PAPERS AND SHORT REPORTS

Dysphagia in acute stroke

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Abstract

A prospective study was undertaken to define the incidence, duration, and consequences of dysphagia in an unselected group of 91 consecutive patients who had suffered acute stroke. The site of the present lesion and of any previous stroke was determined clinically and was confirmed by computed tomography of the brain or necropsy in 40 cases. Of 41 patients who had dysphagia on admission, 37 had had a stroke in one cerebral hemisphere. Only seven patients showed evidence of lesions in both hemispheres. Nineteen of 22 patients who survived a stroke in a hemisphere regained their ability to swallow within 14 days.

Dysphagia in patients who had had a stroke in a cerebral hemisphere was associated in this study with a higher incidence of chest infections, dehydration, and death.

Introduction

Dysphagia in stroke is usually considered to indicate a brain stem lesion caused by vertebrobasilar disease or bilateral corticobulbar fibre damage,[1-3] but it has also been reported in unilateral hemisphere lesions, including stroke.[4-10] There is no reference, however, to such dysphagia in standard medical and neurological textbooks.[2 3 11-16]

This study was designed to establish the incidence, duration, and consequences of dysphagia in stroke, particularly stroke in a cerebral hemisphere.

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Patients and methods

One hundred consecutive patients who had a clinical diagnosis of acute stroke[17] were seen during six months at a district general hospital. Patients were identified by members of staff of the stroke unit who visited the medical wards daily during the week. Medical staff in the hospital were asked to report patients who had a stroke on other wards to one of us (CG). Nine patients were excluded because their stroke had occurred more than 14 days before admission or because the clinical results of further investigation did not support the original diagnosis of stroke.[17]

Ninety one patients entered the study (38 men, 53 women; median age 70, range 26-96). Fifty six patients were seen within 48 hours after the ictus, and a further 26 were seen within 96 hours. The remaining nine patients were seen within 13 days after the stroke. The history was established by directly questioning the patient or a relative or by referring to the medical notes. A basic neurological examination was performed and repeated one week later to distinguish clinically stroke in a cerebral hemisphere from that in the brain stem. Motor power was assessed with the motoricity index,[18] which gives a score from 0 (total paralysis) to 100 (normal power). Forty seven patients underwent computed tomography of the brain. Necropsies were performed on eight of the patients who died during the study.

The cranial nerve function was examined and the degree of dysarthria determined with standard tests.[1 3 19] Apraxia[2] was diagnosed when a patient could not perform a task such as protruding the tongue on request, though the patient may have been able to perform unthinking movements with the tongue, such as licking the lips. Swallowing ability was assessed by asking the patients to drink 50 ml water steadily from a beaker or medicine container (modification of Frenchay dysarthria assessment[19]). The patients sat upright and were supported or helped to hold the beaker if necessary. This test was omitted in patients known to have choked on fluids that day and in those who were unconscious. Patients who had depressed or absent gag reflexes were tested initially with smaller volumes of water. If the patients choked they were allowed to rest for a few minutes, and the test was repeated. Dysphagia was defined as the inability to drink 50 ml water or choking more than once while attempting to drink 50 ml water on two occasions.

The tests of swallowing ability and lower cranial nerve function were repeated at least every 48 hours during the first week after the stroke. Thereafter the tests were repeated twice a week until the patient had no difficulty in swallowing 50 ml water. At each assessment the patients were examined for evidence of chest infection. This was defined as the presence of cough or fever and signs of infection on examination or in a chest x ray film. Charts of fluid balance were kept by the nursing staff, and blood samples were taken every two or three days during the first 10 days in hospital. Dehydration was defined as a negative fluid balance (urine output greater than fluid input), a fluid intake of less than 0·5 litres a day, a packed cell volume of 0·48 or higher, or a urea concentration of 10 mmol/l or higher on at least one occasion.

TABLE I—*Background data on 91 patients with acute stroke*

	Patients with dysphagia (n=41)	Patients without dysphagia (n=50)	Significance
Age (years):			
Median	73	67	
Range	44-85	26-96	p<0·05*
Sex ratio M/F	18/23	20/30	NS†
History:			
Previous cerebrovascular event	23	19	NS‡
Previous dysphagia with stroke	2	1	NS§
Hypertension	13	12	NS‖
Diabetes mellitus	8	4	NS¶

* Mann-Whitney U test. † χ^2=0·03. ‡ χ^2=2·29. § Fisher's exact test. ‖ χ^2=0·34. ¶ χ^2=1·70.

Statistical analyses were performed with parametric or non-parametric tests. The differences in means were assessed with the *t* test or Mann-Whitney U tests, and associations were judged with χ^2 (with correction factor where necessary) or Fisher's exact test. Differences between the groups were considered to be significant if p=0·05 or less.

Results

Forty one patients (45%) had acute stroke and dysphagia. There was no significant difference in the length of time between the stroke and the first assessment between the patients who had dysphagia and those who did not. Table I shows that the patients who had dysphagia were significantly older (p<0·05) than those who did not but that sex and history did not differ between the groups. Twelve patients who had dysphagia did not have a history of cerebrovascular events, hypertension, or diabetes that might have

TABLE II—*Clinical features, duration of dysphagia, appearance of computed tomogram of brain, and necropsy findings in 20 patients with acute stroke*

Case No	Age	Sex	Previous stroke or transient ischaemic attack	Clinical features	Duration of dysphagia (days)	Computed tomogram (days after stroke)	Scan appearance	Necropsy finding	Outcome at six weeks
11	44	M	No	Right hemiplegia, dysphasia, hemianopia upper motor neurone seventh cranial nerve weakness, anarthria, right gag reduced, dyspraxia, sleepy at first	6	10	Infarct of left middle cerebral artery territory	—	Alive
21	70	F	No	Right hemiplegia, upper motor neurone seventh cranial nerve weakness, right gag reduced, very drowsy	10 (Death)	2	Haemorrhage, left basal ganglia	—	Dead
25	65	F	No	Left hemiplegia, dysphasia, hemianopia, upper motor seventh neurone cranial nerve weakness, dysarthria, loss of consciousness at first	6	9	Normal (poor quality)	—	Alive
35	67	F	Yes	Right hemiplegia, dysphasia, hemianopia, upper motor neurone seventh cranial nerve weakness, dysarthria, gag and cough reduced, progressive loss of consciousness	6 (Death)	1	Left temporal and occipital lobe infarcts	Multiple infarcts	Dead
40	65	F	No	Left hemiplegia, hemianopia, upper motor neurone seventh cranial nerve weakness, dysarthria, weak palate and cough, sleepy at first	11	8	Infarct of right middle cerebral artery territory	—	Alive
45	80	F	Yes	Right hemiplegia, dysphasia, hemianopia, dysarthria, drowsy	13 (Death)	2	Multiple infarcts	—	Dead
47	85	M	No	Right hemiplegia, dysphasia, upper motor neurone seventh cranial nerve weakness, dysarthria, sleepy at first	25	1	Left parietal infarct	—	Alive
49	75	M	Yes	Right hemiplagia, dysphasia, dysarthria, no loss of consciousness	2	1	Normal (poor quality)	—	Alive
52	67	F	Yes	Dysphasia, right upper motor neurone seventh cranial nerve weakness, palate weak, gag absent, tongue to right, dyspraxia, no loss of consciousness	6	7	Infarct of left middle cerebral artery territory and old infarct of posterior cerebral artery territory	—	Alive
55	74	M	No	Left sensory loss, no loss of consciousness	1	4	Infarct of right parietal lobe, and infarct adjacent to left lateral ventricle posteriorly	—	Alive
57	61	F	No	Left hemiplegia, hemianopia, upper motor neurone seventh cranial nerve weakness, dysarthria, palate sensation and gag reduced, tongue to left, no loss of consciousness	5	18	Infarct of right basal ganglia	—	Alive
61	65	M	Yes	Right hemiplegia, dysphasia, upper motor neurone seventh cranial nerve weakness, dysarthria, palate sensation, gag, and cough reduced, tongue to right, no loss of consciousness	4	10	Infarct of left temporal lobe	—	Alive
67	78	M	Yes	Right hemiplegia, dysphasia, upper motor neurone seventh cranial nerve weakness, dysarthria, gag reduced, dyspraxia, loss of consciousness at first	6	15	Multiple infarcts	—	Alive
68	84	F	Yes	Right hemiplegia, no gag, unconscious	5 (Death)	—	—	Multiple infarcts	Dead
84	61	M	No	Right hemiplegia, dysphasia, hemianopia, upper motor neurone seventh and fifth cranial nerve weakness, dysarthria, palate, gag, and cough reduced, tongue to right, sleepy at first	2	7	Haemorrhage left basal ganglia	—	Alive
95	60	M	No	Left hemiplegia, dysphasia, upper motor neurone seventh cranial nerve weakness, dysarthria, palate and gag reduced, tongue to left, no loss of consciousness	8 (Death)	—	—	Infarct of left basal ganglia, pneumonia	Dead
100	85	M	No	Right hemiplegia, dysphasia, hemianopia, upper motor neurone seventh and fifth cranial nerve weakness, dysarthria, sleepy at first	7	10	Infarct of left middle cerebral territory	—	Alive
12	73	F	No	Right hemiplegia, dysarthria, weak palate, gag absent, ataxia, no loss of consciousness	9	3	Infarct of superior cerebellar artery territory	—	Alive
33	72	M	No	Left hemiplegia, hemianopia, dysarthria, gag and cough reduced, progressive loss of consciousness	12 (Death)	3	Infarct of right occipital pole	Infarcts of left brain stem, left basal ganglia, and right occipital pole	Dead
82	75	M	Yes	Right hemiplegia, upper motor neurone seventh and fifth cranial nerve weakness, dysarthria, gag, palate, and cough reduced, no loss of consciousness	25	21	Infarcts of brain stem	—	Alive

predisposed them to suffering from bilateral hemisphere lesions or cortico-bulbar fibre damage.

Among the patients who had dysphagia, 23 had had strokes in the left hemisphere and 14 had had strokes in the right. In the group who could swallow normally there were 21 patients who had had strokes in the left hemisphere and 28 who had had strokes in the right. The increased incidence of strokes in the left hemisphere in the group who had dysphagia was not significant ($\chi^2=2{\cdot}42$, df=1, NS). Of the 16 patients who had had a stroke in a cerebral hemisphere and dysphagia and who had suffered a previous stroke, nine had had strokes in the same hemisphere and seven had had strokes in the opposite hemisphere. There were also four patients who had had strokes in the brain stem in the group who had dysphagia and one patient who had had a stroke in the brain stem in the group who could swallow normally.

The diagnoses were made clinically and were confirmed by computed tomography of the brain or necropsy in 40 cases. Eighteen patients who had had an acute stroke and had dysphagia underwent computed tomography; two had normal scans. Necropsy was also performed on two of these patients. A further two patients who had dysphagia did not undergo computed tomography but did undergo necropsy. Table II describes the clinical details, the appearances of the computed tomograms of the brain, and the necropsy findings in these 20 patients. Computed tomograms of the brain in five patients without dysphagia were normal.

On average, the patients who had dysphagia had had more severe strokes than those who could swallow normally. Table III, however, shows that there was considerable variation within each group. For example, two patients who had dysphagia and cranial nerve problems showed no weakness in the arms and legs (average total motoricity score 100), while 13 patients who did not have dysphagia had a profound hemiplegia with a motoricity score in the range 0-32 (total motoricity score range 0-32). Twenty nine patients who had had a stroke in a hemisphere and had dysphagia were alert and able to perform the dysphagia test on at least two occasions, while eight patients who had had a stroke in a hemisphere were unable to cooperate fully with the swallowing test: four patients were unresponsive and four patients were drowsy at all assessment times. These eight were included in the dysphagia group, as they were unable to drink 50 ml water.

TABLE III—*Severity of stroke in 91 patients*

	Patients with dysphagia (n=41)	Patients without dysphagia (n=50)	χ^2 (df)	Significance
Motoricity score for hemiplegic side on admission:				
Mean	27·6	58·3	*	p<0·001
Distribution				
100	2	8		
66-99	3	19		
33-65	6	7		
0-32	23	13		
Unassessable	7	3		
Sitting balance on admission:				
Normal	5	26	17·0 (2)	p<0·001
Less than one minute unsupported or with support only	16	16		
None	18	8		
Unassessable	2	0		
Worst level of consciousness:				
Normal	11	25	8·3 (3)	p<0·05
Mild drowsiness	4	8		
Drowsy but rousable	14	11		
Localised pain or unresponsive	12	6		
Discharged from hospital within two weeks after stroke	4	20	9·1 (1)	p<0·01
Died in hospital within six weeks after stroke	19	11	5·0 (1)	p<0·05

* Mean difference 30·7 (99·9% confidence interval 4 to 58).

There was no significant difference in the incidence of hemianopia, sensory inattention, subjective sensory loss, or dyspraxia between the patients who had dysphagia and those who did not. Five patients who had dysphagia had clear dyspraxia affecting the tongue, lips, and face.

Two patients who had had a stroke in the brain stem and who had dysphagia died. The other two patients who had had a stroke in the brain stem and who had dysphagia on admission were able to swallow normally within 25 days and were discharged home. These patients will not be discussed further here.

Table IV shows that abnormalities of the lower cranial nerves were more common in patients who had had a stroke in a hemisphere and who had dysphagia than in those who did not have dysphagia. Not all of the tests were performed in every patient, owing to some patients being dysphasic, drowsy, or confused. No single abnormality was present in all the patients who had dysphagia, but weakness of the upper motor neurone facial nerve and dysarthria were the most common. The signs did not correspond to recognised brain stem syndromes and were thought to be supranuclear in origin.

TABLE IV—*Cranial nerve abnormalities observed in 86 patients during first week after hemisphere stroke*

	% Of patients with dysphagia (n=26-33)*	% Of patients without dysphagia (n=46-48)*	χ^2	Significance
Fifth cranial nerve weakness	10	0	†	NS
Upper motor neurone seventh cranial nerve weakness	82	50	7·2	p<0·01
Dysarthria	96	51	14·6	p<0·001
Gag reflex	58	10	18·5	p<0·001
Palatal movement	46	11	9·4	p<0·01
Cough	52	15	10·7	p<0·01
Tongue protrusion	50	19	6·2	p<0·05

* Not all tests were performed in every patient owing to dysphasia, drowsiness, or confusion.

In the patients who had had a stroke and survived dysphagia lasted for eight days or less in 15 patients, nine to 14 days in three, and up to 40 days in a further three. The mean duration was 8·5 days. The patients who had had a stroke in a hemisphere and had dysphagia had a higher incidence of chest infections (seven (19%) compared with four (8%) among patients who could swallow normally), but the difference was not significant (χ^2 with Yates's correction=1·32, df=1, NS). All the chest infections developed within one week after the stroke and were not restricted to patients whose consciousness was impaired.

Fourteen patients who had had a stroke in a cerebral hemisphere and who had dysphagia were given fluids by nasogastric tube or intravenously. The fluid balance, however, proved to be difficult to assess, as the other patients who had dysphagia spilt fluid owing to facial weakness and choking, and over half of the patients were incontinent of urine.

Blood tests were performed on 33 of 37 patients who had dysphagia and 31 of 49 patients who did not have dysphagia during the first 10 days after the stroke in a hemisphere. Nine (27%) of the patients who had dysphagia had at least one packed cell volume value of 0·48 or higher compared with four (13%) of the patients who could swallow normally. Nineteen (58%) of the patients who had dysphagia had a urea concentration of 10 mmol/l or higher compared with 10 (32%) of the patients who did not have dysphagia. Serial measurements of packed cell volume and urea concentration in patients who had dysphagia showed that these values varied with fluid intake.

Discussion

This study showed that 41 (45%) of 91 patients who had had an acute stroke had difficulty in swallowing when admitted to hospital. These were unselected patients admitted to a district general hospital. Dysphagia was found in 37 (43%) of 86 patients who had had a stroke in a cerebral hemisphere. This may seem surprising, but there have been several previous reports of dysphagia in unilateral hemisphere lesions.[4-6 9]

During our study Veis and Logemann reported their video-fluorographic studies of patients who had swallowing disorders after a cerebrovascular accident.[7 8] The site of the lesion was confirmed by computed tomography of the brain in about half of the patients. They found that 26 (23%) of 113 patients who had had a first hemisphere stroke in a cerebral hemisphere had dysphagia.[8] They may have failed, however, to identify some patients who had dysphagia after their stroke, as they relied on a computer search to identify patients who had had a stroke and then inspected the medical charts for evidence of difficulty in swallowing. A uniform test of swallowing ability was not applied to all patients who had had a stroke.

Willoughby and Anderson did not find any patients who had had an acute stroke in a cerebral hemisphere and had dysphagia in their study,[20] though 60% had dysarthria. Their patients were assessed up to a month after their stroke in most cases and as late as four and a

half years afterwards in one case. In our study over 90% of patients were seen within four days after their stroke, and 86% of patients who had dysphagia on admission were able to swallow normally two weeks later.

Barer reported dysphagia in strokes in a cerebral hemisphere while studying the effect of oral β blockers in acute stroke.[10] Unconscious patients and those who had severe dysphagia were excluded. Patients were seen within 48 hours after the onset of symptoms, and one third underwent computed tomography of the brain. Barer found that 29 (28%) of 105 patients who had a stroke in only one hemisphere had dysphagia. A week later this had resolved in 13 patients (45%), and seven (24%) had died.

Our results show that stroke in only one hemisphere is a more common cause of dysphagia than is generally believed. In the present study only seven patients who had dysphagia showed evidence of bilateral lesions. Dysphagia was not restricted to those with a history of stroke, transient ischaemic attack, hypertension, or diabetes mellitus that might have caused small lacunar infarcts or degenerative changes in the corticobulbar tracts. There was no significant difference in the incidence of these conditions or of previous stroke and dysphagia between the patients who could swallow normally and those who had dysphagia. As in the studies by Veis and Barer the number of patients who had lesions in the left hemisphere was greater than the number who had lesions in the right hemisphere,[7 8 10] but the difference was not significant.

What possible mechanisms can explain dysphagia in unilateral hemisphere stroke? Though the severity of strokes in patients who had dysphagia was on average greater than in those who had no difficulty in swallowing, there was considerable variation within each group. Thus distortion of the brain stem owing to severe cerebral oedema after a stroke may cause dysphagia in some cases, but it is unlikely to explain dysphagia in all cases.

Dysphagia associated with facial apraxia has been reported.[2 6 21] None of the patients reported on by Meadows had dyspraxia,[4] and apraxia was observed in only five of our patients who had dysphagia. Previous studies have implicated the lowest part of the precentral gyrus in the cortical control of swallowing,[4 6 9 21] and the region responsible for apraxia may lie close to a swallowing area in the lower part of the precentral gyrus. Bruyn and Gathier discussed reports of lesions of the lower precentral gyrus and the posterior part of the inferior frontal gyri that caused a cortical form of pseudobulbar palsy with dysphagia—"the operculum syndrome."[9] This usually affects both hemispheres but can be unilateral. The syndrome is due to ischaemic lesions in parts of the brain supplied by the branches of the middle cerebral artery. The present study suggests that such deficits can be found in patients whose stroke was caused by complete infarction of such parts of the brain. Though swallowing pathways are bilaterally represented, some patients seem to be more susceptible to disturbance of function in one hemisphere, and the other hemisphere takes some days to compensate. Why this should vary among patients is not clear.

Animal studies suggest that non-capsular pathways—namely, the hypothalamus, the limbic system (especially the amygdala), and the basal ganglia—as well as the frontal cortex and corticobulbar tracts, play a part in swallowing.[22 23] Damage to these structures by infarction or haemorrhage probably explains the occurrence of dysphagia in patients who did not have classic lesions in parts of the brain supplied by the middle cerebral artery or stroke in the brain stem in this study.

Veis and Logemann, using videofluorographic studies, showed that the most common abnormality in patients who have had a stroke and who have dysphagia is a delayed swallowing reflex. Decreased control of the tongue and reduced pharyngeal peristalsis were seen in over one half of the cases. Cricopharyngeal dysfunction was rarely seen. These disturbances usually occurred in combination, and about 40% of patients aspirated.

In the present study 19% of the patients who had a stroke in a cerebral hemisphere and dysphagia developed chest infections compared with only 8% in the group who could swallow normally. This difference may have been owing to aspiration or increased immobility in the dysphagia group. There seemed to be an increased risk of dehydration, but more work needs to be done on the complications of dysphagia in a larger number of patients who have had a stroke. If dysphagia is identified early after a stroke happens dehydration and chest infections may be prevented with nasogastric tubes or intravenous fluids until swallowing recovers.

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