Compound muscle action potentials in newborn infants with spina bifida

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The aim of this study was to investigate the relationship between compound muscle action potentials (CMAPs) and neurological impairment in newborn infants with spina bifida. Thirty-one newborn infants (17 males, 14 females, mean gestational age 39wks [SD 2]; mean birthweight 3336g [SD 496]) with spina bifida were investigated at a median age of 2 days (range 1–18d). Motor and sensory impairment and muscle stretch reflexes were assessed and neuroimaging was performed. CMAPs were recorded from the tibialis anterior muscle and the gastrocnemius muscle after percutaneous electrical nerve stimulation. CMAPs were obtained in almost all infants. The area under the curve of the CMAP (CMAP-area) was associated with motor and sensory impairment and with the presence of muscle stretch reflexes, but not with the morphological level of the spinal anomaly. These associations were stronger for the gastrocnemius muscle than for the tibialis anterior muscle. In conclusion, the CMAP-area correlates with neurological impairment in neonatal spina bifida and provides an estimate of residual motor neuron function in affected spinal segments. The assessment of CMAPs after percutaneous electrical nerve stimulation is recommended as an additional instrument to the clinical neurological examination and imaging studies.

See end of paper for list of abbreviations.

Spina bifida is a congenital malformation of the nervous system, which usually results in severe disabilities.[1–4] These disabilities mainly depend on neonatal neurological impairment, especially on sensory impairment.[5] Traditionally, neurological impairment is assessed by clinical examination, but the clinical neurological examination of a newborn infant with spina bifida may be complex and, to a certain extent, subjective. Potentially confounding factors are inconsistencies between patterns of muscle activity and neurosegmental innervation,[6] the distinction between normal leg movements and purely reflex leg movements,[7] and changing movement patterns in the first week of life.[8] In the past few years, neuroimaging is performed in most centres as well, but the morphological level of the spinal anomaly is only partly related to the neurological impairment.[1,8,9] An additional instrument that provides objective information about neurological impairment is desirable and may improve preoperative clinical decision-making in newborn infants with spina bifida.

Motor nerve conduction study may be an appropriate additional diagnostic instrument as it provides a diagnostic guide in several disorders.[10] Previously, we reported on the presence of compound muscle action potentials (CMAPs) in leg muscles after percutaneous electrical nerve stimulation in almost all newborn infants with spina bifida.[11] Therefore, the presence of a CMAP as such is of no diagnostic use, but the magnitude of the CMAP, which reflects the number and size of the activated motor units, may be of diagnostic value.

The aim of the present study was to investigate the association between the magnitude of the CMAP, as represented by the area under the curve (CMAP-area), and neurological impairment in newborn infants with spina bifida considering a potential diagnostic value of the CMAP. We hypothesized that a larger CMAP-area is associated with less neurological impairment. The clinical value, methodological aspects, and pathophysiological considerations are discussed.

Method

PARTICIPANTS

Thirty-one newborn infants (17 males, 14 females) with spina bifida born at or referred to the Radboud University Nijmegen Medical Centre were enrolled in the study. Fourteen of these children were diagnosed antenatally. Most infants were born at term (mean gestational age [GA] 39wks [SD 2]) and had a birthweight appropriate for GA. The mean birthweight was 3336g (SD 496) with a SD score to the population norm of 0.9. The mean head circumference was 35.6cm [SD 2.8] with a SD score to the population norm of −0.3. At the time of investigation, the perinatal period was uneventful for all infants. The Regional Committee on Research involving Human Subjects approved the study protocol. Informed consent was obtained from all parents.

CLINICAL ASSESSMENT

The clinical assessment was performed before surgical closure of the spinal anomaly and was based on repeated physical examinations, and brain and spinal cord MRI within 72 hours after birth. Motor impairment was assessed on each side separately and scored according to the lowest spinal segment with lasting non-stereotypical, non-reflex leg movements. Where motor impairment was thoracic, we did not attempt to assign it to a single spinal segment, because we considered this as too inaccurate. Sensory impairment was assessed on each side

separately and scored according to the lowest dermatome with a behavioural reaction to pin prick. Muscle stretch reflexes were scored as present or absent. On MRI the spinal anomaly was classified according to Tortori-Donati et al.[12] and its morphological level and its size was described by identifying the cranial margin of the spinal anomaly with the corresponding vertebra. Cerebral comorbidity was assessed by the presence or absence of hydrocephalus, Chiari II malformation, and corpus callosum dysgenesis.

NEUROPHYSIOLOGICAL ASSESSMENT

The neurophysiological assessment took place at a median age of 2 days (range 1–18d) before surgical closure of the spinal anomaly. The same assessor performed the procedure in all infants. CMAPs were obtained from the tibialis anterior and the gastrocnemius muscle by supramaximal percutaneous electrical stimulation of the peroneal and the posterior tibial nerve respectively, at the popliteal fossa. CMAPs were recorded using surface electrodes (tendon-belly montage) and an Oxford Synergy electromyograph (Oxford Instruments, Old Woking, Surrey, UK; band-pass filter 20Hz and 3kHz, amplifier range 100mV, and display sensitivity of 0.5mV/division). The latency was measured from the stimulus artefact to the onset of the first negative deflection of the CMAP. The area under curve of the first negative wave was calculated as a measure of the magnitude of the CMAP (Fig. 1).

Measurements of the gastrocnemius muscle were obtained in only 18 newborn infants as it was added to the protocol later during the investigation.

ANALYSIS

As the gastrocnemius muscle was added to the protocol later, two subgroups were present in our study. Possible differences in clinical impairment and CMAP measurements between these subgroups were analyzed using the Mann-Whitney *U* test or the Fisher exact test.

In order to allow statistical tests, the scores for motor and sensory impairment and morphological level were consecutively numbered from 1 (T1) to 22 (S5). These variables were handled as continuous variables. In addition, the scores for motor impairment were dichotomized according to the spinal segmental innervation of the investigated muscles. This dichotomy was achieved by dividing the variable for both muscles separately into impairment cranial to the spinal segments innervating the muscle or impairment at or caudal to these segments. For that purpose the spinal segmental innervation according to Sharrard[13] was applied. This resulted in dichotomization for the tibialis anterior muscle into above L4 and from L4 downward and for the gastrocnemius muscle into above S1 and from S1 downward. The CMAP measurements were summarized in box plots to show similarities, differences, and associations between CMAP and impairment measurements. Associations between CMAP and impairment measurements were further analyzed with Spearman rank correlation coefficients and in case of dichotomous variables with the Mann-Whitney *U* test. In addition, the CMAP-area data were logistically transformed to generate approximately normal distributions. Multivariable linear regression analyses were then performed for the subgroup in which both muscles were investigated. In these analyses, motor and sensory impairment were defined as dependent variables and the CMAP-areas as independent variables. Statistical analyses were performed using SPSS (version 14.0).

Results

CLINICAL IMPAIRMENT

The clinical impairment measurements of the investigated newborn infants are summarized in Table I. In the 31 infants included in the study, motor impairment was thoracic in 10 infants, lumbar in 14, sacral in seven, and clearly asymmetrical in four. Sensory impairment was thoracic in six infants, lumbar in 15, sacral in 10, and clearly asymmetric in five. The patellar reflex was present in 15 infants, the Achilles reflex was present in six of these 15 infants, and in one the Achilles reflex was present and the patellar reflex was absent. In the remaining 15 infants both reflexes were absent. Most spinal anomalies could be classified as myelomeningocele ($n=23$), whereas four anomalies were classified as myelocele. The other four anomalies were other types of spina bifida. The morphological level was thoracic in six infants, lumbar in 24, and sacral in one. Most spinal anomalies covered five or more vertebrae. All infants with myelomeningocele or myelocele had hydrocephalus and Chiari II malformation. Corpus callosum dysgenesis was identified in 24 of these infants.

Regarding the clinical impairment measurements, no differences were present between the subgroup in which only the tibialis anterior muscle was investigated and the subgroup in which both the gastrocnemius and tibialis anterior muscle were investigated.

CMAP

The muscles responded to stimulation in almost all infants: for the tibialis anterior muscle 26 of the 31, and for the gastrocnemius muscle 15 of the 18 infants. When the gastrocnemius muscle did not respond, neither did the tibialis anterior muscle.

The distributions of the CMAP latency and the CMAP-area are depicted in Figure S1 (supplementary material published online). Regarding the tibialis anterior latency and CMAP-area, no differences were present between the subgroup in

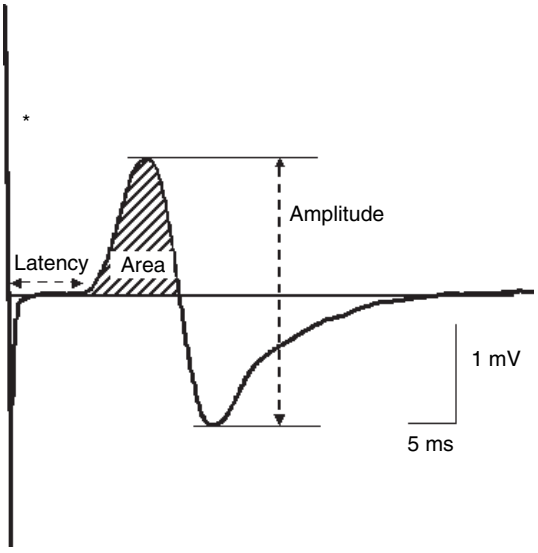

Figure 1: *Measurements of the compound muscle action potential. * Indicates stimulus artefact.*

which only the tibialis anterior muscle was investigated and the subgroup in which both the gastrocnemius muscle and tibialis anterior muscle were investigated.

ASSOCIATIONS BETWEEN CMAP AND CLINICAL IMPAIRMENT

We found strong associations between the CMAP-area and motor and sensory impairment, and muscle stretch reflexes (i.e. the less the impairment, the larger the CMAP-area). No associations were found between latency and impairment, between CMAP measurements and morphological characteristics of the spinal anomaly or between CMAP measurements and cerebral comorbidity (hydrocephalus, Chiari II malformation, and corpus callosum dysgenesis).

Table I: Impairment measurements (*n*=31)

Impairment	*n*
Motor impairment	
Thoracic[a]	10
Lumbar[b]	14
Sacral	7
Sensory impairment	
Thoracic[c]	6
Lumbar[d]	15
Sacral	10
Muscle stretch reflexes	
Both reflexes absent	15
Patellar reflex present, Achilles reflex absent[e]	9
Achilles reflex present[e]	7
Type of spinal anomaly	
Myelomeningocele	23
Myelocele	4
Lipomyelomeningocele	1
Meningocele	1
Other type of closed spina bifida	2
Cranial margin of spinal anomaly	
Thoracic	6
Lumbar	24
Sacral	1
Size of spinal anomaly	
≥10 vertebrae	4
7–9 vertebrae	9
5–6 vertebrae	14
<5 vertebrae	4
Cerebral comorbidity	
Hydrocephalus	27
Chiari II malformation	27
Corpus callosum dysgenesis	24

[a]Two asymmetric (L1-Th; Th-L2). [b]Two asymmetric (L5-S1; S1-L5). [c]One asymmetric (Th12-L1). [d]Four asymmetric (L2-L3 [2]; L4-L5; L5-S2). [e]One asymmetric.

Table II: Spearman's rank correlation of compound muscle action potential (CMAP)-area with motor and sensory impairment and morphological level of the spinal anomaly

CMAP-area	*Motor impairment*		*Sensory impairment*		*Level of the spinal anomaly*	
	Right	*Left*	*Right*	*Left*	*Right*	*Left*
Gastrocnemius, *n=15*	0.78[c]	0.70[c]	0.42[a]	0.58[b]	0.11	0.14
Tibialis anterior, *n=26*	0.46[b]	0.34[a]	0.36[a]	0.30[b]	0.21	0.20

[a]$p<0.10$. [b]$p<0.05$. [c]$p<0.01$. CMAP-area, area under the curve of the first negative wave of the compound muscle action potential.

The associations between the CMAP-area and the muscle stretch reflexes are presented in Figure 2. The CMAP-areas of both muscles were almost negligible when both reflexes were absent. The gastrocnemius CMAP-area was considerably larger when the patellar reflex was present and even larger when the Achilles reflex was present as well. This applied to a lesser extent to the tibialis anterior muscle: the CMAP-area was slightly larger when the patellar reflex was present, but did not increase any further when the Achilles reflex was present.

The associations between the CMAP-area and motor and sensory impairment are specified in Figure S2. Correlation coefficients for these associations are presented in Table II. The associations were stronger for motor impairment than for sensory impairment, but both associations were clearly stronger than the weak associations between the CMAP-area and the morphological level of the spinal anomaly. These findings applied in particular to the gastrocnemius muscle and to a lesser extent to the tibialis anterior muscle.

The analyses concerning motor impairment as a dichotomous variable are illustrated in Figure 3. This figure clearly shows that the CMAP-areas of both the gastrocnemius and the tibialis anterior muscle were statistically significantly larger when motor impairment was at or caudal to the spinal segmental innervation of the muscle in question, than when motor impairment was cranial to these segments.

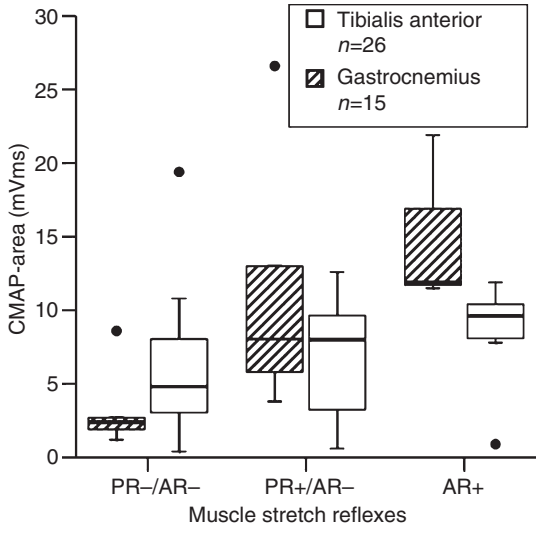

Figure 2: *Associations between compound muscle action potential (CMAP)-area and muscle stretch reflexes. The horizontal bar and the upper and lower border of each box mark median, 25th, and 75th centiles respectively. Error bars mark 5th and 95th centiles. Points lie beyond 5th and 95th centiles. As the results for both sides were almost identical, only data for the right side are presented. CMAP-area, area under the curve of the first negative wave of the compound muscle action potential; AR, Achilles reflex; PR, patellar reflex; plus sign indicates reflex present; minus sign indicates reflex absent.*

Multivariable linear regression analyses showed that the CMAP-areas of both muscles together were a better predictor for motor impairment than for sensory impairment. In all analyses, the gastrocnemius CMAP-area determined the majority of the predictive value for motor and sensory impairment (Table III).

Discussion

The present study shows strong associations between the CMAP and the severity of spina bifida in newborn infants. To be more specific, the magnitude of the CMAP, represented by the area under the curve, relates to the presence of muscle stretch reflexes and motor impairment and, to a lesser degree, to sensory impairment. To our best knowledge, this has not been reported before. Although other authors reported motor nerve conduction studies in neonatal spina bifida,[14,15] the magnitude of the CMAP was mentioned in only one study.[16] Compatible with our results, other authors also reported responses to be present in almost all assessed muscles. This is also compatible with studies using other methods of stimulation, such as electrical neural plaque stimulation,[14,17] faradic muscle stimulation,[18,19] and lumbar magnetic stimulation.[11] The presence or absence of a response cannot be a diagnostic criterion, when responses are present in virtually every case. The results in the present study demonstrate that the CMAP-area is indeed distinctive and that it may provide an additional measure for neonatal neurological impairment. A larger CMAP-area is associated with less neurological impairment.

Before further interpreting the results, some methodological remarks have to be made. To quantify the magnitude of the CMAP, the area under the curve of the first negative wave was calculated. The CMAP-area provides an estimate of the amount of functioning motor units.[20] The area was taken instead of the more commonly used amplitude, because the amplitude is more liable to temporal dispersion resulting in a larger variability in the amplitude compared to the area.[21,22] As additional temporal dispersion due to abnormal myelination can be expected in pathological neurons, the CMAP-area was considered to reflect the amount of activated motor units most appropriately.

Table III: Results of multivariable linear regression for CMAP-area predicting motor and sensory impairment[a] (n=15)

	Right				*Left*		
Step	*Predictor CMAP-area*	R^2	*p value*	*Step*	*Predictor CMAP-area*	R^2	*p value*
Motor impairment							
1	GC	0.52	0.004	1	GC	0.67	<0.001
2	GC-TA	0.60	0.01	2	GC-TA	0.73	0.001
Sensory impairment							
1	GC	0.33	0.03	1	GC	0.16	0.15
2	GC-TA	0.34	0.10	2	GC-TA	0.24	0.22

[a]CMAP-area data were transformed logistically. CMAP-area, area under the curve of the first negative wave of the compound muscle action potential; GC, gastrocnemius muscle; TA, tibialis anterior muscle; R^2, coefficient of determination.
CMAP, compound muscle action potentials; GC, gastrocnemius muscle; TA, tibialis anterior muscle.

In addition, our assessment of neurological impairment needs consideration. We assessed three modalities of neurological impairment (muscle stretch reflexes, motor and sensory impairment). These modalities are to a certain extent interdependent, but each modality can be affected to a different degree. No consensus exists about which modality is most specific or reliable for determining neurological impairment. Therefore, we used all three modalities in the analyses. The cranial demarcation of impairment to a single spinal segment may be arbitrary. However, more reliable methods are not available[23] and categorization of impairment as thoracic, lumbar, or sacral was not specific enough considering the aim of the study.

Contrasting our findings to CMAPs obtained from healthy newborn infants might be interesting from a pathophysiological point of view. However, valid normative data are not available. To subject healthy newborn infants to neurophysiological examinations as applied in the present study, for merely scientific reasons might be considered as ethically unacceptable, as the main aim of our study was to differentiate mildly affected from severely affected infants. That aim, unlike the differentiation from the healthy state, requires only data of affected infants.

The CMAP-area was most strongly associated with motor impairment and with the presence of muscle stretch reflexes, but less strongly with sensory impairment. This difference is plausible, as the CMAP above all represents motor function. However, the association with sensory impairment was more pronounced than the association between the CMAP-area and the morphological level of the spinal anomaly. This is in accordance with the assumption that the neurological impairment only partly relates to the morphological abnormalities in spina bifida.[9]

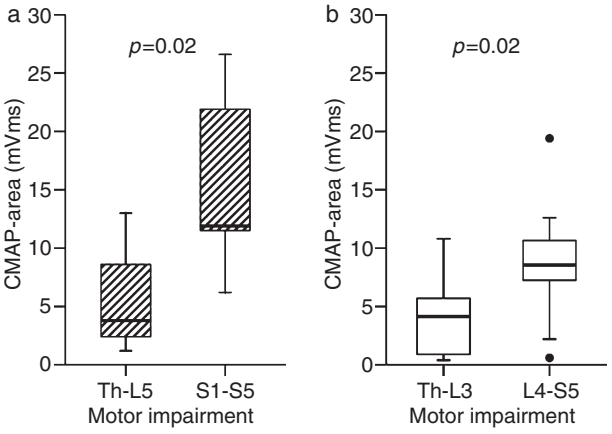

Figure 3: *Associations between compound muscle action potential (CMAP)-area and motor impairment. Data are expressed as described for Figure 2. As the results for both sides were almost identical, only data for the right side are presented. Motor impairment was dichotomized according to segmental innervation of (a) gastrocnemius muscle, and (b) tibialis anterior muscle respectively (see text for details). CMAP-area, area under the curve of the first negative wave of the compound muscle action potential; L, lumbar; Th, thoracic; p, p-value based on Mann-Whitney U test.*

In all analyses the gastrocnemius CMAP-area seems much more specific for neurological impairment than the tibialis anterior CMAP-area. This might be due to the smaller variability in the tibialis anterior CMAP-area compared to the variability in the gastrocnemius CMAP-area (Fig. S1). Considering the spinal segmental organization and the distribution of impairment levels within our study group, the tibialis anterior muscle is usually less affected than the gastrocnemius muscle. Furthermore, the ability to recruit motor neurons from spinal segments cranial to the spinal anomaly applies more to the tibialis anterior muscle than to the gastrocnemius muscle.

Clear associations exist between the presence of muscle stretch reflexes and the CMAP-area with differences between the two muscles (Fig. 2). The neurosegmental association between the gastrocnemius muscle and the Achilles reflex, and the partial neurosegmental association between the tibialis anterior muscle and the patellar reflex may cause this difference. The difference in CMAP-area between the two muscles when the Achilles reflex is present, can be explained by a difference in muscle volume. Furthermore, the association between the gastrocnemius CMAP-area and the presence of the Achilles reflex suggests that non-excitability of a reflex results from an insufficient amount of functioning efferent motor neurons, rather than from an interrupted reflex arc. For infants in whom the Achilles reflex could not be elicited, a CMAP was still obtainable. This proves the integrity of efferent neurons. Evidence for the integrity of afferent neurons is provided by Sival et al.[8]

In spina bifida, both upper and lower motor neuron dysfunction might be present. To what extent the upper or the lower motor neuron determines the neurological impairment remains a matter of debate.[8,14] The CMAP-area provides an estimate of the residual lower motor neuron function in affected spinal segments. The association between the CMAP-area and motor impairment shows that this residual function decreases when more cranial spinal segments are involved in motor impairment. This suggests a cranio-caudal gradient (i.e. a cranio-caudal decrease) in lower motor neuron function in the affected spinal segments. This gradient might be related to the degree of upper motor neuron function. In normal neurodevelopment, the upper motor neuron is involved in the activity dependent regulation of the development of the lower motor neuron, as described by Eyre et al.[24] In spina bifida, the upper motor neuron must pass through disordered spinal segments to synapse to the lower motor neuron in affected spinal segments. In longer tracts the integrity of the upper motor neuron is more vulnerable than in shorter tracts. This might result in a more definite underdevelopment of lower motor neurons in affected caudal segments than in affected cranial segments.

The results on motor impairment as a dichotomous variable show that a large CMAP-area is related to normal leg movements and a small CMAP-area to paralysis, considering our method to assess motor impairment (Fig. 3). The presence of normal movements denotes that the upper motor neuron integrity is at least partially preserved. This implies that the CMAP-area also provides indirect information about the degree of upper motor neuron function in spina bifida.

The above-mentioned considerations imply that the demarcation of motor impairment to spinal segments is a simplification of the actual impairment, because residual motor function is present in affected spinal segments caudally from this demarcation. This residual function might explain the disagreement between patterns of muscle activity and neurosegmental innervation, as described by McDonald et al.[6] Evaluation of the residual motor function by assessment of the CMAP-area may provide a more precise estimate of motor impairment. Since the method as used is easy to perform and well tolerated, we recommend CMAP assessment as an additional instrument in the preoperative neonatal assessment of spina bifida. For clinical use, we suggest that the assessment of only the gastrocnemius muscle would be sufficient, since this muscle is most sensitive. To what extent this method has predictive value for neurological impairment and disability in later life requires further follow-up study. The present results support the hypothesis that the CMAP-area may be indicative of neurological impairment at a later age as well and that a larger CMAP-area may predict a better functional outcome.

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Supplementary material

The following supplementary material is available for this article online:

Figure S1: Distribution of CMAP latency and CMAP-area.

Figure S2: Associations between CMAP-area and motor impairment and sensory impairment.

This material is available as part of the online article from http://www.blackwell-synergy.com/doi/abs/10.1111/j.1469-8749.2008.03041.x (this will link you to the article abstract)

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List of abbreviations

CMAP	Compound muscle action potential
CMAP-area	Area under the curve of the first negative peak of the compound muscle action potential
GC	Gastrocnemius muscle
TA	Tibialis anterior muscle