

Timing as a prominent factor of the Jendrassik manoeuvre on the H reflex

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SYNOPSIS The influence of the Jendrassik manoeuvre on the myotatic reflex was analysed using a strain gauge as an indicator of the upper extremity movement and the H reflex of the soleus muscle as the test reflex. The most prominent factor responsible for the enhancement was not the speed or the strength of the manoeuvre but the timing from the instruction.

Jendrassik (1883, 1885) was the first to report the phenomenon that the knee jerk was coincidentally enhanced if the subject pulled his hands against each other or made other violent movements of the upper extremities at the time the patellar tendon was struck. Since then, this manoeuvre has been widely used with the explanation that 'the procedure merely takes the patient's mind off his extremities and allows them to relax' or that, on the contrary, 'it removes cortical inhibition from the corresponding anterior horn cells'.

Recent papers on the Jendrassik manoeuvre have generally been concerned with the role of the gamma motor system or the speed and strength of the manoeuvre. Of course, the T response as led off with EMG electrodes should be used as a substitute for the ankle jerk, but the mechanical instrument which applies a precise constant tap for the T response is very difficult to adjust compared with the electrical stimulation required for the H response. Furthermore, like Clare and Landau (1964), Gassel and Diamantopoulos (1964a, b), and Landau and Clare (1964a, b), we have found that the enhancement of H reflex is observed even after gamma fibre blockade, with the conclusion that the primary system should be a direct and/or an

internuncial spinal mechanism which causes the alpha motoneurone excitation (Kohn, 1965; Kawamura, 1969).

While looking for any difference between the brisk and powerful contraction and the slow and sustained manoeuvre, we noticed the important correlation between the timing of the Jendrassik manoeuvre and its enhancing effect on the H reflex.

METHODS

Thirteen normal subjects aged 18 to 31 years, 10 patients with spasticity, and five with rigidity ranging in age from 38 to 59 years were examined repeatedly. The subject was placed in a supine position on a bed in an electrically shielded room. An orthopaedic extension frame resembling a Böhler extension frame was used to fix the examined lower limb while the other limb was extended naturally on a bed. The knee joint was flexed at 165° and the ankle joint fixed at 100° by a foot board. A subject can comfortably maintain the same posture for about an hour. The experiment was interrupted when he wished to change the posture especially of his head.

The stimulators and amplifiers were made by Heiwadenshi Co. (HM 305). The strength and speed of the manoeuvre were measured with the strain gauges of Shinkotsushin Co. (DS 6/PX). A simple circuit was connected with the set to synchronize a trigger for the stimulator and a signal lamp for the manoeuvre.

The H response was elicited by stimulation of the tibial nerve in the popliteal fossa by a silver disc electrode 9 mm in diameter applied with electrode

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jelly. An anode, 3×10 cm thin silver plate covered with cotton gauze and soaked in physiological saline solution, was put on the proximal end of the patella of the same leg. The muscle action potentials were recorded oscillographically from two silver electrodes both 9 mm in diameter, placed longitudinally 2 cm apart on the surface of the soleus muscle, along the medial side of the Achilles tendon about 3 cm distal from the point where the two heads of the gastrocnemius join the Achilles tendon. The stigmatic electrode was proximal.

The stimulus was a rectangular electrical pulse of 1 ms duration. The intensity was usually adjusted to get the maximal amplitude of H response while M response was not yet elicited. The intensity was usually about 45 to 55 V by this stimulator (the output impedance 3 k Ω). The intervals between stimuli were more than five seconds. The amplitude was measured at the largest positive and negative deflections.

The H responses elicited by the adjusted intensity were recorded one hundred times for the control and a frequency distribution curve prepared. The curve in the normal subject closely resembled a normal distribution curve, and so the standard deviation may be used for the comparison. In patients, however, the curve was not always symmetrical so we calculated the area of 80% from the modal value and regarded the amplitudes as the range of the control variation in the patient (Matsushita, 1968). The dotted lines

drawn in the following figures indicate the upper and lower limits of the 80% area from the modal value. When the H response was enhanced above the upper limit we assumed that it was due to facilitation, and under the lower limit we termed it inhibition.

The subject was asked to look at a signal lamp while holding with both hands the grips of a cylindrical load cell in which a strain gauge was mounted. Although we measured the movement by strain gauge, it should be described as the relative isometric contraction of the arms because the subject was asked to be in complete relaxation and just put his fingers on the grips before the manoeuvre, and many muscles contracted in various conditions during the manoeuvre. In fact, this was one of the reasons why we had to choose the tension as indicator of the whole Jendrassik manoeuvre. Sometimes a subject was asked to pull the instrument by one hand while the other end was fixed (the one hand method), or to push a microswitch for the circuit by a thumb (the push button method). It was interesting that the reinforcement was remarkable even by this push button method, with slight motion of a thumb.

He was instructed to pull the grips as soon as the signal lamp was switched on and to stop pulling and relax when the lamp switched off. A green miniature lamp of 5 V was used for the signal to avoid the influence of a strong photic stimulation to the subject. When the signal lamp was switched on or off, the circuit was triggered simultaneously to elicit an H

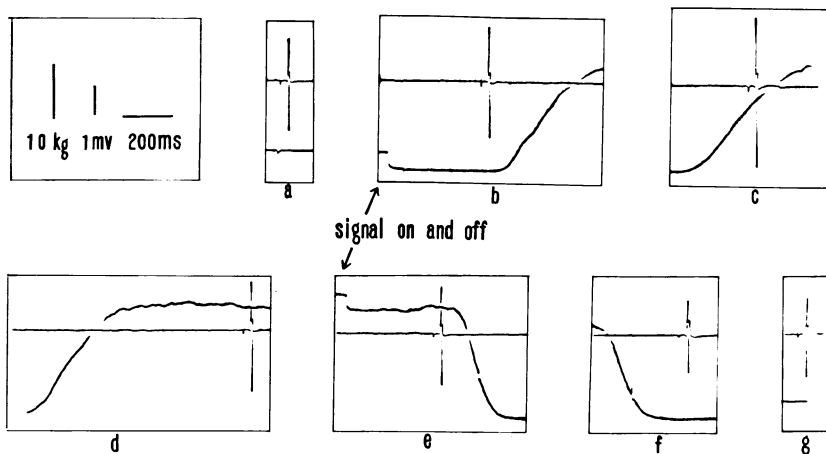


FIG. 1 A and G: control H response. B: enhancement appeared before the deflection of the strain gauge. C: enhancement was pronounced while the strength was increasing. D: effect continued during the manoeuvre, but the enhancement was less than C. E: enhancement declined after the 'off' signal, before the strength had decreased. F: amplitude of H response fluctuated within the lower range of the control variation after stopping the manoeuvre.

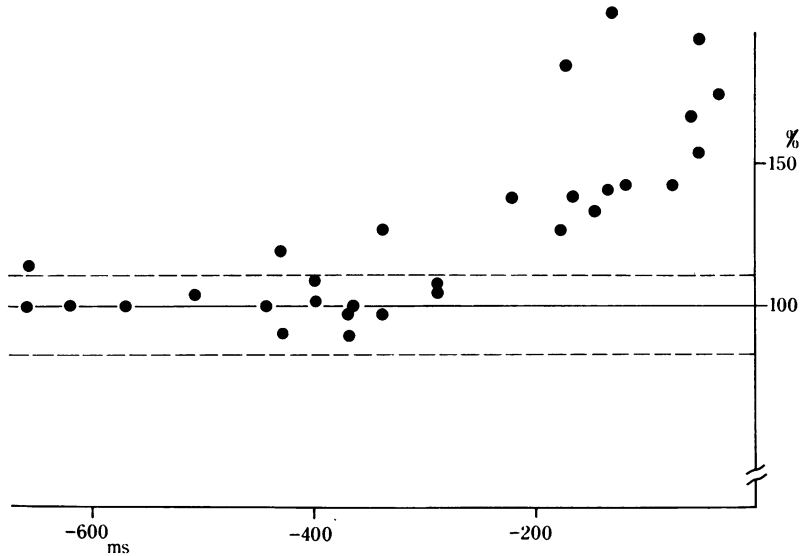


FIG. 2 Effect of 'preceding facilitation' in a normal subject. The enhancement appeared about 300 ms before the movement of the upper extremity. '-600 ms' means the time 600 ms before the initial deflection of the strain gauge signal. 100% in the ordinate means the modal value of the H response amplitude, and the dotted lines indicate the upper and lower limits of the 80% area from the modal value.

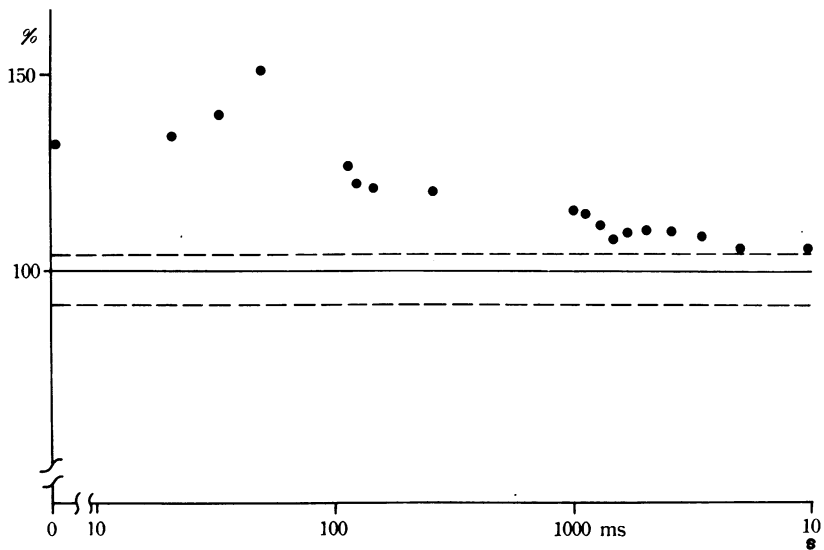


FIG. 3 Effect of sustained contraction of the upper extremity in a normal subject. The enhancement persisted during the manoeuvre, although the effect gradually declined. The abscissa is plotted on a logarithmic scale.

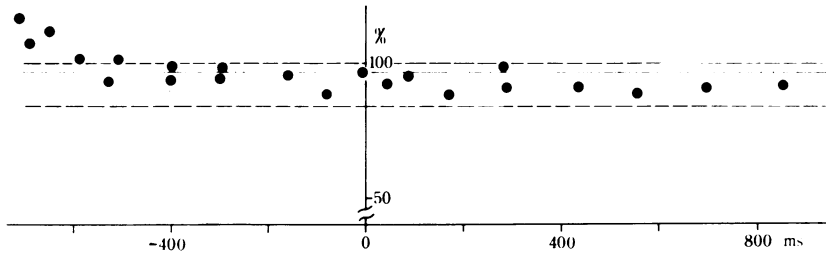


FIG. 4 Effect of 'preceding inhibition' in a normal subject. The enhancement declined after switching the signal 'off' before the relaxation of the upper extremity. Then the amplitude of H response fluctuated within the lower range of the control variation.

response after an optional delay which was set at random.

RESULTS

NORMAL SUBJECTS Thirteen normal subjects were examined repeatedly 36 times. Precaution was taken to prevent any muscle contraction in the neck, shoulders, and legs before the manoeuvre. No voluntary muscle contraction was recorded electromyographically in the soleus muscle during the experiment.

The response time from the recognition of the signal lamp to the onset of the manoeuvre was spread in the range of 260 to 480 ms in the normal subject group. The H reflex was prominently enhanced not only at the onset of the manoeuvre but also before the strain gauge showed any increase of power in the upper extremities (Fig. 1). That is to say the reflex arc of the lower extremities was facilitated from immediately after the perception of the signal which urged him to move his hands. This preceding facilitatory period was observed from about 300 ms before the actual movement of the upper extremities (Fig. 2). There was a slight time lag between the first action potential of the brachioradialis muscle and the deflection of the strain gauge, although it was usually less than 100 ms.

The influence of the photic stimulus of the signal lamp and psychological anticipation of the instruction were examined as follows. The subject was asked to concentrate on the signal lamp but not to start the manoeuvre. While the subject was concentrating on the signal, one hundred H responses were measured. However,

the spread of amplitudes was within the control variation. Another red miniature lamp was then connected with the green lamp. He was asked to pull the instrument only when he noticed the

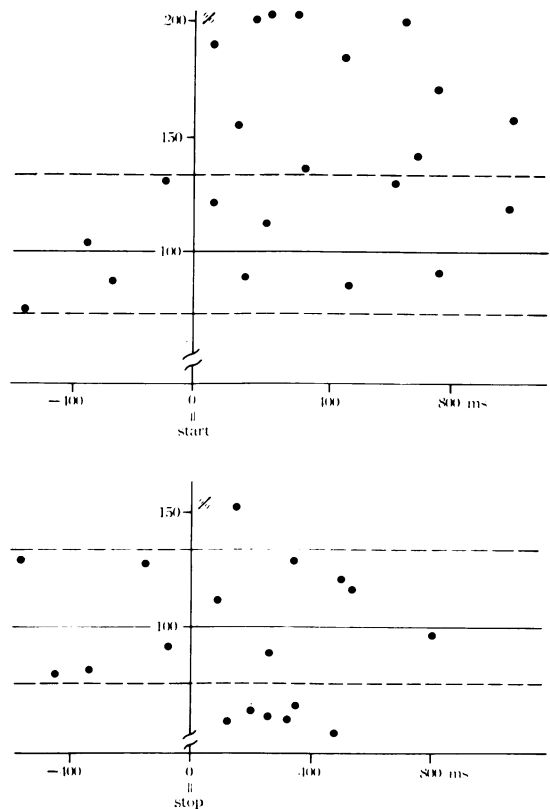


FIG. 5 Effect in a patient with Parkinsonism. The same tendency as in healthy subjects was seen in spite of the large control variation.

green lamp in succession to the red lamp. H responses were elicited at optional intervals from the switching on the red lamp as well as the green lamp. The H responses obtained after the red lamp alone fluctuated within the range of the control variation. On the contrary, those obtained after the green lamp showed marked facilitation even before the movement of the hands and during the manoeuvre.

The duration of the manoeuvre was usually within one second, but it was sustained up to 10 s in some cases. After the onset of the manoeuvre, the H response was augmented most prominently for about 300 ms, then the effect declined gradually, but persisted during the manoeuvre (Fig. 3).

The subject was asked to stop the manoeuvre as soon as the signal lamp was switched off. The augmented H response was diminished before the strain gauge showed a decline of the pulling strength. The response time from the recognition of the signal 'off' to the complete relaxation of the upper extremities ranged from 480 to 880 ms in the normal subjects. The enhancement usually disappeared from 600 to 400 ms before the complete relaxation. After stopping the manoeuvre the amplitudes of H response were spread within the control variation, but they were located near the lower limit (Fig. 4).

PATIENTS WITH PARKINSONISM It was difficult to elicit H responses without concomitant M response in the patients with Parkinsonism so the examination was performed when the amplitude of M response was within one fourth of the H response. Although the control variation was much larger than normal, the H response was significantly enhanced during the Jendrassik manoeuvre. The response time from the signal to the onset of the manoeuvre was longer than in the normal subject. When the patient stopped the movement after the signal 'off', the H responses fluctuated within the broad range of the control variation or were slightly depressed (Fig. 5).

The most prominent sign of the five patients was rigidity, and therefore more consideration should be given to the other cardinal clinical signs of akinesia and tremor.

PATIENTS WITH SPASTICITY Nine patients with hemiplegia due to cerebrovascular accident and

one patient with transverse myelitis were examined. The hemiplegic patients were more or less clumsy in the manoeuvre and sometimes contraction of the arm or shoulder muscles was noted before the strain gauge responded to the

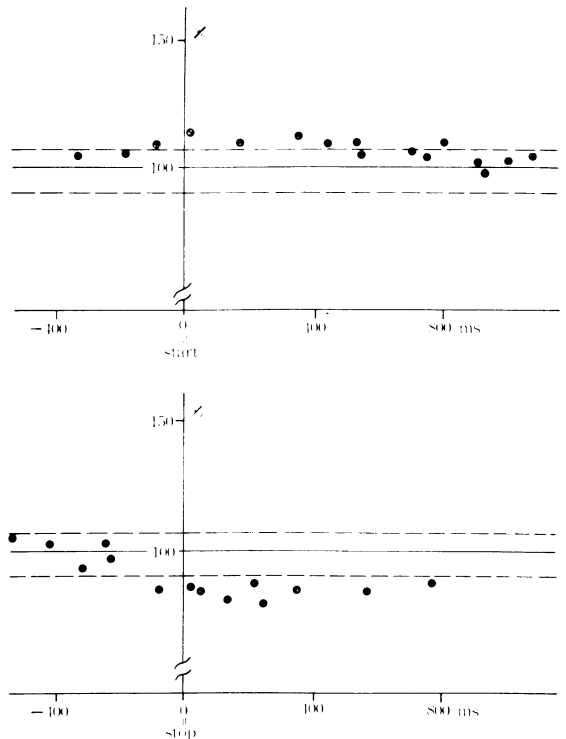


FIG. 6 *Effect in a hemiplegic patient (spastic side.) The maximal H response was of high amplitude and stable in the control variation. The depression after the stopping of the manoeuvre was dominant compared with the initial enhancement. The tendency became more apparent when the intensity of electric stimulus was reduced by half of the maximal H response.*

manoeuvre. Accordingly, the time course of the enhancement was difficult to determine exactly.

The H response of the spastic leg was augmented slightly above the upper limit of the control variation, while in the non-affected side the H response was more enhanced than in the spastic side but less than in normal subjects (Fig. 6). The maximum size of the H response in hemiplegic patients was apparently larger than

in the normal. Therefore, the intensity of the electrical stimulus was reduced to one half of the maximal H response in size, then the enhancement appeared as in the normal subject.

After stopping the manoeuvre, the H response was obviously diminished below the lower limit of the control variation, not only on the hemiplegic side but also on the non-affected side.

A patient with transverse myelitis at the level of D₁₀ cord segment was examined. His H response was not as large as in the hemiplegic patients and it was never influenced by the Jendrassik manoeuvre, even though the stimulus was reduced by half.

DISCUSSION

It is important to calculate the control variation in the amplitude of H response in comparing the influence with respect to amplitude change. We have emphasized that the distribution curve itself should represent one facet of the alpha motoneurone excitability. A standard deviation value is useful, but it does not always show a symmetrical curve of normal distribution in certain pathological cases. In these serial experiments we determined the 80% area from the modal value as the control variation range. The range was sometimes very wide in patients with Parkinsonism, but an influence of the Jendrassik manoeuvre could still be demonstrated. In the group of spastic patients the control variation range was narrow, indicating that most of the alpha motoneurone pool had been excited by the stimulus, with a maximal H response, and that the ratio with the subliminal fringe was re-

duced. The influence of the manoeuvre was more evident when the amplitude of H response had been set to one half of the maximal amplitude, but the influence was significant even with the maximal amplitude leaving a narrow subliminal fringe.

While examining the speed or the strength of the manoeuvre to confirm the original comment of Jendrassik that the extent of reflex enhancement is related to the number of muscles contracted, we noticed that the more important factor responsible for the enhancement was the timing from the instruction. Provided that the pulling strength was limited within 20 kg, the speed or the strength of contraction did not appear to have a significant correlation with the extent of enhancement of the H reflex.

The rate of facilitation was nearly equal in the same subject when using the two hands method, the single hand method, and the push button method. As soon as the subject switched the trigger button lightly with his thumb, the H reflex was enhanced at the same time. No significant difference was noted in the experiments which were done to determine the influence of ipsi- or contralateral thumb movement on the H reflex. Some other trials, such as shrugging the shoulders, had been made to see whether there were any correlation between the selective contraction of tonic or phasic muscles and the extent of the enhancement of H reflex which is elicited from tonic soleus muscle. The tendency was almost the same, but more definitive observations should be made before drawing a conclusion about the correlation.

A prominent factor for the enhancement was

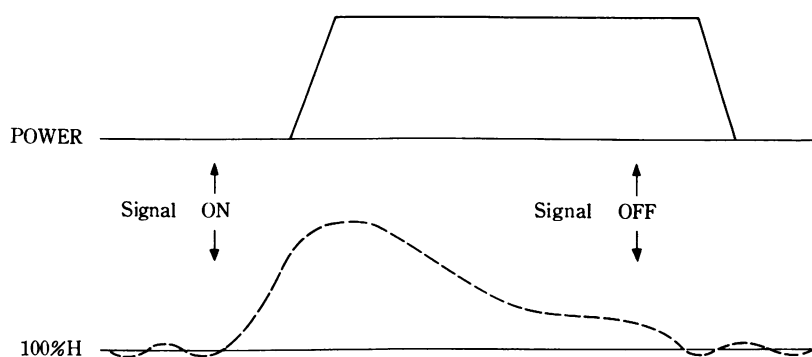


FIG. 7 A schematic diagram of the influence of the Jendrassik manoeuvre on the H response.

the timing between the onset of the manoeuvre and the stimulus for the H response. The time course of the enhancement suggests an influence of the Jendrassik manoeuvre in the following three phases (Figs 1, 7).

The first and most prominent phase was from about 100 ms before the onset of the manoeuvre to about 300 ms after the onset. The H response was already augmented when the subject was preparing himself for the manoeuvre after the signal 'on', before the strain gauge indicated any movement of the upper extremities. There was a slight time lag between the first action potential of the brachioradialis muscle and the deflection of the strain gauge, although it was usually less than 100 ms. The period of this preceding facilitation varied, because of individual differences in the response time for the manoeuvre after the signal. The influence of the photic stimulus by the signal lamp and the psychological expectance were examined, but the preceding facilitation occurred only when the voluntary contraction of the upper extremities succeeded the signal. Thus the intention to do the manoeuvre resulted not only in excitation of the alpha motoneurons of the upper extremities, but also had a facilitatory effect upon the motoneurons of the lower extremity.

Bowditch and Warren (1890) reported that the enhancement was apparent in the early phase of the Jendrassik manoeuvre. Lately, Gassel and Diamantopoulos (1964a) showed the same tendency, but they did not examine the preceding facilitation. After we had finished these experiments, we found that Gottlieb and Agarwal (1973) noticed almost the same preceding facilitation on H response by the Jendrassik manoeuvre. They considered the effect to be related to changes of body posture accompanying the manoeuvre. Postural changes and vestibular influences may certainly have strong effects, but we confirmed the enhancement even by a slight motion of the push button method. The other important finding in their report is that the T response showed continued facilitation, while the H response was depressed after the initial facilitation. Thus they postulated that the prolonged facilitation in sustained contraction must be mediated by fusimotor activation of the muscle spindles.

In the following phase, the enhancing curve declined gradually but a certain elevated level

was maintained for as long as the manoeuvre lasted. Paillard (1955) stated that the H reflex was augmented in brief and brisk contraction and not in sustained contraction. Clarke (1967) mentioned that sustained performance of the manoeuvre probably results in adaptation of activity in the reticular formation. Koguma (1963) and Yajima (1965) reported that depression of the H reflex occurred in the Jendrassik manoeuvre in cases of a brain-stem lesion, but the time factor was not described in their reports.

The last phase is the phenomena seen on stopping the manoeuvre, which were opposite to the first phase. The enhancement of the H reflex disappeared when the subject prepared himself to stop the manoeuvre after the signal 'off', while the strain gauge still indicated that the muscle had not been completely relaxed. After relaxation of the upper extremities the H response rarely drops below the lower limit of the control variation in normal subjects, though Struppler and Preuss (1959) and Gassel and Diamantopoulos (1964a) inferred that the H reflex was somewhat depressed after stopping the manoeuvre.

In the hemiplegic group, the H response was often inhibited after stopping the manoeuvre, not only in the spastic side but also in the non-affected side. Therefore, this overshooting might be regarded as a pathological sign.

There has been much argument about the effect of the Jendrassik manoeuvre in Parkinsonism (Hassler, 1956; Stern and Ward, 1960; England and Schwab, 1961). Recently, Gassel and Diamantopoulos (1964a) commented that patients with Parkinsonism showed enhancement both of the ankle jerk and of the H reflex on vigorous muscular contraction of the upper extremities, and there was no correlation between the extent of enhancement and the severity of the symptoms of Parkinsonism. We also concluded that the manoeuvre was effective on H response beyond the large control variation.

Sommer (1940) postulated that a gamma motor system has a leading role in the Jendrassik manoeuvre. He compared the effect on H and T responses. His conclusion was that the fusimotor sensitization of the muscle spindle was responsible for the facilitation because the H response seemed resistant to augmentation.

There have been several reports to confirm his hypothesis using the method of comparing H and T responses (Hoffmann, 1951; Paillard, 1955; Buller and Dornhorst, 1957; Struppler and Preuss, 1959). Clare and Landau (1964), Gassel and Diamantopoulos (1964a, b), and Landau and Clare (1964a, b), employing the technique of differential nerve blockade, proposed that the facilitatory mechanism might be explained on the basis of a direct effect upon the motoneurone excitability, because the H response which had been depressed by selective gamma fibre blockade, and even when the tendon jerk was abolished, was still enhanced by the Jendrassik manoeuvre. Kohno (1965) and Kawamura (1969) confirmed their findings. Clarke (1967) also agreed with the hypothesis concerning the knee jerk.

Granit and Henatsch (1956) and Bianconi and van der Meulen (1963) reported that the muscle spindle is very sensitive to high frequency mechanical vibratory stimuli. Hagbarth and Eklund (1966) devised a cylindrical vibrator which can be attached to human muscles. They found that the H response was suppressed during vibratory stimulation applied to the ipsilateral Achilles tendon and the suppressed H response was enhanced by the Jendrassik manoeuvre. These phenomena have been confirmed by De Gail *et al.* (1966), Matsuda (1969), and Kawamura (1969). These facts also suggest that the Jendrassik manoeuvre has a rather direct effect upon the alpha motoneurons.

CONCLUSION

The mechanism of the Jendrassik manoeuvre should not be explained merely as mental distraction, but it should be a result of competitive integration of the motor regulating systems including the gamma system; furthermore, the combination has been changed with the time course of the manoeuvre.

One of the reasons, and perhaps the most important, for the controversy about the effectiveness of the Jendrassik manoeuvre upon H or T responses, could be failure to take sufficient count of the timing factor as an important phase of the manoeuvre.

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