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Author Response to Invited Commentary by Heathcock [Commentary for: Gastrocnemius/soleus Muscle Tendon Unit Changes Over the First 12 Weeks of Adjusted Age in Infants Born Preterm]

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Comments
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Author Response

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We thank Heathcock for taking the time to write her commentary[1] regarding our study.[2] Her comments are interesting, and our subsequent investigation into these ideas has led us to uncover some enlightening evidence in relation to the topics raised in the commentary. We will attempt to review each of the topics raised and the evidence associated with these topics.

Measurement of Gastrocnemius-Soleus Muscle Tendon Unit

Heathcock[1] suggests that "the measurements taken in this study were largely of the soleus muscle" secondary to knee contractures. There are 3 important issues to consider when contemplating this perspective. First, we measured infants born full term and infants born preterm over 3 different ages. In the infant born full term at the newborn measurement, knee extension is limited. During data collection, we were careful to ensure that we extended the knee as much as possible, but in the newborn infant born full term, the knee has a small flexion contracture that limits full extension. Although this muscle may not have been fully lengthened, it was lengthened as much as possible. This limitation to extension was not an issue at 6 or 12 weeks of age in the infants born full term or in the infants born preterm at any age.

Second, we do not know which structures are limiting knee extension. If the gastrocnemius muscle was the structure limiting knee extension, then by extending the knee fully, we lengthened the muscle. Based on research by Brown and Swenson,[3] the difference in ankle dorsiflexion in infants born full term at newborn age with the knee flexed to 90 degrees versus fully extended was 3 degrees in boys and 2 degrees in girls. This difference in ankle dorsiflexion with full extension and 90 degrees of knee flexion is small, and changes observed at the ankle with the small knee flexion we had present in our participants should have been even smaller than that found with 90 degrees of knee flexion and full extension, possibly too small to reliably detect via a goniometric measurement.

Third, we were measuring the gastrocnemius-soleus muscle tendon unit. During measurement, palpation occurred at the Achilles tendon, which is composed of the tendinous attachment of both muscles. Distinction between the gastrocnemius and soleus muscles cannot be made in the Achilles tendon, and shortening in either muscle would likely result in changes of the Achilles tendon. Measuring the gastrocnemius-soleus muscle tendon unit gives us information about available ankle motion for function. Clinically, this is a reasonable and accepted measurement. Matjačić et al[4] used a device to artificially create shortening in the gastrocnemius muscle, in the soleus muscle, and in both muscles. The results of their study suggest that during function, plantar flexion at the ankle would be limited by shortening either the gastrocnemius muscle or the soleus muscle or by shortening both muscles, and all 3 of these scenarios would result in toe-walking.

Although the proximal insertions of the gastrocnemius and soleus muscles produce different effects on trunk support, acceleration and deceleration of the trunk and leg during gait,[5] there is significant evidence to suggest that both muscles are implicated in toe-walking.[6–11] Both muscles have been found via electromyographic (EMG) analysis to activate earlier in the gait cycle and remain active for more of the gait cycle during toe-walking.[6–11] During computer-simulated EMG recreations of 10 adults during toe-walking, increased activity of the soleus and gastrocnemius muscles was observed, with the soleus muscle contributing the most to both the vertical and horizontal ground reaction forces in early stance.[10,11] The notion that the soleus muscle does not contribute to toe-walking is not supported by the evidence.

Splinting

Heathcock[1] accurately reports that we suggest splinting. Our suggestion of a splint "that limits plantar flexion and allows active dorsiflexion"[2(p146)] would allow the infant to kick and move in all directions, with the exception of limiting plantar flexion. The splint we suggested would not limit kicking, cause immobilization, or confine the infant's kicking in any direction except extreme plantar flexion at the ankle.

We would like to restate our ideas on timing of the splinting intervention. The most important reason we suggest early intervention is based on animal studies that demonstrated differences in a muscle's response to lengthening in young animals.[12,13] Muscle tendon unit measurements in the full-term infant mimic the response documented in very young infants.[2] If intervention was delayed, the outcome might not be the same, whereas having intervention occur prior to term age matches the timing of the lengthening that occurs in the infant born full term. Physical therapy intervention to lengthen the gastrocnemius-soleus muscle tendon unit immediately following preterm birth also would be more likely to alter movement patterns that may be developing during this period and encourage patterns similar to those of the infant born full term by increasing the similarities of the biomechanical system. Lastly, for practical reasons, this is when the infants may be available for intervention when they are in the neonatal intensive care unit (NICU).

Of course, the infant born preterm is, following birth, external to the uterine environment, and we cannot at this point exactly mimic the uterine environment in the NICU. We can try, however, to decrease influences of this external uterine environment by positioning and providing postural support. Many NICU therapists position infants in flexed positions to physically improve stability and promote self-regulation. These interventions have been the topic of many research articles over the years. Clinically, when we find postural changes in the infant born preterm, we traditionally have attempted to decrease the environmental influences that may result in these postural changes. Our interventions might not perfectly recreate the uterine environment, but they are designed to improve NICU outcomes.

In addition to positioning changes, Heathcock[1] suggests that "infants—especially preterm infants—kick a lot." There is very limited evidence documenting the frequency of kicking in infants born preterm prior to term age. Droit et al[14] examined kicking in infants at low risk for developmental issues who were born preterm prior to term age. They reported (1) a mean of 1.33 bouts of kicking per hour, with an average of 6.5 kicks per bout, at 31 to 35 weeks gestation and (2) a mean of 1.83 bouts of kicking per hour, with an average of 7.4 kicks per bout, at 37 to 39 weeks gestation. Kicking frequency documented at and following term age in the infant born preterm increases to a mean of 3.7 to 28.6 kicks per minute (versus hours as measured prior to term) from term to 4 months after term age.[15–18] None of the research examined kicking over days or weeks; therefore, we have no evidence concerning what an average kicking rate would be over a day or week. Research suggests that kicking frequency changes with arousal level, and when infants are fussing and crying, the frequency of kicking increases.[19] Based on the definition of infant state 1, deep sleep, kicking would not be observed in this state. If the infants born preterm are physiologically stressed, kicking may be less than when the physiological system is stable. Despite the frequency of kicking, the splint we suggested would not limit kicking and would support the infants learning a lower-extremity pattern without the extremes in plantar flexion, similar to the movement of infants born full term.

Heathcock[1] suggests that splinting the ankle would not affect the length of the gastrocnemius muscle because it is a 2-joint muscle. We would like to raise 3 issues regarding this statement. First, changes in ankle dorsiflexion have been documented in children after short leg casting (below the knee).[8,20–23] Some studies documented functional improvements that accompanied the increases in dorsiflexion.[8,22] Second, movement in utero, during the last few weeks of gestation, would most likely not include full knee extension, because the infant would not have enough room to fully extend the knee. Splinting just the ankle would mimic the plantar-flexor lengthening in utero found in infants born full term. Third, infants born preterm are generally more extended. If the infant born preterm has a splint on, which limits plantar flexion, and the knee is generally in a position of more extension, then both ends of the gastrocnemius muscle would be stretched and lengthening of this muscle would be likely. Based on these points, we hypothesize that limiting plantar flexion at the ankle may very well be an effective intervention.

Multiple Births Versus Singleton Birth

We agree with Heathcock[1] that there may be a difference in uterine movement between single and multiple gestations. Multiple gestations may decrease available space for infant movement. Eleven of the 22 infants included in our preterm group were from multiple-gestation pregnancies. It is possible that the infants from multiple births may encounter more cramping earlier in gestation and could have experienced gastrocnemius-soleus muscle tendon unit lengthening as a result of crowding prior to birth. This would have resulted in the preterm, multiple-gestation infants looking more like the full-term group. Significant differences between our 2 groups were found when the multiple-gestation preterm infants were included in the preterm group. If you examine Figure 3 of our article[2], there is a distinct difference between the preterm and full-term groups, which is supported by our statistical analysis.

Two additional points regarding inclusion of infants from multiple-gestation births in research are worth considering. First, there has been a steady increase in multiple births,[24] and the addition of infants from multiple births represented the population of the NICU where we recruited infants. If ankle range and movement is an issue in these children, we as physical therapists should be identifying and addressing it. Second, historical precedence can be found to include multiple-gestation preterm infants in preterm groups. Heathcock et al[18] included 9 multiple-gestation infants out of 10 preterm infants when investigating differences between full-term and preterm groups.

In light of some of the concerns Heathcock[1] raises, we would like to review briefly a study by Griffin et al,[8] who examined children who did and did not idiopathically toe-walk and recorded muscle activations via EMG during gait. The children who toe-walked were placed in short leg casts for a minimum of 6 weeks. Immediately following intervention, these children demonstrated EMG gastrocnemius and soleus muscle activations similar to those of the children who did not toe-walk. The children also demonstrated positive changes in their ability to heel-strike, which continued. It appears, based on this study, that a change in muscle length has potential to alter use of the muscle during function and, in this case, ankle movement during gait.

Our results are consistent with the findings of numerous studies on infant kicking and have the potential to shed light on ankle movement during gait and possibly toe-walking. In addition, this study confirms our previous findings. Although there is still much work to do in this area, we hope that our investigation provides a positive step in the process of understanding the biomechanics of the infant's moving system.

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