Published in final edited form as:

Pediatr Phys Ther. 2013 ; 25(3): 278–289. doi:10.1097/PEP.0b013e31828dc3b6.

Muscle Activation Patterns in Infants with Myelomeningocele Stepping on a Treadmill

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Abstract

Purpose—To characterize how infants with myelomeningocele (MMC) activate lower limb muscles over the first year of life, without practice, while stepping on a motorized treadmill.

Methods—Twelve infants with MMC were tested longitudinally at 1, 6, 12 months.

Electromyography (EMG) was used to collect data from the tibialis anterior (TA), lateral gastrocnemius (LG), rectus femoris (RF), biceps femoris (BF).

Results—Across the first year, infants showed no EMG activity for ~50% of the stride cycle w/ poor rhythmicity and timing of muscles, when activated. Single muscle activation predominated; agonist-antagonist co-activation was low. Probability of individual muscle activity across the stride decreased w/age.

Conclusions—Infants with MMC show high variability in timing and duration of muscle activity, few complex combinations, and very little change over time.

Keywords

electromyography; female; gait; infant; male; motor skill; myelomeningocele; treadmill

INTRODUCTION

Myelomeningocele (MMC) is a common and debilitating form of spina bifida (SB), which is the most frequently occurring of all neural tube defects.¹ Typically, the defect occurs in the lumbar or sacral regions and leads to sensorimotor deficits at and below the spinal lesion. In the U.S., 1,500 to 2,000 babies are born each year with this disorder.² Most infants with MMC are delayed in learning to control their lower limbs sufficiently to acquire basic motor skills, such as sitting, crawling, and walking. For those with sacral level lesions, the likelihood of walking is 92–100%; as lesion level rises, this probability reduces to 38–62%

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Conflict of Interest: The author(s) declare no conflict of interest.

at the L3–L5 levels, and 4–29% for those with lesions at L2 and above.^{3–5} In children with lesions above the sacral level who learn to walk independently, sensorimotor deficits contribute to delays in walking onset and long-term gait difficulties.⁶ For example, the median age of walking onset is 3 years⁷ with concomitant impairments that often require the use of assistive devices to support and stabilize movement.

Multiple factors contribute to the delayed onset of locomotor skills for infants with MMC; an important contributor is their reduced level of spontaneous leg activity.^{8–11} Ulrich and Ulrich¹² reported that infants with typical development (TD) and those with Down syndrome (DS) who spontaneously kicked more often were, those who also learned to walk earlier. Recent data suggest that engaging infants with MMC in moving their legs in patterns of behavior that are functionally relevant, such as alternating kicking or stepping patterns, can help them learn to control their leg movements.¹³

One possible way to engage infants with MMC in moving their legs in functionally relevant patterns is by using partial bodyweight supported treadmill training. Ulrich and colleagues^{14–16} have shown that without being given any treadmill practice, infants with MMC respond with steps when supported in an upright posture on a pediatric treadmill during the first postnatal year. Further, these studies demonstrated the neuromotor adaptability available to infants with MMC early in life; infants changed the quality and frequency of stepping in response to experimenter-driven changes in belt speed and varied external sensory enhancements.^{14–16} Thus, these infants demonstrated that they have sufficient nervous and muscle system resources with which to construct and change their leg actions. What we do not know is how they activated their leg muscles in order to produce these step patterns, over developmental time.

Previous research has shed light on the emergence of underlying muscle activation patterns in the development of trunk and arm actions. Working with infants, Hadders-Algra et al.¹⁷ and Thelen and Spencer¹⁸ found correlations between movement direction and muscle activation when infants with TD were learning to sit independently and to reach for objects. However, they also found that latencies among activations of individual muscles and the frequency with which an individual muscle was activated during each movement cycle were highly variable. These variable onset times and durations of muscle activity settled into more stable and predictable activation patterns as functional practice increased. Similarly, Chang and colleagues^{19–20} observed high variability in the timing, frequency, and durations of muscle activations for toddlers with TD and toddlers with Down syndrome, as they began to walk independently. Several months of walking practice were required before muscle firing patterns became rhythmic, even though kinematic stability of the stride cycles resolved earlier.^{21–22} Over the first 7 months of walking, both toddlers with TD and DS show improvements in gait parameters that mimicked typical motor learning curves, that is, plots of change reflected a steep slope over the first few months, followed by slower and smaller improvements in skill.^{20–22} While we know the spinal sensory and motor nerves are compromised in the SB population, surprisingly little research has documented muscle activity during gait in children with this condition.

In fact, published reports of muscle activation patterns during ambulation in children with SB are nearly non-existent although ample publications report weakness in the lower extremities.^{23–24} Combined with the altered gait kinematics produced by children with SB, such as compensatory movements in the frontal and sagittal planes,^{6,25–26} one might logically expect differences at the level of muscle activation patterns, compared to healthy children. Park and colleagues²⁷ studied percent of time the rectus femoris, medial hamstrings, gluteus medius and maximus were “on” during barefoot walking in 4–17-year-old youth with sacral-level MMC and found increased activation durations for all muscles

compared to children that were healthy. This outcome may emerge from their need to compensate for inherent muscle weakness or instabilities caused by joint malformations. However, the population of persons with MMC is characterized by additional factors that are likely to contribute to the activation patterns that emerge over time, including diminished peripheral sensorimotor pathways, asymmetries in the degree of neuromuscular loss, varying degrees of scoliosis and kyphosis, as well as other skeletal and ligamentous impairments. These factors interact with each other over developmental time, forming potential negative cascading effects. Thus, to understand the developmental trajectory of muscle activation patterns during early, functionally relevant movements, we believe one meaningful approach is to characterize how infants produce stepping patterns very early in life, and over developmental time.

Recently, Teulier and colleagues²⁸ addressed this issue for infants with TD, following them from 1-month through 12-months of age. They examined infants' patterns of activation in 4 major muscles in the legs as they stepped in response to being supported upright on a small motorized treadmill. These researchers reported high variability in the combinations of muscles activated concurrently across stride cycles, with high incidence of all 4 muscles being active simultaneously initially, which reduced significantly with age. Analysis of the probability that a muscle was active at any one point in time across normalized stride cycles was low, consistently less than 50% for the tibialis anterior, rectus femoris, and biceps femoris, suggesting that the timing of muscle activity onset and termination was quite unstable and did not improve with age. The exception to this was the lateral gastrocnemius, which demonstrated probability values similar to those for experienced walkers at all ages. Overall, infants who were healthy produced a wide variety of muscle activation patterns that produced flexion and extension needed to create stepping. Within this variability, changes were also observed over time, suggesting components of adaptive control over the limbs were improving.

The purpose of this study was to characterize how babies with MMC produce steps, that is, activate their muscles over the first year, without direct practice, when supported upright on a motorized treadmill. Based on our previous reports of lower step frequencies in infants with MMC compared to their peers with TD across the first year¹⁶ and the neuromuscular and activity deficits characteristic of this population, we proposed that 1) the timing of the activation of all 4 core gait muscles would fail to show rhythmicity or consistent timing of onset and end of activation within strides; 2) infants with MMC would show a low level of muscle activation when stepping with high reliance on single muscle vs complex muscle combinations and significant portions of the stride when no muscles are active; 3) co-activation of agonist-antagonist pairs would be low; and 4) little change in patterns of muscle activation would be evident over the first year.

METHOD

Participants

Participants included 12 infants (6 female) born with MMC, 10 of whom participated in a larger longitudinal study of their responses to being supported upright on a motorized treadmill.¹⁶ Previously, we tested 12 infants with TD using the same method and conditions as described for infants with MMC. Results for the infants with TD were reported previously.²⁸ Inclusion criteria for all infants with MMC were: lumbar or sacral lesion level, no known disorders beyond those associated with MMC (e.g. hydrocephalus, Arnold Chiari malformation, etc.), gestational age ≥ 32 weeks (group $M = 36.88$ wks, $SD = 1.11$). Families were recruited by physician referrals from hospital clinics in southeast Michigan and northwestern Ohio.

Table 1 provides detailed profiles for each infant. Lesion groups were assigned based on the likelihood infants would become and remain able to ambulate in the community into adulthood.^{5,30} The lesion level groupings were as follows: low-level lesions (L5 and caudally), middle-level lesions (L4), and high-level lesions (L1–L3).

Procedures

Testing occurred in the Developmental Neuromotor Control Laboratory, School of Kinesiology, at the University of Michigan. When families arrived at the lab, we explained procedures and asked parents to sign a consent form approved by the university's Institutional Review Board. Parents also completed a medical status and history survey (including: shunt status, level of lesion, surgeries, status of associated conditions, and other medical complications). Infants were tested at 1, 3, 6, 9, and 12 months of age (corrected age). At each visit, families updated infants' medical status surveys.

To prepare babies for testing, we removed their clothing and diaper and placed retro-reflective markers bilaterally on their lower limbs. EMG data were collected by Therapeutics Unlimited EMG equipment (Therapeutics Unlimited, Inc. Iowa City, IA) (4 channels) at a sampling rate of 1200 Hz, synchronized by a manual trigger with the kinematic and video data recorded at 60 Hz. We placed pre-amplified bipolar surface EMG electrodes (rectangular patch 5×2.5 cm; inter-electrode distance: 0.63cm) over the muscle bellies of the lateral gastrocnemius (LG), tibialis anterior (TA), rectus femoris (RF), and biceps femoris (BF) muscles of the right leg for the first set of trials and moved to the left leg for the second set. To minimize wire movement and interference, a research assistant held the EMG cables above the treadmill surface during each trial. Additionally, EMG values were visualized in real-time both before each trial and while the infant moved, to check for noise in the EMG signal and to reduce the chance that cross talk was recorded.

We used a custom-made motorized treadmill (Carlin's Creations, Sturgis, MI), 18 cm × 42 cm × 82 cm (H,W,L) with a smooth belt surface (30 cm wide). The treadmill was placed on a large table with 3 Peak Motus™ (Peak Performance Technologies, Inc., Englewood, CO) cameras placed on each side of the treadmill to monitor joint marker positions. A digital color video camera (JVC, Yokohama, Japan; model: TK-C1380) was positioned to videotape leg movements for data capture verification purposes.

We held infants just below the axillae so they were upright with feet resting on the treadmill belt for twelve, 20-second trials. Trials were presented in 2 sets of 6 speeds. Trials 1 and 12 were baseline trials in which the treadmill belt was stationary. During trials 2 through 5 (Set 1) and 6 through 11 (Set 2) the treadmill belt speed was increased from .068 to .22 m/s, in increments of .038 m/s. Between sets, and as needed during testing, infants were given rest breaks. Rest time was imposed when transitioning from Set 1 to Set 2 to reposition EMG electrodes and during the test sessions if infants became fussy or had bowel or bladder discharge.

After treadmill testing, we measured infants' total body weight and length; leg length (greater trochanter to lateral malleolus), thigh length (greater trochanter to the lateral knee joint), thigh and shank circumferences. We also administered the motor subscale of the Bayley Scales of Infant Development II to assess concurrent levels of functional motor skill.³¹

The Data

This report includes patterns of muscle activity during alternating strides at 1, 6, and 12 months. Data from infants at 3 or 9 months did not differ from those shown at 1, 6, and 12 months. Selected stride parameters are summarized to characterize the overt quality of

infants' strides. For details regarding step frequency, step type, and leg behaviors produced when not stepping across the first year for these babies, please see Teulier et al.¹⁶ Because EMG was recorded from one leg at a time, we examined whether there was an effect of leg on the muscle activation characteristics. We tested the leg effect and leg by age interaction for each of our analyses and no effects were found for any of the muscles tested. Therefore, leg was removed as a factor in subsequent analyses.

Data Reduction

Stride Parameters—Toe off and touch-down within alternating steps were identified by trained behavior coders using the digital video data. Training required each coder to practice with training tapes and to obtain a coefficient of agreement of 0.85 (interobserver reliability coefficient, kappa) with data sets validated previously by experts in our lab. We normalized stride cycle durations to leg length to account for developmental differences in body size. Data were then transformed to a dimensionless variable with the following formula³²:

$$\text{normalized cycle duration} = \frac{\text{cycle duration}}{\text{square root}(\text{leg length}/\text{gravity})}$$

Coders also identified the part of the infant's foot that contacted the treadmill belt at touch-down and in mid-stance: toe, flat, heel, lateral, or medial. From this information, we calculated the percent of occurrence of each foot posture at touch-down and mid-stance. For each step we also coded infant's leg posture at mid-swing and mid-stance as high flexion (hip and knee flexed > 90° and foot approximately 4 inches or more off the treadmill belt) or low flexion (hip & knee < 90°).

EMG—The number of treadmill strides infants with MMC produce across the first 9 months is low and this is reduced when the entire stride (from toe off through end of stance) needed to occur within our 20 second test trials, therefore we used several procedures to extract strides to analyze. Our goal was to include a minimum of 1 and maximum of 4 strides for each infant. For a stride to be included the entire cycle had to be completed within the 20-sec trial and data for all 4 muscles had to be free from movement artifact and high levels of noise. We prioritized strides that occurred at the middle belt speed (.144m/s), because in our previous work we found this to be the optimal speed across the first year.¹⁶ We then selected strides occurring at .106m/s and .182m/s, and last, if needed .068m/s or .22m/s speeds were used.

Because some infants produced many strides and others produced only a few across all trials (or none in the case of EE at month 1), we only included a minimum of 1 to a maximum of 4 consecutive strides per infant per trial in our analyses to avoid overweighting those who took many steps compared to those who took few steps. For determination of which strides to include, we visually assessed all 4 EMG signals across the entire trial and determined which stride(s) had corresponding usable muscle activations. For those infants who took multiple consecutive strides, we chose the strides that occurred in the middle of the sequence for analyses to minimize the influence of initiation and deceleration on the EMG patterns for all muscles tested. Thus, for infants who took > 10 strides with analyzable EMG, we analyzed 4 strides; for 6–9 strides, we analyzed 3 strides; for 3–5 strides, we analyzed 2; and for those infants who took 1–2 strides, we analyzed 1 stride. After all levels of processing, we were able to analyze strides for 6 infants at 1 mo ($M/\text{infant} = 3$ strides); 7 infants at 6 mo ($M/\text{infant} = 3$ strides), and 10 infants at 12 mo ($M/\text{infant} = 3$ strides). Table 2 presents the number of strides each infant produced that were analyzable and the number of strides included in subsequent analyses (in parentheses) for each infant at each age. Of the 12

infants for which we analyzed EMG data, 10 were tested at all 3 ages, but only 4 had sufficient EMG data that was usable for our analyses at all 3 ages.

To process EMG data, we first applied a band-pass filter with cutoff frequencies set at 75 Hz and 300 Hz.³³ The low-end frequency removed electrical noise associated with wire sway and biological artifacts while the high cutoff eliminated extraneous tissue noise at the electrode site. Next, we rectified the data, then eliminated any high-frequency components added in the rectification procedure by using boxcar averaging with a window size of 7 samples. EMG data were subsequently converted to an on–off designation.

To determine when muscles were activated (on versus off) we used a decision-making process³³ to ensure that these choices were objective and reproducible. We began by using a 50-ms window moved frame by frame across each EMG trace. If the average EMG activity within a window exceeded a minimum noise threshold, the center value of that window was considered ‘on’. To determine the noise threshold, we computed frequency histograms of the amplitudes for each EMG signal for that trial. In addition, we normalized the frequency histograms to the modal amplitude for each trial. We chose to use infant’s modal amplitude because it was virtually impossible to accurately identify if or when infants produced maximum muscle activations. We used a cutoff value of 0.15 of the normalized modal frequency to differentiate EMG on–off activity³³ because we found it to be most sensitive for accurately identifying the onset and offset of muscle activation relative to the occurrence of an identified gait event. Finally, the duration of activity was summed across small segments of activity if the period of inactivity between segments was less than 50 ms.

We used 3 primary approaches to analyze muscle activation patterns. First we applied a muscle state analysis to identify for each frame in the stride cycle which muscles were concurrently active, that is, the “state” of EMG activity as the cycle unfolds.³³ Because we recorded EMG for 4 muscles, 16 combinations ranging from all ‘off’ to all ‘on’ were possible. In addition, this method allowed us to determine the duration and frequency of occurrence for each state, reflecting which muscle combinations appeared most often and for how much of the stride cycle. To compare infants both within and across age groups, the duration of EMG muscle activation was normalized relative to the total amount of time that muscle was ‘on’ (e.g. when all of its’ muscle activations across a stride cycle were summed).

Second, we analyzed for each individual muscle the likelihood that it was active at each point in time across the normalized stride cycle. We summed data for all strides for all infants at each age; thus, a probability value of 1 meant that the muscle was always ‘on’ at that point in the cycle for all infants. A probability of 0.5 meant that the muscle was ‘on’ at that point for 50% of the cycles produced by infants.

To put this in context, for a skilled performer, or when muscles activate with temporal rhythmicity over repeated cycles, the probability data when plotted, should show a pattern similar to the actual EMG traces; if firing is random, the plot should be relatively flat. We calculated probability percentages for each age by summing all ‘on’ values at each of the 100 time points across the cycle for all babies and dividing by the total number of cycles.

Third, we calculated the amount of co-activation between agonist-antagonist muscle pairs because previous research suggests that during nascent skill pattern production infants tend to co-contract agonist-antagonist pairs, which reduces as control improves. To do so we used the following formula:

$$\text{co-activation value} = 2 \times \frac{\text{number of frames when both muscle } A \text{ and muscle } B \text{ are on}}{\text{number of frames when muscle } A \text{ is on} + \text{number of frames when muscle } B \text{ is on}}$$

Note that this formula is derived from Winter,³⁴ but does not address amplitude of muscle activation, only its presence or absence.

Data Analyses

For our statistical analyses, we used PASW version 18.0.3 (IBM Corporation, Somers, NY). To account for grouping of cross-sectional and longitudinal data, because some infants were tested only once whereas others were tested at all ages, we used linear mixed models to test for main effects and interactions followed by Bonferroni corrections. Fixed effects included age (1, 6, 12 months) and phase (stance, swing) with participant as the repeated measure. We set statistical significance at $\alpha < .05$.

RESULTS

Stride Parameters—A full account of the number and types of strides taken each month by each infant and by group is published in Teulier et al.¹⁶ Briefly, the MMC group mean did not change significantly over ages 1–12 months, averaging 14.4 steps/minute (contrasted with infants with TD who performed 40.8 steps/minute) and of all steps produced, a significantly smaller proportion were alternating (~40% across all ages), compared to their peers with TD (~55% at 1-month, increasing to ~98% by 12 months). Although infants with MMC who had the highest lesion levels tended to produce the fewest strides, across other levels step frequency was not predicted by lesion level.

Table 3 indicates that although absolute stride cycle showed a trend toward increased duration with age [$F(2,101.2)=2.4$, $P=.10$], when normalized to leg length, values significantly decreased [$F(2,101.7)=4.7$, $P=.01$]. With age, infants spent proportionately more time in stance [$F(2,99.0)=6.4$, $P=.002$] from 59% at 1 month to 68% by 12 months. And, the frequency with which their legs were highly flexed in swing reduced significantly [$F(2, 65.9)=108.60$, $P<0.001$].

The part of the foot infants used to make initial contact at touch down varied among heel, toe, flat-footed, and the medial or lateral side of their foot. With age, frequency of heel contact, toe contact, and flat-footed contact increased while medial-lateral contact at touchdown significantly decreased [$F(2,10.2)=10.6$, $P=.003$] (see Table 3). Additionally, infants with MMC used 3 different foot contact postures in mid-stance: toe, flat-footed, and the medial or lateral side of their foot. Table 3 shows that flat-footed contact increased significantly with age [$F(2,9.8)=5.9$, $p=.02$]. As flat-footed contact posture increased, both toe and medial-lateral contact postures decreased, though neither was statistically significant.

Exemplar Muscle Activation Traces

Because infants' EMG activation traces during stepping look quite different from those commonly published for highly skilled walkers, and because the timing, duration, and amplitude of activation was highly inconsistent from 1 cycle to the next, we provide exemplars of smoothed and rectified EMG data for all 4 muscles from 2 infants at 1 and 12 months as well as example LG muscle activation traces during individual strides for 4 different infants at 1, 6, and 12 months of age (Figures 1 and 2, respectively). For Figure 1, we selected an infant with a high level lesion who might be expected to take few, if any,

alternating steps across ages and who had a lower probability, due to level of lesion, of showing muscle activity in all 4 of the monitored muscles. We selected a second infant with a low level lesion who might be expected to take multiple, consecutive, alternating steps and had a higher probability of showing activity in all muscles. Both sets of muscle traces show 2 strides (strides not consecutive for the infant with a high level lesion at month 1). In Figure 2, we show smoothed, rectified EMG traces for the LG muscle only in 4 infants with MMC of varying lesion levels. Both figures reveal the degree of variability demonstrated by individuals for assembly of the active and passive forces necessary to produce alternating steps; the active muscle contribution was sometimes high, sometimes low or nonexistent for any individual muscle. Further, illustrated in the figures is the lack of rhythmic activations.

Muscle Activations

Duration—Figure 3 suggests that the percent time each muscle was active tended to decrease with age in swing and to increase with age in stance, and that the majority of muscle activity occurred in stance. The main effect for phase reached significance for: TA [$F(1,229.0)=21.2$, $P<0.001$], LG [$F(1,227.4)=25.2$, $P<0.001$], and RF [$F(1,225.9)=21.2$, $P<0.001$].

Frequency—The average number of times each muscle was activated during a stride was generally well above 1, with very high standard deviations around the mean. The TA mean tended to decrease with age, LG to increase, with BF and RF showing negligible age trends. Only the TA decrease was statistically significant [$F(1,143.6)=3.2$, $P=0.04$].

Analyses of Muscle Activation Patterns

Sixteen muscle states—Figure 4 illustrates the percent of time during stride cycles, collapsed across infants, in which each combination of the 4 muscles were active or inactive. The most frequent muscle activation state for all 4 muscles, was activation of individual muscles in isolation. Periods of time during which 2, 3, or all 4 muscles were active occurred quite infrequently at all ages. High standard deviation bars suggest that the occurrence of all of these states was quite fluid and variable.

Co-activation muscle patterns—To address the question of whether agonist-antagonist muscle pairs showed the typical change from high coactivation early in the development of control that leads to reduced co-activation or reciprocal activation, we selected the 3 agonist-antagonist muscle pairs most commonly reported in gait research, TA+LG, LG+RF, and RF+BF. Figure 5 shows that percent of time within cycles that each muscle pair was co-activated was consistently low (20.86%) and did not change across ages.

Probability of Muscle Activity

Figure 6 presents, for each muscle at each age, across the normalized stride cycle, collapsed over all cycles analyzed for all babies, the probability or likelihood that the muscle was active. Surrounding the mean value trace for each age is the standard deviation. Superimposed is an indication of the average time within the normalized cycle that touchdown occurred.

Several points can be drawn from viewing the patterns that emerge from this treatment of the data. First, for all muscles, infants demonstrated a higher probability of activating their muscles at 1 month than either 6 or 12 months. This is most apparent for activation of the LG and RF, but both the TA and BF showed higher probabilities of being activated during the swing phase and at end stance when infants were 1-month old than at 6 and 12 months. Second, the probability of any muscle being active at any point in the stride cycle never

surpassed 50% and was quite flat by 6 and 12 months. The LG and RF showed tendencies for peak activations early in stance for the LG and in mid-swing for RF at 1 month, which seemed to diminish by 6 and 12 months. Similarly, at 1 month for the TA and BF, relative increases in probability of activation surrounded end of stance and toe off, but also reduced with age. Third, variability, as depicted in the standard deviation envelopes surrounding the mean traces, was high.

DISCUSSION

Our results describe how babies with MMC activate their muscles over the first year, without direct practice, when supported upright on a motorized treadmill support. Overall the results also support our predictions that their activation patterns for the 4 core gait muscles would show arrhythmic and inconsistent timing across strides and low levels of muscle activation, with minimal complexity compared to their peers with TD; and low levels of co-activation across agonist-antagonist pairs with little change in these muscle activation patterns over the first year.

Our data in which we focused on the relative consistency and rhythmicity of muscle activation patterns showed strong similarities to those reported previously for infants with TD.^{16–18} When Teulier et al.²⁸ studied infants with TD stepping on a treadmill across the first year, they, like Thelen & Spencer,¹⁸ used the same objective frame by frame approach as we did to determine when leg muscles were active or not active. Generally their results showed highly variable and arrhythmic muscle activity. The smoothed and rectified EMG traces for infants with MMC (Figures 1 & 2) reinforce that this is also true for infants with this neural tube defect as well. Infants used many variations when, and if, any individual muscle was activated as the other leg pattern formed a step. Thus, we could not generate ensemble averages without masking the inherent characteristics of how infants, in this context, accomplish sequential leg flexions and extensions.

Although the data did not lead to ensemble averages, we used a different qualitative analysis to examine the probability across time, that each muscle was active at each point in the normalized cycle; data were collapsed over strides and babies. Here the data showed greater divergence from those reported previously for infants with TD supported during treadmill stepping.²⁸ First, we found that the probability of any muscle being active at any point was always less than 50%, but disturbingly, these values were lower at 6 months and remained low at 12 months, suggesting perhaps some loss of activation potential.^{8–11} Studies of in-utero activity suggest that fetuses with MMC are as active as infants with TD^{35–37}; post-birth leg activity tends to reduce as babies deal with the antigravity demands on their muscles as well as other medical issues, including surgeries and medications. Second, infants with TD showed patterns of relatively clear peaks and valleys in their probability of activating their muscles, though the timing of these peaks and valleys usually did not match those produced by skilled walkers.^{19–20,28} The exception was the LG, which did parallel that seen during stable walking, across all months. But, our infants with MMC showed relatively flat probabilities across the cycle and across ages for all muscles, with the exception of LG and RF at 1 month. Given that visual observation of the overt behavior nevertheless was that the leg moved forward, touched down, and moved through stance, we suggest that infants were using multiple combinations of active muscle force and passive forces (gravity and motion-dependent torques) in response to the treadmill context. Infants with TD have about 30 leg and foot muscles available to create action. Infants with MMC may have a diminished number of motor units and even loss of function of individual muscles. Clearly, they demonstrated the flexibility to use, in sum, multiple force combinations in order to step.

The flexibility displayed by infants with MMC in using these 4 core gait muscles was more simplistic than that of infants with TD. Our muscle state analysis showed that of the 16 possible combinations of muscles that could be achieved at any point in the stride cycle, no muscle activity and single muscle activations dominated. Babies with TD showed an opposite response.²⁸ For the latter babies, in stance all muscles dominated among potential muscle states, reducing with age to other combinations of muscles; in swing, muscle activity did not increase with age. Infants with MMC showed relatively low levels of co-contraction for agonist-antagonist pairs that changed little over the first year of life post birth.

Infants with TD showed higher levels of co-contraction at month 1 that reduced significantly with age.²⁸ The infants in Teulier et al.'s studies^{16,28} did not practice treadmill stepping and, at 6-months of age were not spontaneously practicing stepping or even pulling to a stand. This observed pattern of reducing the amount of co-activation of agonist-antagonist pairs over time, as control over muscles improves has been reported previously. For example, Chang et al.^{19–20} showed that infants with TD and DS reduced co-activation of gait muscles as their months of walking independently increased. What seems evident here is that for infants with MMC, improvements in their level of control over their core gait muscles is, at least, significantly delayed. They also seem to show unique solutions to the “problem” of responding to this context and activating their leg muscles. From less complexity and very simple activation responses, they may need to learn to bring into play more muscle combinations in order to establish muscles working together in synergies.

Infants with MMC did show some areas of improvement with age, particularly in stride parameters. Like healthy babies, their leg postures shifted dramatically from the more highly flexed “fetal” postures of neonates to more extensor dominance, at least by 6 months with more improvements by 12 months. Foot postures at touchdown shifted from the medial or lateral side of the foot to dominantly flat or toe contact; during mid-stance medial-lateral postures also decreased and flat-footed dominance emerged by 6 months of age. Taken together, the decreased variability in foot placement and position at touchdown and midstance in combination with the increased overall duration of muscle activity during stance and “off” time during swing suggests some improvement in the ability to adapt to the context in which we placed them. Perhaps this reflects gradual improvement in the function of their sensorimotor systems - the ability to perceive sensory input and respond with some change in their motor output.

Additional improvements were evident in that muscle activation durations increased with age. One might be tempted to view this as a negative because muscle activation durations have been shown to reduce with age for babies with TD.²⁸ However, we would argue, an increase in duration for infants with MMC may be a positive adaptation to their decreased inherent capacity to stabilize their posture in upright. Previously, Chang et al.²⁰ showed that toddlers with DS who were learning to walk, despite months of experience, had increased activation durations of these same 4 muscles, but toddlers with TD showed decreased activation durations. Similarly, Park and colleagues²⁷ showed that children with MMC showed increased activation durations for the RF, BF, gluteus maximus and gluteus minimus when walking, compared to children with TD. In both cases, researchers hypothesized this to be an adaptation they acquired to meet their increased needs to stabilize their bodies in upright motion. Given that infants with MMC also showed a significant increase with age in the proportion of time spent in stance, this makes sense. Infants with MMC are making small adaptations, improvements, to their responses when supported upright on a treadmill and stepping, but very slow and very small changes.

The clinical implications from our EMG work with this population must be viewed as in its early stages. This study contributes an understanding of muscle activation patterns that underlie the more overt characterizations of treadmill stepping in infants with MMC that have been published previously.^{14–16} By objectively examining the underlying muscle activation used by infants with MMC to produce stepping behavior across the first year, we provide a foundation from which to better understand the later development of control, characteristic muscle weaknesses, and atypical movement patterns shown by children and adolescents with MMC.^{6,23–27} It is imperative for researchers and clinicians to understand the “starting point” for muscle activity as this contributes to infants’ responses to movement context and eventual improvements or potentially negative cascading effects with regard to their development. Greater understanding and appreciation of these early muscle activation patterns will facilitate design and implementation of effective, earlier interventions that will promote improved and long-term functional mobility.

Our data continue to suggest that lesion level alone will not predict behavioral or neuro-physiological step responsiveness in infants with MMC, other than for infants with the highest level lesions. Our finding that infants with MMC had a higher probability of activating muscles at 1 month than at 6 or 12 months supports our ongoing work to identify an optimal approach for an early and relevant intervention to sustain and enhance residual motor function in these infants. Taken together, the decreased variability in foot placement and position at touchdown and midstance in combination with the increased duration of muscle activity during stance, support that, although muscle activation may decrease with age, these infants do become less passive in producing or controlling steps, particularly in stance. This speaks to an early problem-solving capacity in these infants that has not historically been a in consideration in specifying optimal timing and content of early motor intervention for this population. We propose that subsequent studies examine a practice model of treadmill stepping with these infants, wherein this early problem-solving ability would theoretically interact with experience. The data reported here suggest that both recovery (enhanced activation) and compensation (more efficient combinations of muscle activation and passive forces) are likely mechanisms of change available to these infants, even at the youngest ages. Determining the best approach to tap this potential continues to drive our work.

Limitations

The primary limitation of our study is the small sample size. Also, we only measured surface EMG activity for 4 muscles. However, we chose the 4 leg muscles (2 proximal and 2 distal) that are most commonly cited in the gait literature as being especially relevant to the acquisition and production of stepping and walking. The number of strides for which our EMG data were analyzable varied by baby, but overall was small compared to studies involving infants with TD. We attempted to control for this by including no more than 4 strides per infant. Despite these limitations, we suggest that the neuromotor control issues faced by these infants, while challenging to address in infancy, have direct implications for their early development of lower limb movement and control.

Conclusions

Infants with MMC show highly variable timing of the activation of muscles during treadmill stepping across the first year, similar to that seen in infants with TD. Their overall level of muscle activation, particularly during stance, is low, more often relying on passive forces and single muscle activations, whereas infants with TD use more complex muscle combinations. And, although the overt quality of foot contact patterns and duration of muscle activations show improvement with age, the underlying muscle patterns produced by these infants when stepping show minimal improvement and even some decrement (e.g., in

probability of activation) across these ages. We encourage future research that tests options for early interventions that may enhance muscle activation and quality during this period.

Acknowledgments

We would like to thank the participants and their families for taking part in this research study as well as the physicians and staff at the Myelomeningocele clinics in Northwest Ohio, Detroit Medical Center, and University of Michigan Pediatric Neurosurgery Clinic for their help in recruiting.

Grant support: Funding for this study was provided by NIH NICHD R01 HD047567 awarded to Beverly D. Ulrich. Ms Sansom was supported by grant H424C010067 from the U.S. Office of Special Education and Rehabilitative Services awarded to Dale Ulrich.

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Figure 1.

Exemplar traces of smoothed, rectified EMG for 2 infants, with high and low-level lesions. Traces contain 2 stride cycles for each infant at 1 and 12 months, and show the behavior of all 4 muscles as their legs demonstrated stepping behaviors.

Figure 2. Smoothed rectified LG EMG traces for single stride cycles, normalized to 100%, for 4 infants, at each age. Treadmill belt speed was 0.144 m/s for all steps represented here.

Figure 3.

Percent of time within the stance and swing phase that muscles were active, as a function of age. * Represents significant difference between phases ($P < .05$).

Figure 4.

Distribution of the occurrence of each of the 16 possible muscle activation states, as a function of phase and age. The figure is organized such that moving from left to right across the figure, the number of muscles active concurrently increases. Mean values for infants with MMC, in both stance and swing, reduce in this direction as well. Starting on the left side, no muscle activity (NO) occurred approximately half of the time within stance and swing. Y-axis maximum value is abbreviated due to the high standard deviation values for no muscle activity (NO). Mean and SD values for NO are reported next to each bar. Dashed grey line separates data for NO and single muscle states from combinations of multiple muscles. * Represents significant difference ($P < 0.05$) between stance and swing phases of the stride cycle.

Figure 5. Percent of stride cycle during which agonist-antagonist muscle pairs were simultaneously co-activated, as a function of age.

Figure 6.
Probability means and standard deviation values for each muscle, at each age.

Table 1

Participant profiles

Participant	Gestation Age (wks)	Gender	Lesion Level*	Total Bayley 1 mo	Total Bayley 12 mo	Surgery	Hydro-cephalus	Shunt	Arnold Chiari	Club Feet
AA	34	M	L2/L3	4	35	IU	Y	Y	N	N
BB**	36	F	L4/L5	17 (6 m)**	55	IU	Y	Y	N	N
CC	36.5	F	L5/S1	7	44	EU	Y	Y	N	N
DD	36.5	M	S1/S3	8	55	EU	N	N	N	N
EE	37	F	L1/L2	7	49	EU	Y	Y	Y	N
FF	37	M	L4	6	47	IU	Y	Y	Y	Y
GG	37	F	L4	7	50	IU	Y	Y	Y	Y
HH	37	F	S1	6	53	EU	N	N	N	N
II	37.5	M	L5/S1	7	51	EU	N	N	N	N
JJ	38	M	L3	6	44	EU	Y	Y	Y	Y
KK	38	F	L4/L5	8	59	EU	N	N	Y	N
LL**	38	M	L5	8	U	EU	U	U	Y	N

* Level at which surgical repair was performed.

** Not enrolled in the longitudinal study (BB tested at 6 and 12 m, LL tested at 1 m).

IU=Intra-uterine repair.

EU= Extra-uterine repair.

U=Data not available.

Table 2

Number of strides taken by each infant that were analyzable. In parentheses: Number of strides (1–4) utilized for group Electromyography (EMG) analyses.

Participant	1 m (n=6)	6 m (n=7)	12 m (n=10)
AA	4/(2)	U*	U*
BB	U*	U*	19/(4)
CC	5/(2)	12/(4)	2/(1)
DD	U*	U*	19/(4)
EE	0	2/(1)	3/(2)
FF	U*	6/(3)	38/(4)
GG	U*	U*	13/(4)
HH	12/(4)	8/(3)	9/(3)
II	14/(4)	18/(4)	25/(4)
JJ	U*	3/(2)	2/(1)
KK	15/(4)	39/(4)	66/(4)
LL	3/(2)	D*	D*
Mean	8.8/(3)	12.6/(3)	19.6/(3)

D*: Participant discontinued participation.

U*: EMG data not usable due to technical problems.

Table 3

Description and statistics for stride parameters, by age

	1 mo	6 mo	12 mo
	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>
Cycle duration (seconds)	1.9 (1.1)	2.1 (0.9)	2.1 (0.9)
Normalized Cycle duration	13.8 (7.7) *	13.1 (5.6) *	12.1 (5.3)
Stance phase (% cycle)	59.1 (16.2) ***	68.5 (12.1) *	68.5 (12.0) **
Percent Leg Flexed in Swing (%)	95.9 (8.8) ***	54.4 (35.1) ***	38.0 (22.9) ****
Foot Contact at Touch Down (%)			
Heel	14.4 (13.9)	19.3 (20.9)	19.5 (11.9)
Flat	22.9 (19.7)	25.8 (21.9)	34.1 (21.4)
Toe	20.9 (39.9)	34.0 (27.1)	40.5 (27.9)
Medial/Lateral	41.9 (22.7) ***	20.9 (26.6) *	6.9 (12.1) **
Foot Posture during Midstance (%)			
Flat	21.6 (22.1) ***	50.0 (33.6) *	50.9 (39.6) **
Toe	50.1 (40.4)	42.5 (37.7)	35.2 (32.2)
Medial/Lateral	27.7 (26.0)	6.5 (11.9)	13.8 (27.2)

* Significant difference between 1 and 6 months ($P < .05$).** Significant difference between 1 and 12 months ($P < .05$).*** Significant difference between 6 and 12 months ($P < .05$).

Table 4

Description of stride parameters, by lesion level and age

Lesion		1 mo	6 mo	12 mo
		M(SD)	M(SD)	M(SD)
Normalized Cycle Duration	High	20.4 (8.4)	11.2 (3.0)	15.3 (4.8)
	Middle	14.4 (5.3)	11.7 (4.2)	13.3 (5.4)
	Low	13.6 (4.4)	12.1 (5.3)	13.1 (5.9)
Stance Phase (%)	High	58.7 (12.8)	61.0 (9.0)	61.3 (14.8)
	Middle	65.1 (12.6)	67.8 (8.2)	66.5 (10.3)
	Low	58.7 (15.6)	69.0 (9.9)	69.8 (13.0)
Leg Flexion (%)	High	58.1 (43.4)	57.9 (43.8)	49.0 (32.9)
	Middle	94.4 (10.4)	66.9 (23.9)	37.7 (26.1)
	Low	96.6 (8.4)	58.7 (35.6)	41.1 (18.1)