Ultrasound Obstet Gynecol 2024; **63**: 53–59
Published online in Wiley Online Library (wileyonlinelibrary.com). **DOI:** 10.1002/uog.27536.

Revisiting MOMS criteria for prenatal repair of spina bifida: upper gestational-age limit should be raised and assessment of prenatal motor function rather than anatomical level improves prediction of postnatal function

L. TRIGO[1,2,3,4], R. H. CHMAIT[5], A. LLANES[5], G. CATISSI[6], E. EIXARCH[1,2,3,7], A. VAN SPEYBROECK[8] and D. A. LAPA[6,9]

[1]*BCNatal – Fetal Medicine Research Center, Hospital Clínic and Hospital Sant Joan de Déu, University of Barcelona, Barcelona, Spain;* [2]*Institut d'Investigacions Biomèdiques August Pi i Sunyer (IDIBAPS), Barcelona, Spain;* [3]*Faculty of Medicine and Health Sciences, University of Barcelona, Barcelona, Spain;* [4]*Obstetrics and Gynecology Department, Pourtalès Hospital, Neuchâtel, Switzerland;* [5]*Los Angeles Fetal Surgery, Department of Obstetrics and Gynecology, Keck School of Medicine, University of Southern California, Los Angeles, CA, USA;* [6]*Fetal Therapy Program, Hospital Israelita Albert Einstein, São Paulo, Brazil;* [7]*Centre for Biomedical Research on Rare Diseases (CIBER-ER), Barcelona, Spain;* [8]*Children's Hospital Los Angeles, University of Southern California, Los Angeles, CA, USA;* [9]*Fetal Medicine Department, Hospital Infantil Sabará, São Paulo, Brazil*

KEYWORDS: fetal surgery; fetoscopic repair; meningomyelocele; motor function; myeloschisis; neural tube defect; neurological motor function; open spina bifida; rachischisis

CONTRIBUTION

What are the novel findings of this work?
In this longitudinal evaluation, we found that there is no significant change in neurological motor function in the 4-week interval from first evaluation at 22 weeks of gestation up to the time of prenatal surgery at or beyond 26 weeks of gestation. We also found that the anatomical level of open spina bifida is a weak tool with which to predict postnatal ambulation following prenatal repair, compared with ultrasound assessment of motor function via systematic documentation of lower-extremity movements.

What are the clinical implications of this work?
The use of prenatal motor-function level assessed by ultrasound should replace the anatomical level to predict postnatal motor-function prognosis in fetuses undergoing prenatal repair of open spina bifida. Moreover, the upper limit of gestational age for proposing prenatal surgery should be revisited, as motor function does not change even when surgery is delayed beyond 26 weeks of gestation.

ABSTRACT

Objectives *To determine if the lower-extremity neurological motor function level in fetuses with open spina bifida deteriorates within the 4-week interval between a first prenatal motor assessment at around 22 weeks of gestation and a second evaluation, prior to 'late' prenatal surgery, defined as surgery at 26–28 weeks and, in certain situations, up to 30 weeks, and to assess the association between prenatal presurgical motor-function level, anatomical level of the lesion and postnatal motor-function level.*

Methods *This was a two-center cohort study of 94 singleton fetuses with open spina bifida which underwent percutaneous repair using the skin-over-biocellulose for antenatal fetoscopic repair (SAFER) technique between December 2016 and January 2022. All women underwent two prenatal systematic ultrasound evaluations, approximately 4 weeks apart, with the second one being performed less than 1 week before surgery, and one postnatal evaluation via physical examination within 2 months of birth. Motor-function classification was from spinal level T12 to S1, according to key muscle function. Each leg was analyzed separately; in case of discrepancy between the two legs, the worst motor-function level was considered for analysis. Motor-function-level evaluations were compared with each other and with the anatomical level as observed on ultrasound. Independent predictors of a postnatal reduction in motor-function level were assessed using a logistic regression model.*

Results *Prenatal motor-function level was assessed at a median gestational age of 22.5 (interquartile range (IQR), 20.7–24.3) and 26.7 (IQR, 25.4–27.3) weeks,*

Correspondence to: Dr L. Trigo, Rue de la Maladière, 45, 2000, Neuchâtel, Switzerland (e-mail: trigoxlv@gmail.com)

Accepted: 16 October 2023

with a median interval of 4.0 (IQR, 2.4–6.0) weeks. The median gestational age at surgery was 27.0 (IQR, 25.9–27.6) weeks and the postnatal examination was at median age of 0.8 (IQR, 0.3–5.4) months. There was no significant difference in motor-function level between the two prenatal evaluations (P = 0.861). We therefore decided to use the second prenatal evaluation for comparison with postnatal motor function and anatomical level. Overall, prenatal and postnatal motor function evaluations were significantly different from the anatomical level (preoperative assessment, P = 0.0015; postnatal assessment, P = 0.0333). Comparing prenatal with postnatal motor-function level, we found that 87.2% of babies had similar or improved motor function compared with that prior to prenatal surgery. On logistic regression analysis, lower anatomical level of defect and greater difference between anatomical level and prenatal motor-function level were identified as independent predictors of postnatal motor function (odds ratio, 0.237 (95% CI, 0.095–0.588) (P = 0.002) and 3.44 (95% CI, 1.738–6.813) (P < 0.001), respectively).

Conclusions *During a 4-week interval between first ultrasound evaluation and late fetal surgical repair of open spina bifida, motor function does not change significantly, suggesting that late repair, ≥ 26 weeks, does not impact negatively on motor-function outcome. Compared with the anatomical level of the lesion, preoperative neurological motor-function assessment via ultrasound is more predictive of postnatal motor function, and should be included in preoperative counseling.*

INTRODUCTION

The MOMS trial[1] established prenatal surgery as a standard treatment option for open spina bifida (OSB). Using 25.9 weeks' gestation arbitrarily as the upper gestational age for surgery, it showed benefits that included a reduction in the need for cerebrospinal fluid shunt placement and in the rate of moderate-to-severe hindbrain herniation[1]. In addition, the MOMS trial showed that infants repaired prenatally had improved motor function compared with those that underwent traditional postnatal repair, exhibiting motor function that was two or more levels better than that expected according to their anatomical lesion level[1].

In 2016, Carreras *et al.*[2] proposed a new technique to evaluate prenatally, using systematic ultrasound assessment, the lower-extremity neuromotor-function level in fetuses with OSB. Their data showed that it was possible to assess lower-limb movements by ultrasound, with good correlation to motor function after birth. In 2017, Maroto *et al.*[3] confirmed these findings and found the technique to be easily reproducible. Interestingly, Carreras *et al.*[2] found that the anatomical level did not correlate well with the postnatal motor-function level. Similar results were found by an international consortium for fetoscopic repair of OSB in a study that included 300 cases[4]: only 19% (49/257) of infants had a concordant anatomical level and postnatal motor function. Recently, Maiz *et al.*[5] found similar results in a prospective trial, reporting moderate agreement of fetal motor level of the lesion before prenatal repair of OSB, but only slight agreement of presurgery anatomical level, with motor level at birth. This evidence raises the question as to whether lower-extremity neurological motor-function level assessed by prenatal ultrasound should be used in counseling patients who are considering prenatal repair of OSB.

Moreover, our group has questioned the upper gestational-age limit for offering prenatal surgery, since our previously published results[6], including cases which underwent fetoscopic repair beyond 26 weeks' gestation, were similar to those of the MOMS trial. The median gestational age at surgery in our cohort was 27 weeks, as we took the position that delaying surgery to 26–28 weeks, or, under certain circumstances, even later in gestation, could mitigate the risks associated with extreme prematurity; having delayed prenatal surgery by even a week or two could prove crucial if, for example, an emergency delivery was required during or soon after the surgery[6–8].

Therefore, the aims of our study were: to determine whether the lower-extremity motor function of fetuses with OSB deteriorates between presurgical evaluations in the 4-week interval prior to 'late' prenatal surgery, defined as surgery at 26–27 weeks' gestation, and to compare prenatal presurgical motor-function level with the anatomical level of the lesion and to assess the association between prenatal presurgical motor-function level, anatomical level of the lesion and postnatal motor-function level.

METHODS

The study was approved by the ethics committees of Hospital Israelita Albert Einstein in São Paulo, Brazil, and the University of Southern California, Keck School of Medicine, CA, USA (CAAE: 48991021.0.0000.071 and HS-18-00591, respectively).

Study population

This was a two-center retrospective cohort study of 94 singleton fetuses with OSB which underwent percutaneous repair using the skin-over-biocellulose for antenatal fetoscopic repair (SAFER) technique between December 2016 and January 2022[6,9–11]. Inclusion criteria for surgery were similar to those of the MOMS trial, except that the upper gestational age limit was 28 weeks of gestation (and exceptionally, in a few cases presenting late, up to 30 weeks). All women had at least two presurgical ultrasound evaluations, with the last being performed less than 1 week before surgery. For the purposes of this study we included the last examination before surgery and the one approximately 4 weeks prior

to this. Both evaluations were performed by the same operator in São Paulo (D.L.) and in Los Angeles (R.H.C.).

Evaluation of motor-function level

Prenatal motor-function level was assessed by ultrasound for each leg separately, according to Carreras *et al.*[2], and for each fetus the worse of the two motor-function levels was considered in our analysis. Briefly, motor-function level was classified according to lower-limb movements, as follows: no hip or leg movement, T12; hip flexion, L1; hip adduction, L2; knee extension, L3; knee flexion, L4; dorsal flexion of ankle, L5; and plantar flexion of ankle, S1. Due to the impossibility of evaluating other sacral levels on the prenatal scans, we classified all cases as 'sacral', even if, at postnatal evaluation, they were considered as motor-function level S2 or S3. All prenatal motor-function evaluations were carried out by one of two fetal-medicine experts (D.L., R.H.C.), one from each center involved in the study. Postnatal motor function was classified via physical examination by a physiatrist or a pediatrician specialized in the care of children with OSB. Motor function was considered to have changed significantly if the difference between preoperative and postnatal motor-function level was ≥ 2.

Imaging evaluation

The defect type was classified according to the presence (myelomeningocele) or absence (myeloschisis) of a sac covering the defect. Anatomical level was defined on the first of the two ultrasound examinations (confirmed on the second), in a midsagittal image of the spine, as the highest vertebral level showing splaying of the vertebral ossification centers and without coverage, as described previously[12]. Each lateral ventricle was assessed in the axial plane according to ISUOG guidelines on sonographic examination of the fetal central nervous system[13], and we used the larger of the two for analysis. In all ultrasound examinations, we recorded the absence or presence of clubfoot (unilateral or bilateral).

Surgical protocol

In this study, we included OSB cases that were operated in São Paulo, Brazil, with the original SAFER technique[9,10,14] and by the minilaparotomy technique, a variation of the SAFER technique[11]. Both techniques have been described previously in detail[10,11,14]. Briefly, maternal intravenous anesthesia with minimal general anesthesia was administered. Ultrasound was used to guide the placement of three (rarely four) trocars, of which one was a 5.8-mm balloon blunt-tip system (Applied Medical®, Rancho Santa Margarita, CA, USA) and the others were 11-Fr vascular introducers (Terumo®, Tokyo, Japan). Amniotic fluid was removed and the uterus insufflated with heated and humidified carbon dioxide (CO_2), usually up to a pressure of 15 mmHg. The fetus was then repositioned in a prone position, with the legs spread apart.

The neuroplacode was incised from the transition zone. A biocellulose patch (Bionext®, Bionext, São Paulo, SP, Brazil) was used in São Paulo and a Durepair patch (Medtonic Neurologic Technologies, Goleta, CA, USA) was used in the USA. The patch was placed over the neural placode to protect the medulla. We do not place sutures to fix the patch to any tissue or to close the dura mater directly. If possible, a myofascial flap was created and sutured over the patch using absorbable Quill® (2020 Surgical Specialties Corporation, Westwood, MA, USA)[10,11]. The skin was sutured at the midline with running sutures (nonabsorbable Quill™©, 2020 Surgical Specialties Corporation) either directly above the patch, or above the myofascial flap if it was possible to develop the flap. If the skin was not sufficient to close the defect in the midline, in São Paulo, from Case 76 onwards, we included a skin substitute (Nevelia®, Symatese, Chaponost, France), whereas the Los Angeles group used lateral relaxing incisions. Before Case 76 in São Paulo, Integra® (Dermal Regeneration Template LifeSciences, Plainsboro, NJ, USA) was used, fitted to cover the skin gap.

At the end of surgery, the reserved amniotic fluid or warmed lactated Ringer's solution was returned to the uterine cavity, after the CO_2 had been slowly released. No sutures were placed in the myometrium to close the trocar sites; only the maternal skin was closed, except in cases that underwent percutaneous minilaparotomy[11].

Statistical analysis

Data were stored and analyzed using STATA (Stata Statistical Software: Release 13, 2013; StataCorp LP, College Station, TX, USA). Anatomical and motor-function levels were transformed into numerical data from 0 (T12) to 6 (sacral). Categorical variables are presented as numbers of cases and percentage and were compared by Pearson's χ^2 or Fisher's exact test, as appropriate. Continuous variables are presented as mean and SD or median and interquartile range (IQR), following testing for normality. These were compared by Student's *t*-test if normally distributed and Wilcoxon's signed-rank test if non-normally distributed when comparing matched pairs, or by Wilcoxon's rank-sum test for comparison of two independent populations. Multiple logistic regression was performed to identify independent variables associated with a non-reduced postnatal motor-function level. $P < 0.05$ was considered statistically significant.

RESULTS

Included in the study were 94 fetuses, 49 from São Paulo, Brazil and 45 from Los Angeles, USA. Myelomeningocele was present in almost three quarters of the fetuses (73.4% (69/94)) and myeloschisis was present in the remaining cases (26.6% (25/94)). Prenatal lower-extremity motor-function level was assessed at a median (IQR) gestational age of 22.5 (IQR, 20.7–24.3) and 26.7 (IQR, 25.4–27.3) weeks of gestation, with a median (IQR) interval between prenatal assessments of 4.0 (2.4–6.0) weeks.

Postnatally, babies were evaluated at a median age of 0.8 (IQR, 0.3–5.4) months. Characteristics of the study population are presented in Table 1.

There was no significant difference in motor-function level between the two prenatal assessments ($P = 0.861$), despite the 4-week interval between the evaluations (Figure 1a and Videoclip S1). We therefore decided to use the second prenatal evaluation for comparison with postnatal motor function and anatomical level.

The anatomical level of the lesion was significantly different from the postnatal motor-function-level assessment. Only 22 (23.4%) babies had the same anatomical level when compared with their postnatal motor-function level ($P = 0.026$), whereas 29 (30.9%) had lower and 43 (45.7%) had higher motor-function level (Figure 1b). Overall, prenatal and postnatal motor-function evaluations were significantly different from the anatomical level (preoperative assessment, $P = 0.0015$; postnatal assessment, $P = 0.0333$).

Analyzing prenatal *vs* postnatal motor-function level, 81.9% (77/94) of infants presented a similar motor-function level (i.e. a difference in level of < 2)

Table 1 Maternal and fetal characteristics of study population of 94 singleton fetuses with open spina bifida, overall and according to center where prenatal surgery took place

Characteristic	*Total (n = 94)*	*São Paulo (n = 49)*	*Los Angeles (n = 45)*
Maternal characteristics			
Maternal age (years)	32 (28–35)	32 (28–37)	31 (27–34)
Parity	1 (0–1)	0 (0–1)	1 (0–2)
Body mass index (kg/m^2)	28.2 (25.6–31.1)	27.5 (24.9–30.1)	29.3 (26.9–31.2)
Diabetes	3 (3.2)	2 (4.1)	1 (2.2)
Hypothyroidism	4 (4.3)	3 (6.1)	1 (2.2)
Fetal characteristics			
Type of defect			
Myelomeningocele	69 (73.4)	39 (79.6)	30 (66.7)
Myeloschisis	25 (26.6)	10 (20.4)	15 (33.3)
Anatomical level	L5 (L4–S1)	L5 (L4–S1)	L4 (L4–L5)
GA at first prenatal motor-function evaluation (weeks)	22.5 (20.7–24.3)	22.2 (20.0–24.6)	22.9 (21.0–24.0)
First prenatal motor-function level	L5 (L5–S1)	L5 (L4–L5)	S1 (S1–S1)
GA at second prenatal motor-function evaluation (weeks)	26.7 (25.4–27.3)	26.1 (25.0–27.0)	27.0 (26.6–27.4)
Second prenatal motor-function level	L5 (L5–S1)	L5 (L4–L5)	S1 (S1–S1)
GA at surgery (weeks)	27.0 (25.9–27.6)	26.3 (25.4–27.3)	27.1 (27.0–27.6)
GA at delivery (weeks)	33.4 (31.5–36.1)	33.0 (31.4–35.3)	35.0 (31.9–37.3)
Age at postnatal evaluation (months)	0.8 (0.3–5.4)	5.4 (1.3–9.8)	0.4 (0.1–0.6)
Postnatal motor-function level	L5 (L4–S1)	L5 (L4–L5)	S1 (L5–S1)
Male sex	42 (44.7)	23 (46.9)	19 (42.2)
Anterior placenta	42 (44.7)	25 (51.0)	17 (37.8)

Data are presented as median (interquartile range) or *n* (%). GA, gestational age.

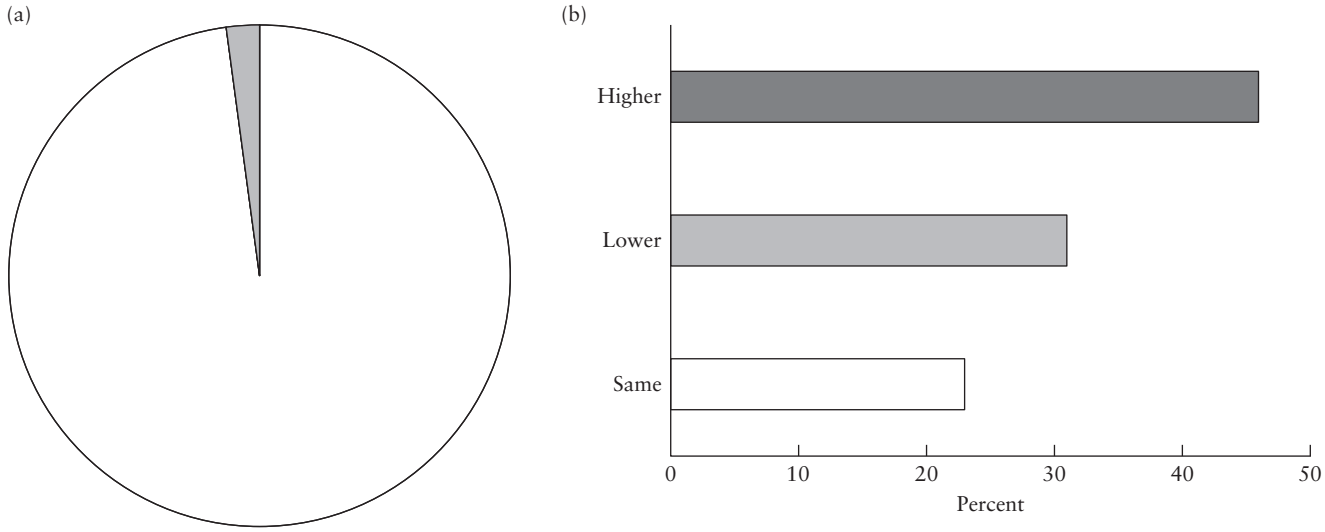

Figure 1 (a) Pie chart comparing prenatal motor function at first *vs* second preoperative evaluation, in 94 singleton fetuses with open spina bifida, showing minimal difference in motor-function level over 4-week interval. Motor-function level: same (□), lower (▩), higher (■). (b) Bar chart showing that anatomical level of defect coincided with postnatal motor-function level in only 23.4% of cases. In remaining (almost 80%) cases, anatomical level and motor-function level were discordant.

Table 2 Fetal characteristics according to postnatal reduction in motor function of two or more levels with respect to prenatal motor-function level in 94 singleton fetuses which underwent surgery for open spina bifida

	Postnatal reduction in motor function		
Fetal characteristic	*Yes (n = 12)*	*No (n = 82)*	P
GA at first prenatal motor-function evaluation (weeks)	23.1 (21.2–24.1)	22.4 (20.6–24.3)	0.803
GA at second prenatal motor-function evaluation (weeks)	26.7 (26.4–27.4)	26.7 (25.3–27.3)	0.594
GA at surgery (weeks)	27.1 (26.6–27.5)	27.0 (25.6–27.6)	0.529
GA at delivery (weeks)	34.2 (32.1–36.2)	33.3 (31.1–36.1)	0.479
Myeloschisis	3 (25.0)	22 (26.8)	1.000
Area of defect (cm^2)	8.5 ± 8.5	4.6 ± 3.2	0.003
Ventriculomegaly ≥ 15 mm	5 (41.7)	26 (31.7)	0.493
Anatomical level	L4 (L3–L5)	L5 (L4–S1)	0.219
Prenatal motor-function level	S1 (L5–S1)	L5 (L4–S1)	0.038
Postnatal motor-function level	L4 (L3–L4)	S1 (L5–S1)	< 0.001
Clubfoot	6 (50.0)	20 (24.4)	0.085
Female sex	6 (50.0)	46 (56.1)	0.692

Data are presented as median (interquartile range), *n* (%) or mean ± SD. GA, gestational age.

Table 3 Multiple logistic regression for reduced postnatal motor function in 94 singleton fetuses which underwent surgery for open spina bifida

Characteristic	*Odds ratio (95% CI)*	P
Myelomeningocele	0.389 (0.067–2.241)	0.291
Anatomical level	0.237 (0.095–0.588)	0.002
Difference between anatomical level and prenatal motor-function level*	3.441 (1.738–6.813)	< 0.001
GA at surgery (weeks)	1.014 (0.582–1.789)	0.960
Male sex	2.174 (0.394–12.000)	0.373

*Anatomical level minus prenatal motor-function level. GA, gestational age.

in prenatal and postnatal evaluations, and 5.3% (5/94) had improved motor function after birth. Thus, in total, 87.2% (82/94) of cases had a similar or improved motor-function level postnatally ($P < 0.001$). Table 2 compares fetal characteristics according to whether there was worse motor function on postnatal than on prenatal evaluation. There was no difference in median gestational age at surgery (27.1 (IQR, 26.6–27.5) *vs* 27.0 (IQR, 25.6–27.6) weeks, $P = 0.529$) or in type of lesion (myeloschisis: 25.0% *vs* 26.8%, $P = 1.000$) in the group with *vs* the group without postnatal reduction in motor function. However, cases with deterioration in motor level had larger mean ± SD defect areas (8.5 ± 8.5 *vs* 4.6 ± 3.2) cm^2, $P = 0.003$).

With respect to the performance of the two different centers, there was no significant difference in the number of cases with reduction of motor-function level postnatally (5 (10.2%) in São Paulo *vs* 7 (15.6%) in Los Angeles, $P = 0.542$).

To identify independent factors associated with the reduction in motor-function level between prenatal and postnatal evaluations, we performed a logistic regression analysis, including type of defect, anatomical level, difference between prenatal motor-function level and anatomical level of defect, gestational age at surgery and fetal sex. This showed that the lower the anatomical level, the higher the chances of maintaining prenatal motor function in the postnatal period (OR, 0.237, $P = 0.002$) and that the larger the difference between anatomical level and prenatal motor-function level, the higher the chances of reduced postnatal motor function (OR, 3.44, $P < 0.001$) (Table 3).

DISCUSSION

Main findings

In this study we found no significant deterioration in fetal lower-extremity neurological motor-function level in the 4-week interval before prenatal surgery, suggesting that delaying prenatal repair up to 28 weeks' gestation does not hamper the preservation of motor function. Moreover, prenatal surgery later than 26 weeks has the advantage of avoiding the risk of extreme prematurity. We also demonstrated that the anatomical level of the lesion is a weak prenatal tool with which to predict postnatal motor function in fetuses with OSB that undergo prenatal repair. Conversely, preoperative motor-function-level assessment by ultrasound was a good predictor of postnatal motor function being concordant in almost 90% of cases.

Review in context of previous studies

Our results are in agreement those of a recent study published by Maiz *et al.*[5]. Anatomical level of the lesion and postnatal motor-function level were concordant in only 23% of cases and, comparing prenatal and postnatal motor-function levels, we found that 87% of infants presented similar or improved motor-function level in the postnatal evaluation. Similarly, Maiz *et al.*[5] reported 88.5% of cases with similar and 1.9% of cases with improved postnatal motor-function level.

Since the MOMS trial, many studies have reported the benefits of prenatal surgery to repair OSB in terms of improvement in motor function[4,15,16], believing that the difference between anatomical level and postnatal

motor-function level demonstrates neurological improvement. However, as we and others have shown, this is not the case. In fact, prenatal surgery allows preservation of the neurological motor function that is present at the time of the prenatal repair. In other words, the MOMS study showed that lack of prenatal repair led to degradation in motor function compared with the level of the anatomic lesion, while prenatal repair allowed maintenance of motor function at the level present at the time of prenatal repair, which represented an apparent improvement compared with the anatomical level.

Taking into consideration the 'two-hit hypothesis' for the impairment associated with OSB, it seems intuitive that longer duration of neural exposure to the intrauterine environment increases the intrauterine spinal cord injury[17,18]. Therefore, early repair of the defect has been suggested to avoid longer exposure of the neural tissue to amniotic fluid. In the MOMS trial, a gestational age between 19.0 and 25.9 weeks was adopted as an inclusion criterion for prenatal surgery[1], and this has been the gold standard so far. However, when the second hit strikes is unknown. Prenatal repair at a later gestational age (> 26 weeks) has been reported, and offers the potential to reduce the risk of complications due to extreme prematurity[8,19,20]. We found that there was no clinically significant deterioration in motor function between the two prenatal time points (~22 and ~26 weeks' gestation), thus validating our protocol of postponing prenatal repair to up to 28 weeks. Thus, it appears that during this period in mid pregnancy, the amniotic fluid may not be sufficiently harmful to exposed nerves that there is significant deterioration in motor function. This observation could explain why the motor-function outcomes from our previous work (which included cases with prenatal repair beyond 26 weeks' gestation)[6] were similar to or better than those of the MOMS study, which performed repair before 26 weeks.

Clinical applications

We have shown previously that central nervous system anomalies do not increase when prenatal surgery for OSB is performed beyond 26 weeks of gestation[21–23]. Our current data, furthermore, support that there is no deterioration in prenatal motor function when prenatal surgery occurs beyond 26 weeks. This finding should be reassuring when a diagnosis of OSB is made late, due to socioeconomic imbalances or lack of appropriate prenatal screening. It also offers the possibility to prevent complications of extreme prematurity that may be associated with earlier prenatal surgery. It is important to consider that, in many countries, termination of pregnancy is not an option, and, in others, although possible, it is not generally considered, for cultural and/or religious reasons. The possibility of offering the affected infant improvement in quality of life via prenatal surgery is key in such settings.

Although the MOMS trial established a comparison between anatomical level and postnatal motor function to assess the efficacy of prenatal surgery with respect to motor function[1], based on our findings we propose that this evaluation should be revisited. Our results provide evidence that the prenatal ultrasound evaluation of motor-function level proposed by Carreras *et al.* in 2016[2] is much more reliable than the anatomical level in predicting postnatal motor function following prenatal repair of OSB, and should be used in counseling prior to surgery.

Strengths and limitations

Our study has several strengths and limitations. Strengths include the relatively large series with minor differences in surgical protocol. This was a two-center study that yielded concordant results between the two centers.

Limitations include that we evaluated the impact of 'late' surgery, defined as surgery at 26–28 weeks' gestation, with regard to fetal motor function and not with regard to the impact of this delay on fetal ventriculomegaly or subsequent need for cerebrospinal fluid diversion. Furthermore, our data evaluated SAFER and percutaneous/minilaparotomy technique results only, so our findings may not be transferable to other repair techniques. Finally, because there is no ultrasound definition of prenatal motor-function level lower than S1 described in the literature, all sacral function cases were grouped together.

Conclusions

We have shown that prenatal motor-function level in fetuses with OSB does not change significantly in the 4-week period from 22 to 26 gestational weeks. Therefore, prenatal repair of OSB beyond 26 weeks of gestation most likely does not alter the prognosis for lower-extremity motor function.

We believe that it is time to revisit the inclusion criteria adopted by the MOMS trial for offering fetal surgery for OSB. We propose that a later gestational age limit, around 28 weeks, should be accepted for surgery and that prenatal motor-function level should take priority over anatomical level of the lesion in prenatal assessment and counseling for pregnancies affected by OSB.

ACKNOWLEDGMENTS

We thank all the families for their time and motivation during interviews and appointments, without whom this project would not be possible. L.T. and E.E were funded by the Erasmus+ Programme of the European Union (Framework Agreement number: 2013-0040). This publication reflects the views only of the authors, and the Commission cannot be held responsible for any use which may be made of the information contained therein. Additionally, the present research received funding from the Cerebra Foundation for the Brain Injured Child (Carmarthen, Wales, UK) and AGAUR 2017 SGR grant n° 1531. E.E. received funding from the Convocatòria Intensificació Interna per als professionals de l'Hospital Clínic de Barcelona 2023, granted by Hospital Clínic de Barcelona.

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SUPPORTING INFORMATION ON THE INTERNET

The following supporting information may be found in the online version of this article:

Videoclip S1 (a) Plantar flexion (S1 level) at first visit. (b) Same fetus, 3 weeks later, showing that S1 motor function level was preserved over this time interval. mie, lower left limb.