1999). Mechanisms of recovery from aphasia. Evidence from positron emission tomography studies. *Journal of Neurology, Neurosurgery and Psychiatry*, *66*, 155–161.
- Warrington, E. (1982). The fractionation of arithmetical skills: A case study. *Quarterly Journal of Experimental Psychology*, *34*, 31–51.

APPENDIX 1

Additions

$5 +$ $3 =$	$16 +$ $4 =$	$27 +$ $5 =$	$55 +$ $89 =$
$376 +$ $125 =$	$3.306 +$ $1.704 =$	$835 +$ $98.279 =$	

Subtractions

$8 -$ $2 =$	$19 -$ $8 =$	$15 -$ $9 =$	$72 -$ $35 =$
$149 -$ $34 =$	$5.000 -$ $354 =$	$60.100 -$ $4.712 =$	

Multiplications

$7 \times$ $8 =$	$11 \times$ $3 =$	$38 \times$ $4 =$	$142 \times$ $5 =$
$37 \times$ $24 =$	$308 \times$ $73 =$	$928 \times$ $726 =$	

Divisions

$6 : 2 =$	$15 : 3 =$	$45 : 9 =$	
$456 : 6 =$	$312 : 13 =$	$8.694 : 69 =$	

Raw scores' correction matrix for the influence of age and educational level

Age (years)	Educational level											
	5	6	7	8	9	10	11	12	13	14	15	> 15
11–15	4.3	3.4	2.4	1.5	0.5	−0.4	—	—	—	—	—	—
16–20	4.7	3.8	2.8	1.8	0.9	−0.1	−1.0	−2.0	−2.9	−3.9	−4.8	—
21–25	5.0	4.1	3.1	2.2	1.2	0.3	−0.7	−1.6	−2.6	−3.5	−4.5	−6.4
26–30	5.4	4.4	3.5	2.6	1.6	0.6	−0.3	−1.3	−2.2	−3.2	−4.1	−6.0
31–35	5.7	4.8	3.8	2.9	1.9	1.0	0.0	−0.9	−1.9	−2.8	−3.8	−5.7
36–40	6.1	5.1	4.2	3.3	2.3	1.3	0.4	−0.6	−1.5	−2.5	−3.4	−5.3
41–45	6.4	5.5	4.5	3.6	2.6	1.7	0.7	−0.2	−1.2	−2.1	−3.1	−5.0
46–50	6.8	5.8	4.9	3.9	3.0	2.0	1.1	0.1	−0.8	−1.8	−2.7	−4.6
51–55	7.1	6.2	5.2	4.3	3.3	2.3	1.4	0.5	−0.5	−1.4	−2.4	−4.3
56–60	7.5	6.5	5.6	4.6	3.7	2.7	1.8	0.8	−0.1	−1.1	−2.0	−3.9
61–65	7.8	6.9	5.9	5.0	4.0	3.1	2.1	1.2	0.2	−0.7	−1.7	−3.6
66–70	8.2	7.2	6.3	5.3	4.4	3.4	2.5	1.5	0.6	−0.4	−1.3	−3.2
71–75	8.6	7.6	6.7	5.7	4.8	3.8	2.9	1.9	0.9	0.0	−1.0	−2.9

APPENDIX 2

Let T1 and T2 denote the mean scores at the first and the second evaluation. In the absence of spontaneous recovery, a bivariate normal distribution with common mean μ , common variance σ^2 , and correlation ρ is assumed.

The dual-effect model decomposes the change score T2–T1 into three component parts, measuring the regression toward the mean $-(1 - \rho)(T1 - \mu)$, the true recovery effect $-\delta$, and the impact of the initial level of performance $-\eta(T1 - \mu)$, respectively, beside a random measurement effect ϵ . Thus, $T2 - T1 = -(1 - \rho)(T1 - \mu) - \delta - \eta(T1 - \mu) + \epsilon$.

In the present case, T1 was recorded on the whole sample of 190 participants and served to estimate μ and σ^2 , and T2 was recorded on the 92 acalculic participants and allowed estimation of ρ , δ , and η . Maximum Likelihood (ML) estimates for the parameters and their variances are given by Naranjo and McKean (2001).

Let m , s^2 , d , h , r , and g be unbiased ML estimates of μ , σ^2 , δ , η , ρ , and γ , where $\gamma = 1 - \eta/\rho$.

$$m = \sum_{i=1}^{190} T1_i / 190 \quad s^2 = \sum_{i=1}^{190} (T1_i - m)^2 / (190 - 1)$$

$$m1 = \sum_{i=1}^{92} T1_i / 92 \quad m2 = \sum_{i=1}^{92} T2_i / 92$$

$$(gr) = [\sum_{i=1}^{92} (T1_i - m1)(T2_i - m2)] / [\sum_{i=1}^{92} (T1_i - m1)^2]$$

$$d = (gr)(m1 - m) - (m2 - m)$$

$$r^2 = 1 - \{ \sum_{i=1}^{92} [T2_i - m2 - (gr)(T1_i - m1)]^2 \} / (92 s^2)$$

$$g = (gr)/r \quad h = r(1 - g)$$

$$SE(d) = [s^2(1 - r^2)/92]^{1/2}$$

$$SE(g) = \{ (1 - r^2)[g^2(1 - r^2) + 2r^2] / (184r^2) \}^{1/2}$$

The hypothesis of no effect of the initial level performance coincides with $\eta = \rho(1 - \gamma) = 0$ or, equivalently, with $\gamma = 1$.

The main hypothesis of no true recovery effect coincides with $\delta = 0$.

Owing to the large sample sizes (190 and 92 participants), statistical testing relied on standard deviations, rather than on the better approximate bootstrap-based confidence intervals.