Physiological mechanisms of rigidity in Parkinson's disease

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SUMMARY Electromyographic responses of triceps surae and tibialis anterior produced by dorsiflexion stretch were studied in 17 patients with Parkinson's disease. Most patients showed increased muscular activity when attempting to relax. A few patients showed an increase of short-latency reflexes when relaxed and when exerting a voluntary plantarflexion prior to the stretch. Many patients showed long-latency reflexes when relaxed and all but one showed long-latency reflexes with voluntary contraction; and these reflexes were often larger in magnitude and longer in duration than those seen in normal subjects. Unlike the short-latency reflex, the long-latency reflex did not disappear with vibration applied to the Achilles tendon. The long-latency reflexes and continuous responses to slow ramp stretches were diminished at a latency similar to the beginning of long-latency reflexes when the stretching was quickly reversed. Dorsiflexion stretch also frequently produced a shortening reaction in tibialis anterior. Of all the abnormal behaviour exhibited by the Parkinsonian patients only the long-latency reflex magnitude and duration correlated with the clinical impression of increased tone. The mechanism of the long-latency reflex to stretch which is responsible for rigidity is not certain, but the present results are consistent with a group II mediated tonic response.

One of the major manifestations of Parkinson's disease is rigidity. The only symptom unequivocally produced by rigidity is a feeling of stiffness. As a clinical sign, however, this term refers to the phenomenon of increased resistance when stretching a muscle passively. Although some features of rigidity have been characterised, the detailed physiology is still unknown. Possible mechanisms include an exaggeration of the monosynaptic stretch reflex, an exaggeration of the long-latency stretch reflexes, the development of a tonic stretch reflex and the development of a shortening reaction.

The possibility of exaggeration of long-latency stretch reflexes has received much recent attention.

Lee and Tatton¹ observed an enhancement of the long-latency stretch reflexes of the wrist flexors and extensors in subjects with Parkinson's disease. These results have been confirmed by Mortimer and Webster^{2,3} in biceps brachii and by Chan *et al*⁴ in tibialis anterior. Marsden *et al*⁵ ascribed an increase of long-latency reflexes of the flexor pollicis longus to the fact that subjects with Parkinson's disease are not truly relaxed at rest and have only an apparent increase of the long-latency reflexes.

We have studied the EMG responses of triceps surae and tibialis anterior to passive dorsiflexion of the ankle in a group of patients with Parkinson's disease. This attempt follows our recent demonstration of long-latency stretch reflexes in triceps surae⁶ and seeks further understanding of which mechanisms are responsible for the rigidity.

Materials and methods

The study was performed on 17 patients with Parkinson's disease ranging in age from 40–80 years, who gave informed consent to participate. The signs, symptoms, and medications of the patients are noted in the table. Rigidity was assessed clinically prior to the study. Methods for

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Table Clinical and physiological data

Patients	Duration of illness (yr)	Rigidity	Background at rest	Magnitude				LLR torque duration (ms)	Medications
				SLR* no torque	LLR†	SLR* torque	LLR†		
1	1½	Normal	6%	3	1	5	1	41	Sinemet 10/100 3 × day
2	3	Mild	6%	5	1	13	1	42	Sinemet 25/250 3 × day
3	4	Mild	11%	81	0	78	2	23	Sinemet 25/250 6 × day
4	14	Mild	10%	40	0	28	2	33	Sinemet 10/100 6 × day Artane 2 mg 3 × day Symmetrel 100 mg 2 × day Inderal 40 mg 5 × day
5	1	Mild	12%	14	1	20	1	43	None
6	2	Mild	9%	50	0	32	1	26	None
7	2	Moderate	25%	8	2	23	0	0	Sinemet 25/250 3 × day
8	1	Moderate	20%	18	1	13	2	73	Sinemet 25/250 4 × day
9	4	Moderate	10%	8	1	20	1	60	Lithium 300 mg 3 × day
10	4	Moderate	12%	19	1	19	2	74	Sinemet 10/100 4 × day
11	4	Moderate	8%	7	2	8	2	79	Sinemet 10/100 5 × day Bromocriptine 2.5 mg 5 × day
12	4	Moderate	4-5%	2	1	5	2	71	Sinemet 10/100 5 × day Artane 1 mg 3 × day
13	8	Moderate	12%	6	0	—	—	—	L-Dopa 3 × day Artane 2 mg 4 × day
14	3	Moderate	19%	10	1	10	1	71	Sinemet 12.5/125 6 × day Symmetrel 100 mg 2 × day Kemadrin 5 mg 4 × day
15	20	Severe	—	—	—	18	2	68	Pagitone 1.25 mg 2 × day
16	4	Severe	20%	33	2	—	—	—	Sinemet 10/100 7 × day
17	4	Severe	10%	34	2	77	2	38	Sinemet 10/100 6 × day

* The magnitude of the SLR for each patient is the value of the EMG at 3600°/s² acceleration which is obtained from linear interpolation from the actual data points.

† For the LLR, 0 means absent, 1 means normal for the amount of background contraction and 2 means abnormally large magnitude for at least one acceleration value.

— means not studied.

studying the EMG responses of triceps surae and tibialis anterior have been described in detail in an earlier paper.⁶ Briefly, the subject sat in a chair with the knee flexed at 90° with the foot strapped to a platform which was attached to the spindle of a torque motor. A strain gauge was incorporated into the platform so that the torque exerted by the subject on the platform could be measured and this information was made available to the subject by deflection of a meter needle. Using a PDP 11/10 computer and a feedback circuit the platform could be programmed to make ankle displacements at different velocities. The platform reached the specified velocity in approximately 50 ms. EMG with surface electrodes was recorded from triceps surae and tibialis anterior. The EMG signals, angular position of the ankle and the torque on the platform, were sampled by the computer at 2 ms intervals. The EMG was rectified and filtered before collection. The latency of the EMG reflexes was measured from the electronic command to move the pedal (thus clearly overestimating the biological latency). The durations were measured by visual inspection and the amount of EMG activity in each burst was measured by integration. The integration values were normalised to the EMG activity produced with a maximal voluntary effort (method 2 from reference 6). Hence activity equalling maximum voluntary effort was given a value of 100.

The initial angle of the ankle was -10° and at random times the ankle was dorsiflexed to +5° at one of three specified velocities, 100°, 150° or 200°/s. Each velocity was repeated 10 times and the results were averaged separately. Since peak velocity was attained more quickly with faster velocities, acceleration was a better single descriptor of the

mechanical event than the specified velocity itself. Hence the results are reported in terms of acceleration. Two conditions were studied. In the first there was no background torque exerted by the patient on the platform. The subject sat in a chair and was asked to relax as much as possible. In the second the subject voluntarily exerted a plantar flexing background torque onto the platform of 20% of his maximum force while waiting for the perturbation.

The voluntary response to muscle stretch was studied with the patient's foot relaxed on the platform. He was given one of two tasks to perform when he perceived the perturbation (1) *push* (plantarflexion of the ankle) or (2) *assist* (dorsiflexion of the ankle). All the perturbations were from -10° to 5° and the specified velocity was 150°/s. Vibration was applied at 100-150 Hz with a physical therapy vibrator (Foredom Electric Company Series 37) to the Achilles tendon prior to the dorsiflexion of the foot, with and without background force exerted by the subject. In other experiments the ankle was displaced in two consecutive phases: (1) from -10° to 5° at 15°/s and then from +5° to -10° at 200°/s and (2) from -10° to +5° at 150°/s and then from +5° to -10° at 150°/s.

Results

STRETCH REFLEXES OF TRICEPS SURAE AT "REST"

A short-latency reflex was present in all the patients studied (fig 1A), and this response increased in amplitude with increased acceleration of stretch. The

Fig 1 EMG response of lateral gastrocnemius muscle for patient 12 to rapid dorsiflexion stretch while the patient was at rest (A) and while exerting 20% background force (B). The top trace is rectified EMG activity, the next trace is ankle angle and the next trace is acceleration of ankle. The traces are the average of 10 single trials.

“magnitude” of the short-latency reflex was calculated for each patient as the value of the EMG at $3600^\circ/\text{s}^2$ acceleration obtained from linear interpolation from the actual data points. Using this method the short-latency reflex was enhanced for 9 out of 16 patients in comparison to normal subjects previously reported⁶ (fig 2). In 12 out of 16 patients the short-latency reflex was followed by EMG activity similar to the long-latency reflexes observed when a normal subject is voluntarily exerting a background force (figs 1A, 3). The amplitude of the long-latency reflexes sometimes increased and sometimes decreased with acceleration. It was not possible to standardise a “magnitude” for the long-latency reflexes since their behaviour was not monotonic. For this reason the long-latency reflexes were simply described as absent, normal magnitude or large magnitude, and the magnitude was considered large if the long-latency reflexes exceeded normal for any acceleration. In our patients with Parkinson's disease the triceps surae were not completely at rest preceding the stretch, but showed a continuous

background contraction. By integrating the EMG activity in the first 100 ms of the record (prior to any reflex response) the patients showed activity varying between 4.5% to 25% of the maximal force (see table). In order to assess the role of background force on the results we carried out further experiments on normal subjects. Background EMG activity at rest was usually less than 4% of maximal force, but could be as high as 7%. The short-latency reflex was present even if the muscle was totally relaxed and did not change much with background force, but increased slightly with background force up to 50% of maximum. The long-latency reflexes (one or more discrete components) were absent at rest, appeared with a background force between 5–10% of maximum, became larger in proportion to background force up to about 30% after which they did not increase. Considering the normal results, all but three patients demonstrated increased background EMG activity at rest. In relation to the short-latency reflex, the three patients with normal background had normal amplitude. Five patients had markedly increased amplitude, more than would be expected even if they were exerting 20% voluntary force, and in none of these cases was the background contraction more than 20%. In four patients the short-latency reflex was increased, but within the range of a normal subject exerting torque. In relation to the long-latency reflexes, the three patients with normal background activity all showed long-latency reflexes at rest and their magnitude was usually within the range of amplitude of those of normal subjects exerting 20% background force. In general the range of background EMG activity was less in those patients who did not show long-latency reflexes at rest (6–12%) and the magnitude in different patients was only loosely correlated with the amount of background force.

The latency of short-latency reflexes varied from 50–80 ms and shortened with faster stretching, and the duration varied from 20 to 40 ms and both of these

Fig 2 Number of patients showing different magnitudes of the short-latency stretch reflex while at rest and while exerting 20% background force. The light bars indicate normal behaviour and the dark bars indicate abnormal behaviour.

Fig 3 Number of patients showing different magnitudes of long-latency stretch reflexes while at rest (light bars) and while exerting 20% background force (dark bars).

parameters are within the normal range. The long-latency reflexes when present appeared at latency of 84–122 ms with durations of 30–75 ms.

STRETCH REFLEXES OF TRICEPS SURAE WITH BACKGROUND FORCE

When the patient voluntarily exerted a background force of 20% of his maximum force, the short-latency reflex was present at slightly shorter latency and with the same duration of the same response evoked without background force (fig 1B). This response increased in magnitude with increased acceleration of stretch but was not often increased in magnitude with respect to the circumstance when the subject was requested to relax (fig 2). Three of 15 patients showed increased magnitude of the short-latency reflex. Long-latency reflexes appeared at latencies of 80–110 ms and were present in all but one of the patients. The long-latency reflexes increased in magnitude in comparison to the long-latency reflexes observed without background force and were enhanced in magnitude with respect to normal values in eight out of 15 patients (fig 3). In three of the patients with enhanced long-latency reflexes an enhancement of short-latency reflex was also present, while four showed no enhancement of the short-latency reflex (one was not studied). It can be noted also that two of the patients with an enhanced short-latency reflex did not show enhanced long-latency reflexes. In normal subjects it was usually possible to divide the long-latency reflexes into two discrete components, but this was often difficult for the patients. Additionally the long-latency activity in the patients often continued without pause beyond a latency of 150–160 ms into the time interval which we considered in normal subjects to be characterised by voluntary activity. With faster velocity of stretch, the long-latency reflexes disappeared in one patient who showed a very large short-latency reflex.

VOLUNTARY RESPONSE

The voluntary plantar flexion movement in response to muscle stretch was studied in nine patients. A large EMG response from triceps surae was present at a latency varying from 170–250 ms. The beginning of the voluntary response often came immediately after a long-latency response without an intermediate pause separating these two bursts. In four patients voluntary dorsiflexion of the foot was performed following the perturbation and an EMG response from tibialis anterior appeared at a latency of 250–300 ms.

EFFECT OF VIBRATION

The effect of vibration applied to the Achilles tendon was studied in seven patients (fig 4). When the patients were trying to be relaxed the short-latency reflex was abolished in all and the long-latency reflexes when present, persisted although diminished in amplitude. When a background force was exerted by the subject the short-latency reflex was markedly suppressed or absent in all, but the long-latency reflexes persisted in all but two patients. The long-latency reflexes in this circumstance were decreased by vibration approximately 10% to 50% in the different subjects.

RESPONSE OF TIBIALIS ANTERIOR

In 11 of the 17 patients a large phasic EMG response was recorded from the tibialis anterior after stretching the triceps surae (shortening reaction).

DOUBLE RAMP DISPLACEMENT

In the first series of experiments the first displacement was a slow dorsiflexion (15°/s) in the attempt to induce tonic EMG activity in triceps surae, and the second displacement was a fast plantar flexion (150–

Fig 4 Effect of vibration applied to the Achilles tendon on the stretch reflex. In each of the four parts of the figure are shown rectified EMG (with identical scaling) from lateral gastrocnemius and ankle position. The displacement is 14°. Each trace is the average of 10 individual trials.

100°/s) of the ankle. Fifteen patients were studied. In eight patients the slow dorsiflexion elicited gradually increasing involuntary muscular activity (fig 5). In all these patients a fast plantar flexion of the foot abruptly abolished or markedly decreased the tonic EMG activity at a latency ranging in the different patients from 100 to 150 ms. In the remaining seven patients no EMG activity was induced by the slow dorsiflexion of the foot.

In the second series of these experiments the ankle was displaced quickly from -10° to 5° at 150°/s and then quickly back to -10° at 150°/s (triangle stretch). Eight patients were studied. This type of stretch differed from the standard stretch mainly by virtue of having no plateau phase; the dynamic dorsiflexion phase was similar. In all patients the short-latency reflex was similar to that seen with the standard maintained stretch, but the long-latency reflexes were diminished.

CLINICAL EVALUATION

The table lists the clinical assessment of rigidity of the patients together with the quantitative results of ankle stretching. The rigidity was classified in four degrees: absent, mild, moderate and severe, but for the purposes of analysis the patients were divided into two groups (1) absent and mild, and (2) moderate and severe. Chisquare tests were carried out to look for correlations between the degree of tone and background contraction (normal or elevated), short-latency reflex (normal or large), long-latency reflexes (present or absent), magnitude of long-latency reflexes with background force (normal or large) and presence or absence of shortening reaction. Correlations with the degree of tone were found only for long-latency reflexes at rest ($p < 0.025$) and

magnitude of long-latency reflexes with background force ($p < 0.01$). The average duration of the long-latency reflexes with background force was 35 ms for the group with absent-mild tone and was 67 ms for the group with moderate-severe tone and this was also a significant difference ($p < 0.01$). This difference might be even more extreme than it appears since long-latency reflexes often merged with subsequent later activity so that the true duration might well be longer than what we have measured.

Discussion

SHORT- AND LONG-LATENCY STRETCH REFLEXES Andrews *et al.*,⁷ Dietrichson⁸ and McLellan⁹ have previously shown that phasic stretch reflexes are normal or slightly increased in Parkinsonian patients which in general is in accord with the common clinical findings. An increase of the stretch reflexes can more frequently be demonstrated during slow and maintained stretching.^{7,8} This phenomenon can be called an enhanced tonic stretch reflex and is the electrophysiological correlate of the clinical sign of rigidity. The observation of Lee and Tatton¹ that subjects with Parkinson's disease have an increase of long-latency stretch reflexes is provocative because it suggests a neuronal pathway apparently different from the monosynaptic pathways that could sustain the rigidity without enhancing the phasic stretch reflex. Subsequently, a direct correlation between the amount of rigidity measured in patients with Parkinson's disease by quantitative slow stretches and the magnitude of the long-latency responses of biceps and triceps has been shown by Mortimer and Webster.^{2,3} Marsden *et al.*⁵ suggested that the increase of the long-latency reflexes of the flexor pollicis longus was due to the fact that rigid patients are not truly relaxed at rest. On the other hand, Tatton *et al.*¹⁰ reported that the increase of long-latency stretches was out of proportion to baseline activity.

Thirteen of 16 patients reported here showed an increase of baseline EMG activity when they were at rest. This baseline activity is important to note, but was not responsible alone for the clinical impression of increased tone. Nine of the 16 showed an increased short-latency reflex at rest and five patients showed an increased short-latency reflex compared to normal even when considering background activity. Three of these nine patients showed an increase of short-latency reflexes when exerting voluntary background force. However, these changes in short-latency reflexes did not correlate with the clinical impression of tone.

The presence of an involuntary background contraction before the stretch could only to some extent explain the presence of long-latency reflexes

Fig 5 EMG response of lateral gastrocnemius muscle to abrupt plantarflexion for $+5^\circ$ to -10° following a slow dorsiflexion ramp stretch which began prior to the beginning of the illustration at -10° . The top trace is rectified EMG in arbitrary units and the bottom trace is ankle position. Each trace is the average of 10 trials.

when the patients were requested to relax, since four patients had long-latency reflexes greater than that seen even with background force. In addition, the presence and the magnitude of the long-latency reflexes were not always directly related to the amount of baseline activity. When the patients were exerting background force, the magnitudes of the long-latency reflexes in eight were greater than that which could be explained by the level of background force. These increases in magnitude and also in duration were correlated with increased tone in agreement with the previous observations of Mortimer and Webster.²³

The short-latency reflex certainly represents the monosynaptic stretch reflex but the pathway for the long-latency reflexes is not clear. A trans-cortical loop has been suggested^{15,11-16} but spinal mechanisms alone can be responsible.¹⁷ We have previously suggested⁶ that the long-latency reflexes of triceps surae arise from serial responses to multiple spindle discharges, but other mechanisms are also possible. The behaviour of long-latency reflexes in patients with rigidity reported here could be explained by at least two hypotheses. The first is an enhancement of proposed normal stretch reflex mechanisms resulting in an increased response to second and third spindle bursts. In favour of this hypothesis is that the short- and the long-latency reflexes sometimes were increased in the same patient. However, often the long-latency reflexes were selectively enhanced in patients with severe rigidity. The experiments using vibration demonstrated that the mechanism of the monosynaptic short-latency reflex differed from the mechanism of at least some part of the long-latency reflexes; this fact suggests that these reflexes in Parkinsonian patients are not completely generated by a monosynaptic reflex mediated by IA fibres.

The second hypothesis for the abnormal long-latency reflexes in Parkinson's disease is that a new phenomenon in patients with rigidity has been superimposed upon the normal responses. This hypothesis can itself be divided into two possibilities. The first one is an enhanced polysynaptic response to the same inputs active in normal subjects.^{10,18} We cannot exclude this hypothesis definitely; however, the triangle stretch experiments which show a reduction in the long-latency reflexes when the stretch is not maintained, suggest that the long-latency responses depend on continuing afferent input. In addition, the vibration experiment can be considered negative evidence if one accepts the notion that vibration has its effect by keeping the IA afferents so active that they cannot respond to phasic stretch.¹⁹ The second possibility is that there is a new response to inputs not active in normal subjects. In this regard we are attracted to the possibility of group

II afferent input. Matthews²⁰⁻²³ showed that group II fibres add to the excitatory influence in the stretch reflex, participating in the tonic stretch reflex of the decerebrate cat. With the spike-averaging technique it has been shown that group II impulses exert monosynaptic excitatory effects on alpha motoneurons in extensor muscles.²⁴⁻²⁷ All these works suggest that group II afferents have a role beyond the flexor reflex afferent system and can probably participate in the stretch reflex. In addition their slow conduction velocity fits well with the latency of the long-latency reflexes. Group II afferents would not be expected to be stimulated significantly by triangle stretches and indeed with this type of stretch these reflexes are reduced. It was to support this hypothesis that we investigated the effect of vibration applied at high frequency to the Achilles tendon. It has been shown that group II fibres are not sensitive to vibration in the cat;²⁸ vibration in man has clearly some effects, but these effects are probably less at high frequency.²⁹⁻³² In normal subjects⁶ vibration of the Achilles tendon before the stretch suppressed the short- and the long-latency reflexes. In patients with rigidity the short-latency reflex was abolished but the long-latency reflexes persisted even if decreased in magnitude. These arguments are not definitive by themselves, but other evidence in favour of group II mechanism being active in rigidity will be discussed in relation to the tonic stretch reflex.

TONIC STRETCH REFLEX

Increased tone clinically is equivalent to a tonic stretch reflex physiologically. There has been no clear understanding about the physiology of the tonic stretch reflex in normal man since this reflex is absent in the normal relaxed state. Lance *et al*³³ have suggested that the tonic stretch reflex can be recorded during isometric contraction or with reinforcement manoeuvres, but this has not led to any clearer understanding of mechanism. Another view about the tonic stretch reflex in man developed with the discovery that the primary spindle endings in cats^{28,34,35} and in man³⁶ are sensitive to vibration and that continuous vibration behaves like prolonged stretching and induces a tonic contraction of the muscle called a tonic vibration reflex. The tonic vibration reflex has been observed in decerebrate cats²⁰ and in spinalised animals after administration of levodopa.³⁷ In man the tonic vibration reflex has been considered a polysynaptic spinal reflex,^{38,39} and not a cortically mediated reflex.^{30,31} Although McLellan⁹ showed that the tonic vibration reflex is facilitated in patients with rigidity, Lance *et al*³² and Hagbarth²⁹ reported that the tonic vibration reflex of Parkinsonian patients does not differ from that of normal subjects.

We have been discussing the long-latency reflexes as a type of phasic stretch response which is later in time than the classic monosynaptic response, but the long-latency reflexes could be viewed as the beginning of a tonic stretch reflex. Our stretch is ordinarily maintained for at least 3 s so that there is opportunity for tonic stretch reflexes to be manifest. Indeed for some patients these reflexes do have the appearance of continuous activity not divisible into components and this activity might well continue into the time period which we have not analysed because it corresponds with what might be voluntary activity. It is possible that patients manifesting enhanced long-latency reflexes have a tonic stretch reflex superimposed upon the normal phasic long-latency reflex mechanism. The results with triangle stretches supports the idea of a tonic quality of the long-latency reflexes.

We investigated the stretch response in this group of patients with slow stretching of triceps surae. This technique which mimics the clinical method of appraisal of tone is not equivalent to the tonic phase of a step-like stretch, but like tonic stretch can be productive of continuous EMG activity which is not seen in normal subjects. In half of the patients studied the slow stretching induced involuntary EMG activity. In general these patients were the ones with greater increased tone and larger long-latency reflexes. A fast plantar flexion of the foot was subsequently delivered in the attempt to measure the time of disappearance of the continuous EMG response which would tell us the latency of the pathway supporting the activity. In all the patients the time was in the latency range of the long-latency reflexes. This suggests that activity is not supported by the monosynaptic pathway but instead by pathways compatible with long-latency phenomena. As noted above, it is possible that in Parkinson's disease there is a new mechanism superimposed on the normal which might be mediated by group II afferents. The data from animals would certainly suggest that enhanced response to group II afferents could be responsible for tonic stretch reflexes and reflexes to slow continuous stretch. In favour of this hypothesis is the work of Dietrichson⁸ showing that the increased tonic reflex response in Parkinsonian patients depend on the integrity of small-sized nerve fibres, either static fusimotor or group II afferents.

Conclusion

The notion that rigidity is simply a result of enhanced supraspinal drive on alpha motor neurons,^{3,40-44} or a result of alpha and gamma motor neurons being co-activated,^{7,32,40} is not supported by our finding of a poor correlation between the level of background

EMG activity at rest and the clinical impression of tone. The notion that rigidity stems from enhanced fusimotor activity,⁴⁵⁻⁴⁸ seems to have been disproved by the findings of Wallin *et al*⁴⁹ and Burke *et al*⁵⁰ with microneurography. These findings suggest that rigidity arises from a change of central nervous system reflex responsiveness.

Two examples of increased reflex mechanisms in Parkinson's disease are the shortening reaction and the monosynaptic reflex. The shortening reaction acts in an opposite direction to what is necessary to produce the clinical impression of increased tone, and indeed there is no relationship between this phenomenon and degree of rigidity as has been shown also by Mortimer and Webster.^{2,3} The short-latency reflex does act in the correct direction to what is necessary to produce increased tone but it is only enhanced in a few patients and it does not correlate with clinical impression.

Long-latency reflexes are remarkably prominent in Parkinsonian patients, appearing commonly at rest, are often increased in amplitude and are frequently long in duration merging with subsequent activity which may be voluntary. Their magnitude and duration correlate well with clinical impression of rigidity. The long-latency reflexes have the appearance and behaviour of a tonic response and the continuous EMG produced by slow ramp stretches is supported by pathways with latencies similar to the long-latency reflexes. Hence these reflexes seem to play a role in tonic stretch behaviour of the Parkinsonian patient. The mechanism of the abnormal long-latency reflexes needs further investigation, but the observations about them at the present time are compatible with a group II mediated tonic stretch response.

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