Lesion Site Patterns in Severe, Nonverbal Aphasia to Predict Outcome With a Computer-Assisted Treatment Program

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**Objective:** To test whether lesion site patterns in patients with chronic, severe aphasia who have no meaningful spontaneous speech are predictive of outcome following treatment with a nonverbal, icon-based computer-assisted visual communication (C-ViC) program.

**Design:** Retrospective study in which computed tomographic scans performed 3 months after onset of stroke and aphasia test scores obtained before C-ViC therapy were reviewed for patients after receiving C-ViC treatment.

**Setting:** A neurology department and speech pathology service of a Department of Veterans Affairs medical center and a university aphasia research center.

**Patients:** Seventeen patients with stroke and severe aphasia who began treatment with C-ViC from 3 months to 10 years after onset of stroke.

**Main Outcome Measure:** Level of ability to use C-ViC on a personal computer to communicate.

**Results:** All patients with bilateral lesions failed to learn C-ViC. For patients with unilateral left hemisphere lesion sites, statistical analyses accurately discriminated between those who could initiate communication with C-ViC from those who were only able to answer directed questions. The critical lesion areas involved temporal lobe structures (Wernicke cortical area and the subcortical temporal isthmus), supraventricular frontal lobe structures (supplementary motor area or cingulate gyrus 24), and the subcortical medial subcallosal fasciculus, deep to the Broca area. Specific lesion sites were also identified for appropriate candidacy for C-ViC.

**Conclusions:** Lesion site patterns on computed tomographic scans are helpful to define candidacy for C-ViC training, and to predict outcome level. A practical method is presented for clinical application of these lesion site results in combination with aphasia test scores.

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**TREATMENT OF patients with aphasias who have no meaningful speech has generally been ineffective using gesture, pantomime, drawing, or picture manipulation.** More than 20 years ago, the first attempts using a nonverbal substituted language based on pictures and icons on cards (visual communication [ViC]) were reported. More recently, ViC was adapted for computer use (computer-assisted visual communication [C-ViC]). Not all patients with severe aphasia have been able to learn the lexical and syntactic rules of C-ViC and use them to independently initiate communication.

Lesion sites that might underlie this difference in response to C-ViC were reported in a pilot investigation of 7 patients in whom a specific left hemisphere lesion site pattern was associated with ability to use C-ViC to initiate communication (best response). A different pattern was associated with inability to use C-ViC to initiate communication, but ability to use C-ViC to answer questions posed by others (moderate response). Patients with best response had either no lesion in, or extensive lesion in only 1 of the following 2 left hemisphere areas: area 1—temporal lobe structures (Wernicke cortical area, or the subcortical anterior temporal isthmus area, containing afferent auditory projections from medial geniculate body to the Heschl gyrus); or area 2—supraventricular frontal lobe structures (supplementary motor area [SMA] or cingulate gyrus area 24, cortical or white matter). Patients with moderate response had extensive lesion in both areas 1 and 2.

In the present study, the lesion site patterns from patients in the pilot study were applied to predict C-ViC outcome for 17 new patients. The primary objective was to test the validity of the original hypothesis that patients with best response would have either no lesion in, or extensive lesion in either area 1 or area 2, and that patients with only moderate response would have extensive lesion in both areas 1 and 2.
PATIENTS AND METHODS

PATIENTS

All patients (N = 17) had had a left hemisphere cerebrovascular accident; 4 patients also had a silent right cerebrovascular accident, first documented on computed tomographic (CT) scan performed at the time of the left cerebrovascular accident. Age at onset of left cerebrovascular accident ranged from 33 to 74 years (mean [SD], 57.8 [12.6] years). Patients were right-handed; all had severe right hemiplegia (except for patients 4 and 6).

Before C-ViC training, patients were tested with the BASA examination15 (Table 1). All patients met the major criterion for entry, eg, severe limitation in speech output, with little or no meaningful spontaneous speech (and writing) in conversation or in picture description. Auditory comprehension was impaired in all patients; however, there was variability, and not all patients were globally aphasic.

Before C-ViC training, most patients had been treated with 1 or more traditional treatment programs without success. The decision to recommend C-ViC was made by the speech/language pathologist administering the BASA examination; no lesion site information was used.

Treatment with C-ViC began during the chronic phase of stroke, ranging from 3 months to 10 years after stroke onset (Table 1). All patients were able to control the computer mouse with the nonparalyzed hand and were seen as outpatients for 1-hour treatment sessions, usually twice per week for about 6 months to 1 year.

THE C-ViC TREATMENT PROGRAM

Patients were prescreened to determine whether they could match 8 icons on the computer screen to the same 8 real objects. Four patients could not perform this icon-to-object matching task, despite 2 to 4 weeks of training; they were classified as no response.

The C-ViC training consists of 2 phases (E.H.B. and M.N., unpublished data, 1998). In phase 1, patients are trained to use the computer mouse to carry out commands given to them in C-ViC (comprehension), to answer simple questions, and, finally, to compose descriptions of simple acts (production). Patients learn to use 3 verb action icons (lift, turn, give), pictures of objects (16-24 icons), and pictures of people (minimum of 3). Faces from photographs are scanned into the computer, including those for patient and therapist. Patients learn to arrange person, action, and object icons in a left-to-right grammatically correct order. Phase 1 is considered complete when a patient can describe events without error, and without guidance, using 2 grammatical constructions: (1) subject-predicate-object and (2) subject-predicate-indirect object-direct object (Figure 1, top).

Phase 2 training focuses on real-life communicative acts, including describing simple acts, expressing needs, asking questions, and making requests (giving commands) (Figure 1, bottom). Patients learn to use up to 23 verb action icons, up to 160 pictures of objects, 5 to 10 pictures of people, and conjunctions and modifiers for a maximum vocabulary of 240 icons. Criterion for mastery of each verb action icon is reached at 75% (or better) correct use without guidance for 2 consecutive treatment sessions. Phase 2 is completed when 7 different grammatical constructions have been mastered.

Quality of communications generated by patients using C-ViC in phase 2 was rated by the clinician administering the program (E.H.B.) using a rating scale based on different from the Porch Index of Communicative Ability (PICA).16 This 8-point rating scale (Visual Index of Communicative Ability; VICA) ranged from 0 to 7 (Figure 2). Phase 2 VICA ratings of greater than 4.5 were considered good C-ViC productions; less than 4.5, poor.

CT SCAN ACQUISITION AND ANALYSES

We have observed that CT scans performed at less than 3 months after stroke onset do not adequately reveal the complete borders of an area of infarction, especially in white matter adjacent to ventricle (C.L.P., M.A.N., R.S., S.M.H., and M.N.P., unpublished data, 1998). These areas are important to examine in relationship to potential for recovery of speech.17 Therefore, all patients underwent noncontrast CT scanning at 3 months after stroke or later (Figure 3).

The lesion areas on CT scan were analyzed with 2 methods: (1) lesion site analysis and (2) total brain lesion size analysis. Lesion site analysis included visual assessment of each neuroanatomical area (eg, Broca area, Wernicke area, etc), where presence or absence of lesion and extent of lesion within that area were determined. The neuroanatomical

Outcome levels following phase 2 C-ViC training are summarized in the last column of Table 1 (VICA rating). Seven patients were able to use C-ViC to initiate communication (best response); 6 patients were not able to initiate communication, but they were able to use C-ViC to respond to questions posed by others (moderate response). Four patients were unable to learn to match 8 icons on the computer screen to the same 8 real objects (no response). Subsequent statistical analyses were performed only between best and moderate response groups.

Table 2 shows that there were no significant differences between best and moderate response groups regarding age, months after stroke onset entering C-ViC, or in number of weeks in C-ViC treatment. There was a trend for the no response group to be older than the other 2 groups regarding age at stroke onset (P = .06).

Table 3 summarizes statistical comparisons between the best response and moderate response groups during C-ViC training. There was variability among patients and a tendency for the data to be skewed; therefore, medians were computed and comparisons were con-
areas examined were those previously observed to be relevant to outcome with C-ViC\(^2\) and to recovery of speech and comprehension.\(^{17-21}\) Lesion size analysis included calculation of the percent lesion size across the whole brain.

**Lesion Site Analysis**

The neuroanatomical areas examined for presence or absence of lesion and extent of lesion are diagrammed in Figure 3. The extent of lesion within each area was visually assessed using a 6-point rating scale, where 0 indicates no lesion present in that area and 5, lesion is present in that entire area.\(^{17}\) Lesion extent values greater than 3 (indicating lesion in greater than half of that area) are considered to be extensive lesions and have been observed to be associated with more severe deficits.\(^{17,20,21}\)

Scans were rated by 2 experienced raters (M.A.N. and C.L.P.); conferenced data were used. Interrater reliability coefficients range from 0.93 ($P<.001$) to 0.97 ($P<.001$) (C.L.P., M.A.N., R.S., S.M.H., and M.N.P., unpublished data, 1998).\(^{22}\) The CT scan analyses were performed in a blinded manner without information regarding C-ViC outcome.

Within area 1, each structure (Wernicke cortical area and the subcortical temporal isthmus area) was examined separately for extent of lesion using the 6-point rating scale. Area 2, the supraventricular frontal lobe structures (SMA or cingulate gyrus area 24), was assessed only for presence or absence of lesion. The location of white matter pathways originating from these cortical areas is not known, thus, it was not possible to know if lesion was present in greater than or less than half of these structures. A plus sign indicated that visible lesion was present in the SMA, the supraventricular cingulate gyrus area 24, or white matter deep to them; minus sign, no visible lesion.

One secondary objective was to define a lesion site pattern that could be identified with candidacy appropriate for C-ViC training, ie, lesion sites compatible with no recovery of meaningful spontaneous speech. Patients who do not recover speech have extensive lesion in 2 subcortical white matter pathway areas\(^{17}\): (1) the medial subcallosal fasciculus area (located deep to the Broca area, anterolateral and adjacent to the left frontal horn); and (2) the middle one third periventricular white matter (middle 1/3 PVWM) area (located deep to the motor and sensory cortex area for mouth, lateral and adjacent to the body of the left lateral ventricle). The medial subcallosal fasciculus area contains, in part, white matter pathways from the SMA and supraventricular cingulate gyrus area 24 to the head of the caudate and are believed to be important, in part, for initiation of speech. The middle 1/3 PVWM area contains efferent and afferent white matter pathways for the mouth, as well as other thalamocortical, intrahemispheric, and interhemispheric pathways believed to be important, in part, for motor and sensory aspects of speech.\(^{17}\) Each of these 2 areas was visually assessed for extent of lesion using the 6-point rating scale. The extent-of-lesion rating for the medial subcallosal fasciculus area was assessed at slice B (Broca) and at slice B/W (Broca and Wernicke) (Figure 3), and a mean across the 2 slices was computed. This mean was added to the single extent-of-lesion rating for the middle 1/3 PVWM area at slice SM (supramarginal gyrus). Maximum possible summed extent-of-lesion rating for these 2 areas combined is 10.

Most patients with aphasia who have a summed extent-of-lesion rating greater than 7 for these 2 areas have no recovery of meaningful spontaneous speech,\(^{17}\) and are therefore likely candidates for C-ViC. Most patients with aphasia who have a summed extent-of-lesion rating less than 7 for these 2 areas have some recovery of meaningful fluent speech\(^{17}\); these patients are candidates for verbal treatment programs.\(^{21-25}\)

Additional left perisylvian areas examined included frontal operculum (Broca area), supramarginal gyrus, angular gyrus, and additional areas shown in Figure 3.

**Total Lesion Size Analysis**

The lesion borders were defined as areas of visible low-density signal, separate from ventricles and fissures.\(^{26}\) If lesion was also present in the right hemisphere, that lesion was combined with the left, to compute a total percent brain lesion size.

**Hemispheric Asymmetries**

The CT scan occipital length asymmetries were measured because some patients with global aphasia with atypical occipital asymmetry on CT scan (equal, or increased right asymmetry) have improved recovery in single-word comprehension, repetition, or naming.\(^{23}\) Conducted using the Mann-Whitney $U$ test. The best response group required significantly fewer sessions to complete phase 1 than did the moderate response group (median, 7.5 sessions vs 18.5 sessions, respectively; $P = .004$). The 2 groups were nearly identical in number of sessions in phase 2, however, patients with best response mastered an average of 20.8 verb action icons, while those with moderate response mastered only 12 ($P = .02$). The number of sessions necessary to reach a common criterion point (12 verb action icons, a number reached by 12 of 13 of the patients) was a median of 28 sessions for best response and 69 for moderate response groups ($P = .004$).

**Table 4** shows that all patients in the best and moderate response groups had only unilateral left hemisphere lesion; the 4 patients with no response had bilateral lesions. (At the time the manuscript was in preparation, a new patient with bilateral lesions was observed to be performing well in phase 2 C-ViC training. The patient is a right-handed woman in her late 40s.) Subsequent testing on lesion site patterns was performed only between the best and moderate response patients.

Hypotheses were tested using discriminant function analysis, and resulting group identification decision matrices were tested for significance by means of the odds ratio (OR) method. The first hypothesis tested the validity of our original neuroanatomical model in which areas 1 and 2 were included. Because the extent-of-lesion data for area 2 were always in a categorical format (plus or minus), the extent-of-lesion data for area 1 were converted to a plus or minus. If the extent-of-lesion rating was greater than 3 (lesion in greater than half of the area), for either Wernicke area or the temporal isthmus area, then area 1
was rated a plus and a rating of 3 or less was rated a minus (Table 4, “Summary Area 1” column.)

The first discriminant analysis was performed by forcing in the categorical data for areas 1 and 2. While 7 of 7 patients with best response were correctly identified, 3 of 6 patients with moderate response (patients 10, 12, and 13) were misclassified as best response (odds ratio, 8; *P* = .20, 2-tailed) (Table 4, “Original Hypothesis, Correct Outcome Predicted?” column). All 7 best response patients had extensive lesion in only 1 of the 2 critical areas (Figure 4 and Figure 5).

The 3 of 6 patients with moderate response who were correctly identified each had extensive lesion in both areas 1 and 2 (Figure 6). Although there may be post hoc explanations for the 3 moderate response patients misclassified as best response (patients 10, 12, and 13; Table 4 footnotes), these results prompted additional discriminant analyses.

There were 2 concerns with the results from the first discriminant analysis based on the original hypothesis. First, the procedure weighted the Wernicke cortical area and the subcortical temporal isthmus area equally. Second, the extent-of-lesion ratings for these 2 temporal lobe structures (Wernicke area and the temporal isthmus area) were treated as categorical, although extent-of-lesion data were available for each area. Therefore, a second discriminant analysis was performed using 3 variables:

### Table 1. Patient Data*

<table>
<thead>
<tr>
<th>C-ViC Outcome Group</th>
<th>Patient No.</th>
<th>Age Entering C-ViC Treatment, y</th>
<th>Poststroke Onset Time Entering C-ViC Treatment</th>
<th>Pre-C-ViC BASA Score, Auditory Comprehension Raw Score (Maximum = 16)</th>
<th>Pre-C-ViC BASA Score, Oral/Gestural Raw Score (Maximum = 21)</th>
<th>Pre-C-ViC Overall BASA Score (Maximum = 61)</th>
<th>Phase 2 C-ViC Performance Rating (VICA Rating)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Best response</td>
<td>1</td>
<td>44</td>
<td>10 y</td>
<td>15</td>
<td>1</td>
<td>39</td>
<td>6.50</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>42</td>
<td>10 y</td>
<td>14</td>
<td>4</td>
<td>44</td>
<td>6.20</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>77</td>
<td>46 mo</td>
<td>10</td>
<td>6</td>
<td>39</td>
<td>5.77</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>53</td>
<td>21 mo</td>
<td>13</td>
<td>5</td>
<td>42</td>
<td>5.43</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>49</td>
<td>10 mo</td>
<td>16</td>
<td>12</td>
<td>49</td>
<td>4.93</td>
</tr>
<tr>
<td></td>
<td>6</td>
<td>69</td>
<td>19 mo</td>
<td>12</td>
<td>12</td>
<td>41</td>
<td>4.59</td>
</tr>
<tr>
<td></td>
<td>7</td>
<td>60</td>
<td>25 mo</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Moderate response</td>
<td>8</td>
<td>47</td>
<td>43 mo</td>
<td>12</td>
<td>43</td>
<td>43</td>
<td>4.40</td>
</tr>
<tr>
<td></td>
<td>9</td>
<td>69</td>
<td>77 mo</td>
<td>12</td>
<td>7</td>
<td>36</td>
<td>3.76</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>59</td>
<td>28 mo</td>
<td>12</td>
<td>11</td>
<td>36</td>
<td>3.59</td>
</tr>
<tr>
<td></td>
<td>11</td>
<td>56</td>
<td>7 mo</td>
<td>5</td>
<td>2</td>
<td>15</td>
<td>3.44</td>
</tr>
<tr>
<td></td>
<td>12</td>
<td>59</td>
<td>9 mo</td>
<td>7</td>
<td>2</td>
<td>27</td>
<td>3.03</td>
</tr>
<tr>
<td></td>
<td>13</td>
<td>64</td>
<td>26 mo</td>
<td>9</td>
<td>6</td>
<td>30</td>
<td>2.73</td>
</tr>
<tr>
<td>No response</td>
<td>14</td>
<td>73</td>
<td>3 mo</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA, N/A</td>
</tr>
<tr>
<td></td>
<td>15</td>
<td>70</td>
<td>14 mo</td>
<td>8</td>
<td>6</td>
<td>30</td>
<td>CND</td>
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<tr>
<td></td>
<td>16</td>
<td>75</td>
<td>7 mo</td>
<td>8</td>
<td>2</td>
<td>20</td>
<td>CND</td>
</tr>
<tr>
<td></td>
<td>17</td>
<td>65</td>
<td>13 mo</td>
<td>12</td>
<td>7</td>
<td>41</td>
<td>CND</td>
</tr>
</tbody>
</table>

* C-ViC indicates computer-assisted visual communication treatment program; BASA, Boston Assessment of Severe Aphasia examination; VICA, Visual Index of Communicative Ability; NA, no data available; and CND, could not do C-ViC. Patients are rank ordered according to their phase 2 C-ViC performance rating.
Figure 3. Location of specific neuroanatomical areas on computed tomographic scan that were examined for presence or absence of lesion, and extent of lesion within that area. Top, Lateral view; bottom, axial view (15° to canthomeatal line) for slices B, B/W, W, SM, and SM + 1. The computed tomographic scan slices are labeled with reference to specific cortical areas present on each slice. B indicates Broca; B/W, Broca and Wernicke; W, Wernicke; and SM, supramarginal gyrus. Each neuroanatomical area on each slice was visually assessed for extent of lesion using a 6-point rating scale (where 0 indicates no lesion; 3, half of area has lesion; 5, entire area has lesion [see text for further explanation]). The areas most relevant to this study were the following: W, Wernicke area; Ti, subcortical, anterior temporal isthmus area; MScF, medial subcallosal fasciculus area; M 1⁄3 PVWM, middle one third periventricular white matter area (see text for additional explanation of these areas). GP indicates globus pallidus; ALIC, anterior limb, internal capsule; P, putamen; C, head of caudate; I, insular structures; T, temporal lobe; PLIC, posterior limb, internal capsule; P-M, premotor; ASm, anterior supramarginal; PSm, posterior supramarginal; and Ang, angular gyrus.
Wernicke area and the temporal isthmus area as 2 separate quantitative variables, and the SMA or cingulate gyrus area 24 as 1 categorical variable. When these 3 structures were each considered, 1 patient with best response was misclassified (patient 3) and 2 patients with moderate response were misclassified (patients 12 and 13) (OR, 12; \( P = .10 \), 2-tailed).

Two additional discriminant analyses were performed with the last 3 variables plus an additional, extent-of-lesion variable. When lesion for the middle 1/3 PVWM area was added to the 3 variables, the discriminant analysis became less reliable, misclassifying 4 subjects—2 with best response (patients 3 and 6) and 2 with moderate response (patients 12 and 13) (OR, 6; \( P = .22 \)). However, when lesion for the medial subcallosal fasciculus area was forced in with the 3 variables (Wernicke area, temporal isthmus area, and SMA or cingulate gyrus area 24), the prediction rate became highly significant, misclassifying only 2 subjects—one with best response (patient 3) and 1 with moderate response (patient 12) (OR, 30; \( P = .03 \)).

Each patient with no response had bilateral lesions. Analysis of the left hemisphere lesion alone would have predicted best response for patients 15, 16, and 17 (extensive lesion only in area 1), and moderate response for patient 14 (areas 1 and 2). With additional right hemisphere lesion, the predicted effect of the left hemisphere lesion site patterns alone was not valid.

Patients who have no recovery of meaningful spontaneous speech usually have summed extent-of-lesion rating greater than 7 for the medial subcallosal fasciculus area plus the middle 1/3 PVWM area. Table 4 shows that 15 (88%) of the 17 patients in the present study had summed extent-of-lesion ratings greater than 7.0 (range, 7.1-9.9). The 2 patients who had ratings of less than 7.0, compatible with recovery of nonfluent speech, were patient 5 (4.99) and patient 6 (3.5). Patient 5 recovered to a phrase length of 3 words (he received some therapy focusing on improving verbal expression concurrently with C-ViC); patient 6 had no recovery of speech.

Unexpectedly, 2 other patients did recover some speech, despite summed extent-of-lesion ratings greater than 7.0, compatible with no recovery of speech (patient 1, 9.9; patient 3, 9.27). Patient 1 was transferred to a verbal treatment program and has a phrase length of 5 to 6 words; patient 3 has a phrase length of 3 words.

Overall, 14 (82%) of the 17 patients had the level of speech expected from their lesion site patterns. Most patients (15/17 [88%]) had summed extent-of-lesion ratings compatible with absence of recovery of speech, and many of these latter patients (13/15 [87%]) were appropriate candidates for nonverbal C-ViC training.

There was no significant difference in lesion size between best response (mean [SD], 10.03% [4.2%]) and moderate response (mean [SD], 16.9% [8.52%]). In addition, there was no significant difference in lesion size between no response (mean [SD], 14.7% [3.8%]) and either of the others.

Most patients (15 of 17) had typical left occipital length asymmetry. The single patient with atypical right occipital length asymmetry (patient 1) had an unexpected recovery of nonfluent speech, despite a

**Table 2. Unpaired t Test Results for Best Response Group vs Moderate Response Group Regarding C-ViC Treatment Time and BASA Scores Before Entering C-ViC Treatment**

<table>
<thead>
<tr>
<th></th>
<th>Best Response Group</th>
<th>Moderate Response Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Range</td>
</tr>
<tr>
<td>Age entering C-ViC, y</td>
<td>56.3 (13.1)</td>
<td>42-77</td>
</tr>
<tr>
<td>Poststroke onset entering C-ViC, mo †</td>
<td>25</td>
<td>10-120</td>
</tr>
<tr>
<td>Total time in C-ViC treatment, wk</td>
<td>42.1 (38.5)</td>
<td>4-99</td>
</tr>
<tr>
<td>Pre-C-ViC BASA scores</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Auditory comprehension</td>
<td>13.3 (2.2)</td>
<td>10-16</td>
</tr>
<tr>
<td>Oral/gestural</td>
<td>7.2 (4.3)</td>
<td>1-12</td>
</tr>
<tr>
<td>Overall score</td>
<td>42.3 (3.8)</td>
<td>39-49</td>
</tr>
</tbody>
</table>

*C-ViC indicates computer-assisted visual communication treatment program; BASA, Boston Assessment of Severe Aphasia examination.
†The value reported here is a median: the Mann-Whitney U test is reported.

**Table 3. Mann-Whitney U Test Results for Best Response Group vs Moderate Response Group Regarding Phase 1 and Phase 2 C-ViC Training**

<table>
<thead>
<tr>
<th></th>
<th>Best Response Group</th>
<th>Moderate Response Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Median (Range)</td>
<td>Median (Range)</td>
</tr>
<tr>
<td>Total No. of sessions to complete phase 1 C-ViC training</td>
<td>7.5 (3-14)</td>
<td>18.5 (11-22)</td>
</tr>
<tr>
<td>Total No. of sessions to complete phase 2 C-ViC training</td>
<td>66 (13-126)</td>
<td>78 (47-94)</td>
</tr>
<tr>
<td>Total No. of verb action icons learned in C-ViC training</td>
<td>20.8 (15-23)</td>
<td>12 (12-20)</td>
</tr>
<tr>
<td>No. of sessions to learn 12 verb action icons in phase 2</td>
<td>28 (9-37)</td>
<td>69 (42-82)</td>
</tr>
</tbody>
</table>

*C-ViC indicates computer-assisted visual communication training program.
Table 4. Lesion Site Data for 17 Patients With Severe Aphasia Treated With the C-ViC Program*

<table>
<thead>
<tr>
<th>C-ViC Outcome Group</th>
<th>Patient No.</th>
<th>Time CT Scan Taken After Stroke, mo</th>
<th>Right Hemisphere Lesion</th>
<th>Area 1 (Temporal Lobe)</th>
<th>Area 2: SMA/Cingulate Gyrus Area 24</th>
<th>Original Hypothesis, Correct Outcome Predicted?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Best response</td>
<td>1</td>
<td>21</td>
<td>-</td>
<td>1.5</td>
<td>5</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>9 y 9 mo</td>
<td>-</td>
<td>1</td>
<td>1.75</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>17</td>
<td>-</td>
<td>4.55</td>
<td>5</td>
<td>-</td>
</tr>
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<td>+</td>
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<tr>
<td></td>
<td>5</td>
<td>14</td>
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* C-ViC indicates computer-assisted visual communication; CT, computed tomographic; SMA, supplementary motor area; MScF, medial subcallosal fasciculus; Middle ½ PVWM, middle one third periventricular white matter area; plus sign, a visible lesion detected; minus sign, no visible lesion detected; R, right; L, left; and equal sign, no asymmetry.
† Extent-of-lesion ratings were based on a 6-point scale where 0 indicates no lesion and 5 indicates entire area has lesion.
‡ Small right frontal, small right parietal.
§ Right lower motor cortex, right lacune, anterolateral to the frontal horn.
¶ Right frontal lacune, right extreme capsule, and claustrum.
# Best response (BR) predicted. Left occipital lesion, not present in other patients.
** Flat affect, severe depression, not present in other patients, never showed emotional improvement following mastery of phase 1 C-ViC training.
†† Medically ill, multiple infections; died a few months after discharge from phase 2 C-ViC training.
‡‡ No lesion in the MScF area; however, an extensive lesion was present at the origin of this pathway (SMA/cingulate gyrus area 24). Thus, the lesion extent rating of 5 was used here.

The BASA scores obtained before C-ViC training were available for 6 patients with best response, 6 with moderate response, and 3 with no response (Table 1). The best response group had significantly better BASA auditory comprehension subscores (P = .03) and overall BASA scores (P = .02) than the moderate response group (Table 2). There were too few patients in the no response group for statistical comparisons.

The BASA scores were subjected to discriminant function analyses for best response and moderate response to determine whether they could be used to predict C-ViC outcome. When the auditory comprehension subscore was entered, 4 patients were misclassified: 1 with best response (patient 3) and 3 with moderate response (patients 8, 9, and 10) (OR, 5; P = .54). When the oral-gestural communication subscore was entered, 6 patients were misclassified: 3 with best response (patients 1, 3, and 5) and 3 with moderate response (patients 8, 9, and 10) (OR, 1; P = .99). When the overall BASA score was entered, however, only 1 patient was misclassified, patient 8 from the moderate response group (OR, 25; P = .04).

The median cutoff overall BASA score for best response was 38 (range, 39-49 of a possible 61). Five of the 6 patients with moderate response had overall BASA scores of less than 38 (range, 15-36; 1 patient had a score of 43 [patient 8]) (Table 1).

The 3 patients with no response had overall BASA scores that were within the range of both best and moderate response. Patient 17 had a BASA score of 41 (best response, >38), and patients 15 and 16 each had overall BASA scores of 20 and 30, respectively (5 of 6 patients with moderate response had scores ranging from 15 to 36).

**COMMENT**

This study tested the validity of a previously identified lesion site hypothesis to predict C-ViC outcome. When this original hypothesis was modified to include not only the 2 original areas—area 1 temporal lobe structures (Wernicke area and the temporal isthmus area) and area 2 supraventricular frontal lobe structures (SMA or cingulate gyrus area 24)—but also the medial subcallosal fasciculus area, 6 of 7 patients with best response and 5 of 6 with moderate response were correctly classified with discriminant function analysis.

The effect of the additional factor, extent-of-lesion within the medial subcallosal fasciculus area, is not completely understood in relationship to C-ViC outcome. For example, 5 of 7 patients with best response and 5 of 6 patients with moderate response had extensive lesion in the medial subcallosal fasciculus area. However, 2 pa-
tients with best response had no lesion in this area and this factor may have been considered important in the discriminant analysis. Lesion in the medial subcallosal fasciculus area could have an additive effect to interruption of the initiation/limbic pathways from the SMA or cingulate gyrus area 24, to the head of the caudate, deep to the Broca area. However, since 5 patients with extensive lesion in this area had best response, it would be difficult to apply this additional lesion factor in a practical manner in the clinic, when predicting C-ViC outcome.

The original lesion site pattern associated with best response (ability to initiate communication with C-ViC) is one that spares large portions of either the posterior systems or the anterior systems involved in language recovery. Sparing of posterior systems, including area 1 (Wernicke cortical area or the temporal isthmus area), may allow enough preservation of left hemisphere structures that pictorial representations can gain access to semantic meaning. Sparing of anterior systems, including area 2 (SMA or cingulate gyrus area 24), may allow enough frontal callosal pathways to be preserved that the overall frontal capacity to learn and execute a novel system is possible.28

It is also possible that sparing of anterior systems has a specific initiation/limbic effect.29 If enough frontal me-
dial limbic structures (including the SMA) are preserved, then patients can probably initiate a semantic system if they have one that is intact (perhaps either in the left posterior temporoparietal area or the right hemisphere). That patients with moderate response could use C-ViC to respond to questions but not to initiate interaction suggests that moderate response was related more to initiation and utilization rather than to semantic production capacity. This would be compatible with an “initiation/limbic” explanation, not a frontal “capacity” account.

The overall BASA score before C-ViC treatment also showed significance in predicting C-ViC outcome, where 6 of 6 patients with best response and 5 of 6 patients with moderate response were correctly predicted. The incorrect prediction involved moderate response in patient 8, whose overall BASA score was 43 (a score of >38 is compatible with best response). This moderate response outcome for patient 8 was correctly predicted, however, from the lesion site data using the original hypothesis. The original hypothesis lesion site data had misclassified 3 patients with moderate response as best response; however, their BASA scores were less than 38 (patient 10, 36; patient 12, 27; and patient 13, 30) and thus compatible with the observed moderate response.

A practical clinical method of predicting C-ViC outcome is proposed from these 2 data sets combined. If both BASA data (overall score, >38) and lesion site data (extensive lesion, area 1 or 2) are compatible with best response, then best response is likely. If both BASA data (overall score, <38) and lesion site data (extensive lesion, areas 1 and 2) are compatible with moderate response, then moderate response is likely. However, if either of these data sets (lesion site data or overall BASA score) is compatible with only moderate response, then moderate response is likely. Both neuroanatomical and behavioral data seem to be necessary for optimal prediction.

This study also examined lesion site patterns in relationship to absence of recovery of speech, thus, likely candidacy for entry into C-ViC. The basic lesion site pattern associated with absence of recovery of speech (summed extent-of-lesion ratings of >7.0 for medial subcallosal fasciculus plus the middle 1/3 PVWM) was observed in 15 (88%) of the 17 patients referred for C-ViC; 13 (87%) of these 15 patients did not recover speech. Most patients without speech who have bilateral lesions do not seem to be good candidates for C-ViC training, although some exceptions will occur.

In addition, the issue of hemispheric asymmetries (potential for anomalous dominance) was examined. One pa-

Figure 5. Computed tomographic scan at 9 years 9 months after stroke onset in a patient (patient 2) with best response who entered the computer-assisted visual communication (C-ViC) program at 10 years after stroke. An extensive lesion was present only in 1 of the 2 areas relevant to outcome level with C-ViC training. An extensive lesion was present in area 2 (supraventricular frontal lobe structures, supplementary motor area or cingulate gyrus area 24, deep white matter). See slices SM + 2 and SM + 3 (black-and-white arrows). A lesion was present in the temporal lobe structures, only small, patchy lesion was present in the temporal isthmis on slice B. Only minimal, equivocal lesion was present in the Wernicke cortical area, located lateral to the third ventricle (slices B/W and W). The lesion site pattern associated with no recovery of spontaneous speech and appropriate candidacy for C-ViC training was present, eg, an extensive lesion in the medial subcallosal fasciculus area, adjacent and anterolateral to the left frontal horn, on slices B and B/W (black arrows), and in the middle one third periventricular white matter area, adjacent and lateral to the body of the lateral ventricle, on slice SM (black arrow). For an explanation of abbreviations, see the legend to Figure 3.

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tient (patient 1) had atypical right occipital length asymmetry and recovery of some nonfluent speech, not expected from his lesion site pattern. This phenomenon was also observed in a previous study, in which 1 patient recovered nonfluent speech, despite a lesion site pattern compatible with no recovery of speech. That patient (B.J.) was left-handed, aphasic from a left hemisphere lesion, and had equal occipital length on CT scan. These 2 patients, however, were young at stroke onset, ages 34 (patient 1) and 43 years (B.J.), and each also had lesion sites compatible with best response. There may have been multiple factors that contributed to their better recovery.

This unusual recovery of some speech may be related to a gradual involvement of the right hemisphere. Recovery of word comprehension in global aphasia may continue for years, and it has been hypothesized that this recovery may reflect gradual involvement of right hemisphere semantic systems in comprehension in chronic aphasia. A gradual pattern of improvement in naming ability (despite left hemisphere lesion site expansion) after 5 to 15 years after stroke also has been observed. The systems involved in this long-term recovery are not known. A recent positron emission tomographic study of recovery in Wernicke aphasia served a bilateral network to be important in functional reorganization of language.

Patients with nonverbal aphasia who have severe deficits at 1 month after onset of stroke have a bleak prognosis for recovery of useful language output. The results from the present study suggest that if a patient with severe aphasia has had no recovery of meaningful spontaneous speech by 3 months after stroke, a noncontrast CT scan plus evaluation with the BASA examination are likely to provide predictive information that may be useful for long-term treatment planning. However, it has been our clinical experience with more than 30 patients with severe nonverbal aphasia that many are not ready for C-ViC until approximately 9 months after stroke.

Computed tomographic scans are currently our first choice for structural imaging where lesion site analysis regarding potential for long-term recovery and treatment planning will be performed. We have attempted to apply our lesion site analysis to magnetic resonance scans. However, the T1-weighted magnetic resonance images tend to underestimate the extent of the lesion near ventricle, and the T2-weighted magnetic resonance images tend to overestimate the extent of the lesion near ven-
tricle, compared with CT images. The lesion site analysis used in this research requires analysis of white matter areas adjacent to ventricle (at the frontal horn and body of the lateral ventricle). Therefore, lesion site analysis on CT scans is preferred.

A study on cost-effectiveness of C-ViC and impact on functional communication in severe aphasia is in progress. There are currently 5 patients using C-ViC outside the therapy setting. The C-ViC computer program is in the public domain and available through one of us (E.H.B.). Cost of a personal computer (<$2000) and cost of twice-weekly treatment sessions for a 6- to 12-month period comprise the major costs. The refinement provided in the present study (C-ViC candidacy and predicted outcome) suggests that future cost savings could be appreciated with patients with severe nonverbal aphasia, in part, through timely intervention with an appropriate nonverbal treatment program likely to promote increased communication, vs long-term intervention with inappropriate verbal treatment programs where nonincreased communication is likely.

The role of augmentative and alternative communication devices in rehabilitation of patients with severe aphasia has been reviewed by Kraat. She suggests these devices show promise, but direct application needs refinement. The present study is one approach toward that necessary refinement.

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