TOPICAL ANESTHESIA: MODULATION OF THE MONOSYNAPTIC REFLEXES BY DESENSITIZATION OF THE SKIN

M.A. SABBAHI and C.J. DE LUCA

NeuroMuscular Research Laboratory, Department of Orthopaedic Surgery, Children's Hospital Medical Center, Harvard Medical School, Boston, Mass. 02115, and Liberty Mutual Research Center, Hopkinton, Mass. (U.S.A.)

(Received for publication: July 12, 1982)

The interaction between skin receptors and their possible effect on the modulation of the α-γ motoneuron excitability has not been widely or thoroughly investigated in man. In this study, the effect of desensitization of skin receptors on the α-γ motoneuron system was investigated by applying topical anesthesia to selected skin areas of the lower limb.

The few relevant reports that have appeared in the literature are not in complete agreement. Hagbarth (1952), working on decerebrated animals which lacked cortical integration, demonstrated that specific skin areas may excite or inhibit the extensor motoneurons in decerebrated cats. These results were later demonstrated in man using noxious stimuli to the skin (Hagbarth and Finer 1963). Unfortunately, their results could not be repeated by other workers (Gassel and Ott 1970). It has also been reported that spinal reflexes such as the H-reflex may be facilitated by low threshold shocks delivered to the human cutaneous nerve which supply the skin area overlying the muscle groups (Hugon 1973). In other studies, skin stimulation was shown to have no relevant effect on these spinal reflexes (Magladery et al. 1951; Isaacs et al. 1968). These discrepancies may be due to the methods used to study the effect of skin receptor areas on the motoneuron pool. In all previously reported studies, electrical or noxious stimuli which are not natural or specific for particular receptors were used to excite the skin.

In our own previous studies using monosynaptic reflexes, we found that natural stimuli to the skin (mechanical and thermal) were more effective in producing inhibition responses (Sabbahi and Sedgwick 1976). In the present study, skin desensitization was shown to have a substantial effect on the H-reflex and the Achilles tendon reflex (ATR). These monosynaptic reflexes provided a direct measure of the α-γ motoneuron excitability. More importantly, in studying the effect that the skin has on the motoneuron pool, our work differentiates between the effect of dermatomal innervation and the innervation of skin areas overlying muscle groups.

In addition to its inherent physiological interest, the interaction between skin sensors and the motor system could provide a natural and convenient access for affecting the behavior of the motor system. The latter concept has important applications in rehabilitation medicine. The utilization of skin receptors as a potential effector organ in the treatment of spasticity is in progress (Sabbahi et al. 1981).

Materials and Methods

Fifty-eight normal adult subjects whose age ranged from 18 to 54 years with a mean and standard deviation of 27 ± 7 years volunteered for the study and signed informed consent forms. None of the subjects had a past history of neurological disorders. The experiment consisted of obtaining measurements of the H-reflex and ATR before and after topical anesthesia was applied to
The H-reflex was elicited by stimulating the posterior tibial nerve unifocally with 1 msec square pulse once every 5 sec with a stimulus subthreshold to the M-response. (In some experiments a small M-response was elicited in order to monitor the changes in the stimulus volley.) The stimulus level was maintained constant throughout the experiment. The stimulation electrode (cathode) consists of a felt sphere, 1.5 cm in diameter, well padded and soaked with 0.9% physiological saline solution. The indifferent electrode (anode) consisted of metal mesh (10 cm x 10 cm) padded with felt and soaked with 0.9% physiological saline. It was positioned on the anterior surface of the thigh just above the patella. The H-reflex was recorded differentially via two cup electrodes, filled with conducting gel, and placed 3 cm apart on the skin overlaying the soleus muscle in line with the Achilles tendon. The reference electrode (ground) for the recording set-up was a rectangular metal plate (3 cm x 5 cm) placed between the stimulation and recording sites to minimize the stimulus artifact. The signal was amplified 1000 times with a bandwidth of 10 Hz to 10 kHz, and was preserved on an ultraviolet sensitive paper for subsequent analysis.

The procedure for recording the H-reflex consisted of stabilizing the amplitude of the response and lowering the threshold of the motoneuron pool until minimal variation in the reflex was obtained. This was achieved by using a threshold stimulus for eliciting the M-response with a frequency of 1 pulse/sec for 3 min, followed by lowering the stimulus frequency gradually to 1 pulse every 2 sec, and then to 1 pulse/5 sec, while reducing the stimulus intensity until the required reflex amplitude was obtained. Stimulation was maintained at this rate and intensity for 3-5 min until maximal stabilization of the reflex amplitude was achieved, at which point measurements could begin. Using these controlled stimuli, which with a few exceptions were subthreshold to the M-response, the resulting maximal H-reflex was measured.

The ATR was elicited by using an electrically operated solenoid plunger to deliver a constant-force impact to the tendon every 10 sec. The response was recorded via the same electrodes used for recording the H-reflex. Recordings were made while the ankle was fixed in a specially designed apparatus which allowed free movement of the foot. In some experiments the foot was passively stretched and fixed perpendicular to the leg by an elastic band which allowed foot movement during reflex contractions. This procedure was carried out in order to test the changes in the H-reflex and ATR during stretching with a presumably higher excitability level of the soleus motoneuron pool.

Control values of the H-reflex and ATR were obtained at the beginning of each experiment. To desensitize the skin, topical anesthesia (20% Benzocaine) was sprayed for 15-20 sec on one of the following 4 skin areas of each subject: the calf skin area (CSA), the anterior tibial skin area (TSA), the quadriceps skin area (QSA) and the hamstrings skin area (HSA). In 3 subjects the total skin area of the lower limb (TSL) was desensitized to test any summation effect on the motoneuron pool. The topical anesthesia was also separately sprayed on each of the following skin dermatomes for each subject: L2, L3, L4, L5, S1 and S2. (Some subjects participated in separate experiments in which different skin dermatomes or skin areas were sprayed with anesthesia.) The outlines of the desensitized skin areas and dermatomes as well as the number of subjects studied for each are presented in Fig. 1. Subsequent to the application of the anesthesia, the H-reflex and ATR were measured at various time intervals up to 40 min.

In 3 other subjects, the calf skin area, quadriceps skin area and hamstring skin area were sprayed separately with a placebo spray (similar in all respects to the anesthesia spray, except that it contained no Benzocaine) and the H-reflex and the ATR were recorded at various time intervals up to 30 min. These control experiments were carried out without prior information given the subjects.

The peak-to-peak amplitudes of the H-reflex and ATR were measured and the average value of not less than 10 consecutive sweeps was calculated. The ratio of the average amplitude of the reflexes after application of the topical anesthesia to the
average amplitude before anesthesia was expressed as a percentage. The two values were compared with a t test, with the assumption that both values are normally distributed.

Results

Topical anesthesia caused partial reduction in skin sensation to superficial touch (or desensitization of the skin) beginning 5 min after its application. The subjects were unable to identify hair movement and their sensation to cotton wool was substantially reduced. However, deeper skin sensation of pin prick was not distinguishably different from that of the contralateral control limb having normal sensation. This effect continued throughout the experiment and diminished gradually afterward. This effect was not noticed by subjects sprayed with the placebo.

As may be seen in Fig. 2 individual subjects typically showed considerable change in H-reflex amplitude after application of the topical anesthesia. This figure represents typical H-reflex responses of one normal subject for each tested skin area and skin dermatome. The mean value and two standard deviations for 10 consecutive sweeps are shown (dotted line). The low values of the standard deviations indicate the high degree of stability of the H-reflexes provided by our method. These reliable responses continued for 30–40 min after the application of the anesthesia. In Fig. 2
the control H-reflexes are shown in the upper row (control), while the reflexes recorded 30 min after application of the anesthesia are presented in the lower row (anesthesia). A t test was performed between the peak-to-peak amplitude of the H-reflexes before and 30 min after the application of the anesthesia to different skin areas and dermatomes. The statistical significance of the difference is presented in the bottom row (P value).

Effects of desensitization of skin areas

H-reflex. As may be seen from Fig. 2A, in a typical individual experiment the H-reflex significantly increased in amplitude (P < 10^-6) after application of topical anesthesia to different skin areas except for the anterior tibial skin. Fig. 3 presents the regression lines for the grand mean, for each group of subjects, and one standard deviation value of the percent of change in the peak-to-peak amplitude of the H-reflex and ATR measurements after topical anesthesia was applied to the calf skin area (n = 6), the anterior tibial skin area (n = 11), the quadriceps skin area (n = 7) and the hamstrings skin area (n = 7). The control value, obtained prior to the application of the topical anesthesia, was considered as 100% at zero time. Table I presents the value of the slopes and the statistical significance of the linear regressions in Fig. 3. A t test was performed on the slopes of the regression lines and a slope of zero. This test measures the level of significance of the time-dependent variation of the amplitude of the H-reflex. It may be seen in Fig. 3 that the H-reflex amplitude increased substantially after the application of topical anesthesia to the skin overlying the calf, quadriceps and hamstring muscles. This facilitation of the reflex was first noticed 5 min after the application of the anesthesia and continued

<table>
<thead>
<tr>
<th>Skin area</th>
<th>Value of slope (% change/min)</th>
<th>P value</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>H-reflex</td>
<td>CSA 1.92</td>
<td>0.04</td>
<td>S</td>
</tr>
<tr>
<td></td>
<td>TSA 0.30</td>
<td>0.55</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>QSA 1.30</td>
<td>0.41</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>HSA 3.44</td>
<td>0.08</td>
<td>S</td>
</tr>
<tr>
<td>ATR</td>
<td>CSA 0.62</td>
<td>0.30</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>TSA 1.26</td>
<td>0.08</td>
<td>S</td>
</tr>
<tr>
<td></td>
<td>QSA 0.12</td>
<td>0.89</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>HSA 0.34</td>
<td>0.52</td>
<td>NS</td>
</tr>
</tbody>
</table>

Fig. 3. Percentage changes in the peak-to-peak amplitude of the H-reflex (HR) and the Achilles tendon reflex (ATR) with time, after application of topical anesthesia to the calf skin areas (CSA), anterior tibial skin area (TSA), quadriceps skin area (QSA) and hamstrings skin area (HSA). The grand mean and standard deviation of the percent of changes in all subjects tested at each skin area is plotted for a period of time up to 40 min post anesthesia. The control value of the H-reflex amplitude before anesthesia was considered as 100% on the ordinate in these plots. Notice that the ordinate scale for the percentage changes in the H-reflex after application of anesthesia to the hamstring skin area is twice that of the other plots.
throughout the duration of the experiment (30–40 min).

Desensitization of the calf skin area facilitated the H-reflex amplitude to \((129 \pm 34)\)% after 5 min, and to \((203 \pm 99)\)% after 40 min. However, 5 out of 6 subjects demonstrated facilitation while one subject showed no apparent changes. Desensitization of the anterior tibial skin area resulted in an inconsistent behavior of the H-reflex. It increased in amplitude in 6 of the 11 subjects, showed substantial inhibition in 3 subjects and did not change appreciably in the remaining two subjects. However, even in those subjects who demonstrated facilitation, the increase of the amplitude was not substantial when compared to the results obtained from the other skin areas. In individual experiments the H-reflex induced by the desensitization of the quadriceps skin area was greater than that induced by the desensitization of the calf skin. Reflex facilitation was noted 2 min after the application of topical anesthesia; after 5 min the amplitude increased to \((170 \pm 75)\)% (Such a substantial and rapid increase in the H-reflex amplitude was only seen to occur in the quadriceps skin area.) Forty minutes after the application of the anesthesia, 5 out of 7 subjects demonstrated substantial reflex facilitation ranging from 150% to 322% with a mean and standard deviation of \((215 \pm 77)\)%; one subject showed less facilitation \((136)\)% and the remaining subject showed no apparent change in the H-reflex \((105)\)%. This results in the large standard deviation seen in Fig. 3. The greatest facilitation was induced by desensitization of the hamstrings skin area. As may be noticed from Fig. 3 the scale of the ordinate of the curve for the hamstring skin area is twice that of other curves. The average amplitude of the H-reflex increased to \((148 \pm 70)\)% after 5 min and to \((279 \pm 200)\)% 40 min after application of anesthesia. The facilitation was observed in most subjects. In one subject, the amplitude increased almost 6-fold over the control value while one subject demonstrated no apparent changes.

**ATR.** Desensitization of 3 of 4 skin areas did not affect the ATR amplitude in most subjects. Only when the anterior tibial skin area was desensitized did the ATR approach a significant increase in amplitude (Table I). The increase in the peak-to-peak amplitude of the ATR continued with time after application of the anesthesia to the anterior tibial skin (Fig. 3).

When the measurements at 30 min post anesthesia were compared with the control values before anesthesia, a *t* test for the mean showed that no significant changes occurred in the ATR, whereas all skin areas, except the anterior tibial skin area, exhibited a significant \((P < 0.04)\) increase in the H-reflex.

**Effects of application of topical anesthesia to various dermatomes**

**H-reflex.** Application of the topical anesthesia to the skin dermatomes showed a substantial facilitation of the reflex for most of the dermatomes tested. As can be seen in Fig. 2B, in the individual experiments the H-reflex significantly increased in amplitude \((P < 10^{-6})\) 30 min after application of the topical anesthesia to each skin dermatome except for that of the S1 dermatome.

In most subjects, however, the degree of facilitation in the H-reflex was not as significant as it was when skin areas were desensitized (Fig. 2A). This is probably due to the fact that the skin dermatomes were physically smaller than the skin areas previously examined. This result appears to indicate the existence of a possible spatial summation of the effect on the discharge of the motoneuron pool. The H-reflex facilitation increased with time following application of the anesthesia (Fig. 4). Both the L2 and L3 dermatomes which overlap with parts of the quadriceps skin area showed an H-reflex facilitation similar to that of the quadriceps skin area. Thirty minutes after application of anesthesia to the L2 dermatome and quadriceps skin area, the grand mean values for the H-reflex were \((214 \pm 44)\)% and \((202 \pm 69)\)% respectively. However, although L3 dermatome desensitization did not result in as much H-reflex facilitation as in the L2 dermatome \((138\% \text{ in L3}, 214\% \text{ in L2}, 30 \text{ min post anesthesia})\), the inter-subject variation (standard deviation) in the L3 dermatome was relatively small (Fig. 4). For both the L2 and L3 dermatomes the value of the slope of the linear regression line was statistically significant \((P < 0.05, P < 0.04)\) (see Table II).
Application of topical anesthesia to the L4 and L5 dermatomes resulted in a substantial increase in the H-reflex amplitude. This reflex facilitation continued with time (Fig. 4). However, the slope of the linear regression line for both cases was not statistically significant from the zero horizontal line (Table II). This was mainly due to the large inter-subject variation which was represented by the large standard deviation. In fact, an H-reflex inhibition or no significant change with desensitization of the L4 or L5 dermatomes might have been expected as parts of these dermatomes overlap with the anterior tibial skin area. However, because both dermatomes cover an area from the anterior and posterior surface of the leg (Fig. 1) and because reflex facilitation appears to be more dominant than inhibition after anesthesia, the net result was an increase in the H-reflex amplitude. This varied behavior was always associated with the largest inter-subject variation seen (Fig. 4). In the L4 and L5 dermatomes 9 out of 10 subjects showed varied reflex facilitation while one subject demonstrated no apparent changes.

In the SI dermatome, skin desensitization in 5 subjects resulted in mild (133% ± 22%, 30 min post anesthesia) but significant (P < 0.05) facilitation of the H-reflex. The reflex amplitude ranged from a mild to moderate degree of facilitation in 4 subjects. One subject demonstrated no significant changes throughout the 30 min following anesthesia. It is important to note that inter-subject variation from the SI dermatome was small when compared to other skin areas and dermatomes (Fig. 4). Moreover, the reflex facilitation, post anesthesia, was not as large as those after

### TABLE II

The slope value of the linear regression line for the changes in the H-reflex and ATR with time post anesthesia to various skin dermatomes (values correspond to those plotted in Fig. 4).

<table>
<thead>
<tr>
<th>Skin dermatome</th>
<th>Value of slope (%) change/min</th>
<th>P value</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>H-reflex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L2</td>
<td>2.44</td>
<td>0.05</td>
<td>S</td>
</tr>
<tr>
<td>L3</td>
<td>1.19</td>
<td>0.04</td>
<td>S</td>
</tr>
<tr>
<td>L4</td>
<td>2.03</td>
<td>0.10</td>
<td>NS</td>
</tr>
<tr>
<td>L5</td>
<td>1.55</td>
<td>0.26</td>
<td>NS</td>
</tr>
<tr>
<td>S1</td>
<td>0.73</td>
<td>0.05</td>
<td>S</td>
</tr>
<tr>
<td>S2</td>
<td>1.35</td>
<td>0.21</td>
<td>NS</td>
</tr>
<tr>
<td>ATR</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L2</td>
<td>0.55</td>
<td>0.70</td>
<td>NS</td>
</tr>
<tr>
<td>L3</td>
<td>0.80</td>
<td>0.65</td>
<td>NS</td>
</tr>
<tr>
<td>L4</td>
<td>0.10</td>
<td>0.92</td>
<td>NS</td>
</tr>
<tr>
<td>L5</td>
<td>−2.35</td>
<td>0.02</td>
<td>S</td>
</tr>
<tr>
<td>S1</td>
<td>0.80</td>
<td>0.43</td>
<td>NS</td>
</tr>
<tr>
<td>S2</td>
<td>−0.50</td>
<td>0.63</td>
<td>NS</td>
</tr>
</tbody>
</table>

Fig. 4. Percentage changes in the H-reflex and ATR with time after application of topical anesthesia to L2, L3, L4, L5, SI and S2 dermatomes. The grand mean and standard deviation of the percent of changes in all subjects tested at each skin dermatome is plotted for a period up to 30 min post anesthesia. The control value of the H-reflex amplitude before anesthesia was considered as 100% on the ordinate in these plots.
desensitization of the calf skin area, L2 or L3 dermatome (Figs. 2B and 4).

The S2 dermatome is equivalent for the most part to the hamstrings skin area. Also, it covers the surface area on the back of the proximal one-third of the leg (Fig. 1). When this dermatome was desensitized, H-reflex facilitation was noticed in 2 out of 3 subjects, while one subject showed no measurable changes. This variability resulted in an average reflex response having no significant changes post anesthesia (Table II).

In those experiments in which a small M-response was recorded, no significant changes were noted. Therefore, it appears that no changes in the stimulus volley occurred during the experiment. Thus the changes in the H-reflexes are attributable to a change in the central excitatory state. Also, no significant changes were noticed in the latencies of the responses.

The results of H-reflex modulation are summarized in Fig. 5. It shows a lateral view of those areas which when desensitized with anesthesia result in H-reflex facilitation (dotted) and those which when desensitized result in either H-reflex inhibition or no significant changes (cross-hatched).

_ATR_. Whenever there was a substantial increase in the amplitude of the H-reflex after topical anesthesia, it was always associated with either a significant time-dependent decrease (_P_ < 0.02) of the ATR or else no significant change. The substantial inter-subject variation of the ATR data concealed the predominant trends of most subjects. However, because the intra-subject variation was minimal, it was possible to group the results obtained from all 24 subjects: 14 showed inhibition, 5 showed some facilitation, and 5 showed no measurable changes. (For details of individual dermatomal effect, refer to Table III.) In these individual experiments the grand mean amplitude of the ATR was less than 50% of the control in 3 subjects and was 53–89% of the control in 11 subjects. In those subjects who showed facilitation of the ATR post anesthesia, the mean value of the amplitude ranged from 117% to 147% of the control value.

### Table III

The changes in the ATR in individual experiments after application of topical anesthesia to various skin dermatomes.

<table>
<thead>
<tr>
<th>ATR changes</th>
<th>L2</th>
<th>L3</th>
<th>L4</th>
<th>L5</th>
<th>S1</th>
<th>S2</th>
<th>Number of subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inhibition</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>14</td>
</tr>
<tr>
<td>No change</td>
<td></td>
<td></td>
<td>3</td>
<td>1</td>
<td>1</td>
<td></td>
<td>5</td>
</tr>
<tr>
<td>Facilitation</td>
<td>1</td>
<td>2</td>
<td></td>
<td>1</td>
<td>1</td>
<td></td>
<td>5</td>
</tr>
</tbody>
</table>
Application of topical anesthesia to the entire skin of the lower limb

When topical anesthesia was applied to the entire skin of the lower limb, the H-reflex was substantially increased in amplitude in all subjects (n = 3). The peak-to-peak amplitude was (211 ± 95)% of the control 30 min post anesthesia. Again, the inter-subject variation was large. With every increase in H-reflex amplitude, a substantial reduction of ATR was noticed; it decreased to (64 ± 39)% of the control value 30 min after application of anesthesia.

No measurable changes in the H-reflex or ATR were noticed after application of the placebo spray in the control experiments. It is worth noting that the above-mentioned results were relatively similar whether the foot was passively flexed (with accompanying stretching of the Achilles tendon) or when the foot was left free in the neutral position.

Discussion

The soleus H-reflex was significantly increased in amplitude after desensitization of different skin areas and dermatomes of the lower limb with the exception of the anterior tibial skin area and its parallel dermatomes. (Note the lack of statistical results from L4 and L5 as shown in Table II.) Most individual experiments, such as the one presented in Fig. 2A, B, clearly demonstrate such a pattern of excitability. These individual experiments show that S1 and possibly S2 are the skin dermatomes with minimal effect on the soleus motoneuron pool after topical anesthesia. It was also noted that whenever there was a facilitation of the soleus H-reflex, it was almost always associated with inhibition or no measurable changes in the ATR of the same muscle.

The H-reflex has been generally considered to be a reliable measure of the excitability of the α motoneurons, while the ATR has been interpreted as a measure for both α and γ motoneuron excitability (W.B. Matthews 1970). However, due to the possible involvement of the presynaptic inhibition in the modulation of these reflexes, it may be claimed that the H-reflex as well as the ATR are not a direct measure of the excitability of the α motoneurons (Wall 1964; Schmidt 1971). Furthermore, although H-reflex technique is an artificial (electrical) method of testing the excitability of the α motoneurons, other properties of this reflex, such as being monosynaptic, spinal and unbiased by the γ-system outweigh its limitation.

Our experiments, which show H-reflex facilitation following the application of topical anesthesia to various skin areas of the lower limb, appear to indicate that decreased sensitivity of skin receptors can cause an increase in the α motoneuron excitability. Furthermore, a companion study involving the use of the H-reflex recovery curve, which is not affected by presynaptic inhibition, also demonstrated an increase in the excitability of the motoneuron pool following topical anesthesia (Sabbahi and De Luca 1981), providing additional credence to the present results.

In a previous investigation, Sabbahi and Sedgwick (1976) demonstrated that two kinds of natural skin stimuli (cooling to stimulate the cold receptors and scrubbing to stimulate the mechanical receptors) both substantially inhibited the H-reflex. The results of our present study are also consistent with these findings since they show that the decrease of skin sensation induced by topical anesthesia resulted in the opposite effect, that is, in H-reflex facilitation. Furthermore, both sets of results are supported by our current clinical studies (Sabbahi et al. 1981). In these recent studies involving patients with stroke, application of topical anesthesia to the skin of the lower limbs resulted in a substantial increase in the active movement pattern associated with substantial increase in the peak-to-peak amplitude of the H-reflex.

Other investigators have also noted modifications in the motor output with varying excitability of the skin receptors. Hagbarth and Finer (1963) have shown that some skin areas of the human lower limb have a facilitatory effect on the flexor reflex, whereas other areas have an inhibitory effect. More recently, Marsden et al. (1979) reported an increase in electromyographic activity in the flexor muscle and a decrease in the extensor muscle of the thumb after applying anesthesia to the skin of the thumb. Animal studies have also supported these findings by showing that the α motoneurons (Hagbarth 1952) and the γ motoneurons (Eldred
and Hagbarth 1954) are affected by skin stimulation. In contrast, other investigators have reported that the skin was without effect on motoneuron excitability (Magladery et al. 1951; Isaacs et al. 1968). This contrary finding may be attributable to differences in the mode of skin stimulation, since the latter investigators used electrical stimulation to the skin in order to study the effect of skin receptors discharge on motoneuron excitability. Electrical skin stimulation, apart from being artificial, is also non-specific to a particular receptor (P.B.C. Matthews 1972).

The apparent effect the skin receptors have on the discharge of the motoneuron pool could vary not only according to the type of receptors stimulated, but also according to the location of these receptors in the skin. In fact, Hagbarth (1952) reported that the motoneurons to the extensor muscles of the cat foot are excited by all skin areas of the lower limb except that skin area overlying the antagonist muscle. This pattern of modulation could be of clinical validity, although it has not been reproduced by other workers (Magladery et al. 1951; Isaacs et al. 1968; Gassel and Ott 1970).

The pattern observed in this study by which skin areas modulate the excitability of the soleus motoneuron pool does not coincide with the findings of Hagbarth and Finer (1963) but is consistent with our previous work (Sabbahi and Sedgwick 1976). The experimental procedure employed in the present experiments is different, and most important, the stimuli are not noxious. Hagbarth and Finer (1963) used noxious stimuli and used the flexor reflex as an index for the motoneuron excitability. In our work, either natural stimuli or desensitization of cutaneous receptors discharge (via anesthesia) were used. Also, many of the subjects tested in Hagbarth and Finer’s experiment maintained muscle contraction at the time of stimulation, whereas ours were relaxed and remained relaxed. Sustained muscle activity involves a number of segmental mechanisms which would be quiescent in our subjects. It could be argued that the flexor reflex as a polysynaptic reflex could be altered by the suprasegmental inputs on the interneurons (Willer et al. 1979) which would conceal the true response of the motoneurons to the afferent signals. Willer et al. (1979) demonstrated a significant change, either facilitation or inhibition, in the nociceptive reflex with supraspinal inflow as set by mental task or stress.

In the individual ATR experiments, it was noted that whenever there was an increase in the H-reflex after topical anesthesia, there was either no significant change or a mild reduction in the amplitude of the ATR. This indicates a concurrent reduction in the excitability of the γ motoneurons (implying a decrease in the sensitivity of the muscle spindles to phasic stretch) to compensate for the increase in the excitability of the α motoneurons. This conjecture is in agreement with similar observations reported by other investigators (Urbscheit and Bishop 1970) and is compatible with the work of Eldred and Hagbarth (1954) who demonstrated an increase in the discharge of the γ motoneurons after skin stimulation. It is also supported by the recent work of Ellaway et al. (1981) who demonstrated an increase in the γ motoneuron discharge after stimulation of the lower threshold group III mechanoreceptor afferents. This simultaneous interaction between the α and γ motoneurons in response to the reduction in skin sensory discharge may be a result of the requirement for maintaining the reflex gain constant.

Recently Abbruzzese et al. (1978) have reported a reduction in the tendon vibration reflex after anesthesia applied to the skin underlying a vibrator placed over the extensor muscles of the lower limb. They attributed these changes to a ‘withdrawal’ of a tonic facilitatory influence from skin mechanoreceptors. This tonic facilitatory effect of the cutaneous afferents was also proposed by Marsden et al. (1976) and may be similar to our findings. However, it is important to note that this ‘withdrawal’ of the tonic facilitatory influence on the γ motoneurons from the skin is always associated with ‘withdrawal’ of the tonic inhibitory influence on the α motoneurons as seen from the present results of the H-reflex. It appears that the skin afferent stimuli have a dual effect on the motoneuron pool: a tonic facilitatory effect as well as a tonic inhibitory effect. This is shown in the phasic and tonic reflexes respectively.

The mechanism which controls the simultaneous interaction between the α and γ motoneurons
after skin desensitization is not yet understood. Segmental and suprasegmental mechanisms could be involved. The possibility that suprasegmental and supraspinal pathways could be involved in modulation of the H-reflex with skin desensitization is supported by the results of Rosén and Asanuma (1972). The projection of cutaneous afferent to the motor cortex and the transcortical theory of the stretch reflex is in agreement with the effect of supraspinal pathways on the α motoneurons during desensitization of the skin. Moreover, Lewis and Porter (1974) demonstrated an increase in the firing discharge of the pyramidal tract neurons after blocking afferent discharge with anesthesia. However, the fact that L5, S1 and S2 dermatomes (the spinal roots of which supply the soleus muscle) and the anterior tibial skin area (L4, L5, S1) showed minimal or no significant changes implies a segmental effect. This was also the case with stimulation of the mechanoreceptors and cold receptors (Sabbahi and Sedgwick 1976). From the present results, however, it is difficult to differentiate the degree of involvement of different kinds of mechanisms resulting in the modulation of the H-reflex and the ATR.

Perhaps the most important point demonstrated by these results is that spontaneous on-going activity from the skin of the lower limb continuously affects the motoneuron pool of the soleus. Spontaneous discharges of cutaneous afferents have been reported in the glabrous and in the hairy skin of humans (Knibestol and Vallbo 1970; Knibestol 1975) as well as the cat (Chambers et al. 1972). Johansson (1978) has demonstrated that such discharges occur below the psycho-physical threshold of the skin. It is postulated that the reduction in this activity or discharge, induced by topical anesthesia, alters the excitability level of the motoneuron pool. The concept of this present conjecture could be of clinical value in modulating the output of different types of motoneuron pools by using the skin as a potential effector in neurological conditions. Such modulation, coupled with the apparent selective connections between skin receptors and specific motoneuron pools could provide the basis for a novel approach in the rehabilitation of spastic patients. The apparent interaction between the α and γ motoneurons should be exploited clinically, particularly for cases of spasticity caused by γ bias. This hypothesis has been tested favorably by Sabbahi et al. (1981).

In conclusion, these results demonstrate the existence of a significant inhibition of the soleus α motoneuron pool by all the skin areas of the lower limb except those overlying the antagonist. The latter skin area had a varied and inconsistent effect on the α motoneurons of the soleus muscle. We speculate that even without overt skin stimulation there is an on-going activity from the skin of the lower limb on the soleus motoneuron pool. The evidence for this speculation is that when skin sensation is reduced or blocked, an overall change in the excitability arrangement of the α and γ motoneurons occurs in response to afferent signals.

Summary

Skin desensitization by topical anesthesia was studied for its effect on the motoneuron excitability of the soleus muscle. Skin areas overlying calf, tibial, quadriceps and hamstrings muscles and skin dermatomes (L2, L3, L4, L5, S1 and S2) were studied separately. Motoneuron excitabilities were measured by the H-reflex and Achilles tendon reflex (for α and γ motoneurons).

It was shown that anesthesia applied to all skin areas and dermatomes, except those overlying the antagonist muscles, resulted in significant facilitation of the soleus H-reflex. In these cases, the ATR showed either slight inhibition or no significant changes. Anesthesia to the skin overlying the anterior tibial antagonist muscle produced varied and inconsistent modifications in the amplitude of the H-reflex. In these cases the ATR was either slightly facilitated or showed no significant changes.

These results appear to indicate the existence of on-going excitatory and inhibitory effects from the skin on the α and γ motoneuron pool probably via segmental and suprasegmental levels. A possible clinical application of these results to modulate the motoneuron pool excitability is proposed.
Résumé

Anesthésie topique: modulation des réflexes monosynthétiques par suppression de la sensibilité cutanée

L'effet d'une 'désensibilisation' de la peau par anesthésie locale a été étudié, sur l'excitabilité des motoneurones du muscle solaire. On a ainsi traité séparément les territoires cutanés situés au-dessus des muscles du mollet, au-dessus du tibial, du quadriceps et de la partie postérieure de la cuisse, ainsi que les dermatomes cutanés L2, L3, L4, L5, S1 et S2. Les excitabilités motoneuronales (α et γ) ont été évaluées à partir du réflexe H et du réflexe achilléen.

L'anesthésie, lorsqu'elle était appliquée à tous les territoires cutanés et aux dermatomes, a entraîné une facilitation significative du réflexe H du solaire, à l'expiration de l'anesthésie des régions cutanées recouvrant ses muscles antagonistes. En même temps, le réflexe achilléen était soit légèrement inhibé soit non significativement modifié. En revanche, l'anesthésie appliquée à la peau au-dessus du tibial antérieur, antagoniste du solaire, n'a modifié le réflexe H que de manière inconstante et variable. Dans ce cas, le réflexe achilléen a été soit légèrement facilité soit non significativement modifié.

Ces résultats suggèrent l'existence d'effets excitateurs ou inhibiteurs toniques de la peau sur les pools de motoneurones alpha et gamma, probablement à travers les niveaux segmentaires et suprasegmentaires. Une possibilité d'application clinique de ces résultats est proposée, en vue de moduler l'excitabilité des pools de motoneurones.

The authors are grateful for the interest and assistance provided by Dr. W.R. Powers throughout this study. We wish to thank Mr. J. Bloom for his help in the analysis of the data, and Mr. L.D. Gilmore for providing technical assistance. The topical anesthesia spray (Americaine) as well as the placebo spray was supplied by American Critical Care. This work was supported by Project Hope and Liberty Mutual Insurance Company.

Références


Matthews, W.B. The clinical implications of the H-reflex and
other electrically induced reflexes. In: D. Williams (Ed.),
Modern Trends of Neurology, Vol. 5, Butterworths, London,
Rosén, I. and Asanuma, H. Peripheral afferent inputs to the
forelimb area of the monkey motor cortex: input-output
Sabbahi, M.A. and De Luca, C.J. Topical anesthesia: H-reflex
recovery changes by desensitization of the skin. Electroenceph.
Sabbahi, M.A. and Sedgwick, E.M. H-reflex modulation by
natural stimulation of the skin. Electroenceph. clin. Neuro-
physiol., 1976, 41: 537.
Schmidt, R.F. Presynaptic inhibition of the vertebrate central
Urbscheit, N. and Bishop, B. Effects of cooling on the ankle
Wall, P.D. Presynaptic control of impulses at the first central
synapse in the cutaneous pathway. Prog. Brain Res., 1964,
Willer, C.J., Bourbeau, F. and Albe-Fessard, D. Supraspinal
influences on nociceptive flexion reflex and pain sensation