Clinical Anatomic Study of Pure Dysarthria

Keiji Ichikawa, MD, and Yasufumi Kageyama, MD

Nine patients with pure dysarthria underwent computed tomography or magnetic resonance imaging. Eight patients had infarcts of lacunar or larger size in the internal capsule: four in the superior portion of the anterior limb or adjacent corona radiata and four in the superior portion of the genu or the adjacent corona radiata. In one patient, there was a small infarct in the bulbar motor cortex. Dysarthria was transient and characterized by poor articulation in all cases. Five patients also had contralateral facial weakness, and three patients with lesions in the genu had minimal and transient involvement of the contralateral fingers. These three cases appeared to be variants of the dysarthria–clumsy hand syndrome. We submit that this syndrome should sometimes be regarded as a stroke syndrome rather than always as a lacunar syndrome. (Stroke 1991;22:809–812)

Pure dysarthria is one of the lacunar syndromes reviewed in 1982 by Fisher, who characterized it as the sudden onset of unaccompanied dysarthria. However, two major concerns regarding the pure dysarthria syndrome remain unanswered. First, it is unclear what “pure” means because a detailed clinical picture of this syndrome has not yet been described, and it is not unusual to detect a few soft neurologic signs in stroke patients who complain only of the sudden onset of dysarthria. Second, the site of the lesion responsible for this syndrome remains uncertain because computed tomographic scans generally are either normal or show multiple lesions and because pathologic verification is lacking. We believe it is more reasonable to define pure dysarthria as the sudden onset of dysarthria caused by stroke, without any other significant symptoms or signs of neuropsychologic abnormality, motor weakness, ataxia, sensory loss, or cranial nerve dysfunction except for those related to articulation. The purpose of this study was to clarify the clinical picture of the pure dysarthria syndrome and to determine the anatomic site of the lesion using computed tomography and magnetic resonance imaging.

Case Reports

During the 5-year period from January 1985 to July 1990, our institution treated 21 patients with cerebral infarcts who presented with the sudden onset of dysarthria. Among them, nine patients met the following criteria for pure dysarthria syndrome: a clinical picture corresponding to that described above, and radiographic evidence of a single infarct or a fresh infarct responsible for the dysarthria. Dysarthria caused by pseudobulbar palsy was excluded.

Four of nine patients had lesions involving the superior portion of the anterior limb of the internal capsule (patients 1 and 4; Figure 1) or the adjacent corona radiata (patients 2 and 3); two of these four patients (patients 1 and 4) had their main lesions in the anterior putamen with anterior extension into the anterior limb of the internal capsule. In four patients (patients 5–8), infarcts involving the uppermost portion of the genu of the internal capsule were demonstrated. One patient (patient 9) had a small infarct in the bulbar motor cortex (Figure 2). Six patients, including one with a cortical infarct, had lesions on the right side, and three had lesions on the left side. The maximal diameter of these infarcts in the horizontal plane varied from 1.0 to 2.7 cm, which meant that they were not always limited to lacunar size (Table 1).

The clinical data are summarized in Table 1. Several observations are worth emphasizing. All of these patients had a similar pattern of dysarthria. Their speech was only mildly or moderately dysarthric, requiring no or minimal effort by the examiner to comprehend individual words. The most prominent speech abnormalities were a “thick” tongue or slurring with incomplete articulation. Lingual, buccal, and palatal sounds were all approximately equally affected. Some patients showed mild slowness of their speech. In these patients, speech was not scanning, explosive, hypophonic, or dysprosodic, and the syntactic structure remained intact. The dysarthria cleared completely over 1–4 months in all patients.

Although none of the patients complained of symptoms other than dysarthria, seven patients had
some associated neurologic signs. Five patients had transient facial asymmetry contralateral to the cerebral lesion; in four it was mild and in one it was moderate, persisting 2 weeks to 2 months. Swallowing was preserved in all patients. In one patient with a motor cortical infarct, there was slight deviation of the tongue to the contralateral side on protrusion. All of these patients had full muscle power in their limbs, but the muscle stretch reflexes were increased in five patients, bilaterally in four and contralaterally in one. Mental state was normal in all patients. None of them complained of clumsiness of the fingers or limb ataxia, but careful examination revealed minimal slowing of finger-tapping on the contralateral side in three patients with lesions in the genu of the internal capsule (patients 6–8). This slow finger-tapping persisted for only 1 day in patient 8, for approximately 10 days in patient 6, and for more than a month in patient 7. Angiography was performed in one patient (patient 8) and disclosed no significant findings.

Discussion

In a strict sense, one might define pure dysarthria as a syndrome associated with no neurologic signs other than dysarthria itself. However, a review of this series of patients with pure dysarthria showed that seven of nine had some associated soft neurologic signs, such as facial asymmetry, increased muscle...
Ichikawa and Kageyama  

**TABLE 1. Summary of Clinical Pictures**

<table>
<thead>
<tr>
<th>Patient/Age/Sex/Handedness</th>
<th>Medical history</th>
<th>Symptoms at onset</th>
<th>Associated signs</th>
<th>Duration of dysarthria (mo)</th>
<th>Site of lesions</th>
<th>Size of lesions (cm)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/70/F/R</td>
<td>HT LVH</td>
<td>DA only, moderate</td>
<td>Shallow L-NLF, mild; bilat. inc. PTRs</td>
<td>2</td>
<td>R-ALIC</td>
<td>2.7x1.7x2.8</td>
</tr>
<tr>
<td>2/63/F/R</td>
<td>HT</td>
<td>DA only, mild</td>
<td>None</td>
<td>2</td>
<td>L-CR adjacent to ALIC</td>
<td>2.0x1.2x1.2</td>
</tr>
<tr>
<td>3/59/M/R</td>
<td>HT Gout LVH</td>
<td>DA only, mild</td>
<td>Bilat. inc. PTRs</td>
<td>4</td>
<td>R-CR adjacent to ALIC</td>
<td>2.4x1.2x2.0</td>
</tr>
<tr>
<td>4/55/M/R</td>
<td>DM</td>
<td>DA only, mild</td>
<td>Shallow R-NLF, mild; bilat. inc. DTRs, mild</td>
<td>1</td>
<td>L-ALIC</td>
<td>2.7x0.5x1.4</td>
</tr>
<tr>
<td>5/46/M/R</td>
<td>HT</td>
<td>DA only, mild</td>
<td>None</td>
<td>2</td>
<td>R-GIC</td>
<td>2.4x1.2x1.0</td>
</tr>
<tr>
<td>6/68/F/R</td>
<td>HT</td>
<td>DA only, mild</td>
<td>Shallow R-NLF, mild; inc. DTRs on R-upper limb; slow R-finger-tapping, minimal</td>
<td>1</td>
<td>L-CR adjacent to GIC</td>
<td>1.7x1.0x1.4</td>
</tr>
<tr>
<td>7/51/M/R</td>
<td>HT DM</td>
<td>DA only, mild</td>
<td>Inc. bilat. DTRs, mild; slow L-finger-tapping, minimal</td>
<td>2.5</td>
<td>R-CR adjacent to GIC</td>
<td>1.0x1.0x1.2</td>
</tr>
<tr>
<td>8/46/M/R</td>
<td>None</td>
<td>DA only, moderate</td>
<td>Shallow L-NLF, mild; slow L-finger-tapping, minimal</td>
<td>2</td>
<td>R-CR adjacent to GIC</td>
<td>0.7x1.2x0.8</td>
</tr>
<tr>
<td>9/59/F/R</td>
<td>DM</td>
<td>DA only, mild</td>
<td>Shallow L-NLF; deviated tongue to L (5 mm)</td>
<td>3</td>
<td>R-bulbar motor cortex</td>
<td>0.8x1.4x1.0</td>
</tr>
</tbody>
</table>

F, female; R, right; HT, hypertension; DA, dysarthria; L, left; NLF, nasolabial fold; bilat., bilateral; inc., increased; PTR, patellar tendon reflex; ALIC, anterior limb of the internal capsule; LVH, left ventricular hypertrophy; CR, corona radiata; M, male; DM, diabetes mellitus; DTR, deep tendon reflex; GIC, genu of the internal capsule.

*Anteroposterior x transverse x rostral caudal dimensions.

stretch reflexes, or mild asymmetry of finger-tapping. Therefore, these patients may not be precisely described as having the pure dysarthria syndrome. Nevertheless, it seems reasonable for there to be some objective signs associated with dysarthria, such as facial weakness or mild deviation of the tongue, when patients are seen early after the onset. The two patients in this study with no associated signs (patients 2 and 5) were evaluated somewhat later than the remaining patients, who were seen within 10 days of the onset of dysarthria. Accordingly, we feel that pure dysarthria should be defined as mentioned above; otherwise, this syndrome could become so rare in clinical practice as to be ignored.

Whether or not the patients with asymmetrical finger-tapping (patients 6–8) should be considered as cases of the dysarthria–clumsy hand syndrome is also worth debating. The original report of the syndrome by Fisher2 described clumsiness, awkwardness, and slowness of fine manipulation of the hand associated with slight weakness, difficulty in writing, and ataxia on the finger–nose test. However, our patients only had clumsy fingers detectable by careful examination, changes too soft to qualify as clumsy hand syndrome. Furthermore, the asymmetrical finger-tapping in patient 8 was observed only on the day of onset of dysarthria, suggesting that the time interval between onset of dysarthria and neurologic examination is important in detecting the pure dysarthria syndrome. Nevertheless, it is probably best to regard these three cases as variants of the dysarthria–clumsy hand syndrome because some patients with this syndrome had lesions in the uppermost portion of the genu of the internal capsule on computed tomography1,3 that were similar in location to those observed in our three patients.

The characteristics of the dysarthria observed in our nine cases were approximately uniform. The dysarthria was relatively mild and usually subsided after 1–4 months, following the general rule that dysarthria due to unilateral involvement of the corticobulbar fibers is transient. It was not possible to distinguish dysarthria due to anterior capsule lesions from that caused by lesions of the genu or the bulbar motor cortex.

The lesions responsible for the pure dysarthria syndrome were located in three different anatomic structures in our series: the anterior capsule in four patients, the genu in four, and the bulbar motor cortex in one. Previous reports of this syndrome are uncommon,4,5 and no pathologic verification has been obtained. Ozaki et al6 presented five cases of the pure dysarthria syndrome with computed tomography correlations. Lacunar infarcts in the anterior limb of the internal capsule were present in four cases, and an infarct in the genu was seen in one case, similar to the findings in our study. The lesions in their patients were all situated in the superior portion of the internal capsule or the adjacent corona radiata, and the same applied to our eight patients with subcortical infarction. This could be an important factor in producing the pure dysarthria syn-
drome due to the lesions in the genu of the internal capsule, because the pyramidal tract occupies a broader area in the superior capsule or corona radiata than in the inferior capsule, making it possible for the corticobulbar fibers to be disrupted while sparing the corticospinal fibers. No previous reports have mentioned the pure dysarthria syndrome being due to a small infarct in the bulbar motor cortex.

Previous findings taken together with our study suggest that this syndrome is caused by the disruption of one of the following parts of the motor systems necessary for articulation: the frontopontine fibers in the anterior capsule, the corticobulbar fibers in the genu, or the bulbar motor cortex. It might well be argued that a small infarct in the brain stem, especially in the basis pontis, could produce this syndrome, although no such case has yet been reported.

Recently, the clinical definition of so-called lacunar syndrome or infarcts has become a source of controversy because their symptoms are not entirely specific for cerebral infarction.6-9 It also has been suggested recently that large artery disease and embolism may be more important in lacunar infarction than was previously assumed,10 especially in the cases of infarcts larger than 1.5 cm in diameter.11 Five of our patients (patients 1–5) had infarcts larger than that size.

Finally, the dysarthria in patient 9 was not caused by a lacunar infarct but by a small cortical infarct. We should consider the pure dysarthria syndrome as possibly being a stroke syndrome in some cases, rather than always a lacunar syndrome like pure motor hemiparesis, ataxic hemiparesis, and other such lacunar conditions.

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