Clinical Aphasiology, Vol. 24, 1996, pp. 159–172

# Evolution of Acute Aphasia as Measured by the Western Aphasia Battery

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Aphasiologists agree that aphasia recovery is dynamic in the 0- to 3- month post stroke period but differ as to the character of aphasia evolution. Debate about the evolution of aphasia syndromes pertains to the definition of aphasia syndromes, the evaluation instrument used, the time post onset that aphasia tests are given, the interface between aphasia severity and aphasia type, and the relationship of magnitude of change to frequency of evolution of aphasia from one type to another.

Kertesz and McCabe (1977) studied evolution of aphasia using the Western Aphasia Battery (Kertesz, 1979, 1982). Based on Western Aphasia Battery, aphasia quotients obtained in a serial fashion (at 45 days and at 3, 6, and 12 months post onset), 39 of 93 patients (41.9%) changed aphasia type as they recovered. Extent of recovery was reported to be good in Anomic, Conduction, Transcortical Motor, and Transcortical Sensory aphasia; fair to good in Broca's and some Wernicke's; fair to poor in most Wernicke's; and poor in Global aphasia. Basso and colleagues (1979) gave two nonstandardized aphasia examinations to patients with fluent or nonfluent aphasia. They found no significant difference in extent of recovery between these two types of aphasia when initial severity was controlled. Vignolo (1964), Kertesz and McCabe (1977), and Kertesz (1988) also acknowledged the importance of initial severity of aphasia, noting a ceiling effect in mild aphasia and a greater potential for change in severe aphasia.

Pashek and Holland (1988) prospectively examined 43 aphasic patients with the Western Aphasia Battery at 1, 2, 3, 4 to 6, and 7 to 12 month intervals. Using descriptive criteria rather than the Western Aphasia Battery taxonomy to define aphasia type, they found that 19 of 32 (59.4%) patients followed 6 months or longer changed aphasia type, and that older subjects were both more severe initially and less likely to evolve from one aphasia type to another than younger subjects.

Some researchers question the validity of aphasia classification (Arbib, Caplan, and Marshall, 1982; Caramazza and Badecker, 1989), while others are critical of the psychometric properties of current tests designed to identify classical aphasia syndromes (Crary, Wertz, and Deal, 1992; Swindell, Holland, and Fromm, 1984; Wertz, Deal, and Robinson, 1984). Still others minimize the value of classifying aphasia when assessing prognosis. Basso, for example, wrote: "Level of impairment soon after onset is a significant indicator of the level of ultimate recovery. The type of aphasia cannot be studied independently of the severity, since it is intrinsically related to it." (Basso, 1992, p. 342). The resultant bias against aphasia classification—while based on reasonable theoretical and psychometric arguments—is discordant with the fact that many clinicians find it useful (Kertesz, 1994, personal communication). In fact, a number of aphasia treatment texts use aphasia type as an organizing principle (Davis, 1993; LaPointe, 1990; Perkins, 1983). Thus, the intent of this research was not to enter the debate regarding the validity of classical syndromes or the characteristics of available tests, but rather to explore further the clinical usefulness of scores and profiles derived from the Western Aphasia Battery. The specific purpose was to define the relationship of aphasia type evolution to magnitude of change in aphasia quotients and to baseline aphasia type.

# METHOD

## Subjects

Over a 2 1/2 year period, 456 left hemisphere stroke patients were evaluated by speech–language pathologists at Duke University Medical Center. Because of its reported reliability and validity (Kertesz, 1979, 1982; Shewan and Kertesz, 1980), the Western Aphasia Battery was administered to most aphasic individuals who engaged in treatment programs. A retrospective analysis of these patient files yielded 39 who met the following selection criteria: single left hemisphere stroke (either ischemic or hemorrhagic) confirmed by head CT or MRI; at least two Western Aphasia Batteries within the acute recovery phase (within 3.5 months post onset); and, at least a 30-day interval between Test 1 and Test 2. Subjects were excluded if they had multiple or bilateral strokes, history of prior stroke, head trauma, dementia, delirium, or psychiatric illness requiring hospitalization. Finally, patients were excluded if initial aphasia quotients were 93.8 or greater, suggesting their performance to be within normal limits. Handedness, education, gender, or amount and duration of aphasia treatment were not stipulated among the selection criteria. Subject characteristics are summarized in Table 1.

**Table 1. Characteristics of the 39 Aphasic Subjects.**

| | | *Nonfluent* | *Fluent* | *All Subjects* |
|---|---|---|---|---|
| | | *(N = 21)* | *(N = 18)* | *(N = 39)* |
| *Handedness (R;L)** | | 20;1 | 18;0 | 38;1 |
| *Sex (M;F)** | | 14;7 | 9;9 | 23;16 |
| *Stroke Type (I;H)** | | 18;3 | 16;2 | 34;5 |
| *Age (yrs)* | *Mean* | 64.2 | 66.1 | 65.1 |
| | *Range* | 49–79 | 27–88 | 27–88 |
| | *SD* | 8.13 | 16.59 | 13.05 |
| | *Median* | 65 | 68 | 67 |
| *Education (yrs)* | *Mean* | 10.3 | 10.3 | 10.3 |
| | *Range* | 0–16 | 4–16 | 0–16 |
| | *SD* | 3.69 | 3.00 | 3.41 |
| | *Median* | 12 | 10 | 11 |
| *Speech-language pathology sessions* | *Mean* | 43.9 | 38.1 | 41.2 |
| | *Range* | 2–98 | 0–78 | 0–98 |
| | *SD* | 22.50 | 22.12 | 22.53 |
| | *Median* | 39 | 45.5 | 40 |
| *Days post stroke at initial Western Aphasia Battery* | *Mean* | 25.6 | 27.3 | 26.4 |
| | *Range* | 7–37 | 3–42 | 3–42 |
| | *SD* | 8.52 | 9.15 | 8.85 |
| | *Median* | 25 | 28 | 27 |
| *Days post stroke at final Western Aphasia Battery* | *Mean* | 67.0 | 70.3 | 68.5 |
| | *Range* | 33–101 | 46–169 | 33–169 |
| | *SD* | 18.80 | 28.09 | 18.45 |
| | *Median* | 70 | 67 | 70 |
| *Aphasia Quotient Change* | *Mean* | 16.1 | 15.4 | 15.8 |
| | *Range* | 1.5–49.4 | −1.7–34.9 | −1.7–49.4 |
| | *SD* | 12.2 | 11.3 | 11.8 |
| | *Median* | 16.3 | 12.8 | 15.6 |

*Note:* Aphasia *type* and *fluent* versus *nonfluent* classifications were determined at baseline examination using the Western Aphasia Battery (WAB) taxonomy (Kertesz, 1982).
*Handedness: R: right; L: left; *Sex:* M: male; F: female; *Stroke Type:* I: ischemic; H: hemorrhagic

## Procedures

Entered on a database were demographic data, days post stroke at initial and final test, aphasia quotients, and subtest scores comprising the aphasia quotient (i.e., speech content, speech fluency, auditory comprehension, oral repetition, and oral naming). Data were taken from existing files and calculations for the adjusted subtest scores and the total aphasia quotients were verified prior to database entry.

Performance change scores were computed relative to the group as a whole and to nonfluent versus fluent classifications. Aphasia type and evolution of aphasia type were subsequently derived. Aphasia type was determined using the Western Aphasia Battery taxonomy (Kertesz, 1979, 1982) and as such was based on scores pertaining to speech fluency, comprehension, repetition, and naming. Using this taxonomy, the fluent aphasias potentially identifiable by the Western Aphasia Battery were Transcortical Sensory, Wernicke's, Conduction, and Anomic; the nonfluent aphasias were Global, Isolation, Transcortical Motor, and Broca's. For the present study, evolution of aphasia type was defined as a change in the aphasia type between Test 1 and Test 2.

To assess inter-rater reliability, 312 scores were printed from the database (39 subjects $\times$ 4 subtests $\times$ 2 tests). Judge 1 (FBM) and Judge 2 (JH) independently entered these data into the Western Aphasia Battery classification matrix (Kertesz, 1982). These data allowed 78 possible aphasia type comparisons (39 subjects $\times$ 2 tests), about which the two judges independently agreed on the aphasia type 76 of 78 times (97.4%), for 38 of 39 subjects (97.4%). The disagreement reflected a systematic error in data entry and neither an error on the raw scoresheet nor an error in computation of subtest scores. Thus, of 312 scores, 310 (99.4%) were verified to be correct as compared to the raw data sheets. Once the errors were corrected, the interjudge agreement about aphasia type improved to 100%.

## Statistical Analyses

For each patient, an aphasia quotient change score was derived by subtracting the baseline aphasia quotient score from the aphasia quotient at reevaluation. To test whether the group as a whole improved, a paired t-test was used on the change scores. To further investigate factors that might influence this change, a linear regression analysis was performed with the aphasia quotient change score as the dependent variable and with baseline aphasia quotient, age, sex, and days post onset at the time of baseline examination as the independent variables. A similar regression analysis of the factors influencing whether a patient changed aphasia type

between Test 1 and Test 2 was also performed. Because the dependent variable in this case was dichotomous (i.e., aphasia type changed, or it did not), a logistic regression analysis was used with the same independent variables as in the previous analysis. The intent of using linear and logistic regression procedures was to assess the significance of an independent variable while controlling for the potentially confounding effects of all of the independent variables. The number of independent variables was limited to assure an appropriate subjects-to-variables ratio.

# RESULTS

## Timing of Evaluations

The Western Aphasia Battery was administered in three time periods: first time period, 0 to 30 days; second time period, 31 to 60 days; and, third time period, 61 to 102 days. Though a minimum of 30 days intervened between Test 1 and Test 2 for all subjects, the time of initial test varied. Cohort 1 was comprised of 26 subjects who were initially tested during the first time period. Of these subjects, 13 were reevaluated in the second time period, and 13 in the third time period. Cohort 2 was comprised of an additional 13 subjects who were evaluated initially within the second time period and reevaluated within the third time period. Cohort 1 and Cohort 2 were comparable in age (65.1 and 64.9 years, respectively). Cohort 1 achieved modestly higher aphasia quotients than Cohort 2 both at baseline and at follow-up examinations (42.5 and 59.7 for Cohort 1; 34.4 and 47.8 for Cohort 2, respectively). To assess the significance of time post onset at the time of baseline examination, the variable days post onset at baseline examination was entered into the linear and logistic regression analyses.

## Baseline and Final Aphasia Type

Among the 39 subjects, 21 (54%) had nonfluent aphasia and 18 (46%) had fluent aphasia at the time of baseline examination. The fluent aphasias were represented by Wernicke's (N = 7), Anomic (N = 5), Transcortical Sensory (N = 3), and Conduction (N = 3); the nonfluent aphasias by Global (N = 13), Broca's (N = 6), and Transcortical Motor (N = 2). At the time of final examination, the fluent aphasias were represented by Wernicke's (N = 3), Anomic (N = 14), Transcortical Sensory (N = 2), and Conduction (N = 6); the nonfluent aphasias by Global (N = 4) and Broca's (N = 9), while one patient evolved to the nonaphasic range of performance.

## Aphasia Quotients, Aphasia Types, and Evolution

The mean change in aphasia quotient for all subjects (Table 1) was 15.8, yielding a significant difference between Test 1 and Test 2 using the paired t-test ($t = 8.216$; $df = 38$; $p = .0001$). Among our 39 subjects, 26 (67%) experienced an evolution of aphasia type; 13 (33%) did not. The mean aphasia quotient change score for those who evolved from one aphasia type to another was 19.7; the score for those who did not evolve was 8.0 (Table 2).

Using linear regression, with aphasia quotient change as the dependent variable, initial severity (baseline aphasia quotient) was not associated at a statistically significant level with the aphasia quotient change scores, but revealed a significant effect for age ($t_{29} = 3.75$, $p = .0007$) and days post onset at baseline examination ($t_{29} = 3.49$, $p = .0014$). (For the linear regression, $R^2 = .525$.) Using a logistic regression statistic, with change in aphasia type as the dependent variable, age was shown to be significantly associated with an evolution of aphasia type between Test 1 and Test 2 ($X^2 = 4.12$, $p = .04$), as was days post onset at baseline examination ($X^2 = 5.20$, $p = .02$).

The following descriptive statistics help clarify these findings for age and time post onset relative to aphasia quotient change and aphasia type evolution. The average aphasia quotient change score for younger subjects ($< 65$ years) was 22.1 and for older subjects ($\geq 65$ years) was 11.8. The difference between mean aphasia quotient change scores was associated with different frequencies of aphasia type evolution: 13 of 15 (86.7%) in younger subjects as compared to 13 of 24 (54.2%) in older subjects. For subjects evaluated in the 0- to 30-day period (Cohort 1) the aphasia quotient change score was 17.2; for those initially evaluated in the 31 to 60 day period (Cohort 2), the aphasia quotient change score was 13.4. The difference between mean aphasia quotient change scores was associated with higher frequencies of aphasia type evolution: 19 of 26 (73.1%) in Cohort 1 as compared to 7 of 13 (53.8%) in Cohort 2.

## Nonfluent versus Fluent Aphasia and Evolution

Our 21 nonfluent subjects, as a group, had lower aphasia quotients—both initial and final—than our 18 fluent subjects (Table 3). Nevertheless, the magnitude of change associated with evolution of aphasia type was similar regardless of the fluent-nonfluent classification. Nonfluent subjects who evolved from one aphasia type to another showed a mean aphasia quotient change score of 18.3; fluent subjects, 21.6 (Tables 1 and 3). Nonfluent subjects who did not evolve showed a mean aphasia quotient change score of 10.5; fluent subjects, 5.8. Despite the fact that nonfluent subjects were more severe initially than fluent subjects, the frequency with which aphasia type evolved between the two tests was similar: 15 (71%)

**Table 2. Mean Performance on the Western Aphasia Battery Subtests and Aphasia Quotient Contrasting the Change Scores of Those Patients Whose Aphasia Type Evolved (N = 26) Versus Did Not Evolve (N = 13) during Acute Recovery**

| | | *Subjects Whose Aphasia Type Evolved (N = 26)* | | | *Subjects Whose Aphasia Type Did Not Evolve (N = 13)* | | |
|---|---|---|---|---|---|---|---|
| ***Subscore*** | ***Max*** | ***Initial*** | ***Final*** | ***Change*** | ***Initial*** | ***Final*** | ***Change*** |
| *Content* | 10 | 3.5 | 5.7 | 2.2 | 4.7 | 5.6 | 0.9 |
| *Fluency* | 10 | 3.6 | 5.5 | 1.9 | 4.6 | 5.5 | 0.9 |
| *Comprehension* | 10 | 4.9 | 7.1 | 2.2 | 5.5 | 6.2 | 0.7 |
| *Repetition* | 10 | 3.7 | 5.5 | 1.8 | 4.2 | 5.0 | 0.8 |
| *Naming* | 10 | 2.9 | 5.0 | 2.1 | 4.1 | 4.9 | 0.8 |
| *Aphasia Quotient* | 10 | 36.7 | 56.4 | 19.7 | 45.9 | 53.9 | 8.0 |

*Note*: Aphasia *type* and *fluent vs. nonfluent* classification were determined at baseline examination using the Western Aphasia Battery taxonomy (Kertesz, 1982)

*Table 3. Mean Performance on Western Aphasia Battery Subtests and Aphasia Quotient Contrasting the Change Scores of Patients Whose Aphasia Type Evolved Versus Did Not Evolve During Acute Recovery When Patients Were Categorized as Fluent Versus Nonfluent Aphasia*

| | | *Nonfluent Aphasic Subjects (N = 21)* | | | | | | *Fluent Aphasic Subjects (N = 18)* | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | *Type Evolved (N = 15)* | | | *Type Did Not Evolve (N = 6)* | | | *Type Evolved (N = 11)* | | | *Type Did Not Evolve (N = 7)* | | |
| *Subscore* | *Max* | *Initial* | *Final* | *Change Score* | *Initial* | *Final* | *Change Score* | *Initial* | *Final* | *Change Score* | *Initial* | *Final* | *Change Score* |
| *Content* | 10 | 1.7 | 4.3 | 2.6 | 0.8 | 2.2 | 1.4 | 5.9 | 7.6 | 1.7 | 8.0 | 8.6 | 0.6 |
| *Fluency* | 10 | 1.7 | 3.7 | 2.0 | 1.0 | 2.5 | 1.5 | 6.2 | 7.8 | 1.6 | 7.7 | 8.1 | 0.4 |
| *Comprehension* | 10 | 3.9 | 5.9 | 2.0 | 2.3 | 3.3 | 1.0 | 6.2 | 8.8 | 2.6 | 8.1 | 8.7 | 0.6 |
| *Repetition* | 10 | 2.0 | 3.9 | 1.9 | 0.6 | 2.0 | 1.4 | 5.9 | 7.7 | 1.8 | 7.3 | 7.6 | 0.3 |
| *Naming* | 10 | 1.5 | 3.2 | 1.7 | 0.3 | 1.2 | 0.9 | 4.9 | 7.3 | 2.4 | 7.4 | 8.1 | 0.7 |
| *Aphasia Quotient* | 10 | 22.0 | 40.3 | 18.3[a] | 10.1 | 20.6 | 10.5[b] | 56.8 | 78.4 | 21.6[c] | 76.6 | 82.4 | 5.8[d] |

[a]median = 17.2 (range 3.9–49.4)
[b]median = 5.0 (range 1.5–40.8)
[c]median = 20.5 (range 6.6–34.9)
[d]median = 3.2 (range −1.7–13.5)
*Note:* Aphasia *type* and *fluent vs. nonfluent aphasia* determined at baseline using WAB taxonomy (Kertesz, 1982).

for nonfluent subjects; 11 (61%) for fluent subjects. Thus, neither the aphasia quotient change scores nor the frequency of aphasia type evolution appeared to be related to the fluent-nonfluent classification.

A final observation (derived from Table 3) is that nonfluent subjects who evolved from one aphasia type to another were less severe than their nonfluent counterparts who did not evolve (as judged from the subgroup mean aphasia quotients). In contrast, fluent aphasic subjects who evolved were more severely impaired than their fluent counterparts who did not evolve (as judged from the subgroup mean aphasia quotients). Close inspection of the data in the bottom row of Table 3 shows that an evolution of aphasia type was less likely to occur when mean aphasia quotients fell either below 20 or above 80, than when aphasia quotients fell between 20 and 80.

## Consolidated Aphasia Type Evolution

While recognizing differences in methodology among Kertesz and McCabe (1977), Pashek and Holland (1988) and the present study, as well as differences in cell sizes among the aphasia types, we consolidated these data about the frequency with which different subjects evolve from one aphasia type to another. This consolidation (Table 4) reveals that about 50% of all aphasic patients evolved from one type to another during the course of recovery. However, the relative frequency of aphasia evolution depending on baseline aphasia type, ranged from 39.3% for Broca's aphasia to 100% for Transcortical Motor aphasia. Among our sample of 39 subjects, the patterns of aphasia type evolution were as follows (Table 4). Of 13 patients with Global aphasia, four remained Global, seven evolved to Broca's, and two evolved to Wernicke's. Of six patients with Broca's aphasia, two remained Broca's, one evolved to Transcortical Sensory aphasia, two evolved to Conduction, and one evolved to Wernicke's (the latter due to a change in the speech fluency rating). Both patients with Transcortical Motor aphasia evolved to Anomic. Of three patients with Transcortical Sensory aphasia, one remained Transcortical Sensory, while the other two patients evolved to Anomic. Of seven patients with Wernicke's aphasia, two evolved to Conduction, and five evolved to Anomic. Of three patients with Conduction aphasia, two remained Conduction, and one evolved to Anomic. Of five Anomic aphasia patients, four remained Anomic, and one evolved to the range of nonaphasic performance.

# DISCUSSION

The main findings follow. First, the magnitude of aphasia quotient change was significant from Test 1 to Test 2. Second, evolution of aphasia from one type to another was associated with the magnitude of aphasia quotient

## Table 4. A Consolidation of Aphasia Type Evolution: Initial and Final Aphasia Diagnoses as Measured by the Western Aphasia Battery[a]

| *Author*[b,c] | *Initial Aphasia Type* | *Final Aphasia Type* | | | | | | | | | *Relative Change by Type* |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | *Global* | *Broca's* | *TMA* | *Isolation* | *TSA* | *Wernicke's* | *Conduction* | *Anomic* | *Nonaphasic* | *(Percent)* |
| | Global | | | | | | | | | | |
| Kertesz & McCabe, 1977 | 22 | 17 | 2 | 1 | — | — | — | 1 | 1 | — | |
| Pashek & Holland, 1988 | 7 | 2 | 2 | — | — | — | 1 | — | 1 | 1 | 45.2 |
| McDermott, Horner & DeLong, 1996 | 13 | 4 | 7 | — | — | — | 2 | — | — | — | |
| Total | 42 | 23 | 11 | 1 | — | — | 3 | 1 | 2 | 1 | |
| | Broca's | | | | | | | | | | |
| Kertesz & McCabe, 1977 | 17 | — | 13 | 1 | — | — | — | — | 3 | — | |
| Pashek & Holland, 1988 | 5 | — | 2 | — | — | — | — | — | 1 | 2 | 39.3 |
| McDermott et al., 1996 | 6 | — | 2 | 0 | — | 1 | 1 | 2 | 0 | — | |
| Total | 28 | — | 17 | 1 | — | 1 | 1 | 2 | 4 | 2 | |
| | TMA | | | | | | | | | | |
| Kertesz & McCabe, 1977 | 3 | — | — | — | — | — | — | — | 2 | 1 | |
| Pashek & Holland, 1988 | — | — | — | — | — | — | — | — | — | — | 100.0 |
| McDermott et al., 1996 | 2 | — | — | — | — | — | — | — | 2 | — | |
| Total | 5 | — | — | — | — | — | — | — | 4 | 1 | |
| | Isolation | | | | | | | | | | |
| Kertesz & McCabe, 1977 | 2 | | | | | | | | 1 | — | |
| Pashek & Holland, 1988 | — | — | — | — | 1 | — | — | — | — | — | 50.0 |
| McDermott et al., 1996 | 0 | — | — | — | — | — | — | — | — | — | |
| Total | 2 | | | | | | | | 1 | — | |
| | | — | — | — | — | — | — | — | | | |
| | | — | — | — | 1 | — | — | — | | | |

| *Author*[b,c] | *Initial Aphasia Type* | *Final Aphasia Type* | | | | | | | | | *Relative Change by Type* |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | *Global* | *Broca's* | *TMA* | *Isolation* | *TSA* | *Wernicke's* | *Conduction* | *Anomic* | *Nonaphasic* | *(Percent)* |
| | TSA | | | | | | | | | | |
| Kertesz & McCabe, 1977 | 3 | — | — | — | — | — | — | — | 1 | 2 | |
| Pashek & Holland, 1988 | 1 | — | — | — | — | — | — | — | 1 | — | 85.7 |
| McDermott et al., 1996 | 3 | — | — | — | — | 1 | — | — | 2 | — | |
| Total | 7 | — | — | — | — | 1 | — | — | 4 | 2 | |
| | Wernicke's | | | | | | | | | | |
| Kertesz & McCabe, 1977 | 13 | 1 | — | — | — | 1 | 7 | — | 4 | — | |
| Pashek & Holland, 1988 | 5 | — | — | — | — | — | 2 | 1 | 2 | — | 64.0 |
| McDermott et al., 1996 | 7 | — | — | — | — | — | 0 | 2 | 5 | — | |
| Total | 25 | 1 | — | — | — | 1 | 9 | 3 | 11 | — | |
| | Conduction | | | | | | | | | | |
| Kertesz & McCabe, 1977 | 8 | — | — | — | — | — | — | 1 | 2 | 5 | |
| Pashek & Holland, 1988 | 2 | — | — | — | — | — | — | — | 2 | — | 76.9 |
| McDermott et al., 1996 | 3 | — | — | — | — | — | — | 2 | 1 | — | |
| Total | 13 | — | — | — | — | — | — | 3 | 5 | 5 | |
| | Anomic | | | | | | | | | | |
| Kertesz & McCabe, 1977 | 25 | — | — | — | — | — | — | — | 13 | 12 | |
| Pashek & Holland, 1988 | 3 | — | — | — | — | — | — | — | 1 | 2 | 45.5 |
| McDermott et al., 1996 | 5 | — | — | — | — | — | — | — | 4 | 1 | |
| Total | 33 | — | — | — | — | — | — | — | 18 | 15 | |
| *GRAND TOTALS* | *155* | *24* | *28* | *2* | *1* | *3* | *13* | *9* | *49* | *26* | *53.5%* |

[a]Pashek & Holland (1988) tested patients with the *Western Aphasia Battery*, but used composite syndrome definitions rather than the *WAB* taxonomy to assign aphasia type. Kertesz & McCabe (1977) and McDermott, Horner & DeLong (1994) used the *Western Aphasia Battery* classification approach (Kertesz, 1979, 1982).

[b]Patients from Kertesz & McCabe (1977) with aphasia due to trauma, aneurysm, or AVM were excluded from this table; patients sustaining ischemic and hemorrhagic strokes only were included.

[c]Patients from Pashek & Holland (1988) who were defined as unclassifiable or dementia were excluded from this table.

change. Third, aphasia quotient change scores were associated significantly with age of subjects and days post onset at initial examination, but not with initial severity (baseline aphasia quotient), or patients' gender. Fourth, the fluent and nonfluent subgroups behaved similarly in that an aphasia quotient change of approximately 20 points predicated aphasia type evolution in approximately two-thirds of the patients in each group. Fifth, the observed interaction between fluent-nonfluent aphasia classification and aphasia severity suggests that there may be a dynamic range within which aphasia evolution occurs. Sixth, consolidated data showed that aphasia type evolved for about half of the subjects.

The overall significant improvement of our subjects between Test 1 and Test 2, though not unexpected for a group of acutely aphasic individuals, confirms that our group of 39 subjects is typical of those reported in the aphasia recovery literature. The 0- to 3-month post stroke period was a potent period of recovery for our patients. During this period, younger patients and patients evaluated earlier experienced more significant improvement (and therefore, a higher frequency of aphasia type changes) than older patients and those evaluated later.

Patients who evolved from one aphasia type to another had more substantial aphasia quotient changes than those who did not, suggesting that improvement in language performance (in this case, as captured by the aphasia quotient) is a necessary condition for evolution of aphasia type. The subgroup-by-subtest data (see Table 3) suggest that aphasia quotient gains reflected uniform gains across all subtests. Visual inspection of these data might lead one to conclude that evolution of acute aphasia is predicated on across-the-board improvement. Our close inspection of individual subject data, as well as our clinical experience, suggests that this is not necessarily the case. For example, in instances that Global aphasia evolves to Broca's aphasia, the critical gain occurs in auditory comprehension; in instances that Conduction aphasia evolves to Anomic aphasia, the critical gain occurs in oral repetition. Thus, overall gains are necessary to occasion aphasia type evolution, but subtest-specific gains are essential relative to specific patterns of evolution.

Though we confirm Basso et al.'s findings (1979) that both fluent and nonfluent aphasia improve to a similar extent, we also observed that the fluent-nonfluent classification may interact with aphasia severity. Among our subjects, nonfluent patients who evolved from one aphasia type to another were less severe than their nonfluent counterparts who did not evolve, while fluent aphasic subjects who evolved were more severe than their fluent counterparts who did not evolve. We suggest that the aphasia quotient range of 20 to 80 (out of 100 possible) may be the dynamic range in which aphasia type evolution is most likely. Thus, clinicians may find it valuable to consider aphasia classification in conjunction with severity rather than to base prognostic estimations on absolute levels of performance alone.

The potential value of knowing the aphasia type at the time of initial evaluation is strengthened by the consolidated data. The precise direction of evolution during acute recovery appears to operate under some constraints (e.g., Global to Broca's; Wernicke's to Anomic), and the relative likelihood of aphasia type evolution appears to vary with baseline aphasia type. Despite the small cell sizes in some instances (due to the rarity of certain aphasia syndromes), the trends are beginning to emerge. The data are convincing that the aphasia subtypes differ in their evolution.

In closing, our data agree in part with Basso's (1992) observation. Type of aphasia is related to severity of aphasia. Despite compelling evidence in the literature that initial overall severity has a potent influence on recovery, it is clearly not the only influence. In our study, magnitude of change, and not initial severity, was the critical variable underlying aphasia evolution. To suggest that the aphasia type is intrinsically related to aphasia severity and therefore aphasia type should be ignored (Basso, 1992) is to undermine the potential value of knowing the options regarding patterns of evolution and anticipating the likelihood of evolution. Just as advancing age and time post onset are important prognostic considerations, so too are the qualitative aspects of the aphasia as captured in aphasia classification.

We think aphasia type has potential value for both prognosis and treatment. Regardless of the aphasia classification system, knowing aphasia type increases our sensitivity not only to the early profile of strengths and weaknesses (both qualitative and quantitative), but also to the evolving profile. Therefore, aphasia severity and aphasia type are best analyzed in tandem.

## ACKNOWLEDGMENTS

Amy Chaney, M.S.; Joy Crowder, M.A.; Ann Cyptar, M.S.; Mary Ann Eller, M.A.; Debra A. Girod, M.S.; Carolyn Reus, M.S.; and Andrew Kertesz, M.D., F.A.C.P.(C)

*This research was partially funded by a grant from the Mellon Foundation (David Matchar, M.D., and Jennifer Horner, Ph.D., Co-P.I.s)*

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