Electromyographic recording of the jaw reflex in multiple sclerosis

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The clinical value of the jaw reflex is well recognized in the detection of upper motor neurone disease. This sign was first described in 1886 (de Watteville, 1886) and has also been called the 'temporomasseter reflex', 'mandibular reflex', and 'jaw jerk', although Wartenberg (1945) felt that 'jaw reflex' was the appropriate term.

Interruption of the reflex arc is less easily recognized clinically. Thus, the reflex may be reduced in normal subjects and, although the reflex arc is predominantly unilateral and suggested by experimental evidence to be monosynaptic (Corbin and Harrison, 1940; Szentágothai, 1948; McIntyre, 1951; Hugelin and Bonvallet, 1956; McIntyre and Robinson, 1959), it can rarely be lateralized by clinical testing. Mild to moderate wasting or weakness of the jaw muscles is also difficult to assess, especially when bilateral or when malocclusion is present. Therefore, although involvement of the sensory division of the fifth cranial nerve may be detectable clinically, a lesion of the reflex arc or motor division may not be apparent.

Electromyographic recording of the jaw reflex has been used to detect abnormality of the fifth nerve which may not be evident clinically, this method also allowing comparison between the response on each side to a single mechanical stimulus.

The association of trigeminal neuralgia with multiple sclerosis has been known since 1905 (Berger, 1905; Adson, 1926; Parker, 1928). Recent papers suggest that 1 to 2% of patients with trigeminal neuralgia later develop multiple sclerosis (Müller, 1949; Peet and Schneider, 1952; Dereux, 1952; Ruge, Brochner, and Davis, 1958; Rushton and Olafson, 1965) and that a similar percentage of patients with multiple sclerosis give a history of facial pain indistinguishable from trigeminal neuralgia except that it is more often bilateral. For this reason it appeared probable that some patients with multiple sclerosis, even without clinical indication of fifth

nerve involvement now or in the past, might have abnormality of the fifth cranial nerve.

METHOD

A consecutive unselected series of 32 patients referred to the University of Michigan Medical Center with multiple sclerosis was studied. Each patient was examined by one of us (L.O.T.), particular note being made of any clinical evidence of fifth nerve abnormality, then the jaw reflex was recorded by the other (C.J.G.) without prior knowledge of the clinical findings. The jaw reflex was elicited by a tap with a reflex hammer on the midpoint of the patient's chin, the hammer triggering the sweep on which the two electromyographic tracings were displayed. The recordings were made simultaneously from each side with surface electrodes over each masseter muscle. The mean normal latency as previously reported (Goodwin, 1968) was 8.4 msec on each side with a standard deviation of 1.0 msec and a range of 6.0 to 10.5 msec. In that study the reflex was found to be present bilaterally in each normal subject and the difference in latency between the two sides was not more than 2 msec, in 82 out of the 86

FIG. 1. Normal jaw reflex, three successive responses. This also shows the small decrease in latency on both sides with increasing strength of the tap on the chin (1 to 3).

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subjects being 1.0 msec or less. The recordings in both that study and this were read to the nearest 0.5 msec (Fig. 1).

Amplitude and duration of response were also measured but gave no additional information in this series and so are not reported.

RESULTS

Twelve of the 32 patients (38%) with multiple sclerosis showed an abnormality, this being a delay or absence of the jaw reflex on one or both sides (Fig. 2) as detailed in Table I. The abnormalities

FIG. 2. Abnormal jaw reflex in woman of 46 with multiple sclerosis. Latency left 13.0 msec. Right 9.0 msec.

were equally distributed between the two sides. Table II shows the mean age and duration of multiple sclerosis compared with the presence or

TABLE I
ABNORMAL REFLEX IN 12 PATIENTS

	Left	Right	Bilateral	Total
Delay	5	2	0	7
Absence	1	3	3	7

Two patients showed delay on one side and no response on the other.

TABLE II
AGE AND DURATION OF ILLNESS OF PATIENTS WITH MULTIPLE SCLEROSIS

Jaw reflex	No.	Ages (yr)			Duration of multiple sclerosis (yr)		
		Mean (S.D.)	Range		Mean (S.D.)	Range	
Abnormal	12	33.1 ± 8.4	21-48		5.2 ± 3.2	3-12	
Normal	20	41.0 ± 12.8	19-58		4.7 ± 2.7	1-10	
Total	32	38.0	19-58		4.9	1-12	

absence of an abnormal reflex, there being no significant correlation with either of these two factors. However, it is interesting to note the lower mean age in the abnormal group.

The clinical history was compared with the jaw reflex findings (see Addendum) and again there was no correlation between the occurrence of an abnormal reflex and a particular clinical finding on history or examination, and no increased incidence of abnormality with a history of cranial nerve involvement. At the time of this examination no patients had evidence of fifth nerve abnormality and in none was there a history of facial pain. In each patient in whom an abnormal reflex was found electromyography of each masseter was carried out; in no case was there any evidence of denervation. One patient with multiple sclerosis and diabetes mellitus had normal findings on peripheral nerve conduction studies, which together with the absence of clinical history or sign of peripheral neuropathy makes this a very improbable cause of the abnormal jaw reflex.

Figure 3 shows a graph of the findings in all the patients, the darkened area being that of ± 2 standard deviations from the normal mean of 8.4 msec. Each of those showing a delayed reflex on one side also showed a marked increase in the difference between the two sides above the upper limit of normal of 2 msec.

Case 25 was of particular interest because at the first examination the reflex was unobtainable on the right and of prolonged latency (11.0 msec) on the left, but on the repeat examination three months later the reflex was found to be present and of normal latency on each side, though this was not accompanied by clinical remission of the spastic paraplegia which was her major neurological problem. In two other patients with an abnormal jaw reflex, repeat examination showed no changes.

DISCUSSION

The results show that electromyographic recording of the jaw reflex can detect abnormality of the fifth cranial nerve when none is detectable clinically. But abnormal clinical findings such as diminished sensation or an absent corneal reflex can occur with a normal jaw reflex, as seen in another patient not included here who had intracranial secondaries from carcinoma of bronchus. This may well be because of the different pathways involved.

Sensation and the afferent side of the corneal reflex are carried in the sensory root of the fifth nerve. This arrangement is in contrast with that of the jaw reflex, whose afferent and efferent sides are both contained in the motor root. The afferent path-

FIG. 3. Normal range of latency of jaw reflex compared with findings in 32 patients with multiple sclerosis.

way of this stretch reflex originates in the muscle spindles of the masticatory muscles, the afferent fibres enter the brain stem in the motor root of the fifth cranial nerve and go to the large, unipolar cells of the mesencephalic trigeminal nucleus. These fibres are distinct from those arising in the Golgi tendon organs, which travel with the sensory root (Szentágothai, 1948; McIntyre and Robinson, 1959).

Abnormality of the jaw reflex was detected in 12 of the 32 patients with multiple sclerosis examined, there being no correlation between this and the duration of the disease or the age of the patient. In no patient was there clinical evidence of fifth nerve involvement, and no patient gave a history of facial pain. One patient, Case 25, showed return of the reflex on examination four months after the initial testing, although this was not accompanied by clinical improvement. In two further patients repeat examination still showed the same abnormality.

Multiple sclerosis is a disease almost exclusively of the central nervous system. The rarity with which it affects the peripheral nervous system was noted by McAlpine, Compston, and Lumsden (1955), who found only two definite reports in the literature describing apparent involvement at this level. Also, in the present series, every patient with an electrically

abnormal jaw reflex had a normal electromyogram of the masseter muscles. These considerations make the possibility of peripheral neuropathy a most improbable cause of our findings. The exact site of the lesion responsible for these findings awaits pathological detection, but it is suggested that this probably involves the reflex arc at some point between the mesencephalic and motor nuclei of the trigeminal nerve. Figure 4 shows a hemisection of the pons taken from another patient with multiple sclerosis showing an area of demyelination in this region. It is suggested that such a lesion could cause the abnormalities that have been discussed.

It may be impossible to confirm the early diagnosis of multiple sclerosis because of lack of dissemination of the lesions in space or time, and objective evidence of a lesion outside the area of clinical involvement might help to support the diagnosis. However, we do not know how early in the disease the jaw reflex may show abnormality as the mean duration of the disease in our patients was 4.9 years, and in only one was it less than three years. This technique could be especially useful in patients whose main neurological difficulty was optic neuropathy, or spastic paraplegia with no signs involving the cranial nerves—particularly in this last group where the findings

FIG. 4. Necropsy material from case of multiple sclerosis. Hemisection through the tegmentum of the pons at the level of the sensory root of the trigeminal nerve, showing demyelination of the sensory root and root entry zone, and involving also the cells of the motor nucleus of trigeminal. Lesions in this latter distribution may be reflected by the reflex abnormalities described. (An incidental finding is demyelination of the descending corticospinal tract.)

of an abnormality above the level of the cervical cord would be strong evidence against a tumour or other lesion in that region.

It is planned to extend this investigation further, both to allow more prolonged serial study of patients with multiple sclerosis and also to study the jaw reflex in patients with structural lesions affecting the fifth cranial nerve. Studies have begun and suggest that in these cases also this test may show abnormality. Abnormality of the jaw reflex in mesencephalic lesions has also been documented by Hufschmidt and Spuler (1962).

SUMMARY

Electromyographic recording of the jaw reflex has shown absence or increased latency on one or both sides in 12 of an unselected series of 32 patients with multiple sclerosis. In none of these was there any evidence of lower motor neurone fifth cranial nerve involvement on clinical examination. Suggestions are made as to the probable site of the lesion causing this finding and its value in diagnosis.

It is planned to extend this investigation and to study the jaw reflex in other lesions of the fifth cranial nerve.

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ADDENDUM

CLINICAL SUMMARIES AND JAW REFLEX LATENCIES IN 32 PATIENTS WITH MULTIPLE SCLEROSIS

Case No.	Age	Sex	Duration (yr)	Jaw reflex latency			Clinical summary
				L	R	L-R	
1	21	F	3	18.0	Abs	—	Left optic neuropathy 3 yr before admission, with remission, followed by spastic paraparesis. Also diabetes mellitus
2	33	M	3	14.0	8.0	6.0	Spastic paraparesis 3 yr before admission, with remission. Later developed cerebellar ataxia
3	45	M	5	8.0	7.0	1.0	Chronic spastic paraparesis, with periods of exacerbation and remission. Uninhibited neurogenic bladder
4	50	F	5	7.5	7.5	0	Left retrobulbar neuropathy 5 yr before admission, with remission. Later had posterolateral sclerosis syndrome, with partial remission of the spastic component
5	21	F	4	8.5	6.5	2.0	Right optic neuropathy with remission, followed by cerebellar ataxia, truncal mainly
6	32	F	10	9.0	7.0	2.0	Right retrobulbar neuropathy at onset, with remission, followed by slow development of spastic tetraparesis (on ACTH)
7	32	F	10	7.0	Abs	—	Right retrobulbar neuropathy 10 yr previously, followed by mild spastic paraparesis
8	47	F	10	7.5	7.5	0	Chronic, severe, spastic paraplegia. Normal CSF and myelogram
9	25	F	3	8.0	9.0	-1.0	Right retrobulbar neuropathy with remission, followed by right 6th nerve palsy, with remission. Also cerebellar ataxia, mild
10	25	F	3	8.5	14.0	-5.5	Cerebellar ataxia, dissociated nystagmus and spasticity of lower limbs. Course of exacerbation and remission
11	21	M	3	Abs	Abs	—	Exacerbations (3) and remissions of severe spastic paraparesis. Also cerebellar ataxia and posterior column involvement in lower extremities. Negative myelogram
12	42	M	4	7.5	7.0	0.5	Chronic spastic paraplegia. No evidence of mass lesion
13	52	M	9	8.0	8.5	-0.5	Chronic spastic paraplegia. Myelogram: No evidence of mass lesion
14	49	F	3	9.5	8.5	1.0	Episode of severe truncal ataxia, followed by mild spastic paraparesis. Myelogram normal
15	46	F	12	13.0	9.5	3.5	Episode of retrobulbar neuropathy. Severe spastic paraplegia. Cerebellar ataxia
16	48	F	6	Abs	8.0	—	Chronic paraplegia (intense lower limb spasticity). Normal myelogram
17	29	F	1½	7.5	8.0	-0.5	Right optic neuropathy 1½ yr previously, with remission. Recurrence eight weeks before admission with dissociated nystagmus and right nuclear facial palsy
18	58	M	10	8.0	8.0	0	Chronic spastic paraplegia, without myelographic evidence of mass lesion
19	30	F	3	10.5	10.0	0.5	Mild spastic paraparesis, without myelographic abnormality
20	35	F	3	Abs	Abs	—	Spastic paraparesis with remission. Later had diplopia, and was found to have internuclear ophthalmoplegia
21	33	F	3	9.0	13.0	-4.0	Severe cerebellar ataxia, mainly truncal. No cause found on investigation
22	42	M	3	13.0	9.0	4.0	Incapacitating cerebellar vermis syndrome 'dentato-rubro-thalamic' type tremor. No evidence of structural cause
23	57	F	3	8.5	9.5	-1.0	Spastic paraparesis. Negative myelogram
24	51	F	6	9.0	9.0	0	Moderate spastic paraparesis, with negative myelography
25	32	F	3	11.0	Abs	—	Spastic paraplegia. Negative myelography ¹
26	19	F	3	8.5	9.0	-0.5	Recurrent episodes of spastic paraparesis, with some posterior column and cerebellar signs in lower limbs
27	29	M	3	7.5	6.0	1.5	Spastic paraparesis, with moderate cerebellar ataxia
28	29	F	10	Abs	Abs	—	Spastic paraparesis (negative myelogram)
29	54	F	3	8.0	8.0	0	Mild spastic paraparesis (negative myelogram)
30	51	F	3	8.0	8.0	0	Mild spastic paraparesis (negative myelogram)
31	54	F	3	8.5	8.0	0.5	Right spastic hemiparesis, with mainly truncal ataxia, proprioception impaired lower limbs
32	25	F	3	9.0	9.0	0	Truncal ataxia, scanning speech and hyperreflexia

¹Bilaterally normal latencies and duration at re-examination four months later, despite absence of clinical change.