

The Dysarthria–Clumsy Hand Syndrome: A Distinct Clinical Entity Related to Pontine Infarction

Jonathan D. Glass, MD, Allan I. Levey, MD, PhD, and Jeffrey D. Rothstein, MD, PhD

Using magnetic resonance imaging, we studied 6 patients with the dysarthria–clumsy hand syndrome. All were found to have pontine infarctions contralateral to the symptomatic side. Clinically, these patients exhibited dysarthria; “clumsiness,” characterized by dysmetria, dysrhythmia, dysdiadochokinesia and sometimes truncal and gait ataxia; and mild ipsilateral weakness. Previous clinical-anatomical correlations for this syndrome are limited by inconsistencies in clinical diagnostic criteria and low-resolution imaging methods. In our patients, and in a review of the literature, the overwhelming majority of patients with the dysarthria–clumsy hand syndrome had pontine infarcts. We conclude that if rigid clinical criteria are used, the label of the dysarthria–clumsy hand syndrome predicts a lesion in the contralateral basis pontis.

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Intriguing studies of patients with ipsilateral pyramidal and cerebellar signs have been reported sporadically under the titles of “The Dysarthria–Clumsy Hand Syndrome,” “Ataxic Hemiparesis,” and “Homolateral Ataxia and Crural Paresis.” Fisher [1–3] coined each of these terms to identify separate stroke syndromes based on different constellations of symptoms and signs, although it is clear from his most recent review [4] that the term ataxic hemiparesis is meant to replace the term homolateral ataxia and crural paresis. The clinical distinction between dysarthria–clumsy hand syndrome and ataxic hemiparesis is outlined in Table 1. The “clumsiness” and “ataxia” may look the same, as noted by Fisher [3], but the lack of dysarthria, prominent hemiparesis, and frequent sensory symptoms and signs in ataxic hemiparesis serve to distinguish the two syndromes.

Numerous publications have suggested varied anatomical localizations responsible for the clinical syndromes, and these data have led to a generally accepted conclusion that both syndromes can be caused by a lesion of either the basis pontis or the internal capsule. This conclusion is based on evidence limited by two factors: (1) Some authors have used the labels “dysarthria–clumsy hand” and “ataxic hemiparesis” interchangeably, not adhering to Fisher’s original descriptions and thus confusing the clinical distinction. (2) There is virtually no pathological data available for

anatomical confirmation of lesions. The majority of clinical-anatomical correlations have been based on computed tomographic (CT) images that are notoriously insensitive for detecting pathological conditions of the posterior fossa.

High-resolution *in vivo* brain imaging by magnetic resonance scanning (MRI) is an invaluable tool for clinical-anatomical correlation, especially for diseases such as lacunar strokes, which have low mortality and thus provide few immediate specimens for pathological study. We collected a series of 6 patients who adhere to Fisher’s original description of the dysarthria–clumsy hand syndrome and found that all showed pontine infarction by MRI. A patient with ataxic hemiparesis had both pontine and capsular infarcts on MRI. We suggest that if rigid clinical criteria are used, the label of dysarthria–clumsy hand syndrome predicts a lesion in the contralateral basis pontis.

Methods

Patients were selected from the inpatient populations of the three hospitals served by the Johns Hopkins Neurology program. All were examined by at least one of us. Inclusion into the study was made solely on the basis of the patients’ clinical appearance; MRI data, even if available, were not considered in the selection of patients. Rigid clinical criteria were used in order to enhance the specificity of the clinical-anatomical correlation. Patients selected as having the dysarthria–

From the Department of Neurology, The Johns Hopkins Hospital, Baltimore, MD.

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Address correspondence to Dr Glass, Neuropathology Laboratory, Pathology 509, The Johns Hopkins Hospital, 600 N. Wolfe Street, Baltimore, MD 21205.

Table 1. Distinguishing Features between Dysarthria-Clumsy Hand and Ataxic Hemiparesis

	Dysarthria-Clumsy Hand	Ataxic Hemiparesis
Dysarthria	Moderate to severe	None
Hemiparesis	Mild (if any) "pyramidal" weakness	Moderate, frequently leg weakness > arm weakness > face weakness
Clumsiness/ataxia	Ipsilateral clumsiness characterized by dysmetria, dysrhythmia, dysdiadochokinesia, and frequently gait ataxia	Similar to dysarthria-clumsy hand
Sensory	None	Frequent ipsilateral paresthesias, sometimes with objective sensory signs
Other	+/- tongue deviation on protrusion, palatal weakness, and Babinski sign	

Table 2. Summary of Clinical Findings

Patient No.	Age (yr)	HTN, DM	Dysarthria	UMN VII	Tongue Deviation	Palate Deviation	Weakness	Babinski Sign	Lesion Location
1	63	+	++	+			+	+	Pons
2	72	+	++	+					Pons
3	57	+	++	+	+	+	+	+	Pons
4	63	+	++	+			+		Pons
5	37		+++	+	+		+	+	Pons
6	49	+	++	+	+		+	+	Pons
7	59	+		+			++		Pons/capsule

HTN = hypertension; DM = diabetes mellitus; UMN VII = central facial paresis; + = present; ++ = moderate impairment; +++ = severe impairment.

clumsy hand syndrome met the following criteria: (1) dysarthria without dysphasia; (2) unilateral "central" facial weakness with ipsilateral clumsiness appearing as a cerebellar-type ataxia (dysmetria, dysrhythmia, dysdiadochokinesia, gait ataxia), with mild or no weakness; and (3) no sensory symptoms or signs. Other findings were variable (Table 2). MRI scans were done 2 to 7 days after the onset of symptoms using either 0.4- or 1.5-tesla machines and standard acquisition settings and times.

Results

Six patients with the dysarthria-clumsy hand syndrome were identified. There were 4 women and 2 men with a mean age of 57 years (range, 37-72 years). All patients were found by MRI to have paramedian pontine lesions. One patient with ataxic hemiparesis had both a pontine and a capsular lesion.

Case Reports

PATIENT 1. A 63-year-old, right-handed woman complained of sudden onset of thick speech and unstable walking. She noticed severe difficulty with writing and signing a check and described her right hand as "clumsy." On examination she was awake, alert, and oriented. Blood pressure was 220/110 mm Hg and there were bilateral carotid bruits. Speech was thick and slow without dysphasia. There was a

right "central" facial paresis. Tongue and palate movements were normal. Appendicular strength was normal except for minimal (5-/5) weakness of the finger abductors in the right hand. Deep tendon reflexes (DTRs) were symmetrical and normally active except for bilaterally diminished ankle jerks. She had a right Babinski sign. Results of a sensory exam were normal. On the right, finger-to-nose (FTN) and heel-to-shin (HTS) movements were dysmetric and rapid alternating movements (pronation/supination RAM) were slow and irregular. Gait was wide based and she fell to the right.

Brain CT scan on the day of admission had a normal appearance. A MRI scan the following day showed an abnormal high-intensity signal on T2-weighted images in the left paramedian basis pontis (Fig 1).

PATIENT 2. A 72-year-old, right-handed diabetic man had difficulty walking after arising from bed and complained of "weakness" of his right side and a "thick tongue." He had a "stroke" 1 year previously, with right-sided weakness but no speech difficulties. On examination he was awake and oriented but slow to respond. He recalled only one of four cities in 5 minutes but performed well with the remainder of the bedside mental status testing. Speech was thick but there was no dysphasia. There was mild right facial weakness. Strength was normal and DTRs were symmetrical. Plantar reflexes were flexor bilaterally. There was a mild sensory polyneuropathy. He performed poorly on FTN, HTS, and RAM on the right. Gait was wide based but steady.



Fig 1. Patient 1. Figures 1 through 6 are horizontal plane MRIs through the pons, revealing varied degrees of paramedian infarction contralateral to the patients' symptomatic side.

Brain CT scan on the day of admission was normal. MRI done on the third hospital day showed a left paramedian pontine infarct (Fig 2).

PATIENT 3. A 57-year-old, right-handed hypertensive woman noted the gradual onset over an hour of weakness in the right leg and then thick, slow speech late one evening before going to bed. The next morning her symptoms persisted, with additional complaints of difficulty using her right hand and a headache in the left occipital region. When examined later that morning her blood pressure was 150/90 mm Hg; there were no bruits or murmurs. Mental status was clear, and speech was dysarthric but not dysphasic. There was a "central" facial paresis and palatal and tongue weakness on the right. She had a mild right hemiparesis (4/5, arm weakness > leg weakness). Tendon reflexes were symmetrical and there was a right Babinski reflex. Results of a sensory examination were normal. She had poor RAM and an intention tremor on the right. Gait was more unsteady than could be expected from the degree of weakness.

Brain CT scan on the day of admission was normal. MRI on the third hospital day showed a left paramedian pontine infarct (Fig 3).

PATIENT 4. A 63-year-old, right-handed alcoholic, hypertensive man noticed weakness of his right arm and leg when arising from a chair. He complained of a bifrontal headache and his family noted a right facial droop and slurred speech. His symptoms had largely resolved when he was first examined. Blood pressure was 160/100 mm Hg. Speech was dysarthric but not dysphasic. The right nasolabial



Fig 2. Patient 2.

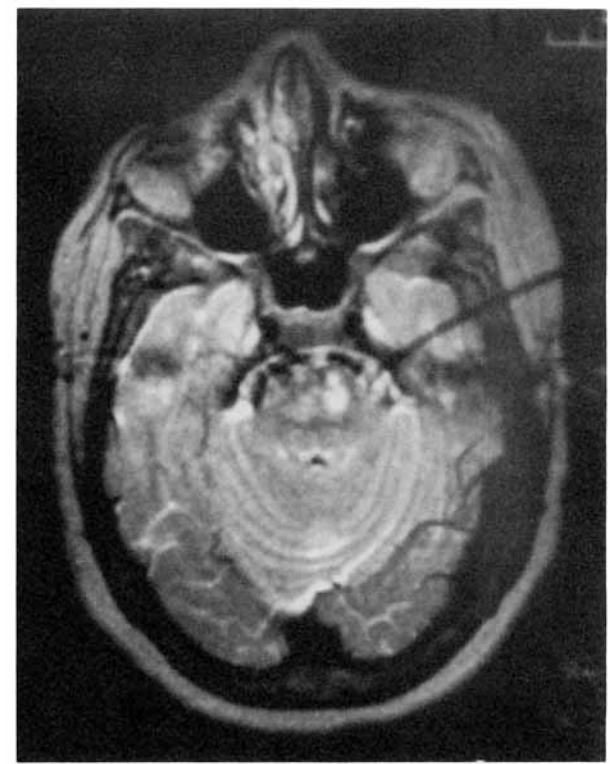


Fig 3. Patient 3.



Fig 4. Patient 4.

fold was flat and the tongue and palate were normal. There was a right pronator drift and mild (4+/5) weakness of the right triceps and wrist extensors. Tendon reflexes were brisker on the right and plantar reflexes were flexor. He had a mild stocking and glove peripheral neuropathy. RAMs were dysrhythmic and slow and on FTN testing he had mild dysmetria on the right. Gait was wide based and he could not tandem walk.

Brain CT scan on the day of admission was normal. On hospital day 7 an MRI showed extensive infarction of the left paramedian basis pontis (Fig 4).

PATIENT 5. A 37-year-old, right-handed woman with a history of migraine headaches experienced the sudden onset of speech difficulty right-sided weakness, and headache in the left temporal region. Her blood pressure was 120/70 mm Hg and a general exam revealed normal findings. She had severe dysarthria but no deficit in language. There was a right "central" facial paresis and the tongue deviated to the right. Palatal elevation was symmetrical. She had a moderate right hemiparesis (arm 3/5, leg 4/5) with symmetrical DTRs and a Babinski sign on the right. Findings on sensory examination were normal. Bilateral dysmetria and dysrhythmia were present, much more severe on the right. Gait was wide based and ataxic.

Brain CT scan and lumbar puncture on admission revealed normal findings. Head MRI on day 2 showed a left paramedian pontine lesion consistent with infarction (Fig 5).

PATIENT 6. A 49-year-old, right-handed woman with a long history of poorly controlled diabetes and hypertension awoke with slurred speech and an inability to walk. Her symptoms resolved over 1 hour but recurred 24 hours later and persisted. Her blood pressure was 140/90 mm Hg and

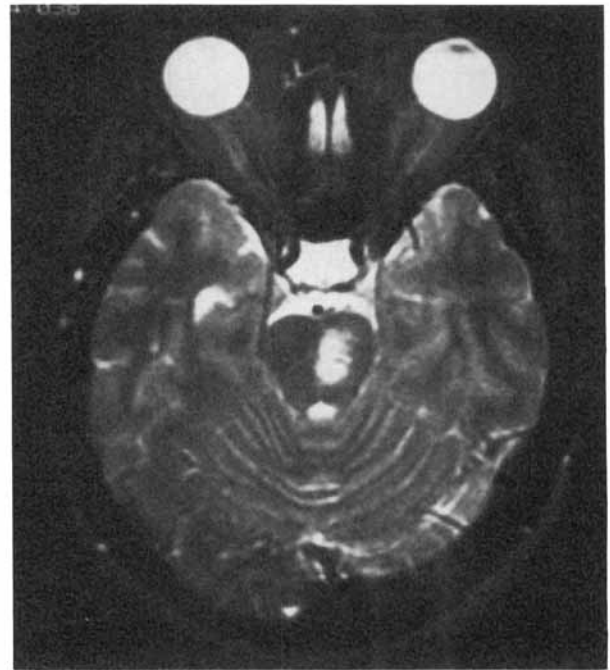


Fig 5. Patient 5.

she had a grade 2 cardiac ejection murmur. Speech was slow and thick but there was no dysphasia. Mental status was normal. Saccadic eye movements were hypermetric to the right. She had weakness of the right lower face and the right side of her tongue. Also present were a right pronator drift, mild (4+/5) weakness of the intrinsic muscles of the right hand, and minimal weakness of the right hamstrings and foot dorsiflexors. Tendon reflexes were brisker on the right and there were bilateral Babinski reflexes. She had a mild stocking polyneuropathy. She performed poorly on RAM and FTN on the right and her gait was broad based and unsteady.

Brain CT scan on admission was normal. Head MRI on day 2 showed a left paramedian pontine infarct (Fig 6).

PATIENT 7. A 59-year-old, right-handed hypertensive woman complained of sudden "heaviness" in the right arm and leg, unsteady walking, and a "clumsy" right hand. She had no difficulties with her speech. On examination, her mental status and speech were normal; blood pressure was 150/85 mm Hg. There was minimal flattening of the right nasolabial fold and other cranial nerves were normal. She had a right hemiparesis, with the leg (3/5) weaker than the arm (4+/5). Tendon reflexes were symmetrical and plantar reflexes were flexor. Results of the sensory examination were normal. She exhibited past pointing on FTN and dysrhythmic RAM in the right upper extremity. Gait was not tested.

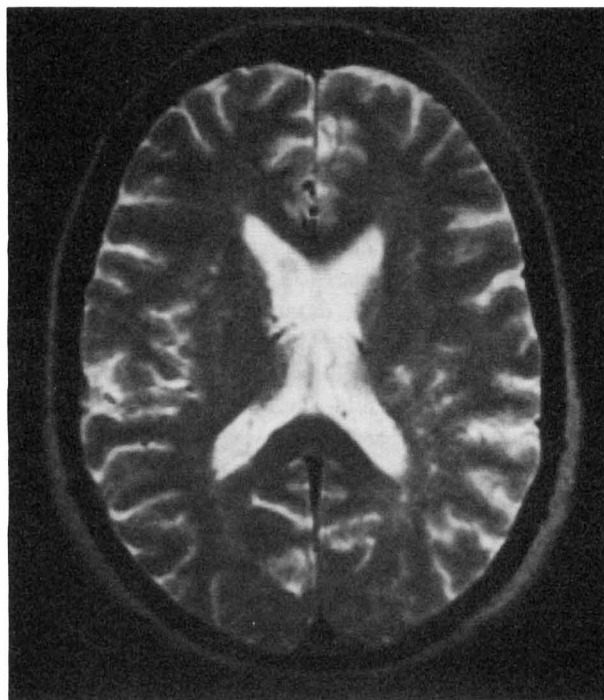
Several hours after admission the right hemiparesis progressed to hemiplegia. A brain CT scan was normal. She was then started on continuous intravenous infusion of heparin. Her weakness began to improve after 24 hours and improved to baseline by 72 hours. Examination again confirmed dysmetria on the right.

Head MRI on day 7 showed lacunar lesions in the left corona radiata and left pons (Fig 7).



Fig 6. Patient 6.

Fig 7. Patient 7, who had ataxic hemiparesis. Small infarcts are seen in the pons and internal capsule, both contralateral to the symptomatic side.



Discussion

Since Fisher's original articles describing syndromes with unilateral pyramidal and cerebellar signs [1-3], there have been numerous case reports of similar patients, bearing the titles of "dysarthria-clumsy hand," "ataxic hemiparesis," and "homolateral ataxia and crural paresis." Though Fisher's case descriptions served to separate these patients into at least two distinct groups, several authors have used the terms interchangeably or have chosen to lump all cases under the label of "ataxic hemiparesis" [5-10]. Some authors have added qualifiers such as "hypesthetic" [11], "painful" [12], or with "trigeminal" weakness [5, 13], creating new categories of "ataxic hemiparesis." Since nearly all publications suggest clinical-anatomical correlations, the specificity of the labels, necessarily based on clinical symptoms and signs, is quite important. Inconsistent applications of these labels have made the literature on this topic confusing and nonspecific, serving to support Landau's recent conclusion that the term ataxic hemiparesis has ". . . no operational meaning, and no utility at all" [14].

Using Fisher's original description of the dysarthria-clumsy hand syndrome, we studied 6 consecutive patients using MRI in an attempt to define an anatomical site where a lesion would produce the associated symptoms and signs. We made a point of clinically separating these patients from those with ataxic hemiparesis (homolateral ataxia and crural paresis) as defined by Fisher's original report of 14 patients [1]. Distinguishing features of ataxic hemiparesis are tran-

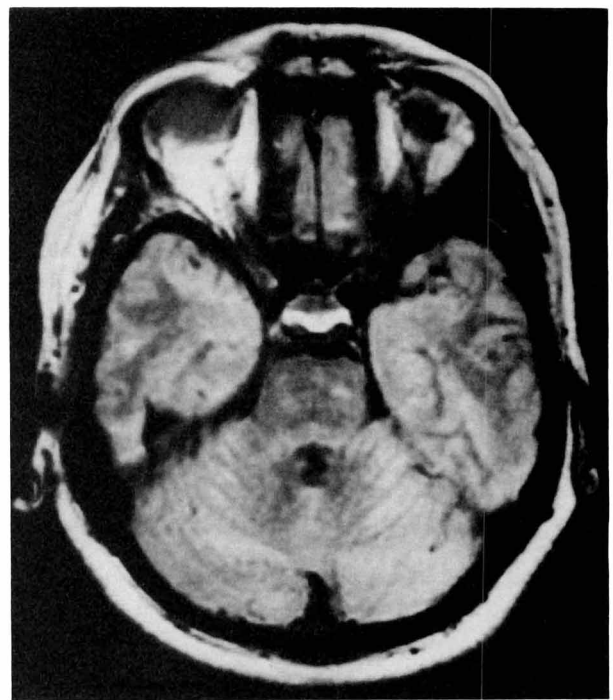


Table 3. Previous Reports of Dysarthria-Clumsy Hand and Ataxic Hemiparesis^a

Author(s)	Year	As Reported ^b		Reevaluated ^{b,c}		Method	Location
		AH#	D-CH	AH	D-CH		
Fisher and Cole [1]	1965	14		14		Clinical	
Fisher [2]	1967		3		3	Pathological	Pons (1)
Fisher [3]	1978	3		2	1	Pathological	Pons
Spertell and Ransom [25]	1979		1		1	CT	Capsule
Perman and Racy [38]	1980	1		1		CT	Capsule
Bendheim and Berg [29]	1981	1		1		CT	Midbrain (leukemia)
Sakai et al. [5]	1981	1 "trigeminal weak"			1	CT	Pons
Ichikawa et al. [26]	1982	1		1		CT	Capsule
Iragui and McCutchen [23]	1982	1		1		CT	Capsule
Schnapper [6]	1982	1		1		CT	Pons
Tuhrim et al. [41]	1982		1		1	CT	Pons
Jabbari et al. [35]	1983	1		1		CT	Capsule
Jacome [7]	1983	1		1		CT	Capsule
Jokelainen and Pike [20]	1983	1		1		CT	Corona radiata
Kobatake and Shinohara [8]	1983	2			2	CT	Pons
DeRenzi et al. [16]	1983	2		2		CT	Capsule
Sage and Lepore [9]	1983	3		2	1	CT	Corona radiata (2), capsule (1)
Van Buggenhout [42]	1984	1		1		CT	Pons
Biller and Scardigli [30]	1984	2		2		CT	Corona radiata
Bougouslavsky et al. [12]	1984	1 "pain"		1		CT	Thalamus
Donati et al. [33]	1984	4		4		CT	Capsule/corona
Huang and Lui [24]	1984	5		4	1	CT	AH: capsule (4); "D-CH": no lesion (1)
Huang and Chan [17]	1984	1		1		CT	Pons
Mori et al. [19]	1984	1		1		CT	Capsule
Tredici et al. [21]	1983	1		1		CT	Capsule
Delgado et al. [31]	1985	1		1		CT	Pons
DeVries and Sno [32]	1985	1	1	1	1	CT	Capsule
Mori et al. [37]	1985	5	3	n/a	n/a	CT	AH: capsule(4), pons(1); D-CH: putamen(3)
Verma and Maheshwari [11]	1986	2 "numbness"		2		CT	Thalamus
Sakai et al. [18]	1986	1			1	CT	Pons
Schleimer et al. [40]	1986	1		1		CT	Capsule
Ambrosetto [13]	1987	1 "trigeminal weak"			1	CT	Pons
Koppel and Weinberger [36]	1987	1	1		2	CT	Pons
Nabatame et al. [22]	1987	3		3		MRI	Pons
Roy III et al. [39]	1987		1		1 (no VII)	CT/MRI	Cerebellum
Huang et al. [34]	1988	2			1	CT	Pons

^aTerms in quotations refer to qualifiers in case descriptions.

^bNumber of patients.

^cRevised diagnosis after review of published case histories.

AH = ataxic hemiparesis or homolateral ataxia and crural paresis; D-CH = dysarthria-clumsy hand syndrome; n/a = case histories not published.

Table 4. Clinical-Anatomical Correlation in Previous Reports

Localization	As Published		Reevaluated	
	AH	D-CH	AH	D-CH ^a
Pons	19	3	9	11
Capsule/corona	29	2	24	3
Other	4	4	4	1
Totals	53	9	37	15

^aNote: 3 patients with D-CH had no reported anatomical correlation.

AH = ataxic hemiparesis; D-CH = dysarthria-clumsy hand.

sient paresthesias; more prominent hemiparesis, with the leg frequently being weaker than the arm and face; and relative lack of dysarthria. All of our patients who were labeled as having dysarthria-clumsy hand syndrome had lesions consistent with small infarcts (lacunes) in the paramedian basis pontis contralateral to the symptomatic side. Patient 7 was diagnosed as having ataxic hemiparesis and was included for comparison.

Our data support Fisher's original pathological findings [2] and suggest that the dysarthria-clumsy hand syndrome is usually caused by a lesion of the paramedian basis pontis. The current literature, however, does not support this hypothesis, with more than half the cases reported correlated with lesions outside the pons (Table 3). Because of the question of seman-

tics in labeling patients, we reviewed all published case histories and relabeled them based on more rigid criteria as dysarthria-clumsy hand, ataxic hemiparesis, or neither (Table 4). Patients for whom there was insufficient data for clear diagnosis were left as the authors' original designation. It is interesting that no patient reported to have "dysarthria-clumsy hand" was relabeled while 9 patients with "ataxic hemiparesis" were redesignated to have dysarthria-clumsy hand, supporting the view that authors have tended to group all patients under the general term of ataxic hemiparesis. After reevaluation, in only 3 of 15 patients was dysarthria-clumsy hand correlated with lesions outside the pons.

Our analysis of other authors' published material must be interpreted with caution because we did not examine the patients nor did we review their charts and radiological images. However, these data are useful for the interpretation of our data in comparison with others.

This series of patients focuses only on clinical-anatomical correlations in the dysarthria-clumsy hand syndrome. There is no doubt that lesions of the basis pontis cause other neurological syndromes, including some that may resemble the dysarthria-clumsy hand syndrome (i.e., "ataxic hemiparesis").

It remains debatable which system(s) is (are) responsible for the motor abnormalities in patients with the dysarthria-clumsy hand syndrome [15]. Clinically, our patients with pontine lesions showed signs suggestive of cerebellar dysfunction. Corticospinal tract findings were not prominent and did not appear to be a significant cause of the "clumsiness" in our patients. This further supports the view [8, 9, 11, 16-28] that there is disruption of cerebellar pathways accounting for at least some, if not all, of the symptoms. It remains unclear why a unilateral pontine lesion, which should equally affect nuclei and crossing fibers, does not give bilateral cerebellar signs. One patient (Patient 5) in this series did have bilateral ataxia. Her signs, however, were notably asymmetrical, raising the same functional question.

Our conclusions are: (1) The dysarthria-clumsy hand syndrome is distinguishable from other syndromes of unilateral pyramidal and cerebellar signs. (2) The most likely anatomical location for a lesion causing the dysarthria-clumsy hand syndrome is the contralateral basis pontis. Strict adherence to clinical criteria coupled with the wide availability of MRI technology will enable clinicians to quickly test this hypothesis.

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References

1. Fisher CM, Cole M. Homolateral ataxia and crural paresis: a vascular syndrome. *J Neurol Neurosurg Psychiatry* 1965;28:48-55
2. Fisher CM. A lacunar stroke: the dysarthria-clumsy hand syndrome. *Neurology* 1967;17:614-617
3. Fisher CM. Ataxic hemiparesis: a pathologic study. *Arch Neurol* 1978;35:126-128
4. Fisher CM. Lacunar strokes and infarcts: a review. *Neurology* 1982;32:871-876
5. Sakai T, Murakami S, Ito K. Ataxic hemiparesis with trigeminal weakness. *Neurology* 1981;31:635-636
6. Schnapper RA. Pontine hemorrhage presenting as ataxic hemiparesis. *Stroke* 1982;13:518-519
7. Jacome DE. Homolateral ataxia and crural paresis. *Arch Neurol* 1983;40:659-660
8. Kobatake K, Shinohara Y. Ataxic hemiparesis in patients with primary pontine hemorrhage. *Stroke* 1983;14:762-764
9. Sage JI, Lepore FE. Ataxic hemiparesis from lesions of the corona radiata. *Arch Neurol* 1983;40:449-450
10. Bamford JM, Warlow CP. Evolution and testing of the lacunar hypothesis. *Stroke* 1988;19:1074-1082
11. Verma AK, Maheshwari MC. Hypesthetic ataxic hemiparesis in thalamic hemorrhage. *Stroke* 1986;17:49-50
12. Bogousslavsky J, Regli F, Ghika J, et al. Painful ataxic hemiparesis. *Arch Neurol* 1984;41:892-893
13. Ambrosetto P. Ataxic hemiparesis with contralateral trigeminal nerve impairment due to pontine hemorrhage. *Stroke* 1987;18:244-245
14. Landau WM. Ataxic hemiparesis: special deluxe stroke or standard brand? *Neurology* 1988;38:1799-1801
15. Landau WM. Ataxic hindbrain thinking: the clumsy cerebellum syndrome. *Neurology* 1989;39:315-323
16. DeRenzi E, Nichelli P, Crisi G. Hemiataxia and crural hemiparesis following capsular infarct. *J Neurol Neurosurg Psychiatry* 1983;46:561-563
17. Huang CY, Chan KY. Pontine ataxic hemiparesis, a lateral penetrator syndrome? *J Neurol Neurosurg Psychiatry* 1984;47:1046-1047
18. Sakai F, Aoki S, Kan S, et al. Ataxic hemiparesis with reductions of cerebellar blood flow. *Stroke* 1986;17:1016-1018
19. Mori E, Yamidori A, Yukata K, et al. Ataxic hemiparesis from small capsular hemorrhage. *Arch Neurol* 1984;41:1050-1053
20. Jokelainen M, Pilke A. Ataxic hemiparesis. *Arch Neurol* 1983;40:326
21. Tredici G, Graziella B, Sanguineti I. Capsular ataxic hemiparesis. *Arch Neurol* 1983;40:326
22. Nabatame H, Fukuyama H, Akiguchi I, et al. Pontine ataxic hemiparesis studied by a high-resolution magnetic resonance imaging system. *Ann Neurol* 1987;21:204-207
23. Iragui VJ, McCutchen CB. Capsular ataxic hemiparesis. *Arch Neurol* 1982;39:528-529
24. Huang CY, Lui FS. Ataxic hemiparesis, localization and clinical features. *Stroke* 1984;15:363-366
25. Spertell RB, Ransom BR. Dysarthria-clumsy hand syndrome produced by capsular infarct. *Ann Neurol* 1979;6:263-265
26. Ichikawa K, Taustsumishita A, Fujioka A. Capsular ataxic hemiparesis. *Arch Neurol* 1982;39:585-586
27. Soria ED, Fine EJ, Paroski MW. Lacunes: the pervasive strokes. *NY State J Med* 1987;87:650-655
28. Kapelle LJ, van Gijn J. Lacunar infarcts. *Clin Neurol Neurosurg* 1986;88:3-17
29. Bendheim PE, Berg BO. Ataxic hemiparesis from a midbrain mass. *Ann Neurol* 1981;9:405-407
30. Biller J, Scardigli K. Ataxic hemiparesis from lesions of the corona radiata. *Arch Neurol* 1984;41:136-137

31. Delgado G, Gallego J, Zubieta JL. High-resolution CT scan in pontine ataxic hemiparesis. *J Neurol Neurosurg Psychiatry* 1985;48:1069
32. DeVries L, Sno HN. Ataxic hemiparesis and 'dysarthria-clumsy hand', two related syndromes caused by a lacunar cerebral infarction. *Ned Tijdschr Geneesk* 1985;129:1628-1631
33. Donati E, Callea L, Faggi L, et al. Ataxic hemiparesis: further CT confirmation on an old localizing hypothesis. *Ital J Neurol Sci* 1984;5:275-278
34. Huang CY, Woo E, Yu YL, et al. Lacunar syndromes due to brainstem infarct and haemorrhage. *J Neurol Neurosurg Psychiatry* 1988;51:509-515
35. Jabbari B, Gunderson CH, McBurney JW. Improvement of ataxic hemiparesis with trihexyphenidyl. *Neurology* 1983;33:1627-1628
36. Koppel BS, Weinberger G. Pontine infarction producing dysarthria-clumsy hand syndrome and ataxic hemiparesis. *Eur Neurol* 1987;26:211-215
37. Mori E, Tabuchi M, Yamadori A. Lacunar syndrome due to intracerebral hemorrhage. *Stroke* 1985;16:454-459
38. Perman GP, Racy A. Homolateral ataxia and crural paresis: case report. *Neurology* 1980;30:1013-1015
39. Roy EP III, Keefover RW, Riggs JE, et al. Dysarthria-clumsy hand syndrome and cerebellar hemorrhage. *Ann Neurol* 1987;21:415-416
40. Schleimer J, Galasko D, Stern BJ. Ataxic hemiparesis with intact sensory modalities. *Arch Neurol* 1986;43:8
41. Tuhim S, Yang WC, Rubinowitz H, et al. Primary pontine hemorrhage and the dysarthria-clumsy hand syndrome. *Neurology* 1982;32:1027-1028
42. Van Buggenhout E. Pontine ataxic hemiparesis. *Arch Neurol* 1984;41:16