The purpose of this communication is to report on the analysis of the effects of muscle pressure on altering motoneuron excitability. Motoneuron excitability was assessed by measuring changes in the H-reflex in 30 neurologically healthy individuals. The results indicate that muscle pressure is excitatory, but of such low intensity as to be of dubious therapeutic benefit. Methodological limitations specific to muscle pressure stimulation limit the interpretation of our results. These limitations are discussed, and a suggestion is made for an alternative approach to evaluate the effects of muscle pressure on motoneuron excitability.

**Key Words:** Motor neurons, Muscle tonus, Physical therapy.
subject was tested twice. In one series of tests, the electrical stimulus intensity used to elicit the H-reflex produced a minimum M wave and a near-maximum H wave. Because the M wave is a direct motor response, influenced primarily by the placement of the stimulating electrode, consistency in the M wave was used to judge stability in the stimulating conditions. The second series of tests used a greater stimulus intensity that produced nearly equal M and H waves.

The results for both sets of assessments are summarized in the Figure. At stimulus intensities that elicited minimum M waves (Figure, illustration A), 26 of 30 subjects demonstrated a mean initial reduction in the H-reflex of 64% of the control values, which returned to 86% of the controls within 5 seconds and to 90% within 30 seconds of pressure application. A concomitant reduction in the M wave to 65% of the controls persisted until cessation of pressure. The M wave reduction signified a possible change in stimulating conditions and prevented us from concluding that muscle pressure inhibits the motoneuron pool.

A second series of assessments, therefore, was made to compensate for these M-wave changes. The electrical stimulation intensity was adjusted so that the M and H waves were about equal. For 23 of the 30 subjects, the H-reflex displayed a mean initial reduction of 90% of the control values, overshoot baseline levels within 5 seconds of pressure application, and reached a maximum of 115% of baseline levels within 20 seconds (Figure, illustration B). These findings suggest that muscle pressure is mildly excitatory to the motoneuron pool. The excitation is almost immediate and within 5 seconds of pressure application displays a plateau at levels 10% to 15% greater than baseline levels. Whether muscle pressure is excitatory or inhibitory to motoneurons, therefore, remains unclear.

When standard H-reflex stimulating conditions were used (Figure, illustration A), sustained muscle pressure produced a transient depression of the H-reflex. A nearly identical finding was reported earlier for sustained tendon pressure, which was interpreted as an inhibition of the soleus muscle motoneuron pool. In our findings, that interpretation is complicated by the unexpected M-wave changes. As shown in the Figure (illustration A), a mean M-wave depression to 35% of the controls also was observed on pressure application. This depression remained constant until the pressure was released. Because consistency of M-wave amplitude was a major criterion for stable experimental conditions, variations in the M waves jeopardized the conclusion that an inhibitory effect was induced by muscle pressure. The plateauing of the M-wave depression during pressure application could indicate a shift in baseline conditions to which any effect of the H-reflex should be referenced. The 36% initial reduction in the H-reflex (compared with the 35% M-wave reduction), thus, could indicate merely a shift in baseline conditions, and the subsequent increase in the H-reflex amplitude might indicate an actual excitation. A partial resolution to this problem required accounting for the M-wave changes.

The most plausible explanation for these M-wave changes is that they were artifacts caused by tissue distortion under the recording electrode during pressure application. To test for recording electrode artifact, pressure was applied directly over the recording electrode. Under extremely high pressures, several times greater than that of the experimental stimulus, no changes in the M wave were observed. This finding undoubtedly is due to the on-site preamplification system, which was imbedded, together with the electrodes, in a rigid epoxy mount. Such a system was designed originally to eliminate movement artifacts for myoelectrically controlled prostheses.

Another possible explanation for the observed M-wave changes is that a change occurred in the stimulating conditions. When pressure was applied to the gastrocnemius-soleus muscle complex, a slight distortion in the tissue within the popliteal fossa could be palpated. Optimal stimulation could be maintained by repositioning the electrode during pressure application. By repositioning the stimulating electrode, however, a subsequent loss of prepressure and postpressure con-

![Figure](image-url)
controls occurred. Rather than manipulate the electrode assembly, therefore, the intensity of stimulation was increased so that nearly equal H and M waves were generated. In so doing, two inherent problems of the initial analysis (where the M wave was minimal) were addressed. The first problem was that M waves were of much greater amplitude and, therefore, decreased the variability inherent to the measurement of small signals. The approximately 50% reduction in the standard errors for M-wave measurements for the higher stimulation voltages (Figure, illustrations B vs A) indicates this improvement in the consistency of response. The second problem was that the greater intensity of stimulation created a larger electrical field around the tibial nerve that could offset some of the effects caused by stimulating electrode displacement. The average decrease in M-wave response during pressure application was 10% less for the higher stimulation voltage (Figure, illustration B vs A). This smaller M-wave change is indicative of a greater number of motor axons being stimulated, which most likely was due to a larger electrical field surrounding the tibial nerve.

By compensating for the change in stimulating conditions, the H-reflex response to muscle pressure was indicative of an excitation rather than inhibition of the soleus muscle motoneuron pool. This excitation occurred within the first 5 seconds of pressure application and, because of the lack of statistical differences (p < .05) among the values at times 5, 10, 20, and 30 seconds, may indicate a plateauing of the response. Overall, the H-reflex response was weak; only the response recorded at 20 seconds of pressure application was significantly different from baseline levels.

**CONCLUSIONS**

These findings must be regarded as tentative because of the methodological considerations discussed. Even if the conclusion that muscle pressure is excitatory to motoneurons were accepted, the rather small changes recorded (10%-15% above baseline levels) raise the question of the therapeutic effectiveness of such a stimulus. Resolution of this problem may not be possible at the level of H-reflex analyses. Because pressure applied to the skin may excite the entire spectrum of afferent fiber types, it might be more informative first to ask specific questions about the relative contributions of cutaneous mechanoreceptors and muscle and tendon receptors on pressure-induced changes in motoneuron excitability. The cross-correlation techniques used to analyze motor unit spike trains10,11 could provide the most useful information in this regard. When a more thorough understanding of the contribution of these various afferent groups to motoneuron excitability is obtained, their combined contributions, as encountered in clinical situations, may be analyzed more effectively. Until then, the effectiveness of muscle pressure in exciting motoneurons remains unresolved. The tentative findings presented here suggest that muscle pressure produces an excitatory effect, but of such low intensity that the clinical benefits are dubious.

**REFERENCES**