APHASIA IN ACUTE STROKE/Brust et al. 167

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Aphasia in Acute Stroke

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SUMMARY Previous surveys of stroke populations have offered only cursory information on language disturbance, and, conversely, few surveys of aphasic populations have dealt exclusively with stroke or with acute phenomena. This paper describes aphasia in 850 acute stroke patients consecutively registered by the Harlem Regional Stroke Program, of whom 177 (21%) were aphasic; of these, nine were of Broca's type, 24 were of Wernicke's type, 14 were anomia, ten were conduction, seven were of "isolation" type, and 107 were "mixed." An unexpected finding was a significant overrepresentation of men among the nonfluent aphasics.

During the following four to 12 weeks, 12% of fluent aphasics died, and 12% remained moderately or severely impaired; among survivors, aphasia improved in 74%, and in 44% it cleared completely. During the same period, 32% of nonfluent aphasics died, and 34% remained moderately or severely impaired; among survivors, aphasia improved in 52%, and in only 13% did it clear completely. In both fluent and non fluent groups, hemiparesis and/or visual field cut were associated with poor prognosis.

Introduction

APHASIA is a common accompaniment to stroke, and is often the most disabling sequela. Yet surveys of stroke populations have tended to offer limited information as to the type and degree of aphasia present. Conversely, aphasia studies have seldom involved only stroke patients, or patients acutely affected. The present study analyzes the incidence of aphasia types among acute stroke patients seen at Harlem Hospital, and relates the clinical findings to early prognosis.

Methods

From 1971 through 1973, 850 patients were admitted to Harlem Hospital with the diagnosis of acute stroke. Criteria for inclusion in the registry of the Harlem Regional Stroke Program have been previously described. Upon admission the patient was examined by members of the Medical House Staff, plus a neurology consultant. In addition, within 12 to 48 hours and then at periodic intervals an examination was performed by a member of the Stroke Program Team and recorded on standardized forms. The data of this report are

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Reprint requests to Dr. Brust, Harlem Hospital Center.
based upon review of both the Stroke Program forms and the patients' hospital records.

Spontaneous speech was studied for fluency, articulation, the presence of paraphasia, alterations in grammar, and the degree of comprehensibility of the patient's speech to the examiner. Nonfluent speech was defined as an abnormal decrease in the number of words used per minute, with hesitations on initiating speech and abnormalities of rhythm; also there was usually impaired prosody, the infection and "music" of speech. Comprehensibility could be limited in severely nonfluent speech simply because of the limited number of words used, or because of poor articulation. Comprehensibility could be quite adequate despite severe nonfluency, the words sometimes consisting largely of substantives delivered "telegraphically." With fluent speech the limiting factor to comprehensibility was often the number of verbal or literal paraphasias, which, when dominating speech, produced incomprehensible jargon.

The patient's own comprehension of spoken speech was tested by giving commands plus asking the patient to answer obvious questions with yes or no and to indicate objects named by the examiner. Ability to name was assessed for objects, body parts, and, in many cases, colors. Ability to repeat was tested with a simple phrase such as: "Today is a very nice day," plus a syntactically loaded phrase such as: "No ifs, ands, or buts." Writing was tested mainly to dictate; reading was tested aloud and for gross comprehension.

Fluency, auditory comprehension, naming, and repetition were judged to be normal, mildly abnormal, or severely abnormal based on the subjective impression of the examiner. More quantitative assessment was not attempted. Aphasia was called mild, moderate, or severe based on combinations of these features, especially speech comprehension, plus the degree to which the patient's speech could be understood. With Broca's aphasics severity was based upon speech output alone.

The aphasia classification of Geschwind was used because of the simplicity and brevity of testing required. Broca's aphasia is defined in terms of nonfluency not explained by anoma, and with comprehension preserved.

**Table 1** Aphasia Findings in 850 Acute Stroke Patients, by Type

<table>
<thead>
<tr>
<th>Finding</th>
<th>No.</th>
<th>% of specified group</th>
<th>% of all aphasics</th>
<th>% of all pts.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fluent aphasia</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wernicke</td>
<td>24</td>
<td>13.6</td>
<td>2.8</td>
<td></td>
</tr>
<tr>
<td>Conduction</td>
<td>10</td>
<td>5.6</td>
<td>1.2</td>
<td></td>
</tr>
<tr>
<td>Anomic</td>
<td>10</td>
<td>5.6</td>
<td>1.2</td>
<td></td>
</tr>
<tr>
<td>Isolation</td>
<td>7</td>
<td>3.9</td>
<td>0.8</td>
<td></td>
</tr>
<tr>
<td>Uncertain</td>
<td>6</td>
<td>3.4</td>
<td>0.7</td>
<td></td>
</tr>
<tr>
<td>Group total</td>
<td>57</td>
<td>32.1</td>
<td>6.7</td>
<td></td>
</tr>
<tr>
<td>Nonfluent aphasia</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mixed</td>
<td>107</td>
<td>60.5</td>
<td>12.6</td>
<td></td>
</tr>
<tr>
<td>Broca</td>
<td>9</td>
<td>5.1</td>
<td>1.1</td>
<td></td>
</tr>
<tr>
<td>Anomic</td>
<td>4</td>
<td>2.3</td>
<td>0.5</td>
<td></td>
</tr>
<tr>
<td>Group total</td>
<td>120</td>
<td>67.9</td>
<td>14.1</td>
<td></td>
</tr>
<tr>
<td>Total of aphasics</td>
<td>177</td>
<td>–</td>
<td>20.8</td>
<td></td>
</tr>
<tr>
<td>No aphasia</td>
<td>673</td>
<td>–</td>
<td>79.2</td>
<td></td>
</tr>
<tr>
<td>Total pts.</td>
<td>850</td>
<td>–</td>
<td>100</td>
<td></td>
</tr>
</tbody>
</table>

Other features, e.g., poor articulation, agrammatism, "recurring utterance," or relative preservation of emotional speech, may be present. Fluent aphasia subtypes are based on combinations of abnormal naming, comprehension, and repetition. In Wernicke's aphasia comprehension and repetition are both impaired to the same degree. In anomic aphasia they are both preserved. In conduct aphasia repetition is a good deal more abnormal than comprehension. In "isolation of the speech area" aphasia (transcortical sensory aphasia) the reverse holds.

We are aware that this classification represents an oversimplification of a probably far more heterogeneous population, and that classifications based on broader psychological testing may be closer to the truth. In a city hospital with frequent emergency admissions a system to be useful has to be short and straightforward.

**Results**

Table 1 shows that of 850 total stroke patients, 177 (21%) had aphasia acutely. In 57 (32%) speech was fluent, and in 120 (68%) it was nonfluent.

In nine of the nonfluent patients, gross comprehension was preserved, and other features characteristic of Broca's aphasia were present. These additional features, however, varied markedly from one patient to the next. In most of the Broca aphasics naming was only mildly impaired and repetition was normal. (Those with preserved repetition would be classified by some as "transcortical motor aphasia."")

Four patients had hesitant nonfluent speech with normal comprehension, but here nonfluency seemed secondary to impaired naming, other features suggesting Broca's aphasia were absent, and the patients were therefore classified as nonfluent anomics.

One hundred seven patients had mixed aphasia with nonfluent speech plus impaired comprehension, mild in 35 and severe in 72 (global aphasia). Of the 35 with comprehension only mildly impaired, naming and repetition were either normal or only mildly impaired in 16; six had severely impaired repetition and thus appeared to have combined features of Broca's aphasia and conduct aphasia. Of the 72 mixed aphasics with comprehension severely impaired, naming was, surprisingly, only mildly impaired in six. The rest had either severe impairment of both naming and repetition, or else one or the other modality was not tested, usually because the patient would not attempt the task.

Twenty-four of the fluent aphasics met the criteria for Wernicke's aphasia; 13 of these were severely affected and 11 only mildly affected. Ten patients had conduct aphasia; eight had severe and two mild impairment of repetition, but in only one was repetition severely impaired in the

**Table 2** Average Ages of Fluent and Nonfluent Aphasics and of Nonaphasics

<table>
<thead>
<tr>
<th></th>
<th>Years</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fluent</td>
<td>65</td>
<td>40-100</td>
</tr>
<tr>
<td>Nonfluent</td>
<td>68</td>
<td>33-94</td>
</tr>
<tr>
<td>Broca's</td>
<td>60</td>
<td>33-76</td>
</tr>
<tr>
<td>Anomics</td>
<td>62</td>
<td>50-77</td>
</tr>
<tr>
<td>Nonaphasics</td>
<td>65</td>
<td>28-95</td>
</tr>
</tbody>
</table>
presence of normal comprehension. Ten patients had fluent
anomia, mild in nine. Seven patients had "isolation of the
speech area" aphasia; in four of these there was severely im-
paired comprehension, but in only one was it accompanied
by entirely normal repetition, and in none was there frank
echolalia. In six of those with fluent speech, repetition was
not assessed, and their aphasia types therefore remain uncertain.

As with the nonfluent group, naming in the fluent patients
often failed to parallel either comprehension or repetition.
Moreover, the speech output within each of the fluent
groups varied widely, especially among the Wernicke
aphasics, in whom it ranged from intelligible speech with
preserved patient insight to either verbal or neologicistic
jargon and anosognosia.

Table 2 shows no significant difference in average age
between fluent and nonfluent aphasics or between aphasics
and nonaphasics. Table 3, however, shows that men were
significantly (Chi-square corr = 19.06, P < 0.001) more com-
mon in the nonfluent group. Seventy-five percent of all aphasics were either moderately or severely impaired, and this degree of impairment was significantly (Chi-square corr = 19.06, P < 0.001) more common in the nonfluent group.

Table 7 shows that at 4 to 12 weeks seven (12%) of the
fluent aphasics had died and seven (12%) were still moderatly or severely impaired. Of 50 survivors at 4 to 12
weeks, aphasia improved in 37 (74%), and in 22 (44%) it

cleared completely. Twelve of the 38 nonfluent aphasics who
died had bloody CSF on admission. Six had moderate to
severe hemiparesis, and five had a visual field cut. Conversely, all those without hemiparesis and those without
a field cut survived.

Table 8 shows that at 4 to 12 weeks 38 (32%) of the non-
fluent aphasics had died and 41 (34%) were still moderately or severely impaired. Of 82 survivors at 4 to 12
weeks, aphasia improved in 45 (55%), but in only 11 (13%) did it
clear completely. Twelve of the 38 nonfluent aphasics who
died had bloody CSF. Thirty-four had moderate to severe
hemiparesis, and 20 had a visual field cut. (Only one appeared to have normal visual fields; 17 were uncertain.)

Conversely, the six nonfluent aphasics without hemiparesis
(including one pure anomia) survived, as did 26 of the 27
lacking a visual field cut.

Thus, nonfluency carried a significantly (Chi-
square corr = 6.67, P < 0.01) worse prognosis for mortality,
and within both the nonfluent and fluent groups mortality
was associated with hemiparesis and/or visual field cut.

One nonfluent patient with a mild deficit on admission
became moderately impaired following a second stroke. Of
always run in parallel. Severity was graded on admission ex-
cept for several patients initially stuporous or mute who
became testable within a few days. (An interesting exception
was a man who remained mute up to his discharge after
many weeks. He showed poor auditory comprehension,
writing, and reading, but was cooperative and had relatively
preserved non-language mental functioning. He has been in-
cluded in the mixed aphasia group.)

Table 6 records severity of aphasia on admission. Seventy-five percent of all aphasics were either moderately or severely impaired, and this degree of impairment was significantly (Chi-square corr = 19.06, P < 0.001) more common in the nonfluent group.

Table 4 Hemiparesis in Fluent and Nonfluent Aphasics

<table>
<thead>
<tr>
<th></th>
<th>Moderate to severe</th>
<th>Mild</th>
<th>None</th>
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<tbody>
<tr>
<td>Fluent</td>
<td>17(30%)</td>
<td>25(44%)</td>
<td>15(26%)</td>
</tr>
<tr>
<td>Non Fluent</td>
<td>97(81%)</td>
<td>17(14%)</td>
<td>6(3%)</td>
</tr>
<tr>
<td>Broca's</td>
<td>77(78%)</td>
<td>2(22%)</td>
<td>0</td>
</tr>
<tr>
<td>Anomics</td>
<td>2(30%)</td>
<td>1(25%)</td>
<td>1(25%)</td>
</tr>
<tr>
<td>Mixed</td>
<td>88(80%)</td>
<td>14(43%)</td>
<td>3(3%)</td>
</tr>
<tr>
<td>Total</td>
<td>114(64%)</td>
<td>42(24%)</td>
<td>21(12%)</td>
</tr>
</tbody>
</table>

Table 5 Homonymous Hemianopia in Fluent and Nonfluent Aphasics

<table>
<thead>
<tr>
<th></th>
<th>Present</th>
<th>Absent</th>
<th>Uncertain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fluent</td>
<td>18(32%)</td>
<td>24(42%)</td>
<td>15(26%)</td>
</tr>
<tr>
<td>Non Fluent</td>
<td>66(55%)</td>
<td>27(23%)</td>
<td>27(22%)</td>
</tr>
<tr>
<td>Broca's</td>
<td>4(44%)</td>
<td>5(56%)</td>
<td>-</td>
</tr>
<tr>
<td>Anomics</td>
<td>3(75%)</td>
<td>1(25%)</td>
<td>-</td>
</tr>
<tr>
<td>Mixed</td>
<td>5(56%)</td>
<td>21(20%)</td>
<td>27(25%)</td>
</tr>
<tr>
<td>Total</td>
<td>84(47%)</td>
<td>51(29%)</td>
<td>42(24%)</td>
</tr>
</tbody>
</table>
the two mildly impaired patients who died, one had a second stroke and the other a myocardial infarction.

Of the 82 survivors from the nonfluent group 52 (63%) were still nonfluent at 4 to 12 weeks. While speech became fluent in one Broca’s aphasic, there persisted mild dysarticulation and anomia.

Only 20 fluent and 33 nonfluent patients had reading and writing checked. Many patients would not attempt either task. The great majority of those checked were impaired in both skills. One fluent and two nonfluent patients had grossly normal reading and writing, but extensive testing to detect fine abnormalities was not done.

**Discussion**

The incidence of symptoms and signs has been reported in a number of large stroke populations. Details on language function seldom have been given, however. For example, Marquardson’s retrospective study of 769 acute stroke patients noted that 133 patients, or 33% of the immediate survivors, were aphasic. The only elaboration offered was that aphasia was usually a “mixed amnestic deficit,” and “severe” in two-thirds. In 106 cases aphasia was combined with hemiparesis. Seventy-one of 88 patients followed improvement did not occur. Indeed, Benson and Geschwind have stressed the unreliability of classifying patients too early after an acute insult, since a particular type of aphasia may evolve only after weeks or months.

Of two reports on the natural history of stroke in Rochester, Minnesota, one, analyzing patients from 1945 through 1954, did not state the incidence of aphasia. The other, dealing with the years 1955 through 1969, noted that 10% of survivors at six months were aphasic and that each of these “also had other disabilities.” The incidence of aphasia acutely was not defined, nor was there any breakdown as to the type of language disturbance later present.

Omae’s et al. study from Japan was limited to patients with cerebral infarction. Aphasia was present in 23 of 98 cases (24%), and was assessed by the short Minnesota Test. Modifying Schuell’s classification, the authors considered four patients to have “simple” aphasia, three with “aphasia with scattered lesions,” six with “irreversible aphasia,” two with “partial auditory imperception,” five with “mild aphasia with persisting dysarthria,” and two with “severe aphasia unclassifiable” because of confusion. Further explanation of these types was not given.

David and Heyman’s report of 100 consecutive cerebral infarctions noted that 48 cases were in the carotid territory, 32 were in the vertebrobasilar territory and 20 were “uncertain;” aphasia frequency was not given. Robinson’s et al. study of the natural history of cerebral thrombosis in Worcester, Massachusetts, listed aphasia as a “minor deficit” (examples of severe deficit were hemiplegia or hemiparesis), and did not give aphasia incidence, acutely or chronically. Gurdjian’s et al. analysis of 600 stroke patients similarly treated aphasia in a cursory fashion.

There have been, conversely, many series of aphasics, but they have not concerned stroke patients exclusively. Head based his elaborate aphasia classification on only 26 patients, most of whom had sustained gunshot wounds and were examined after their symptoms had stabilized. Investigators who have similarly studied largely or entirely head injury cases include Kleist, Goldstein, Schiller, Wepman, Russell and Espir, Hecaen and Angelergues, and Luria. Studies based mainly on stroke patients, but including cases of head trauma or neoplasm, include those of Weisenberg and McBride, Alajouanine, Schuell, Jenkins and Jimenez-Pabon, Marks, Taylor and Rusk, and Brown and Simonson. Sarno et al. evaluated 31 stroke patients, but deliberately selected only those severely affected. Moreover, aphasia studies, whether with stroke patients or otherwise, have largely been conducted weeks, months, or years after the acute insult. Weisenberg and McBride’s patients, for example, were studied from two days to ten years afterward, with an average of several months. Wepman’s cases were all assessed after six months because of the authors’ belief that at that time spontaneous improvement did not occur. Indeed, Benson and Geschwind have stressed the unreliability of classifying patients too early after an acute insult, since a particular type of aphasia may evolve only after weeks or months.

<table>
<thead>
<tr>
<th>Aphasia Severity on Admission</th>
<th>Severe</th>
<th>Moderate</th>
<th>Mild</th>
<th>Cleared</th>
<th>Died</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fluent</td>
<td>13(23%)</td>
<td>16(28%)</td>
<td>28(49%)</td>
<td>24(39%)</td>
<td>7(12%)</td>
</tr>
<tr>
<td>Nonfluent</td>
<td>49(86%)</td>
<td>21(30%)</td>
<td>1(2%)</td>
<td>3(5%)</td>
<td>2(3%)</td>
</tr>
<tr>
<td>Broca’s</td>
<td>1(2%)</td>
<td>3(5%)</td>
<td>1(2%)</td>
<td>2(3%)</td>
<td>0</td>
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<tr>
<td>Anomia</td>
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<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Mixed</td>
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<td>2(5%)</td>
<td>2(5%)</td>
<td>14(13%)</td>
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</tr>
<tr>
<td>Total</td>
<td>84(47%)</td>
<td>49(28%)</td>
<td>14(13%)</td>
<td>10(5%)</td>
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</table>

<table>
<thead>
<tr>
<th>Aphasia Severity on Admission</th>
<th>Severe</th>
<th>Moderate</th>
<th>Mild</th>
<th>Cleared</th>
<th>Died</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fluent</td>
<td>1(20%)</td>
<td>2(40%)</td>
<td>2(40%)</td>
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<td>0</td>
</tr>
<tr>
<td>Nonfluent</td>
<td>21(42%)</td>
<td>21(42%)</td>
<td>5(10%)</td>
<td>4(8%)</td>
<td>2(4%)</td>
</tr>
<tr>
<td>Broca’s</td>
<td>0</td>
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<td>2(40%)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Anomia</td>
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<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>2(4%)</td>
<td>4(8%)</td>
<td>2(4%)</td>
<td>4(8%)</td>
<td>2(4%)</td>
</tr>
</tbody>
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<table>
<thead>
<tr>
<th>Aphasia Severity on Admission</th>
<th>Severe</th>
<th>Moderate</th>
<th>Mild</th>
<th>Cleared</th>
<th>Died</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fluent</td>
<td>1(4%)</td>
<td>1(4%)</td>
<td>3(12%)</td>
<td>1(4%)</td>
<td>0</td>
</tr>
<tr>
<td>Nonfluent</td>
<td>16(64%)</td>
<td>8(32%)</td>
<td>1(4%)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Broca’s</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
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</tr>
<tr>
<td>Anomia</td>
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<td>0</td>
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<td>0</td>
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</tr>
<tr>
<td>Total</td>
<td>16(64%)</td>
<td>8(32%)</td>
<td>1(4%)</td>
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<th>Aphasia Severity on Admission</th>
<th>Severe</th>
<th>Moderate</th>
<th>Mild</th>
<th>Cleared</th>
<th>Died</th>
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<tbody>
<tr>
<td>Fluent</td>
<td>1(17%)</td>
<td>1(17%)</td>
<td>3(50%)</td>
<td>1(17%)</td>
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<tr>
<td>Nonfluent</td>
<td>17(68%)</td>
<td>7(27%)</td>
<td>1(4%)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Broca’s</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Anomia</td>
<td>0</td>
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<td>0</td>
<td>0</td>
<td>0</td>
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<td>Mixed</td>
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<tr>
<td>Total</td>
<td>18(78%)</td>
<td>8(34%)</td>
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<table>
<thead>
<tr>
<th>Aphasia Severity on Admission</th>
<th>Severe</th>
<th>Moderate</th>
<th>Mild</th>
<th>Cleared</th>
<th>Died</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fluent</td>
<td>1(17%)</td>
<td>1(17%)</td>
<td>3(50%)</td>
<td>1(17%)</td>
<td>0</td>
</tr>
<tr>
<td>Nonfluent</td>
<td>17(68%)</td>
<td>7(27%)</td>
<td>1(4%)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Broca’s</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Anomia</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Mixed</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>18(78%)</td>
<td>8(34%)</td>
<td>1(4%)</td>
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</table>
The present study deals solely with unselected, consecutive stroke patients, each of whom was evaluated within hours of acute insult. Twenty-one percent of all stroke patients had aphasia acutely, and while, not surprisingly, the majority were of mixed type, there were nonetheless a substantial number of "pure forms": 5% Broca's, 6% anomia, 14% Wernicke's, 6% conduction, and 4% "isolation" or transcortical sensory.

There have been nearly as many classifications of aphasia as there have been investigators, and often the same term has been used differently by different workers. Most classifications have been based upon either a psychological or an anatomical approach. Following Broca's 1861 reports suggesting the existence of a motor speech center in the left inferior frontal convolution, the late nineteenth century was dominated by localizers. Wernicke in 1874 classified aphasia into three types: sensory, from a lesion in the posterior first temporal convolution (the putative "speech comprehension center"); motor, from a lesion in Broca's area; and central or "conduction" aphasia ("leitungsaphasie," characterized by a paraphasia in spontaneous speech and on repetition), from a disconnection between the two. The notion that the elements of speech, including reading and writing, reside in discrete interconnected centers was re-enforced by such workers as Bastian, Lichtheim, and Broadbent.

As early as 1864 Jackson had warned, "Speaking is not simply the utterance of words ... speaking is propositionizing," stressing that disturbances of language should be studied in terms of psychological phenomena, not areas of anatomical destruction. His views were in the minority until 1906, when Marie declared that Broca's aphasia was simply dysarthria plus a general intellectual deficit especially affecting language. In 1913 Pick defined, psychologically, four stages in the transition from thought to speech, and classified aphasia in terms of arrests along this hierarchical system. Head in 1926 defined aphasia as a disturbance of "symbolic formulation and expression." He recognized four aphasia types: verbal ("defective power of forming words, whether for external or internal use"), syntactical ("lack of that perfect balance and rhythm necessary to make the sounds uttered by the speaker easily comprehensible"), nominal ("difficulty in appreciating the nominal significance of words"), and semantic ("want of recognition of the ultimate significance and intention of words and phrases apart from their direct meaning").

Goldstein viewed aphasia as "dedifferentiation of brain performance," and anomia in particular as "loss of abstract attitude." He recognized several varieties of aphasia. Three were expressive: peripheral motor, with preserved writing; central motor, corresponding to Broca's aphasia; and "transcortical" motor, with relatively preserved repetition. Three were receptive: pure word deafness; "cortical" sensory, corresponding to Wernicke's sensory aphasia; and "transcortical" sensory, with, again, relatively preserved repetition. Goldstein also recognized "central" aphasia, corresponding to Wernicke's conduction aphasia, and "amnestic" aphasia, or word-finding difficulty.

Brain saw different varieties of aphasia as disturbances of hierarchical "schemas," a concept he derived in part from Kant and from Bergson. Luria has based his aphasia classification upon Pavlov's concept of cortical analyzers. Schuell and Jenkins, while dividing aphasia into five categories, saw them as quantitative variants of a single basic disturbance. Bay recognized only one kind of aphasia, believing that Broca's type represented aphasia plus dysarthria and that Wernicke's sensory aphasia represented aphasia plus a general mental disorder.

A number of classifiers in this century, for example Hensch and Kleist, continued to emphasize anatomy. Nielsen, in an attempt to encompass within a classification anatomical, physiological, and psychological features, developed categories such as "agnosia, auditory, temporal, verbal" and "aphasia, visual, semantic, external capsular," and counted 87 types. In a recent review Meyer has pointed out that many workers considered holist, for example Head, have in fact associated particular anatomical lesions with their clinical subtypes. Nonetheless, systems such as Head's are difficult to utilize clinically. As Weisenberg and McBride stated, "Head's classification ... seems superior to (others) in theory and inferior in practice."

It should be stressed that basing the study of an aphasia population on any classification is unlikely to produce new insights into whether the system being utilized accurately detects the physiological or psychological similarities or differences within the population. Spreen has said, "the nosological system chosen determines the scope and amount of detail available for analysis. No such schema is likely to produce more than a confirmation or a lack of confirmation for the classification system used by the researcher."

Benson and Geschwind have observed that "the rate of agreement on what is being described in the different classifications is actually very high." There are exceptions to this statement. For example, there is no real counterpart in Geschwind's system to Head's semantic aphasia. Moreover, by compartmentalizing patients on the basis of one or two shared signs, it is implied that such aphasias are mechanistically or physiologically the same. The extreme variability of speech output, qualitatively and quantitatively, in, for example, Broca's and Wernicke's aphasia, suggests this assumption may be fallacious. Geschwind's classification is based furthermore upon anatomical assumptions which are debatable (Brown has made a persuasive argument against the concept of conduction aphasia as secondary to a lesion in the arcuate fasciculus, the putative cable connecting Wernicke's and Broca's areas). Finally, while Geschwind's emphasis on fluency versus nonfluency has linguistic foundation, it is not always easy to call speech either fluent or nonfluent.

A major advantage of Geschwind's system is the ease it lends to examination. Many test batteries have been devised for aphasia patients, some complex and time-consuming, and some testing more than language. Head's systematic series of tests included naming common objects, color naming, and simple reading, plus a clock-setting test, a "coin-bowl" test (requiring following directions involving number and space), and a "hand, ear and eye test" (requiring the following of directions and movement imitation). Goldstein used specific language tests (spontaneous speech, series recitation, repetition, word-finding, auditory comprehension, "responses to everyday questions and comments," and following directions of increasing complexity),
The lower incidence of hypertension in our patients compared to those of the Framingham study, of whom only 15% with acute brain infarct had normal blood pressure, may reflect lack of premorbid information in many of our cases.

The smallest incidence of hemiparesis in fluent than nonfluent patients parallels previous observations of others. The large number of fluent patients lacking a field cut and the large number of Broca's aphasics showing one came as a surprise (although a recent pathological study suggests the area of infarct causing Broca's aphasia extends considerably beyond the so-called Broca's area).

Nonfluency carried a worse prognosis, for both mortality and persisting severe deficit, than did fluency. Since nonfluent aphasia was mixed in 107 of 120 cases, and since a brain lesion associated with both anterior and posterior cortical signs is likely to be larger than a lesion associated with more restricted findings, it is likely that this aspect, rather than any special feature of nonfluency, accounts for the poor prognosis. A similar explanation can be proposed for the poor prognosis associated with either hemiparesis or field cut.

Seventy-four percent of the early surviving fluent aphasics and 55% of the early surviving nonfluent aphasics improved. Such early improvement following acute aphasia is well known. More controversial, and not possible to determine from the present series, is how long aphasia continues to improve, a matter obviously relevant in assessing the value of speech therapy. Also not answerable because of the frequent omission of repetition testing during follow-up examinations was how often one aphasia type evolved into another during improvement.

We did not detect, during this period, any examples of such rare language disturbances as cortical anarthria, pure word deafness, or alexia without agraphia. Nor did we find aphasia with a lesion in the territory of an artery other than the middle cerebral. (Penfield and Roberts have shown transient aphasia to occur with lesions of the supplementary motor area.)

A number of probably aphasic patients were not included in the present series because gross dementia or stupor made meaningful language testing impossible. To what extent language dissolution per se disrupts non-language mental functioning remains controversial. Certainly, many of our severe aphasics showed impaired intellectual abilities in areas other than language.

Aphasia, occurring in 21% of 850 stroke patients over three years, and initially moderate or severe in 75%, represents an enormous clinical problem at Harlem Hospital. It is essential that all physicians dealing with such patients recognize aphasia and its varieties and try to communicate this understanding to the patient and his family. Indeed, it is not unusual for aphasia, especially when accompanied by other obvious neurological signs, to be called psychosis. One of our patients was considered schizophrenic until careful testing, especially of writing, revealed the nature of his problem. Another patient, not included in the present series because she was not properly examined acutely, was sent to a mental hospital, and her aphasia was appreciated only after she was sent back to Harlem Hospital for better blood pressure control. From the standpoint of a patient's ability to function in society, severe aphasia is more
disabling than, for example, hemiplegia or blindness. Kurzke\(^6\) has estimated the incidence of stroke in the United States to be 207/100,000 per year. Thus, based upon a 1970 U.S. population of approximately 210 million, it can be estimated that roughly 400,000 new strokes will occur per year, that at least 21% (or 84,000) of these patients will have aphasia, and that at 4 to 12 weeks the language disturbance in 2.5% (or 10,000) will still be severe.

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An In Vitro Study of Prolonged Vasospasm of a Rabbit Cerebral Artery

SUE PIPER DUCKLES, PH.D., ROSEMARY D. BEVAN, M.D., AND JOHN A. BEVAN, M.D.

SUMMARY Longitudinal stretch of the rabbit basilar artery produces local injury followed by prolonged circular constriction. After stretching and rapid release in vitro localized constrictions promptly occurred. This could be prevented by prior treatment with cyanide or calcium-free solution. Once produced, constrictions persisted for more than 72 hours. Previously induced constriction was not reversed by treatment for two hours with cyanide or by removing calcium. Histological observation indicated that constricted areas were associated with a discrete circumferential rupture of the internal elastic lamina and disruption and thinning of the underlying media.

MOST WORKERS agree that release of vasoactive substances from subarachnoid blood is a major cause of diffuse cerebral vasospasm after rupture of an intracranial aneurysm. Yet there is experimental evidence that spasm may be more prolonged and severe when puncture of a cerebral vessel is combined with injection of blood into the subarachnoid space than when blood is applied without injury. Studies of mechanical stimulation of cerebral vessels have found the resulting constriction to be short lived, lasting less than 30 minutes. It is this failure to demonstrate prolonged spasm after mechanical stimulation of or injury to cerebral vessels that has led to the conclusion that these factors cannot contribute to the etiology of cerebral vasospasm.

The present paper demonstrates that longitudinal stretch of the rabbit basilar artery can produce local injury followed by prolonged discrete circular constriction. The accompanying histological changes are described, and the role of smooth muscle in the production of constriction is confirmed. These findings demonstrate that under certain conditions, such as injury, cerebral arterial smooth muscle may undergo an essentially irreversible contraction. Such a mechanism could contribute to the pathogenesis and physiology of prolonged cerebral vasospasm after subarachnoid hemorrhage or traumatic injury.

Specific catecholamine fluorescence at the adventitio-medial junction was unchanged in constricted areas. The relationship between smooth muscle cell length and resting tension of artery segments with and without constrictions was compared. Segments with constrictions had a shorter muscle length for any given resting tension, which confirms that constriction was not due to passive collapse of the vessel wall. These findings suggest that injury of cerebrovascular smooth muscle may result in essentially irreversible vasospasm. Such a mechanism could contribute to the pathogenesis of prolonged cerebral vasospasm after SAH or traumatic injury to the cerebrum.

Methods

New Zealand white rabbits (weight 2 to 3 kg) were stunned by a blow on the nose and bled from the neck. The entire brain with attached arachnoid membrane and blood vessels was removed and placed in Krebs bicarbonate solution at room temperature containing (mM): Na+, 144.2; K+, 4.9; Ca++, 1.3; Mg++, 1.2; Cl-, 126.7; HCO3-, 25.0; SO4, 1.19; ethylene diamine tetraacetic acid, 0.027; and glucose, 11, and bubbled with 95% O2 and 5% CO2. The basilar artery was observed and further dissection was carried out using a Wild stereomicroscope. Photographs were taken with a 35 mm camera attachment. In some experiments Na CN (200 mg per liter) was added to the Krebs solution and glucose was omitted. In other experiments calcium was omitted from the Krebs solution and 2 mM EGTA (ethylene glycol-bis [β-amino ethyl ether] N,N-tetraacetic acid) was added.

HISTOLOGY

After dissection and diagrammatic recording of the presence and site of areas of constriction, the basilar artery was cut open longitudinally and laid flat on a small card. This was then fixed in buffered formaldehyde, dehydrated...
Aphasia in acute stroke.
J C Brust, S Q Shafer, R W Richter and B Bruun

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