Lumbar hyperlordosis of neuromuscular origin: pathophysiology and surgical strategy for correction

Raphaël Vialle · Nejib Khouri · Christophe Glorion ·
Joël Lechevallier · Christian Morin

Received: 4 February 2006 / Revised: 22 May 2006 / Accepted: 15 June 2006 / Published online: 12 September 2006
© Springer-Verlag 2006

Abstract Lumbar hyperlordosis of neuromuscular origin is rare and requires surgical treatment in order to preserve a good sitting posture. We report twenty-seven cases of a preponderantly sagittal hyperlordosis deformity of the lumbar spine in patients with neuromuscular disorders and identify the indications and results of treatment.

Seventeen males and ten females, aged 13 to 27 years, underwent operations for a lumbar hyperlordosis of

neuromuscular origin responsible for major difficulties in sitting. In all patients, the sacrum was horizontal and associated in twenty-six cases with marked pelvic anteversion. Eleven patients were treated surgically by a posterior approach. The sixteen remaining patients had a preliminary discectomy, followed by posterior correction and fusion. Lumbar hyperlordosis was reduced from 8° to 77° between L1 and S1. The horizontal sacrum was

R. Vialle · N. Khouri
Department of Paediatric Orthopaedics, Université Pierre et Marie
Curie-Paris 6, Armand Trousseau Hospital,
26, avenue du Docteur Arnold Netter,
75571 Cedex 12 Paris, France

R. Vialle · C. Glorion
Department of Paediatric Orthopaedics,
Necker-Enfants Malades Hospital,
149 rue de Sèvres,
75015 Paris, France

R. Vialle
Department of Orthopaedic and Pediatric Surgery,
Fondation Hôpital Saint Joseph,
185 rue Raymond Losserand,
75647 Cedex 14 Paris, France

J. Lechevallier
Department of Paediatric Orthopaedics, Rouen Hospital,
76031 Rouen Cedex, France

C. Morin
Department of Paediatric Orthopaedics,
Institut Calot,
rue du Docteur Calot,
62600 Berck-sur-mer, France

R. Vialle (✉)
105 avenue André Morizet,
92100 Boulogne Billancourt, France
e-mail: raphael.vialle@trs.aphp.fr

Present address:
R. Vialle
Department of Paediatric Orthopaedics,
Institut Calot,
rue du Docteur Calot,
62600 Berck-sur-mer, France

Present address:
N. Khouri
Department of Orthopaedic and Pediatric Surgery,
Fondation Hôpital Saint Joseph,
185 rue Raymond Losserand,
75647 Cedex 14 Paris, France

Present address:
C. Glorion
Department of Paediatric Orthopaedics,
Necker-Enfants Malades Hospital,
149 rue de Sèvres,
75015 Paris, France

Present address:
J. Lechevallier
Department of Paediatric Orthopaedics, Rouen Hospital,
76031 Rouen Cedex, France

Present address:
C. Morin
Department of Paediatric Orthopaedics,
Institut Calot,
rue du Docteur Calot,
62600 Berck-sur-mer, France

partially reduced with an improvement from 8° to 50° . Consequently, patients recovered a comfortable sitting position. One patient died of respiratory complications six weeks after surgery.

Surgical correction is a demanding procedure which can be performed by a posterior approach. It is mandatory to analyse the spino-pelvic balance to avoid iliac retroversion and the loss of the role of the ischia in the sitting position.

Résumé L'hyperlordose lombaire d'origine neuro musculaire est rare et nécessite un traitement chirurgical afin de conserver une bonne station assise. Nous rapportons 27 cas de déformation sagittale avec hyperlordose chez des patients présentant des troubles neuro musculaires afin de déterminer les indications et les résultats de ces traitements. 17 patients étaient de sexe masculin et 10 de sexe féminin avec un âge s'échelonnant de 13 à 27 ans, tous opérés pour une hyperlordose lombaire d'origine neuro musculaire avec difficulté dans la position assise. Chez tous les patients le sacrum était horizontalisé et associé dans 26 cas sur 27 une antéversion pelvienne très marquée. 11 patients ont été traités par voie postérieure simple, les 16 restant ont été traités par voie postérieure avec correction et greffe après une discectomie préliminaire. L'hyperlordose lombaire a été réduite à 77° entre L1 et S1. L'horizontalisation du sacrum a été améliorée de façon partielle de 8° à 50° . Tous les patients ont retrouvé une position assise confortable, 1 patient est décédé de complications respiratoires six mois après la chirurgie. Le traitement chirurgical de ces déformations postérieures peut être réalisé par abord postérieur. Il est nécessaire d'analyser en pré-opératoire la balance rachidienne et pelvienne afin d'éviter une rétroversion iliaque et une perte du rôle de l'ischion dans la position assise.

Introduction

Spinal deformities of neuromuscular origin are secondary to imbalance between postural and muscular forces applied to the growing axial skeleton. These deformities are often progressive and unresponsive to bracing [1]. In non-ambulatory patients, associated pelvic obliquity can be responsible for difficulty with sitting. In cerebral palsy, abnormal spasms of the axial muscles and secondary contractures cause a progressive scoliosis which can be associated with sagittal imbalance. Most reported cases of sagittal imbalance secondary to neuromuscular disorders describe scoliosis with concomitant kyphosis [2]. We report 27 patients with neuromuscular disorder and a preponderantly sagittal deformity with hyperlordosis of the lumbar spine, a rare situation. We discuss the underlying mechanisms explaining the onset and progression of this rare deformity. Treatment of this deformity is difficult

and requires careful analysis of trunk-pelvis relationships with consideration of the pelvis as a pelvic “vertebra”. We present and discuss two different surgical strategies.

Materials and methods

Twenty-seven patients presenting with neuromuscular disorders associated with severe spinal sagittal deformity with lumbar hyperlordosis were studied by review of clinical data and analysis after digitalisation of all spinal radiographs during treatment using the Spineview software package (Surgiview, Paris, France). Lumbar lordosis between L1 and S1 and pelvic incidence were measured for all patients. We analysed the sagittal spinal profile taking into account pelvic-hip relationships such as pelvic tilt and sacral slope (Fig. 1). In all cases, the deformity was progressive with worsening during the 18 months before surgical treatment. In all cases, lumbar hyperlordosis was preponderant and associated with paralytic thoracolumbar scoliosis in seven cases. In all the cases, the lumbar hyperlordosis was responsible for sitting difficulties which made wheelchair use impossible. In one case, the hyperlordosis was responsible for digestive disorders with occlusive syndrome.

We performed a statistical analysis of the data between patients surgically treated by a posterior approach only (group 1) and patients surgically treated by anterior and posterior approaches (group 2). Angular parameters were collected in a computerised database and analysed by the

Fig. 1 Methods of measurement of pelvic incidence, pelvic tilt and sacral slope

Table 1 Main epidemiological data of the series

Case	Age (years)	Gender	Aetiology	Neurological status	Evolutionary symptoms	Hip flexion contracture	Initial L1S1 lordosis (°)	Initial sacral slope (°)	Initial pelvic tilt (°)	Initial pelvic incidence (°)
1	13	F	Cerebral palsy	Spastic quadriplegia	Loss of sitting posture	30° left and 20° right	111	93	-32	61
2	17	M	Cerebral palsy	Spastic quadriplegia	Loss of sitting posture	60° bilateral	97	77	-36	41
3	16	M	Cerebral palsy	Spastic quadriplegia	Loss of sitting posture	50° left and 10° right	91	67	-41	26
4	14	F	Cerebral palsy	Spastic quadriplegia	Loss of sitting posture	30° left and 40° right	114	107	33	140
5	19	M	Cerebral palsy	Spastic quadriplegia	Loss of sitting posture	50° left and 70° right	120	97	-62	35
6	16	M	Cerebral palsy	Spastic quadriplegia	Loss of sitting posture	30° left and 40° right	86	62	-33	29
7	14	F	Cerebral palsy	Spastic quadriplegia	Loss of sitting posture	50° bilateral	108	89	-23	34
8	16	M	Congenital myopathy	Trunk hypotony	Loss of sitting posture	none	94	106	-54	52
9	17	M	Cerebral palsy	Spastic quadriplegia	Loss of sitting posture and digestive symptoms (occlusion)	40° bilateral	108	94	-60	35
10	15	F	Cerebral palsy	Spastic quadriplegia	Loss of sitting posture	50° left and 60° right	94	82	-16	66
11	17	M	Cerebral palsy	Spastic quadriplegia	Loss of sitting posture	45° to 50° bilateral	81	78	-21	57
12	19	M	Cerebral palsy	Spastic quadriplegia	Loss of sitting posture	80° bilateral	100	87	-54	32
13	14	F	Cerebral palsy	Spastic quadriplegia	Loss of sitting posture	none	110	95	-21	74
14	16	F	Occipital meningocele	Spastic quadriplegia	Loss of sitting posture	60° left and 45° right	132	90	-43	47
15	14	M	Cerebral palsy	Spastic quadriplegia	Loss of sitting posture	40° bilateral	115	88	-20	69
16	20	F	Cerebral palsy	Spastic quadriplegia	Loss of sitting posture	60° bilateral	94	77	-14	63
17	17	M	Cerebral palsy	Spastic quadriplegia	Loss of sitting posture	40° bilateral	127	102	-41	61
18	16	M	Myelomeningocele	Flaccid L3 paraplegia	Loss of sitting posture	30° left and 40° right	96	59	-5	54
19	18	M	Cerebral palsy	Spastic quadriplegia	Loss of sitting posture	30 to 45° bilateral	79	64	-24	41
20	13	M	Cerebral palsy	Spastic quadriplegia	Loss of sitting posture	80° right and	109	96	-17	79

Table 1 (continued)

Case	Age (years)	Gender	Aetiology	Neurological status	Evolutionary symptoms	Hip flexion contracture	Initial L1S1 lordosis (°)	Initial sacral slope (°)	Initial pelvic tilt (°)	Initial pelvic incidence (°)
21	14	M	Cerebral palsy	quadriplegia Spastic	Loss of sitting posture	75° left 30° left and 20° right	86	88	-1	87
22	15	F	Cerebral palsy	quadriplegia Spastic	Loss of sitting posture	40° bilateral	83	74	-9	65
23	13	M	Cerebral palsy	quadriplegia Spastic	Loss of sitting posture	40° left and 30° right	101	86	-29	58
24	22	M	Cerebral palsy	quadriplegia Spastic	Loss of sitting posture	30° bilateral	94	70	-53	17
25	19	F	Cerebral palsy	quadriplegia Spastic	Loss of sitting posture	25° left and 45° right	127	105	-53	52
26	27	M	Congenital myopathy	Trunk hypotony	Loss of sitting posture	30° left and 40° right	110	90	-43	40
27	14	F	Cerebral palsy	Spastic quadriplegia	Loss of sitting posture	30° bilateral	104	80	-29	46

SPSS software (SPSS Inc., Chicago, Illinois). We used a paired t-test for equality of means to compare preoperative and postoperative angular parameters and an unpaired t-test for equality of means to compare the results between group 1 and group 2. Differences with P values less than 0.05 were considered statistically significant.

Results

The studied cohort comprised seventeen males and ten females aged 13 to 27 years with an average age of 16.5 years. Neuromuscular disorder was cerebral palsy in 23 cases, congenital myopathy in two cases, lumbar meningocele in one case and occipital meningocele in one case. Patients with cerebral palsy presented a spastic quadriplegia in all the cases with various communicative and responsive statuses. The neurological status and main epidemiologic data of the studied cohort are summarised in Table 1.

The reported patients presented with a severe lumbar hyperlordosis which measured between 79° and 132° from L1 and S1 (Fig. 2). In 25 of 27 cases, bilateral hip flexion contractures of between 10° to 80° with asymmetrical contractures in twelve of 24 cases. In all cases, the sacrum was very horizontal, with sacral slopes from 59° to 107° while seated. Pelvic incidence values were relatively homogeneous in 26 cases and associated with pelvic anteversion with a “reversed” pelvic tilt between 1° and 62°. In one case, the pelvic incidence was 140° but the pelvic ring was not very tilted and pelvic tilt was only 33°. The detailed values of preoperative angular parameters are summarised in Table 1.

All patients were treated surgically by a posterior approach. In 16 cases, multiple-level lumbar discectomy and interbody fusion was performed through an anterior approach (thoracophrenolombotomy). In two cases (patients 11 and 12) an anterior osteosynthesis was done during the anterior approach. Except for one patient, the anterior and posterior procedure were performed separately, with a few days interval. Deformity correction was done with segmental instrumentation (CD Horizon-Medtronic Sofamor Danek Inc., TN, USA). Three patients were not felt to require sacral fixation because of a well balanced pelvis. Lumbar hyperlordosis correction was carried out using two rather different procedures. The first method used a small interval rod-hook construction placed at the apex of the curve. The small rod was approximated to the two principal rods by transverse traction devices (Fig. 3). This first technique was exclusively used in eight of 27 cases. The second method used pedicular screws placed at the apex of the curve and progressively pulled backward to reach the two principal rods (Fig. 4). This second technique

Fig. 2 Preoperative lateral radiograph in a patient presenting with a severe lumbar hyperlordosis measuring 111° between the caudal endplate of T11 and the cranial endplate of S1

was used exclusively in fourteen of 27 cases. The five remaining cases were treated by means of segmental instrumentation without a specific instrumented strategy. In all the cases, the frontal balance was obtained by

Fig. 3 The first surgical technique used to reduce lumbar hyperlordosis. A little distraction rod is placed at the apex of the lordosis (a) allowing reduction of the apical area. A principal rod is then placed between the sacrum and the thoracic spine. Distraction manoeuvres were made to correct sacral horizontal position (b). The small rod was then brought closer to the principal rod by means of the transverse traction devices (c). On each drawing, the levels concerned by the reduction procedure are indicated by the ® sign and the force applied by the arrows

Fig. 4 The second surgical technique used to reduce lumbar lordosis. A principal distraction rod is placed between the sacrum and the thoracic spine (a). Reduction of the residual hyperlordosis is made by pulling backward a long pedicular screw placed at the apex of the deformity (b). Other pedicular screws could be placed at the end of the procedure to improve the final correction (c). On each drawing, the levels concerned by the reduction procedure are indicated by the ® sign and the force applied by the arrows

distraction and compression manoeuvres applied to the spinal instrumentation. Fusion was carried out in all the cases with autologous bone graft. The postoperative course was uneventful in seventeen patients. One patient developed a thrombosis of the left common iliac vein secondary to Cockett's Syndrome (abnormal compression of this vein by the right common iliac artery) with uncomplicated resolution of the thrombus following treatment. Three patients presented a limited zone of skin necrosis in the lumbar area with favourable outcome. In one of the two cases, the skin necrosis was associated with superficial soft tissue infection. One patient presented a deep haematoma requiring surgical treatment. Two patients had deep infections requiring removal of implants in one case. One patient had a partial osteosynthesis failure secondary to a lumbar pseudarthrosis and responsible for a progressive frontal imbalance. The outcome was favourable after repeat osteosynthesis and bone grafting. One patient died from respiratory failure six weeks after the surgical procedure. The postoperative course was pejorative despite continuous artificial ventilation in the intensive care unit. The details of surgical procedures and complications are reported in Table 2.

Functional status improved in 26 patients. Sagittal balance and sitting posture were improved. Lumbar hyper-

Table 2 Details of surgical procedures and complications. Patients surgically treated by a single posterior approach are presented with a grey background

Case	Aetiology	Surgical treatment	Complications	Initial L1S1 lordosis (°)	Final L1S1 lordosis (°)	Final sacral slope (°)	Final pelvic tilt (°)	Final pelvic incidence (°)	Follow-up	Comments
1	Cerebral palsy	T9S1 posterior correction and fusion	none	111	62	43	6	49	1 year	none
2	Cerebral palsy	L1L5 anterior discectomy / T9L5 posterior correction and fusion	none	97	49	38	-2	36	5 years	none
3	Cerebral palsy	T9S1 posterior correction and fusion	Left common iliac vein thrombosis	91	65	54	-26	28	2 years	Favourable outcome after heparin therapy
4	Cerebral palsy	T3S1 posterior correction and fusion	none	114	79	84	35	119	1 year	none
5	Cerebral palsy	T8S1 posterior correction and fusion	none	120	90	69	-27	41	8 years	none
6	Cerebral palsy	L1L5 anterior discectomy / T9L5 posterior correction and fusion	none	86	39	30	12	43	14 years	none
7	Cerebral palsy	T10S1 posterior correction and fusion	none	108	70	60	2	62	15 years	none
8	Congenital myopathy	L1L5 anterior discectomy / T10S1 posterior correction and fusion	none	94	46	61	-34	28	12 years	none
9	Cerebral palsy	L1L5 anterior discectomy / T2S1 posterior correction and fusion	Early haematoma	108	57	49	-3	47	1 year	Favourable outcome after posterior debridement
10	Cerebral palsy	T12L5 anterior discectomy / T2S1 posterior correction and fusion	Late deep infection	94	47	40	24	64	5 years	Favourable outcome after posterior debridement, osteosynthesis removal and antibiotherapy
11	Cerebral palsy	T11S1 anterior discectomy with anterior osteosynthesis / T2S1 posterior correction and fusion	none	81	53	54	0	55	2 years	none
12	Cerebral palsy	T6S1 posterior correction and fusion / L1S1 anterior discectomy with anterior osteosynthesis	Early deep infection and muscular necrosis	100	51	53	-38	15	8 years	Favourable outcome after posterior debridement and antibiotherapy
13	Cerebral palsy	T1S1 posterior correction and fusion	Small area of skin necrosis	110	92	87	-17	70	3 years	Favourable outcome after local medical care
14	Occipital meningocele	L2L4 anterior discectomy / T2S1 posterior correction and fusion	none	132	97	76	-28	48	5 years	none

15	Cerebral palsy	T1S1 posterior correction and fusion	Small area of skin necrosis and superficial infection	115	76	62	-34	28	7 years	Favourable outcome after local medical care
16	Cerebral palsy	T1S1 posterior correction and fusion	none	94	79	64	2	66	4 years	none
17	Cerebral palsy	L1L5 anterior discectomy / T2S1 posterior correction and fusion	none	127	94	80	-9	72	11 years	none
18	Myelomeningocele	T1S1 posterior correction and fusion	Small area of skin necrosis	96	88	72	-18	54	5 years	Favourable outcome after local medical care
19	Cerebral palsy	T10L4 anterior discectomy / T2S1 posterior correction and fusion	none	79	33	38	-15	24	1 year	none
20	Cerebral palsy	L1L5 anterior discectomy / T2S1 posterior correction and fusion	none	109	79	70	15	85	3 years	Bilateral femoral extension osteotomies because of residual 65° hip flexion contracture
21	Cerebral palsy	T11L5 anterior discectomy / T2S1 posterior correction and fusion	none	86	76	90	-6	84	3 years	none
22	Cerebral palsy	T2S1 posterior correction and fusion	Osteosynthesis failure and lumbar pseudarthrosis	83	60	60	-17	43	5 years	Favourable outcome after repeat osteosynthesis and fusion
23	Cerebral palsy	T11L5 anterior discectomy / T1S1 posterior correction and fusion	none	101	83	86	-30	55	4 years	none
24	Cerebral palsy	L1L4 anterior discectomy / T2S1 posterior correction and fusion	none	94	68	74	-39	35	9 years	none
25	Cerebral palsy	L1L4 anterior discectomy / T2S1 posterior correction and fusion	none	127	50	44	-28	16	7 years	Left total hip replacement because of painful hip stiffness
26	Congenital myopathy	L1L4 anterior discectomy / T2S1 posterior correction and fusion	died	110	68	67	-12	38	none	Died from respiratory failure
27	Cerebral palsy	T9S1 posterior correction and fusion	none	104	67	58	-10	36	1 year	none

Table 3 Statistical comparison between preoperative and postoperative angular parameters (paired t-test)

Angular parameter variation (preoperative vs postoperative)	Mean	Standard deviation	t-test value	P value	Significance
Lumbar lordosis	35.29	15.16	12.09	<0.0001	s
Sacral slope	23.70	17.32	7.10	<0.0001	s
Pelvic tilt	-18.66	18.04	-5.37	<0.0001	s
Pelvic incidence	4.44	15.80	1.46	0.156	ns

s: significant

ns: non significant

lordosis was reduced by 8° to 77° between L1 and S1. In 21 cases, sacral slope was partially reduced with an improvement by 8° to 50°. In 20 cases, pelvic anteversion improved with an improvement of the pelvic tilt from 2° to 47°. In fifteen cases, pelvic incidence was modified significantly. In one case whose initial pelvic incidence was 140° and initial pelvic tilt 33°, modification of pelvic alignment reduced pelvic incidence to 21° associated with a reduction of sacral slope from 107° to 84°. The final values of the angular parameters are reported in Table 2.

The follow-up range varies from 1 to 15 years. All patients' functional status improved with a comfortable sitting posture and good sagittal balance. Comparison between preoperative and postoperative measurement showed a statistically significant variation of the lumbar lordosis, the sacral slope and pelvic tilt values; however, pelvic incidence was not modified significantly (Table 3). The comparison between patients surgically treated by posterior approach only (group 1) and patients treated by anterior and posterior approach (group 2) showed no difference between the two groups regarding preoperative

angular parameters and postoperative angular parameters improvement (Table 4).

Because of a severe residual hip flexion contracture, one patient underwent bilateral femoral flexion osteotomies with favourable outcome. In one case, a patient developed a stiff and painful hip and had a total hip replacement five years after spinal surgery.

Discussion

Treatment of spinal deformities from neuromuscular origin must obtain and maintain a well-balanced spine above a well-balanced pelvis in all planes. Treatment must be started as soon as a progressive imbalance is diagnosed, even after skeletal maturity [3]. Spinal deformities in cerebral palsy are commonly severe scoliosis associated with kyphosis and a pelvic obliquity [4, 5]. Isolated lumbar hyperlordosis is rare with only one documented case reported [6]. Cases with thoracic hyperlordosis are reported in association with idiopathic scoliosis [7]. Congenital

Table 4 Statistical comparison between group 1 and group 2 preoperative angular parameters and angular variations after surgical correction (unpaired t-test for equality of means)

Angular parameter	Group	n	Mean	Standard deviation	t-test value	P value	Significance
Preoperative lumbar lordosis	Group 1	11	104.18	11.62	0.457	0.652	ns
	Group 2	16	101.56	16.32			
Preoperative sacral slope	Group 1	11	84.18	14.21	-0.350	0.730	ns
	Group 2	16	86.06	13.40			
Preoperative pelvic tilt	Group 1	11	-20.27	23.72	2.027	0.053	ns
	Group 2	16	-36.12	17.00			
Preoperative pelvic incidence	Group 1	11	60.63	30.65	1.169	0.253	ns
	Group 2	16	49.62	18.35			
Lumbar lordosis improvement	Group 1	11	28.90	12.18	-1.905	0.068	ns
	Group 2	16	39.68	15.77			
Sacral slope variation	Group 1	11	19.36	15.67	-1.083	0.289	ns
	Group 2	16	26.68	18.25			
Pelvic tilt variation	Group 1	11	-10.81	18.15	1.976	0.059	ns
	Group 2	16	-24.06	16.38			
Pelvic incidence variation	Group 1	11	6.45	18.00	0.541	0.594	ns
	Group 2	16	3.06	14.55			

s: significant

ns: non significant

lumbar hyperlordosis and lumbar hyperlordosis secondary to lumboperitoneal shunts have been reported [8, 9]. In 22 of the 26 reported cases, lumbar hyperlordosis was secondary to cerebral palsy with spastic quadriplegia. Muscular hypertony of the *erector spinae* muscles could be responsible for the onset of the lumbar spine deformity. Hip flexion contractures play an associative and a causative role in the onset and evolution of lumbar hyperlordosis secondary to abnormal forces exerted between the lumbar spine and pelvis (Fig. 5). In severe lumbar hyperlordosis, we think that the *psoas* muscle could have a pejorative effect because its abnormal course between the spine and the proximal end of the femur changes its function. Instead of being flexor of the hip, it becomes a lumbar spine extensor (Fig. 6). Lumbar hyperlordosis in cerebral palsy seems to us to be related to the association of proximal (spinal) and distal (pelvic) causes. In cerebral palsy as well as in other neurological or muscular aetiologies, trunk collapse produces poor sitting posture and a typical clinical appearance (Fig. 7). In cases of severe hip flexion contracture, associated procedures such as soft tissues release or femoral extension osteotomy may be necessary to achieve the planned correction. In the twenty-six reported cases, spinal correction alone achieved a good sitting posture but one patient required bilateral femoral extension osteotomies to reduce severe hip flexion contracture.

Analysis of pelvic position and modifications of lumbar-pelvic sagittal alignment is difficult, but a fundamental part

Fig. 5 Hip flexion contracture (a) could be responsible for the onset of lumbar hyperlordosis due to constraints exerted on the lumbar spine (b)

Fig. 6 The two possible actions of the psoas muscle. In normal condition (a), it acts as a hip flexor. In cases of severe hyperlordosis (b), it can become lumbar spine extensor

of preoperative assessment. In a vast majority of cases, the lumbar hyperlordosis is responsible for pelvic “anteversion”, i.e. anterior flexion of the pelvic ring. Although analysis was done of lateral radiographs with the patient supine or sitting and not standing, pelvic “anteversion” was well visualised and pelvic tilt appeared reversed with a negative value. Sacral slope was high, sometimes $>90^\circ$ with a horizontal sacrum. In one case, there was a dissociation of the lumbar-pelvic sagittal balance with lumbar hyperlordosis responsible for a very horizontally positioned sacrum with a sacral slope of 107° . The pelvic “anteversion” in this case was related only to the sacrum and not the ilium (Fig. 8). Pelvic tilt measured 33° and lateral radiographs demonstrated the dissociation between a very horizontal sacrum and the rather “physiological” position of the iliac wings and the ischii. Our analysis of this lumbar-

Fig. 7 Clinical preoperative (a) and postoperative (b) aspect of a patient presenting with a severe lumbar hyperlordosis. Note the preoperative lumbar spine “break” aspect

Fig. 8 In the vast majority of the cases, lumbar hyperlordosis was responsible for an anteverged pelvic ring (a). In one case (patient 22) we noted a dissociation between a very horizontal sacrum and the rather “physiological” position of the iliac wings and the ischia (b)

pelvic disorder was that there was rotation of the sacrum inside the pelvic ring through the sacroiliac joints. Frontal pelvic radiograph showed widening of the sacroiliac articular space. This progressively increased mobility of the sacroiliac joint was responsible for the increased pelvic incidence which measured 140° (Fig. 9a).

Our surgical strategy required analysis of the hyperkyphotic flexibility which was very difficult to assess. In many cases, the flexibility is poor because of the posterior muscles’ stiffness and spasticity. In these cases, it seemed necessary to us to perform preliminary anterior disc excisions. However, we think that extensive posterior soft-tissues release including the muscular and ligaments attachments as well as the *ligamentum flavum* between each posterior arch could provide the necessary correction and avoid the demanding anterior procedure. The advantage of a single posterior surgical procedure is to minimise the respiratory consequences of a long surgical procedure. Especially in cases of preoperative muscular impairment as in myopathy or in adult patients, phrenotomy could be responsible for severe respiratory dysfunction (as in case 26). The final correction obtained in eleven cases by the posterior approach only was satisfactory and allowed reduction of the horizontal sacral and lumbar lordosis which enabled patients to sit comfortably. The difference between combined anterior and posterior procedures and the posterior procedure alone was not statistically significant in terms of correction. Of course, the advantage of the combined anterior and posterior approach is to perform circumferential bone grafting. We experienced one case of lumbar pseudarthrosis after a single posterior approach but

Fig. 9 **a** The progressive motion of the sacroiliac joint in patient 22 was responsible for a pelvic incidence measuring 140° contrasting with rather normal pelvic tilt. **b** Pelvic incidence was decreased by 21° after surgical correction of lumbar hyperlordosis without significant modification of the pelvic tilt

the outcome was good after repeat osteosynthesis and bone grafting by the posterior approach.

It is important to appreciate the spine-pelvis interaction with all segments of the pelvis. In eleven of our cases, unstable sacroiliac joints caused pelvic incidence variations of 11° to 40° after surgical correction of lumbar hyperlordosis. In the case with preoperative dissociation between a very horizontal sacrum and the rather “physiological”

position of the iliac wings and the ischia (Fig. 9b), it would have been incorrect to completely improve the sacral orientation because that would have caused iliac and ischial retroversion and loss of the ischia’s role in sitting. This case demonstrated the importance of preoperative analysis of spinal-pelvic balance to establish an appropriate surgical strategy to correct the severe lumbar hyperlordosis.

Acknowledgement The authors gratefully acknowledge the assistance provided by Dr. Carl Stanitski in the preparation of this manuscript.

References

1. Eicher PS, Batshaw ML (1993) Cerebral palsy. *Pediatr Clin North Am* 40:537–551
2. Ferguson RL, Allen BL Jr (1988) Considerations in the treatment of cerebral palsy patients with spinal deformities. *Orthop Clin North Am* 19:419–425
3. Thometz JG, Simon SR (1988) Progression of scoliosis after skeletal maturity in institutionalized adults who have cerebral palsy. *J Bone Joint Surg Am* 70:1290–1296
4. McCarthy RE, Bruffett WL, McCullough FL (1999) S rod fixation to the sacrum in patients with neuromuscular spinal deformities. *Clin Orthop* 26–31
5. Rinsky LA (1990) Surgery of spinal deformity in cerebral palsy. Twelve years in the evolution of scoliosis management. *Clin Orthop* 100–109
6. Song EW, Lenke LG, Schoenecker PL (2000) Isolated thoracolumbar and lumbar hyperlordosis in a patient with cerebral palsy. *J Spinal Disord* 13:455–460
7. Bradford DS, Blatt JM, Rasp FL (1983) Surgical management of severe thoracic lordosis. A new technique to restore normal kyphosis. *Spine* 8:420–428
8. McIvor J, Krajchich JJ, Hoffman H (1988) Orthopaedic complications of lumboperitoneal shunts. *J Pediatr Orthop* 8:687–689
9. Steel HH, Adams DJ (1972) Hyperlordosis caused by the lumboperitoneal shunt procedure for hydrocephalus. *J Bone Joint Surg Am* 54:1537–1542