Development of the Stretch Reflex in the Newborn: Reciprocal Excitation and Reflex Irradiation

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MYKLEBUST, BARBARA M., and GOTTLIEB, GERALD L. *Development of the Stretch Reflex in the Newborn: Reciprocal Excitation and Reflex Irradiation.* CHILD DEVELOPMENT, 1993, **64,** 1036–1045. The stretch reflex is a spinal cord pathway between a muscle's stretch receptors and its own motor units. This reflex is thought to play an important role in normal motor function, because it is unique as a monosynaptic pathway, and because its hyperactivity is a hallmark of many motor disorders. We previously reported a difference in the stretch reflexes in healthy newborn infants and nonambulatory individuals with cerebral palsy (CP): these reflexes are characterized by responses from the stretched muscle *and* from the reciprocal or antagonist muscle. We proposed the existence of a functional spinal cord pathway that connects both agonist and antagonist muscles at a single joint. We hypothesized that this "reciprocal excitation" is a functional pathway of all newborn infants, which during the normal course of development of motor skills in infants is eliminated. If the CNS is damaged at birth, as in CP, the pathway of reciprocal excitation endures and is reinforced. In the current study of newborns, we recorded stretch reflex responses from *all* major muscle groups of the lower limb. This "irradiation of reflexes" is a normal phenomenon of the newborn CNS. This pathway becomes suppressed during normal maturation and control of coordinated limb movements.

Classic assessments of infant neuromotor functioning have focused on the integrity of the so-called infantile or primitive reflexes. This diverse group of movements, which include the Moro, stepping, placing, rooting, and palmar and plantar grasp reflexes, observed in newborn infants, wane over the first months of life, and eventually disappear. There is considerable controversy over whether the early patterns are inhibited by higher brain maturation or are incorporated into later movements, or indeed whether some of them can be considered "reflexes" at all (Barnes, Crutchfield, Heriza, & Heardman, 1990).

The continuity and importance of one newborn infant reflex, the stretch reflex, is incontrovertible, however. Muscle stretch reflexes are responses to rapid stretch. Consider the familiar case of the tap below the knee cap with the reflex hammer and the subsequent reflex extension of the lower leg. The tap on the tendon briefly but rapidly stretches the quadriceps muscle group to which it is attached. Receptors in the muscle detect the stretch and signal the spinal cord by way of afferent fibers which synapse directly with motor units, a *monosynaptic* connection. The muscle begins to contract soon (35–40 ms) after the stretch is delivered, leading to the "jerk" of the leg. Normally the afferent fibers also connect through an interneuron to the motoneurons of the antagonist muscle group, the hamstrings, but this connection is inhibitory. This causes the extensor muscle to contract, while the flexor muscle relaxes. This pattern of the mutual excitation of the agonist and inhibition of the antagonist is called *reciprocal inhibition* (Fig. 1, left side).

Stretch reflexes are presumed to be an essential part to normal coordinated and controlled movement, because they provide rapid detection and modulation of active

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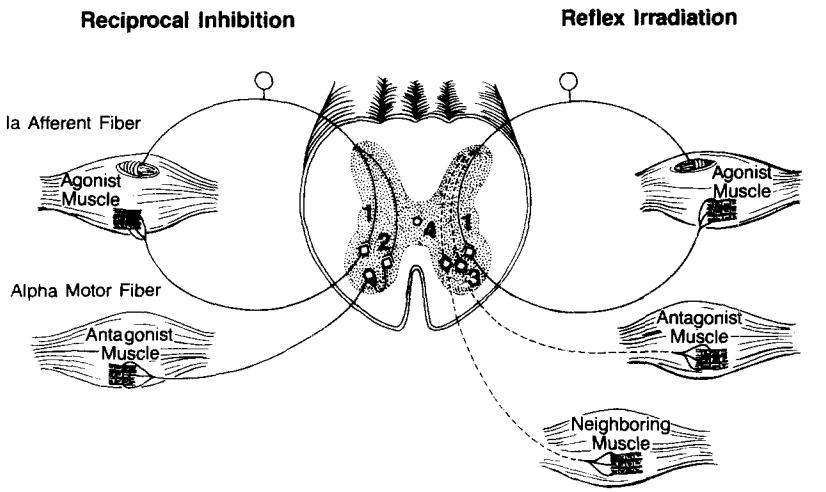

FIG. 1.—Schematic representation of spinal cord circuitry, illustrating the normal stretch reflex pathway of reciprocal inhibition (left) (Paths 1 and 2), and hypothetical functional pathways of reflex irradiation (right) (Paths 3 and 4) in the newborn infant. The normal adult response to stretch results from the firing of Ia afferent fibers which are excitatory on the agonist motor fiber (Path 1) and simultaneously inhibitory on the antagonist muscle through an interneuron (Path 2). We propose that in the newborn, stretch of a muscle is simultaneously facilitory on the stretched muscle and its antagonist by monosynaptic or polysynaptic pathways (reciprocal excitation, Path 3), and also facilitory on neighboring limb muscles by way of segmental interneurons (Path 4). The role of modulation of reflexes by descending tonic inhibition cannot be discounted.

muscle force as a function of the external forces acting to lengthen or shorten the muscles. People with disorders of movement often have abnormal stretch reflexes. For example, in individuals with cerebral palsy (CP), the stretch reflexes are often hyperactive. In addition to this hyperactivity, these individuals may also have *reciprocal excitation* of antagonist muscles, when both the agonist and antagonist muscles are excited, rather than inhibition of the antagonist muscles seen in normal individuals (Gottlieb, Myklebust, Penn, & Agarwal, 1982; Myklebust, Gottlieb, Penn, & Agarwal, 1982). Thus, taps of the Achilles tendon evoke brief electromyographic (EMG) responses from both the stretched soleus muscle *and* the antagonist tibialis anterior muscle. These responses occur in both muscles at similar monosynaptic latencies and comparable amplitudes. To explain these data in individuals with cerebral palsy, we proposed that the connections from muscle spindle afferent neurons simultaneously excite both agonist and antagonist motoneuron pools (see Fig. 1, right side, pathways 1 and 3).

Cerebral palsy is the result of injury to higher brain centers at or near the time of birth. Why do these individuals have abnormal spinal reflexes? The conventional explanation is that both the abnormal reflex profile and the more significant deficits in voluntary motor activities are the direct result of the cerebral injury. We believe that a developmental explanation is more likely (Leonard, Hirscheld, & Forssberg, 1988; Myklebust, 1986, 1990; Myklebust, Gottlieb, & Agarwal, 1986)—that is, that reciprocal inhibition is an acquired pattern, and that the cerebral injury causes the child to retain an infantile reflex pattern.

The basis for this conclusion is our discovery of reciprocal excitation, similar to that seen in CP patients, in lower limbs of normal newborn infants (Leonard et al., 1988; Myklebust, 1986, 1990; Myklebust et al., 1986). Like the CP individuals, newborns respond to an Achilles tendon tap with excitation at both the soleus and anterior tibialis. This suggests that the pathways mediating the reflex are different for newborns than for adults; this raises the possibility that the reflex hyperactivity in CP is the result of damage to the spinal cord that develops subsequent to the cerebral injury. Equally important is the developmental progression from the diffuse newborn pattern of the reflex to the more differentiated adult reflex.

If, in newborns, excitation from a specific stretch stimulus spreads to the antagonist muscle, it is important to ask whether

this effect is localized only to the antagonist muscle, or is more widely distributed. Thus, in this study, we tested the spread of reflex EMG elicited by a tendon tap to other muscle groups of the lower limb (soleus, tibialis anterior, quadriceps, and hamstrings). We found diffuse responses that go beyond the agonist-antagonist pair.

The results of our study have led us to expand our concept of immature reflexes from "reciprocal excitation" of muscle antagonists at a single joint, to one of "reflex irradiation." By this we denote a reflex with one or at most a few synapses at adjacent levels of the spinal cord which link muscles at more than one joint. Reflex irradiation is a normal characteristic of the newborn nervous system.

Method

Tendon jerk reflexes were tested in seven healthy male newborns from 1 to 3 days old (Table 1). For each, the full-term pregnancy and delivery were uneventful. Developmental examination confirmed neuromuscular and physical maturity; gestational age of 39–42 weeks was determined by physical examination. The size of each infant was appropriate or large for gestational age, with birth weights from 2,807 to 4,111 grams. Apgar scores were 8 or 9 at 1 min, and 9 at 5 min for all infants.

During testing, the infant lay supine, with the hips flexed and externally rotated; the knees were flexed, and the ankles were in a neutral position. The head was maintained in the midline for sleeping infants; they turned their heads to the right when awake. The infants were observed in various states of consciousness—awake or light sleep (Brazelton, 1973). Tendon taps were administered only when the infant was lying quietly and not kicking. No differences in reflexes were found with state of consciousness (O'Sullivan, Eyre, & Miller, 1991), except that reflexes could not be obtained consistently during deep sleep (see Fig. 4). This is consistent with studies of the H-reflex (Hodes & Grebitz, 1962). Due to large-amplitude background EMG activity during kicking, reflex EMG responses would not have been identifiable during these movements.

Taps were applied no more often than every 15 sec to the Achilles and patellar tendons. The number of taps applied depended on the infant's cooperation. Testing lasted up to 30 min.

Tendon jerks were elicited with a modified reflex hammer instrumented to trigger the computerized data collection system (Myklebust, Gottlieb, & Agarwal, 1984; Myklebust et al., 1986). Surface EMGs were recorded from soleus (SOL), tibialis anterior (TA), quadriceps (QUAD), and hamstrings (HAM) muscles. Boston elbow myoelectrodes (MYO-111; Liberty Mutual Research Center, Hopkinton, MA) were secured to the limb with Velcro straps. These electrodes had a gain of 3000, and they filtered out signals below 4 Hz and above 500 Hz. Electrodes were 0.5 cm in diameter. There was 1.5 cm between the centers of the electrodes.

The signals were digitized by computer at a rate of 1,000 samples/sec. The maximum peak-to-peak amplitude of the biphasic response was measured by computer.

Measurements of peak EMG amplitudes and of latencies were made in all four

TABLE 1

CHARACTERISTICS OF NEWBORN INFANTS TESTED BY STRETCH REFLEX

Infant No.	Test Age (Days)	Developmental Size (AGA or LGA)[a]	AGE (Weeks) by Exam/Dates[b]	APGAR Score, 1 Min/5 Min	Birth Weight (gm)
1	3	AGA	40/42	8/9	3,300
2	3	AGA	39/40	8/9	2,807
3	3	LGA	39/40	8/9	4,111
4	1	LGA	41/40	9/9	3,912
5	1	AGA	40/40	9/9	3,572
6	3	LGA	40/40	9/9	4,034
7	3	LGA	40/42	9/9	3,920

[a] AGA: appropriate for gestational age, LGA: large for gestational age (based on birth weight, length, and head circumference).

[b] Based on developmental assessment of physical maturity and time since conception.

muscles. To quantify the degree of simultaneous (or near-simultaneous) activation of the antagonists, we computed the ratio of the peak-to-peak, stretch-evoked EMG voltages, or the TA/SOL reflex ratio for Achilles tendon taps (Myklebust et al., 1982, 1986), and the HAM/QUAD reflex ratio for patellar tendon taps (Myklebust et al., 1986).

Results

In the healthy adult, a tap to the Achilles tendon evokes a stereotypic synchronous burst of EMG in the stretched soleus muscle at short (monosynaptic and polysynaptic) latency of 35–40 ms. In this interval, relative electrical silence is observed in the antagonist tibialis anterior muscle, as well as in the neighboring limb muscles quadriceps and hamstrings (Fig. 2). This pattern reflects the unique, strong, and monosynaptic nature of this reflex (Burke, Gandevia, & McKeon, 1984; Magladery & McDougal, 1950). The latency and waveform of the EMG response are very stable on successive taps (Myklebust, 1986). The ratio of the peak-to-peak EMG response measured in the TA and SOL muscles (i.e., the TA/SOL reflex ratio) was calculated for each tap. This ratio is usually less than 0.1 in the normal adult (e.g., from Fig. 2, $(0.15\ mV_{TA})/(3.6\ mV_{SOL} = 0.042)$) (Myklebust et al., 1982, 1986). This means that the tendon jerk reflexes are at least 10 times greater in the tapped muscle than in its antagonist. Similarly, in response to a patellar tendon tap, the HAM/QUAD reflex ratio is also usually less than 0.1 in the normal adult.

In healthy newborns, tendon jerk reflex responses are significantly different from those in the adult. There are monosynaptic latency EMG responses in agonist, antagonist, *and* neighboring limb muscles, with greater trial-to-trial variability in the amplitude and latency of these evoked responses (Myklebust, 1986).

Figure 3 is a composite graph from one infant illustrating typical sets of four EMG recordings from the limb muscle groups (gastrocnemius-soleus, tibialis anterior, quadriceps, and hamstrings) in response to three sequential Achilles tendon taps. The amplitude of the response (and the absence of muscle activity) is variable among successive tendon taps. That is, the first tap may evoke a reflex from the agonist and antagonist muscles, while the neighboring muscles are quiet; the next tap may evoke reflex responses from agonist and neighboring muscles, while the antagonist muscle is quiet; and the following tap may evoke a response from the neighboring limb muscles, and none from the agonist muscle. The results for seven infants are summarized in Tables 2 and 3. The key feature of these data is that while the SOL amplitudes in Table 2 and QUAD amplitudes in Table 3 are not unexpected responses to the delivered stimuli, all the other amplitudes would, in a normal adult, be very close to zero. The average of the individual response ratios is shown, not the ratio of the averages.

The tables shows that, in response to the Achilles tendon taps, SOL and TA responses

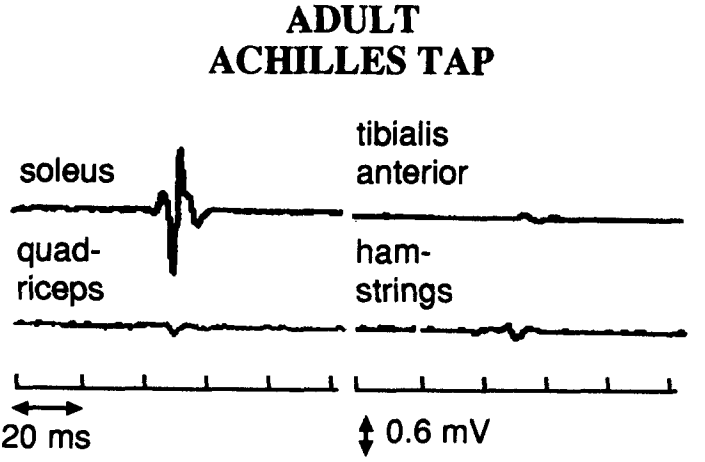

FIG. 2.—An Achilles tendon tap to a healthy adult subject evokes short latency reflex EMG in the agonist soleus muscle; there is relative electrical silence in the antagonist tibialis anterior muscle, as well as in the proximal quadriceps and hamstrings muscle groups.

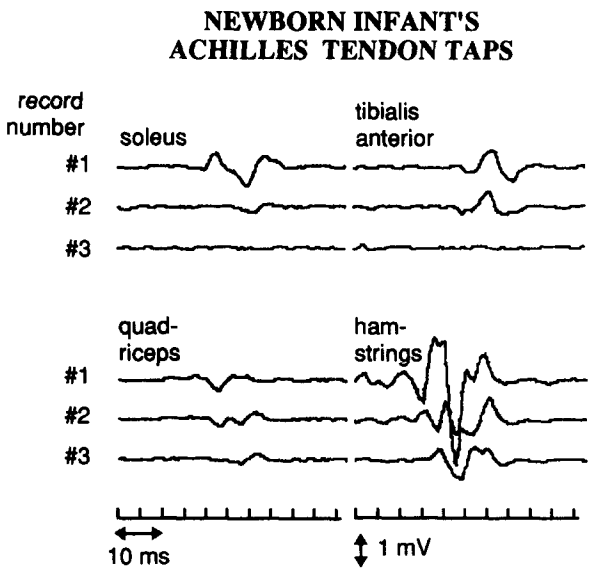

FIG. 3.—EMG responses from the soleus muscle, tibialis anterior muscle, quadriceps, and hamstrings muscle groups following three sequential Achilles tendon taps to one newborn infant. Trial-to-trial variability in the amplitude of the reflex response in all four muscle groups is a common finding in all the infants tested.

occurred at monosynaptic latencies.[1] Note that QUAD and HAM latencies are shorter. This is consistent with a shorter neural conduction path from the tapped SOL muscle to the responding HAM and QUAD muscles. Conversely, latencies of HAM and QUAD muscles following patellar tendon taps were monosynaptic, while latencies of SOL and TA muscles were longer.

Discussion

The data confirm and expand our previous findings about lower limb reflexes in a different, larger population of newborns. Compared to healthy adults, newborns differ in at least four significant ways: (1) The mean TA/SOL reflex ratios for Achilles tendon taps, *and* the mean HAM/QUAD reflex ratios for patellar tendon taps, are higher for newborns. (2) Sequential reflexes are more variable in latency, amplitude, and waveform (see Fig. 3). (3) There is more variability in the relative excitability of the agonist and antagonist muscles that is not related to state of consciousness of the infant. (4) A tendon tap can evoke short-latency EMG responses from *neighboring*, as well as antagonist, muscles. These results demonstrate that the difference between adult and the newborn reflex response to a tendon tap is not merely excitation of agonist and antagonist muscles (i.e., reciprocal excitation). It is broader and better characterized as *reflex irradiation* to neighboring limb muscles. These observations are quite similar to a recent report on reflexes in the upper limbs of newborns, children, and adults (O'Sullivan et al., 1991).

The origin of the variability could be due to many factors. These include variability in contraction levels of muscles of the leg just prior to the tap to the limb, variability of the excitability of the sensory receptors (muscle spindles), the variability resulting from incomplete myelination of the reflex pathways, the variability of synaptic connections, and the variability of descending inputs from higher centers.

Despite the similarity of the infants to each other by clinical (developmental and neurological) examination, there are significant differences in reflex responses recorded

[1] Anthropomorphic data on neonates give the rump-to-sole distance as 23.1 ± 1.9 to 28.8 ± 2.6 cm in 40 week gestation infants (Snyder et al., 1977). Nerve conduction studies for the tibial nerve of term infants are 25.8 ± 2.0 to 28.8 ± 2.0 m/sec (Goeshchen, Purta, Rothe, & Saling, 1983; Schulte, 1978). The resulting approximate conduction time from the ankle to the lumbar spine and back [(2 × 23.1 cm/28.8 m/sec to 2 × 28.8 cm/25.8 m/sec] is 16 to 22 ms.

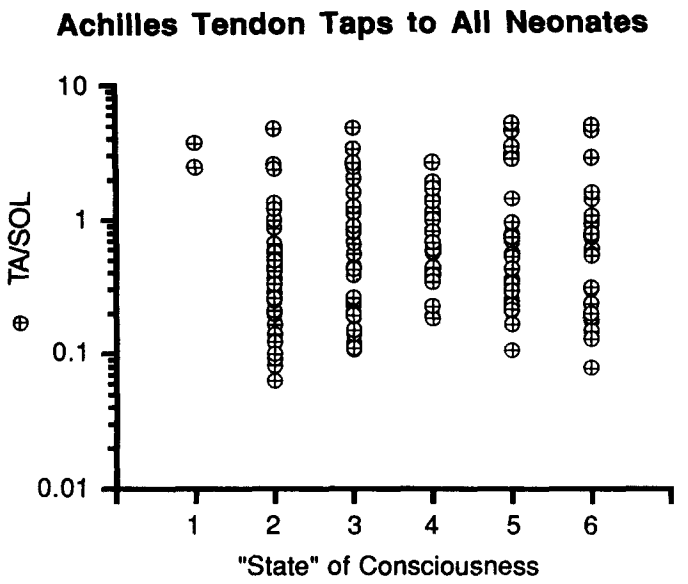

FIG. 4.—TA/SOL reflex ratios for Achilles tendon taps to infants, plotted with respect to "state of consciousness" (Brazelton, 1973): state 1: deep sleep, no movement, regular breathing; state 2: light sleep, eyes shut, rapid eye movement, irregular respiration, some limb movements; state 3: drowsy or semidozing, eyes opening and closing, activity variable, movements usually smooth; state 4: awake, alert, with bright look, eyes open, minimal motor activity; state 5: wide awake, periodic vigorous movement; state 6: crying.

by EMG of the leg muscles. Moreover, the variability in reflex responses *between* infants is as great as the variability in reflex responses *within* an infant.

Three possible mechanisms might produce irradiation of responses:

1. *Volume conduction* of the electrical signal to remote-recording electrodes (Myklebust et al., 1982): Volume conduction of electrical signals is a passive and stable phenomenon. An electrical signal, like the EMG, attenuates rapidly with distance; an EMG electrode that is remote from the excitable tissue always records a *smaller* signal than the electrode that directly overlies the stretched muscle. That is, following an Achilles tendon tap and stretch of the SOL muscle, the muscle activity recorded at the SOL electrode will always be larger than at any other electrode on the limb that is picking up volume-conducted EMG activity from the SOL muscle. This is not the case in our studies: for Achilles tendon taps, the TA EMG is often *larger* than the SOL EMG, and SOL responses can be as large following Achilles taps as patellar tendon taps. These findings cannot be explained by volume conduction alone.

Second, when an electrical signal is volume conducted through a "constant" medium (i.e., the leg of the infant remains unchanged during the test), the differences in waveform characteristics (amplitudes and latencies of EMG activity) between any two recording electrodes should be constant (as long as the electrodes are not moved). That is, the onset time and shape of the EMG signal at the SOL electrode should always have the same relation to those waveform characteristics at the TA electrode, with each and every tap. Examination of individual EMG records fails to demonstrate this consistency.

Third, volume conduction is a passive property of the material in which the electrical signal is transmitted. Therefore, the volume conduction of the SOL signal to the TA electrode is the same as the volume conduction of the TA signal to the SOL electrode. If volume conduction were the only property responsible for the data, then the relation between agonist versus antagonist EMG *waveform* should always be the same, regardless of the tap site. Furthermore, the *onset latency* differences between the agonist and antagonist muscles would be constant from trial to trial at each tap site; this is not the case in any one infant. Moreover, the onset latency differences at neighboring muscles should be constant from trial to trial

TABLE 2

STRETCH REFLEX EMG AMPLITUDES AND LATENCIES FOR ACHILLES TENDON TAPS IN NEWBORN INFANTS

Infant No.	Number of Taps	SOL Amplitude	SOL Latency	TA Amplitude	TA Latency	TA/SOL Reflex Ratio	QUAD Amplitude	QUAD Latency	HAM Amplitude	HAM Latency
1	13	.70 ± .44	19.1 ± .8	.26 ± .19	20.4 ± 2.3	.48 ± .29	.14 ± .09	19.2 ± 4.5	.19 ± .13	17.9 ± 2.0
2	20	.62 ± .39	18.2 ± 1.6	.18 ± .16	18.4 ± 1.7	1.62 ± 1.47	.19 ± .11	15.3 ± 1.4	.52 ± .33	15.0 ± 1.5
3	14	.42 ± .53	18.3 ± 2.9	.34 ± .31	17.9 ± 3.1	.30 ± .10	.29 ± .19	15.2 ± 3.2	1.72 ± 1.47	14.3 ± 2.3
4	16	1.51 ± 1.36	19.3 ± 2.0	1.15 ± .21	22.3 ± 3.2	.58 ± 1.04	.11 ± .06	21.3 ± 4.1	.30 ± .34	20.2 ± 4.2
5	32	.29 ± .26	20.8 ± 2.7	.39 ± .60	21.6 ± 2.0	1.59 ± 1.25	.44 ± .27	15.2 ± 1.7	2.30 ± 1.04	14.2 ± 1.7
6	20	2.69 ± 3.29	15.7 ± 2.2	.68 ± .73	16.3 ± 2.0	.66 ± .78	1.00 ± .40	14.6 ± 1.5	4.68 ± 1.77	14.2 ± 1.3
7	18	1.22 ± .92	17.5 ± 2.5	2.01 ± 2.35	17.2 ± 2.4	1.19 ± 1.28	3.45 ± 2.66	13.9 ± 1.3	.62 ± .35	14.8 ± 1.7

TABLE 3

STRETCH REFLEX EMG AMPLITUDES AND LATENCIES FOR PATELLAR TENDON TAPS IN NEWBORN INFANTS

Infant No.	Number of Taps	SOL Amplitude	SOL Latency	TA Amplitude	TA Latency	QUAD Amplitude	QUAD Latency	HAM Amplitude	HAM Latency	HAM/QUAD Reflex Ratio
1	9	.22 ± .31	19.7 ± 2.0	.10 ± .05	18.6 ± .1	2.17 ± 2.60	14.5 ± 1.3	.30 ± .33	16.7 ± 1.5	.26 ± .17
2	13	.09 ± .03	13.8 ± 1.2	.45 ± .31	13.7 ± 1.3	1.81 ± .89	12.7 ± 1.3	.34 ± .17	16.1 ± 2.2	.18 ± .03
3	5	.05 ± .02	19.7 ± 2.0	.26 ± .03	18.9 ± 2.0	2.52 ± 1.72	14.2 ± 1.9	.23 ± .23	14.8 ± 3.2	.19 ± .04
4	5	.05 ± .01	23.4 ± 1.0	.07 ± .03	23.1 ± 1.0	1.65 ± .30	13.1 ± .5	2.65 ± .65	13.0 ± .5	1.45 ± .13
5	12	.54 ± .81	21.1 ± 2.9	.20 ± .18	20.8 ± 3.1	2.34 ± 2.09	13.9 ± 1.2	1.25 ± 1.04	14.3 ± 2.2	.97 ± 1.16
6	11	.73 ± .47	18.0 ± 1.8	.80 ± .41	18.1 ± 2.1	7.45 ± 2.79	12.2 ± 1.1	1.66 ± .91	12.2 ± 1.1	.29 ± .23
7	14	3.08 ± 2.98	19.5 ± 2.2	2.76 ± 1.97	19.6 ± 2.4	2.49 ± 3.38	14.5 ± 1.6	4.07 ± 1.86	14.5 ± 1.2	5.30 ± 3.65

for each tap site; this also is not the case. (See Fig. 3: QUAD and HAM EMGs are at variable latencies following successive Achilles taps, despite consistency in onset latencies in TA EMGs.)

For these reasons, volume conduction of muscle signals is neither a sufficient nor a likely explanation of our data. However, the exclusive role of volume conduction in these data cannot be assessed, and it cannot be excluded as a contributing factor.

2. *Mechanical dispersion* of the stimulus to neighboring muscles (Myklebust et al., 1982): With each mechanical tap of a tendon, vibration is mechanically transmitted from the tap site through the rest of the limb. If this vibration is intense enough, it may stimulate the muscle spindles of all the muscles in the leg and produce a reflex response. Taps to a bony prominence, like the medial malleolus, should be much more effective at causing a vibration of the leg than taps to soft tissue, like a tendon. However, EMG signals from all limb muscles were always significantly smaller when we tapped the medial malleolus than when we tapped a tendon (Myklebust et al., 1986). While we cannot exclude the contribution of vibration of the limb in evoking muscle responses at recording sites other than from the agonist muscle, our data do not substantiate this mechanism as the primary contribution to the EMG signals recorded from distant electrodes.

3. A *neural mechanism* that includes segmental circuitry (see Fig. 1). While we cannot absolutely exclude the possibility that volume conduction and mechanical dispersion may contribute to the signals we record in the infant, it is unlikely that the widespread reflex EMGs recorded in these infants are explainable solely by these phenomena.

Unlike volume conduction and mechanical dispersion, the functional spinal cord circuitry that we call "reflex irradiation" is capable of explaining the reflex responses in all of the limb muscles following stretch to any one of those muscles. In this model of the infant's stretch reflex, we hypothesize that there exist multiple excitatory connections across one or more spinal levels, linking muscles at one or more joints. In addition, these pathways may have labile synaptic thresholds, and may be modulated by immature descending supraspinal signals.

Anatomically, it is not unreasonable to presume that there are simple reflex connections between muscles of the calf and the thigh (Eccles & Lundberg, 1958). The innervations of the gastrocnemius-soleus muscle (by the tibial nerve), the tibialis anterior (by the common peroneal nerve), and the hamstrings muscles (by the sciatic nerve) are all derived from the L4-5 and S1-2 spinal levels (and S3 for the tibial nerve). The quadriceps innervation (from the femoral nerve) is slightly higher, at L2-4. We suggest that intersegmental neural circuits may exist between lumbar segments in the infant, or the reflex transmission may be at the L4 level, which is common to all these limb muscles.

Functionally, it is also reasonable to expect that some interaction exists between calf and thigh muscles in the infant. For example, the gastrocnemius and hamstrings muscles are both knee flexors. That they can be activated simultaneously by a reflex pathway does not seem unlikely.

Furthermore, many of the infant's voluntary limb movements are performed in a synergy; that is hip, knee, and ankle flexion occur together in some kicking movements (Thelen, 1985). The gastrocnemius muscle is an ankle extensor, and the quadriceps muscle is a knee extensor. That gastrocnemius and quadriceps can be activated simultaneously in spontaneous kicking and by reflex tendon tap also seems reasonable. Thelen and Fisher (1983) also demonstrated that, in infants, kicks are often initiated by strong, phasic activation of hip and ankle flexor muscles, *and* by coactivation of antagonist muscle groups, including tibialis anterior and gastrocnemius muscles. The coactivation of antagonist muscles is a property of spontaneous leg movements, as well as reflex responses in the infant.

If reciprocal excitation and reflex irradiation accurately describe the functional spinal cord circuitry of the infant, the question remains about the changes that must occur in childhood for the development of the adult pattern of the stretch reflex pathway. Studies of development in animals have not reported changes in spinal cord circuitry that would explicitly support our hypotheses; however, reorganization of the connectivity in the cerebral cortex (Stanfield, 1984) and at the neuromuscular junction (Bagust, Lewis, & Westerman, 1973; O'Brien, Ostberg, & Vrbova, 1978) has been reported in

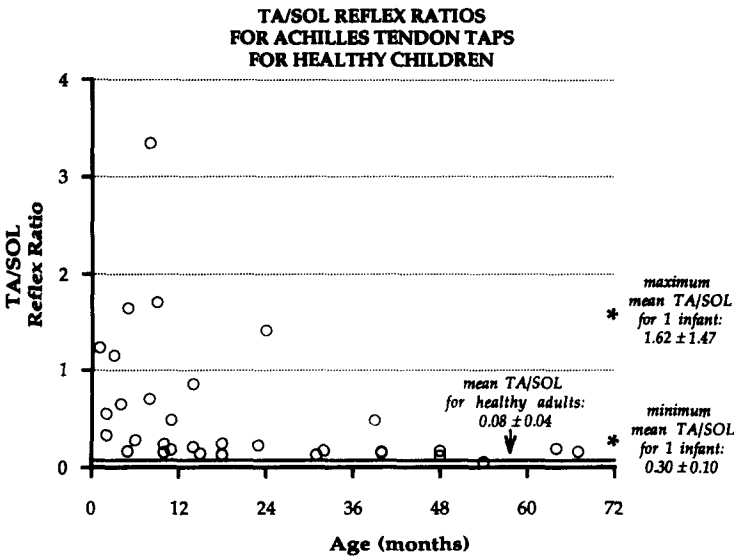

FIG. 5.—Average TA/SOL reflex ratios for Achilles tendon taps of 15 children, ages 1 to 67 months (adapted from Myklebust, 1990). For comparison, the mean TA/SOL reflex ratio for healthy adults and the maximum and minimum TA/SOL reflex ratios for individual infants are also illustrated.

the postnatal period of cats and rats. In addition, plasticity of the spinal cord via activation of previously ineffective synapses in adult decerebrate spinal rats has been proposed by Wall (1988).

While there are very few data on the reflex patterns in healthy children, preliminary studies of both lower and upper limbs (Myklebust, 1986; O'Sullivan et al., 1991) suggest that the pattern of EMG evoked by reflex irradiation is extinguished during the first year of life. This may be of significance in the control of the legs, as this is the time when children begin independent ambulation. Reflexes appear to become more focused with maturation, with suppression of reflex irradiation. Previous work from this laboratory (Fig. 5) shows that reciprocal excitation persists as an identifiable reflex pattern until 4 to 6 years, but the reflex ratios fall significantly during the first year.

These data about infants and children suggest that functional spinal circuitry may be modulated in concert with the development of motor coordination in the lower extremities. This "motor control" includes the ability of the child to make independent joint movements, and to execute coordinated tasks involving multiple joint segments of the limbs, as in walking. If the changes in spinal pathways in children are correlated in time and related to the development of limb motor control, then it is also possible that the neural pathways must mature before normal motor coordination is possible. Alternatively, it may be that the development of normal motor coordination *causes* the changes in spinal circuitry. Possibly, the failure to abolish functional reflex irradiation is contributory to the deficits of voluntary movement in cerebral palsy.

In any case, knowledge that stretch reflex patterns gradually change in early childhood makes it reasonable that the complex segmental circuitry of reciprocal excitation and reflex irradiation exists in the infant, and that these pathways are subjected to modulation during normal growth and development. The monitoring of these reflex patterns is not possible by clinical examination, but only by electrical recordings. Such monitoring should be considered, at least for children at risk for motor disorders.

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