Lower limb cutaneous polysynaptic reflexes in the child, according to age and state of waking or sleeping

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SUMMARY An electromyographic study of reflex responses elicited by stimulation of an area of skin in the lower limb was undertaken in awake or sleeping children from 3 days to 3 years of age. Recordings were made on the tibialis anterior and the short head of the femoral biceps. In the awake child, electrical stimulation of the cutaneous area around the toes evoked polysynaptic discharges (R II and R III) in both muscles. From birth to one year of age, the threshold for the tibialis anterior was much lower than for the short head of biceps, and the flexion reflex pattern predominated. After 20 months of age, the recruitment pattern for polysynaptic responses was different: the threshold for tibialis anterior increased and became higher than for the short head of biceps, as in the adult. In sleeping children, the most striking feature was the depression of R II responses. In non-REM sleep, R III responses also were depressed, with a similar threshold in both muscles, and even disappeared during deep sleep. In REM sleep, R III responses were present in babies, but seemed to be abolished in older children.

In a healthy adult, cutaneous electrical stimulation evokes integrated spinal polysynaptic motor reflex responses. Two reflex response components are obtained according to the intensity or type of stimulation: a short latency response, R II, evoked by stimulation of group II cutaneous fibres, and a longer latency and duration response, R III, evoked by nociceptive stimulation of group III cutaneous fibres. The study of these polysynaptic reflexes is important in children, especially in the early years of life when motor and behavioural adaptation take place through exteroceptive experiences. During ontogenesis, control mechanisms progressively develop, so that the exteroceptive reflexes present at birth are modified, and Babinski’s sign disappears. Few publications have dealt with this subject. Hogan and Milligan7 and Katiyar et al8 attempted a clinical study of the plantar reflex in the newborn, and in infants during the first year of life. Prechtl et al9-11 studied mechanically-evoked exteroceptive skin reflexes and their relation to various behavioural states in the newborn. The present study concerns electrically evoked cutaneous motor reflexes during the first 3 years of life and assesses their variations with age and in different spontaneous states of waking and sleeping in order to consider the mechanisms which underlie and control medullary polysynaptic reflex activity. Our results should be compared with those published earlier12 on the reduction of monosynaptic reflex activity during ontogenetic development and, at any age, during sleep.

Subjects and methods

The 55 healthy children (ranging from 3 days to 3 years of age) examined during this study were born at term and were free of neurological disorder. They may be divided into the following age-groups: 3 days to 3 months (n = 18), 3 months and one day to 10 months (n = 12), 10 months and one day to 20 months (n = 16) and 20 months and one day to 36 months (n = 9). The purpose of the procedures was explained and informed consent was obtained from a parent for each infant.

Polysynaptic reflexes were studied in the lower limbs according to Hugon’s method.10 They were evoked by cutaneous stimulation, using ring electrodes. The negative electrode was placed around the big toe and the positive electrode around the three small toes. Stimulation was produced by bursts of 5-1 ms rectangular electric shocks, separated by a 2 ms interval with a delay of at least 5 s between bursts, each burst being delivered when the muscles were relaxed (it is impossible to obtain constant and regular contractions from children). Recordings were taken using either bipolar surface electrodes or subcutane-
ous needles (Alvar Anestho), from the tibialis anterior muscle and the short head of the femoral biceps. The children were placed in the supine position in a room with a constant temperature of 23°C. The lower limb was most often free, with 120° flexion of the knee and 90° flexion of the ankle. Electromyographic reflex responses were studied during various states of waking and sleeping, which were monitored by means of continuous polygraphic EEG recording (Reega Alvar VIII apparatus), four channels being used for the EEG, one for recording eye movements and one for the electromyogram of the chin muscle. The two remaining channels were used for the EMG of the tibialis anterior and short head of the femoral biceps. An aide was present to take care of the children who were in no way distressed by the procedure. Twenty-eight children fell asleep during the investigation.

The latencies of reflex responses were measured from the beginning of the stimulation burst to the first clear deviation from the baseline. The amplitude of responses was not calculated as an appropriate instrument (integrator) was not available, but was estimated visually and given values of 1 (+) to 5 (+++++). For each group of subjects, measurement of the averages and standard deviations was made assuming Student’s distribution. Differences between the two groups were assessed using the t test. To determine non-linear relationships between two parameters, individual values plotted on the graph were divided into zones along the X axis, each zone containing the same number of points. In each zone, the mean was computed according to X and Y values of the points, and the standard deviation apart from the mean from Y values. The increment between two adjacent zones was defined by a step so as to permit a recovery of 90 to 95% of the points in each zone.

Results

1 Cutaneous polysynaptic reflexes in awake children, from birth to three years of age

At any age, stimulation evoked two reflex responses of short and long latency which could be recorded separately for the two muscles studied. If the two responses occurred together, they were separated by a silent period.

In the newborn and in infants to the age of 10 months, reflex responses were recorded first from the tibialis anterior (fig 1), the mean threshold of the early reflex response being 5·5 ± 2·3 mA. At a threshold of 6·5 ± 2·5 mA the long latency reflex response was evoked from the same muscle. When stimulation intensity reached 1·5 times the tibialis anterior reflex response threshold value, short head of biceps responses were evoked (table 1). The variable (16 out of 30) short latency response (9·6 ± 2·5 mA) was recorded at the same threshold as the late responses (9·6 ± 4·4 mA). For children over 20 months of age, reflexes were evoked first in the short head of the biceps femoris (fig 1). The R II response was inconstant and was elicited in only 50% of the cases. It appeared at nearly the same threshold (7·6 ± 3·4 mA) as the late response. When the stimulation threshold was 1·5 times the biceps femoris reflex response threshold value, responses appeared in tibialis anterior (11 ± 2·4; 12 ± 4 mA) (table 1). Thus, in the older children the order of the responses was reversed, and at the same time stimulation intensities required to evoke muscle responses changed, becoming markedly higher for the tibialis anterior muscle while remaining relatively unchanged, or decreasing slightly, for the biceps. Between 10 and 20 months of age, cutaneous reflex responses followed one or other of these patterns. Nine of the 15 children investigated in this age-group presented the reflex response pattern of babies and the six others that of older children. Between 15 and 20 months of age, the striking feature was that the late responses were elicited from both muscles at the same threshold.

At threshold, reflex response latencies changed very little, though they tended to decrease, during the first 10 months of life (fig 2). At birth and during the first three weeks of life, the mean of the early response latencies was 32·5 ± 4·4 ms for the biceps and 37·2 ± 6·5 for the tibialis anterior. Between 3 and 6 months, the latencies diminished becoming 26·2 ± 3·6 ms for the biceps and 29·4 ± 6·6 ms for the tibialis anterior. The decrease in latency be-
Lower limb cutaneous polysynaptic reflexes in the child

Table 1  Mean values and standard deviations for thresholds (mA) of lower limb polysynaptic reflex responses in awake children according to age. For statistical studies, results were compiled according to 2 age groups and response patterns: from 3 days to 20 months old (infant type responses) or group A and from 10 to 36 months old (adult type responses) or group B

<table>
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<tr>
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<th>R II</th>
<th>R III</th>
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<tr>
<td></td>
<td>TA</td>
<td>Bi</td>
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<tr>
<td>3 days—3 months</td>
<td>n = 17</td>
<td>m = 6.1 ± 2.7</td>
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<tr>
<td></td>
<td>m = 6.1 ± 2.7</td>
<td>n = 10</td>
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<tr>
<td>3 months—10 months</td>
<td>n = 11</td>
<td>m = 4.9 ± 1.3</td>
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<tr>
<td></td>
<td>m = 4.9 ± 1.3</td>
<td>n = 6</td>
</tr>
<tr>
<td>10 months, 1 day—20 months infant responses</td>
<td>n = 7</td>
<td>m = 4.6 ± 1.4</td>
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<tr>
<td>A</td>
<td>n = 35</td>
<td>m = 5.4 ± 2.2</td>
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<tr>
<td>10 months, 1 day—20 months adult pattern</td>
<td>n = 3</td>
<td>m = 11.5 ± 2.5</td>
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<tr>
<td>20 months—36 months</td>
<td>n = 6</td>
<td>m = 11.0 ± 2.4</td>
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<td>B</td>
<td>n = 9</td>
<td>m = 11.2 ± 2.3</td>
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<tr>
<td>A—B</td>
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<td>NS</td>
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NS: Not statistically significant.
†Significant p < 0.01.

between the two age groups was statistically significant (p < 0.02) for each muscle. This latency decrease of R II responses continued, in some children, until about the age of 10 months. Between 10 and 16 months, the mean latencies did not change. Conduction times increased between 16 and 36 months of age and, then, the mean of the early response latencies was 46.7 ± 8.3 ms for the biceps and 47.3 ± 9.9 ms for the tibialis anterior. The difference between the mean of R II response latencies in children from 3 to 16 months of age and that of R II response latencies in children from 16 to 36 months of age was statistically significant (p < 0.01) for each muscle. At threshold, late response latencies were similar for both muscles and followed the same course as the early response latencies. The mean of the late response latencies during the first 3 weeks of life was 127.3 ± 30.1 ms for biceps and 130.5 ± 19.5 ms for tibialis anterior. Between 3 and 16 months of age, the mean of the late response latencies was 98.8 ± 29.2 ms for biceps and 101.3 ± 25.3 ms for tibialis anterior. The difference between the two age groups was statistically significant (p < 0.02) for the two muscle responses. After the age of 16 months, the latencies increased, the mean becoming 114.6 ± 20.99 ms for biceps and 114.3 ± 22.5 ms for tibialis anterior. The difference in the mean of late reflex latencies between the group of children 3–16 months old and that 16–36 months old was statistically significant (p < 0.05) only for tibialis anterior response latencies. Late response latency values differed greatly from one child to another, especially during the first months of life, as indicated by the SD values.

Latencies of polysynaptic reflex responses changed according to the stimulus strength. When the stimulus intensity was changed from a threshold level up to a value just giving a maximal response, the latency of R II reflex responses exhibited an inconstant and small variation (5 ± 2 ms). In contrast, R III latency was sharply reduced (32 ± 9 ms of decrease), and the duration of the response strongly increased. In some cases, the final compo-
nents of the R II response and the first components of the R III response tended to overlap. These changes appeared rather similar, whatever the age of the child.

Reflex response amplitude was estimated visually. At any age, late responses (++++ to ++++++) were always larger and more prolonged than early ones (+ to ++ +). Between birth and 10 months of age, reflex responses elicited from the tibialis anterior were larger than those (particularly early ones) from the femoral biceps. After 20 months of age, the amplitude of each reflex response was similar for both muscles. While the amplitude of the reflex responses varied only slightly with age, it increased with stimulus intensity.

In summary, in our population reflex responses appeared in a different order according to age. After 20 months of age, cutaneous polysynaptic reflex responses occurred as in adult subjects. Reflex response latencies changed little, though they decreased between birth and 16 months of age, and then increased. Reflex responses had a higher amplitude and are more prolonged when stimulus intensity was increased, whereas latencies were reduced. Early response amplitudes always were lower than those of late responses.

2 CUTANEOUS POLYSYNAPTIC REFLEXES IN CHILDREN, ACCORDING TO STATE OF WAKING AND SLEEPING

Three experimental situations were encountered. For each, our results take into account examinations performed on the same children in different states of waking and sleeping.

Table 2  Mean values and standard deviations for threshold (mA) of lower limb polysynaptic reflex responses in children, according to state of waking and sleeping. Statistical results compare the mean threshold in each state of vigilance with that for the same children in quiet wakefulness

<table>
<thead>
<tr>
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<th>3 days—10 months</th>
<th>20 months—36 months</th>
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<tbody>
<tr>
<td></td>
<td>Active wakefulness</td>
<td>Non REM sleep</td>
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<tr>
<td>Tibialis anterior</td>
<td></td>
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</tr>
<tr>
<td>R II</td>
<td></td>
<td></td>
</tr>
<tr>
<td>n = 11</td>
<td>m = 4.3 ± 1.9</td>
<td>m = 9.4 ± 4.2</td>
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<tr>
<td>R III</td>
<td></td>
<td></td>
</tr>
<tr>
<td>n = 11</td>
<td>m = 5.4 ± 2.3</td>
<td>m = 9.6 ± 3.4</td>
</tr>
<tr>
<td>Short head of the femoral biceps</td>
<td></td>
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<tr>
<td>R II</td>
<td></td>
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<tr>
<td>n = 11</td>
<td>m = 6.0 ± 2.1</td>
<td>m = 13.5 ± 3.8</td>
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<tr>
<td>R III</td>
<td></td>
<td></td>
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<tr>
<td>n = 11</td>
<td>m = 6.9 ± 2.9</td>
<td>m = 11.3 ± 5.9</td>
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NS: Not statistically significant.
*Significant p ≤ 0.05.
†Significant p ≤ 0.01.

A Active wakefulness (n = 19) Some children were studied when exhibiting restless behaviour, with tonic muscular activity, most often provoked by the first pulse trains delivered to the toes, even at threshold intensity. Reflex responses elicited by increasing stimulation showed a similar pattern to that in awake and relaxed children, with variations according to age. However, early responses were easier to elicit since for both muscles they always appeared before the late responses. Comparison of the threshold of each reflex response showed that it was 15% to 50% lower in children during active wakefulness (table 2). This reduction was evident regardless of age but is more apparent in babies. Late reflex responses were more prolonged, and their latencies reduced. The latencies sometimes were difficult to evaluate because reflex responses could be marked by tonic muscular activity, or late responses could be mixed with the early ones. In active wakefulness, latencies of late responses, at threshold, were from 10 to 20 ms shorter than in quiet wakefulness. Between birth and 3 weeks of age, mean values were 119 ± 23.5 ms and 114.6 ± 21 ms; between 3 and 16 months of age, the mean values were 80.5 ± 9.9 ms and 80 ± 12.8 ms for tibialis anterior and biceps respectively and after 16 months of age, 102 ± 16.4 ms for both muscles. The amplitude of the responses was higher, especially for early responses. This was obvious even at threshold and in comparison with the amplitude of early responses elicited in relaxed waking at the same stimulus intensity. These results were contrary to those obtained during quiet sleep.

B Non-REM sleep (n = 25) The early responses
for both muscles most often disappeared (in 17 out of 25 children investigated during quiet sleep). When the early responses were present, their thresholds were higher than during relaxed waking, especially for early reflex responses from the tibialis anterior in babies (table 2). Late responses for all the children tended to appear simultaneously, their amplitude being similar for both muscles (fig 3). In only one case, that of a six-month old baby, did reflex responses appear first for the biceps. In general, thresholds were higher during quiet sleep than during relaxed waking and increased when sleep becomes deeper. In six children, no reflex responses were elicited in deep sleep, even when stimuli reached 20 and 30 mA, at which point the children awakened. Latencies were prolonged for late reflex responses, increasing from 8 to 25 ms, at threshold, over those of relaxed waking. Thus, from birth to 21 days of age, the means of late response latencies were, respectively, for tibialis anterior and biceps 140.7 ± 13.3 ms and 140 ± 16 ms; between 3 and 16 months of age, they became 123 ± 30.9 ms and 117-75 ± 27.8 ms; and after 16 months, 129-6 ± 12.7 ms and 124 ± 9.3 ms. At the same stimulus intensity, the amplitude and area of responses were less pronounced during non-REM sleep than during waking. This was true at any age once quiet sleep begins but was more marked as sleep becomes deeper. Thus during non-REM sleep, early reflex responses disappeared in 68% of cases and late responses appeared simultaneously for both muscles at a higher threshold than in the waking state. Response latencies increased, amplitudes were lower and reflex excitability decreased.

C REM sleep In all cases, stimuli were delivered when there were ocular movements, no muscular activity and no discontinuous though classical jerks. Exteroceptive reflex responses during REM sleep varied according to age. Of the 12 children investigated during this type of sleep, five who were between 5 and 27 days old always presented exteroceptive reflexes. In five children from 45 days to 6 months of age, exteroceptive reflex responses were variable, being present in three cases and absent in two. The reflex was not evoked in two children over 13 months old. When exteroceptive reflexes were present, they had the same pattern as described during the waking state. Early responses were never evoked easily. If non-existent during the waking state, they did not appear during REM sleep; if present during relaxed waking, they usually remained during REM sleep, but appeared at a higher stimulus intensity (table 2). Late responses in REM sleep were similar to those in relaxed waking, appearing at the same or even a lower threshold. The different response latencies were very similar in relaxed waking and in REM sleep, the mean value of the early response latencies being 36.7 ± 5.8 ms for tibialis anterior and 30 ms for biceps and that of the late response latencies 134.25 ± 27 for both muscles (n = 8). At threshold, early responses had a lower amplitude in REM sleep than in relaxed waking. On the contrary, late responses, which were always elicited, had a higher amplitude and a larger area than during waking. It is difficult to draw general conclusions from a relatively small population of children; however, it would appear that exteroceptive reflexes were always present in REM sleep during the first month of life and then become variable before disappearing. When evoked, late responses were similar to those during waking, but early responses were weaker or abolished.

Discussion

POLYSYNAPTIC REFLEX RESPONSES DURING WAKING ACCORDING TO THE AGE OF THE CHILD

Our results make it possible to obtain quantitative data concerning changes in polysynaptic reflexes during the early years of life. During the first year, polysynaptic reflex responses, which are present from birth, appear initially in the tibialis anterior, a muscle permitting dorsiflexion of the foot, at a mean intensity of 6 mA. Then, when stimulation intensity
is raised to 8.5 to 10 mA, responses occur in the biceps, the thigh extensor muscle. These responses have a rather constant latency, with a greater amplitude for the tibialis anterior response. Beginning at 20 months of age, the order of appearance of the reflex responses is reversed, with motor reflex responses appearing first in the biceps and then in the tibialis anterior at higher stimulation thresholds of around 11 or 12 mA. The latencies of the responses increase continually, and amplitude becomes similar for both muscles.

These various changes are in keeping with what is known about the ontogenesis of the nervous system. The cutaneous afferents involved in these reflexes gradually increase in diameter during maturation. Likewise, the number of myelinated nerve fibres gradually increases in the sural nerve and the dorsal roots in the newborn human as well as in the kitten. This development is faster in fibres of larger diameter. These facts could explain why the latency of early responses remains rather constant although a statistically significant decrease is observed during the first 16 months of life: the increase in the length of the reflex arc is offset by the increase in conduction velocities. They could also explain the similar changes in conduction time value over the monosynaptic reflex pathway. Progressive inhibition of the dorsal flexors of the lower limb seems to be in accordance with clinical findings, in particular, in accounting for the disappearance of Babinski’s sign in the young child. It has been shown that Babinski’s sign is part of the flexion reflex, being in man comparable to the defensive flexion reflex after nociceptive stimulation that Sherrington described in animals. All the children of less than 10 months of age that we examined demonstrated Babinski’s sign, the manifestation of their reflex responses being similar to that described in the spastic adult. Nevertheless, it should be noted that there are differences in clinical interpretation of Babinski’s sign during the first year of life and by different authors, just as there are many parameters likely to contribute to the cause of these reflex responses in the child, the normal adult or the adult suffering from lesions of the pyramidal tracts. The works just cited, as well as a number of neurophysiological studies in animals, in healthy or pathologically affected humans, or in the newborn, suggest that supraspinal control over polysynaptic cutaneous reflexes may develop during the early years of life.

In animals, the pyramidal tracts exercise a presynaptic inhibitory influence over afferent neurons responsible for polysynaptic flexor reflexes, as well as a postsynaptic facilitative influence over spinal interneurons responsible for polysynaptic extensor reflexes. Experimental studies in humans suffering from lesions of the pyramidal tracts emphasise the facilitation of the polysynaptic flexor reflex upon removal of an inhibition, whereas the polysynaptic extensor reflex is not affected. This accounts for the changes in response reflexes and Babinski’s sign. In the normal adult, there is presumably a cerebral control at the level of the spinal interneurons which regulates transmission, not only over the reflex arcs but also over the ascending spinal pathways, which can themselves influence descending cerebral control of the internuncial reflex transmission. In the child, Schulte has shown the predominance of flexion patterns, even though the principal spinal mechanisms are functional from birth and the influence of supraspinal control is evident within the first week of life. Brain and Wilkinson have drawn a parallel between the progressive reduction in the receptor field of the flexor reflex during the first year of life and the cephalocaudal progression of myelinisation, which, for the corticospinal tract is completed between the 9th and 24th months of life. During the first two years of life, the inhibitory mechanisms of pyramidal suprasegmentary origin are thus in place, modifying the excitability of interneurons or acting on the afferents of the flexor reflex. It would thus seem of interest to relate these changes in polysynaptic reflexes to the decrease in excitability of the monosynaptic reflex pathway that we have described in the child during the early years of life. This phenomenon, also observed in the kitten, would not seem to be due to peripheral factors, but rather to the progressive rise in the subliminal fringe— with age and to the establishment of a supraspinal inhibitory tonic control.

POLYSYNAPTIC REFLEX RESPONSES DURING DIFFERENT STATES OF WAKING AND SLEEPING

Hyperpnoea or crying facilitate early and late responses, regardless of the child’s age. There is a lowering of thresholds and an increase in response amplitude. This situation is characterised by an intense awake state and a basal motor activity. Thus, it is difficult in this case to know which control mechanisms are responsible for the variations in polysynaptic reflex responses. What is known is the influence of the limb’s position and of muscle contraction on polysynaptic reflexes.

Spontaneous sleep often occurs in the child. The study of polysynaptic reflexes during different states of sleep allows mechanisms of spinal, particularly reticular, control to be considered. Our results show a strong and constant reduction, even an abolition, of early responses. Late responses are also reduced or sometimes abolished during deep slow-wave
Lower limb cutaneous polysynaptic reflexes in the child

sleep. They are retained during desynchronised sleep in the first six months of life, then seem to disappear after the age of one, except for muscle twitches inherent in this type of sleep. These findings are in agreement with clinical studies which conclude that there is, in the newborn, a persistence of polysynaptic reflexes of a protective order, regardless of the waking state. In the healthy adult, the evolution of the polysynaptic nociceptive reflex, as studied by attempts to elicit Babinski's sign, has led to differing results. Babinski's sign is said to be present during slow-wave sleep with some authors claiming that it persists during desynchronised sleep and others that it disappears. In the adult suffering from lesions of the pyramidal tracts, Babinski's sign, present during waking, is said to be even more marked during slow-wave sleep and to disappear during REM sleep. These facts suggest that the control mechanisms of such a reflex are different during waking and desynchronised sleep. The sharp depression in early R II responses during sleep suggests that there is a preferential and probably active inhibition of messages transmitted to the spinal cord by large diameter, low threshold cutaneous afferents. On the other hand, the awake state or tonic activity facilitate these responses. Experimental studies carried out by Pompeiano et al show that during desynchronised sleep polysynaptic reflex activity (like that of the monosynaptic reflex as well) is inhibited. During this type of sleep, control mechanisms of spinal motor activity are linked, at least in the cat, with the activating of descending inhibitory controls. These controls descend, on the one hand, from the pontine reticular formation exercising their influence postsynaptically on the motoneurons, and, on the other probably from the median and descending vestibular nuclei, acting on interneurons. These same vestibular nuclei are essential in evoking the rapid eye movements in this type of sleep.

During slow-wave sleep, depression of polysynaptic early responses has also been reported by Shahani in the normal adult, whereas these responses are retained in the Parkinsonian patient. In comparing his results with those of various animal experiments, Shahani argues that changes in responsive in the healthy adult are connected with the activation of a monoaminergic, dopaminergic or noradrenergic, pathway inhibiting transmission of tactile afferent messages at the interneuronal level. Finally, in the child less than one year of age, slow-wave sleep provokes a distribution of long-latency polysynaptic reflex responses which are comparable with those of the older child. Moreover, we have shown elsewhere that monosynaptic reflex excitability decreases with age and, at a given age, during sleep. Thus, spinal excitability changes with age, and slow-wave sleep always has a depressing effect.

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