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The Palmomental Reflex in Parkinson's Disease

Comparisons With Normal Subjects and Clinical Relevance

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• **We tested 356 normal subjects and 109 parkinsonian patients for the palmomental reflex. The total incidence of the reflex was 16.3% in normal subjects, increasing with age. In parkinsonian patients, the overall incidence of the reflex was 71.5%, without clear effect of age. A positive correlation was found between degree of akinesia and incidence as well as intensity of the reflex. In the dyskinetic patients, the reflex was seldom elicited, and, if so, it was small. Modifications of the characteristics of the response could be disclosed in parallel with variations of the patient's clinical status. These findings suggest that the presence of a palmomental reflex in parkinsonian patients could indirectly reflect the decrease of dopaminergic activity in the nigrostriatal pathways.**

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The palmomental reflex (PMR)[1] is defined as a fleeting unilateral contraction of the chin muscles on stimulation of the thenar eminence of the ipsilateral hand.[2] This reflex has been often observed in patients suffering from Parkinson's disease,[3-6] but little is known about a possible relationship between the characteristics of the reflex and the patient's clinical status, the treatment, or the evolution of the disease.

To solve these problems, we conducted a clinical study of the palmomental reflex in normal subjects of different ages and in parkinsonian patients.

SUBJECTS AND METHODS

Three hundred fifty-six normal subjects of different age groups and 109 parkinsonian patients (mean age, 69.2 years; mean duration of the disease, 4.8 years) were tested for the palmomental reflex by the same investigators.

Parkinsonian patients were tested as outpatients in most cases. Several variables were recorded for each patient at each visit: duration of the disease, disability score (Webster's scale)[7] ongoing treatment, presence or absence of tremor and rigidity, and degree of akinesia (scored 0 to 4), using the following measures: quality of speech, step length, writing, and time needed to walk 5 m. Particular attention was paid to the presence of levodopa-induced dyskinesia and impairment of cognitive functions, the latter being evaluated with the Mini Mental State Test.[8] Parkinsonian patients with a score under 20 on this test were considered to suffer from associated dementia.

The PMR was detected by careful visual inspection of the chin muscles with the subject at rest with slightly opened mouth. The stimulus was a rapid, nonpainful stroking over the thenar eminence given with the tip of a ballpoint pen.

For each normal subject and parkinsonian patient tested, the following characteristics of the reflex were noted: presence or absence, unilaterality or bilaterality, intensity (scored as 1+, 2+, or 3+), and habituation. The reflex was considered to habituate if no response could be elicited after five successive stimuli repeated at 1-s intervals.

Among the 109 parkinsonian patients, 84 were tested several times, for up to two years in 36 of them. Moreover, 14 "de novo" parkinsonian patients (mean age, 56 years; mean Hoehn and Yahr stage, 2.1)[9] were tested several times during two years after onset of levodopa therapy. Modifications of the reflex during that time were noted.

Of the 109 patients, 12 were hospitalized because of side effects or loss of efficacy of the treatment or for marked disability. They were tested daily for the PMR, and possible modifications of the reflex during "on" and "off" periods were noted, as well as changes observed during episodes of dyskinesia. Patients were considered "off" when parkinsonian symptoms were more evident, despite levodopa therapy.

RESULTS

Among the 356 normal subjects, 58 (16.3%) had a definite PMR. It was bilateral in all cases and was always weak (1+). All normal subjects with a PMR showed habituation. The incidence of the PMR increased with age.

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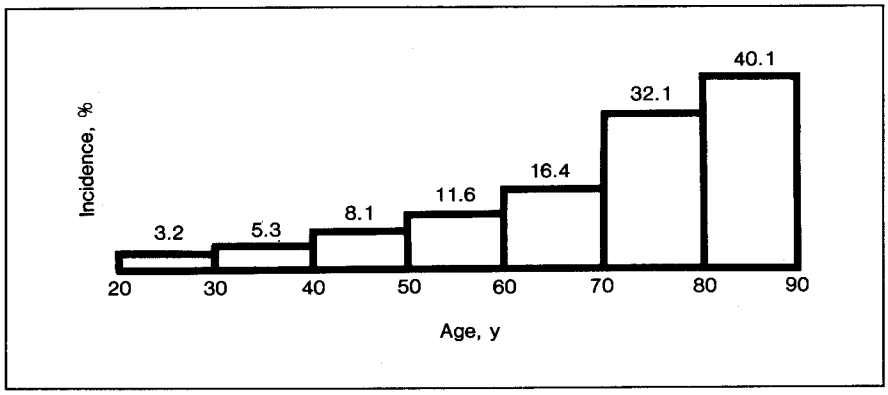

Fig 1.—Incidence of palmomental reflex in 356 normal subjects by age group.

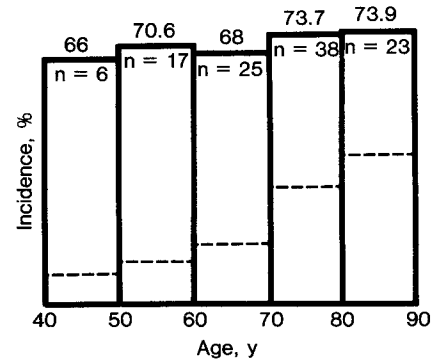

Fig 2.—Incidence of palmomental reflex in 109 parkinsonian patients by age group.

Table 1.—Incidence of Palmomental Reflex and Disability Score by Duration of Parkinson's Disease

	Parkinson's Disease		
	<1 y	>5 y	De Novo Untreated Patients
No. of patients	18	37	14
Mean Webster's score	10.6	18.3	11.1
Palmomental reflex, No. (%)	12 (67)	30 (81)*	10 (71)

*$P < .005$ vs patients with Parkinson's disease for less than one year, χ^2 test.

Table 2.—Relationship Between Degree of Akinesia and Incidence and Intensity of Palmomental Reflex in Nondyskinetic Parkinsonian Patients

	No. of Patients	Palmomental Reflex,* No. (%)	Intensity of Reflex,* No. of Patients			
			0	1+	2+	3+
All patients	85	75 (88)	10	16	30	29
Degree of akinesia						
1	12	8 (67)	4	5	2	1
2	19	15 (79)	4	3	8	4
3	34	32 (94)	2	6	13	13
4	20	20 (100)	0	2	7	11

*1+ indicates mild; 2+, moderate; and 3+, severe.

From 3.2% in the third decade, it reached 40% in the ninth decade (Fig 1).

In the 109 parkinsonian patients, the overall incidence of the reflex was 71.5% (78/109), without a clear increase with age (Fig 2). The intensity of the response was 1+ in 18 patients (16.5%), 2+ in 31 (28.4%), and 3+ in 29 (26.6%). No habituation could be disclosed in 53 patients (48.6%). The difference of incidence of the reflex between parkinsonian patients and normal subjects was significant for each age group (χ^2 test).

The PMR was observed in 12 (67%) of 18 patients who had suffered from Parkinson's disease for less than one year and in 30 (81%) of those 37 who had been affected for more than five years. The difference between the two groups was statistically significant ($P < .005$, χ^2 test). However, if one takes into account the disability score (Webster's scale) at the visit, the incidence of the reflex was similar for groups of patients with the same disability score, whatever the duration of the disease (Table 1). The same was observed for intensity and habituation of the reflex.

Possible relationships between the PMR and the major symptoms of Parkinson's disease were sought. Tremor did not seem to influence the incidence and characteristics of the reflex; PMR was present in 56 (71.8%) of 78 patients with tremor and in 22 (71%) of 31 patients without tremor (not significant). Lack of habituation was observed in 37 patients with tremor (47.4%) and in 16 patients without tremor (51.6%) (not significant). No significant difference in the intensity of the response could be disclosed between the two groups. Rigidity can be hard to quantify and is linked to akinesia in most cases, so we were not able to evaluate the influence of this isolated sign on the PMR.

A clear correlation was found between degree of akinesia at the visit and incidence as well as intensity of the reflex (Table 2). In the same way, lack of habituation was observed in only one third of patients with mild akinesia (scored 1) and in 80% of those who were highly akinetic (scored 4) ($P < .001$, χ^2 test). For this evaluation, parkinsonian patients presenting with levodopa-induced dyskinesia were omitted, for a reason explained below. Among 16 patients with marked unilateral predominance of parkinsonism, 11 (68.8%) had a PMR and eight showed a more intense ipsilateral response.

The most striking feature of this study was that patients suffering from levodopa-induced dyskinesia usually had no elicitable reflex. A response was observed in only three patients (12.5%), with intensity scored 1+ in two and 2+ in one. Habituation was normal. The difference with the main group was highly significant ($P < .001$, χ^2 test). If these patients are excluded from the whole group, the incidence of the reflex in nondyskinetic subjects reached 88.2%, and no habituation was observed in 62.3% of them.

In 13 demented parkinsonian patients, the incidence of the reflex was 69% (nine patients). Lack of habituation was found in seven patients (54%). Dementia associated with Parkinson's disease does not seem to modify the incidence of the reflex in this study (not significant, χ^2 test).

Of the 109 parkinsonian patients, 84 were tested several times for the PMR. Nine of the subjects with a definite PMR at the first test developed dyskinesia during the following months. The reflex disappeared in eight of them and reappeared after reduction of the doses of levodopa in six, within a delay of two months and concomitant with a disappearance of dyskinetic movements.

Seven patients were initially seen

with a low or moderate akinesia score (mean, 1.6) and devoid of PMR. Two years later, these were more markedly akinetic (mean score, 2.4), and four of them showed a clear PMR (1+ in one, 2+ in three). After reinforcement of their treatment, the score for akinesia was reduced (mean, 1.9) and the reflex disappeared in three patients when seen three months later.

In 21 patients with a present reflex, the doses of levodopa had to be increased because of increased disability, and the reflex either disappeared or had a lower intensity in 13 of them seen three months later, in parallel with an improvement of the clinical status.

Fourteen untreated parkinsonian patients were followed up for two years. Their mean score for akinesia at the first visit was 2.1 and the incidence of PMR was 71.4% (ten patients). No habituation was found in six (42.8%), and the intensity of the reflex was 1+ in three, 2+ in four, and 3+ in three. After six months of therapy, the mean score for akinesia was 1.3 and the reflex disappeared in four patients. Among the remaining six, three had a less marked response (1+ in three, 2+ in two, and 3+ in one). Lack of habituation was found in two. The four patients whose reflex disappeared had nearly complete resolution of parkinsonian signs at that time. After two years, the mean score for akinesia was 1.6, and eight patients then showed a PMR (1+ in four, 2+ in three, and 3+ in one). No habituation was observed in three.

Among the 12 patients who had been hospitalized and checked daily for the reflex, no modification of its characteristics could be disclosed over a single day, between "on" and "off" periods. However, in three subjects with an unquestionable reflex on admission (1+ in one, 2+ in two), the response disappeared with a delay of one week after treatment was reinforced. At that time, dyskinetic movements were obvious in two of the three patients.

COMMENT

In previous studies,[1-3,5,6] reported incidences of PMR were variable, as were the conclusions about possible clinical relevance of this sign. These discrepancies can be partly explained by a lack of standardization of the methods. Gossman and Jacobs,[4] using a stimulus similar to ours, did not find increased incidence of PMR in parkinsonian patients compared with age-matched controls. However, they gave no indications about the number of patients presenting with dyskinesia, a fact that might have influenced the incidence of the PMR. The same authors did not disclose a correlation between incidence of the PMR and the clinical status of the patients, but no particular attention has been paid to the degree of akinesia. On the contrary, Klawans and Paulson[6] found, as we did, an increased incidence of the reflex in Parkinson's disease, but they considered PMR present only if habituation was lost. This perhaps explains why they did not observe modifications of the response after the start of levodopa therapy, the intensity of the response being not taken into account. They observed an increased incidence of PMR in more severely affected patients but did not mention a particular relation with akinesia. They concluded that there is no relationship between presence of PMR and the dopaminergic systems.

A very low incidence of the PMR in dyskinetic parkinsonian patients has not been reported in previous studies. This should not be interpreted as a masking of the response by involuntary movements, for we have also observed lack of response in subjects whose dyskinesia did not involve chin muscles. Moreover, as observed for other polysynaptic reflexes, a small contraction of the chin muscles should enhance and not reduce the response. A low incidence of PMR has already been reported in senile demented patients with or without spontaneous or tardive dyskinesia.[10] Thus, it is unlikely that diffuse neuronal loss is an acceptable explanation for the occurrence of the reflex. Moreover, parkinsonian patients with associated dementia do not show increased incidence of the reflex.[6] The PMR is not disease specific and can be observed in various neurologic disorders such as motor neuron disease, stroke, and various encephalopathies.[2,3] so that the mechanisms involved in its genesis are likely multiple.

The pathways involved in the appearance of this reflex are not well known, except for the peripheral ones.[2,11] Afferents are probably through nociceptive and tactile sensory fibers originating from the thenar eminence and the fingers, for the reflex can be elicited by stimulations of the fingers, without participation of proprioceptive Ia fibers. Moreover, the electrophysiologic and sometimes clinical response is reduced or abolished after procainization of the reflexogenic zone and an ischemia of the forearm that does not suppress the tactile sense.[2,11] The peripheral efferent pathway is the facial nerve.[11]

However, the central relays of this reflex are still unknown. One might speculate that thalamic nuclei are involved. Projections from the striatum to the thalamus could modulate the characteristics of the reflex with, in parkinsonism, suppression of inhibition usually exerted on the circuits of the reflex.

The results of this study show that the PMR is seldom observed clinically in normal subjects, while in parkinsonian patients it is often elicited, its incidence and intensity being correlated with the degree of akinesia. Conversely, it is usually absent in dyskinetic parkinsonian patients. In a single patient, changes in the clinical status can be reflected by modifications of the reflex appearing within a few weeks. Tremor and associated dementia do not modify the incidence of the PMR in parkinsonian patients.

An increased incidence of the reflex in Parkinson's disease could indirectly reflect the decrease of dopaminergic activity in the nigrostriatal pathways. In clinical practice, looking for this reflex might help determine whether the treatment correctly matches the dopamine deficit and could easily give an objective though indirect evaluation of the patient's dopaminergic status.

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