

Oral presentation

Open Access

## Muscle echogenicity is increased in fetuses with spina bifida aperta

Renate J Verbeek<sup>1</sup>, Johannes H vd Hoeven<sup>2</sup>, Natasha M Maurits<sup>2</sup>,  
Krystyna M Sollie<sup>3</sup>, Arend F Bos<sup>4</sup>, Oebele F Brouwer<sup>2</sup>, Wilfred F den Dunnen<sup>4</sup>  
and Deborah A Sival\*<sup>1,4</sup>

Address: <sup>1</sup>Pediatric Neurology, University of Groningen, University Medical Center, Groningen, The Netherlands, <sup>2</sup>Neurology, University of Groningen, University Medical Center, Groningen, The Netherlands, <sup>3</sup>Obstetrics, University of Groningen, University Medical Center, Groningen, The Netherlands and <sup>4</sup>Pediatrics, University of Groningen, University Medical Center, Groningen, The Netherlands

Email: Deborah A Sival\* - d.a.sival@bkk.umcg.nl

\* Corresponding author

from 51<sup>st</sup> Annual Meeting of the Society for Research into Hydrocephalus and Spina Bifida  
Heidelberg, Germany. 27–30 June 2007

Published: 20 December 2007

*Cerebrospinal Fluid Research* 2007, **4**(Suppl 1):S11 doi:10.1186/1743-8454-4-S1-S11

This abstract is available from: <http://www.cerebrospinalfluidresearch.com/content/4/S1/S11>

© 2007 Verbeek et al; licensee BioMed Central Ltd.

### Background

In spina bifida aperta (SBA), movements caudal to the meningocele (MMC) are present in utero, but they disappear shortly after birth. Insight in onset and progression of movement loss is therapeutically relevant. Determination of muscle echogenicity (or density (MD)) is used to estimate onset and progression of various neuromuscular disorders in children. In prenatal SBA, we hypothesized that MD assessment could indicate onset and progression of muscle damage in relation to movement loss. Objective: To estimate onset and progression of muscle changes in fetal SBA by MD assessment.

### Materials and methods

MD (muscle/bone pixel-density) was obtained in calibrated images (by standardized ultrasound methods). In SBA (n = 6; GA 22–37 wks) and control fetuses (n = 11; GA 17–36 wks), we assessed MD in gluteus/gastrocnemius (L5-S1), tibialis ant. (L4-5), quadriceps (L2-4), and biceps/triceps (C5-8) muscles. MMCs were at Th12 (n = 1), L4-5 (n = 1) or L5-S1 (n = 4) level. In SBA fetuses, MD was compared between muscles from myotomes caudal and cranial to the MMC. Additionally, MD was compared between SBA fetuses and aged matched controls (n = 6 and median GA 33 wks in both groups), and between pre- and postnatal SBA (<1 week after birth). In succumbed

fetuses, MD values were related to histological observations in affected muscles (3 SBA fetuses).

### Results

During MD assessment, movements caudal to the MMC were present in all SBA fetuses. MD values correlated with gestational age, both in controls and in SBA myotomes cranial to the MMC (r = 0.44, p < 0.01; r = 0.50, p < 0.05; resp.). In contrast, MD values in SBA myotomes caudal to the MMC did not correlate with gestational age (r = 0.26, p = 0.13), and were significantly higher than cranial to the MMC (median +19%, p < 0.01) and controls (+45–60%, p < 0.01). Longitudinal pre- and postnatal MD values caudal to the MMC did not significantly differ (mean 14%). MD values in SBA myotomes caudal to the MMC corresponded with histological assessment (extent of atrophic/hypertrophic muscle fibres).

### Conclusion

Despite persisting leg movements in fetal SBA, MD in myotomes caudal to the MMC is increased prenatally. Early postnatal disappearance of leg movements is unrelated to an additional increase in perinatal MD. Present data support the concept that permanent movement loss is related to acute spinal damage rather than to intrinsic muscle impairment.